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CHEMICAL AND BIOLOGICAL WARFARE

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The London Conference on CBW
held under the auspices of the
J. D. Bernal Peace Library

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CBW

CHEMICAL AND BIOLOGICAL WARFARE

London Conference on CBW

edited by

STEVEN ROSE

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FOREWORD

This book is based on the collection of papers presented to the Conference on Chemical and Biological Warfare which was held in the Bonnington Hotel, London, on February 22nd and 23rd, 1968. The aim of the Conference was to provide an authentic assessment of the present level of development of chemical and biological weapons, their manner of use in war, and the possibilities of defence against them; to consider the status of such weapons in international law; and to discuss ethical problems, especially the responsibility of the scientist, in relation to their development and use.

The Conference was the first major event to be sponsored by the J. D. Bernal Peace Library, an educational trust set up to collect source material and to provide information of assistance in the struggle for peace and toward ensuring the realization of the fullest potentialities of science in building a world prosperous and without war—aims toward which J. D. Bernal has made a unique contribution.

The Conference took place against a background of the use of defoliants and so-called non-toxic chemical weapons in Vietnam and the reported use of more lethal gases in the Yemen. The discussions at the Conference indicated the practicability of the renunciation of CBW by all the major powers at a time when there is still no major investment of money and personnel in this field. It is in the hope that the spread of factual knowledge about the nature and menace of these weapons will further the campaign for their elimination and for more complete disarmament that these papers are being published.

E. H. S. BURHOP

LONDON
May, 1968

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PREFACE

The aim of the Conference whose proceedings form this book was a clear one. While we were concerned to amass as substantial a body of information as possible on current developments in chemical and biological warfare, our object was primarily to help inform opinion and stimulate discussion on the political, military, legal, and moral implications of the research, and on development, stockpiling, and use of chemical and biological weapons in a period of nuclear stalemate when it appears military strategists are placing increasing weight on the potential of these weapons. We attempted —and I have attempted as editor of this book—to separate the presentation of the facts about CBW as a potential and actual war technology, from our assessment of its implications. Such a distinction is of course at best artificial, and its artificiality will be apparent to the reader. It is not really possible to isolate a weapon or a technology from the political or moral climate in which it is made or used. None the less, in military and legal terms, and in the popular imagination, the chemical and biological weapons, like nuclear weapons, have been so isolated, presenting a separate class of problems.

In the Conference, each of these several themes was dealt with in a separate paper, and discussion—often lengthy—followed. In converting the conference texts into book form, I have omitted some few of the papers which seemed to me to be too specialized for a general readership, have slightly edited the individual papers to reduce them in length and to impose a certain uniformity in presentation, and have ruthlessly pruned the discussions which followed the papers, either incorporating points wholesale into the texts, or summarizing the essential themes as they appeared to me at the time or on rereading the transcripts afterwards. All the participants have cheerfully acceded to this procedure, and I must thank them for their participation. I can only hope that the resultant book is readable without losing completely the flavour of a Conference with its implied differences of viewpoint and of personality.

In the task of both planning the Conference and turning its proceedings into a literate and readable text, the Conference committee, chaired by Professor Eric Burhop and including Mr Bill Carritt, Mr Robin Clarke, Dr John Humphrey and Dr Vic Sidel, has provided consistent support; Mr David Pavett has undertaken the major tasks of checking manuscripts and references; and the services provided by Mr Colin Sweet and the Bernal Library have immeasurably lightened the load. I must particularly thank Mr Bert Ede, who recorded our proceedings, and Mrs Margaret Stern, Mrs Anita Taylor, and Mrs Naomi Wolff, who typed and transcribed against an ever-pressing time limit. Mr. Peter Sommer, of Harrap's, has most efficiently handled the task of getting a book which was being almost continuously updated even as it passed through the printer's hands so rapidly into press.

Because of the rapidity with which events have moved even in the few months since the Conference, and the amount of further information that has become available, it has proved possible and necessary to revise some of the papers; statements that were merely guesses in February—particularly about the activities of Porton—can now be documented accurately. This revision has been done where possible without affecting the format of the individual papers by means of editorial insertions. In one or two cases, especially Chapter 9, it has seemed proper to insert substantial additional material. I must take full editorial responsibility for any errors, of fact or interpretation, that these changes may have produced.

STEVEN ROSE

LONDON
August 1968

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The '8-ball' aerosol test sphere.

6,000 sheep accidentally destroyed by nerve gas.
Fort Detrick, Maryland.

Chemical Defence Experimental Establishment, Porton Down.

CBW in Use

Grenade of CS.

Gas-mask

The 'Mighty Mite'

A USAF C-123 *Provider* on a defoliation mission.

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INTRODUCTION

Lord Ritchie-Calder

It is most appropriate that this Conference should be one of the initial activities of the J. D. Bernal Peace Library. In fact I can say: "This is where I came in . . ."

As my MI5 record shows, over thirty years ago, I was involved with Desmond Bernal and a group of scientists who were exposing the near-cynicism of the Air Raid Precautions. We had managed to get hold of one of the gas-masks which were to be issued in the event of war. I was writing about it, but I was also addressing meetings, tailed by a faithful shadow from the Special Branch. One of the things which I was showing was that the charcoal snout was no protection against arsenical smokes. I stood in front of a screen, had a spotlight thrown on me, and filled my mouth with tobacco smoke and put on the gas-mask. I then breathed out through the canister, and the audience could see the smoke curling up. The then Home Secretary, Sir Samuel Hoare, took a poor view of these activities and he denounced me in the House of Commons. He said arsenical smokes would only make one sick. Whereupon I challenged him to go into a gas-chamber and have the experience of being sick in a gas-mask! But the result was that when the millions of civilian gas-masks were issued on the outbreak of war they all had anti-smoke wafers strapped on to the snouts with sticking-plaster.

That was kindergarten stuff compared with the situation today, but security is still as childish. There has been a conspiracy of silence about these activities which are as fraught with mischief and ultimate peril as the development of nuclear armaments. The Doomsday Bug is under wraps!

The conspiracy is not confined to military security. Some scientists themselves shy away from the implications. One knows of distinguished scientists who can be as forthright as the rest in denouncing nuclear weapons but will avoid the subject of chemi-

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cal and biological warfare, especially biological warfare. It is quite irrational. Although they may not be dealing with official secrets and their own academic thinking is not classified, they can only guess, with uncomfortable perception, at the military applications. It is a psychological inhibition. They simply do not want to believe that their fundamental work can be perverted.

Perhaps they are preserving their innocence, like the schoolboy who said he was going to be a biologist: "Physics is bombs. Chemistry's insecticides. Biology is about life." Perhaps they do not want to believe it is about death.

This is another crisis of the scientific conscience. Between the Wars Sir Richard Gregory, at the British Association for the Advancement of Science, pointed out that in a chemical dictionary mustard gas and chloroform were on the same page—the malevolent and the benevolent. But the chemist who discovered mustard gas was just as guiltless as the chemist who discovered chloroform; others took the results of science and perverted them. That was relatively true until the nuclear physicists took the initiative over the bomb. It was they—including Einstein, the lifelong pacifist—who impressed upon the policy-makers and the generals the possibilities of turning the laboratory discovery by Hahn and Strassman into a cataclysmic weapon. And the physicists were left with that guilt-sense.

But the same sort of thing was also happening in biology during the War. It has been said that agricultural science has produced an innocent weed-killer which has been transformed into a defoliant. But from my wartime experience I recognized the post-war commercial weed-killer as one of the secret weapons which wartime scientists had dreamed up as anti-crop warfare.

Certainly scientists enlisted for war were not overburdened with scruples. Perhaps the Allied scientists did not lend themselves as the Germans did, to putting people into gas-chambers or using concentration-camp victims for trying out nerve gases and new diseases, but they 'went along' with biological warfare. One of the principals in Canadian Chemical and Biological Warfare revealed in 1949 that "one bacteriological weapon developed late in the second World War could wipe out all human life in a given area within six hours and yet leave the area habitable afterwards. . . . While it kills everybody in six hours, it itself is oxidized in twelve hours, leaving the ground perfectly safe to occupy. . . ."[1]

In fact, it was *botulinus* toxin that was being described. Botulin became part of a double bluff during the War. Supplies existed

and might have been used, but only if the Germans had first used something of the same—a principle affirmed by both Churchill and Roosevelt in relation to CBW. So we contrived to 'serve notice' on the Germans. Certain Canadian servicemen were briefed that if taken prisoner they would, in addition to name, rank, and number, reluctantly disclose that they had been vaccinated against botulin. A dicey game of poker! It could have meant (as we intended it to) "Don't do anything rash, chum, or else. . . ." Or it might have provoked a pre-emptive strike, and we would have been in a chemical and biological war.

By that time, we were nervous about CBW. Hitler's major secret weapons—the V1 and V2—had failed, and he was being pushed towards that ultimate culmination in the Berlin bunker. Gas had not been used by either side, but it certainly existed—a fact of which the disaster to the gas-supply ship in Bari was a reminder. Another was when a German shell hit an Allied gas-shell dump in the Anzio bridgehead and the gas started drifting towards the German lines. The Allied commander had to use the hot-line technique to his opposite number saying it was an accident and there was no intention to use gas. We may have had to wait until our scientific search parties went into defeated Germany to discover such refinements as the anti-cholinesterases *sarin, tabun,* and *soman,* but even allowing for any imprecisions of our espionage system, we knew they had them. Why? Because, of course, we had similar chemical weapons. That is how one works in CBW—seeing the other side's potential in the mirror of one's own. What we feared was that Hitler, once he had decided that the German people were not worthy of him, would unleash a CBW *Gotterdamerung.* Then, apart from gas-destruction, we might have had a modern Black Death.

Frederic Joliot-Curie suggested 'The Secret War' over twenty years ago. He extended the example of nuclear security to biological security. Behind the sky-high walls of secrecy the scientists had produced the Bomb. They had condoned the secrecy, so that it became the physicists' Bigger and Better Bomb, without any account being taken of the biological effects of radiation, without any of the premonitions, safeguards, and interdisciplinary reminders that come from free scientific exchanges—and without any sanctions on its use which proper information exerts. One recalls Attlee's statement, as one who had concurred in the dropping of the bomb on Hiroshima: "We knew nothing whatever at that time about the genetic effects of an atomic explosion. I knew nothing at all

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about fall-out and all the rest that emerged after Hiroshima. As far as I know, President Truman and Winston Churchill knew nothing of these things either, nor did Sir John Anderson who co-ordinated research on our side. Whether the scientists directly concerned knew, or guessed, I do not know. But if they did, then so far as I know they said nothing of it to those who had to make the decision." And in the entire documentation of the British atomic energy effort there was only one brief paper (by the Medical Research Council) on genetic effects, and that apropos not of the bomb but of the use of radioactive elements as a form of biological warfare.[2]

What Joliot-Curie did was to extend the lessons to biology. He pointed out that such a war need never be declared. It could go on for years with only the hidden aggressors knowing what was happening. Then, gradually, successive crop failures, devastation of herds by disease, human epidemics (unexplained because the organisms weren't in the books), and a catastrophic fall in the birthrate, through the sterilization of women by tampering with the water supply, would reveal the truth.

What Joliot-Curie was arguing for was a full scientific disclosure to alert mankind. But in the 'Through-the-looking-glass' world of military expediency the same arguments are used to justify secrecy —Don't put ideas into people's heads!

Thus 'defence' becomes building up stockpiles of chemical and biological weapons. If the Microbiological Research Establishment at Porton is no more than a means of protecting us against threatened diseases, why is it not under the Ministry of Health? If we pursue this further, and we must, we shall find, as the Americans found for their own institutions, that there is a whole network of relationships between Porton and the universities and research institutions, contracts and grants ostensibly for academic research with the right to publish—but not if the Ministry of Defence chooses to classify it! (This prophecy has now proved correct. *Ed.*)

The issues involved are not only the ultimate threat of world war with no holds barred—the ABC war, Atomic, Biological, Chemical—but the corrosion of our attitudes to evil. I return again to the censorship, self-imposed, by the scientists who 'just don't want to think about it.' Many of them, unless they are bereft of imagination, must know that their fundamental work, their 'pure' science, is being corrupted in this way. They knew the implications of DNA, molecular biology, gene manipulation, of their basic chemistry, and they must know also that intensive work on the

practical applications of such work is going on in 'defence' establishments. Even if they are not participating, they are accessories before the fact, and they must be frank about this.

But we cannot be too hard on them when those who are concerned with peace and war also duck this issue. One may say "Oh, Lord, I've enough to worry about without *that*!" but while we are wrestling with non-proliferation and nuclear restraints—very important it is true—no systematic work towards CBW control is proceeding. Pious resolutions, yes, but that is where it stops. It belongs to that tomorrow when we shall get agreements on general and complete disarmament.

Meanwhile, as they say in the United States, "C.B.W. ought to have better public relations". What they mean by that is that people should be taught to approve of napalm, defoliation, 'police-gas', and so on. This aims to produce the acquiescence of ignorance, which leads to escalation and the legitimization of the next stage—the point where the military can do what they like. It is an easy progression from justifying the defoliation of jungle so as to deprive guerrillas of cover to 'treating' hundreds of thousands of acres of rice-crops to deprive guerrillas—and the people—of food. And to awarding the highest U.S. civilian medal to a woman scientist who discovered a better riceblast fungus —more effective, that is, than the natural disease which has in the past destroyed Asian rice-crops and killed millions by famine.

We get bland terms like 'humane' and 'anti-bellicose' gases, which include two categories which, in U.S. Army Chemical Corps circles, are jocosely called 'off the rocker' and 'on the floor'—a homely way of describing psychochemicals and paralysers. And if they won't stay on the floor there are the proper lethal gases too.

We have had conventions. At least the Americans are 'legal'; they have not signed the Geneva Protocol. But Britain, Australia, and Canada are signatories, although they exercise the reservation on the right of reciprocity. If one is going to reciprocate one must have something to reciprocate with, and thus one justifies peacetime research and stockpiling. And Britain, Australia, and Canada have a quadripartite agreement with the United States.

But surely the greatest conscience casualty is science itself. While one group of scientists are bending their energies to prevent diseases, another is devising man-made epidemics. While one set is discarding chemicals saying "We dare not use that," another is picking them out of the trash-can and saying "You bet we can!"

This is the Jekyll and Hyde of science.

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PART ONE

THE WEAPONS

1	Chemical Weapons	J. Perry Robinson
2	Psychedelics	C. R. B. Joyce
3	Napalm	V. W. Sidel
4	Biological Weapons	I. Màlek
5	Defoliants	A. W. Galston
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1. Chemical Weapons

J. Perry Robinson

Most of the weapons used today are chemical. The explosion of TNT is a chemical reaction, so is the combustion of napalm. But we are concerned here with those weapons which are based on the toxic properties of chemicals rather than on the energetics of their interaction. In arming ourselves with them we are following the lead of the humble ant or snake.

A weapon-system based on toxic chemicals may be looked at as the sum of four parts: (i) a system to deliver the munitions; (ii) munitions to disseminate the chemical agent; (iii) the agent itself; and (iv) the part played by the environment in transporting the disseminated chemical to each target individual. It must be stressed that each target is an individual, and that the weapons we are talking about are purely anti-personnel.

Each of the four parts is dependent to a greater or lesser extent on the other parts. If an attacker is relying on the atmosphere to transport the agent to the target individual's lungs, the agent chosen must be one which can be made airborne in a form which will penetrate the lungs. If the chosen agent is one which is sensitive to heat, then the chosen munition must avoid it. This interdependence must be kept in mind in what follows.

The available chemical agents

In 1956, Edgewood Arsenal, the U.S. chemical warfare research centre, published a four-volume *Report on Properties of War Gases.*[1] This report, which presumably corresponds to the 'Red Book' of the British CW station at Porton, is classified secret, but the assumption is that it contains collected data on all chemicals likely to be of any use in chemical warfare. The titles of the volumes are *G-agents, Blood and Nettle Gases, Vomiting and Choking Gases, and Lachrymators,* and *Vesicants.*

I shall describe two or three of the chemical agents which can be assumed to be in each volume, and also mention known agents

which would now be placed in supplementary volumes, notably the V-agents and the incapacitating agents.

VOMITING GASES AND LACHRYMATORS. In the training manuals these are classified as riot-control or harassing agents. They are irritants[2, 3] and are lethal only in very high concentrations. Some are particularly irritating to the eyes (lachrymators), some to the inner surfaces of the nose and upper breathing tubes (sternutators), some to the skin (orticants), and some, if swallowed, to the upper reaches of the alimentary canal (vomiting agents). The body attempts to counter the irritation either by secreting fluids or by initiating reflex actions, for instance, vomiting, or the desire to scratch. It is the incapacitating nature of these responses which gives the agents concerned their harassing effect and their common names. In general any one irritant will provoke all of these responses to a greater or lesser extent.

Nowadays, the principal requirements made of these agents are an immediate action, a lack of persistency in the field, and a brief duration of effect. Those agents which can be disseminated as smokes (inevitably non-persistent) are thus preferred. The principal one is *ortho-chlorobenzalmalononitrile*, known as CS. In order of occurrence its effects are quoted as "extreme burning sensation of the eyes and copious flow of tears; coughing; difficult breathing and chest tightness; involuntary closing of the eyes plus sinus and nasal drip; nausea and vomiting."[4] In the words of the British Government's patent on its use[5], "A concentration of between one part in ten million and one part in a million is enough to drive all but the most determined persons out of it within a few seconds". That is to say, it is instantly effective at field concentrations between 1 and 8 thousandths of a gram per cubic metre.

CS is more effective than the older agent CN (*chloroacetophenone*) in that it supplements the latter's lachrymatory action with a more severe skin irritation, and is more stable on storage. Its effects are shorter lived and quicker starting than those of the other preferred agent, DM or adamsite. The agent known as CNS[6] is a pre-war formulation of CN which has increased persistency in the field. It contains chloroacetophenone and the vomiting agent *chloropicrin* dissolved in chloroform.[7, 8, 9] A lengthy exposure to these agents is exceedingly unpleasant and painful. Soldiers have been driven mad by the pain. Some have tried to kill themselves to avoid it.[10]

CHOKING GASES. The choking gases are those which act by irritating the lower reaches of the breathing apparatus. Far from being

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merely harassing, their irritancy is sufficient to destroy the more delicate membranes of the lungs or respiratory passages. On the one hand, this may mean that the body's defences against invading micro-organisms are breached; on the other, it may lead to a total blockage of oxygen uptake and hence death either from pneumonia or bronchitis, or from asphyxia. The symptoms are prolonged and excruciating; the final crisis may be delayed for several hours.

The choking gases are the classical agents of chemical warfare. They include *chlorine, phosgene, trichloromethyl chloroformate,* and more recently *disulphur decafluoride.*[11] They are unlikely to be used in a modern chemical war, for their initial irritancy or smell immediately warns of their presence, and gas-masks can be donned before a lethal exposure. In addition, their toxicity is nowadays too low. The lethal exposure to phosgene is around 3200 mg-min/m^3 [12] (a measure of dose and time of exposure combined; e.g., of 1 mg/m^3 for 3200 minutes, or of 3200 mg/m^3 for 1 min, or any intermediate combination. *see* Glossary).

NETTLE GASES. The nettle gases are primarily skin irritants, but more painful than those we have already discussed. An exposure to one of them has been compared to being thrown naked into a bed of stinging nettles, hence their name. They were first seriously studied in the 1930's by the Germans, who sometimes referred to them as "Red Cross" agents.

A typical nettle gas is *dichloroformoxime.* The Russians are said to have stockpiled it during World War II.[13] At low concentrations it is a typical skin irritant and lachrymator, but at higher concentrations it blisters the skin and penetrates it to enter the blood circulation. From then on its effects may be lethal; it will reach the lungs and cause pulmonary oedema (swelling due to uptake of water), characteristic of phosgene to which it is chemically related. A 1955 report[14] from Edgewood Arsenal quotes a lethal toxicity in guinea pigs of 25 mg. per kg. body-weight when placed on the skin.[15] Its combination of highly painful skin attack with the possibility of a lingering death by asphyxiation undoubtedly makes it one of the nastiest agents available.

There is probably no tactical need at present for the nettle gases. Lethal agents must nowadays be imperceptible and therefore non-irritating, while riot-control agents must be non-lethal.

BLOOD GASES. Dichloroformoxime can act as a circulatory poison if it enters the bloodstream, and this action is a special characteris-

tic of the blood gases. These are principally intended for inhalation in the field; once inhaled, they are rapidly absorbed into the blood circulation where in very small doses they can completely block oxygen circulation around the body. To quote the long-out-of-print British official Medical Manual of Chemical Warfare,[16] they "may cause death with dramatic rapidity".

The principal blood gases are *hydrogen cyanide* and *cyanogen chloride* which replaced phosgene in certain types of munition in the U.S. arsenals during World War II.[17] The Russians managed to compound hydrogen cyanide into a form which could be successfully sprayed from aircraft, a considerable achievement.[18] The blood gases are not quite as lethal as phosgene, but are far quicker acting and somewhat harder to protect against.[12]

VESICANTS. These agents injure every sort of body tissue with which they come into contact. Their principal targets are the skin and the eyes, which they burn and blister. Because they are not very volatile substances they persist in the field for long periods and can be effective for days or weeks after dissemination. If they are used in sufficiently high concentration lethal doses may be absorbed through the skin. If they can be made sufficiently airborne lethal doses may also be absorbed through the lungs. In fact their inhalation toxicities are generally much higher than those of the other lethal agents which have so far been described.

There are two main classes of vesicant—the *arsenicals* and the *mustards*. The arsenicals, such as *Lewisite* and the other dichloroarsines have sharp, irritating odours and cause immediate eye pains.[19] In contrast the mustards have practically no smell and cause no initial pain or irritation. None the less, their damage is done within a few minutes of exposure, and after three or four hours the victim may be blinded or so blistered as to be incapable of action. Although the arsenicals give enough warning of their presence for protective clothing to be donned in time, the mustards do not, and it is for this reason that they are still in the arsenals fifty years after their introduction.

There are several different mustards, ranging from the comparatively volatile *bis(β-chloroethyl) sulphide* or *tris(β-chloroethyl) amine* to the highly persistent *α,ω-bis(β-chloroethylthio)alkanes*.[20] One of the most dangerous is the agent Q, which will burn or blind at an exposure of less than 50 mg-min/m^3, and kill if inhaled (as an aerosol for it is a solid) at dosages of 200 mg-min/m^3. Although its tactical use would probably be for skin attack its

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inhalation toxicity approaches that of the older nerve gases. In the words of an Edgewood Arsenal report[21] its lung action is "a bonus effect".

G-AGENTS. The first nerve gases, or G-agents, were developed in secret by the German chemical industry shortly before and during World War II. They are quick-kill agents of tremendous potency. Some, like *tabun*, are persistent in the field. Others, like *sarin*, are non-persistent. They reduced bomb-loads to one-twentieth or less of what was previously necessary. April, 1942, when tabun first went into large-scale production,[22] was an even more important date in chemical warfare than July, 1917, when mustard shell were first fired. The very largest targets were now vulnerable to chemical attack.

The nerve gases are *anti-cholinesterase* agents, working by blocking the enzyme which the body uses to destroy one of its chemical nerve signal transmitters after it has done its job. This has two effects. One is that control is lost over the affected part of the nervous system. The other is that a large concentration of the chemical transmitter rapidly builds up within the body, and that chemical is itself a powerful poison. The body is first incapacitated, and then forced to poison itself.

The symptoms of nerve-gas poisoning are diverse and spectacular. In a comparatively inactive man an exposure to sarin of 15 mg-min/m^3 dims the vision, the eyes hurt and become hard to focus. This may last for a week or more. At 40 mg-min/m^3, the chest feels tight, breathing is difficult, there is coughing, drooling at the mouth, nausea, heartburn, and a twitching of the muscles. At 55, there is a strangling tightness and aching of the chest, vomiting, cramps, tremors, and involuntary defaecation and urination. At 70, severe convulsions will set in followed closely by collapse, paralysis, and death.[23]

Of the better known G-agents tabun is about half as toxic as sarin, and *soman* about twice as toxic.[24] It is a moot point whether tabun is still considered worth stockpiling. Its toxicity is not as high as the other G-agents, but it has a persistency in the field which may be considered tactically useful, that is to say, midway between sarin and the V-agents, so that it can presumably provide a vapour hazard for some days after dissemination. Again, although it is more quickly destroyed by moisture than are the other G-agents, the resultant hydrolysis product, hydrogen cyanide, is highly poisonous, as is the cyanogen chloride formed when it is treated with

decontaminating solutions. In addition, its casualties are rather less amenable to the use of the oxime-type antidotes than those of sarin,[25] but equally there are other G-agents, such as soman[25] and *cyclohexyl methylphosphonofluoridate* (GF)[26] which are even harder to counter.

The Germans stockpiled tabun rather than sarin because they found it easier to make. The final stage in their large-scale process for sarin demanded high fluoride concentrations, and even with elaborate silver-lined apparatus[27] they could not get round the corrosion problems. Although they made some hundreds of tons of its intermediates, they could produce no more than half a ton of sarin itself,[22] as compared with the 12,000 or so tons of tabun.[28] The Americans at least were later able to solve the corrosion problems, and have been making sarin ever since. In 1954 the amount of the substance *phosphoryl chloride,* produced as a by-product, began to prove a severe commercial embarrassment.[29]

NERVE GASES. In some circumstances, lethal doses of G-agents can be absorbed speedily by the skin from airborne field concentrations. Since World War II, new nerve gases have been developed which are absorbed through the skin faster and more reliably than the G-agents.

The new nerve gases are known to the Americans as V-agents. Although they have not yet officially disclosed the chemical structures, it is a reasonable assumption that the V-agents are the *S-dialkylaminoethyl alkyl methylphosphonothiolates.* If this is so then it is interesting to note that the first openly published[30] percutaneous toxicity figures for them were given by Academician M. M. Dubinin, a staff member of the Karpov Institute in Moscow.

The Swedish Ministry of Defence states that between two and ten mg. of F-gas, presumably another name for V-agent, are fatal when placed on a man's skin.[24] Such a dose is so small as to be almost invisible.[31] Their inhalation toxicity is given as about 5 times that of soman.[24] Thus the V-agents are about 2000 times as toxic by skin absorption as mustard gas, and about 300 times as toxic through the lungs.

The V-agents are reputedly a British discovery. Certainly it was ICI who in 1955 applied for a patent[32] to cover the phosphonothiolates mentioned earlier. They thereby prevented Bayer from getting one on their rather later application.[33] One of the inventors named in the Bayer application was the same Gerhard Schrader who was responsible for the nerve gases in the first place.[34]

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INCAPACITATING AGENTS. Most people have heard of the 'psychogases'; some have seen the film of the cat cowering before a mouse under the influence of one—LSD in that particular film. The idea behind such agents is to put the enemy out of action for several hours without permanently harming him, preferably in such a way that he needs no medical attention. Such agents have long been the ideal of chemical warfare theorists; within a few months of the first German chlorine attack in 1915 the specification for a British patent was filed on the idea.[39] Parts of it read very much like a Chemical Corps press release of the late 1950's. The pharmaceutical industry has indeed produced a great range of compounds which have such effects.[40] But the chemical warfare laboratories demand that they act at very small dosages, exposures of less than 100 mg-min/m^3.[41] If a drug is as powerful as this it will almost certainly be lethal at higher exposures, and no field commander is going to make any great effort to avoid over-hitting a target. Thus it seems most likely that the purely incapacitating agent is more a weapon of public relations than of war.

None the less, the Americans are expending considerable effort in pursuing this particular ideal, both inside the defence laboratories and in those of contractees. Among the earliest of the outside contracts must be that with the Shell Development Company, dating from 1952, which called for an examination of synthetic cannabis derivatives for both incapacitating and lethal properties.[42]

Incapacitation can be brought about by interference with either the mind or the body. Mental incapacitation, particularly by the hallucinogens, has been more in the public eye, so much so that the terms 'incapacitating agent' and 'psychogas' are regarded as synonomous. But for the reasons discussed in the next chapter militarily useful incapacitants are more likely to be found among the body-incapacitating agents—the *paralysants*, for denying movement, the *hypotensives*, for causing fainting, the *emetics*, the *convulsants*, the *laxatives*, and so on, perhaps supplemented by mental derangement. It looks as though the Americans' recently standardized incapacitating agent BZ, upon whose use there are "critical limitations,"[43] falls within this class. Its symptoms include a slowing of mental and physical functions, dizziness, disorientation, and hallucinations, coupled with skin flushing, a speeding up of the heart-beat, urinary retention, and constipation.[43] These seem to be very similar to the effects of certain of the anti-cholinergic drugs,[44] and it may be that BZ came to light during the search for nerve-gas antidotes related to atropine. It may even be such an antidote. How convenient that

would be. Guesses at its structure should probably include the *3-quinuclidinyl phenyl glycollates.*

CURRENT DEVELOPMENTS. Before leaving the chemical agents we should indicate one or two lines of research into them which are at present being followed.

One is the use of mixtures of chemical agents with either physiologically active ingredients or inert ones. In the former category come those mixtures in which the toxicity of the main component is increased so that the lethal dose of the mixture is lower than the sum of the lethal doses of the components.[46] This is a well-known technique in the formulation of insecticides. Another possibility is a mixture in which one of the components hampers the medical treatment of the poisoning produced by the other component. Thus Russian workers have found that thiourea considerably slows down the reactivation of acetylcholinesterase inhibited by nerve gas.[47] A third example is the use of adjuvants to promote the penetration of the skin by chemical agents. Thus Canadian workers have shown that in guinea-pigs the lethal dose through the skin of a 50 per cent solution of soman in dimethyl sulphoxide is six times lower than that of pure soman.[48]

In the second category, mixtures containing an inert ingredient, comes the very considerable body of work on the encapsulation of toxic agents, that is the coating of minute particles or droplets of the agents with some other component.[49] There are several reasons for trying to do this, among them the protection of the agent from thermal decomposition during dissemination, or for the sustained release of the agent within a target area so as to maintain active concentrations within it for a predetermined length of time, or to make capsules designed to rupture when trodden on.

Munitions

The job of a chemical munition is to create a toxic environment over as much of the target as is compatible with the toxicity of its charge. It must convert its bulk load either into an even distribution of liquid or solid particles of optimal sizes and velocities, or into a cloud of vapour, or into both. It must do this within a certain time. And for economy it must have a high chemical-to-weight ratio. These are severe demands, and they are made more severe by the diversity of chemical agents now in the stockpiles. Each such agent has a combination of physical characteristics and toxic behaviour which is unique. None the less, all munitions work on the same

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basic principle. They cause the transfer of energy from a store to the chemical load. Generally the store is either an explosive or pyrotechnic charge or a supply of compressed gas. Its size and mode of transfer depend on the physical characteristics of the chemical agent, its volatility, viscosity, surface tension, as well as the sensitivity of the agent to heat and shock. The simplest chemicals to disperse are the volatile, non-persistent ones such as phosgene. These require little more than a small explosive charge to break open their container. The hardest ones are probably the heat-sensitive solid agents which are intended for lethal effect by inhalation, which we have not in fact described here at all; they include such things as *ricin*, a vegetable protein more toxic than the nerve gases and for which the U.S. Army has developed a cluster-bomb system,[50] and the bacterial toxins and biological agents discussed in Chapter 4. Here, we describe briefly only three known types of modern munition, varying in role and scale.

The nerve-gas artillery shell, such as the American M121 series of 155 mm. projectiles[51] have been in production since the middle 1950's. Each shell is about 2 feet long, weighs about 100 lb. and holds about 6 lb. of chemical. Along the axis is a cylindrical, high-explosive burster tube containing about 3 lb. of the explosive Tetrytol. This is a heavy charge; when detonated for an air-burst (by a proximity or time fuse) the nerve gas is atomized into a cloud of small droplets which hang in the air and, in the case of sarin, quickly vapourize. If a non-volatile nerve gas is used, such as VX, part will remain in aerosol form for lung penetration, and part in droplet or spray form for skin effects. Field trials of sarin-filled shell show that for an air-burst 15 feet above the ground a concentration of 3500 mg-min/m^3 will be set up in under 10 seconds over an area 20 yards in radius. A couple of breaths of this will kill. In about 25 seconds the concentration 50 yards from the burst is about 100 mg/m^3, to which a less than 10-second exposure is likely to be fatal.[24]

The 10-lb. mustard-gas thermogenerator cluster bombs are about 18 inches long and perhaps 3 inches wide.[52] On impact a pyrotechnic charge is ignited in the nose; this sends a stream of hot exhaust gas at high speed out through coarse nozzles in the tail section. The mustard gas, about 2·3 lb, is sucked into this stream and shattered into small droplets and vapour which on mixing with the surrounding air form an aerosol cloud of exceedingly small and penetrating particle size. Within five minutes of the scattering of a 64-bomb cluster of such munitions under suitable atmospheric conditions, the

concentration of mustard gas over 8000 square yards would be enough to cause a week-long temporary blindness following a 5-second exposure, or death following the skin absorption of a 2-minute exposure.[52] A B-52 bomber can hold about a hundred such clusters. V-agents can presumably be substituted for the mustard gas if necessary.

As a final example, there are chemical warheads for the larger missiles. Chemical warheads are made for, amongst others, the U.S. Sergeant missile, which has a range of about 100 miles, but the actual details of the warheads are classified.[53] What is known is that the Americans have standardized a number of different types of bomblet for clustering within such warheads. Shortly before the missile reaches the target, the warhead breaks open to scatter these bomblets. There may be several hundred per missile. In appearance, the various types of bomblet look alike; they are spherical, and their surface is vaned to impart rotation during descent, the rotation being used both for flight stabilization and to activate the fuses. Various dissemination mechanisms are used: high explosive burst, gas propellant and nozzles on the American's E120 BW bomblet,[54] and so on. One type which would be particularly well suited to such labile substances as the botulinal toxins consists of two halves held together by a spring-biassed bellows. As the bomblet falls the bellows expand under the influence of increasing atmospheric pressure until, at a pre-determined height, the two halves fly apart.[55] The load of micro-encapsulated powdered agent falls slowly to the ground as a fine cloud.

Delivery Systems

One of the assets of chemical warfare is that it does not depend on extraordinary delivery systems. Chemical munitions may be adapted for delivery by almost any means—grenade-throwers, artillery, aircraft, missiles, and so on. Indeed, in some cases the delivery system may be the environment itself—the chlorine cylinders of World War I for instance, or the flow of conditioned air or drinking water in a modern building. An example of one of the few modern delivery systems designed specifically for chemical munitions is the U.S. Army's M55 truck-mounted rocket launcher which launches salvoes of up to 45 small rockets in a space of 30 seconds.[56]

Missiles are well suited to chemical payloads. A warhead which distributes its load widely tends to counter the inherent inaccuracy. Whether missiles are in fact preferable to artillery or aerial bombing from a cost-effectiveness point of view is another matter.

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THE METEOROLOGICAL PROBLEM. Once a chemical munition has been set off its user has no further control over it. This is of course true for any other munition, but whereas the effects of, say, high explosive, follow within a fraction of a second of detonation, those of a chemical may be delayed for minutes, hours, or even days. In this lies both the strength and the weakness of chemical warfare. On the one hand a toxic atmosphere may be set up which will envelop the whole target area, seeping into tunnels and bunkers, and permeating buildings. On the other hand, the entire load may be blown uselessly away by a sudden wind, or become so diluted by it as to be impotent.

A chemical weapon system must take these problems into account. By and large a random scatter of small munitions over the target area will be preferred to the delivery onto it of a single large munition, for not only is the former easier to achieve, it will also tend to be self-compensating in an adverse environment.

It must be emphasized that, however well-designed the weapon system is, its effectiveness will still depend critically on the prevailing weather conditions. Thus a 1944 report from the Dugway Proving Ground in America[57] indicates that to produce lethal effects in unmasked personnel over a third of a square mile with 1000-lb. M79 phosgene bombs between 17 and 192 bombs may be required, depending on conditions. On a calm, clear night, using M79's, about 3 tons of phosgene would be needed, but during a breezy, sunny day, upwards of 35 tons—the difference between $\frac{1}{4}$ of a B-52 bomb load and $2\frac{1}{2}$ such loads. Not that phosgene would be used nowadays, of course, nor that a single bomber could produce the necessary spread.

All this implies that previous knowledge of target conditions is essential to a chemical attack. An unexpected wind profile or temperature gradient over the target could easily render the whole attack useless. Modern chemical troops are therefore equipped with meteorological apparatus, and a great deal of the research carried out at chemical warfare laboratories is concerned with improving that apparatus and extending its successfulness. Certainly the Chemical Defence Experimental Establishment (CDEE) at Porton maintains close ties with the Meteorological Office. It was not for nothing that the 12th Earl of Dundonald consulted that Office in 1914 before revealing to Lord Kitchener his grandfather's plans for chemical warfare.[58]

Defence

It could be that the introduction of the V-agents and mixed systems goes a long way towards restoring chemical weapons to that position of strategic capability which they lost when the first gas-masks were supplied to counter chlorine in 1915. We said earlier that with the introduction of tabun the very largest targets became vulnerable to chemical attack. With the introduction of the V-agents those targets can no longer be adequately defended, at any rate not without extraordinary and debilitating effort. One should therefore consider briefly what are the available defences against a chemical attack.

Broadly speaking, there are three methods of defence—physical, chemical, and medical.

PHYSICAL. The physical defence is the oldest. It involves putting up physical barriers against the entry of toxic agents into the body. For the individual, it means a gas-mask to protect the eyes and lungs, and chemically impermeable clothing to protect the skin. For collective protection, it means air-conditioned shelters. Over the past fifty years the necessary equipment has in fact been developed to a high degree of efficiency.

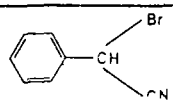
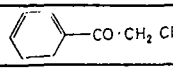
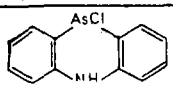
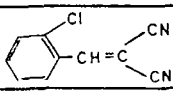
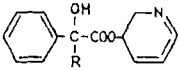
CHEMICAL. In chemical defence, the toxic agent is chemically decomposed before it can enter the body. For the individual, this means ointments containing reactive chemicals for smearing over the skin before an attack, or powders for dusting onto contaminated skin after an attack. Or it means clothing impregnated with reactive chemicals. For collective protection, it means the equipment of anti-gas squads with chemicals and spraying apparatus for destroying toxic agents deposited on the terrain. Or it means the chemical purification of contaminated food or drinking water. The reactive chemicals used are generally alkalis or chlorine-liberating substances,[35] as in general the biochemical mechanisms whereby toxic agents exert their effects mean that the toxic agents react with these substances. The chemical purification of contaminated air is less easy, but suitable decontaminants are becoming available, for example aqueous solutions of compounds of *iodine monochloride* with *polyvinyl pyrrolidone*[36] for spraying into mustard-gassed air.

MEDICAL. Medical defence (or prophylaxis) involves the destruction of the toxic agent *within* the body before it can exert its toxic effect, or the reversal of its biochemical effect, or the biochemical protection of susceptible sites within the body. All these require the

presence within the body before an attack of the appropriate drug at the appropriate site. With one notable exception, *British Anti-Lewisite*,[37] this is almost impossible to achieve satisfactorily. But a fourth, less difficult, possibility exists, namely the application of drugs which will poison the body in the *reverse* direction to the toxic agent. Thus, part of the normal treatment of nerve-gas poisoning which results in an accumulation of the chemical transmitted, acetylcholine, is to block those sites which are attacked by the accumulating chemical transmitter, using as a blocking agent the drug *atropine*, the active ingredient of deadly nightshade. Obviously the application of antidotes which are themselves poisons poses severe problems, which when coupled with the speed of action of modern chemical warfare agents greatly weakens the potential of medical defences.

I think it is true to say that the only reliable defences are the physical ones. But these have the drawback that they are only effective if they can be adopted *before* an attack takes place. They cannot protect an individual once he is poisoned. And while in the past the smell or initial irritancy of chemicals gave enough warning, chemical agents have now been developed specifically to deny such warning even at lethal concentrations. This means that it is possible for a lethal concentration to be set up over a target without its occupants being aware until too late. If those occupants are to be immune, they must either wear all-enveloping, nerve-gas impermeable clothing and gas-masks, and wear them all the time, or they must be warned by sensitive and automatic chemical alarms to so protect themselves. Alarm systems being studied range from the adaptation of the laser to long-path infra-red spectroscopy to the training of animals. Dogs have been taught to bark at sarin concentrations of about 1 mg/m^3, but BZ apparently defeats them.[45] There can be no doubt whatever that any man not so protected would be beyond help before medical aid could reach him.

Certainly it is true that the necessary impermeable clothing has been developed and indeed supplied to troops as standard equipment. It is true also that gas alarms are available, but even the best of them give no more than a few seconds warning. During the pre-war rearmament period the British Government did of course provide everyone in the country with a civilian-type gas-mask. The cost of this ran into tens of millions of pounds.[38] In view of today's extra requirements of impermeable clothing and alarms, not to mention defences against nuclear attack, is it likely that any government, British or otherwise, would still be able to foot the bill?

	US Army Code	Trivial Name	Chemical Structure	Normal Physical State	Smell	Disseminated Form	Symptoms of Intoxication
LETHAL AG	CG	phosgene	$Cl_2C=O$	colourless gas	new-mown hay; imparts metallic taste to tobacco smoke	gas	**lethal** coughing, retching, frothing at mouth, cyanosis, asphyxia; pneumonia
	AC	prussic acid	HCN	colourless liquid	bitter almonds	vapour	**lethal** giddiness, convulsions, unconsciousness, asphyxia
	HD	distilled mustard	$S(CH_2CH_2Cl)_2$	colourless to amber oily liquid	faint garlic	vapour liquid	**harassing** eyes : inflammation, photophobia, ulceration, blindness
	T		$O(CH_2CH_2SCH_2CH_2Cl)_2$	oily liquid	none	liquid aerosol	skin : redness, irritation, blisters
	Q	sesqui-mustard	$CH_2SCH_2CH_2Cl$ / $CH_2SCH_2CH_2Cl$	solid	none	aerosol	**lethal** resembles CG in its action on lungs; other systemic effects
	HN3	nitrogen mustard	$N(CH_2CH_2Cl)_2$	colourless to amber, oily liquid	faint geranium smell	vapour liquid aerosol	
NERVE GASES	GA	tabun	$(CH_3)_2N-P(=O)(CN)-OCH_2CH_3$	colourless to dark brown liquid	none to fruity	vapour liquid aerosol	**harassing** eyes: pupils constrict vision blurs and dims eyeballs hurt
	GB	sarin	$CH_3-P(=O)(F)-OCH(CH_3)_2$	colourless liquid	almost none	vapour liquid	respiration: chest tightness, difficulty in breathing
	GD	soman	$CH_3-P(=O)(F)-OCH(CH_3)C(CH_3)_3$	liquid	slightly fruity to camphor-like	vapour liquid aerosol	
	GE			liquid		vapour liquid aerosol	**lethal** drooling, sweating, nausea, vomiting, cramps, involuntary defaecation or urination, twitching, jerking, staggering, headache, confusion, drowsiness, coma, convulsions, asphyxia
	GF	CMPF	$CH_3-P(=O)(F)-OCH(CH_2CH_2)_2$	liquid		liquid vapour aerosol	
	VE		$CH_3-P(=O)(OR)-SCH_2CH_2NR_2$ (R = alkyl)	liquid		liquid aerosol	
	VX			liquid		liquid aerosol	
HARASSING AGENTS	CA	BBC Camite		pinkish to brown oily liquid	soured fruit	vapour aerosol	**harassing** burning feeling in mucous membranes, severe eye irritation and lachrymation, headache
	CN	CAP		white crystals	apple blossom	aerosol	**harassing** burning feeling on moist skin, copious lachrymation
	DM	adamsite		canary yellow to brownish-green crystals	almost none	aerosol	**harassing** headache, sneezing, coughing, chest pains, nausea, vomiting
	CS	OCBM		white crystals	peppery	aerosol	**harassing** stinging and burning feeling on skin, coughing, tears, chest tightness, nausea
Incapacitating Agent	BZ			solid		aerosol	slowing of physical and mental activity, giddiness, disorientation, hallucinations, occasional maniacal behaviour

PRINCIPAL PROPERTIES of the major known chemical weapons are outlined the table above. Smell and normal physical states vary greatly with the y of the agent, and lethal and incapacitating concentrations depend

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Time of Onset of Symptoms	LD_{50} * Percutaneous Absorption (mg per man)	LCt_{50} * Inhalation (mg-min/m³)	LCt_{50} * Percutaneous Absorption (mg-min/m³)	Incapacitating Dosage (mg-min/m³)	First Use or Country of First Development	Remarks
generally delayed for several hrs.		3200			Germans 1915	produced 80% of WW1 gas fatalities; extensively stockpiled in WW2
immediate		5000	200,000-1,000,000		France, about 1865 ; French, 1916	stockpiled by U.S.A. from 1942 on
delayed 1-48 hours	(blisters : 0.032)	1000	>10,000	7-day blindness: 200 2-week skin burns : 1000	Germans, 1917	most widely stockpiled agent of WW2
delayed	(blisters : 0.004)	about 400			UK, USA pre-WW2	used mixed with mustard; their production methods yield such mixtures ; the British stockpiled 60/40 HT during WW2
delayed	(blisters : 0.0003)	about 300			UK, USA, Germany, pre-WW2	
delayed	(blisters : about 0.06)	1000		7-day blindness: 200 2-week skin burns : 1000	UK, USA Germany, pre-WW2	less smell than HD, so even more insidious
up to 10 min following inhalation, or up to half an hr. following percutaneous absorption	1500 (30 'drops')	150		20 (unmasked)	Germany, 1937	standard German tabun contained 20% chlorobenzene ; to be aerosol dispersed
	2000 (40 'drops')	70	15,000	>20 (unmasked)	Germany, 1938	Germans stockpiled large quantities of its intermediates but could make only ½ ton
	about 1250	about 70			Germany, 1944	highly resistant to oxime therapy
					UK,USA Canada late 1940's or early 1950's	
						harder to treat than GB
	small					
	small					
immediate		3500		30	French 1918	useful as a persistent harassing agent
immediate		8500		80	USA 1918	
up to 3 minutes		30,000		20	UK, USA 1918	
immediate		very large		10	UK early 1950's	
					USA, middle 1950's	only standardized incapacitating agent by 1963

on the rate of breathing and the size of the aerosol particles; figures given are for a breathing rate of 15 litres/min (typical of mild activity). Optimum aerosol size, for maximum lung penetration is 0.5 to 3 microns. * define glossary

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Performance and Evaluation

We have quoted figures to illustrate the performance of certain munitions, and also for the same munition under different weather conditions. The great divergence that these show should indicate just how difficult it is to prepare in advance to launch a chemical attack. And the meteorological factor is by no means the only performance variable. Chemical weapons are purely anti-personnel weapons, so a commander must know just how mobile his target is, how well-protected and disciplined it is, not to mention where it is. He must know the type of ground which it occupies, whether it is open or wooded, dry or damp.

Although it is very probable that a Sergeant or a Scud* tactical missile loaded with nerve gas can produce 30 per cent casualties among a fully deployed battle group of 1400 men, and within seconds and under almost any conditions, there is doubt. Performance figures for chemical weapons such as those which suggest 30 per cent casualties over 100 square miles per B-52 bomb-load, are, although often quoted, in fact based on nothing more than an assumption.[59]

If an adequate prediction of a weapon system's performance under all likely conditions cannot be made, then, however potent that system may be, a commander will be reluctant to employ it, for long term planning will be logistically impossible. It is this fact more than any other which is likely to keep toxic chemicals off future battlefields.

One way to evaluate a chemical system is to carry out small-scale field trials and to blow them up to life-size with all the modern techniques of operational research—sensitivity analysis, war gaming, and so on. But this is not enough; there are too many imponderables, at any rate for today's techniques. The only reasonably certain way is by large-scale field trials carried out under all foreseeable types of condition. This would be a huge operation. Fortunately the political pressures against it are equally huge.

* The Russian equivalent of the Sergeant—*see* p. 131. *Ed.*

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2. Psychedelics

C. R. B. Joyce

There are at least two conflicting reasons for giving an early place to psychedelic drugs in a catalogue of potential agents of chemical and biological warfare: that they are useless; or that they are non-lethal and therefore in certain circumstances ideal. Here, we will briefly examine both these claims.

The first is easy to support or refute at the semantic level. The term 'psychedelic' is the result of an attempt to coin a word for what some regard as a characteristic effect of this kind of drug—its power to "expand consciousness". A psychedelic compound was first defined as "one, like LSD or mescaline, which enriches the mind and enlarges the vision."[1] But the word seems always to have been used since as a qualifier or as a collective term ('psychedelics' or 'psychedelia') for the chemicals or physical objects that are employed to produce the experience. It thus contains a rather sad implication that man cannot attain such a desirable state, an aspect of which is detachment from material preoccupations without material assistance.

However, if for the moment we accept this definition for lack of a better, it is clear that any psychedelic drug is one that enlarges the vision. A sufficiently enlarged enemy vision will contain all other points of view. Since one of these will be the objective, as far as the enemy is concerned, of any political commander, a psychedelic drug should be an ideal chemical warfare agent for major strategic use.

At a lower strategic level, the techniques of what is inelegantly called brain-washing usually have a similar end in view for an individual or small group. Thus, whatever the possibilities may be for successful widespread use of these substances, it would be surprising if some have not already been tried out in face-to-face interrogations of prisoners, the obvious test situation before wider use is contemplated at all. Although we have no evidence that psychedelics have been used in this way, hypnotics certainly have. The parallel with psychotherapy is obvious, and LSD has been

extensively used as an aid to psychotherapy. For the most part, the results have been as deeply equivocal as their interpretation has been partisan.

On the other hand it can also be maintained, at least for the sake of argument, that psychedelics would be worse than useless in warfare because the possessor of a truly enlarged vision would realize that the standpoints of both sides are equally absurd. The major use in wartime for a true psychedelic would be its impartial application to both sides by a disinterested third party. If a recent news report has any foundation, cannabis at least is not such a substance: or alternatively, such drugs are not always the pacifiers their civilian users claim. According to this report at least 75 per cent of NLF (Vietcong) and American Forces are regular users. Psychedelics are more commonly known as psychotomimetics or hallucinogens, although in fact drug-induced psychoses can be distinguished from naturally occurring ones by outside observers, although not so easily as by those experiencing them, and even at the height of their experiences most drug-users are aware that these have no external reality, and so are illusory rather than hallucinatory. The three terms will be used henceforth interchangeably.

Mind-enrichment is not experienced by everyone who takes LSD, and it may be that there is no such universal psychedelic. Short of death or of some major toxic effect which untreated would be fatal, there is no drug which is absolutely consistent in the type of its effects from one individual to another, or even reliable from one occasion to another in the same individual. One well-known hallucinogen, *trimethoxyamphetamine* (TMA) was found by one group, contrary to the experience of others, to cause "unexpectedly anti-social responses" such that "provocation would have precipitated homicidal violence,"[2] an unsatisfactory characteristic for a drug intended to reduce the will of the adversary to resist. Not only do people have different thresholds, but these are affected by experience, by expectations, and by previous exposure to the same drug. The development of tolerance to LSD itself, for example, is extraordinarily rapid—a highly significant degree is induced by a single dose. The subsequent loss of tolerance also occurs rapidly, and is complete in two to four days. Together, these phenomena mean that LSD cannot be taken effectively more frequently than once every three days or so: within that time a second attack with the substance after a first had failed would itself be bound to fail.

In discussions of this problem it often seems to be assumed, at least by those not privy to official secrets, that an effective 'psy-

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chochemical' must be of the lysergic acid diethylamide (LSD) type. This is not necessarily true. There are many other candidates for the job. A recent dictionary of psychotropic drugs[3] contains the formulae of "all compounds which have been reported to have any psychotropic activity", of three specified kinds: tranquillizing, energizing, and hallucinogenic. It excludes only certain large but well-established categories, such as the opiates and barbiturates, that have by now rather well-defined properties. Of 690 compounds in this collection no fewer than 90, or 13 per cent, are reported to have hallucinogenic activity. The name of at least one manufacturer is given for about half of these; the proprietors of the others are anonymous, but one or two manufacturers' names, such as Sandoz, Lakeside, Penick and Dow (who are also large-scale manufacturers of napalm[4] and of at least one chemical—which has found use among American 'hippies/the notorious DOM, or STP) appear relatively frequently among those which are named. Some 15 of the 370 companies listed as supplying at least one psychotropic drug are responsible for at least one hallucinogen each.

Although certain chemical groupings, such as the indoles, are closely associated with hallucinogenic activity, there is no single structural feature that is common to all reported hallucinogens: even nitrogen is not essential, as the cannabis derivatives demonstrate. On the contrary, of the major groups of psychotropic drugs there is not one that lacks a hallucinogenic member. Of 140 relatives of phenothiazine, for example, two are said to be hallucinogenic; of 66 phenylethylamines, 13, while 11 out of 17 LSD derivates are hallucinogenic. Hallucinogenic properties are therefore widespread. Even the anti-depressive *imipramine* is said to have caused hallucinations at doses of over 300 mg,[5] an amount not infrequently given to depressed psychiatric patients. Just as there often appears to be a dose of any so-called sedative that is excitatory, it may be that a further increase in dosage of most drugs active on the central nervous system will produce the kind of disruption known as psychedelic. If this is so the effect of changes in chemical structure will be shown upon such features as potency, or the ease with which the agent reaches its site of action, rather than upon the behaviour elicited.

Several well-known examples are shown in Table 2. It is very important that many of them are known to be lethal. If the claim often made for riot-control or disabling substances—that their effects are entirely reversible—is to be taken seriously, the organic fluorophosphonates are not eligible as 'off the rocker' agents—those under discussion. On the other hand, their effects resemble

those reported for the American gas BZ[7, 8] more closely than do those of most other contenders (Table 3), (See also Chapter 1). One additional comment may also be made about BZ. When the drug known as STP, originally a Dow product, began to circulate among the 'hippie' communities in California there was some suggestion that it might be, at least tentatively, identified with BZ. But this perhaps is no more than an indication of the difficulty of this area —for instance at one point the U.S. Food and Drug Administration also identified BZ as the herb drill, an innocuous enough substance. But it is very difficult to be sure how many substances, or synonyms for the same substance are used; it may be that the real BZ is a mixture of a number of the compounds described in Chapter 1.

Even those indoles previously thought to be virtually non-lethal are now believed to be responsible, albeit indirectly, not only for deaths by suicide or under psychotic delusion, but to have delayed effects extending through the machinery of chromosomal damage to the offspring of those exposed.[9, 10] This may prove to be a general effect of psychedelics: work is in hand at the moment to examine the possibility. It cannot be too often repeated that there is, in any case, no such thing as a safe chemical. Even water can kill.

Table 2 also makes it clear that the range of doses of different substances which may be given for their hallucinogenic effects is about a million. This is a thousand times larger than that for their duration. The two aspects are not correlated. There is a wide range of extremely potent substances available—at least one is more potent than LSD[11]—some short- and some relatively long-acting. Potency alone is unlikely to be a major consideration in the development of new psychedelic agents. One bomber or rocket with a ten-ton payload can deliver thirty-three effective doses of LSD, or one effective dose of atropine, for every present inhabitant of the earth. However, such a delivery would only effectively saturate a much smaller area: perhaps some 400 square miles, which is enough for many purposes. On the other hand, more potent agents do have the advantage that they take up less storage space and hence are also less easily discovered, whether at their site of manufacture or in transit.

The other properties listed in Table 2 for the relatively non-lethal substances—their speed of onset and the time for which their actions last—are of more interest in determining the circumstances under which they could be used. In general, these factors are directly related: the faster the onset, the faster the recovery. The route by which the drugs are administered is also relevant, as in most pharmacological situations, but substances with powerful effects on the

brain are in general readily absorbed, whether given by mouth or by injection, and they are of course particularly well absorbed from gases or aerosols. Only the skin unmarked by lesions may be an effective barrier, and then not to all—not, for example, to the mustards and the phosphonates.

Some ingenuity is probably being shown, and not only by the writers of thrillers, in developing selective delivery systems for drugs with psychedelic properties. Contamination of a major water supply has often been discussed, and dismissed as too crude, too unpredictable in its effects because of the variable affinity for water of different human beings—especially, perhaps, the decision-makers who are the major targets. Air-conditioning systems, pepper-pots, a prepared cigarette are progressively more selective and hence more suitable for use in small groups, particularly when these contain certain members of both the attacking and the attacked communities. A microphone prepared to deliver an aerosol could help to discredit a politician in the eyes of his own community without any of his adversaries being present. At times one is inclined to imagine that some technique of this kind has already reached a considerable degree of sophistication. But if so it is disappointing that the method does not seem to work better. However, according to the *U.S. Field Training Manual*[7] the information available about BZ shows that the means for delivering and dispensing it are intended for non-selective use on a medium but indiscriminate scale.

The psychological objectives of theoretically reversible chemical warfare agents are summarized in Table 4. In the absence of hard scientific evidence it can be no more than a personal opinion, but it seems that the objectives, which may be worthwhile and easily attainable by pharmacological means, are not to be achieved with psychedelic drugs. They seem at the moment to be a side issue if not a side-effect, more attractive for propaganda purposes because of the fantasies about mind-control that they may arouse in the minds of the opposition, than for the military or political usefulness of the actual experiences the engender. Since, however, it is relatively easy to produce new compounds with activity on the central nervous system and it is not very difficult to synthesize many of them in quantities that would make their large-scale employment feasible, a measure of effective political support or of successful 'selling' by scientists involved in their production may well be enough to guarantee their continued research and development.

This chapter has been obliged to concentrate upon the little that is known about American experience with military psy-

chedelics. Next to nothing is known about experience in other countries. An important review of the properties of psychotomimetic compounds by a former member of the CDEE at Porton, subsequently on the staff of the DRS at the British Embassy in Washington, contained no reference to the uses of such substances for defence or attack.[12] Such silence, at the time as well as subsequently, can be constructed in any way one wishes. It is impossible, therefore, to be sure how chemicals with these actions are seen militarily and politically.

To discuss the other major problems of protection, treatment, detection of use, detection of manufacture, and international control of manufacture would be to pile fantasy upon a foundation that is already far too speculative. A form of protection might be chronic medication with the agent expected to be used, or one similar to it, in order to achieve a degree of tolerance or cross-tolerance, but the consequences might be dangerous.

The best treatment of non-fatal poisoning is inactivity. No drug can restore uncomplicated normal function faster than healthy inactivation and excretion. To attempt to hurry it on with analeptics, even after an overdose of morphine or of barbiturate, is often misguided. There is some suggestion that to treat methoxyamphetamine poisoning with a tranquillizer—the approved way to abort a 'bad trip' on LSD—may deepen the intoxication.

To detect in use such drugs as these, which have their characteristic effects upon behaviour, is particularly difficult. For the most part they are tasteless, odourless, and colourless, and active in minute quantities. Sensitive animal indicators (the analogue of the miner's canary and fire-damp), such as the Siamese fighting-fish which responds characteristically to LSD, do exist. It is said that dogs have already been trained to detect the smell of cannabis. But a general detector based on physical techniques, for the presence of bacteria and vesicants as well as psychedelics seems more useful.

The question of international control is a depressing one: at least some of the substances discussed are still prepared, when required on an appreciable scale, from natural sources—cactus, mushroom, tree-bark, or fungus. But they are synthesizable; most have been synthesized; and the syntheses are continually becoming simpler. Although illicit LSD manufacture can sometimes be detected, prohibition of manufacture is another matter. Meanwhile there are additional risks: supplies of psychedelics, like poison gases, can leak. Whether they leak or not, they have an equally disastrous history, present, and future.

TABLE 2 Some Properties of Selected Psychedelic Substances

Class	Example	Human Ed_{50}*	Onset hr	Termination hr.	Lethality	Antidote
ANTI-SEROTONINS						
Indoles [12, 13]	LSD	50 μg	0.5–3	6–24	Indirect; delayed	Tranquillizers
	DMT	50 mg	0.1	1–4		
ANTI-CHOLINERGICS						
Piperidyl benzilates [14]	Benactyzine	100 mg	0.1–0.5	6–24	Small	
Tropine derivatives [13]	Atropine	10 mg	1.0–6	12–72	Appreciable	Prob. unnecessary
ANTI-CHOLINESTERASES						
Organic phosphonates [11, 13]	DFP	10 mg	0.5	120+	High	Oximes
G-V-agents[11]	Tabun (GA)					
ANTIMONOAMINE OXIDASES						
Phenylethylamines[3]	TMA	200 mg	0.5	12	?	Tranquillizers
	DOM (STP)	5 mg?	0.5?	?	? Indirect	? Unknown
CANNABIS DERIVATIVES[14]	Δ^1—THC	5 mg	0.1	5	? None	Hypnotics Tranquillizers
ANTIMITOTICS[15, 11]	Dimethylacetamide	100 g	72+	100+	High	?
	BZ	10 μg?	0.1?	250?	?	?

* Dose effective in 50 per cent of population.

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TABLE 3 Identification of BZ by Comparison of Activity Profiles

	SIGNS								
	Hallucinations or Illusions	Paralysis	Hypothermia	Mental Slowing	Vertigo	Hypotension	Headache	Lacrimation Salivation Sweating	Diarrhoea Vomiting
BZ [7,8,17]	+	+	+	+	+	+	+	?	?
LSD [13, 16]	+	—	?	+	?	?	—	—	—
Anticholinergics [13, 16]	+	—	+	?	?	—	—	—	—
Anticholinesterases [11, 13]	+	+	+	+	+	+	+	+	+
Cyanogens [8, 11, 13]	?	?	?	—	+	+	+	+	?
Arsenicals [8, 13]	+	—	—	?	+	+	+	+	+
Benzothiazoles [11]	?	+	?	?	?	?	?	?	?

TABLE 4 Some Objectives of Reversible CW Agents

Time-Scale	Technique	Agent	Objective	Behaviour-type disorders
1. **LONGTERM**	Conversion Brain-washing, psychotherapy	Conflict resolution → Psychological Warfare →	Enlightenment Acceptance	Motivational
	Starvation, chronic cytotoxic anoxia	Defoliants →	Apathy Aging, mutations	Somatic
2. **INTERMEDIATE**	'Psychedelic' experience Intoxication	Indoles Anticholinesterases Piperidyl benzilates →	Disengagement (hallucinations, illusions, derealization, amnesia)	Cognitive
3. **BRIEF**	Skeleto-motor paralysis Autonomic crisis	Nerve gases Hypotensives Laxatives →	Immobilization	Motor

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3. Napalm

V. W. Sidel

There are two reasons for a brief discussion of napalm. First, circumstances in Vietnam have led to an association of napalm with chemical weapons. But it is important in developing control measures that the issues in dealing with incendiary weapons, such as napalm, be separated from the problem of dealing with chemical weapons, for different issues are involved in the control of the development, production, or use of the two different types of weapons, and confusion of the two may lead only to weakening controls against either. Second, one of the major issues in the development of chemical and biological weapons is the role of secret weapons research in universities. The development of napalm is a fascinating case study of applied weapons research by a university chemist on a well-known university campus.*

The Weapon

Napalm is gelled petrol or, in the American terminology, gelled gasoline. Originally, the term 'napalm' denoted the thickener that produced a gel when added to petrol; later it was broadened by usage to denote the incendiary gel itself. The name is derived from the first syllables of *naphthenate* and *palmitate*, two fatty acids first thought to be the active principles of the thickener. However, the material used in the original synthesis was mislabelled, and actually contained the soaps of all the fatty acids of coconut oil. These other acids, especially *lauric acid*, were found to be essential to the gel. Although literally a misnomer, the name napalm has been retained as a generic one for weapons of this type.[2, 3] Recently the term has also been applied to a gel consisting of petrol, benzene, and polystyrene which is also called 'incenderjell' or Napalm-B.[4]

When mixed with thickener petrol changes from a thin volatile

* Much of this material has been gathered in co-operation with Dr Peter Reich; some of it has previously appeared in the *New England Journal of Medicine*.

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liquid into a tough, stable, sticky gel. Raw petrol is useless as an incendiary weapon; napalm is ideally suited for this purpose. In contrast to many modern weapons, napalm is produced from cheap common ingredients and can be delivered in simple devices.[5]

Incendiary techniques and the development of napalm

Incendiary weapons have a long history in warfare, references to the use of fire in war going back to 2400 B.C. Although the introduction of explosives in the fourteenth century temporarily eclipsed the use of incendiaries, the advantages of fire over blast were well known to military strategists. Blast is self-limiting; incendiary agents are self-propagating, have a prolonged duration of action, and are capable of producing far more disruption and terror than an equal weight of explosives. But it was not until the advent of aerial warfare and the development of efficient incendiary substances, notably napalm, that fire reclaimed its role in war.

Incendiary agents were used in World War I, and an attempt was made by both the German and the Allied Forces to use petrol in flame-throwers.[6] This was hazardous and unsuccessful, but it led to a recognition of the potential danger of incendiary warfare. The treaties of Saint-Germain-en-Laye in 1919 and Trianon in 1920 prohibited the use and manufacture of the flame-throwers along with that of chemical agents. Several provisions against the use and manufacture of flame-throwers and chemical weapons were included in the separate peace treaties negotiated by the United States with the Central Powers. However, it is of interest that the Geneva Protocol of 1925 which prohibited the use of poison gases and bacteriological warfare did not deal with the use of incendiary weapons.[7]

At the time of the outbreak of World War II, the United States Army Chemical Warfare Service was interested in developing an improved incendiary mixture. They enlisted the aid of Professor Louis Fieser of Harvard University, a distinguished organic chemist. He first supplied the Chemical Warfare Service with a formula for a petrol gel using raw rubber as a thickener. This was a moderately effective agent, but the Japanese invasion of the East Indies cut off the supplies of natural rubber and necessitated the development of a synthetic thickener. The research programme that followed, conducted at Harvard University, was presented subsequently in great detail in Fieser's book, *The Scientific Method*,[3] and Fieser has never expressed concern for his part in its development. The first successful napalm detonations were conducted on a Harvard University

games field and behind the football stadium. It is thus an excellent example of applied weapons research in the universities.

The napalm gel proved far superior to the original rubber-based gel, and napalm was used extensively by the United States in incendiary raids on Japan during World War II.[8] Napalm was also used in Korea, where it was called the United States' "best all around weapon,"[9] and it has been used extensively in Vietnam.[10]

Effect of napalm

Napalm casualties are caused primarily by heat and by carbon monoxide poisoning.[11] The adhesiveness, prolonged burning time and high burning temperature of napalm favour third-degree burns, and such burns are likely to be deep and extensive, often resulting in severe scarring and deformities, especially when they occur under conditions making early skin-grafting difficult. Kidney failure may be another serious complication. The igniting agent in napalm weapons, white phosphorus, may become embedded in the tissues and continue smouldering and re-igniting long after the initial trauma. It was reported in Korea that panic is more likely to be observed among napalm victims than among those wounded by other agents.[5] The weapon caused far more deaths in Japan than were caused by the atomic attacks on Hiroshima and Nagasaki.[8]

Conclusion

Although certain incendiary weapons such as flame-throwers were coupled with chemical weapons when mentioned in peace treaties after World War I and although napalm has, due to circumstances in Vietnam, been coupled with chemical weapons in recent years, the most important control document on chemical and biological weapons, the Geneva Protocol of 1925, does not link the two. Control would be strengthened by clearly maintaining the separation of the two types of weapons—that is, chemical and biological on the one hand and incendiary on the other—in measures for control and disarmament. Although explosives are also chemicals there has been no attempt to link them with 'chemical weapons' in arms control documents (see Chapter 1). Perhaps control would be most facilitated by recognizing both the nature of the weapon and the modality by which it has its effect. Thus explosives and incendiaries are 'physical' weapons, producing their effects by blast and heat. Chemical weapons and biological weapons have special toxic modes of action which are not mediated through such 'physical' effects. Although the distinction is a scientifically

inexact one, it may be useful in developing controls: the special quality of nuclear weapons—their radiation effects—has proved useful in control efforts. I believe the special toxic quality of chemical and biological weapons which may permit their definition and control should not be blurred by coupling them with incendiaries.

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4. Biological Weapons

I. Màlek

In this chapter I wish to deal with the most important and most dangerous types of biological weapons—those that use micro-organisms (e.g., bacteria, bacilli, viruses, rickettsias, and fungi). This should be more accurately termed microbiological than biological warfare, as some biological agents can doubtless be devised that do not fall into these categories. I will indicate the scope of the potential weapons and the possible defences against them. Lastly, I will consider possible means of controlling the use of such weapons.

The extent of the threat

There are two extreme points of view on biological weapons. One view underestimates them and regards them as weapons whose effects and potential danger to humanity cannot be compared with those of nuclear weapons. The other stresses not only their danger, but their relative ease of production and use (compared to nuclear weapons), especially in local wars. Such a view also stresses the difficulty of controlling the production and use of biological weapons, thus enhancing their potential danger.

This second view is strengthened by the knowledge that some countries are spending enormous sums on the research and development of biological weapons with huge research institutes, often with better facilities than microbiological institutes, studying the peaceful utilization of micro-organisms. As Elinor Langer points out in Chapter 10, even if these sums seem small in comparison with annual expenditure on research and development of nuclear weapons and rockets, when one examines the scale to which biologists are accustomed in conducting their research they are substantial.

The reality of the threat is sharpened by the persistent memory of the terrible plagues which in the course of history have taken a far greater toll of human lives than war itself. The Spanish influ-

enza epidemic after World War I is still relatively recent, and people are regularly reminded of it by the after-effects of similar epidemics which, though less dangerous, occur even today. In addition, contemporary techniques make it relatively easy to produce infectious aerosols and to disperse them over wide areas. Much research effort has been devoted to a study of exactly how such an aerosol cloud spreads, how long it remains in the atmosphere in an active state, and what is its most effective means of dispersal. Certainly, much of the work at Porton and Detrick is directed to this end.

Without overdramatically raising apocalyptic visions of the effectiveness of biological weapons, it must be apparent that I, as a microbiologist, believe that there is a greater danger from underestimating biological weapons than from overestimating them. After all, these are potential weapons which can easily affect the balance of forces in any conflict, especially in those cases where the control of epidemics is difficult, whether in territories with poorly developed health services or a terrain that has not yet been sufficiently investigated epidemiologically, or when the normal life and physical resistance of the people have been severely damaged by nuclear attack. I do not wish at this point to go into the technical details of the conditions under which biological weapons can be used. We can be sure however that in the research on biological weapons, on which the military spend such enormous sums, attention is being paid to these questions of strategy. That is why I cannot side with the fatalistic view that in a situation where the effective restriction of the use of biological weapons is difficult we must be content to rely on effects of public opinion and the fact that as yet the agents may not be sufficiently controllable to make them as reliable as chemical weapons.

The scope of the weapons

The idea of using microbiological weapons goes back to the experience with infections and epidemics, which have been a serious military problem throughout the history of wars. Despite the development of vaccines, sera, and antibiotics, they remain, even without artificial introduction and dispersion, an important and difficult military problem. There are in fact several examples of the use, or alleged use, of micro-organisms against populations even in the pre-microbiological era; for example, the introduction of smallpox to the American Indians by the early settlers who gave or sold them infected blankets or trinkets. Allegations of 'the poisoning of wells' form a similar class.

ADVANTAGES AND DISADVANTAGES OF BIOLOGICAL WEAPONS. The most powerful argument in favour of the use of microbiological techniques in warfare aimed at population destruction is the relatively great *variety* of possible agents, often with completely different routes of infection, ways of spreading, incubation periods, and very limited possibilities of prevention, defence, and cure.

Some of the infectious agents enter the human body most effectively by way of the respiratory tract, and these can be either bacteria (e.g., plague, *tularaemia*), spores of *bacilli* (e.g., anthrax), *rickettsiae* Q-fever, viruses (e.g., *influenza, psittacosis*, different types of *encephalitis*), or even fungi (e.g., *coccidiomycosis, histoplasmosis*). Naturally each of these infections needs a completely different diagnostic approach and demands a different cure. Specific prevention, where this is possible, varies from case to case. Such infections can, in principle, be very easily spread under favourable meteorological conditions over vast areas by the use of aerosols.

Other infective or toxic agents can penetrate the human body directly through mucous membranes, such as the eye or nose. Among these are several viruses and some bacteria (e.g., those causing tularaemia, *brucellosis, rickettsias*, and *botulism*). Some others are most effectively absorbed orally with food or contaminated water (e.g., *salmonellosis, dysentery*, and *cholera*). And to make the situation still more complicated, these agents can be used in mixtures, thus causing extremely complex diagnostic problems for those attempting to identify and combat the disease.

All this, together with the minute dimensions of the organisms—of the order of one micron in diameter (a micron is 0·001 mm)—and the often complicated and slow methods of detection and identification, means that a population subjected to a microbiological attack would probably not be able to identify its cause before the infection had become widespread.

The second important quality of these microbiological agents is that they are alive and can reproduce. Thus a chain of infection from one infected person to others can easily occur. This is especially dangerous with respiratory infections, where the time of greatest infectivity often begins in the incubation period when the symptoms are very slight and non-specific, and when infection spreads through minute exhaled droplets to all persons in the vicinity of the infected persons. Even in communities not unbalanced by war conditions such spreading of infection can be very hard to control. Experiences with the recent foot-and-mouth disease in cattle in Great Britain, with smallpox epidemics introduced from countries where it is not

adequately controlled, or with myxomatosis—originally introduced artificially to limit the rabbit population and afterwards spreading uncontrollably—are examples of this problem even in highly organized societies. All these problems can be still more severe in countries with insufficiently developed medical and public health services or, of course, under war conditions.

A further advantage of the biological agents as weapons is that some are very resistant to external conditions and can remain latent but potentially infective for many years. An example is the anthrax bacillus, whose spores may remain infective for a hundred years or more—as evidenced by the case of the Scottish island, Gruinard, discussed by Robin Clarke in Chapter 9. With other organisms, conditions can be created to make them sufficiently resistant to survive in the form of aerosols for long periods of time.

Because of the minute dimensions of micro-organisms, they can be easily dispersed in the form of aerosol clouds of great dimensions which can move for very long distances. Field tests have been carried out in the United States, using either fluorescent particles or non-pathogenic bacterial spores of bacilli, showing that such aerosol clouds generated by spraying from a ship along a 150-mile stretch of coast, spread out over 55,000 square miles of land, over which a minimum dose of 15 particles and a maximum dose of 15,000 particles were inhaled by members of the affected population. In spite of the rather unfavourable conditions during this experiment, the cloud could be followed about twenty-three miles in the direction of the wind with viable cell densities corresponding to sufficiently high infective doses, even inside buildings. To achieve this it was sufficient to spray some 500 litres of a suspension of a harmless bacteria (*bacillus subtilis*).[1] The infectious effectiveness of such aerosols depends greatly on the size of the dispersed particles or droplets: the smaller the particles, the more effective they are. With the techniques available today it is easy to produce highly effective aerosols with relatively small devices—perhaps small enough to make possible individual sabotage without real danger for the saboteur, particularly as the incubation period for the disease would give him time to disappear from the scene before the first symptoms were noticed. Similarly it would be much easier to carry massive amounts of such infective agents in aeroplanes than, say, bombs. It can be objected that these are mostly hypotheses made on the basis of experiments with animals and with experience gained in the laboratory. But even these results show that the danger caused by infectious aerosols, especially with some micro-

organisms where the inhalation route is not the natural one, could be greater than supposed.

The infectious or toxic dose of some micro-organisms or their toxin (*i.e.*, the poison they produce) can be very small, so a very small amount of dispersed material could infect or intoxicate large numbers of people. Thus the aerosol doses of *Pasteurella tularaemis* on humans was experimentally evaluated at about 25 to 50 cells; in the case of Q-fever, only one micro-organism (*Coxiella burneti*) might be sufficient to cause infection, so that in one gram of dispersed material many million infectious doses can be present. Drinking 100 ml. of water from a reservoir of some 5 million litres capacity would cause serious infection or intoxication if as little as half a kilogram of Salmonella, 5 kg. of *botulinum* toxin, or 7 kg. of *staphylococcal enterotoxin* had been introduced.[1] Of course, these are only illustrative data upon which it would be possible to enlarge further by considering other infections and other conditions of dispersal.

There is also the possibility of the spread of infection by use of living vectors, such as insects, ticks, or lice, thus forming persistent foci of infection under favourable conditions. Very much is now known about the ecology of such vectors and the way in which they participate in the dissemination of infection. Allegations about the use of germ warfare by the United States in Korea claimed that animal carriers had been used in this way.[5]

A minor but important potential advantage of biological agents is that they do not require large, expensive, and complicated equipment for their use, especially in comparison with equipment necessary for nuclear weapons. Any well-equipped microbiological laboratory with normally skilled technical personnel can readily produce large quantities of the agents if infectious strains of micro-organisms are available.

In the case of intended microbiological attack it is possible to prepare one's own personnel, for instance by vaccination against selected micro-organisms, so that they would not be seriously endangered when entering the infected area.

The possibility of clandestine use of the agents must also be raised, for identification of the artificially introduced infection is often not easy and can take a long time, while allegations of their use are not easily proved or disproved—witness the allegations about Korea[5]—especially where the epidemiological background of the country is not sufficiently known and its medical services are not fully competent.

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And one final advantage: biological weapons are directed against a population (human or animal). They cannot cause any material damage; they lead only to great human losses and produce profound effects on morale, and cause panic. This alone could make the microbiological weapons attractive to a potential aggressor.

These then are the main reasons which make the weapons extremely dangerous and could make them attractive to an attacking force under some special conditions. Bearing these in mind why then have biological weapons not been already used, since nuclear and even chemical ones have been? The answer lies in the fact that these weapons also have their disadvantages, particularly, perhaps, in comparison with chemical weapons. The most important of these is that their effectiveness depends on not only the agent itself, but also on ecological and meteorological conditions, over which the aggressor has no control. In addition the relatively slow development of most infections diminishes their short-term tactical value to a commander concerned with immediate advantage. The effect of biological agents lies in the strategic dimension only.

But I believe that it is not merely because of these disadvantages nor of insufficient knowledge that the weapons have not been used. I am convinced that those huge installations and organizations which study their potential with such efficiency have been able to gather sufficient technological experience to be able to use these weapons on a large scale, fully corresponding to the level of highly sophisticated contemporary military techniques.

Defence against biological warfare

Over the past hundred years a number of techniques for controlling the spread of infectious diseases have been evolved. Among the major achievements have been the ability to isolate and identify a great many infectious agents, the discovery of how these agents behave, how they are able to enter the human body, and the mechanism of their pathogenic activity. This knowledge has created the possibility of a variety of epidemiological measures, notably isolation, disinfection and sterilization, and most important, immunization and vaccination. These last depend on the introduction either of mitigated or dead infectious agents or of specific antimicrobial agents such as chemotherapeutics or antibiotics. All these possibilities are naturally also at our disposal for use against microorganisms disseminated as weapons. But there are difficulties which

make all this highly elaborate system less effective in the case of massive artificial infection with microbial weapons.

Firstly, despite the great success in controlling infectious diseases, there still exist dangerous infections where control is not yet very efficient, either because of the difficulty of diagnosis, as is the case with some virus diseases, or because we do not have sufficiently efficient preventive vaccination or therapeutic measures of chemotherapy or antibiotics. It must be assumed that precisely these microbial agents will find favour in a potential attack.

Secondly, even where epidemiological knowledge is sufficient, in the case of massive dispersion of agents, any counter measures will inevitably lag behind the speed of the outbreak. It will be possible to identify the infection only after the appearance of clear clinical symptoms, and this is usually too late, as the greatest infectivity often occurs during the later stages of the incubation period and before the clinical symptoms have become clear. If one adds the time necessary for exact laboratory identification it is obvious how difficult it will prove to cope sufficiently with the speed of an induced epidemic.

This is still more true of any attempt at preventive immunization. It is hard to imagine that all the population at risk by exposure to microbial attack could be vaccinated against *all* possible agents. And if the vaccination is begun after attack with an identified agent it would take days or even weeks before sufficient immunity could be achieved.

Another difficulty is connected with the fact that nearly all the dangerous micro-organisms likely to be used as biological weapons can exist in *mutant* forms for which further preventive measures would be necessary. Mutant forms can develop naturally, as in the ominous case of penicillin-resistant organisms, but it is also possible to create them artificially by means of exposure to radiation or toxic chemicals.

And the last difficulty results from the fact that immunity—be it natural or artificially induced by vaccination—can readily be destroyed by exposure to radiation. Thus all the advantages of prior vaccination would be lost and all the induced infection would occur in much more dangerous forms in the case of nuclear attack. Thus the greatest danger of the possible use of microbial weapons is in conjunction with the dropping of nuclear bombs.

That is why, in spite of the fact that suitable strains are available for use as vaccines or in the production of antigens, which could be used for immunization against many potential biological agents, a

full immunization programme will probably never become a general prophylactic measure.[1]

Thus it becomes necessary when thinking about defence to seek ways which would enable us to make it more efficient and which would diminish these gaps. The most important would be the development of methods to make the detection of possible microbial attack more efficient. Hedén[1] subdivides detection into three stages, those of warning, sampling, and identification. All of these must be accomplished so as to shorten the time necessary for active defensive steps. Modern microbiological methods and knowledge offer several possibilities which have probably already been explored in the existing special military institutes. An important step towards international confidence in this area would be to distribute this information as widely as possible. There are available, for instance, devices for sampling of air probes with a very high capacity, which can gather some 10 cubic metres of air per minute into a liquid flow of 10 ml., making rapid warning of the presence of infective aerosol clouds possible and effective.[1] Or a method using fluorescent labelled anti-bodies (substances which would bind onto specific microbiological strains) could be used for rapid identification of the agents present in such a cloud. Other possible protective systems are of the mechanical variety—efficient, positive pressure shelters, or disinfection procedures and, in the case of suspected insect vector attack, repellents and so forth. But these must inevitably have a limited role in any protection of the population at large.

Thus biological weapons provide, by comparison with chemical ones, a much greater diversity of agents and methods of induction, they can only be identified with much greater delay, and in spite of considerable knowledge about the agents and the possible defence against them in principle, in practice they are much more difficult to control. They could probably cause much greater disaster among people for a smaller expenditure of effort. On the other hand the chemical weapons are much better defined, more easily manageable, and their effect more immediate. Thus they have a potential *tactical* significance which the biological weapons lack.

One of the characteristic features of biological weapons is that it is difficult to distinguish work done purely for defensive ends from that which is mainly offensive. Furthermore, if defence is to be effective and prepared in time it must be based on knowledge that can easily be transferred to offensive uses. That is why military establishments working on the development of these weapons do

it mostly under the label of defence. Biological weapons, therefore, can be used as an excuse for not reaching agreement on general disarmament, especially in the absence of an early and thorough study of the problems involved, to enable the arguments on the impossibilities of disarmament and inspection to be refuted (*see* Chapter 13).[3, 6, 7]

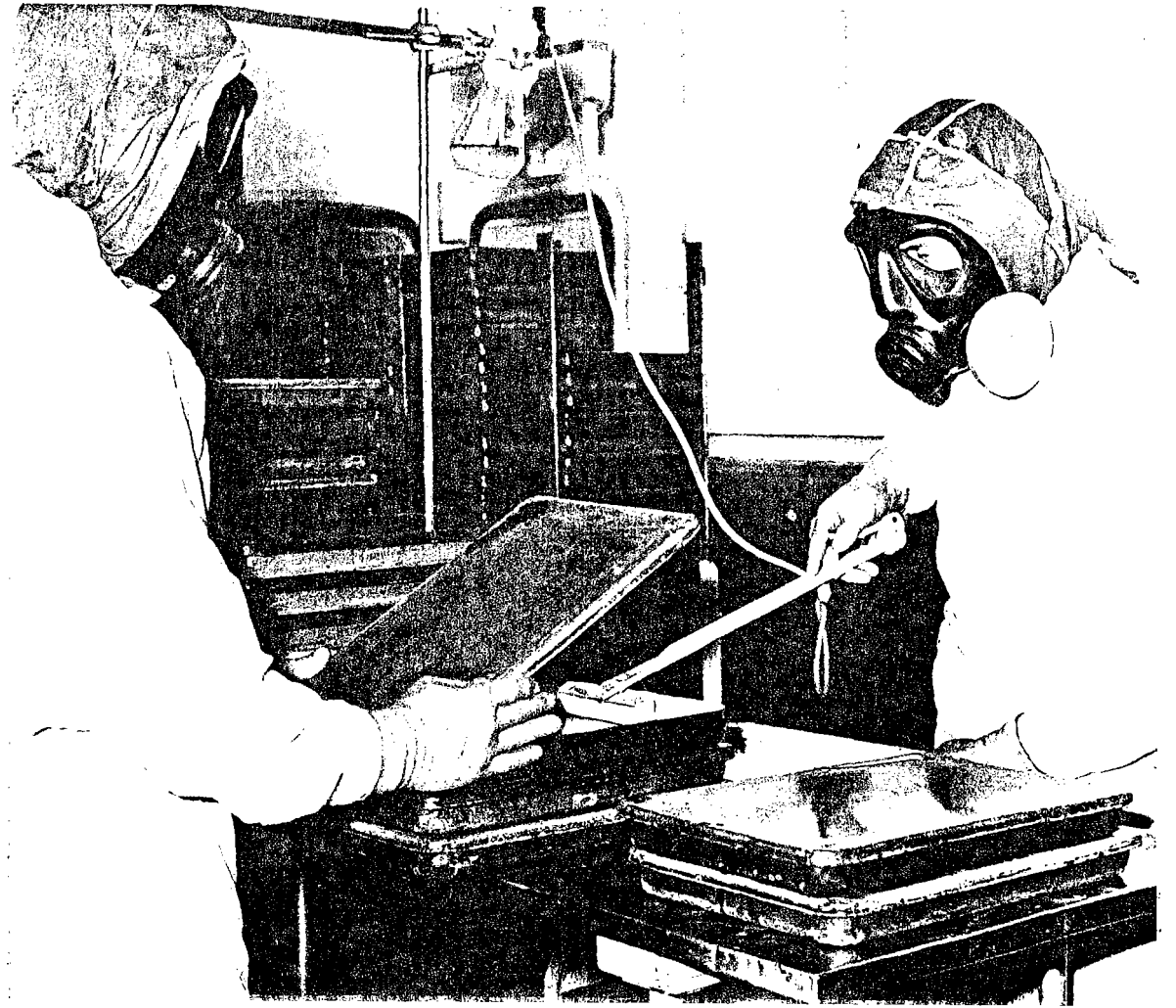
Such considerations have led to the establishment of a study group attached to the international scientists' Pugwash Movement, with the objects of (a) identifying the questions that had to be solved; (b) contributing to their solution on a contemporary scientific level; (c) helping spread knowledge of the actual danger of biological weapons and the struggle against the secrecy of their preparation and production; and, (d) seeking ways and means of speeding the banning of biological weapons and disarmament.[3, 6, 7]

The work of the Pugwash study group was directed also towards three questions: new methods for the quick detection of any biological weapon attack; the possibility of control and inspection of institutes in countries voluntarily giving up any activity which could be misused for the development of biological weapons; and, finally, the general tactics of a campaign leading to complete biological disarmament.

Interesting experimental results were obtained on the second question. By trial inspections of four microbiological institutes in different countries that volunteered (Sweden, Denmark, Czechoslovakia, Austria), relatively effective methods of inspection were worked out. The conditions were also defined under which such a voluntary ban on work on biological weapons and control of such a ban would be effective, while insuring protection against their use by another party. At the same time, it was estimated to what extent the experience of the International Atomic Energy Agency in Vienna, in the control of the peaceful uses of nuclear energy, could be used for a similar system of control of means of biological warfare and the use of micro-organisms for peaceful purposes.

Finally, a significant contribution was made when the Stockholm Peace Research Institute (SIPRI), founded and financed by the Swedish Government, chose as one of its research projects the problem of biological weapons.* It has started work on the legal implications of biological weapons and their ban. Rapid detection

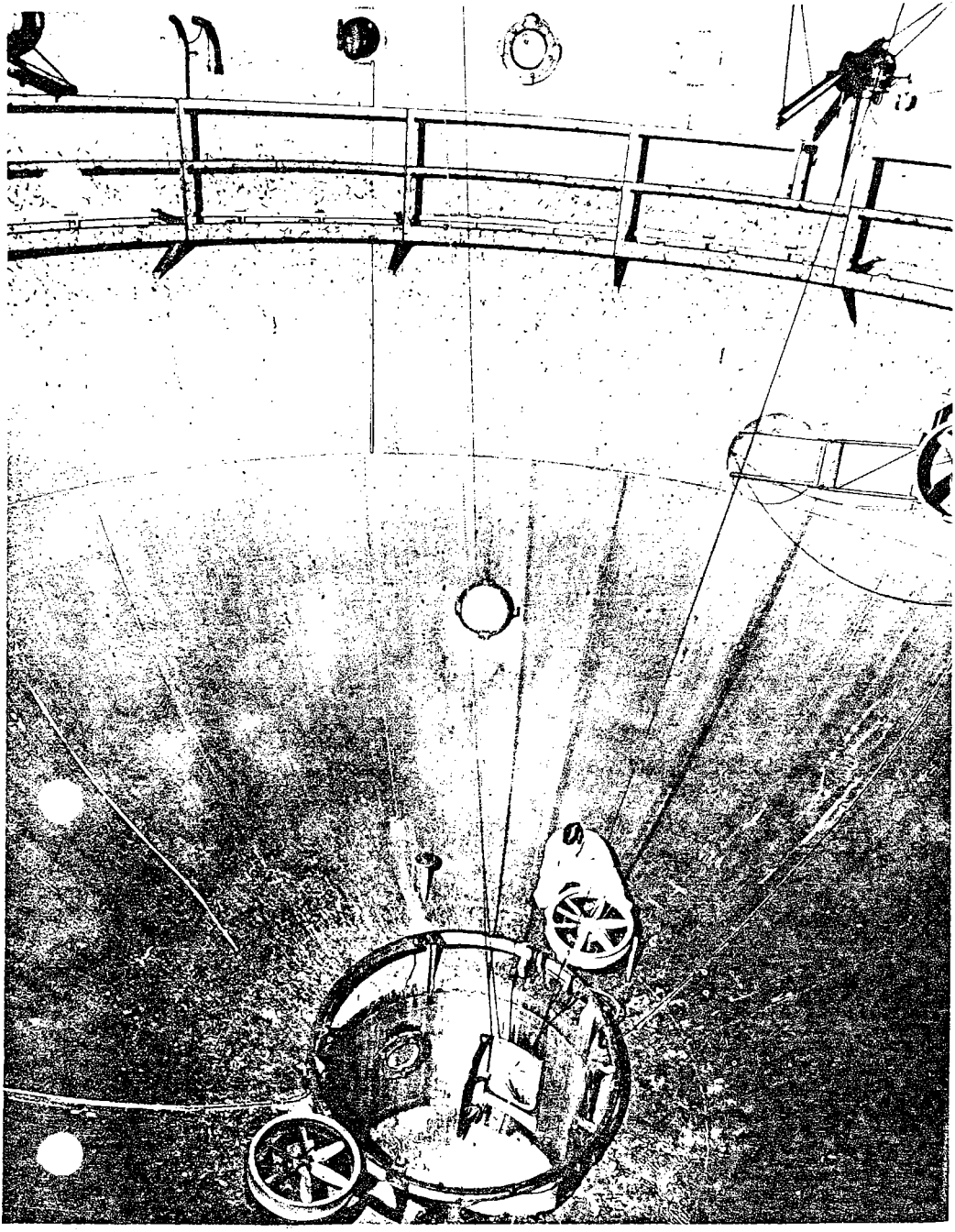
* Representatives of SIPRI were present at the Conference, including Dr Bjornerstedt and Dr Nemec, and were able to give a preliminary account of this study, which, however, has not been included as such in this text. *Ed.*



CBW Research

The mass-production of anthrax by the batch method at the Microbiological Research Establishment, Porton Down, Wilts.

21101



CBW Research

U. S. Army

The dispersal capabilities of bomblets are tested inside this vast aerosol test sphere nicknamed 'the 8-ball'

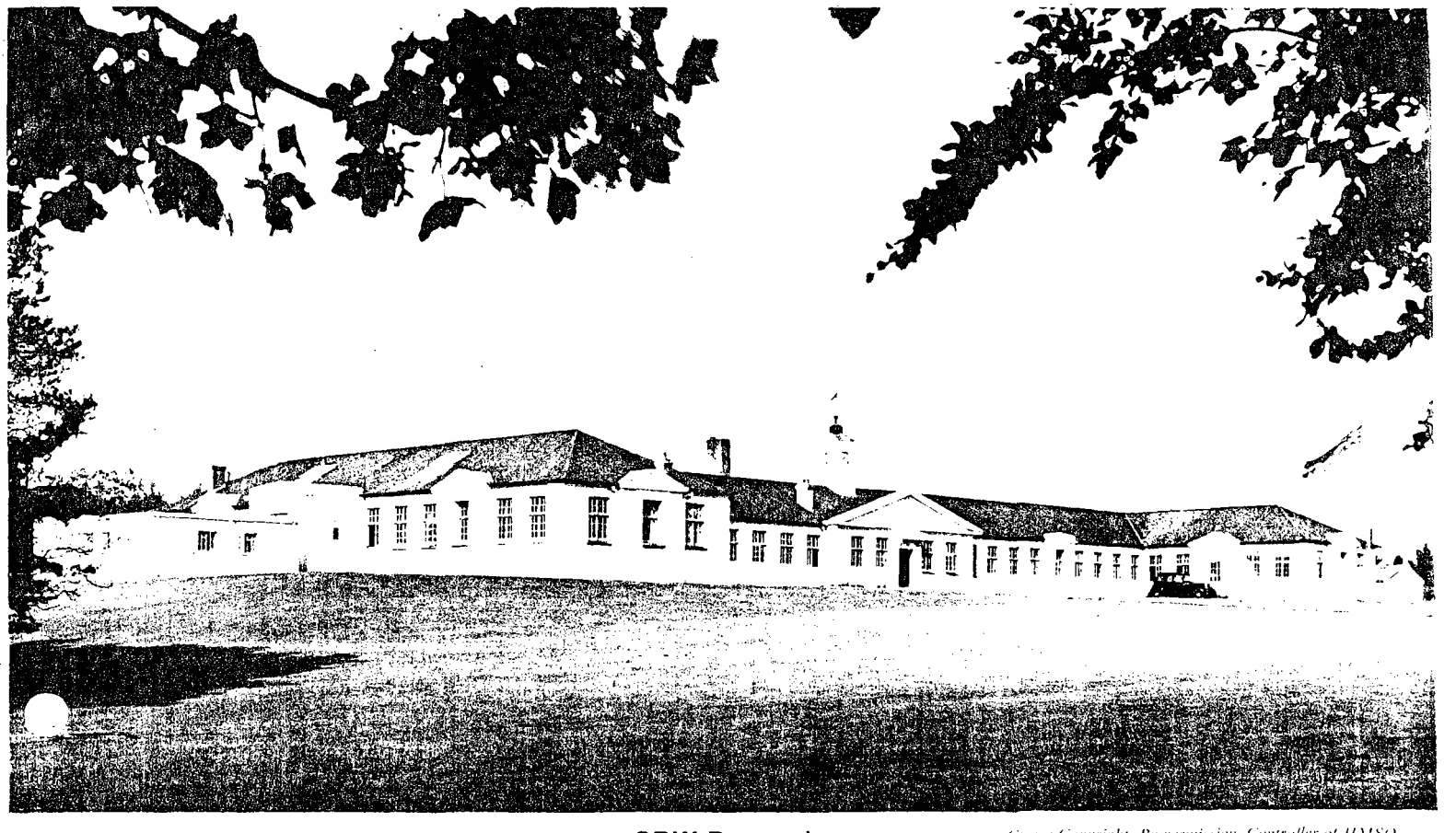
CBW Research *Science*

During recent tests of a new nozzle for the dissemination of nerve gas 6000 sheep were accidentally destroyed on lands adjacent to the Dugway proving ground. This communal grave holds 1200 pregnant ewes

CBW Research

U.S. Army

A portion of Fort Detrick, Maryland



CBW Research

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Headquarters Building, Chemical Defence Experimental Establishment,
Porton Down, Wilts.

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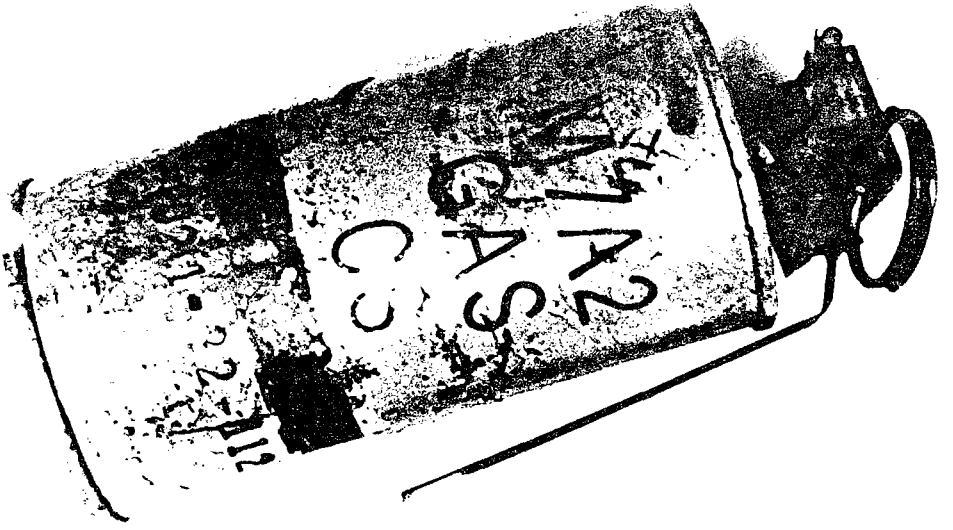


CBW in Use

Above
Grenade of CS gas as used in Vietnam

Right
Gas-mask approved for use in Vietnam

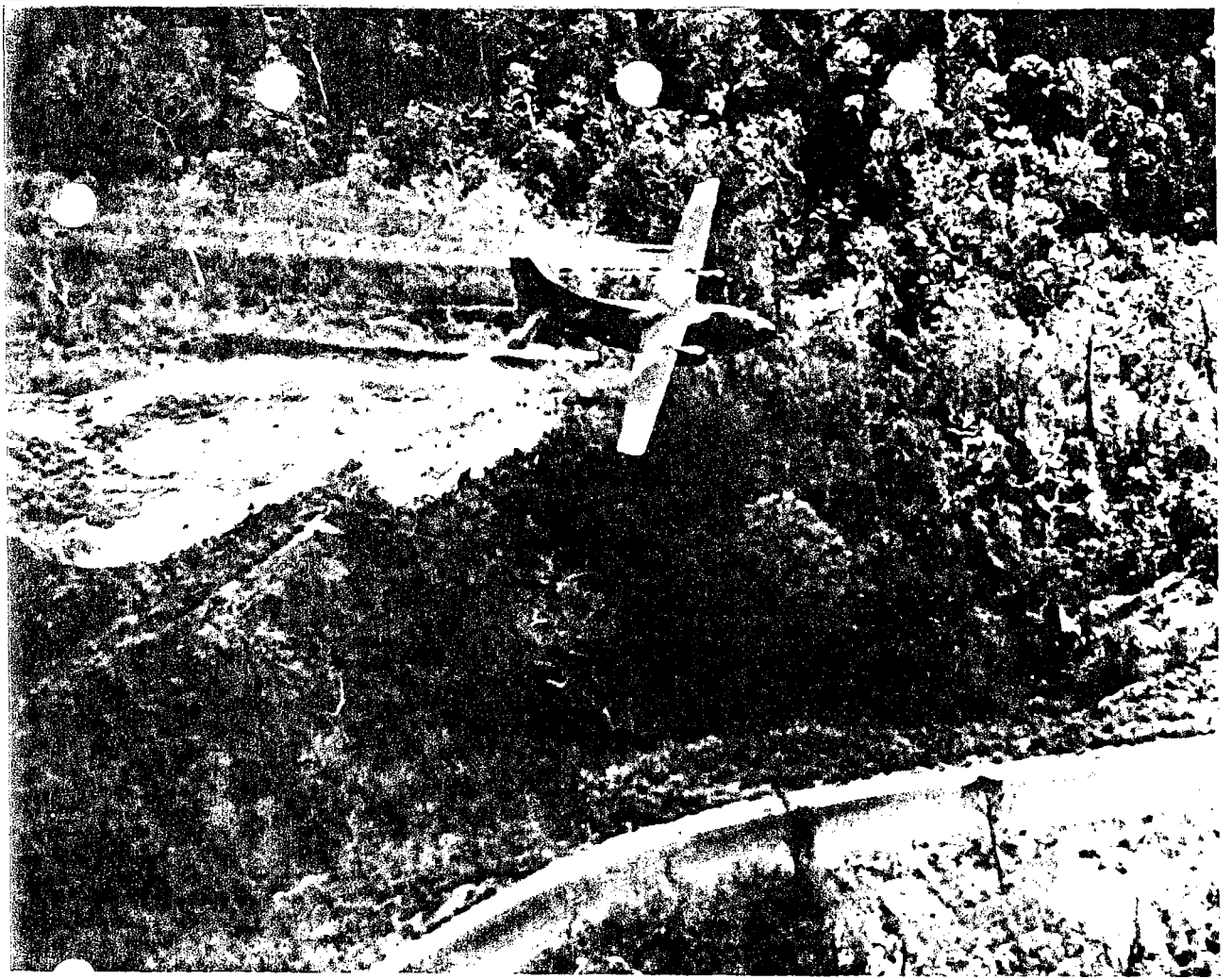
U.S. Army

CBW in Use

The 'Mighty Mite' is in effect a fan which can blow gases (*e.g.* CS, or in this case DDT) in a concentrated stream

U.S. Army

21104



CBW in Use

A U.S. Air Force C-123 *Provider* sweeps low along a road in mountainous central Vietnam on a defoliation mission.

USAF

methods and the possibilities and forms of inspection have also been studied, but the findings are so far only in a preliminary stage.

These are the main tactics of real defence against biological warfare. In my comments on the limitations of conventional defence against these weapons, I tried to make clear that a consistent and co-ordinated international effort leading towards the complete ban of these weapons is the only truly efficient way of defence of mankind against a disaster which will bring it back to medieval times where pestilence can decimate populations.

DISCUSSION

The major issues of the discussion were whether the state of biological weapons research had indeed reached the point where the weapons were technically feasible, and whether inspection techniques of the sort outlined by Academician Malek were likely to prove effective.

Dr Humphrey, while not denying that biological weapons could be developed to a stage where they could be useful strategically or tactically on a large scale, queried whether the difficulties which face their use had been solved for the simple reason that it is extremely difficult to try them out on man. All biologists are aware that one cannot argue from another species like guineapig or pig to man.

In the case of chemical weapons, we have fairly precise data on their properties and behaviour, but biological weapons are considerably less predictable. From the point of view of the field commander biological weapons fall into two categories: the first are those which carry a high rate of infectivity but which would not then normally develop, passing from person to person; and the second, those which rely for their effect on spreading, plague-like, through the population. An example of the former is the incapacitating Dengue fever which produces severe symptoms two or three days after infection, knocking out the fighting potential of the victims. Dengue fever would not, however, spread from person to person as it is a systemic disease, does not produce sputum, and is normally carried by an insect, the sandfly. In the absence of sandflies, therefore, the effects of the use of the weapon die out within a few days. This is one sort of weapon that has achieved a degree of popularity in certain circles on the grounds that it is a humane, non-killing weapon, but it is hard to put this theory to the test without involving a large number of people.

The other sort of biological weapon, analagous to the plague, is even more difficult to test. It is clear, however, that such weapons would have no military value until they had achieved epidemic proportions. In addition it would be necessary for the military commander to ensure that his own troops were specifically immunized.

Professor Meselson pointed out that if biological weapons do become militarily significant one would see it developing because the tests required would have to be under combat conditions. The chance of having an effective biological weapon developed secretly is very remote. The only way one can do the kinds of tests that would develop a predictable weapon would be under wartime conditions, and if that went on one would surely know it. Even development is a fairly conspicuous process, whereas simple manufacture of an already perfected weapon might be more easily concealable (the very conspicuousness of the process made it amenable to inspection techniques such as that pioneered by SIPRI).

But as both Dr Humphrey and Professor M. Pollock pointed out, one still knows very little about the real problems involved in dissemination, in maintenance of biological agents (other than anthrax spores) in a stable form, and so on. The problems are similar, but still more complex than those, outlined by Mr Perry Robinson, for chemical weapons, with the additional complication that the biological systems are self-reproducing ones. To Professor Pollock this lack of knowledge that anyone had of the possible spread and mutation of such infective agents would be enough to make any country think twice about their use, in case they rebounded. Dr Viney, by contrast, raised the spectre of the potential use of biological warfare agents against comparatively isolated targets—garrison islands or ships at sea—which could presumably be attacked with a fair degree of optimism that the attack would not rebound onto the attacking side.

A second point raised by Professor Pollock was whether it was really possible to distinguish between offensive and defensive research on biological weapons. Such a distinction seemed tenuous to him. It does not, for instance, seem possible to devise a maximally efficient defence against bacteriological agents without studying in detail the particular agents a potential aggressor might use. But, as Dr Viney pointed out, offensive research does not necessarily imply an offensive capacity; this might still not be necessary and provide a point at which control could be exercised.

The final issue concerned the feasibility of actually detecting the use of biological weapons. Professor Pollock thought the problem a greater one than Academician Malek had perhaps allowed. With the case of Korea in mind, unless one actually has the opportunity of finding evidence, for instance an aeroplane shot down filled with infected flies or a bacterial bomb clearly marked with its label of origin, would it not be extremely difficult to know definitely whether in fact biological weapons had been used?

But, as Dr Viney said, despite the difficulties of such an international inspectorate, it must not be dismissed even by the sceptics and hard-headed, because the real function of such a body would be quite as much to disprove allegations as to prove them. From the point of view of persuading politicians who are not likely to believe that a committee, in actual war circumstances, could do scientific work on the spot fast enough for it to matter (especially as it is quite likely that biological attack would come after a nuclear attack or after a conventional attack, so that the questions might have rapidly become irrelevant), it would still be very useful for such a body to exist for the purpose of disproving panic allegations or even malicious allegations. Hence the value of the SIPRI studies.

DISEASE	ORGANISM	INFECTIVITY	EFFECTS	SURVIVAL	TRANSMISSION
BACTERIAL DISEASES					
anthrax	Bacillus anthracis	20,000 organisms of ·:2μ inhaled	respiratory form normally fatal if untreated	spore forming highly stable	inhalation (also from animals) skin infection
·ucellosis	Brucella melitensis	high 1300 organisms ?	long lasting recurrent severe fever; rarely fatal	stabilized with dextrin and protein products	inhalation ingestion (also from animals)
cholera	Vibrio cholerae	low by ingestion	severe intestinal infection; sometimes fatal		ingestion (also from animals)
glanders	Malleomyces mallei	high 3200 organisms inhaled	in acute form, severe fever often fatal		inhalation ingestion (also from animals)
melioidosis	Whitmorella pseudomallei	high	normally fatal fever, producing mania and delirium		inhalation ingestion (also from animals)
plague	Pasteurella pestis	high 3000 organisms	very severe; often fatal		inhalation ingestion injection by fleas
tularaemia	Pasteurella tularensis	very high <50 organisms inhaled ?	severe fever; 5-8% fatal	unstable	inhalation ingestion injection by insects
'RAL DISEASES					
breakbone fever	dengue viruses	high single mosquito bite 2 organisms inhaled ?	most incapacitating fever known; very rarely fatal	high	injection by mosquito inhalation
mumps		high	incapacitating but not severe		inhalation
poliomyelitis		low	severe, permanent disability; sometimes lethal	85% survive 23h at 21-24°C as aerosol	ingestion inhalation of moist air
psittacosis		high	mild to severe fever; sometimes fatal		inhalation ingestion injection by insects
·allpox	Poxvirus variolae	high a 'few' organisms	severe; often fatal		inhalation ingestion
yellow fever		high single mosquito bite	Jaundice type fever; 30% mortality		injection by mosquito inhalation
RICKETTSIAL DISEASES					
Q-fever	Coxiella burnetii	very high 1 organism inhaled ? 10^{-6}g infected tissue	fever for 1 week; 1% mortality	stable	inhalation ingestion injection by ticks
epidemic typhus	Rickettsia prowazeki	high	severe; often fatal	poor	injection by louse inhalation ? ingestion ?
FUNGAL DISEASE					
coccidioidomycosis	Coccidioides immitis	1350 spores	mild to severe fever; rarely fatal	spore forming highly stable	inhalation
·XIN					
botulism	Clostridium botulinum	toxic dose 0·12μg ?	severe poisoning; 60-70% mortality	decomposes in 12h in air	inhalation ingestion

CHARACTERISTIC PROPERTIES of some biological agents are shown in the table above. The properties are those of the naturally occurring strains which can, of course, be altered; a highly infective strain of poliomyelitis, for instance, has been produced. Relatively few of the properties of the orga ·ns causing diseases such as tularaemia and yellow fever, for instance, wou ·ave to be changed whereas those of epidemic typhus are almost

VACCINES	THERAPY	STORAGE	EXTENT OF IMMUNITY	EPIDEMICITY	REMARKS
available	antibiotics	as spores	limited ?	low	one of the most stable agents
available in USSR	antibiotics	several months	?	low	affects both man and domestic animals
vaccine reduces severity and incidence	difficult		present to unknown extent	high	unlikely to be effective via water systems
unsatisfactory	antibiotics		limited	low	affects both man and domestic animals
unsatisfactory	difficult		?	low	very rare and little known disease
available	antibiotics		present to unknown extent	high	only pneumonic (respiratory) plague likely to be of BW use
available	antibiotics		present	none	good BW agents apart from doubts concerning stability
available	difficult	stable eight years at 41°F	present	low	might be useful as an incapacitating agent
mass produced	difficult		widespread	high	little use as BW agent because of widespread immunity
mass produced	difficult		widespread	low	limited by low infectivity and widespread immunity
unsatisfactory	antibiotics		present to unknown extent	high	birds act as reservoir of disease; immunity may be fairly wide-spread
mass produced	difficult	stable when freeze dried	widespread—on decline in Europe and N. America ?	high	generally immunity too widespread
mass produced	difficult	stable at 0-4°C	present	none	naturally a sub-tropical disease ; a strain which could survive in temperate climate might be dangerous
available	antibiotics	stable	present	none	very high infectivity
mass produced	antibiotics	difficult	present	high but cannot spread man-to-man	unlikely BW agent ; poor stability
unsatisfactory	antibiotics ?	as spores	present	low	highly stable ; suitable agent if a vaccine were produced
available as toxoid	difficult	bacteria as spores or toxin *in airtight* container	none	none	acts more quickly than any other BW agent ; troops could *invade after 24 h*

entirely unsuitable. Agents against which vaccines are in mass production are not likely to be used in BW; on the other hand, an agent would not be used in BW unless a vaccine were *available*. The psittacosis organism is no longer technically classed as a virus, but is included under that heading for convenience. Mumps is unlikely to be used in BW but is included for comparison.

5. Defoliants*

A. W. Galston

The programme

Chemicals are being used against forested and agricultural lands in Vietnam as part of United States military strategy and tactics. This is the first time that chemicals designed to damage or kill plants have been used in war. To damage or kill a plant may appear so small a thing in comparison to the human slaughter every war entails as to be of little concern. But when we intervene in the ecology of a region on a massive scale we may set in motion an irreversible chain of events which could continue to affect both the agriculture and the wildlife of the area—and therefore the people—long after the war is over.

The purpose of using herbicides, as stated by a U.S. Government spokesman, is twofold:

> Chemical herbicides are being used in Vietnam to clear jungle growth and to reduce the hazards of ambush by Viet Cong forces. . . .
>
> Destruction of food is undertaken only in remote and thinly populated areas under Viet Cong control and where significant denial of food supplies can be effected by such destruction. . . .[1] †

A precedent is being set which is important for two reasons: In the first place once chemicals become accepted weapons in the arsenals of nations, it may be difficult to draw the line between one chemical and another; between one use of chemicals and another. Secondly, the widespread aerial spraying of herbicides can affect the land, water, and living things which must support the people in peace or in war, in independence or under foreign domination, and whatever their form of government. It is this aspect of herbicide use which concerns us here.

Assistant Secretary of State Dixon Donnelly, the U.S. Government

* The basic data in this chapter first appeared in *Scientist and Citizen*, August–September, 1967. Used by permission.

† But *see* pp. 152–53.

spokesman quoted above, also said that the chemicals in use in Vietnam are "used extensively in most countries of both the Free World and the Communist Bloc for selective control of undesirable vegetation. They are not harmful to people, animals, soil or water." The implication of the word 'selective' is that these herbicides are also not harmful to plants other than the target species.

Less reassuring were the words of the twelve plant physiologists who wrote to President Johnson that the massive use of chemical herbicides could upset the ecology of an entire region. In the absence of more definite information than we now have about the effects of such use, they suggested that such an upset could be catastrophic.[2]

With two such diametrically opposite interpretations before us, an examination of what is known and what is unknown about these chemicals is in order.

American biologists who visited Vietnam in 1966 observed damage to a variety of plants.[3] This ranged from serious damage to fruit trees and other commercial plants in rural areas not deliberately sprayed to effects on various species in and around Saigon. Without a controlled study it is difficult to relate plant damage to source; in some cases other agents, such as jet-plane exhausts, may be partly responsible for the effects. There has also been some spraying with malathion for mosquito control. Species affected are listed in Table 6.

It is important to note that spraying is being done quite close to Saigon, and that plants are being affected within a range of about thirty miles from the target areas. The information contained in the Table comes from informal observations by scientists who have been in Vietnam. There appears to be no formal study of the effects of the spray programme on the many interconnected plant and animal species of Vietnam.

The fact is that even in limited and controlled conditions we do not fully understand the basis for the altered behaviour of a plant when we apply various chemicals from outside the plant. To add to the uncertainty, only a few species have been investigated intensively, and as we shall see shortly, species differ greatly in their response to synthetic growth regulators. As a result, when we spray a synthetic chemical from an aeroplane over a mixed population of exotic plants growing under uninvestigated climatic conditions—as in Vietnam—we are performing the most empirical of operations. We learn what the effects are only after we perform the experiment, and if these effects are larger, more complex, or otherwise different

from what we expected there is no way of restoring the original conditions.

The scale

The 12th Air Commando Squadron, in the first nine months of 1966 alone, defoliated a Vietnamese area of 1000 square miles, equivalent to the size of the entire State of Rhode Island in the United States, Derbyshire in Great Britain, or the entire state of Luxembourg.[4] Included in this area were about 70,000 acres of crops, mostly rice. The acreage of cropland rendered unproductive by sprays had risen to more than 150,000[5] by the beginning of 1967. In the early part of 1967 the special Spray Flight of the 309th Aerial Commando Squadron expanded operations to include the so-called demilitarized zone separating North and South Vietnam, as well as war zones C and D and areas adjacent to Laos and Cambodia, and during 1967 a total area of 965,000 acres was sprayed. This is expected to rise to 2 million acres in 1968.

The spraying programme, named 'Operation Ranch Hand', started in 1961 with two C123 transport planes, and has gradually grown to this much larger programme. The chemical most used is a mixture of esters of *2, 4-D* and *2, 4, 5-T*[6] released from high pressure nozzles beneath the wings and just under the upswept tail of the planes. Each plane carries about 1000 gallons of spray and approximately 3 gallons are released per acre, usually in the early morning, when the air is calmest and the danger of drifting is least. Within twenty-four hours the foliage begins to wither and turn brown. By the end of six weeks the leaves fall off the trees. For defoliation, reapplication is usually necessary at the beginning of each new growing season.[7]

In 1961 sixty spray sorties were flown; 1962 saw the number increased to 107, which included defoliation along canals and rivers in a large part of the heavily populated Mekong delta and a mountain pass near Qui Nhon, the port city north of Saigon. In 1966 combined defoliation and crop-spraying operations had increased to the point where approximately 1,324,430 gallons of herbicides were sprayed over more than half a million acres of land, and plans were to triple the programme.[6]

During 1967, 965,000 acres of land were affected, perhaps 450,000 acres of this being cropland. This would be about five per cent of South Vietnam's eight million acres under cultivation, and the programme has continued unabated into 1968.

The amount of herbicides being used is suggested by a *Business*

TABLE 6 Plants Damaged Outside Sprayed Areas (Vietnam)

FOOD CROPS AND COMMERCIAL PLANTS

Common Name	Genus (and Species when known)
Rubber-trees	Ficus elastica
Cassava	Manihot esculenta
Breadfruit	Artocarpus integrifolius
Papaya	Carica papaya
Starapple	Chrysophyllum cainito
Figs	Ficus
Sweet Potato	Ipomoea batatas
Beefwood, or South Sea Ironwood, cultivated for lumber	Casuarina
Mango	Limnanthes mangifera

PLANTS IN THE VICINITY OF SAIGON

(perhaps thirty miles from the nearest sprayed area)

Common name	Family	Genus (and Species when known)
Sweet cassava	Euphorbiaceae	Jatropha
Cassava	Euphorbiaceae	Manihot esculenta
Starapple	Sapotaceae	Chrysophyllum cainito
Piney tree	Guttiferae	Calophyllum
Willow	Salicaceae	Salix
Beefwood	Casuarinaceae	Casuarina equisetifolia
Papaya	Caricaceae	Carica papaya
Hibiscus	Malvaceae	Hibiscus
Flowering maple	Malvaceae	Abutilon indicum
Tropical or Indian almond	Combretaceae	Terminalia catappa
Petarboom	Leguminosae	Peltrophorum ferrugineum
Royal Poinciana (peacock flower)	Leguminosae	Poinciana regia
Silk Cotton Tree	Bombacaceae	Eriodendrum anfractuosum
Dogbane	Apocynaceae	
Sweet potato	Convolvulaceae	Ipomoea
Breadfruit	Moraceae	Artocarpus integrifolius
Figs	Moraceae	Ficus

Week item announcing the disappearance from the U.S. domestic market (except in small packages for home owners) of 2, 4-D and 2, 4, 5-T. The military demand is said to be four times present U.S. capacity.[8] Total production in 1965 of 2, 4-D and 2, 4, 5-T was close to seventy-seven million pounds.[9]

It was announced on July 11th, 1967, that contracts for $57,690,000 worth of chemicals for defoliation and crop destruction have been awarded by the Defense Supply Agency. The quantity of chemicals being purchased was not announced, but even allowing for some price increase the amount in dollars suggests a purchase of between six and seven million gallons. (The Air Force budget for the fiscal year that ended June 30th, 1967, provided $39,500,000 for about five million gallons. If prices remained the same the new contracts would be for more than seven million gallons,[10] according to official Pentagon figures.) Meanwhile the value of British exports of herbicides and associated substances to the United States has risen from $730,986 in 1964 to $2,739,949 in 1967.

Targets are selected by U.S. or Vietnamese officers and must be approved by a province chief, the Vietnamese Army general staff, the U.S. Military Assistance Command, and finally the American Ambassador.

Effects of defoliation

There are reports[6, 11] that *cacodylic acid,* an organic arsenic-containing compound, is being used against elephant grass and rice; both of these are the narrow-leaved type of plant which would be expected to be fairly resistant to the usual formulations of the 2, 4-D type of herbicide.

Of the three herbicides mentioned above cacodylic acid is probably the most toxic to man. According to the authoritative Merck index[12] it is *dimethylarsenic acid*, contains 54.29 per cent arsenic, and is extremely poisonous. The lethal subcutaneous dose in dogs is one gram per kilogram body-weight. (Other sources suggest that cacodylic acid may be even more poisonous with an LD_{50} of 184 mg/kg —*Ed.*)[14]* If the same toxicity held for man about seventy grams, or slightly over two ounces, would kill the average 150-pound man if administered subcutaneously.

Smaller doses could result in nausea, diarrhoea, headache, muscular pains, weak pulse, and coma. All these symptoms flow from

* *See* glossary.

the paralysis of capillaries and degeneration of the lining of the intestinal tract known to be induced by arsenic. In view of the persistence of this material and the cumulative nature of arsenic toxicity, its wide use certainly may pose dangers for the civilian population of Vietnam. And indeed, eye-witness reports from individuals in the sprayed areas have apparently described such symptoms (*see* Chapter 7). The additional danger that arsenic may be accumulated by plants which would be eaten by man must also be investigated, since such an effect would greatly magnify possible toxity. It has already been reported[13] that injection of cacodylic acid into trees can kill the bark beetle, *Dendroctonus*, infecting the trees.

Official statements have referred usually only to the less toxic 2, 4-D and 2, 4, 5-T, but Assistant Secretary of Defense, Cyrus Vance, when asked in 1965 whether arsenic and cyanide compounds were being sprayed over the rice fields of South Vietnam, replied, "We are making limited use of them in the southern part of Vietnam but not yet in the north."[14] It is hard to escape the conclusion that aerial spraying of cacodylic acid produces some harm to the human and animal life below.

The basic formulation for 2,4-D and related herbicides varies with the region, the climate, and the target plant. For example, the Dow Chemical Company product called Esteron R 245 O.S. designed "for the control of trees, bush, and broadleaved weeds" contains 67·7 per cent of 2,4,5-T. This material is usually mixed with either diesel oil, No. 1 or No. 2 fuel oil, or kerosene before spray application, although water sprays are sometimes recommended. About one gallon of Esteron per one hundred gallons of water is a usual mixture. If applied by aeroplane, the practice in Vietnam, about one to four pints of Esteron per acre is the recommended amount.

The label on this Dow preparation carries a "WARNING" on which the following statements are found:

> Do not contaminate irrigation ditches or water used for domestic purposes.
>
> Caution. May cause skin irritation. Avoid contact with eyes, skin, and clothing. Keep out of reach of children.

Another Dow product, called Formula 40 R, which contains alkanolamine salts of 2,4-D and is designed "for the selective control of many broad-leaved weeds in noncrop areas and in certain crops" carries a more definitive statement:

> Causes irritation of skin and eyes . . . In case of contact, flush eyes with plenty of water for at least 15 minutes and get medical attention; wash skin with soap and plenty of water. Remove and wash contaminated clothing before re-use. Do not wear contaminated shoes.

Herbicides have not been successfully designed to act against only a single plant or group of plants. If carried by wind, drift, or even vaporization they can destroy neighbouring plots of useful plants. The warning on Esteron states:

> Do not apply Esteron 245 O.S. directly to, or otherwise permit it to come into direct contact with . . . desirable plants which are sensitive to 2, 4, 5-T, and do not permit spray mists to drift onto them, since even minute quantities of the spray may cause severe injury during both growing and dormant periods. . . . Applications by airplane, ground rigs and hand dispensers should be carried out only when there is no hazard from drift. Do not apply by airplane in the vicinity of cotton, grapes or other 2, 4, 5-T susceptible vegetation. At higher temperatures, vaporization may cause injury to susceptible plants growing nearby. Do not graze to dairy animals within seven days after treatment (to avoid contamination of milk).

Effects on birds and animals were noted in a Reuter dispatch from Saigon which said that "Chemical sprays have played havoc with bird life, destroying vegetation and the insects on which birds feed. Monkeys and deer have also been affected."[15]

The extent of the damage that may be caused by the widespread and indiscriminate use of herbicides can only be approximated because of the paucity of direct experimental data. The reassuring statements about the innocuous nature of the herbicides in use in Vietnam are based on laboratory and field tests in the U.S.A. These have shown the selective nature of these chemicals—their effectiveness against one kind of plant, while leaving some other kinds unharmed. (Although, as noted above, there are limitations to this selectivity.) These tests have also demonstrated that 2,4-D and 2,4,5-T do not persist for a long time in the soil. A serious ecological upset is nevertheless possible.

The main points of concern are as follows:

The mechanism of action of the defoliants is imperfectly understood. We are therefore setting in motion on a large scale a biological process whose action and consequences we cannot altogether foresee or control.

Most experience with these herbicides has been in environments very different from that in which they are now being used. Their effect on individual plants indigenous to that part of the world has not been studied nor have we information about their movement through the food chains in that area.

While experiments performed in Great Britain and the United States indicate that numerous species and strains of common soil micro-organisms are effective in breaking down these compounds, in this process they are transformed into new compounds which may, under some circumstances, have deleterious consequences.

Let us look at each of these problems in more detail.

Mechanism of action

Defoliation with herbicides mimics the natural seasonal defoliation. Leaves of deciduous plants are attached to the stem by a narrow stalk called the petiole. Through this petiole runs vascular tissue which conducts water and nutritive materials into the leaf and organic matter out of the leaf. As long as the leaf-blade produces the hormone *auxin* in moderate concentrations the leaf remains on the plant. In normal concentrations auxin plays a useful role in various aspects of plant growth. If, for natural reasons, such as the shortening days of autumn, or for artificial reasons, such as injury to the leaf blade, the production of auxin in the leaf-blade ceases or slows down measurably, then a layer of large, weak, thin-walled cells which are easily ruptured is formed at the petiole and results in leaf fall. (This is called the abscission layer.)

The knowledge that lowered auxin levels in the leaf-blade result in leaf fall suggested to chemical plant physiologists that the deliberate application of auxin 'antagonists' or compounds which lowered auxin levels in the leaf could furnish a means of controlling leaf fall. This suggestion was put to work in agricultural practice many years ago when defoliants were applied to cotton plants several days in advance of harvest of the bolls, so that the mechanical cotton-picker would not be clogged by the undesirable leaves. The reverse practice, that of prolonging the retention of leaves and fruits on trees to promote better growth and ripening, can be accomplished by spraying with carefully controlled concentrations of substances with auxin activity. The concentration must be carefully controlled, because if it becomes too high undesirable side effects are produced which may even kill the plant. We will deal with these later.

There are still many gaps in our knowledge of the control of leaf

fall. (For example, there are three other classes of hormones (*abscissins, gibberellins,* and *cytokinins*) in addition to the auxins, which play a role—as yet unknown—in leaf fall.

Recent investigations[16] have revealed that high, toxic levels of auxins, which kill some plants and alter growth patterns in others, probably work through the control of the synthesis of still another plant hormone, *ethylene.* This substance, which can cause leaf fall, abnormal flowering, and altered growth patterns in various plants, has long been used to ripen fruits artificially by causing the sharp increase in respiration (the climacteric) which normally precedes ripening. Ethylene is normally absent, or present in very small quantities, in growing plants. The application of abnormally high auxin levels or some appropriate stimulus from the outside world can cause its production in the plant. Application of chemicals with high auxin activity, such as 2,4-D and 2,4,5-T, might have this result.

Ethylene is volatile and effective in several parts per thousand million of air; it can readily migrate from plant to plant and cause growth aberrations. The old observation that one rotten apple in a barrel causes them all to spoil is based on the fact that ethylene is produced in greater quantities in infected and injured fruit tissues; once produced it can induce adjacent fruits to ripen and spoil prematurely. Thus, however carefully a herbicide might be applied to a restricted area, if it gave rise to significant quantities of ethylene the results in neighbouring crops could be catastrophic.

Effect in the South-east Asian environment

2, 4-D and 2, 4, 5-T are especially effective against broad-leaved plants. In fact, this selectivity is so marked that 2,4-D is sold for application to grass lawns, where appropriate concentrations will kill dandelions, plantains, and other common lawn weeds without affecting the growth of the grass. Of course, as many home gardeners have discovered to their sorrow, raising the 2,4-D concentration somewhat can result in greatly inhibited grass growth. Thus, the same 2,4-D and 2,4,5-T which are being used extensively as tree defoliants may be used, with somewhat different, stronger formulation, for the purpose of killing or rendering unproductive various food crops, including rice. Unless carefully controlled, both as to area and concentrations sprayed, the jungle defoliation aspect of the herbicide programme in Vietnam can kill crops in areas where such an outcome is *not* intended.

In addition to the observations cited earlier, there is other

evidence that aerial sprays have done considerable unwanted damage as a result of drifting and volatization of sprayed materials. For example, it is widely known that rubber-trees at a Michelin plantation were inadvertently killed by drifting spray. The United States has compensated the French owners at about $87 per tree.[17]

On December 13th. 1965, three aircraft flew over Thoi An Dong, a village of the Phong Phu district, spraying defoliant extensively. Watermelon only 20 days from full maturity were seriously damaged, as were rice, vegetables, and fruits. Crops in the adjacent villages of Phuce Thoi and Tong Tuyen were also heavily damaged. The entire area was supposed to be 'secure' from the United States point of view, and thus should not have been sprayed at all. Con Son, an island half a kilometer from Can Tho, was subjected to defoliation treatment six times between June and December, 1965. Papaya, jack fruit, milk fruit, coconut, watermelon, mustard cabbage, and beans were adversely affected. At An Nghiep hamlet, beans, cabbage, and tomato were adversely affected. The damage ranged from 40 to 100 per cent, rendering the crops unprofitable for harvest. Some farmers also decided not to replant, thus amplifying the economic loss.[18]

Another problem is that herbicides may affect a susceptible link in an important food chain. For example, fish are an important element in the Vietnamese diet and are frequently bred in rice paddies.[19] Herbicides may not be directly toxic to fish, yet may affect them indirectly if they prove toxic to the micro-organisms on which the fish feed. The appearance of chlorophenols (derived from 2,4-D and 2,4,5-T) in a water supply, at concentrations as low as one part per thousand million renders the water unpalatable, and possibly harmful.[20]

The toxic levels of sixteen aquatic herbicides for the micro-crustacean *Daphnia*, which is a fish food and thus an important elementary link in the food chain of streams, lakes, ponds, and rivers, have recently been measured.[21] Among the more important conclusions are the following: (i) While herbicides are in general less toxic than insecticides, the high concentrations required for effective action suggest that several (not including 2,4-D) could be dangerous to *Daphnia* under field conditions. (ii) Lethal action may be delayed until long after the test compound has been removed. This suggests that previously determined inhibitory levels may be seriously in error. (iii) Mammals (such as laboratory rodents) cannot be used to estimate damage on *Daphnia*, and vice versa. (iv) ". . . even brief exposure of *Daphnia* to certain compounds

Environmental Alteration, under the chairmanship of David R. Goddard, was set up to examine both the MRI report and an analysis of it to be prepared by the National Academy of Sciences. Before either document could be examined the Pentagon released its own summary of the report, and through its director of plant science research at Fort Detrick, Dr C. E. Minarik, minimized the ecological impact of herbicides and defoliants used in Vietnam. Two articles quoting extensively from Dr Minarik appeared in articles written by Walter Sullivan in the *New York Times* of January 4th and 7th; Dr Minarik was also heard over CBS news, repeating the same message. Clearly, the Pentagon felt some need to respond to the challenge.

The Pentagon's *Summary Digest of the MRI Report* did reveal that relevant scientific studies in the ecological area are very scanty and that practically no scientific reports dealing with the Vietnamese area's response to applied herbicides are available. They also admitted to the use of another compound, *picloram,* a derivative of *picolinic acid,* which the Dow Chemical Company's own house organ *Down to Earth* reveals is remarkably long-lived in the soil. In one study, less than 5 per cent of the picolinic acid derivatives had disappeared from soil in well over a year. The D.O.D.'s comment on this is "Picloram . . . is persistent in soils but will tend to leach to depths of two to four feet under average rainfall and soil conditions." In commenting on the danger that humus removal from tropical soils may reduce the soil to an impervious rock (an almost irreversible process known as laterization, and a risk of the defoliation campaign which had been expressed by several scientists), the summary says: "No evidence has been obtained that such irreversible changes have resulted in areas in Vietnam subject to defoliation. Observers in Vietnam have indicated that the vegetational succession following defoliation in tropical forests is one in which grasses rapidly cover the ground in dense stands followed by rank growth of weeds and vines which are effective in minimizing soil change." If this is true, soil laterization is probably not a danger . . . but, then, the entire purpose of the defoliation operation is vitiated by an even denser ground cover than existed before.

The National Academy of Sciences assigned the report to Dr A. Geoffrey Norman, head of the Division of Agriculture of its affiliate, the National Research Council. Dr Norman, himself former head of plant research at Fort Detrick, in turn appointed a committee composed of four academic people, one U.S. Department of

Agriculture researcher, and the director of plant science research at Dow Chemical Company to advise him. Their report, forwarded to the D.O.D. on January 31st, 1968, was called "only a first step in investigating further the ecological effects of intensive use of herbicides" by Frederick Seitz, president of the National Academy of Sciences. In the *New York Times* of February 13th, 1968, Walter Sullivan again wrote an article under the headline "Defoliation Study Casts Doubt on Long-Term Damage in Vietnam" indicating that: "There are no clear indications that widespread aerial spraying to strip Vietnamese war zones of foliage will do long-term damage." He admitted that too little is known to have confidence in these conclusions and that all data were gathered from literature review and interviews, rather than on-the-spot experience. Quite a different picture was painted by Thomas O'Toole of the *Washington Post* who wrote on February 20th, 1968, under the headline "Report paints grim picture of Defoliation in Vietnam": "The chemical defoliation of Vietnam's jungles and forests may be doing permanent damage to its wildlife, soils and streams, a Pentagon-commissioned report acknowledges. At a minimum, two rare species of monkeys and four other animals are in danger of extinction because of the spray programme. The danger of soil laterization is present, and damage to water resources cannot be excluded." These contrary views of the same report summary indicate the need for continued study of this complex situation.

As of now, we await the AAAS evaluation of the full report. But whatever the outcome, the decision, it is clear, will be based on insufficient data. To quote the report, "The extent and pattern of herbicide treatment in Vietnam have no precedent. Therefore, it is difficult to predict the effects with any accuracy."

6. Starvation as a Weapon* Jean Mayer

A United States Government spokesman explains that food is as important to the Viet Cong as weapons, and that herbicides are used "where significant denial of food supplies can be effected by such destruction."[1, 2]

There is as yet little direct evidence from Vietnam of the effects of crop destruction on the NLF, no data on starvation of persons who can be categorically defined as 'Viet Cong', no reports that NLF prisoners have been found to be physically incapacitated by malnutrition, no clear evidence of a lessening of the NLF's will to fight. Yet it is clear that malnutrition is common among Vietnamese civilians, whether due to diet deficiences unrelated to the war, to food problems resulting from other war conditions, military and economic, to the conscious efforts of denying food to the NLF, or to a combination of all three.

Information justifying the programme has never been released by military or other U.S. Government sources. In the absence of such data we must turn to other, less direct information and to historical inferences. In spite of the paucity of information from Vietnam, the effects of food denial as a weapon are no mystery. We can turn to well-documented sources for answers to the questions: How does a food shortage affect a population? Which elements of the population are most affected? Is starvation an effective strategic weapon? The answers to these questions can then be related to the situation in Vietnam.

The effects of starvation on the human body are well known and were described in detail in a number of populations immediately following World War II. Famine affects different elements of the population in different ways and to different degrees; this has been observed in famines occurring in peace-time as well as in war. The author has personally observed famines on three continents, one of them Asia.

* The basic data in this chapter first appeared in *Scientist and Citizen*, August–September, 1967. Used by permission.

Finally, although herbicides have not been used in previous wars, the creation of famine through blockade has been frequently used, and there is historical evidence of its effects.

Effects of Starvation

The first and most obvious effect of starvation on the human body is the wasting of its fat deposits. A nutrition survey of South Vietnam in 1959 found that the average weight of civilian males was 105 pounds,[4] suggesting that such body-fat deposits would generally be meagre in Vietnamese to start with.

The stomach and intestines, heart and lungs are affected next; the size of the liver is drastically diminished. The intestinal lining becomes thin and smooth, thereby losing some of its absorptive capacity, and diarrhoea results. Thus starvation is a self-accelerating process, particularly in children; because of intestinal damage, the food that is available is poorly absorbed, undernourishment increases correspondingly. The damaged lining of the stomach fails to secrete hydrochloric acid, which is important for digestion. Both blood pressure and pulse rate fall.

Early effects of starvation are cessation of menstruation in women and impotence and loss of libido in men. Hair is dull and bristling, and in children abnormal hair grows on the forearms and back. The skin acquires the consistency of paper and not infrequently shows the irreversible dusty brown splotches which are permanent marks of starvation. In extreme cases, particularly among children, the lips and parts of the cheeks are destroyed. The body becomes susceptible to infection and disease. The psychologic state deteriorates rapidly; the individual becomes obsessed with food, mentally restless, apathetic, and self-centred.

A recent paper prepared by U.S. Physicians for Social Responsibility, for Senate hearings on the refugee problem, summarized the medical problems in South Vietnam.[5] It pointed out that malnutrition is widespread among South Vietnamese civilians; beri-beri, night blindness, and anaemia are found frequently; Kwashiorkor, a form of protein malnutrition, occurs and is a major component of the problems of wound-healing and resistance to infections; infant and child mortality is high. Kwashiorkor is a deadly disease affecting children after weaning. It causes degeneration of the liver, pancreas, and intestines, oedema, and eventually death. Diseases associated with malnutrition, such as tuberculosis, are rampant. Although it is impossible to know to what extent these problems stem from the

crop-destruction programme, there can be no doubt that if the programme is continued these problems will grow.

In many parts of South-east Asia, there are food shortages in the best of times, and any strain on the food supply, whether from political factors or natural disasters, may result in famine. A general consequence of famine is the social disruption, including panic, which accompanies it. Starving people attempt to journey to other areas where they hope to find food, and chaos increases. Weakened by lack of food they are susceptible to disease, and these factors interact with one another; disease adds to social disorganization which in turn makes disease more difficult to combat.

In Vietnam, migration has been set in motion by military attacks, or fear of such attacks, on villages or towns, and by the destruction of agricultural lands. At Senate hearings on refugee problems in South Vietnam and Laos, Frank H. Weitzel, Acting Comptroller General of the United States, gave the number of refugees in South Vietnam in the fiscal year 1965 as 600,000 six times what had been expected.[9] In November an additional statement from Mr Weitzel gave a total of 719,000, but said 258,000 were classified as resettled. At the end of 1966, the *New York Times* put the figure at a million, growing at a rate of about 70,000 a month.[10] A news item on July 3rd, 1967, states that almost two million refugees were now in government resettlement camps—one in every seven South Vietnamese.[11]* These recently uprooted people are a different population from the 1955 refugees from North Vietnam who, according to the South Vietnamese Government, are now resettled. In 1965 refugees were almost 100 per cent women, children, and older men.[12]

Twenty-six years of almost uninterrupted war have placed strains on the food supply, especially severe in the last two years. South Vietnam, which exported 49,000 metric tons of rice in 1964,[13] must now import it. Figures for 1966 are not yet available, but 240,000 tons were imported in 1965.[14] At the time of writing infant mortality is estimated at 25 to 30 per cent, more than ten times that of the United States or Britain. Maternal death rate is twenty-five times that of the United States. Life expectancy at birth is about thirty-five years. In an environment where sanitation is primitive and medical facilities are in short supply, a great additional hazard is the risk of epidemics which can grow like wildfire in a weakened, starving, and migrating population.

* More recent figures do not appear to be available; but the aftermath of the Tet offensive must have greatly increased them.

Bubonic plague is endemic, and although only eight cases were reported in 1961, the number is said to have risen to 4500 in 1965.[5] Malaria is also endemic, and the appearance of a form of the disease which does not respond to traditionally effective drugs is a matter of grave concern. Cholera and smallpox have been habitual fellow-travellers of Asian famine with influenza and relapsing fever also frequent. In 1965 the number of cases of cholera in Vietnam increased by 25,000 according to the World Health Organization.[15] In that same year Dr Howard Rusk reported that among refugees "tuberculosis is highly prevalent, as are skin infections, intestinal parasites, trachoma, and other diseases of the eyes, typhoid, and leprosy."[16]

A study of three examples from wars fought within the past hundred years goes a long way towards indicating the effect of food denial as a weapon.

THE SIEGE OF PARIS. Paris was under siege by the Germans for 129 days in 1870–71, during the Franco-Prussian War. One of the reasons given by the Government for surrender was the lack of food within the city. However there was a desperate military situation in the rest of the country. Prior to the siege one of the main French forces was defeated at Sedan; during the siege the other was defeated at Metz. Thirty-six new divisions were organized and equipped from Tours, but a number of them were driven into Switzerland, where they were disarmed and interned.

According to Baldick,[17] the total number of deaths in Paris rose from 3680 in the first week to 4465 in the third—and presumably rose higher as the siege dragged on for eighteen weeks. The winter was severe and people suffered from cold as well as hunger; epidemics swept the city, with smallpox the biggest killer.

Melvin Kranzberg describes the effect of the food shortage on the people of Paris: "With the exception of the dent made in their pocket-books the rich did not suffer from famine during the siege. . . . As for the poor, the men were not badly off, but the women and children suffered. The men could get enough to eat and perhaps too much to drink merely by enlisting in the National Guard."[18]

BLOCKADE OF THE CENTRAL POWERS. In the early days of World War I, the Western Allies were optimistic that the hunger engendered by the blockade of the Central Powers—Germany, Austria-Hungary, and the smaller countries allied with them—would help win the War quickly. After the War, the importance of the Blockade may have been exaggerated by German historians in order

to play down military defeats; it may have been underestimated by British, French, and American historians. The fact remains that it took four years of the combination of blockade and military action to defeat the Central Powers.

Famine oedema, a relative increase in the water content of the body, was observed in civilians in Hamburg in the winter of 1916–17, in Berlin in January, 1917, and in Vienna and the Rhineland later the same year. In 1918 it became common throughout Central Europe. Tuberculosis, which is closely related to malnutrition, began to rise in 1914 and continued to rise throughout the War. In Vienna, the mortality rate from tuberculosis rose almost 100 per cent; in Germany, 44 per cent.[19] The excess of deaths in the civilian population during each of the War years over the number of deaths for the year 1913 totalled 762,796 (*see* Table 7).

These figures represent the number of deaths which under 'normal circumstances' presumably would not have happened. They were probably due to a combination of food shortage with other factors. Medical care of civilians suffered because of the army's drain on medical personnel and facilities. There was a shortage of fuel because importation of coal was reduced by the Blockade and internal distribution was disrupted by the War. Although most of the War was fought on French and Russian soil, the Austro-Hungarian Empire was invaded, with some if its villages and countryside becoming a battleground.

If the figures below are compared with deaths in the army it can be seen that civilian deaths in excess of normal may have been about half as great as the army losses. However, it must also be said that the very war conditions which cause excess civilian deaths make reliable statistics difficult to assemble.

George A. Schreiner, an Associated Press correspondent, spent the first three years of the War in Germany and the nations allied with it, including considerable time with the armies on both Eastern and Western fronts. He states that many men in the army received better food than they had as pre-war civilians. He says that the army "came first in all things", and that when it became necessary to reduce the bread ration, this was made good by increasing the meat and fat ration.[20] Schreiner quotes a "food dictator" as saying that thousands of the poor aged were going to a premature death.

THE SIEGE OF LENINGRAD. The most recent, the most lethal, and yet the most completely ineffective use of starvation as a strategic

TABLE 7[20] Deaths during Blockade of the Central Powers (1914-18)

	Deaths in the Army		Excess deaths in the Civil Population
	On the battlefield and through wounds	Through sickness	Due to the Blockade (presumably)
1st War Year	451,506	24,394	88,235
2nd War Year	330,332	30,329	121,174
3rd War Year	294,743	30,190	259,627
4th War Year	317,954	38,167	293,760*
Post-war due to the war	62,417	10,902	
Total	1,456,952	133,982	762,796

* To end of 1918.

weapon of war was the siege of Leningrad by the Nazis in World War II. It closed around the three million people of the city on September 8th, 1941. For four months only 45,000 tons of food were brought in by water, air, and finally by the road across the ice of Lake Ladoga, and this was expected to sustain the military as well as the civilian population. Late in January, 1942, a corridor was opened which permitted both the importation of food and the evacuation of large numbers of people, but the siege was not completely lifted until two years later. By this time almost a million people—about a third of the city's population—were dead from hunger, cold, and their attendant diseases, and from the bombing and shelling of the city.

As in the previous cases, the soldiers defending the city had better rations than the civilians, although their rations, too, had to be cut in November, 1941, when things were at their worst.[21] Hospital records for the starvation period show some of the effects on infants and pregnant mothers: an increase in stillbirth and premature birth and a rise in neo-natal mortality.[22]

The early and worst parts of the siege were accompanied by German victories elsewhere in the nation; German armies came within a few miles of Leningrad homes and factories where people

continued to live and work. Nevertheless, the troops, besieged along with the city, defended it successfully and eventually broke the blockade.

While historians differ in assigning significance to these blockades as effective military techniques, it is clear from all three of these examples that food denial in war affects the fighting man least and last, if at all, and is therefore unsuccessful unless accompanied by military victories by the blockaders. It is hardest on civilians, particularly children and the elderly; where economic class divisions are sharp, it is particularly hard on the poor.

Destroying food in Vietnam

The increasing use of herbicides in Vietnam referred to in the previous chapter suggests that the U.S. Military plans to enlarge the area where food crops will be destroyed.

News stories have reported other methods being used in the food-denial campaign. In areas under the political control of the NLF, U.S. and South Vietnamese troops may establish temporary military control long enough for a 'harvest protection' operation. This is carried out by entering the area at harvest time, holding off NLF rice-collectors, while peasants are required to sell their surpluses to the government or to the commercial market, and then withdrawing.[23]

The agricultural area in the demilitarized zone and just to the south of it has been rendered completely unproductive, as have special areas in the immediate proximity of Saigon (Operations Junction City and Cedar Falls in the 'iron triangle'). As many as 600,000 Vietnamese have been removed from agricultural productive labour and at the time of writing are residing in camps.[24]

Rice that has already been harvested may be destroyed. Sometimes it is dumped into large pits and covered with shark repellent or other obnoxious compounds; attempts have been made to burn or scatter it. Captured rice has been dumped into the Rachbenggo River by U.S. troops.

According to General William W. Berg, U.S. Air Force Deputy Assistant Secretary of Defense, "Our combat units are well aware of the food shortages in South Vietnam and are not wantonly destroying captured rice whenever it can be salvaged and put to local use. However, in a fluid combat situation, available time, manpower, and transportation will not always permit removal of captured goods to a safe area."[25]

Charles Mohr has reported in the *New York Times* that the

troops have found rice to be "one of the most maddeningly indestructible substances on earth. Even with thermite molten-metal grenades, it virtually will not burn. The scattering of rice does not prevent its collection by patient men."[25]

These practical difficulties suggest one reason for the use of chemical sprays. Another, and perhaps the most important reason, is that it entails a more efficient use of personnel.

"What's the difference between denying the Viet Cong rice by destroying it from the air or by sending in large numbers of ground forces to prevent the enemy from getting it?" a Pentagon spokesman asked. "The end result's the same; only the first method takes far less men."[26]

Whatever method is used, the examination of past wars and famines makes it clear that the food shortage will strike first and hardest at children, the elderly, and pregnant and lactating women; last and least at adult males, and least of all at soldiers.

That these conclusions have applied to Vietnam as well is suggested by the Vietnamese nutrition study, carried out in 1959 by Americans and South Vietnamese under the latter's Committee on Nutrition for National Defence, in which an equal number of army and civilian Vietnamese were compared.[27]

"In the general sense, the nutritional status of the military is superior to that of the civilian population, without appreciable differences between Army, Navy, and Air Force," says the study. While the average civilian male weighs 104·3 pounds, his counterpart in the military weighs 113·0 pounds. And, for those who might suppose the difference results from military selection procedures favouring bigger men from the general population, the study reports:

> Inductees (Quang Trung) weighed 107 pounds on the average, the lowest weight among any of the military. A group of similar men completing their basic training (Quang Trung) has an average weight of 114 pounds, suggesting that the change from a civilian to a military diet resulted in a prompt weight gain, in spite of the strenuous activity of basic training. Considering the combined military services, continuation of such weight gain during the first year of military life was further evidenced by the weight gain from an average 107 pounds for those in the service less than three months to an average of 118 pounds for those with six months to one year of service.

South Vietnamese army medical care was also superior to that available to civilians, as Dr John Reed of the U.S. Public Health Service testified on his return from working with Vietnam refugees:

". . . there are only about 800 qualified physicians in the Republic of South Vietnam. Of this 800, 500 are in the military service. Of the remaining 300, approximately half, or 150, are in private practice in Saigon, so this leaves only about 150 doctors for the entire rural population in South Vietnam."[28]

This refers to the South Vietnamese government side, but on the other side, NLF soldiers were likewise expected to get the fighter's share of whatever food there was. Whether extra rations were enforced by an organized government structure or confiscated by armed bands of guerillas, the end result was the same. Unless direct evidence to the contrary from U.S. observations in Vietnam is forthcoming, this conclusion seems unavoidable: from a military viewpoint, the attempt to starve an enemy can be expected to have little or no effect. What it can be expected to do is to add to the flow of refugees already far beyond the capacity of the programme designed to care for them.

The history of modern war has been one of increasing involvement of civilians. Starvation as a weapon is an aspect of such involvement, one which has the peculiar property of inflicting suffering on civilians while doing little damage to the military. To destroy crops—with herbicides or in any other way—is therefore to employ a weapon whose target is the weakest element of the civilian population.

I would strongly suggest that the proscription of famine as a weapon, whether by chemicals, through food destruction, fire, or mechanical methods, or through food denial by blockade, seizure of foodstuffs, or imposed excessive rationing, be the subject of a separate international convention. The manner in which food is destroyed or denied is less important than the final result. Abandonment of this method of waging war should be all the easier in that it is cruel while at the same time of no definite military value.

DISCUSSION

In the discussion to this Session, Dr Lindop suggested that international scientific pressure, such as through a group of scientific assessors, from the International Confederation of Scientific Unions (ICSU), the major international scientific body, could report on the

defoliated areas and predict the possible damage produced. Dr Galston agreed that this would be desirable, and that the more pressure that was put on the Pentagon in particular, the more the chances that some teams of inspectors would be sent out there. There had been a few visiting scientists sponsored by the Department of Defense to survey the effects of drifting chemicals and to take a look at possible upsets in the ecology and disappearance of certain rare species. The argument that was used by the Department of Defense against the journey to Vietnam of experts who might like to take a look for themselves is that a great many of these operations had been carried out in areas controlled by the NLF, and that the U.S. Army could not then in good conscience release a group of people into an area over which they had no control militarily. Dr Lindop pointed out, however, that International Red Cross inspection teams were sent into combatant areas without guarantee of safety. The influence of an international team would be immense.

02112

PART TWO

CBW IN USE

7 Vietnam — M. F. Kahn

8 The Yemen — M. Meselson & D. E. Viney

7. Vietnam

M. F. Kahn

Sources

In this report I shall deal mostly with practical aspects of the use of CBW in Vietnam. I will also report on my personal experience as an investigator for the International War Crimes Tribunal (IWCT) set up by Bertrand Russell, and also as a member of the Scientific Commission of the Tribunal.

I would like first to describe the methods used by the Tribunal investigators and also the sources of the information made available to us.

Our sources were as follows: (i) Reprints or photostats of the main U.S. reports and papers, appearing in both the general and technical American press, dealing with CBW in Vietnam. (ii) Texts of all the dispatches issued by American war correspondents in Vietnam, including those which were not (for whatever reason) published by the news agencies. (iii) Official reports of the Commission of Enquiry on the American War Crimes in Vietnam, a body set up by the Democratic Republic of Vietnam (DRV) (North Vietnam) with a technical appendix of material evidence and numerous pictures. (iv) Official reports of the Committee for the Denunciation of War Crimes Perpetrated by the U.S. Imperialists and their Puppets in South Vietnam, an organ of the NLF of South Vietnam with technical appendix of material evidence and pictures. (v) Reports of the investigation teams, sent by the IWCT to both the DRV- and NLF-controlled areas of South Vietnam. (vi) Reports of the members of the Scientific Commission of the IWCT, including a technical appendix and results of chemical analyses and of animal experiments.

I shall add to this documentation data collected through my personal experience as a member of both investigation teams in the DRV and South Vietnam (NLF-controlled areas) and as a member of the scientific commission.

Comment on the use of Napalm and Phosphorus

Although napalm and phosphorus are not in the strict sense chemical or biological weapons (see chapter 3) since they are mainly incendiary weapons, it is worth pointing out that this classification is somewhat arbitrary.

As far as napalm is concerned, numerous reports show that this compound, when burning, gives rise to large amounts of carbon monoxide. This happens mostly when napalm is burning indoors, when its combustion is incomplete. Thus there are reports of Japanese soldiers killed during World War II by napalm without any visible burns. While in South Vietnam, I had the opportunity to observe the following case: M. Nguyen Van Ba, male, 36 years old, had received a small amount of napalm while trying to protect himself in a shelter during a U.S. Air Force attack over the village of Ta-Bang (Province of Tay-Ninh) in 1966. He had felt a burning on his hand, and then became comatose for several hours. When he awoke, according to the people who discovered him, he was in a bad state of complete disorientation and had hallucinations. Since then he had had a tremor with hypertonia, exaggeration of postural reflexes, and complained of significant loss of vision. On examination I found a very large concentric narrowing of his eye field on both sides; these symptoms are suggestive of the results of intoxication, similar to domestic gas poisoning. The burning scar was very tiny, and the effects induced by this burning were obviously not sufficient to explain his state; other people with him in the shelter were found dead.

It is worth pointing out that carbon monoxide intoxication greatly increases the lethality of napalm since it prevents the victim from escaping the fire.

As far as phosphorus is concerned, as well as horrible burning this compound causes a severe intoxication and hepato-nephritis (liver and kidney poisoning), which in most cases is fatal, even when the burning appears superficial. In fact, phosphorus penetrates deeply into the skin and the subcutaneous tissues, since it produces phosphoric acids which are very acidic, and spreads all over the body.

In addition to these toxic effects on man one has to consider the effects on cattle, poultry, and fish. Fish forms the bulk of the protein intake of the Vietnamese peasant, and poisoning results when a phosphorus bomb or shell falls into any lake or area of water used for breeding fish.

Defoliants and Herbicides

Chapters 5 and 6 have already discussed these compounds and their use in Vietnam. I shall mention only some particular aspects of their use which I noted during my investigations in South Vietnam.

TARGETS. The province of Tay-Ninh, where I stayed mostly, had been heavily attacked by defoliants. I saw large areas of rice fields and of jungle which had previously received amounts of defoliant. Over the jungle the results of the attack were hardly noticeable. I never saw the cover provided by the leaves completely destroyed. However I did see in some places an abnormal amount of freshly fallen leaves on the earth and occasionally some more heavily damaged trees, proving that the defoliants had been used. Most of the places attacked in the jungle areas were in fact on the edge of the jungle close to the rice fields. On the other hand, I saw heavy damage caused by defoliant on plantations including trees and papayas. Dessicated leaves, swollen, cracked, and fissured stems, and stunted fruits unfit for eating were evident. The witnesses who testified to our Commission were unanimous in their accounts of the spraying of chemicals and the resultant damage to crops. As far as the rice fields were concerned, our evaluation was less precise as the results of the chemical spraying were mixed with the results of neglect caused by the non-stop land and air bombardment in the 'free-killing zones', but here, too, the testimony of the Vietnamese peasants was unanimous concerning the damage caused to the rice by the chemicals.

Thus, for the investigation team on the spot it was beyond doubt that concentrated attacks on the food supplies of the Vietnamese people was the main target of the so-called defoliants in Vietnam. The obvious ineffectiveness of the chemicals on the declared target —the jungle—contrasts sharply with the relative efficiency against crops and food and trees.

TOXICITY OF THE DEFOLIANTS FOR HUMANS AND ANIMALS. Assistant Secretary of State, Dixon Donelly, quoted by Galston in Chapter 5, said that the chemicals in use in Vietnam ". . . are not harmful for people, animals, soil or water". Despite this, several dozen witnesses heard by our Commission in South Vietnam told us of the physiological troubles they underwent following the spraying of chemicals. In most of the cases it is true that these troubles were relatively mild or transitory in adults. But we were told about lethal

cases occurring among children. In fact adults take care, being informed by the medical services of the NLF, to avoid eating fruits and herbs after chemical spraying. But children do, on occasion, eat fruit coated with chemicals. Miss Thuy-Ba, M.D., chief of the medical staff of a provincial NLF hospital, described to us a lethal case she observed. A five-year-old boy was brought to the hospital after he had eaten contaminated fruit. He had severe abdominal pain, vomiting, then diaorrhea with blood in his stools, followed by collapse and death. On pathological examination, the post mortem revealed disseminated necrosis of the intestinal mucosa.

We collected numerous reports of the heavy losses inflicted on cattle and poultry by the chemicals. Buffaloes, pigs, hens, and ducks died by the dozen after spraying. These animals—like the children—were not reluctant to eat foodstuffs, even heavily coated by chemicals.

The Commission of Inquiry of the DRV has carried out experiments to test the animal toxicity of these compounds, and in a first set of experiments the animal tested was the duck. The chemical was DNOC (Dinitro-ortho-cresol). Each animal weighing 0·75 to 1·0 kg. received either 20 or 100 mg. of the substance. Death subsequently occurred from 5 minutes (120 mg.) to 14 minutes (20 mg.). Pathological findings in these animals revealed extensive damage to the digestive tract with haemorrhages. One has to bear in mind that an amount of 20 to 100 mg. may easily* be ingested by animals feeding in a chemical-coated area.

Thus localized vitamin deficiencies were sometimes associated with these symptoms, and one has also to consider the possible, yet unpredictable, delayed consequences not only for the exposed people, but also for their descendants.

Gases in use in Vietnam

THEIR NATURE AND DISSEMINATION METHODS IN USE. Despite the first official denials concerning the use of gases in Vietnam, the use of CN (chloroacetophenone); DM (adamsite), an arsenical compound; and CS (ortho-chlorobenzalmalononitrile) has now been admitted by the Americans, and there is a persistent report that the

* Some of the conference participants were less certain that it would be easy to build up an intake of quite so large a dose as this. However there now exists, in the French literature, a case of the near fatality of a small French girl and the death of her dog following the use of 2, 4,-D as a herbicide near Lille, France.[14] *Ed.*

hallucinogen, BZ, was used on at least one occasion, in Bong-san, in March 1966. The method of dissemination of these gases calls for further comment.

It has been reported that the chemicals are used mostly in the form of aerosols.[11] In fact, at least for CS on which I was able to collect precise data and material evidence, the methods of dissemination involve the dispersion of solid particles which undergo direct sublimation from solid to gas. They are not dispensed as droplets, or true solution dispersed with an aqueous phase, which is the definition of a true aerosol. In the projectiles, such as hand grenades of either cylindrical or spherical form, CS is present as a powder which is disseminated in small particles by the explosion of a standard detonator. This detonator is the same as the detonator of the explosive hand grenade. The heat caused by the explosion helps the sublimation of the solid compound. This factor is important when attempting to calculate the concentration obtained by the use of these projectiles in an enclosed space.

Another way of dissemination which is especially adapted to a closed area is the dispersion of the powder by means of a high velocity wind machine, nicknamed 'Mighty Mite' by the Americans. This machine provides a very powerful air stream, and can very easily build up high concentrations of gases in caves and shelters.

In some cases gases are used in the open air, thrown in cluster canisters for example, or in numerous small projectiles dispersed by a special type of multi-barrelled portable mortar. One of these was shown to the IWCT in Roskilde by the North Vietnamese delegation. But reports from the American side acknowledge the fact that this kind of use was rather ineffective because of the quick dispersion of the gas in the atmosphere and the fact that Vietnamese troops were able to protect themselves with gas-masks.

But it is the alternative use of the gases which is more disturbing —their injection into shelters and caves to force out people from their safe underground protection. In fact, as many reports have confirmed, virtually the whole population (both soldiers and civilians) is obliged to take protection in caves or underground shelters to escape shells, bombs, and the less conventional weapons such as cluster-bomb units, napalm, and phosphorus.

When gases are thrown into such shelters a condition is obtained which differs completely from that following use in the open air. All the data available for CN, DM, and CS are calculated for low concentrations obtained by dispersion in the open air. But it must be emphasized that when powder is thrown into a closed area

much higher concentrations build up. In fact, these concentrations cannot actually be calculated by the users of the gas. For a given dose the concentration of gas in the air depends on temperature, humidity, and above all, the volume of air into which the gas is dispersed, and it is obvious that this volume cannot be either calculated or even estimated by troops using the gas.

When applying the gases soldiers inevitably pump in the maximum amount they feel possible. Such machines as 'Mighty Mite' provide the possibility of blowing in more than 22 lb. of CS into one shelter. A very high concentration of this compound within the shelter can easily build up.

ARE THE GASES 'HARASSING' OR LETHAL? This point has been discussed, particularly by the Americans themselves, in the context of the fact that 'riot-control' and tear gases are not prohibited by the Geneva Convention. However, some semi-official U.S. sources acknowledge that even if a gas is formally labelled as 'harassing', death may sometimes result from their use. Thus in his book *Tomorrow's Weapons*[1] Rothschild states that CS has a *low* lethality potential. For CN, DM, and CS, toxicity figures indicate a lethal concentration of 100 to 300 mg/m^3 of air.*

For DM, which has been reported in use over the South Vietnam town of Hue in February, 1968, it is stated that this compound . . . "is not approved for use in . . . any operation where deaths are not acceptable" (e.g., riot control). However the field manual of the U.S. Army reports that it may be used combined with CN in munitions and in "military or paramilitary operations, in counter-insurgency operations, or in limited or general war . . . where *possible deaths are acceptable*" [my italics]. One has to remember that the gases used in World War I, in 1915, and now prohibited by international law were not, by far, 100 per cent lethal gases; mortality was only about 10 per cent of those affected.[2]

THE FACTUAL REPORTS. The first information appeared in March, 1965 in the *New York Times*[3] when George Reedy of the Presidential Press Office stated, "The gases are rather standard types of riot-control agent".

On the same day Secretary of State Robert McNamara stated that these gases "could be obtained through commercial channels." He

* Mr Robinson has also pointed out that toxicity data on CS is also to be found in the open literature[12, 13]. The use of DM as a harassing agent by British forces has been ruled out on the legal grounds that its use contravenes the General Protocol, 1925, according to Porton Technical Paper No. 651, declassified in June 1968. *Ed.*

displayed an illustrated catalogue issued by one manufacturer, and it was stated that "Mr McNamara is understood to have tried the effectiveness of CS gas personally to compare it with types used in World War II."[3] He added that: "Rather than use firepower, thereby jeopardising the lives of non-combatants, to drive the Vietcong out of the area the South Vietnamese troops dispensed riot-control agent". Dean Rusk, quoted by the *New York Times*, stated that, "It wasn't very effective. When the wind blew it away, it was dissipated, it did not achieve the purpose", and he concluded, "the anticipation is, of course, that these weapons be used only in those situations involving riot control or situations analogous to riot control".

As it can be seen from these declarations, the use of these gases in shelters or caves was not under consideration, officially at least, in March 1965.

The following month, *Time* magazine stated: "Compared with napalm bombs that incinerate whole villages, or a white phosphorus shell that burns a man to the bone, the temporarily disabling gases used in Vietnam seem more humane than horrible".[4] There followed many reports from Trung Lap[5, 6] describing how the gases were injected into tunnels and shelters, including those where civilians were hiding.

Then came the story about Robert Bowtell, given as follows in the *New York Times*: "Non toxic (sic) gas and smoke being used against Vietcong guerillas, in tunnels north-west of Saigon, have killed one Australian soldier and sent six others to hospital, officials said today. Cpl. Robert Bowtell, 21, of Sydney, died of asphyxiation although he was wearing a gas mask".[7] This information was confirmed from different sources.* It was the first official admission of the possible lethality of the so-called tear gases.

As a matter of fact, dispatches from Vietnamese sources alleged that in Han Nghia (Long Hu province) troops belonging to the 173rd U.S. Brigade together with Australian and New Zealand troops had used gases extensively against the civilian population hidden in shelters. From January 8th to 15th, 1966, more than one hundred people were killed by the gases in the shelters.

It can be seen that the two reports fit completely, and provide

* Since the conference my attention has been drawn to a rather more detailed account in the *Courier Mail* (Australia), January 13th, 1966. The report suggests that a build-up of carbon monoxide in the tunnel may have been a contributory factor in Bowtell's death. The involvement of CS, though, is not disputed. *Ed.*

TABLE 8

N.5

FRONT NATIONAL DE LIBERATION
DU SUD VIET NAM

Comité pour la dénonciation des crimes de guerre commis par les impérialistes US et leurs valets au Sud Viet Nam

EPANDAGE DE GAZ TOXIQUES

Date	Faits et localités	Dommages
28 janvier	- Plusieurs bombes à gaz toxique larguées sur le hameau de Phu Lac, village Hoa Hiệp, district de Tuy Hoà (province de Phu Yên)	- 100 habitants morts plusieurs autres intoxiqués
27 février 1965	- Plusieurs ballons à gaz toxiques largués sur les villages de Binh Hoa, Thanh Phuong (province de Kiên Tuong)	- Plusieurs enfants ayant ramassé ces ballons ont été gravement intoxiqués
Février 1965	- Epandage de gaz toxiques sur la localité de Bồng Son (province de Binh Dinh)	- Plusieurs habitants intoxiqués
9 mars 1965	- Epandage de gaz toxiques sur les habitants de la localité de Tân Uyên (province de Biên Hoà)	- Plusieurs habitants morts
Fin Mars 1965	- Epandage de gaz toxiques sur la localité de Bồi Loi (province de Tây Ninh)	- Des centaines d'habitants intoxiqués dont plusieurs femmes et enfants morts dans la suite.
3 avril 1965	- 4 bombes à gaz toxique larguées sur le village de Phuoc Thu, district de Tiên Phuoc (province de Quang Nam)	- Presque tous les habitants du village gravement intoxiqués.
9 avril 1965	- Des bombes à gaz toxique larguées sur le village de Phuoc Son (district de Thang Binh) province de Quang Nam.	- 72 habitants intoxiqués.
13 mai 1965	- Dans une opération de ratissage, épandage de gaz toxique à Vinh Châu (province de Soc Trang)	- Des centaines d'habitants intoxiqués dont 30 morts dans la suite.

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Date	Faits et localité	Dommages
26 Août 1965	- Insufflation de gaz toxique par 4 pulvérisateurs sur la population groupée au canal de Rach relevant du village de An Ninh (province de Long An)	- 146 habitants intoxiqués, 30 morts
5 septembre 1965	- Dans une opération de ratissage au hameau de Vinh Quang, village de Phuoc Son, district de Tuy Phuoc (province de Binh Dinh) insufflation de gaz CN par 67 pulvérisateurs dans les abris de la population.	- Plus de 100 habitants intoxiqués dont 26 enfants de moins de 10 ans, 26 femmes, 18 vieillards gravement atteints, 35 morts, 7 ont les yeux crevés.
8 septembre 1965	- Ratissage, lancement de plusieurs grenades à gaz toxique dans la localité de Ba Lang An (province de Quang Ngai)	- 78 habitants morts dont presque tous sont des vieillards, des femmes et des enfants.
6 octobre 1965	- Insufflation de gaz toxique sur la population de la localité de Bến Cat sud (province de Binh Duong)	- Plusieurs habitants intoxiqués, 3 morts.
22, 23 Novembre 1965	- Bombes et grenades à gaz toxique larguées sur la localité de Thach Thu, district de Mo Duc (province de Quang Ngai)	- Plusieurs habitants intoxiqués et atteints des maladies graves.
2 janvier 1966	- Ratissage à Bầu Trai (province de Long An) insufflation par mighty mite de gaz toxique parmi la population	- Des centaines d'habitants intoxiqués, plusieurs morts dans la suite.
2 janvier 1966	- Ratissage à Duc Hoa, Duc Hué (province de Cho Lon): lancement de grenades à gaz toxique dans des abris de la population.	- Des centaines de femmes, d'enfants et de vieillards intoxiqués faisant plusieurs morts.

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Date	Faits et localité	Dommage
De 8 à 15 janvier 1966	- Les GI's de la 173ème brigade et les mercenaires de l'Australie, de la Nelle Zélande en opération de ratissage à Hậu Nghĩa (province de Long An) ont insufflé dans les abris de la population des gaz toxiques à forte dose.	- Plus d'une centaine d'habitants morts dont la plupart des femmes et d'enfants. A cette même opération le sergent américain Robert Bowtell mourut lui-même d'intoxication. 6 soldats d'Australie intoxiqués ont dû être transportés à l'hôpital.
9 - 14 janvier 1966	- Ratissage à Cu Chi (province de Gia Dinh) insufflation de gaz toxique dans les abris de la population	- Plus d'une centaine d'habitants morts, des centaines d'autres intoxiqués.
10 janvier 1966	- Ratissage au hameau de Kim Tai district de An Nhon (province de Binh Dinh). Les GI's et mercenaires Sud Coréens ont lancé des grenades à gaz toxique dans les abris où ils ont parqué des femmes, vieillards et enfants.	- 40 femmes, enfants et vieillards morts
27, 30 janvier 1966	- Des ballons de gaz toxique larguées sur les villages de Khanh Hung, Trân Hoi, Khanh Binh (province de Bac Liêu)	- Plus de 1.000 habitants intoxiqués dont plusieurs enfants, 6 parmi ont perdu la faculté de parler.
1 février 1966	- Insufflation de gaz toxique dans des abris de la population du village de Ky Anh, district de Tam Ky (province de Quang Nam)	- 14 enfants morts, plusieurs personnes intoxiquées.
14 février 1966	- Au cours d'une opération de ratissage à Bồng Son (Binh Dinh) où étaient engagés 22.000 GI's et mercenaires, des hélicoptères de la division de cavalerie mobile US N°1 ont lancé 600 grenades à gaz toxique sur les différents hameaux du district.	- Des centaines d'habitants intoxiqués.

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Date	Faits et localité	Dommage
26, 28 février 1966	- GI's et mercenaires Sud coréens opérant dans 11 hameaux du district de Binh Khê (province de Binh Dinh) ont lancé des grenades à gaz toxique sur la population, ont chargé les gens à la baïonnette ou les ont ligotés dans des maisons qu'ils incendièrent par la suite	- 1.188 habitants morts dont la presque totalité est constituée de femmes, de vieillards et d'enfants (137 femmes, 60 vieillards, 70 enfants)
21 février 1966	- A Bồng Son nord (province de Binh Dinh) lancement de centaines de grenades à gaz toxique et épandage d'[illegible]	- Plusieurs habitants intoxiqués, nombre de morts non encore déchiffré.
Mars 1966	- A Bồng Son (province de Binh Dinh) lancement sur la population civile de 2.000 grenades à gaz toxiques.	- Des centaines de personnes intoxiquées
13 avril 1966	- A Dầu Tiếng (province Binh Duong) lance vat de 10 grenades à gaz toxiques en répression d'un meeting tenu par 300 ouvriers de la plantation de caoutchouc de Dầu Tiếng (province Binh Duong)	
30 Avril 1966	- Au village de Hoa Hiệp district de Chau Thanh (province de Tây Ninh) lancement de nombreux fûts de poudre toxiques	- Nombreux habitants intoxiqués, nombreux animaux domestiques [illegible]
Première demaine de mai 1966	- Lancement de 1½ tonnes de poudres toxiques dans le Nord-Ouest de Tây Ninh au cours de l'opération "Bingingham"	

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Date	Faits et localité	Dommage
Juin	- Epandage de poudres toxiques sur les villages de Ham Cuong, Ham Liên, Hàm Phu, Hàm Ninh, Hàm My (district de Hàm Thuận) Tân Hiệp (district de Hàm Tân) province de Binh Thuận	- Des centaines d'habitants intoxiqués
5 juillet 1966	- Epandage de toxiques en poudre sur la localité de Da Bao (Tây Ninh)	- Nombreux habitants intoxiqués, unhort.

Sud Viet Nam, le 1er octobre 1967

Le Secrétaire général
Signé : Dang Ngoc Ky

Pour traduction conforme :
P. Le Chef de la délégation permanente du FNL du Sud Viet Nam Nord Viet nam et P.O
Le Chef du Secrétariat,

TRAN KY HAI

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official evidence of the lethality of the gases used in the tunnels and shelters. From Vietnamese sources, more data was obtained which indicated an extensive use of gases, and numerous deaths. The NLF in South Vietnam prepared a detailed report, parts of which are reproduced on pp. 94–95. In fact, the NLF claim that there have been a minimum of thirty well-authenticated cases of the lethal use of gas prior to December, 1967. Deaths must run into several hundreds and those less affected into many thousands. These are inevitably minimum figures. One may recall that the mortality rate from gas in World War I was only about one in ten, yet the post-war records of those in France and Britain who did not die from gas at the time made it clear that the after-effects may persist, and many deaths attributable to the gas may occur years after the gas attack itself. For instance, any kind of bronchial irritation may increase the rate of lung cancer incidence in the affected population.

During our inquiry in Vietnam in Tay Ninh Province, peasants told us that they found big barrels that the local officers had identified as probable gases. We went to see these barrels and found a large, heavy container, easily identified, since the contents were clearly labelled. Each barrel was said to contain eighty lb. of CS. It is noteworthy that the label put on these barrels read "RIOT CONTROL". I remember our thoughts when looking at these words; they were obviously misleading, since it is difficult to imagine how the average policeman could handle an 80-lb barrel in an attempt to control any kind of riot. It is possible that this barrel was intended to feed a 'Mighty Mite'—which is not intended for use against riots. We made a small hole in the barrel and took away a sample of grey powder. Very soon we became severely ill with tearing coughs, agonizing abdominal pains, vomiting, and headaches. This happened despite the fact that we were in the open air, had protected our faces with a kind of mask made of plastic, and were well aware of the toxicity of the compound. We were sick for several hours.

The samples we took back to France were formally identified by Professor Lederer's laboratory in Paris and were used for the experiments described in the following section.

Experiments done with the gases used by the United States in Vietnam[8]

As has been said, it appears that the gases used by the United States in Vietnam were not simple harassing agents, owing to the way in which they were used in confined and closed areas where

the concentration becomes high. The potential lethality of these gases is, as we have shown, recognized by the United States (reluctantly and indirectly) and by the Vietnamese. To further elucidate this point, experiments were carried out on animals.

The first set of experiments were carried out at Hanoi, by the members of the Vietnamese Commission on the U.S. War Crimes. Dr Vigier of France attended the experiments.[9] All these experiments were filmed and the film was shown in Copenhagen for the Tribunal.

The experiments were as follows: DM was tested on a monkey at a concentration of 15,000 mg/m^3. The monkey showed severe respiratory seizures within 20 minutes and died within 45 minutes. Pathological findings included severe liver damage. CS was tested first on a cat, at 15,000 mg/m^3. The animal died within 30 minutes. Pathological findings included severe lesions, mainly of liver, brain, and kidneys. The same compound was tested on a monkey at a concentration of only 5000 mg/m^3. Death occurred in 25 minutes, with severe lesions similar to those seen in cats.

The second set of experiments were carried out in France by Professor Roussel in his laboratory devoted to industrial and environmental toxicology. He made a precise study of the toxicity of CS on mice, and found that at a concentration of CS powder of 200 to 2,500 mg/m^3 of air, two-thirds of the exposed mice died in 10 hours. The pathological findings included severe lesions in kidney, liver, and respiratory tract.

Conclusions

It is not possible at the time of writing to assess whether other gases or toxic agents have been used by the United States in Vietnam. Many reports have referred to the possibility of use of compounds such as LSD as a weapon in Vietnam, and it must be emphasized that if any such experiment has been carried out by the United States in Vietnam it would be difficult to collect sufficiently precise data to present reliable evidence here.

Moreover, American statements on the use of these weapons have been consistent only in their evasiveness. Thus even when they were trying to make the world believe that they were using only "tear gas" in Vietnam, it was revealed that DM was prohibited even in the open air for riot control, because it was known that fatal casualties could result from its use. In the *New England Journal of Medicine,* Sidel and Goldwyn have stated:

> Even if a "humane weapon" is developed, its humanity will require the delivery, as in the laboratory, of a precisely measured dose to a standard victim. Both these requisites have thus far been impossible to obtain in the field. Chemical and biological weapons are notoriously uneven in their dispersal and therefore in the amount absorbed by each recipient; to ensure that every person receives an incapacitating dose, some will have to receive an overdose. Furthermore, the young, the elderly and the infirm will be the particularly susceptible victims.[10]

There is little to change in this statement. Only a word. Instead of "will be", we have to put "are".

ADDENDUM

The following information has since come to my notice. M.F.K.

Recent scientific reports have shown that defoliants like 2, 4-D do not need to be ingested in order to be toxic; they are able to penetrate the skin so that mere contact is sufficient. (LACOMBE, A., *Revue Française de pédiatrie*, 1967, III, 207. *See also* NEILSEN, K., Fatal poisoning in man by 2, 4-D. *Acta Pharmacol.*, 1965. 22. 224–234.)

A first-hand testimony of the lethality of the so-called riot-control gases was made available to me by Dr Alje Vennema. While serving in Quang Ngai Provincial Hospital he saw many cases of fatalities following exposure to the gases. The principal cause of death was respiratory. See *New York Review of Books*, May 9th 1968.

8. The Yemen

M. Meselson

The History

Since 1963 there have been sporadic reports of the use of poison gas in the Yemeni civil war. It has been alleged that the gas was being used by the Egyptians on behalf of their allies, the Yemeni Republicans. The charges have come from the Yemeni Royalists, their supporters the Saudi Arabians, from various journalists, and, most authoritatively, from the International Red Cross.

So far as the outside world is concerned suggestions of the use of gas in the Yemen began on July 8th, 1963, with an article in the London *Daily Telegraph* by Richard Beeston stating that there had been a gas attack on the village of Al Kanma. Following this story, U Thant initiated an inquiry "to determine whether poison gas has been used in the fighting in the Yemen." A United Nations observation team in the Yemen produced a preliminary report stating they had no evidence that gas had been used but that inquiries were continuing. No definite conclusion was ever announced by the U.N.

I find no further reference in the press to the use of gas in the Yemen until November, 1966, when Wilfred Thesiger, a British archeologist and explorer who had spent some time with the Royalists, stated that he had come into a village shortly after a gas attack. Thesiger reported seeing twenty victims of what he termed "blinding gas" and said that he treated a boy suffering from "blister gas" that might have been a form of mustard.

The first alleged use of nerve gas took place on January 5th, 1967, on the village of Kitaf, the military headquarters of the Yemeni Royalists. This would be the first use in any war of nerve gas, a class of compounds far more deadly than any other known war gases. Judith Listowel, writing in the *Statist*, reports an interview with the survivors and states that 155 persons were killed and 200 more severely poisoned. There happened to be a Red Cross team in the vicinity of Kitaf at the time, and they are alleged to have

sent a cable to their superiors reporting the gas attack and requesting gas-masks. The Red Cross did not officially acknowledge the telegram, but the Red Cross Director in Geneva stated that all parties had been appealed to not to use illegal weapons. On January 24th, Jordan threatened to withdraw recognition of the Yemeni Republican government unless the use of gas was stopped.

On January 31st, the United Arab Republic Minister of National Guidance, Mohammed Fayek, denied that the U.A.R. had ever used gas and furthermore said that Egypt would welcome a United Nations fact-finding commission to investigate the charges.

Over the next three months, the Saudi Arabian ambassador to the U.N. urged U Thant to speak out against the use of gas in the Yemen apparently not wishing to place the matter before the U.N. himself. The Saudi Arabians did however produce several pages of medical and chemical evidence purporting to show that nerve gas had been used at Kitaf. The evidence includes measurements of the phosphate content of the blood of human and animal casualties, supposedly an indication of the use of nerve gas. However, some of the figures seemed far too high to be reasonable.

On May 10th, 1967, according to a report published in the *New York Times* and attributed to the International Red Cross, there was an attack using gas on two Yemeni villages, Gadafa and Gahar. On the following day the International Red Cross team in Jidda, Saudi Arabia, received an urgent request for help. They arrived in Gahar on May 15th after being attacked on the road by military aircraft. The team conducted an autopsy and examined several survivors. Their report claims that 75 people were killed by the attack and 273 more by attacks on other villages later in May. The autopsy results were sent to the Faculty of Forensic Medicine at the University of Berne for evaluation, where it was concluded that the victims described had indeed been killed by poison gas, the most likely agent being mustard. Although these details have never been officially acknowledged by the Red Cross, on June 2nd, 1967, it issued a statement saying:

> The International Committee of the Red Cross has again received, from delegates in the Yemen, reports of bombing by toxic gas. A medical team led by the head of the R.C. Mission in the Yemen went on May 15 and 16 to a village in the northern part of the country to attempt to give aid to the victims of bombing which had taken place some days previously and as a result of which, according to the survivors, many inhabitants had died of asphyxiation. Delayed by air raids to which the convoy was victim, I.C.R.C. doctors on arrival at the site immediately

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> gave treatment to some of the wounded and collected various indications pointing to the use of poison gas. Extremely disturbed and concerned by these methods of warfare, which are absolutely forbidden by codified international and customary law, the International Committee of the Red Cross at once communicated its delegates' reports to all authorities concerned in the Yemen conflict requesting them to undertake a solemn engagement not to resort in any circumstances whatever to the use of asphyxiating gases or any other similar toxic substances.

The salient points in this record are that poison gas has been used in violation of the Geneva Protocol, that it has been used against civilians, and that nerve gas may have been employed in some of the attacks. The evidence indicates that Egypt is responsible, but Egyptian officials have denied the charge and have asked for an international investigation.

There have been no reports of gas warfare in the Yemen since the Arab-Israeli war of last year. However, it was not until about that time, with the first official Red Cross confirmation that gas had been used, that world reaction began to assume serious proportions. This, together with a variety of political reasons, may explain the failure of the world community to take more decisive steps to enforce the 1925 Geneva prohibition against gas warfare.

The Yemeni events and the United Kingdom

D. E. Viney

The first official British comment on the accumulating evidence of the use of toxic gas in the Yemen came on January 31st, 1967, when the Prime Minister agreed in the House that "such evidence as I have suggests pretty strongly that poison gas may have been used". It was immediately after this that Egyptian papers invited the U.N. to investigate, and the U.A.R. National Assembly was told by its Arab Affairs Committee (February 6th) that the allegations were a defamatory invention put out by Jordan and Saudi Arabia.

After unauthorized versions of the International Red Cross team's report had reached the press, and interest in the subject had been enhanced by Israeli apprehensions during the Middle East conflict in June, the Government was again pressed for comment. Minister of State, George Thompson, stated on July 3rd: "We have received well substantiated reports that gas was used several times. Both a mustard type and a choking type have been used on different occasions."

When the possibility of a British initiative was raised Foreign Secretary, George Brown, reiterated that "we condemn the use of gas in the Yemen," but argued that it would be most appropriate

for some Arab country, such as Saudi Arabia, to take action in the first place. That was on July 6th. Later in the month over two hundred Members of Parliament nevertheless signed a letter requesting that the Government should do something, and Mr Brown told an all-party delegation of three that the United Kingdom would consult with other powers as to "the best means of putting a stop to this clear breach of generally accepted rules of conduct".

Immediately after this Parliament went into recess. When it met again the Egyptians were pulling out of the Yemen; the British Government made it clear that none of the other powers consulted was interested in initiating further action and that it considered in any case that such an initiative might now exacerbate the situation in the Yemen rather than assist a settlement. It was of course widely assumed that a desire to avoid further unpopularity with the Arab world in the wake of the Middle East war played a major role in setting this attitude, and the West would no doubt have found itself isolated if it had impugned Egypt. The Soviet Union, Czechoslovakia, and other countries who had taken up a strongly pro-Arab line could not have been expected to lend any diplomatic aid for such a purpose, even though they were better placed to exert persuasion on Arab countries than the British were. If they made private representations in Cairo on the undesirability of flouting the Geneva Protocol—and it cannot be excluded that they did so—they evidently had little success.

Since the expert evidence of the use of lethal gases in the Yemen was not made public, no-one without special access to the facts can point the finger dogmatically at Egypt. However, the evidence was apparently found persuasive by British Ministers who had every political reason not to inflate the issue. The reasons why no international action was in the end taken are understandable enough. But the net result is that the Geneva Protocol, that lone achievement in the history of attempts to proscribe particular classes of weapons, is seen to have been flouted with impunity in the very kind of military confrontation where, because of the extreme inequality of strength and the impossibility of deterrence or reprisal in kind, respect for law is almost the only motive for restraint; and indeed to have been flouted (it seems) by a state which signed the original Protocol in 1925, reaffirmed its adhesion on becoming a Republic in 1953, and joined in the general acclamation of the ban on lethal chemical weapons during the United Nations debate in 1966.

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PART THREE

RESEARCH POLICY

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9. United Kingdom

Robin Clarke and
J. Perry Robinson

The organizers of this conference felt it important that representatives from the Ministry of Defence or its chemical and biological research centres at Porton Down, Wiltshire, should be asked to attend the conference and give papers on official United Kingdom policy. In consequence, representatives of both the Chemical Defence Experimental Establishment and the Microbiological Defence Establishment at Porton, as well as the Director of Chemical and Biological Defence, were invited to deliver papers on research policy and on matters of defence.

Unhappily, all three were unable to get permission to do so, although there have been television appearances both before and since.[1] What follows, therefore, is an attempt by two outsiders interested in the problem to summarize the results of some fairly extensive detective work and present what little is known about the British Chemical and Biological Warfare effort and the policies that lie behind it. Later, we shall indulge in a little speculation, but first we shall list what little is already public knowledge.

The research establishments

CHEMICAL DEFENCE EXPERIMENTAL ESTABLISHMENT. Much the oldest is the Chemical Defence Experimental Establishment (CDEE), as it is now called, which began on its present site in Wiltshire in 1916[2]—shortly after the first chemical weapons were used in World War I. As its name implies, it was from the first designed to be a defensive organization for all three military services. It now comes within the compass of the Ministry of Defence. Its budget is not publicly known and nor is the size of its staff. According to a press release[2] which was prepared in 1964 on the occasion of a press visit to Porton, much of its work is devoted to the solving of peace-time problems in civilian life; for example, it gives advice on the design of respirators for protection against industrial dusts and poisonous

vapours and gives assistance in the treatment of poisoning from accidents in the use of agricultural chemicals.

However, undoubtedly one of the main contributions of the CDEE has been the improvements made by it in the respirator. From the respirators used in the last war, the CDEE developed a number of new models, of which the Light Respirator Mark 7 was in service in the early 1960's and which has now been replaced by the S6. A third type was developed at the CDEE for civilian use, and others have been developed for special service requirements, such as those of air crew. But the respirator is likely to be insufficient protection in time of chemical attack. The CDEE has also produced air filtration units for use where respirators may not be practicable, and these units are already installed in certain buildings and ships. One of these units, built entirely from non-magnetic material, has been designed for use on mine-sweepers.

The CB suit, which is said to be impervious to radioactive dust and to all chemical and biological weapons, was also developed at the CDEE. It is a remarkable advance on other protective CB suits in that it is very light and porous to allow passage of both air and moisture. Various other types of protective overclothing have also been developed there, sometimes as a result of close collaboration with the American, Canadian, and Australian establishments.[3] Alarm equipment to go with these protective devices has also been produced. It includes alarms said to give virtually instantaneous warning of the presence of nerve gases in either liquid or vapour form, another device for indicating when it is safe to remove the respirator, as well as detective pads which will indicate the presence of liquid chemicals by changing colour.

The CDEE has also been at the forefront in the therapy of nerve-gas poisoning.[4] Like the Swedish and the American establishments, it has developed devices[5] for the automatic injection of a standard dose of atropine into the bloodstream through the thigh. The injector is claimed to be superior to either of the other two models available in that the needle is pressure-sensitive. One of the first drawbacks of such injectors was the danger of the needle striking the bone with consequent medical complications. The British injector is designed so that pressure on the needle of the kind likely to be caused by contact with the bone causes the needle to halt its advance. A pill to be taken at the same time as the injection has also been developed. It contains the oxime known as P2S and has also found uses in treating cases of agricultural poisoning from phosphorus-containing insecticides.[2]

This side of the CDEE's activities, at least, is publicly known and is, indeed, described extensively in its scientific literature. Development work of this kind is also backed up by a pure research programme into the classical subjects of chemical and biological research centres—the physics of aerosols and sprays, micrometeorology, measurement of the size of particles and their concentration in the atmosphere, etc. Perhaps the only other publicly known fact—and certainly the only publicly known fact of offensive relevance—is that the riot-control agent CS was developed in the 1950's at the CDEE.[6] [The information that has become available about CS since the conference is quite extensive and is summarized in an addendum to this paper. *Ed.*]

MICROBIOLOGICAL RESEARCH ESTABLISHMENT. The other main unit at Porton, the Microbiological Research Establishment (MRE), is much more recent in date and probably better known if not to the public then at least to civilian scientists. In 1945, some forty-five people[7] were employed there, of whom fifteen were scientists, but the laboratory facilities were at that time far from ideal. The laboratories were therefore expanded, a job which was completed in June 1951,[8] and which was apparently carried out so that microbiologists, which the defence effort needed, could be attracted. This move was clearly successful, and the scientific work of the establishment gained rapid recognition. Clearly the MRE also grew in size, and though its exact complement of scientific staff is not publicly known, we know that in 1964, for instance, forty-one people authored openly published scientific papers from the establishment.[9] [In 1966, forty scientific papers were published by staff of the MRE, according to a Parliamentary Answer.[54]] The MRE has a reputation for scientific excellence, and this could not have been gained behind closed doors. On the contrary, access to the MRE is relatively easy, and it opens its doors—or at least some of them—to around 2000 visitors every year.[1] Its policy is equally liberal towards the publication of research results. According to the Director—and a glance at the microbiological literature tends to confirm what he says—about 80 per cent of the work that goes on at the MRE is published in the open literature. Perhaps, in case there is any confusion about it, something should be said about the scientific nature of this unclassified work.

Perhaps the best known concerns the continuous cultivation of bacteria,[8] a technique if not actually pioneered at Porton then certainly followed up very quickly by workers there. The net result

of this—and similar work, of course, in Czechoslovakia and the United States—was the ability to grow bacteria indefinitely at a constant rate under standard conditions and with constant composition. This technique has proved particularly valuable in industry for the preparation of vaccine material and for growing the fungi used in the production of antibiotics.

Another field of important work has concerned the pathogenicity of disease-causing organisms. Scientists at the MRE, for instance, identified a specific toxin liberated by the anthrax bacillus and later proved their ability to produce it *in vitro*. They also resolved it into three components.[10] Similarly, in work first reported in 1961, they found that the bacterium *Brucella abortus* which causes brucellosis, or Malta fever, has a predeliction for foetal tissue.[11] The MRE workers found that this was because such tissue contained erythritol which stimulated the bacterial growth.

A substantial number of papers have also been published by workers at the MRE on the subject of the survival of bacterial species, particularly in aerosol form. In 1966 a single issue of the *Journal of General Microbiology* carried four articles by MRE scientists.[12, 12a] One dealt with the nutritional requirements of the plague bacillus and related species, two were on the survival of bacterial aerosols and the factors affecting it, and the last was on the survival of bacteria in suspension. Though the very fact that these papers were published at all virtually proves they are not representative of the growth, storage, and dispersal of British biological plague bombs, they do show that there is more than one way of interpreting the nature of the research effort at Porton. Officially, of course, all work there is either concerned with defence or is so basic that it has no military implications at all. Equally, and several workers at the MRE have admitted this, there is no clear dividing-line between offence and defence in these matters. Even to study defence requires that scientists learn how to produce samples of various weapons so that different defensive measures can be evaluated. The production of these samples may provide information useful to a nation which wishes to produce sample weapons on a larger scale—and perhaps one should recall that Porton has very few secrets, if any, from similar establishments in Australia, Canada, and the United States.

Is there, then, any concrete evidence that any of the work carried out at Porton, other than that done during World War II, has been done for offensive purposes? Early in World War II Defence officials sprayed anthrax on the island of Gruinard off the north-west coast

of Scotland.[13, 14, 15] This was clearly a field trial of a biological weapon and one of the few that has ever been openly recorded. But the aim of the exercise, according to the present Director of the MRE,[1] was to establish whether or not biological warfare was at that time feasible. Dr Gordon Smith, Director of MRE, has said that it was then that several officials really began to believe that biological warfare was practically possible.[1] The experiment, incidentally, also demonstrated one of the great drawbacks of biological warfare in that the island that was sprayed in the 1940's is still contaminated and, according to Dr Gordon Smith, is likely to remain so for another hundred years. There is no evidence that this experiment, however, was the first step in the production of biological agents for use in war. We know of no other fields trials until the 1950's, when inanimate particles were sprayed from aircraft in the Bahamas area in 1954.[16, 17] However, for chemical weapons, which do not necessarily require field testing over large areas, we are aware of several field trials. Thus Porton Technical Paper No 239 of 1951 is entitled "The Lethality to Rats of GB and GE from HE/Chem. Weapons in the Field",[18] while No 424 of 1954 is entitled "The Production of Casualties in Monkeys with GB Vapour".[19] These two papers are not openly published. But again it could be easily argued that the trials were to test various defensive measures.

Detailed discussion of the exact balance of offence and defence in exercises of this kind is somewhat futile until such time as the officials concerned can be persuaded to reveal more about them. The overwhelming evidence at the present time is that both units are concerned primarily with defence and that a relatively small proportion of their work, possibly inevitably, may also have offensive implications. Neither is there any evidence of the manufacture of any weapons at Porton. We know, of course, that the equipment for the manufacture of the raw material for biological warheads is available at the MRE at Porton. Indeed, during the Asian 'flu epidemic of 1957, the MRE produced more than 600,000 doses of 'flu vaccine.[8] This means they could equally well produce 600,000 doses of a biological warfare agent. In that sense, the existence of the MRE must be reassuring to officials concerned with BW policy. For although Britain has both signed and ratified the Geneva Protocol[20] forbidding the use of chemical and biological weapons in war, the Protocol does not preclude retaliation in kind. While Britain has never shown any indication that she intends to break the Geneva Protocol, officials must have considered the possibility

that a potential enemy might do so first. If they did, and it was decided to retaliate in kind, the MRE at Porton would probably provide the necessary biological material.

Chemical Weapons

If this seems obvious the situation with regard to chemical weapons is much less so. We do know that Britain possessed chemical weapons, but never used them, throughout World War II. After the War they were mostly disposed of, about 100,000 tons in all, some off the west coast of Ireland[21] and some in the Bay of Biscay.[22] The last load was sunk in September 1948,[21] under the direction of the Ministry of Transport. Captured German stocks were for the most part also sunk, tens of thousands of tabun shells, for instance, were sunk in the Baltic.[23] Captured German gas bombs were still being sunk in 1957, by then in the Atlantic.[24]

Have any chemical weapons been manufactured since then? Except for various riot-control agents, there is no evidence of this. There was a pilot plant built for the manufacture of DFP, Britain's rather weaker version of the German nerve gases, at Sutton Oak, in Lancashire,[25] which began operating in 1945, but has since been resited at Nancekuke, Cornwall. In fact, we know of next to no evidence of the manufacture of chemical weapons nor of any arrangements with industry for such manufacture if it should be required.* Such arrangements existed before the last War and indeed were widely proclaimed. However, in view of the similarity of the nerve gases to some of the modern insecticides, there is no doubt that the chemical industry could go over to nerve gas manufacture without much difficulty.

Optimistically, one is tempted to conclude that Britain has no operational stocks of chemical weapons, but the psychology of the situation is obviously so delicate that no official is ever going to confirm or deny such a suggestion, whichever way the truth lies.

Britain's Defence Potential

On the other hand, there is absolutely no doubt of Britain's determination to defend herself against chemical and biological attack. We are not only carrying out an expensive research and development programme into the best ways of achieving this, but we have, for instance, some several million post-World War II gas-masks already stocked for civilian use.[26] Furthermore, chemical

* But *see* p. 114. *Ed.*

defence equipment is now standard for troops in specific areas, particularly the British Army of the Rhine. Every soldier in areas where chemical warfare is thought likely is now issued with the S6 respirator and with three auto-injectors loaded with atropine for the treatment of nerve gas poisoning. In addition, British soldiers involved in chemical warfare would have chemically sensitive pads sewn on their battledresses, which would respond by colour changes if liquid chemical became splashed on to them. Soldiers are also issued with specially impregnated pads with which to wipe off any chemical that comes into contact with skin. The chemical and biological suit that was developed at Porton is now being made available to soldiers in special areas, and a residual vapour detector —a device to tell when the atmosphere is free from contamination— is being made available to section leaders, *i.e.* the leaders of every ten men.

Much of this equipment will also double as protection against biological weapons, but a pack of broad spectrum antibiotics would also be issued to soldiers for use as a 'blanket cure' should they become involved in a biological attack.[26] One can only hope that the scientists who recommended this particular policy have since looked closely at recent figures on infective drug resistance.

From information such as this, and from some knowledge of the research activities at Porton, it would be tempting to conclude that Britain is genuinely concerned only with defence against chemical and biological weapons.

Future policy

There is, however, one area in which there is still room for doubt. That area concerns the exploitation of discoveries made at Porton during the various research programmes. The normal way of exploiting inventions and discoveries is through the medium of patents, but when one examines those in which the British Defence authorities have had a hand, a number of dubious points emerge.

Most of the Ministry of Defence patents which have been published have an obvious usefulness in industry, sometimes of great significance. These patents are justifiably exploited to their full commercial worth. They include those for British Anti-Lewisite[28] for the treatment of metal poisoning, artificial breathing-machines,[29] cascade impactors[30, 31] for studying air pollution, the spinning-top atomizer[32] for producing accurately dimensioned particulate sprays, an improved process for making dialkyl phosphorohalidates,[33] (essential raw materials in the production of many insecticides), and

plague vaccines.[34] Most of these have been exploited through the conventional channel for doing so, the National Research Development Corporation (NRDC). Indeed, one of the purposes of the NRDC is just that, to exploit commercially discoveries made in Government laboratories.

The decision as to whether the Ministry or the NRDC should handle a Government patent application rests with the Ministry, although the NRDC's advice is being increasingly sought. In general, the Ministry will retain those patent applications in a field in which it can operate better than the NRDC, for example a field in which only its contractees are likely to need patent protection. Thus, the Ministry has retained the title in the patent[6] on the riot-control agent CS and in that on a novel process for making the nerve gas sarin.[35] Occasions do arise when a patent application is assigned to the NRDC who then finds that there is no normal commercial outlet for it, in which case it may well be assigned back to the Ministry, perhaps for future use.

But there are occasions when this process of assignment and re-assignment looks odd, particularly if it is so timed that the patent is published in the name of the NRDC. It might almost be said that this process was intended solely to conceal the Ministry's connection with the patent, particularly if the patent goes on to live its full life. There are two particularly pertinent instances of this. One such patent[36] discloses a method of forming carbon-phosphorus bonds by way of what is now called the 'Perrin-Kinnear complex' after its discoverers at Porton. This patent was subject to security measures during its early years, and was kept in force for its full term. It describes an extremely simple method of making nerve-gas intermediates. The second patent[37] discloses a continuous process for making methylphosphonothionate esters, compounds which are possible intermediates in V-agent manufacture.

In view of the continued payment of annual taxes on these patents, and of the convoluted history of their proprietorship, it is reasonable to assume that the Ministry thinks them important. It is not in the least improbable that the Ministry is licensing their inventions, profitably, to other nations or to industry. But there is little further evidence to support this, apart from the existence of overseas equivalents to some of the patents concerned.[38, 39]

The position over the exploitation of Porton discoveries under international exchange agreements is much clearer. In 1960 the Ministry of Defence confirmed[40, 41] a newspaper report[42] that information had been supplied to the United States under such an

arrangement, and that that information formed the basis of a process for making a nerve gas which was about to go into large-scale production, at the Newport nerve-gas plant. It was not disclosed which nerve gas it was, but since the Americans had then a very satisfactory process of their own for mauufacturing sarin or GB, the assumption is that it was their other now-standardized nerve gas, the V-agent VK. If this is so it may be that the information passed included that disclosed in the last patent referred to.[37]

Conclusions

There appears to be no evidence directly available that the United Kingdom is producing lethal offensive chemical weapons or even that she has stockpiles of them. The patent literature, however, leaves room for doubt about a number of connected issues. There is very little doubt that the United Kingdom has in the recent past supplied know-how to the United States of direct relevance to the manufacture of at least one nerve gas which is being stockpiled there. And, finally, the presence of a number of patents in the literature suggests that the United Kingdom is in a position to sell toxic weapons, or at least components of them, to other countries. We know this is done for some of the lachrymatory agents; it is also possible that it could be done for some of the lethal agents.

It is interesting to compare the British Government's security classification of chemical defence work with that of the Americans. Until recently the British attitude was almost invariably to keep it secret. Even the official history of the World War II weapons development programme makes practically no mention of chemical warfare equipment, beyond admitting that "it absorbed a great deal of energy and many notable developments occurred."[43] The Americans on the other hand follow a very erratic course. There are times when it has seemed that their classification system has broken down. On several occasions the Department of Commerce have published in one of their information journals abstracts of still classified reports.[44] On another occasion they published through unclassified channels an index[45] to an entire range of classified reports published over a period of a year.

During General Creasy's great public relations campaign[42, 46] to get both the public and Congress to accept the idea of toxic warfare, a great quantity of material was declassified, some of it describing still classified British work. As the campaign backfired, the declassification programme slowed down until the present time,

when the U.S. Department of Defense is possibly more cagey than our own Ministry. In several instances, declassified reports[47, 48] which had been made available in our scientific libraries were recalled to the United States, never to be seen again.

The same sort of thing happened immediately after World War II. Thus in 1946, reports of the interrogation of German chemical warfare workers were published,[49] only to be quickly recalled. Similar Japanese reports remained published however. On the British side, the German reports remain classified, but the Japanese ones were released a short time ago.[50]

The final point that should be made concerns the reason why the two units at Porton have remained steadfastly classified since World War II. The hope that they might become civilian establishments has been raised several times[51, 52, 53] since then, and indeed a number of scientists have left Porton simply because they were under the impression that the units would be declassified and they then realized that this was not going to happen. Why not? From what has been described of the work there very little if any would be out of place in any major civilian chemical or microbiological research centre. Why, then, does such a move not take place? Our guess is that the Ministry of Defence wishes to continue having its cake and eating it. Under the present arrangement it can maintain a front of having only defence interests with an immediate option of diverting its efforts, via Porton, into totally different channels. It is difficult to see what other reason there could be for not declassifying the establishments and thus allaying the largely unnecessary suspicion which still lingers round them.

ADDENDUM

Since the Conference, further information about the activities at Porton Down has become available, partly as a result of the publicity following the Conference itself, partly as a result of the publication of the evidence of the House of Commons Select Committee on Science and Technology, which has been examining the role of Britain's Defence Research Establishments. The Select Committee visited Porton on May 6th 1968 and heard evidence from Mr G. N. Gadsby and Dr C. E. Gordon Smith, Directors respectively of the CDEE and the MRE. Part of the information given in these hearings was published in "The Observer", May 26th 1968, and was subsequently judged to be a breach of Parliamentary Privilege. The full report of the Committee was, however, eventually published on July 8th 1968, although certain sections of the evidence were omitted, apparently

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on security grounds. According to "The Observer", July 28th 1968, the Ministry of Defence originally wanted to leave out rather more of the evidence, but changed its mind both in view of the prior "Observer" leak and the adverse publicity which Porton was receiving. What follows is a summary drawn from this evidence, the replies to a number of Parliamentary Questions and related news stories (see, e.g. "The Times", June 1st 1968).

—Editor.

Cost, facilities and policy

Of the two Establishments at Porton, the CDEE costs £1,600,000 annually and employs some seventy scientists and engineers (including medical doctors). The MRE costs £900,000 a year and houses forty-eight scientific officers and seventy-six experimental officers (an item, presumably relating to medical doctors, was censored from the Minutes here). Some of this cost, to both Establishments, is recovered in sales to other government Departments, and also commercially, by way of patents let through the National Research Development Corporation.

Porton is advised by a group of external scientists, drawn partly from the universities; the current list of twenty-two (1968) includes: Professors N. K. Adam, F.R.S., F. Bergel, F.R.S., E. Boyland, J. I. G. Cadogan, N. B. Chapman, R. C. Cookson, D. H. Everett, R. B. Fisher, D. J. E. Ingram, C. A. Keele, P. L. Krohn, F.R.S., G. J. Kynch, W. V. Mayneord, F.R.S., M. D. Milne, P. R. Owen, A. E. Ritchie, H. N. Rydon, J. B. Speakman, R. H. S. Thompson, S. Tolansky, F.R.S., M. Weatherall, A. Wilson, and Dr R. D. Keynes, F.R.S.

As well as these external advisers, Porton has attached to it a number of Service officers—thirteen altogether on the establishment of the CDEE, from Army, Navy, and Air Force, whose function is to specify Service requirements in detail; amongst these officers are medical doctors. There is also a member of the U.S. Standardisation Office, U.K., permanently located at Porton, a standing reminder of the existence of the Quadripartite Agreement between the U.S., U.K., Australia, and Canada on information-sharing. Another reminder was, incidentally, provided recently when a paper jointly authored by two workers at the MRE and one at Fort Detrick on gene transfer between two strains of *Pasteurella Pseudotuberculosis* (a relative of the plague bacillus) appeared in the *Journal of General Microbiology*. Other versions of this Quadripartite Agreement, both tripartite and bilateral, exist with other NATO countries.

Despite these links, the Directors of both CDEE and MRE have categorically stated that the work of their Establishments is entirely defensive, and that stockpiles and retaliatory capacity, should Britain be attacked, are both lacking. Porton's responsibility, according to the Director of CDEE, is confined to providing defensive techniques for the Forces; Dr Gordon Smith extends his definition of the defensive role of MRE to the civilian population as well. So far as secrecy is concerned, MRE publishes, according to its Director, some 80 to 90 per cent of the work done, but "it is an extremely difficult figure to calculate with any degree of accuracy." To a question as to whether the actual output from MRE, if what was published amounted to some 80 to 90 per cent was not somewhat meagre, the Director replied: "I think our publication record is quite good. I think it could be improved if some members of staff who are rather slow at writing papers would hurry up with them . . . it is our practice to try and offer a substantial piece of work before we publish it." No figures are available for the proportion of CDEE's work which is published, but it was generally agreed to be considerably less than that of MRE.

Nonetheless, secrecy at Porton has become a matter of considerable public concern. The Select Committee asked whether Porton held 'open days' and

was told that its directors had asked to hold them but that permission had not been granted. Even before the Minutes had been published, though, it was announced in Parliament that open days at Porton would be instituted. A later amplification by Mr John Morris, Minister of Defence for Equipment, made it clear that the first open day was to be for MRE only and that attendance would be by invitation to "representatives of universities, learned societies, research associations, local authorities, industry, the press" and "a number of organisations likely to be interested in the work of the establishment." There appears to be no intention of extending the open day to the work of CDEE as well.

Porton and the Universities

In addition to its external advisers, Porton has another type of link with the universities. This consists of a series of collaborative projects with individual members of university staffs and of specific research contracts. An example of the type of collaborative project is that which resulted in a joint publication by two workers at St Thomas's Hospital and the Director of MRE in *The British Medical Journal* (*January 29th* 1966) which contains a description of research in which a series of patients suffering from terminal cancers were experimentally infected with live Langat virus—which causes encephalitis, though is normally not infective in humans—and which for various reasons the authors hoped might serve to alleviate some of the effects of the cancers (which were mainly leukaemic); the research findings were negative, however; all the patients subsequently died.

So far as the research contracts go, the CDEE has twenty-seven, including those with Birbeck College, London, University College of Wales, Oxford, Oxford College of Technology, Birmingham, King's College, London, Welsh College of Advanced Technology, Institute of Neurology, Middlesex Hospital Medical School, University of Liverpool, Chelsea College of Advanced Technology, Queen's University, Belfast, Portsmouth College of Technology, University of Edinburgh, University of Southampton, University of Sheffield, St Mary's Hospital Medical School, University of Exeter, University of Kent, and University of St Andrews.

Exact details of many of these contracts are not available, but a summary is provided in the Table. Examples include a contract to Professor Henry Rydon, Exeter, (who is also on the Advisory Board and previously worked at Porton) to study the isolation, purification, and structure of the plant toxin ricin—rather analogous to botulinus and tetanus toxins, though much less powerful; to Dr Mary Whittaker, Kings College London, for work on acetylcholinesterase enzymes (those whose activity is blocked by the nerve gases); to Dr B. Jacques, Portsmouth College of Technology, on the synthesis of 4-hydroxyl, 1, 2, 3, 4 tetra-hydroisoquinolines (substances with related structures are known to be hallucinogens); and to Professor Harry Smith, Birmingham, on the fractionation of compounds of microbial origin important in the pathogenesis of infectious diseases.

None of these contracts appears to be for a very large amount of money, and most appear to be administered so as to allow a fair degree of freedom to the recipients; that is, they are not specifically classified. Indeed Professor Rydon has laid down as the criteria under which he would accept any external contracts coming into his department (at a Teach-In on CBW held at Exeter University on June 11th 1968) that the work must be of relevance to the interests of the Department and must have no restrictions on the right to publish. Such criteria are certainly wholly admirable; nonetheless it must be noted that what the Ministry of Defence means by unclassified is given in a Parliamentary answer by the then Under-Secretary of State for Defence on February 26th 1968:

> When arranging a research contract the Ministry of Defence agrees with the university who is to supervise the work, but the Ministry does

not normally have a voice in deciding who should carry it out. The results of research for defence are, in accordance with general scientific practice, published freely *provided there are no overriding considerations of national interest, including security*. Universities undertake to consult the Department before publication. Over the past two years, no university has declined to undertake a research contract offered to it. [Our italics].

One must also assume, that however the recipients of the Porton contracts read the significance of their work, those who awarded them the contracts in the first instance must have felt that the work was in some way related to Porton's prime mission; it certainly has no brief to function as a charitable foundation, awarding money for projects irrelevant to its interests.

Nancekuke and the CS controversy

Apart from Porton, other 'in-house' developmental work on chemical warfare is carried out at the Porton outstation of Nancekuke (Portreath) in Cornwall, where pilot-plant facilities for the manufacture of chemical agents exist. Two substances at least are known to be manufactured there, the nerve gas VX, currently made in test quantities only, and the harassing agent CS (see Chapter 1). CS is manufactured in quantities of around four to six tons a year. Some at least of this is transported to the factory of Schermuly Ltd. (a subsidiary of the Charterhouse group), near Dorking in Surrey; here the CS canisters are made and filled, CS is exported to about sixty foreign countries at a price of 35 shillings per pound. In addition, it is supplied to thirty-six of the eighty police forces in England and Wales, as well as to some in Scotland. Police instructions as to the use of the CS is that it is "only to deal with armed criminals or violently insane persons in buildings from which they cannot be dislodged without danger of loss of life, or as a means of self-defence in a desperate situation, and that in no circumstances should they be used to assist in the control of disturbances". (Mr Buchan, Under-Secretary of State for Scotland.) If the evidence of Dr Kahn (Chapter 7) is taken into account, though, it is clear that the use of CS in the type of confined space envisaged in this instruction to police might not be without its hazards. Taking the figure quoted by Porton for the L Ct_{50} of CS, one might expect that the explosion of one Porton CS grenade in the average-sized suburban living room would result in the build-up of a dose lethal to half the exposed population within twenty minutes—(assuming the CS powder remained dispersed in the air) doubtless less if those exposed were at all unhealthy, or included young children (see also the discussion of the toxicity of CS in *Science Journal*, August 1968).

It is certain, though, that the CS used in Vietnam is not of British manufacture. Nor is the gas used during May 1968 by the Paris police, which, code-named CB, the French version of CS, is apparently produced in that country. British-made CS is apparently not exported either to France or the USA. It may be noted in passing that the French CB grenade, according to an analysis made in May 1968 by Dr Kahn, holds a charge of no more than 1–2 grams of CS. The Porton-patented canister described in the preceding chapter contains 30-40 grams, whilst the Mighty Mite used in Vietnam may contain 80 pounds (that is, 35,500 grams). Nonetheless, even though in Paris the gas was used on the open streets, several casualties from the use of CN and CS have been reported from Paris, including allegations of partial or total blindness and at least one death. It is likely that most of these were CN casualties, but it must be emphasized that a proper medical evaluation of this data is not currently available in England. But additional evidence as to the potential hazards of these agents, and also the American combined agent, containing mainly CN, and known as Mace has come from a study made at the University of Michigan, and reported in the *New Scientist* (June 20th 1968), has made clear that the spread of their use should not go unquestioned.

Table 9 CBW Research Contracts let to British Universities*

Chemical Defence Experimental Establishment

Nature of Research	*Location*
Research on Fluorohydrocarbons	University of Birmingham
The isolation purification and structure determination of physiologically active peptides	University of Exeter
The Development of Ultra Microbiochemical Techniques to explore the mode of action of drugs	University of Liverpool
Synthesis of Components of Pharmacological or Therapeutic interest	University of Manchester I. of S. and T.
A study of the processes concerned with the metabolism of drugs and toxic substances	St Mary's Hospital Medical School
Determination of the Structure of Pharmacologically Active compounds	University of Sheffield
Studies in the chemistry of organo-phosphorous compounds	University of St Andrews
Synthesis of 4 Hydroxy 1, 2, 3, 4 Tetrahydriosoquinolines	Portsmouth College of Technology
Histopathological Studies in Neurotoxicity Institute of Neurology	London University
Research on the optical properties of Aerosols	University College of South Wales
The Absorption and Removal of Hydrocyanic Acid Gas by Solids	University of Bristol
Metabolism and Toxicity of Highly Fluorinated Cyclohexames	University of Birmingham
Effect of prolonged inhibition of Cholinesterase in Animals	Middlesex Hospital Medical School
A combined behavioural electro-physiological and biochemical investigation of the central action of certain drugs	University of Birmingham
Investigation of the preparation and properties of fibrous absorptive carbons	University of Bristol
A Physico-chemical investigation of the reaction of Nucleophiles with unsaturated gases	King's College, London
An investigation of the ability of some biological systems to inactivate acetyl chlorine and of the mechanism of such inactivation	Chelsea College of Science and Technology
Genetica Factosa and the effect of infantile stimulation on the activity of the acetycholine—cholinesterase and other systems in the brain of the rat	University of Southampton
Nucleophilic Catalysis in Relation to the treatment of Organophosphate poisoning	University of Kent
Some aspects of the Biochemistry of the skin	Queen's University, Belfast
Factors influencing the gelation of liquids with special reference to organic liquids and gels without chemical cross-links	Welsh College of Advanced Technology
The effect of drugs on neuro-muscular transmissions and contractile force of mammalian skeletal muscle	University of Bristol
Studies relevant to the reactivation of Organo-phosphorus inhibited enzymes	University of St Andrews

Microbiological Research Establishment

Nature of Research	*Location*
Investigation of Fluorocarbohydrates	University of Oxford
Diffusion controlled electrodes for use in biological systems	Birkbeck College, London University
Investigation of the use of Hydrated Calcium phosphate and related materials for the purification and fractionation of viruses and viral compounds	Oxford College of Technology
Fractionation of Compounds of Microbial Origin important in the pathogenesis of infectious diseases	University of Birmingham
Electrophoretic Behaviour of Bacteria	University of Liverpool

* Source Hansard, May 29th 1968

10. United States

Elinor Langer

Recently research on the chemical and biological weapons establishment in the United States has made considerable progress. Research within the establishment has undoubtedly made progress as well, but that is beyond the control of the concerned scientists, campus activists, and reporters who are responsible for the upsurge in public attention to the CBW question. As a result of these labours we can begin our discussion on a somewhat more sophisticated level, and specifically by clearing the air of a number of points of official propaganda concerning CBW that, while not necessarily technically untrue, have a cumulatively misleading effect. Briefly, these official contentions are that the CBW programme is marginal; that it is inexpensive; that it is unsuccessful; and that it is defensive in nature.

The terms "marginal" and "inexpensive" are relative ones, applicable to the CBW programme only when compared with the nuclear weapons programme, the military space programme, or any of the other multi-billion-dollar American defense activities that involve the substantial acquisition of hardware. Because chemical and biological weapons development is by its nature less costly, comparative expenditures reveal nothing of the scope of the programme, the intent of its sponsors, or their degree of commitment to it. The scientific resources of the United States are so great that commitment of even a fraction of its energy holds promise of significant results. In other words, the CBW programme can be both marginal and capable of vast destruction.

Second, "unsuccessful". For many years certain sections of the U.S. Defense Department have made an effort to communicate the sense that the difficulties in developing CBW weapons, particularly biological weapons, were insurmountable. (The CBW enthusiasts, housed chiefly in the Chemical Corps, have always resented this and have tried to put their contrary view across, but have been somewhat restrained by security precautions in doing so.) The origins of this contention lie in part in public relations and in part

in the genuine dilemmas encountered by the research programme in its early years, particularly during and after World War II. To be usable as weapons systems, biological organisms must possess a number of complex characteristics simultaneously. In the early post-war years it was not clear that the necessary combinations could be achieved, and there was honest debate over feasibility. There is still debate and much of it is still honest; however there is also an obvious tendency on the part of Government officials and researchers to use the earlier difficulties as a smoke-screen for the substantial progress that has been made. Men of enormous ability have been working prodigiously on these problems for a number of years. References in the open literature and interviews with those close to military developments leave little doubt that these workers have met with much success, particularly in understanding techniques of aerosol dissemination. In addition, references in military manuals strongly suggest that at least some biological weapons systems have become available. In the United States the answers to two key questions that would help establish the truth ("What is the current view of the feasibility of biological weapons?" and "Which biological agents have gone into the production stage?") are classified. Granted that classification can conceal failure as well as success and is frequently used in that fashion, the point is that we have no reason to assume failure.

Third, "defensive". With a few exceptions, such as development of detection and protective equipment, little CBW research can be accurately described as defensive—a word that may conceivably apply to the intentions of the policy-makers (*i.e.*, we are doing it because others are doing it) or to the occasion of CBW's use (*i.e.*, we don't mean to use it first). Because of the nature of chemical and biological weapons, research even in seemingly 'pure' areas, such as the development of vaccines, has at least equal implications for offensive and defensive use. This central point has in fact played a key role in early (and continuing) internecine fighting about the CBW programme. When the biological warfare programme was expanded around 1942 the Army Surgeon-General took the position that only the defensive aspects should be studied. The Chemical Corps argued successfully that offensive and defensive aspects could not be separated, and was awarded control of the entire programme. For years the disapproval of the Army Medical Corps of the CBW programme was so intense that they refused to station a medical team at Fort Detrick, the BW research centre. In recent years, however, opposition has waned, and the Army doctors have

been represented at Detrick, though the medical unit appears to centre its attention on medical aspects of CBW research such as immunization and therapy. As one of the military's CBW manuals points out "CB defence is a prerequisite to an attack capability."[1]

Development of chemical and biological research

The current healthy state of the U.S. CBW research programme is intimately related to a larger trend in U.S. weapons policy, the swing away from the 'massive retaliation' doctrine that began in the late 1950's and reached its fruition under Kennedy in the New Frontier. Before that time the Army Chemical Corps was something of an orphan, a victim of the uproar against gas warfare that set in after World War I. The Corps believed in itself and went about touting statistics demonstrating that gas casualties in the War suffered less than other types of casualties (the statistics are still frequently recited), building up its constant theme that chemical and biological weapons are more "humane" than other forms of warfare. But the audience was small and made up mostly of the already committed. The Corps was given only crumbs of the military budget, usually around $35 million a year. There was another peak, during the build-up of Detrick at the time of World War II, but that concentration dissipated too in the demobilization after 1945. In 1959 the Corps invented and carried out an expansionist mission "Operation Blue Skies", designed to win public support for its latest enthusiasm, "incapacitating" weapons, particularly psychochemicals. It was that campaign, endorsed by potent interest groups such as the American Chemical Society, that dovetailed with the decision of the Kennedy administration to acquire a weapons arsenal more versatile than the nuclear stockpile. CBW budgets began to rise, along with the budgets for a variety of other non-nuclear armaments which seemed to offer more flexibility in fighting limited wars. Between 1961 and 1964 the CBW budget (Research and Development) nearly tripled, rising from $57 million to about $158 million. The bulk of R & D money is spent by the Army, although the Navy and Air Force in recent years have also undertaken CBW research. In 1961 about another $136 million was spent (by the three services) for procurement; procurement figures for more recent years are not available. In addition to the funds for R & D and procurement, there is a large standing capital investment in CBW. How large it is impossible to say as the figures are tucked away under various camouflaged titles in the military budget. However, according to its own advertisement, Fort Detrick

alone, the centre of U.S. biological warfare research, occupies 1300 acres of land (near Frederick, Maryland), has a building complex valued at $75,000,000, "one of the world's largest animal farms", and "facilities for conducting research with pathogenic organisms that are among the best in the world".

Civil involvement

For a variety of reasons the military has found it necessary and desirable to contract out a substantial portion (perhaps as much as half, or more) of its CBW research and development to universities, research corporations, and private industry. The reasons are interesting and reveal something about the climate in which work in CBW is performed. The Chemical Corps has a public relations problem even within the Pentagon. Defense Department employees, who have attended high-level Pentagon briefings on a whole range of U.S. weapons systems, report that seasoned generals and admirals who can calmly devote whole mornings (or whole careers) to the business of nuclear destruction become queasy and indignant when it is the Chemical Corps' turn at the blackboards. The reluctance of competent researchers to submit to the conditions of work on military-controlled bases has posed considerable problems of recruitment for 'in-house' research. In addition, there has been considerable interest on the part of industry in getting a share of the work. Reluctance to work on a military base is not the same thing as scruples about the substance of the research, and the contracting-out operation until recently ran into scant opposition. In a sense it has permitted university and other private researchers to have what they might well consider the best of two worlds: military money and private prerogatives.

An item-by-item count of who is doing what has been built up over the last few years, and part is shown in Table 10. But the conclusion may be briefly summarized: virtually any topic we could think of that would facilitate the development of usable weapons systems is under study in the United States. Labour is rationally divided, with basic scientific research being performed chiefly at universities; research on the means of delivery by industry, particularly the aero-space industry; and a variety of functions of both sorts by the non-profit organizations. It appears that at least fifty universities, perhaps twenty-five companies, and about a dozen research institutes are now holding or have recently held CBW contracts. The work ranges in scope from the million-dollar research on weapons-dissemination at the University of Pennsylvania to

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individual medical research contracts of perhaps $10,000 a year; it ranges in apparent lethality from the study of the "supersonic delivery of dry biological agents" (Litton Industries) to investigation of the "chemical and physical principles applicable to BW detection" (The University of Buffalo). The list of co-operators includes not only universities, non-profit organizations, and industry, but some of the most prestigious organizations of American science, including the National Academy of Sciences, which runs a fellowship programme, and the American Society for Microbiology which has for years provided Fort Detrick with advisory services. The U.S. Public Heath Service maintains close liaison with Fort Detrick, and receives a few hundred thousand dollars a year for its efforts. A number of foreign university researchers have also accepted contracts and grants stemming from the CBW research programme.

Defense establishment

Notwithstanding all the outside assistance, the internal capacity of the Government remains substantial. Fort Detrick, with a staff of 120 Ph.D.'s, 110 M.S.'s,* 320 B.S.'s,* 34 veterinarians, and 14 physicians, is the principal CBW station with a major research function. Only about 15 per cent of the research findings are published through conventional scientific channels. Detrick is particularly proud of the three or four occasions when, in co-operation with other scientific societies, it organized research symposia (the principal topic: airborne infection). Another of these occasions—a joint symposium with the American Institute of Biological Sciences in celebration of Detrick's 25th birthday—has recently taken place and—a symptom of changing tempers—was boycotted by a large percentage of the intended participants. But most of the research done at Detrick becomes part of a secret literature managed by the Defense Department and available to other Government agencies or contractors only on a 'need to know' basis.

Fort Detrick, in addition to its research activities, is involved in process development, small scale production, and design and operation of pilot plants. Closely related to Detrick is the Dugway Proving Ground which employs about 900 people (and occupies an area in Utah almost as large as the county of Hampshire). Dugway is the principal station for field assessment and testing of

* The American M.S. and B.S. degrees are similar to the British M.Sc. and B.Sc. *Ed.*

chemical and biological munitions. According to Pentagon officials there are no large-scale trials of chemical and biological agents on human subjects. Limited testing is done at Detrick using volunteers—Seventh Day Adventists who serve in the Armed Forces only as non-combatants—and experiments have from time to time been performed on prisoners. But the military evidently believes that the value of testing is outweighed by the dangers of contamination and limits its trials to animals or to non-pathogenic simulants of active biological warfare agents. Sometimes testing on animals is inadvertent, as with the accidental slaughter of 6000 sheep during recent tests of a new nozzle for dissemination of nerve gas at Dugway (the sheep were grazing on ranch lands 30 miles away). It is worth noting that the Pentagon insists—contrary to opinions voiced by some in the conference—that these trials would form an adequate basis on which to test deployment of chemical and biological weapons.

Biological munitions are produced at Pine Bluff Arsenal, a 15,000 acre installation outside Pine Bluff, Arkansas, which employs about 1400 people. Pine Bluff also produces toxic chemical munitions and riot-control munitions. Its activities range from manufacturing the agents to filling and assembling the weapons. Research and development on chemical weapons and some production and assembly take place in a number of components of the Edgewood Arsenal, in Edgewood, Maryland. A variety of chemical munitions, including nerve gas, mustard gas, 'incapacitants', and anti-crop weapons are produced at the Rocky Mountain arsenal in Denver. The U.S. also operates a major manufacturing plant—at an estimated annual cost of $3·5 million—in Newport, Indiana, where the nerve gas, sarin (see Chapter 1) is produced and loaded into rockets, land mines, and artillery shells. The plant is managed by the Food Machinery Corporation, has 300 employees, and is reported to have been operating twenty-four hours daily since 1960. Additional chemicals were manufactured during the middle 1950's at a plant in Muscle Shoals, Alabama, now closed. Training of officers and troops in the use of chemical and biological weapons takes place at the U.S. Army Chemical Centre and School at Fort McClellan, Alabama; related activities in the Air Force occur at Eglin Air Force Base, in Florida.

Most of the procurement of the chemical defoliants and herbicides in use in Vietnam is done on a commercial basis.

In the summer of 1967 the Pentagon announced the award of $57·7 million to the following chemical companies to purchase

defoliants for Vietnam: Dow, Diamond Alkali, Uniroyal, Thompson Chemical, Hercules, Monsanto, Ansul, and Thomas Hayward. About another dozen companies also sell chemicals, including various tear gases, to the Government.

Chemical weapons are produced in forms fit for the requirements of all military services. They are available in a variety of manifestations from artillery shells to the Sergeant missile, which has a range of 100 miles, the Honest John and Little John rockets, and chemical land mines. They are also available as bombs for delivery by conventional military aircraft. Detailed information on delivery systems for biological agents is classified, but the unclassified manuals suggest that they are available as warheads for missile systems (for large area attacks), as cluster bombs, and as spray tanks and dispensers mounted on aircraft.

The justification—the protesters

Recent protests in the United States have called forth a variety of defences from those in CBW and related research and development. Some representative samples: "We are an electric utility and our contract with the Federal Government covers the supplying of electric services for the Dugway Proving Ground. That is as far as our relationship with the Dugway Proving Ground goes and what the power is used for is beyond our knowledge." E. A. Hunter, Vice-President, Utah Power and Light Company.[2]

("Government contracts represent a big part of our research efforts—it's a way of life." H. Ridgely Warfield, director of John Hopkins Institute for Co-operative Research, recipient of over $1 million in contracts for "studies of actual or potential injuries or illnesses, studies on diseases of potential BW significance, and evaluation of certain clinical and immunological responses to certain toxoids and vaccines."[3])

"In no way do we feel that our firm is being 'morally compromised' by handling CBW work for the Pentagon. We feel we have an obligation to respond to Pentagon requests for assistance." R. L. Carnahan, vice-president, Travelers Research Center, a major recipient of contracts involving study of factors affecting dissemination of chemical and biological weapons, and author of a study of public reaction to CBW use.[3] A similar response was made by Dow Chemical Company following protests over its manufacture of napalm. Dow issued a statement which read in part: "Dow accepted this contract because we feel that simple good citizenship required that we supply our Government and our military with those goods

they need when we have the technology and capability and have been chosen by the Government as a supplier."

"No war was ever fought with the intent to be kind. Each weapon as it is used to kill makes man quite as dead, his family quite as bereaved. As a weapon is effective, so it will be used, and the resources of the entire nation will be utilized by the Government on request, or by requisition, to produce those weapons deemed by the Military to be most effective in concluding the war . . . Scientists make their peculiar contribution to man's welfare. At times it is used well. At times, inventions for survival in one time of history become agents for genuine human betterment and even become powerful agents to impress on man his need to abrogate force. But other men than scientists also direct the course of civilization. If you spend your powers assaulting one aspect of a problem which cannot be solved by that aspect, you waste your powers. By your reasoning, nearly every facet of American life should be halted, from the production of breakfast food to air transport, since almost all contribute to the prosecution of this war which no one wants. Stop the war with adequate international action or accept the reality that man is still primitive, and that a man or a woman or a child is as dead from mortar fire or shrapnel or a simple rifle." Melvin Calvin, Nobel prize winner, professor of chemistry and molecular biology, University of California, Berkeley, member, Board of Directors, Dow Chemical, manufacturers of napalm. (Calvin's letter appeared in the campus paper, Daily Californian, after a graduate student had challenged him to state his views on the manufacture of napalm and its use in Vietnam.)

"I submit that there is almost no major corporation that is not contributing in some way to the Vietnam war regardless of the points of view of its executive". Thomas Watson, President, IBM. (Letter to Brown University campus paper criticizing students there who attacked Dow Chemical.)

In general, university protesters over CBW and related war research have divided into three groups—a 'left' or radical minority willing directly to confront the issues of the moral use of research results for war; a 'rightist' or conservative minority (usually in the engineering sections) fearful of the damaging consequences to themselves of ending military contracts on any grounds; and a liberal 'centre' willing to oust the research but chiefly on the libertarian or educational grounds that faculty members must not interfere with one another's interests. The Dow argument (the Government needs us) is also heard with regard to university and CBW, though less frequently.

A number of universities have attempted, in the wake of unfavourable publicity, to divest themselves of their CBW research projects (the University of Pennsylvania) or of all university-sponsored military or secret research (Cornell, the University of Michigan). In each case, divestiture has provoked considerable tumult, and efforts have been made to find grounds for cancellation not involving moral censure. Instead, dialogue has focussed on the issues of 'publishability' and 'relevance of the research to the university's teaching mission'. The contrary argument, made by—among others—University of Pennsylvania President, Gaylord Harnwell, is that by tradition American universities guarantee their faculties 'academic freedom' to research whatever questions they choose; the administration's job is to insulate the faculty from changing political winds.

Policy

U.S. policy regarding CBW see-saws between two poles—the declaration of no first use by President Roosevelt in 1943 and the refusal of the Pentagon and State Department to endorse a similarly worded declaration in 1960. What about Vietnam? So far, *known* U.S. operations have been confined to spraying the jungle and NLF food crops, and to the use of theoretically non-lethal gases, albeit not necessarily in a non-lethal fashion. (The use of BZ has been alleged, but to my knowledge not proven. The same is true of alleged experimental use of other chemicals in underground tunnels, etc.) [But *see* Chapter 7.] According to a recent report by the Midwest Research Institute (commissioned by the Pentagon in response to scientists' protests), enough herbicide was used in 1967 to 'treat' 965,000 acres, although because many areas were treated more than once, the total acreage defoliated was less. Its conclusion, briefly, is that the possibilities of long-term adverse effects are minimal and that the spraying may even be beneficial in some instances. Is this chemical and biological warfare? U.S. officials repeatedly say it is not: others insist that it is. But perhaps the importance of the question is overrated. Surely the important thing is not to name these activities, but to understand what they, along with the other barbarities, are doing to that country. The question in the use of herbicides, as Jean Mayer has pointed out (Chapter 6) is the ethics of starvation, not the ethics of the means of destroying crops. The issue in Vietnam is not the vehicle of destruction but the destruction itself.

I think that this raises an important question: Why are we spend-

ing so much time on CBW? I have had to think about this a great deal myself in the course of my own work and have come to reject most of the conventional answers. I do not believe that CBW is less moral than other means of war, or even that it is necessarily a more unpleasant way to die. I do not accept the argument that the tenuous restraints on CBW which have been followed up until now indicate that man is capable of 'civilizing' warfare; I believe that the United States—as well as other countries—would use any means of war that they believed, overall, would advance their interests.

I believe that CBW is a violation of international law, but I believe that 'resettlement' of villagers in Vietnam is a violation of equal proportion, and that law is as unlikely to restrain one as the other. I agree with those who say that CBW is a perversion of science, but I believe that with Hiroshima and other developments science had already demonstrated sufficient perversion and CBW does not appear unique.

I think those who argue that CBW should be watched because it is of special utility in the counter-revolutionary wars the United States and other imperial powers will undoubtedly be fighting have so far done no more than assert their point. Finally, I think that to confine talk about the risks of 'escalation' in Vietnam or elsewhere to chemical and biological weapons at a time when escalation is taking other equally dangerous paths is academic.

Why then bother about CBW? I think there are a few main reasons, although perhaps they will seem rather ethnocentric ones. First, I think that many people believe that some or all of the arguments above are true, including people who, for a variety of reasons, are unwilling to engage in direct anti-war activity or to oppose the government's policies overtly. This is true, for instance, of the scientists' coalition in the United States as well as of a number of university-based protesters. As a result CBW has become a good tactic for dissidents; it is a vulnerable point in the military-industrial complex. A related reason is that it exposes how some of America's structural and civil liberties, such as 'academic freedom', are inadequate to guarantee an ethical scholarly climate.

Concern about CBW may, in consequence, help to promote serious university reform. The same may be true in Britain and in a number of continental countries that co-operate with the U.S. defense-research effort. A final reason is that CBW is small enough and manageable enough for outsiders to come to grips with the problem as a whole, and reform seems possible; it weighs less

heavily than the tangled mass of the Vietnam war, on the one hand, or the arcane mysteries of nuclear defense, on the other. Progress in opposing CBW seems possible. But the fact is that we could abolish CBW altogether without making the world much safer or our universities more wholesome. At the risk of sounding trite, CBW is only a symptom. Perhaps these are not good words for a CBW conference. But it is important to remember that CBW is only a battle; it is not the war.

TABLE 10 U.S. university involvement in CBW research, 1961-68*

		SELECTED EXCERPTS	
University	Department	Researcher	Project
Boston	Chemistry	Dr. Mason	**CW agents**
California (Berkeley)	Chemistry	Dr. Rapoport	**Lethal and incapacitating agents**
California (Los Angeles)		Dr. Taylor	**Medical aspects of CW**
Connecticut	Pharmacology		**Synthetic products of psychedelics**
Illinois (Urbana)		Dr. Finger	**Crop and food destruction agents**
Illinois Institute of Technology		Dr. Ehrlich	**Improved biological aerosols**
Indiana		Dr. Rudsell	**More effective toxic compounds**
Johns Hopkins			**Disease of potential BW significants**
Maryland		Dr. Woodward	**Vulnerability of man to BW**
Massachusetts		Dr. Kunsberger	**Emetics as possible incapacitating agents**
Oklahoma			**BW in remote conflict areas**
Pennsylvania		Drs. DuBois, Koelle, Haugoard	**Applied research on CBW systems including evaluation of defoliants in Vietnam**
Virginia Medical College		Dr. Hoff	**New lethal and incapacitating agents**
Washington State		Dr. Hendrix	**Anti-crop warfare**

This list is selected from a directory of 57 universities, institutes all having U.S. Department of Defense contracts: the total of all D.o.D. contracts to U.S. universities amounted to $ 380 million in 1966. Other colleges etc. include:—Brooklyn, Buffalo, California Medical Schools, Chicago, Clarkson, Columbia, Cornell, Delaware, George Peabody, George Washington, Georgia, Hahneman, Iowa, Kansas, Maryland, MIT, Michigan, Minnesota, New York, N. Carolina, Ohio, Oregon, Pittsburgh, Rutgers, St. Louis, Stanford, Tennessee, Texas, Utah, Washington, Western Reserve, William and Mary, Wisconsin, Yale.

* Data compiled from details published by Michael Klare, *Viet Report*, January, 1968.

11. Soviet Union

D. E. Viney

This chapter seeks only to assemble a few salient facts publicly known about the potential for chemical and biological warfare of the Soviet Union and the Government's apparent attitude towards it; and to add a minimum of what seems to the author reasonable speculation.

Potential

Soviet spokesmen have, since World War II, repeatedly forecast that future world conflicts are bound to involve the use of both chemical and biological weapons (if only because the 'aggressor' would initiate it), and as the Soviet Union is patently capable of producing many kinds, it would be surprising if she had not proceeded from intensive research to the stockpiling of whatever agents she had decided to rely upon. This further step would of course not necessarily follow if the Soviet Union came to regard a conflict between CBW-capable powers as unlikely, or if she abandoned faith in the efficiency of deterrence, or the legitimacy of retaliation, in the CBW field; there is no sign of her adopting these positions. There is no reason to think she would stockpile newly discovered agents of dubious value; one can be less sure that she would destroy old stocks of agents which, for her, had been outdated by more sophisticated ones, but might again prove useful in special circumstances or be made available to friendly nations with different military requirements.

It is well known that the Russians acquired a headstart in the business of nerve gases at the end of World War II by overrunning a German plant manufacturing the least toxic of the nerve gases then known, tabun, and seizing a stock of some 12,000 tons of it. In view of their subsequent interest in organo-phosphorus compounds generally, it would be surprising if they had not kept at least level with the West in perfecting the manufacture of more toxic ones, and of both more and less volatile agents than tabun. Perry

Robinson (Chapter 1)[1] mentions that the Russians were, surprisingly, the first (in 1960) to publish percutaneous toxicity figures for the V-agents.

Among the less advanced agents, the same chapter mentions evidence that the Russians stockpiled what might be called the 'lethal-irritant' dichloroformoxime, in World War II, as well as adapting for field use hydrogen cyanide (which in the West has often been dismissed as a possible weapon because of its volatility).

Around 1960 U.S. sources, in the course of a campaign to make CBW more acceptable, published statements claiming that some 15 per cent of all ballistic warheads on the Warsaw Pact front were chemical ones—an estimate neither confirmed nor denied by official quarters in any other country (nor, incidentally, attacked as provocative by Soviet spokesmen). Some Warsaw Pact exercises have certainly been conducted on the assumption that chemical weapons would be employed, alone or in conjunction with nuclear attack. The use of chemical warheads with tactical ballistic systems such as the SCUD missile (range up to 150 miles) or the FROG free-flight rocket (range of later versions up to 30 miles) would represent a formidable capability, each missile, assuming a warhead filled with nerve gas, being able to produce a high proportion of fatalities over an area of 1 sq.km. or more among target personnel, unless they were provided with the most advanced protective clothing and were wearing it at the time of attack.

American writers have spoken of Soviet preparedness for delivery of chemical agents by such other means as normal artillery shells and aircraft bombs. They have also mentioned spray-tanks for dissemination from aircraft,[2] and though it might be thought unlikely that much faith would be placed on aircraft flying at vulnerably low altitudes and speeds in a European theatre of conflict, their military utility could be higher elsewhere; it is in any case known (*see* Chapter 1) that the Russians adapted hydrogen cyanide for dispersal by just such means.

Nothing is known, on the other hand, of any Soviet success in developing feasible incapacitating agents. As for biological agents, intensive research can be assumed if only for the same reason as with other advanced countries, *i.e.*, the need to produce vaccines. It is widely reported that the Soviets have field-tested mild pathogens, and they obviously have ample territory for the purpose. Soviet research into the possibilities of mass vaccination from the air must have suggested military applications. However, the crucial facts require no detailed evidence; the Soviets are clearly in a posi-

tion to embark on large-scale manufacture of biological agents if ever they decide to; and equally, there is no reason to assume that they have formulated a military requirement of this kind, or ever will. The felt need for a deterrent, *i.e.*, retaliatory, capability does not mean a felt need for the means of replying to every agent with the same agent, or even to every whole class of agents with the same class.

As for Soviet defensive equipment against chemical or biological weapon attack, it was said in 1964[2] to include a very advanced gas-mask, adequate qualities of protective clothing against chemical agents, detection and identification equipment similar to the West's, but no automatic warning systems. Civil defence preparedness is actively promoted by paramilitary and youth organizations through which perhaps half the population have at some time received thorough classroom training in defensive techniques. Underground shelters with air-filtration exist in major population centres, though their capacity is no doubt just as inadequate in terms of general war contingencies as in any other country.

Military doctrine

Doctrine can best be inferred from deployment, so that the following remarks apply only to the Soviet Warsaw Pact front, and only to chemical weapons.

Until a few years ago it may well be that chemical warheads were seen by Russians partly as a substitute for tactical nuclear warheads, which were harder to develop than the more powerful nuclear warheads used on long-range missiles, and so were not available in sufficient quantities to counter the known American capabilities. Assuming however that there is now some balance on the tactical nuclear level, a lethal chemical weapon capability would still make sense in these roles: (i) tactically, to destroy personnel without destroying installations and communications to one's own detriment, and without rendering large areas inaccessible to one's own side for such long periods as would nuclear weapons; (ii) to furnish a military option above the conventional level, but still short of nuclear escalation; and (iii) to deter chemical weapon attack by an opponent.

Of these three roles, the first two only remain attractive as between nuclear opponents on the assumption (by no means certain) that the use of advanced chemical weapons would avert nuclear escalation and not simply precipitate it. But the third, deterrent, role would still remain valid, and even the existence of large, deployed

stockpiles on the Soviet side would be consistent with a Soviet view that deterrence was their primary role, but that the capability must remain a large one in case deterrence failed. Although a distinction can be made of course between use of a chemical weapon capability to deter *any* attack and to deter a chemical weapon attack, this does not invalidate the foregoing argument.

Exaggerated inferences can be drawn from the statements of Soviet military men, who are after all just as liable—sometimes more—to inject political and empire-making considerations into their analyses as those of other nations. Marshall Zhukov's 1956 *obiter dictum* before a Party Congress that "a future war, should it be unleashed, will be characterized by the massive use of . . . means of mass destruction such as atomic, thermo-nuclear, chemical, and bacteriological weapons" cannot seriously be used in 1968 to argue an active Soviet interest in possessing an offensive capability in all parts of the spectrum. Marshall Sokolovsky says more soberly in the 1963 edition of *Soviet Military Strategy* that in a future war "one must expect the aggressor to use chemical and bacteriological weapons in conjunction with nuclear ones". A prefatory note by the publishers of *Bacteriological Weapons and How to Defend Against Them*[3] even more cagily says that "future war is considered by the aggressive circles of the USA to be a total war in which means of mass destruction will be widely used". This booklet is recommended for use by "agitators, propagandists, lecturers and teachers" and is largely devoted to a description of epidemic diseases and means of controlling them. It is possible, then, that instruction based on such books is seen primarily as a means of inculcating general hygienic practices and knowledge, with the anti-Western implications as a propaganda bonus. It is interesting, however, that this particular book does not repeat the charges lavishly made by Russians and Chinese alike at the time of the Korean War, that the Americans had actually used biological weapons—in particular insect-borne agents—at that time. Its historical review jumps from alleged Japanese activities during World War II to the use of defoliants and anti-crop compounds in Vietnam.

Arms-control doctrine

The USSR is one of the fifty or so countries which have signed the Geneva Protocol of 1925 binding them not to use "asphyxiating poisonous or other gases", nor "analogous liquids, materials or devices", nor "bacteriological methods of warfare" against fellow-signatories. Like Britain and many others, the Soviets put in a

reservation releasing them from these restrictions if they found themselves at enmity with a country "whose forces, or the forces of whose allies" failed to respect the Protocol. Strictly, then, the USSR is not inhibited by the Protocol vis-à-vis the U.S. (as a non-signatory), nor would she be against British forces allied to U.S. forces in a conflict in which the United States was not respecting the Protocol. Of course, the United States may be held to have a virtual 'no first use' policy regarding CBW; and some jurists argue that the Geneva Protocol, though contractual in form, has entered into the body of international customary law (*see* Chapter 12) through long (if not universal) observances, so that it is binding among non-signatories as much as signatories. Possibly some Soviet jurists would take this view, and the tendency in Soviet legal writing is certainly to take the Protocol seriously and grant it some efficacy as a factor which, alongside mutual deterrence, prevented chemical agents from being used in World War II.

These considerations do however draw attention to the greatest East-West bone of contention in regard to CBW, *i.e.*, whether non-lethal chemical agents are in fact banned under the Geneva Protocol. It is outside the scope of this chapter to mention the many arguments that have been adduced on each side. Suffice it to point out that though the Soviet authorities have used tear gas for riot-control just as Western states have done, it by no means follows that they, or we, must logically condone the use of the same agents in wartime against enemy forces. The last major international debate on CBW was that in the U.N. at the end of 1966, where the Hungarians and various African delegates made most of the anti-American running; the Soviet delegates do not appear to have spoken at all. (The debate ended with a general appeal for all countries to adhere strictly to the Protocol, but cast little light on the points at issue.) The Soviet Union is meanwhile on record with its 1962 plan for General and Comprehensive Disarmament (GCD) which proposes complete abolition of both chemical and biological weapons, during the second of three 3-year disarmament stages; against this, the corresponding Western blueprint advocates study of the special difficulties of chemical and biological arms-control in Stage I, a halving of stockpiles in Stage II, and elimination of the rest and of manufacturing facilities in Stage III. The Soviet plan makes no mention of inspection difficulties, nor of discrimination problems, and by analogy with the Soviet stand over inspection in other fields one must assume that at present they would not contemplate giving alien inspectors the right to do anything but observe

the actual destruction of agreed quantities of agents. It seems a long time, too, since those inter-war disarmament negotiations when the Soviets proposed that factory committees in each country should ensure the non-production of prohibited weapons.

It must be feared in any case that implementation of the GCD plans is not a thought uppermost in the minds of negotiators of any major power today, so doggedly do the obstacles seem to have grown instead of shrinking. Meanwhile the Soviets are probably happy to stand pat on the Protocol as they see it, plus their known capability in those CBW fields which they consider effective. In regard to the contentious non-lethal agents, they may feel themselves to be in somewhat of a public relations quandary. As occasional users of riot-control agents they could not approve of any statements appearing to condemn these as intrinsically inhumane. On the other hand they probably do not foresee any great military need for these or other non-lethal agents on their own side (expecting to be on the guerrilla, not counter-guerrilla, side of limited wars) and can hardly oppose any criticism of the Americans in Vietnam. It is therefore not likely at present that the Russians will be eager partners in any efforts to secure a redefinition of terms as a means of strengthening the Protocol. And even where lethal agents are used in clear violation of the Protocol, as in the Yemen, political considerations will put a severe brake on Soviet reactions. It is not suggested that those seeking ways of constraining CBW, and especially of constraining the spread of CBW capabilities, should give up: simply that they should bear the political background in mind.

DISCUSSION

Two major issues were raised in this discussion. First, the nature of the quadripartite and tri-partite agreements between Britain Canada, Australia and the United States were questioned by Professor Wall, who quoted an interview given by Dr Pennie, Deputy Chairman of the Canadian Defence Research Council to a Canadian magazine.[1] This interview gave two important items of information.

One was a fairly detailed description of the Suffield Proving Ground north of Medicine Hat in Alberta, Canada. It consists of a set of closed laboratories and also a thousand square mile tract of

land which is used specifically, according to Dr Pennie, for studying the spread of organisms. Equipment from all parties to the agreement was tested there. Asked how the pact works, Dr Pennie replied: "There are regular meetings of scientific staff engaged in this particular line of work in all three countries, and there is free disclosure of information in all these areas. We also attempt to divide the work between laboratories whether they be in Britain, the United States or here (Canada) wherever the work suits. You can understand that you need specialized facilities for this type of work. There are facilities in Canada which don't exist in Britain; there are some in Britain which do not exist in the United States so it makes good sense and sound logic to try and divide up the work."

Professor Wall went on to point out that the existence of an agreement of this kind places equal responsibility on all four members of this quadripartite pact.

The second major theme concerned the issue of secrecy: should work at Porton continue to be secret? If it was exclusively defensive in character, why could it not be declassified?

The general arguments in favour of secrecy were indicated by Dr Viney and Professor Meselson who pointed out that there were two sorts of secrecy: conspicuous and clandestine. The former variety could lead to opinions in other defence establishments that there was something worth investigating. The very knowledge of the existence of secret research is a powerful deterrent to one's enemies. But the clandestine sort also has considerable advantages —there is less danger of spying and political control is easier.

Countering the move to declassify secret research at Porton, Dr Viney pointed out that if one disclosed all the facts about a genuinely defensive capability, then the research becomes undermined because the potential enemy knows what one is equipped to defend oneself against. Further, revealing all that is known about CBW would amount to a proliferation of dangerous knowledge. Whilst this does not mean that secrecy must be eternal in a world of sovereign states, there are sound, peace-loving reasons for maintaining secrecy until such time as it can be assumed that all countries have given up offensive intentions.

Finally, Professor Meselson showed that, from the point of view of a British Minister of Defence, Porton probably offered an extremely high return on investment. In exchange for a small expenditure at Porton and the maintenance of the quadripartite agreements, he had access to the results of the much larger programme of the United States. For this reason alone, the Ministry would probably fight quite hard to maintain secrecy.

After some theorizing as to the actual percentage of secret work carried out at Porton, the Conference heard the arguments in favour of declassification. Mr T. Dalyell (Member of Parliament for W. Lothian) suggested that many of the scientists at Porton might prefer to work in the open in order to achieve healthier relations with university scientists. Dr Sidel felt that the chances of the development of offensive weapons by any power would be

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much reduced in a world where a policy of complete openness was pursued. Dr Rose pointed out that throughout the history of science, secrecy or no secrecy, there are many instances of simultaneous discovery. For example, it is now abundantly clear that the Soviet Union would have developed its own atomic weapons within a very short space of time irrespective of the leakage of secrets that occurred in the 1940s and early 1950s. An exactly comparable situation applies today to CBW—the South Africans are surely capable of making many of the weapons, whilst one can be reasonably confident of the ability of the universities and chemical industry in Western Germany to be sure that they require very little help from Porton's publications. In fact, one could say that the synthesis of the chemical agents described by Perry Robinson are within the scope of any reasonably competent organic chemist. As far as biological agents are concerned, the central problem is that of mass production and this should be within the capabilities of any drug house with a pilot plant.

Mrs Galston attempted to deal with Dr Viney's concern that scientific information should not fall into the wrong hands by pointing out that in our present world every hand at this point and for this type of weapon is the 'wrong hand'.

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PART FOUR

LEGAL ASPECTS OF CBW

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12. Legal Aspects

I. Brownlie

General features of the law

The first and major characteristic of the law of war is its paradoxical nature. The United Kingdom *Manual of Military Law*[1] states that the development of the law has been determined by three principles:

> First, the principle that a belligerent is justified in applying compulsion and force of any kind, to the extent necessary for the realisation of the purpose of war, that is, the complete submission of the enemy at the earliest possible moment with the least possible expenditure of men, resources and money; secondly, the principle of humanity, according to which kinds and degrees of violence which are not necessary for the purpose of war are not permitted to a belligerent; and thirdly, the principle of chivalry, which demands a certain amount of fairness in offence and defence, and a certain amount of mutual respect between the opposing forces.

In spite of the element of paradox, the law has produced certain factors of discrimination in the means of carrying on war. The principal factors are, first, the prohibition of weapons which cause unnecessary suffering in proportion to their destructive effect, and, secondly, a distinction between combatants, whether regular or irregular, and non-combatants.

The law and the statesmen who influence its development utilize categories which may not seem very sophisticated or up to date. In many ways legal categories are bound to have rough edges partly because they set standards for a great variety of people most of whom are neither scientists nor lawyers. However, the legal categories have in general maintained a sufficiently close relationship with technical developments. The law has employed the category of chemical warfare since at least 1919, though the legal reference is usually the formula which prohibits "the use in war of asphyxiating, poisonous or other gases, and of all analogous liquids,

materials or devices." The *Dictionary of U.S. Army Terms*, published in 1953, defines chemical warfare as "the tactics and technique of conducting warfare by use of toxic chemical agents." The weapons may be thought to include incendiary fuels[2] and smoke gases, but this is a matter of doubt. The indisputable content of the category is the list of anti-personnel gases; incapacitants, for example, hallucinatory drugs; choking gases, such as chlorine and phosgene; nerve gases, including sarin and tabun; gases which affect the oxygen supply in the blood; and blister gases including mustard gas and lewisite.

Since 1925 the legal materials have contained the category of "bacteriological methods of warfare". The modern technical concept of biological warfare includes bacteriological methods, but extends to a wider range of agents. The *Dictionary of U.S. Army Terms* defines biological warfare as "the employment of living organisms, toxic biological products, and chemical plant growth regulators to produce death or casualties in man, animals or plants; or defence against such action." The *U.S. Army Manual,* published in 1956, describes biological warfare as "the military use of living organisms or their toxic products to cause death, disability, or damage to man, his domestic animals or crops" and continues: "It includes the employment not only of bacteria but also of other micro-organisms and higher forms of life, such as insects and other pests . . . While certain chemical anti-crop compounds are not truly biological warfare agents, they are so considered as a matter of convenience." It seems to be a matter of historical accident that the use of gases has been regarded as chemical warfare while the use of bacteria and 'toxic biological products' is classified separately.

Other features of the law require brief notice. Much of the law consists of treaties, and in some connections states are concerned to point out that they are not a party to this or that treaty. Nevertheless, international law may develop rules of customary law which are evidenced by state practice in the form of protests, official warnings, manuals of military law, and various diplomatic materials. A treaty may by its terms provide evidence of the state of general international law in two ways: by containing statements purporting to be declaratory of existing law; and independently of the first method, by being accepted as a statement of the law even by non-parties. Finally, the modern principle is that the law of land warfare applies to international conflicts whether or not the parties characterize an armed conflict as a 'state of war'. An insurgency may constitute an armed conflict, and, although it will not be

of an international character unless there is substantial foreign intervention against the lawful government, it is now generally accepted that the general principles of the humanitarian law of war apply to internal conflicts.

The prohibition of chemical warfare in customary international law

There is a great deal of evidence in support of the view that chemical warfare as a *genus* is prohibited by customary international law. The peace treaties of World War I and the Treaty of Berlin of August 25th, 1921, between the United States and Germany, contain provisions which prohibit the importation or manufacture by Germany of certain gases and other liquids and devices, and in doing so treat *as already prohibited* "the use of asphyxiating, poisonous and other gases and all analogous liquids and devices." In 1922 the Washington Treaty was concluded.[3] This contains a provision the substance of which was to be repeated in the Geneva Protocol of 1925. Article V prohibits "the use in war of asphyxiating, poisonous or other gases, and all analogous liquids materials or devices." The United States ratified the Treaty on June 9th, 1925,[4] but it failed to come into force because of the absence of French ratification. The French failure to ratify, it may be noticed, had nothing to do with the reference to chemical warfare, but flowed from the limitations on submarines in the Treaty.

The Geneva Protocol signed on June 17th, 1925

The Protocol is the most important instrument concerning prohibited weapons. It provides as follows:

> . . . the undersigned Plenipotentiaries, in the name of their respective Governments:
>
> Whereas the use in war of asphyxiating, poisonous or other gases, and all analogous liquids, materials or devices, has been justly condemned by the general opinion of the civilised world; and
>
> Whereas the prohibition of such use has been declared in Treaties to which the majority of the Powers of the World are Parties; and
>
> To the end that this prohibition shall be universally accepted as a part of International Law, binding alike the conscience and the practice of nations;
>
> Declare:
>
> That the High Contracting Parties, so far as they are not already Parties to Treaties prohibiting such use, accept this

prohibition, agree to extend this prohibition to the use of bacteriological methods of warfare and agree to be bound as between themselves according to the terms of this declaration.

International lawyers of great authority have held that insofar as the instrument relates to chemical weapons it is declaratory and affirmative of the customary law at the time.[5] Forty-nine states signed the Protocol, which came into force in 1928. In 1944 there were forty-four parties by ratification or accession. Since then another fourteen states have become parties, including India, Pakistan, Cuba, and a number of African States. China's accession of 1929 has been recognized by the People's Republic of China. The United Kingdom, France, the USSR, and a number of other states are parties subject to a condition of reciprocity.[6] The United States did not become a party to the Protocol, but the Protocol has acquired considerable prestige and is referred to as evidence of the general law.[7] Thus in the *Shimoda* case[8] a Japanese court concerned, *inter alia,* with the legality of the atomic attacks on Hiroshima and Nagasaki, referred to the Protocol as a source of relevant rules although neither the United States nor Japan were parties to it.

The United States and CBW

The position of the United States obviously calls for further examination since its non-participation in the Geneva Protocol is a companion to that country's scientific and military resources and proven readiness to prepare and resort to chemical warfare and related techniques.

The failure to ratify the Protocol did not stem from any decisive negative stand on its subject matter but from a desire to avoid an omnibus prohibition. However, since World War II the United States policy has been more equivocal. The *U.S. Army Field Manual*[9] expressly states that "the United States is not a party to any treaty, now in force, that prohibits or restricts the use in warfare of toxic or non-toxic gases . . . or of bacteriological warfare".

American official sources have in fact adopted a fairly clear view on these matters on a number of occasions in the past. In 1932, on the basis of proposals by President Hoover, the General Commission of the Disarmament Conference adopted a resolution which condemned the use of gas, bacteriological, and incendiary warfare as methods of warfare. A study by the U.S. Naval War College in 1935[10] concluded that "the use of poisonous gases and those that

cause unnecessary suffering is in general prohibited; the use of smoke-screens and of tear gas has not been included in the category of prohibited acts, but the use of bacteriological warfare has been prohibited". Of interest is the robust statement of President Roosevelt which is printed in the *Department of State Bulletin* dated June 12th, 1943.[11] This statement includes the following passages:

> From time to time since the present war began there have been reports that one or more of the Axis powers were seriously contemplating use of poisonous or noxious gases or other inhumane devices of warfare.
>
> I have been loath to believe that any nation, even our present enemies, could or would be willing to loose upon mankind such terrible and inhumane weapons. However, evidence that the Axis powers are making significant preparations indicative of such an intention is being reported. . . Use of such weapons has been outlawed by the general opinion of civilised mankind. This country has not used them, and I hope that we never will be compelled to use them. I state categorically that we shall under no circumstances resort to the use of such weapons unless they are first used by our enemies.

In a debate in the United Nations General Assembly on October 16th, 1952, Dean Acheson, then United States Secretary of State, declared:[12] "We will not commit aggression with chemical weapons or bacteriological weapons, which we have been falsely and slanderously accused of using."

The American position would seem to be far from consistent, and in the case of chemical weapons the *U.S. Army Field Manual* ignores the evidence for the existence of a customary rule prohibiting use. In any case the American position, in so far as it appears to deny the existence of prohibitions, relates to non-prohibition of chemical and biological weapons as a class, *tout court*. Thus it is subject to various specific rules as to methods of conducting war in general. I shall enlarge on this aspect of the matter later on.

The 1966 Resolution of the United Nations General Assembly

On December 5th, 1966, the General Assembly of the United Nations adopted the following resolution:[13]

> *The General Assembly*
>
> *Guided* by the principles of the Charter of the United Nations and of international law:
>
> *Considering* that weapons of mass destruction constitute a

danger to all mankind and are incompatible with the accepted norms of civilisation.

Affirming that the strict observance of the rules of international law on the conduct of warfare is in the interest of maintaining these standards for civilisation;

Recalling that the Geneva Protocol for the Prohibition of the Use in War of Asphyxiating, Poisonous or Other Gases, and of Bacteriological Methods of Warfare, of 17 June 1925, has been signed and adopted and is recognised by many States.

Noting that the Conference of the Eighteen-Nation Committee on Disarmament has the task of seeking an agreement on the cessation of the development and production of chemical and bacteriological weapons and other weapons of mass destruction, and on the elimination of all such weapons from national arsenals, as called for in the draft proposals on general and complete disarmament now before the Conference.

1. *Calls for* strict observance by all States of the principles and objectives of the Protocol . . . signed at Geneva on 17 June 1925, and condemns all action contrary to those objectives;

2. *Invites* all States to accede to the Geneva Protocol of 17 June 1925.

The resolutions of the General Assembly are normally not binding as such on member states, but resolutions on legal questions provide clear evidence of the state of the law, since the resolutions represent the deliberate and collective view of those states giving affirmative votes. This resolution is expressly concerned with legal issues and was adopted by 101 votes. No state voted against, but three—Cuba, France, and Gabon—abstained.

The significant features of the resolution are as follows. First, it assumes that the Geneva Protocol today constitutes general international law and no longer a mere contract for actual parties to it. Secondly, this assumption extends both to chemical and bacteriological warfare. Thirdly, the prohibition seems to be of these types of warfare *tout court* because the weapons concerned are weapons of mass destruction. On this third point it might be argued that the reference to weapons of mass destruction cuts down the effect of the resolution because not all chemical or bacteriological weapons are necessarily of this type. However, the intention of the resolution is clearly to deal with these weapons as types; hence the focus on, and emphatic reference to, the Geneva Protocol. In general the resolution clearly militates against the former apparent reservations in the position of the United States. The United States took part in the process of amending the original proposal and voted for the resulting text.

The question of conditional legality

The materials examined so far relate primarily to the issue of prohibition unconditionally of the classes of chemical and bacteriological weapons. It is important to tackle the problems from a different point of view for a number of reasons: the United States might renew its stand against prohibition *tout court*; the observer, and especially the non-lawyer, may feel that the alternative to categorical prohibition is that CBW is *lawful* without qualification. This cannot be so on any view of the legal position.

Insofar as particular chemical and biological weapons have indiscriminate effects, or cause unnecessary suffering, they can only have conditional legality. The *U.S. Army Manual* necessarily adopts the principle of conditional legality, since any more permissive principle would be utterly untenable. Paragraph 36 of the *Manual* thus provides:

> The use of weapons which employ fire, such as tracer ammunition, flame throwers, or napalm, and other incendiary agents, against targets requiring their use is not violative of international law. They should not, however, be employed in such a way as to cause unnecessary suffering to individuals.

Although the *U.S. Army Manual* is equivocal in dealing with both chemical and bacteriological weapons, the general implication is that in its regime a conditional legality exists as in the case of fire weapons.

The position of conditional legality must be given some elaboration. It is an uncontestable principle of customary law that it is forbidden to use arms, projectiles, or material calculated to cause unnecessary suffering.[14] Moreover, numerous rules of customary law, repeated and developed in the Geneva Conventions of 1949, would be inevitably broken if weapons are used which produce extensive, indiscriminate, and possibly unknown and uncontrollable results. Two Conventions in particular are involved here: the Geneva Convention for the Amelioration of the Condition of the Wounded and Sick in Armed Forces in the Field,[15] and the Geneva Convention Relative to the Protection of Civilian Persons in Time of War.[16] The indiscriminate character of certain types, at least, of chemical and biological techniques needs stressing. Moreover, the elements of persistence and consequential action may aggravate the problem.

Assessment of the legal position

It is not very useful to examine the various forms of special pleading to be found in some of the literature in favour of freedom of action and choice of weapons. The fact is that the vast majority of writers in various languages take the firm view that both chemical and bacteriological weapons are prohibited and that the Geneva Protocol now represents general or customary international law.[17] The current United Kingdom *Manual of Military Law* contains a careful codification of *The Law of War on Land* prepared with the assistance of Sir Hersch Lauterpacht, later the British judge on the International Court of Justice. Paragraph 111 of *The Law of War on Land* provides that "the use of poison and poisoned weapons, asphyxiating, poisonous or other gases, and of all analogous liquids, materials or devices, and bacteriological methods of warfare are forbidden." This view of the law receives recent and very substantial reinforcement from the General Assembly resolution of 1966. In the debate on that resolution the United States reacted to charges relating to the conduct of the war in Vietnam by saying that the only gases it had used were irritant gas, tear gas of the type normally used in riot control.

The United States' position is interesting because it does not lay stress on permissiveness, but pleads that an exception exists to the overall prohibition. The exception does create some difficulty. In all probability the definition in the Protocol was intended to be comprehensive for the *genus* of substances with which it dealt. In November 1930, the United Kingdom presented a memorandum to the Preparatory Committee of the Disarmament Conference which stated that the Protocol definition included non-toxic gases and notably irritant gases.[18] On the other hand, if the *genus* concerned is confined to those weapons which cause unnecessary suffering tear gas may not be included as such, and everything will depend on the type and the way it is used. It may be noticed that the U.S. Naval War College Study of 1935 states that the prohibition does not include smoke-screens and tear gas.

A more serious threat to the legal regime comes from the doctrine of reprisal. Several parties to the Geneva Protocol, including the United Kingdom and the USSR, made a reservation of reciprocity, and the customary law contains the principle that reprisals may be employed in reaction to a prior violation of the law committed by the enemy. However, the doctrine of reprisals is limited in that a reprisal must not be in excess of the degree of violation com-

mitted by the enemy and reprisals directed against wounded and sick prisoners-of-war and civilians, protected under the Geneva Convention of 1949, are unlawful and constitute war crimes. A further consideration is the possible effect of the strong terms of the General Assembly resolution: the consequences may be that the rules in the Protocol are now *jus cogens* and not subject to the doctrine of reprisals and also that reservations on the same theme are void.[19]

Some other relevant legal principles

The use on a substantial scale of weapons which cause indiscriminate harm to combatants and non-combatants and which in any case cause unnecessary suffering will involve the commission of crimes against humanity. Crimes against humanity were declared to be crimes under international law in the Agreement on Military Trials of 1945,[20] which provided for the establishment of an International Military Tribunal at Nuremberg. The definition contained therein refers, *inter alia*, to "murder, extermination, enslavement, deportation or other inhumane acts committed against any civilian population." The Agreement on Military Trials had twenty-three parties, and a unanimous resolution of the United Nations General Assembly affirmed "the principles of international law recognized by the Charter of the Nuremberg Tribunal and the Judgment of the Tribunal."[21]

Depending on the effects of the particular weapon and the conditions in which it is used, to resort to chemical and, more especially, biological warfare might constitute a breach of the Genocide Convention.[22] However, the intention in such a case must be "to destroy, in whole or in part, a national, ethnic, racial, or religious group, *as such* . . ."[23] Finally, if large scale and reckless use of weapons and techniques produced effects which spread over frontiers the state using weapons in this way would be in breach of the duties toward neutral states which the customary law imposes on belligerents. Article 1 of the Hague Convention of 1907, concerning the rights and duties of neutrality, provides that "the territory of neutral Powers is inviolable."

Defoliation and crop destruction

The practice of aerial spraying for purposes of defoliation and crop destruction was clearly not foreseen by the various legal controls relating to the conduct of war. Moreover, the rationale behind

the existing rules is the causing of indiscriminate and unnecessary suffering to people by direct means. Nevertheless, practices which must result in depriving peasant communities permanently of their food resources constitute a crime against humanity and if persisted in, when large-scale distress is manifest, would amount to genocide. Large-scale destruction of the fertility of a countryside is an operation which is probably more strikingly indiscriminate as between combatants and non-combatants than any technique other than resort to nuclear weapons. A 'scorched-earth' policy which is carried out in circumstances in which specific material resources are kept out of enemy hands and which is accompanied by a taking of responsibility for the subsistence of civilians from the area concerned is not necessarily illegal. Of course, the normal scorched-earth technique is not aimed at the ecology of a region in any case. A further point is that pillage of captured places and unnecessary destruction of property are forbidden by rules of undisputed authority. Large-scale crop destruction must fall foul of these rules, more especially when it is carried out from the air.

Incendiary weapons

The classification of weapons has been a matter of conventional and historical association. Neither the legal definition nor, it seems fairly clear, the scientists include fuels and incendiary weapons in the category of chemical weapons. As Dr Sidel points out in his paper on napalm,[24] explosives and incendiary weapons are 'physical' weapons, producing their effects by blast and heat. As a class it is doubtful if such weapons are prohibited since they do not appear to fall within the terms of the Geneva Protocol.[25] Nevertheless, the normal rules of the law apply: napalm and similar weapons can be used lawfully only against military targets and "their use against personnel is contrary to the law in so far as it is calculated to cause unnecessary suffering."[26]

Control of armaments and disarmament

It is well known that, in spite of adherence to rules limiting or prohibiting the use of certain types of weapons, states may manufacture and/or, possess the weapons concerned. Disarmament is the better form of insurance therefore. Recent proposals for disarmament have shown particular concern for the prohibition of the production, possession and use of chemical and bacteriological weapons. This concern is perhaps an index of the growing role of

such weapons in military preparations. In this connection it is worth recalling an existing treaty provision in Protocol III of 1954 to the Brussels Treaty of 1948.[27] Article One of the Protocol stipulates that the Federal Republic of Germany shall not manufacture in its territory atomic, biological, and chemical weapons. In Annex II the following definitions occur. A chemical weapon is defined as "any equipment or apparatus expressly designed to use, for military purposes, the asphyxiating, toxic, irritant, paralysant, growth-regulating, anti-lubricating or catalysing properties of any chemical substance." A biological weapon is defined as "any equipment or apparatus expressly designed for use, for military purposes, harmful insects or other living or dead organisms, or their toxic products."

Concluding remarks

It will suffice to make certain general points.

(i) If the Geneva Protocol is regarded as declaratory of the customary law only in respect of chemical warfare, then the distinction between chemical and bacteriological warfare may matter since the prohibition of the latter type may be less firm. The chemical-biological distinction is difficult to maintain in some ways at least. Thus the existence of non-biological poisons, the low molecular weight toxins, blurs the line with chemical warfare. However, the general practice of states and the evidence provided by the United Nations resolution of 1966 justifies the conclusion that both chemical and biological warfare are prohibited by general, *i.e.*, customary, international law.

(ii) The prohibition existing on the basis of the Geneva Protocol and the United Nations resolution is clearly not restricted to lethal gases. It almost certainly includes all anti-personnel gases.

(iii) Even if the Geneva Protocol was regarded as in some way defective, and even if it were to be denounced by some of the parties to it, it cannot be emphasized too strongly that the ordinary laws of warfare still apply. The layman, who knows about the Protocol but is ignorant of the law of war in general, is ready to assume that everything turns on the Protocol. The overriding principle, which applies to all weapons, is that weapons should not be used which are calculated to cause unnecessary suffering or are inevitably indiscriminate as between combatants and others. These principles may be found in any statement of the law of land warfare, including the British *Manual of Military Law*, Part II.

DISCUSSION

Discussion of Dr Brownlie's paper ranged extensively over many of the issues he raised; two particular topics may perhaps be singled out, the legality of the U.S. use of chemical weapons and defoliants in Vietnam, and the general question of whether the activities of international lawyers, in building a legal framework in which war could be conducted, helped reduce its risk.

On the first point, the use of the weapons in Vietnam, Dr Brownlie reiterated that attempts to discriminate between lethal and non-lethal weapons failed; the definition of chemical weapons was concerned not so much with lethality but with the concept of "asphyxiating, noxious or causing unnecessary suffering".

Dr Galston asked what the position was regarding the use of food deprivation as a weapon, and if the deliberate destruction of food, either by the dumping of captured bags of rice or by the destruction of rice plants in the field, were challenged and the United States asked to supply a basis for its actions, what likely legal basis would be used.

In reply Dr Brownlie pointed out that there is no legal provision on scorched earth policy as such. The *British Manual*, for example, which is quite detailed, not only in its actual provisions but in the comments on provisions, where it suggests examples of the scope of particular provisions, says that the poisoning of wells is illegal but the diversion of watercourses is allowed.

It comes back to the general principle that one cannot use means of conducting a war which cause unnecessary and indiscriminate harm to the civilian population. It thus becomes a question of how this general principle is applied to the particular facts. And this is a problem no matter what area of war is discussed. If a war zone is declared and treated without the slightest attempt to sort things out, then it would be very difficult to find a legal basis to say that this behaviour came within the accepted rules. On the other hand if a given war zone, before declaring it a war zone, is claimed to be occupied by foreign forces and the civilians were behind the lines somewhere (where one has lines), then there are no legal risks at all; one just treats everything as an open target.

Mr M. Leitenberg pointed out that the *U.S. Field Manual* (USFM 27.10.1949 para. 24 and 1956 para. 40, 41) was very precise on this point. Thus it states that destruction of food crops and food supplies is prohibited unless it can be shown that these are for the use of enemy combatant personnel. The United States has said repeatedly (i) that intended victims of this campaign are male Viet

Cong combatant personnel who are in isolation from the community and (ii) that to make sure that there is no unnecessary suffering caused to civilians they are warned in advance and told where they can go. Now, while this may not be the case in practice, there remains a very interesting connection between the U.S. law of warfare and what the U.S. Government claims to be doing. But according to Donald Hornig, President Johnson's Science Advisor, the real purpose of the anti-crop programme has been directed at moving the population out of NLF-controlled areas into those controlled by the Saigon regime. This puts quite a different perspective on the programme and is borne out by a survey of defoliated areas. Crop destruction has been most marked not in the sparsely-occupied areas where the effect would be largely confined to the NLF guerillas but to the densely-populated, fertile Mekong Delta —the 'Rice Bowl of South East Asia'.

Thus, as Dr Brownlie concluded, it all depends on the evidence on the particular case concerned. In the Vietnamese case, the U.S. policy of declaring war zones and then treating everything in the zone as a free target might be quite good evidence on this particular point.

The second theme was raised by Professor Arnott, who asked whether Dr Brownlie felt that the body of international law relating to warfare produces any inhibitory effect at all on the behaviour of governments when it suits them otherwise. Those who were engaged in the early battles to contain nuclear weapons, claimed Professor Arnott, do not believe that the fact that they haven't been used is in any way due to any international agreements, test bans, or whatever, but because so much is known about these weapons that they are politically impossible; this is the sanction which prevents their use. The same would be true for CBW as well.

Dr Brownlie replied to this challenge that his answer could not be his opinion as a lawyer, because this was a matter of politics and sociology. There was a difference between talking about rules and their efficacy. If one brought a criminal lawyer into the discussion, he would talk about criminal law, but he would not have said the law is probably not worth talking about because only ten per cent of warehouse break-ins in London ever come to conviction. Rules of themselves could not dictate in matters of high policy, but could have some sort of moral imperative which affect governments in this sphere. The problem of enforcement is a complicated one—the rules themselves are never self-enforced—they cannot be, and the rules of warfare present more of a problem than there would be in some other areas of law. But one of the ways out is to remind governments publicly that they have in fact said these things and signed these treaties.

One of the problems is the sociological question of why law is observed at all. Governments are not individuals—they are like rather vast and complex corporations. Within a government one can have different views on crucial matters of international law. In times past the Admiralty has had one view and another sector

has another view. In the United States, on some leading questions of international law the State Department has a different view from the Department of Justice—on sovereign immunity, for instance, immunity of foreign states and their agencies. So if one is talking about states, one must remember they are not individual beings. The problems are different from those of making internal laws. On the world scene there is a newer type of community, a definite sort of community consisting of 130 large corporations which can behave as very complex units. Thus despite its inadequacies, the codification of international law remains a powerful tool.

PART FIVE

ETHICAL PROBLEMS

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13. Preventing CBW (a) J. H. Humphrey

In discussing what might be done about the danger that chemical and biological weapons may become accepted as part of the normal armoury of nations, it will be more useful if we consider them from the point of view of those who regard it as their business to prepare for wars as well as from that of those who regard resort to war as an unnecessary and unacceptable way of settling differences between nations. So long as national governments, and those who maintain them in office, continue to prepare for war rather than repudiate it, arguments about whether one way of killing people or of putting them out of action is more beastly than another are liable to appear academic and to be ineffective.

While I accept that to take part in the development and use of chemical and biological weapons poses ethical problems for the scientists involved and that to take part in 'public health in reverse' is indeed directly contrary to a physician's role, the ethical problems seems to me to be personal and not essentially different from those which face a physicist or engineer working on ballistic missiles or a bomb sight. Further, I doubt whether to those at the receiving end an attack with chemical or biological weapons could be much more unpleasant than one with napalm or saturation bombing with anti-personnel missiles. Once the weapons are such that the person who uses them is not made aware of the direct consequences of his actions, and especially if he has been taught to regard his enemies as creatures whose lives are incomparably less important than his own, he can escape such emotions as pity, disgust, or horror which might make him hesitate to use the weapons except perhaps in the heat of battle. To me and, I suspect, to military planners it seems illogical to set chemical and biological weapons in a category apart from other modern weapons on grounds of beastliness alone. The question is, are there other grounds on which these weapons can be distinguished? I think there are.

The first and most important, as explained by Dr Brownlie, is that the people in all countries and the politicians in their public

pronouncements recognize such a distinction—even though it may be illogical and based on an accident of propaganda and history. The terms of the Geneva Protocol, publicly supported at the United Nations in December 1966, by the representatives of all the major powers including the United States and Japan, forbid the first use of chemical and biological weapons in warfare, although they do not forbid research on or possession of such weapons. Despite its obscurities and the tenuousness of any guarantees of observance the Geneva Protocol provides a statement of good intentions which it has not been possible to achieve in respect of any other weapons.

The second is that investment in CBW, although by no means inconsiderable, is still small compared with investment in conventional and nuclear weapons. At present, so far as one can tell, CBW is regarded rather as a useful adjunct to the existing armoury, although the situation might change if both wars and the nuclear stalemate continue. Some of the advantages claimed for weapons of this kind have been set out in General Rothschild's book *Tomorrow's Weapons*, and include cheapness, minimal destruction of property and fixed installations, flexibility (including the capacity to reach persons protected against all but very powerful conventional weapons), and, in a strange way, humaneness, since the non-lethal weapons may incapacitate the enemy sufficiently to destroy his fighting power temporarily. Although lethal chemical and biological weapons could conceivably be used against more or less specific targets, they are nevertheless essentially indiscriminate in their action and are classified as weapons of mass destruction. The status of non-lethal chemical and non-lethal biological weapons (if the latter are under serious consideration) has been left blurred, and it is arguable whether they should be defined as weapons of mass destruction. These are the weapons which are put forward by their protagonists as being humane. Since few people—at least in peacetime—relish the idea of killing, the humaneness claimed for CBW could lead to its being regarded as acceptable, despite the Geneva Protocol. I find it hard to understand—and this doubt is confirmed by the example of the use of non-lethal chemical weapons in Vietnam—how non-lethal weapons can deter or defeat a determined enemy, except when used as a means of making conventional weapons more effective. This is a matter which requires careful discussion and hard thinking.

The third difference is that much of the research which goes into the development of chemical and biological weapons, or into defence against them, is of a very similar nature to research which

would legitimately be pursued without any consideration of CBW. For example, the toxic properties of the organo-phosphorus insecticides need to be studied for the sake of the persons who make and use them; similarly important is work aimed to discover antidotes and to devise adequate protective clothing. The defoliants used in Vietnam are standard agricultural chemicals, and spraying them from aeroplanes to defoliate trees—rash though this may appear to ecologists—has already been done in Africa in attempts to clear the tsetse fly, vector of trypanosomiasis, from parts of the 'tsetse belt.' What sets apart work on chemical weapons is the deliberate and secret investigation of properties which increase their toxicity for man, the preparation and manufacture of devices for use in war, and the stockpiling of chemicals for this purpose. Similarly the development of biological weapons requires research into factors controlling the pathogenicity of microbes and their mode of dissemination; study of techniques for rapid detection and identification; large-scale culture methods; preparation of protective vaccines; and so on. Nearly all the research required could quite properly be undertaken as non-military projects in universities, medical research institutes, or enterprises engaged in the manufacture of vaccines. Even intensive investigations on uncommon and exotic highly virulent microbes could be entirely justifiable in a wholly peaceful world, both on scientific grounds and because of the possibility that some time, somewhere, such microbes might become disseminated and give rise to serious epidemics such as have repeatedly ravaged man and other animals in the past. As with chemical weapons, microbiological research and development become biological weapon research only insofar as techniques are devised for deliberately spreading pathogenic infective agents in war.

A fourth feature of chemical and biological weapons is that their manufacture does not require elaborate or highly specialized installations which would be difficult to conceal. Indeed the potential activity per unit weight of these weapons, especially of biological weapons, is so great that quite small factories or parts of factories could suffice to supply the military requirements. Apart from the need for precautions essential for the handling of highly dangerous materials nothing obvious need distinguish such factories from others making ordinary fine chemicals or, say, antibiotics or bacterial vaccines. Consequently, inspection and verification to ascertain whether the ingredients of such weapons were being made would present much greater difficulty than in the case, for example, of fissile material for nuclear weapons.

With these four general points in mind, it seems to me that there are a number of ways in which the further development of chemical and biological weapons might be halted and the danger of their further use in war diminished.* The first is by ensuring that the support publicly given to the Geneva Protocol by almost all the national delegations at the General Assembly of the United Nations in December, 1966, is strengthened and not eroded. This means that the issues involved must be clearly discussed and widely enough understood to put political pressure on the governments of those countries which have not ratified the Protocol to do so. Without minimizing the unpleasantness of CBW or the ethical objections to weapons of this kind, I think that the most valid reason for doing this is that the Geneva Protocol, if not a giant confidence trick, is a real beginning in that limitation of weapons which all countries in the United Nations have declared they wish to seek. Until the difficulties presented by inspection and verification have been got round it may be difficult to extend the Protocol to forbid research on or possession of chemical and biological weapons, although states could be encouraged to renounce these for themselves, as Austria has done.

But even if all countries had adhered to the Geneva Protocol allegations of infringement in times of tension or war might still be made, and would require rapid investigation, since the right of retaliation in kind would remain. There is need for a recognized organization prepared to do this. It might be the International Red Cross Organization, or perhaps the World Health Organization because of its accepted expertise in epidemiology and its non-partisan reputation.

All this would minimize the danger of the future use of CBW, but it would not prevent countries from researching on and preparing such weapons as a precautionary measure to permit retaliation. The next and more important goal is for all countries to renounce the use of CBW altogether. This would imply that no secret research, by government or industry, could be done on potential weapon agents, and that open research on them in scale and content must have a clearly non-military aim. Renunciation of CBW could be made soon—indeed it might become much more difficult later—because no country has yet made such a major in-

* Many of the issues mentioned here have been raised under the stimulus of discussions held under the auspices of the Pugwash Movement.

vestment in these weapons as to feel its security threatened by renouncing them as part of a general agreement. It would be a gesture of mutual confidence which could lead to other and more important steps towards disarmament in more sensitive areas.

Finally, what should be the future of the establishments which have been built up to study and prepare for national defence (and, to a greater or lesser degree, offence) in relation to CBW? It is important to consider this question, since on the one hand they are likely to have a powerful vested interest in their own continued existence, with the possibility of arguing for this at the top level, and on the other hand they represent a sword which could be changed, with a minimum of beating, into a magnificent ploughshare. I will consider our own establishments, the MRE and the CDEE at Porton Down. I will start with the assumption that their own public image of themselves is broadly correct. That is, that their function is to study potential offensive agents only in relation to devising means of defence against them; that the great bulk of the original scientific work done there is published; and that most of the scientists take jobs there not only to earn a reasonable salary, but because they are given excellent facilities to study scientifically interesting problems and because they have a genuine belief that their work is important for the military defence of the country. Furthermore, I know that they are willing to help other civilian institutions with advice and the provision of materials and facilities. Nevertheless, in accepting this image I think it necessary to make two reservations. The first is that the study of defensive measures may not be so innocent as it might appear if studies of offensive measures and discoveries relating to these are left to our American allies on a reciprocal basis. In the absence of other information it may be wiser to assume that the collaboration which began during World War II has not ended.

The existence of an agreement between Britain, Canada, Australia, and the United States, and of shared proving grounds for CBW research at Suffield, Alberta, and Innisfail, Queensland, are examples. It is important to know what are the general terms and duration of this quadripartite agreement. The second reservation is that the output of published work from Porton would appear somewhat meagre if it represented the total useful output from an ordinary research institute with a comparable staff and facilities, and this is bound to give the impression that more goes on there than is revealed. Despite these reservations, the fact remains that at Porton are collected teams much of whose work has a direct relation to the problems of

civilian life, but has much less impact on these than it might do if the primary object of the establishments were not to undertake partly classified work for the Ministry of Defence.

I do not of course, know, what classified work goes on at Porton nor what is the purpose of keeping it secret. Let us suppose, hypothetically, that significant defensive advances have been made, such as the development of a very effective general purpose gas-mask or of a rapid and extremely sensitive method for detecting airborne microbes. Would the revelation of such developments diminish their defensive value? Or might not this actually be increased, by showing that it would be difficult for one country to steal a march on another in this field? The answers do not seem to me to be obviously 'yes' and 'no' respectively.

It would be unrealistic to expect that the Ministry of Defence could abolish its establishments at Porton out of hand, or even declassify all the past work which has been done there, unless other countries known to be interested in CBW such as the United States and the USSR were prepared to do likewise. However this does not mean that no initiative can be taken. Provided that our Government were not contemplating becoming engaged in a war within the next few years involving CBW—despite the Geneva Protocol—and especially if it were meanwhile prepared to make a real effort at the international level to reach agreement to renounce these weapons, I suggest that for a trial period of not less than five years all the future work carried out at Porton should be declared declassified. Because, I suspect, biological weapons are presently less important than chemical weapons it might be easier to declassify the MRE first. The CDEE might follow suit later, after allowing time to observe whether there was a favourable international reaction to the first initiative, and time to sort out possible complications about chemical weapons shared with our allies. Work on the defence of man and animals against highly virulent and pathogenic organisms or work on toxic chemicals could proceed much as at present and might well be more effective because collaboration with other workers would be easier and more natural. Of course the results would not be the exclusive property of the Ministry of Defence, but there would be nothing to prevent the armed forces from obtaining advice and making use of them as they wished. Even during the trial period I would like to see at least a part of MRE positively devoted to international collaboration, for example with the World Health Organization over problems of mutual interest (of which there are many) or with the

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Food and Agriculture Organization over such projects as the large-scale production of micro-organisms as food additives for regions where dietary protein is inadequate. Similarly part of the Chemical Defence section might act as a Research Association for the by no means economically insignificant protective clothing industry (which at present has none). Collaboration of this sort would not only have the advantage of promoting goodwill but could help towards defraying the cost of keeping Porton going.

To make such a gesture would carry some risk, but it would be an act of leadership by this country which could produce quite disproportionate gains by helping to break the deadlock over disarmament. Furthermore, by making the gesture for an initial trial period, without disbanding the teams which have been built up, even the risk would be minimized.

(b) M. Meselson

Half a century has passed since the world's only major outbreak of poison gas warfare. Large-scale germ warfare has never been attempted. Gas and germ warfare are explicitly prohibited by international law in the Geneva Protocol of 1925. Considering the enormous scale of gas warfare in World War I, it is remarkable how well the Protocol has been respected. There have been only two instances of verified poison gas warfare since 1925—in Ethiopa, and in the Yemen. In Vietnam the United States has been employing a powerful but generally non-lethal anti-riot agent, maintaining that the Protocol does not forbid it.

When compared with the recent history of other forms of warfare the record shows that the governments and peoples of the world have come to practise and expect a degree of restraint against the use of chemical and biological weapons not found for any other class of weapons, except nuclear ones. The chief factor justifying that restraint is the same for both nuclear and CB warfare—apprehension that, once begun, it would open up an unfamiliar and highly unpredictable dimension of warfare that might lead to the extermination of very large numbers of troops and civilians, especially one's own.

Destructiveness of CBW

There is no doubt that existing nuclear weapons could destroy entire populations. Although the performance of chemical and biological weapons in any particular attack would be less predic-

table than that of nuclear weapons, they too have very great potential for mass killing. The most effective method of strategic CBW attack would presumably entail the production by bombers or missiles of a cloud of toxic or infectious material over or upwind from a target, to be inhaled or absorbed through the skin by persons in the attacked population. Although masks, protective suits, and special shelters can provide effective protection against known chemical and biological agents, the cloud would readily penetrate dwellings and other ordinary structures.

An attack by a single bomber, dispensing one of the more deadly nerve gases, could kill most unprotected persons within an area of at least five square miles, this being the size of the zone of high mortality caused by the Hiroshima and Nagasaki atomic bombs. Although nerve gases are among the most poisonous substances known to be suitable for military use, it may well be possible to devise weapons containing far more poisonous materials, perhaps toxins or related substances. Weapons based on such super-poisons might become as destructive to unprotected populations as thermonuclear weapons of equal size.

Poisonous as nerve gases are, virulent micro-organisms and viruses can be a million or more times more so, in terms of the amount that can cause incapacitation or death. Although many infectious agents are rapidly inactivated or lose their virulence when dispersed in the atmosphere, this obstacle to the development of biological weapons can probably be circumvented or overcome with sufficient research effort. If so, biological weapons could surpass thermo-nuclear bombs in terms of the area coverage possible for a weapon of specified size. However, even after very extensive research, the performance of biological weapons is likely to remain subject to great uncertainty. Their effects would depend in large measure on poorly understood and highly variable factors that determine man's resistance to infection. A biological attack intended to be highly lethal might actually kill very few persons, and, conversely, an attack expected only to cause temporary incapacitation could cause high mortality.

Although biological warfare agents might be chosen from among those that are not highly contagious under natural circumstances, this would not preclude the unexpected initiation of a widespread epidemic under the very unnatural conditions inherent in military use. Indeed, it is possible that bacteria or viruses disseminated in an aerosol cloud could subsequently emerge from the exposed population of humans, insects, birds, rodents, or other animals with in-

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creased persistence, contagiousness, and virulence to man. Large-scale operations in regions populated by many persons or animals would be more risky than small operations in desolate places, and viruses might be more hazardous than bacteria. However, we cannot evaluate the risks with any confidence in any of these situations. Therefore, the field testing of live biological weapons and especially the outbreak of actual biological warfare would constitute a menace to the entire human species.

The uncontrollability of CBW

A major uncertainty in predicting or controlling the course of CBW, once it is begun, would arise from the great variety of possible weapons and targets, from the incapacitating to the highly lethal and from the local battlefield to entire continents. Once begun at any level in earnest it would be very difficult to predict how far CBW might go. Distinctions and stopping places would be very difficult to define and to keep. The preparations and training required for one form of CBW would facilitate and therefore tempt escalation to larger scale and more deadly CBW operations. The breakdown of barriers to weapons once regarded as illegal and peculiarly uncivilized can inspire and encourage methods of warfare even more savage than those underway at the time.

The vulnerability of troops or civilians to CBW attack depends very much on the availability and effectiveness of protective facilities, the rigour of defensive training and discipline, and the performance of early-warning systems. All of this may act to place an unusually high premium on surprise or clandestine attack and on the use of novel or unexpected agents or means of dissemination. Once the effect of surprise has worn off and defensive precautions have been instituted, CB warfare might continue on a large scale but with relatively inconclusive effects until new weapons are introduced or until conventions against the attack of previously inviolate targets are transgressed.

The myth of humane CBW

It is well known that some chemicals such as tear gas are able to incapacitate a man for a short time with little risk of killing. Some people have concluded from this that the introduction of non-lethal chemicals and even of biological weapons thought to be non-lethal might actually make war more humane. The argument has shown considerable appeal both for thoughtless zealots who

wish to advance the practice of CBW in any form and also for persons who genuinely hope to make war less savage. Although it is true that some chemical warfare agents are relatively non-lethal in themselves, it seems to me almost certain that their use would definitely not make wars on the whole less savage and would in fact risk making them much more so.

It is naïve to expect that in a real war non-lethal agents would be used by themselves. Once introduced into a combat area, the pressure would be very great to utilize them in any manner that increased the overall effectiveness of general military operations. Non-lethal chemical weapons would be used to increase the effectiveness of lethal ones. Tear gas can reduce the accuracy of enemy rifle fire, allowing one's own forces to approach more closely, increasing the accuracy and intensity of their counterfire. It can be used to force men out of protective cover and into the line of fire or the path of bomb and shell fragments. Under the desperate pressures of a war fought with artillery, bombs, napalm, and other lethal weapons, it is only reasonable to expect that "non-lethal" weapons once introduced will come to be used in order to kill. This has happened in Vietnam where the U.S. forces have spread riot gas over large areas to force persons from protective cover to face attack by fragmentation bombs. It happened in World War I when both sides used tear gas and other non-lethal chemicals in grenades and artillery shells to facilitate conventional infantry and artillery operations. This is not to say that non-lethal weapons would necessarily make war any more savage than it already is. However, it seems unrealistic to expect that they would make it any less so.

In any case, if tear gas or similar agents should prove at all effective when first used both sides would introduce protective devices and tactics, making subsequent use of such agents much less effective. Thus, except perhaps when they are first introduced, non-lethal chemical weapons are unlikely to have much effect except to set the stage for more deadly CBW operations.

The conduct of non-lethal CBW can greatly facilitate preparations and training for the use of lethal chemical and biological agents. When combatants learn to protect themselves against the effects of mild or 'conventional' agents the temptation will be strong and the means will be at hand to experiment with more deadly ones. During the first year of World War I both sides used tear gas and other harassing agents until the Germans introduced lethal chlorine gas. Following that, both sides tested a large number of

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poison gases seeking to find ones that would be decisive in battle. The first attack with poison gas had a devastating effect. The Allied front was broken, and 5000 of the 15,000 gas casualties died. However, even though more effective gases were introduced in great quantity by both sides, advances in defensive preparations prevented gas from being a decisive weapon in World War I. Advocates of "humane" gas warfare often point out that, at least toward the end of World War I, gas produced casualties with proportionately less mortality than did high-explosive weapons. However, this was not because commanders on both sides wished to fight without killing, but rather because the most effective gases then known caused more wounds than deaths. Modern nerve gases are vastly more lethal than the old World War I gases. Can anyone have much confidence that skin-penetrating nerve gas would not have been used in World War I had it become available in 1917? The difficulty of allowing the limited employment of gas without running the risk of bringing the whole chemical and biological arsenal into use has been concisely stated by T. C. Shelling in his book *Arms and Influence* (Yale University Press, 1966).

> 'Some gas' raises complicated questions of how much, where, under what circumstances; 'no gas' is simple and unambiguous. Gas only on military personnel; gas used only by defending forces; gas only when carried by projectile; no gas without warning—a variety of limits is conceivable . . . But there is a simplicity to 'no gas' that makes it almost uniquely a focus for agreement when each side can only conjecture at what alternative rules the other side would propose and when failure at coordination on the first try may spoil the chances for acquiescence in any limits at all.

These principles appear to have been understood by the leaders of both sides in World War II, during which neither lethal nor non-lethal gases were employed. At the outbreak of the War, both sides exchanged assurances that they would observe the Geneva Protocol of 1925. Later, in 1943, President Roosevelt declared:

> Use of such weapons has been outlawed by the general opinion of civilized mankind. This country has not used them and I hope that we never will be compelled to use them. I state categorically that we shall under no circumstances resort to the use of such weapons unless they are first used by our enemies.

Although many rules of war were violated in that conflict, it is fortunate for all sides that the rule against gas was observed. Ger-

many had secretly developed and produced a large quantity of nerve gas. Although the Allies had no weapon of comparable deadliness, they could have produced vast quantities rather soon after becoming aware of its existence. Since the previous restraints against anti-city warfare had already broken down, the introduction of nerve gas in the midst of World War II would almost certainly have caused a death toll vastly greater than it was.

Chemical and biological weapons by their very nature are suited to the attack of large areas; their natural targets are people rather than military equipment; important military personnel can be equipped and trained to use protective devices far more easily than can civilians. For all of these reasons, civilians are the most natural and most vulnerable targets for CBW attack. If the barriers against CBW are broken down, civilians are likely to become its main victims.

Chemical and biological weapons and minor powers

The development and initial production of a new weapon usually requires much greater sophistication and effort than is needed to reproduce a weapon already possessed by another. The accessibility of chemical and biological weapons to smaller powers will depend very much on the CBW programmes of great powers and, for a limited time, on measures to keep the results of those programmes secret. With chemical and biological weapons as with other weapons, great powers will probably lead the way unless they deliberately refrain from doing so.

The chemical compositions of several nerve gases are published in the open literature, and detailed manufacturing procedures could be specified by competent chemists and chemical engineers. Although no thorough cost-analysis has been published, it would appear that a considerable number of smaller nations could produce and integrate nerve gas weapons into their artillery and air forces without great economic strain. Commercial transport aircraft could be modified without great difficulty to drop or spray the gas. No small power is definitely known to produce nerve gas or to have been supplied with it by another, although there have been newspaper reports that Egypt has used a nerve gas on a small scale in the Yemen conflict.

The acquisition of nerve-gas weapons would greatly increase the destructive potential of a small nation's military forces, but it might also greatly reduce its overall security by provoking its neighbours to arm themselves similarly. This they might do by producing the

gas themselves or by demanding it from their great power allies. If nerve gas warfare should ever break out between two small states, the population of one or both could be largely annihilated within a short space of time, and the intense feelings provoked around the world might well ignite a much larger conflict.

The attempt to develop biological weapons of reasonably assured characteristics would require a costly and technically sophisticated effort and an elaborate testing programme. Indeed, only use in war itself would provide the kind of information that responsible military men would require before placing much reliance on a radically new type of weapon. It seems unlikely that a small power would attempt the development of biological weapons except perhaps as a deterrent threat. However, this would be an extremely risky posture for a small power unless large powers had already legitimized the possession and threatening display or use of biological weapons.

Why single out CBW for special prohibitions?

As long as wars continue to be fought with high explosive weapons and napalm, what sense does it make to maintain special constraints on CBW? The question is understandable, but it seems to me that some substantial answers are contained in the remarks above. We realize that special rules are required for nuclear weapons. The distinction between conventional weapons and nuclear ones of any size is a real one, and the importance of maintaining it is generally understood. Chemical and biological weapons share with nuclear ones the attribute of potentially overwhelming destructiveness. Biological weapons could pose a threat to the entire human species. Both chemical and biological weapons place a high premium on clandestine and surprise attack, thus lessening stability. Once developed, chemical and biological weapons can be exceedingly cheap, relatively easy to produce, and quick to proliferate. They would threaten civilians especially. Their use would violate the oldest major arms control treaty still in force.

Finally, there are moral arguments, although each person must evaluate them for himself. It is widely held that the use of chemical and biological weapons would be cowardly and inhuman. Perhaps this is because their use could make mass killing and subjugation technically easier than it already is. Some have said that poison-gas warfare would be more humane than other forms of lethal combat. Perhaps so. But it may also be more acceptable to the onlooker and to those who might consider themselves responsible. Is it not partly

in order to relieve the conscience of society that many civilized countries perform legal executions by means that are considered humane? Indeed, it may be that men inwardly dread the eventual consequences to civilization of introducing methods for the killing or subjugation of large numbers of their fellows easily and 'humanely'.

Preventing the use of chemical and biological weapons

It is important for nations to understand that it is in their long-term interest to prevent the use of chemical and biological weapons. This principle could be understood better and more widely than it is today. Discussions and publications by private persons can help. Studies by competent national and international organizations could command wide attention and respect.

The United Nations might consider forming contingency plans for the investigation of allegations concerning the use of chemical and biological weapons. Some of the political difficulties standing in the way of such investigation might be eased if the machinery were partly set up in advance and if its purpose were to establish whether or not chemical and biological weapons had been used rather than to identify the user. It might be agreed that the request of the alleged victim would be sufficient to initiate an investigation. If this were not acceptable it might be required, instead, that all combatants in a given conflict agree to the holding of any inspection. At least this would place the aggressor in the position of having to oppose an investigation.

Some nations or areas might declare themselves to be free of chemical and biological weapons. The Austrian State Treaty prohibits that country from developing or possessing such weapons. West Germany has given assurances to its allies in the Western European Union that it will not produce chemical and biological weapons, and the Union's Arms Control Agency conducts periodic inspections throughout its European territory to provide assurance that no member is producing these weapons. Obviously, there would be many problems and choices involved in setting up CBW-free areas. However, the possibility should be carefully investigated. A start in this direction has been made by the Pugwash Biological Warfare Study Group which has conducted experimental inspections for the non-production of biological weapons in NATO, Warsaw Pact, and unaligned countries.

As in most arms-control matters, the decisions lie mainly with the super powers, although the initiative may be taken in part by

other nations. Nevertheless, it is vital that the super-powers understand that the use of chemical and biological weapons anywhere in the world would constitute a threat to their interests. Possessing nuclear weapons, they do not need chemical and biological weapons as a major deterrent.

There may be a certain symmetry in the view that biological weapons are needed to deter the possible use of such weapons by an enemy. However, in the nuclear environment, the proposition is devoid of logic. The super-powers have no need for biological weapons, and by producing and displaying them they would only make trouble for themselves in the future.

The Geneva Protocol provides a relatively clear and unique standard to guide both the practice and the expectations of member nations. The Protocol has been ratified by all major powers except Japan and, ironically, the nation which proposed it at Geneva —the United States. Many of the states organized since World War II, including the People's Republic of China and both Republics of Germany, have ratified the Protocol or have agreed to be bound by the ratification of their predecessors. Less than two years ago, in December, 1966, the General Assembly of the United Nations passed without opposition a resolution calling for strict observance of the Geneva Protocol and appealing for universal accession to it. The United States and Japan voted in support of the General Assembly resolution along with 99 other states. It is important to secure the actual ratification of Japan, the United States, and other nations that have not yet ratified the Protocol. Means should be found to make clear that viruses as well as bacteria and non-lethal as well as lethal chemical and biological weapons are meant to be included under its prohibition. But great care must be exercised to make sure that attempts to further clarify the scope of the Protocol do not result in weakening its universal authority.

14. Medical Ethics

V. W. Sidel

Discussion of the role of the physician in relation to biological warfare is complex and difficult. One must consider not only the ethical implications of the various roles which the physician may be called upon to play directly in relation to such weapons, but one must also be concerned in more general terms with the roles of the military and civilian physician.*

It is first of all important to make clear that it is not simply a straw man that is being attacked—that is, it must be made explicit that biological weapons are being developed and produced[4] and that physicians are being asked to contribute to their development. Examples of physician participation are clear. Professor Rosebury wrote of his experiences: "At Detrick a certain delicacy concentrated most of the physicians into principally or primarily defensive operations; the modifiers *principally or primarily* [italics in the original] are needed because military operations can never be exclusively defensive."[1] An editorial in *Military Surgeon*, apparently commenting on nations other than the United States, says: "In certain positions, a military surgeon or civilian doctor might be assigned to jobs other than healing. He is perhaps required to perform or to participate in scientific investigations, the purposes of which are not new remedies for the sick, but more and more power-

* I am indebted to many people in the development of these ideas. Dr Theodor Rosebury, a microbiologist who directed research at Camp Detrick during World War II, has since repeatedly warned of the dangers of biological weapons and has discussed their ethical implications.[1] My colleagues in Physicians for Social Responsibility in Boston, especally Drs Robert Goldwyn, Bernard Lown, Peter Reich, and David Savitz, have contributed greatly to the discussions of these problems. Many of the ideas presented here have been drawn from papers written in collaboration with Dr Goldwyn: one was published in 1966 in the *New England Journal of Medicine*[2] and one will be included in a book on medical ethics to be published later in 1968 in the United States by Little, Brown, & Co.[3]

ful atomic, bacterial and chemical weapons of aggression."[5] At Fort Detrick, the major biological warfare research establishment in the United States, there are currently employed 14 M.D.'s, 120 Ph.D.'s, and hundreds of other scientists and technicians.[4] It is very likely that other physicians are associated with the many secret research projects in academic institutions which are listed in Chapter 10.

Before turning to the specific roles of the military and the civilian physician, the argument that it is unethical for a physician to participate in the development, production, or use of biological weapons must be briefly reviewed.

(1) The dangers to both civilian and military population of biological weapons have already been discussed. In summary, the dangers that have relevance to medical ethics are:

(a) They are likely to be used against non-combatants. Major General Thomas J. Hartford stated in a signed editorial in *Military Medicine*:

> It appears that . . . (biological weapons) would be used primarily for strategic rather than tactical purposes. It would be an excellent means of producing non-effectiveness without causing damage to material things. Thus it is possible that the civilian and not the military population might be the prime target. If an enemy were attacking the New York–New Jersey coastal area he would be more interested in producing disability in the millions of industrial workers than in the few thousand military personnel stationed in the target area. . . .[6]

(b) They are unpredictable. Beyond the immediate consequences there may be ecological, medical, and social results impossible to foresee. A biological agent introduced into a susceptible population may cause disease or mortality on a scale not visualized by the attacker. An example is the measles virus which, although usually relatively benign, can cause a large number of deaths under certain conditions—for example, in an unprotected population such as the Fiji Islanders, of whom 20 to 25 per cent died from measles or its consequences in 1875, or in a population weakened by war such as the women and children in Boer concentration camps in 1900.[7] In contrast to other weapons, biological agents may make the human being not only a victim but also a propagator. An example in another species occurred in May, 1952, in the Department of Eure et Loire in France. One or two rabbits were inoculated with myxomatosis virus; by the end of 1953 the disease had spread through 26 departments in France, and through Holland, Belgium, Germany, and Switzerland, killing between 60 and 90 per cent of the rabbit population.[8]

(c) They are truly 'public health in reverse'.[9] They are designed to undo much of the work that physicians and other medical workers have done to eliminate and control infectious disease. They are, therefore, clearly genocidal, and furthermore may be genocidal in the sense of damaging vast groups of the human race rather than a single people.

(2) There are specific passages in widely respected medical oaths which appear to argue against participation by medical doctors in such research. For example, the Hippocratic oath states: ". . . I will use treatment to help the sick according to my ability and judgment, but never with a view to injury and wrong-doing. Neither will I administer a poison to anybody when asked to do so, nor will I suggest such a course. . . ."[10] The Oath of Geneva, a modernization of the Hippocratic Oath, formulated by the World Medical Association in 1948, states: ". . . I will not permit considerations of religion, nationality, race, party, politics, or social standing to intervene between my duty and my patient; . . . even under threat I will not use my medical knowledge contrary to the laws of humanity."[11] Even more specifically, the Code of Ethics in Wartime of the World Medical Association states: ". . . It is deemed unethical for doctors to weaken the physical and mental strength of a human being without therapeutic justification and to employ scientific knowledge to imperil health or destroy life."[12]

(3) These professional oaths codify a body of professional conduct which has served through the centuries to make the physician better able to perform his healing role. Any member of the profession who weakens the ethical standards of the profession by taking part in activities which contribute to disease, disability, or untimely death only weaken the professional status, and therefore the healing potential, of all physicians.

Turning first to the role of the military physician, the entire role of the physician in the armed forces is an ambiguous one. One role for the military physician was eloquently stated in 1965 by Sir Theodore Fox, former editor of *The Lancet*: "A nation's lawyers must execute its laws even when this means the execution of a human being; a nation's soldiers must kill whomsoever their leader commands; but the doctor—perhaps because of his religious origins—is excused such destruction. Even in war, he may be permitted to believe in human brotherhood—to treat all men without political or racial or religious distinction."[13] And many physicians have indeed distinguished themselves in war by devoted and sometimes heroic service to both friend and foe.

But there is another side to the physician's participation in war or in the preparation for war. *Field Manual 8–10* of the U.S. Army states clearly that "the Army Medical service is a supporting service of the combat elements of the Army primarily concerned with the maintenance of the health and fighting efficiency of the troops." Its mission, the Manual continues, is to "conserve manpower . . . (for) early return to duty" and to "contribute directly to the military effort by providing adequate medical treatment and rapid orderly evacuation for the sick and wounded".[14] Dr Morris Fishbein, the former Executive Secretary of the American Medical Association, presents this side of the military physician's role even more strongly: "Military philosophers say there could never be wars if there were no doctors. No doubt they are right. The anguish and destruction of the human body in war with modern weapons would be impossible to contemplate without the healing ministrations . . . that modern medicine provides."[15]

Within this overall conflict of the healing and the combat-promoting roles of the military physician, there are a number of individual conflicts. Attempts have been made to resolve some of these dilemmas by formal codes of conduct. The best known of these are the Geneva Conventions of 1949 which stipulate that medical personnel are to be considered non-combatants; they are, if captured, to be returned to their own forces as soon as possible; and, in return for this special status, they are forbidden to use arms except to defend themselves and their patients.[16] It is clear that these Conventions perform a useful military function; the special non-combatant status of medical personnel enables them better to carry out their healing role. It would appear to be in the self-interest of the combatants to preserve the clear non-combatant role of the physician.

An example, not directly related to CBW, of the types of ethical problem which can arise is provided by the recent court-martial of Howard W. Levy, M.D., Captain in the Army of the United States. Although other issues were involved, one of the major charges on which Levy was convicted and sentenced to three years hard labour was his refusal to obey an order to train Special Forces troops in specialized techniques. This refusal was based in part on Levy's belief that such men, functioning in the dual capacities of combat soldiers and "medical personnel", would break down the distinction between combatant and non-combatant necessary to effective application of the Geneva Conventions; in training them, he felt, he would produce a net increase in disease, disability, and death. He

also believed that such poorly-trained, unsupervised men might do more medical harm than good and that, since their primary duty was as combat soldiers rather than medical personnel, they might be required to abandon sick and wounded patients or otherwise behave in a manner contrary to medical ethics and/or the Geneva Conventions. Whether Levy was right or wrong in these views is not relevant to our discussion here; what is relevant is that as a military physician he was ordered to do something he felt he could not ethically do as a physician and, upon refusing to do so, he was court-martialled and convicted. The defence that the order was contrary to Levy's view of his medical ethical responsibilities was held irrelevant and was not permitted as a defence at his trial.[17, 18]

The result of the Levy trial forces a re-evaluation of the role of the military physician. Until his conviction it may have been possible for the military physician to hold the view that he was still the arbiter of his own medical ethics; that he might do those things which contributed to health and were therefore ethical, and that he might refuse to do those things which he felt to be professionally unethical. Since the result of the Levy trial, if allowed to stand, suggests that the American military physician is no longer free to make such ethical decisions once he has entered the military service, the American physician may have no choice at all except to refuse to enter military service. This line of reasoning forces us to return to a paragraph written in 1938 by Professor John Ryle, Regius Professor of Physics at the University of Cambridge:

> It is an arresting, if at present a fantastic thought, that the medical profession which is more international than any other, could, if well co-ordinated, of its own initiative put a stop to war, or at least increase its uncertainties, and hamper its aims considerably so as to give pause to the most bellicose of Governments. It is everywhere a recognized and humane principle that prevention should be preferred to cure. By withholding service from the armed forces before and during war, by declining to examine and inoculate recruits, by refusing sanitary advice and the training and command of ambulances, clearing-stations, medical transport, and hospitals, the doctors could so cripple the efficiency of the staff and aggravate the difficulties of campaign and so damage the morale of the troops that war would become almost unthinkable. Action of this kind would also produce a profound effect on the popular imagination. In such refusal of service (if such refusal were decided upon in the last resort) there would be no inhumanity comparable with the inhumanity which medicine at present sanctions and prolongs. But let the dream pass and fantasy make room for facts.[19]

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It would be ironic indeed if the court-martial of Captain Levy should be one of the events which would turn Professor Ryle's fantasy into fact.

The problem of the civilian physician, whose need to be guided by medical ethics is uncomplicated by a concurrent need to obey military orders, is quite different. Again a model may be found in circumstances which were not directly related to CBW, the conduct of German physicians during World War II. Although only a relatively small number of German physicians actually took part in the infamous medical crimes for which the Nazi physicians were convicted at Nuremberg, hundreds of others knew of them. As a 1946 editorial in the journal of the American Medical Association put it:

> Perhaps most serious of all is the failure of German medical organizations and societies to express in any manner their disapproval of these widely known experiments. Physicians have a right to expect that men trained in the traditions of medicine would refuse to participate in any way in such acts of inhumanity and brutality. In some instances the defence has been offered that these experiments were conducted under the highest authority of the German state. That cannot possibly be considered in the slightest an extenuation of the failure of these physicians to act in accordance with the principles and traditions of their profession.[20]

Thus the responsibility of the physician is visualized as far broader than simply refusing to take part in an activity which he finds unethical. He must not only refuse himself to participate but he must actively "express his disapproval". Failure to do so represents complicity. One of the greatest dangers in regard to CBW may simply be the apathy toward this problem of most of the medical profession.

What guidelines should the physician use with regard to his or another physician's, participation in research, development, production, or use of CBW? There seems no doubt of the unethical nature of the physician's participation in development, production, or use of offensive weapons of any type; the fact that chemical or biological weapons are the ones with which the physicians may become engaged and the ones about which he has specialized knowledge gives him a special responsibility not only to refuse to work on but also actively to protest against the development, production, or use of such weapons.

But what about research and development of defensive measures

against chemical or biological agents? 'Defence' has often been given as justification for the work being done by physicians and scientists on such weapons. We have already noted that Dr Rosebury, who has participated in such research, feels that the distinction between offensive and defensive research is ethically and technically blurred. What guide-line should be used by the physician who wishes to work on defence against such or, a much more common problem, wishes to conduct research in fields which might in some unforeseeable way contribute to the development of weapons? It seems to me that the least ambiguous guide-line is that of free access of information. If the physician engages in research which is freely published, whether it be on micro-organisms in general or in defence against biological warfare in particular, he cannot be ethically faulted. It is when he engages in 'defensive' or 'pure' research in *secret* that he cannot be distinguished ethically from those who work on the development or production of weapons.

In summary, it seems to me that the ethical physician must continually ask himself whether what he is doing will lead to a net increase or decrease in disease, disability, and untimely death. For the military physician, it may at one time have been possible to consider his activities as producing a net benefit; I believe that, in the United States at least, recent events have made this view untenable, and the ethical physician may have to refuse to participate at all.

Finally, there appears to me to be no question that for the civilian physician ethical principles can best be protected by a refusal to participate in, and an active protest against, secret research in general and work on biological weapons in particular. As Rosebury has suggested: "Ethical principles are not a luxury; the essence of ethics—concern for the value of man—is indispensable for the survival of medicine as a profession and doubtless also for the survival of mankind as a species."[1]

DISCUSSION

Dr Sidel's paper on medical ethics raised isues that were discussed at length by the Conference.

Professor Mayer raised the point that he, like Dr Sidel, was a

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witness, in fact the opening defence witness, at the Levy trial. A feature of the court-martial was that the United States Army was abiding by the ordinary legal rules of having Captain Levy tried by a jury of his peers, but it considered that his peers were officers of similar rank, and the twelve officers who were sitting on the court-martial were, in fact, infantry officers. There was not a single medical officer on the jury. This is extremely significant because it means that the quality of a medical officer as a physician was not something which was considered relevant in his trial. The testimony on medical ethics, the history of the Geneva Conventions and their significance was, in effect, already discounted by the very choice of the court-martial. The American Medical Association and other medical bodies were extraordinarily indifferent to the fact that a physician could be tried on a question of medical ethics by people who were not themselves physicians and where physicians were not even represented. This suggests an abdication by the medical profession of its responsibility to look into possibilities of medical ethics being involved in a conflict with a civil power.

A second, still broader question raised by Dr Sidel is the need for a better definition of who in fact is a physician. Perhaps the definition of a physician is out of date if it implies that a physician is anybody who is a doctor of medicine or has an equivalent degree. Obviously the Hippocratic oath should apply to them in all situations including those in which they are in effect working on chemical warfare. If on the other hand the definition of a physician is one which involves only those people who are actively engaged in therapy a number of people who are doctors of medicine will not be covered, but at least in the United States many people who are in professions not usually considered as specifically medical, such as clinical psychologists, might well come under that classification. But many researchers on CBW would then be excluded from this ethical debate.

Mr M. Birnstingl was doubtful whether the moral responsibilities of the doctor could be restricted in this way. There is surely a danger of medicine allowing itself to become a tool of the current political ideology, when seen only in terms of the Hippocratic ethical tradition, without considering the wider issues. The Hippocratic tradition is concerned with the doctor's ethical and humanitarian role in relation to a single individual, his patient, and gives him no guidance as to his position regarding, for instance, the war in Vietnam. Modern warfare would be impossible without the co-operation of doctors, and they are able to take part without any violation of their ethics. They withdraw and wait while their fighting friends drop toxic gas or napalm, after which they may help the victims who survive. To a physician trained to prevent suffering, such a role may appear irrational, but it is sanctioned by medical ethics, through its apparent humanitarian function. But the wider situation has been well described by Dr Howard Levy as "Kill, Kill. Cure, Cure." and this is the situation which the doctor's presence supports. A doctor may need to do this kind of thing, but he cannot

shelter behind his humanitarian role. He is always an accomplice to the wider act, and it is his relation to this which he must consider.

When civilian populations are being annihilated and other norms of human conduct disregarded a doctor must decide whether he can continue to subjugate medicine to such a policy. Professor Mayer has pointed out elsewhere that Howard Levy's stand was not solely a matter of medical ethics, although the circumstances of his trial may have forced him into making his defence on these narrow grounds. But Levy was also concerned with moral questions extending far outside his professional code, namely the submission that no doctor has a right to allow himself to be used in what he believes to be a genocidal war. And its corollary, an attempt to prevent medicine from being subjugated to a political purpose and becoming just a cog in a wheel of bad and degraded ideology.

Finally, that the doctor's dilemma was also the scientist's was stressed by Professor Burhop, who pointed out that in practice questions of the social responsibility of scientists could not be separated from assessments of political and moral factors. It was difficult to adopt simple-minded criteria applicable in all circumstances. During World War II he, like other scientific colleagues who had earlier been active in the movement against war and fascism, saw no contradiction in working on the development of the first nuclear weapon. It seemed clear that the development of a nuclear weapon by German scientists was entirely feasible and that, possessing such a weapon, Hitler would have had no compunction about holding the whole world to ransom in order to achieve his aims.

Similarly, today, it should not be difficult for an American scientist, mindful of his social responsibilities, to decide that to give freely of his knowledge and talents to assist his Government to subjugate the people of Vietnam, whether by the development of CBW or by the improvement of napalm or anti-personnel weapons, was evil and reprehensible.

Unfortunately, the situations one usually has to face are less clear-cut. For example, what attitude should a university adopt toward the question of accepting funds from defence agencies for open peace-time research in basic science? Perhaps the question of whether the research is open or secret could provide a useful criteria. Scientists should not participate in secret work, whether in university or government institutions. Secrecy is far more important for the development of new offensive than defensive weapons.

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15. Scientific Responsibility C. F. Powell

By way of an introduction to this theme, I want to comment in general terms on the place and responsibility of scientists in our present situation. I have no technical competence in CBW but I believe that this issue and the problems raised in the earlier chapters should be seen as only a part of the general problem of avoiding a catastrophe for our whole civilization. What is involved here is not only the future of humanity but also matters of great importance for the future of science itself—for its moral standing and effectiveness. It is possible, even if we avoid a general war, for scientific research to decline, and this research is our indispensable instrument for overcoming the grim prospect of widespread hunger in the next twenty years to which Bernal has quite recently again directed our urgent attention.

We were brought up in a situation in which science enjoyed immense respect and prestige. I believe this derived from the aims and methods which characterized the development of science under the stimulus of the Baconian philosophy and the work of those in Europe who thought like him—Leonardo, Galileo, Descartes. The new method was to replace the appeal to ancient authorities by reliance upon the observation of nature and by acting upon it, by experiment. And the new aim was to be the improvement of the human condition. It was Bacon's passionate conviction that the discovery of printing, the compass needle, and gunpowder gave only a faint intimation of what was becoming possible for mankind if the search for fruitful knowledge were pursued in a systematic and organized way.

Contrary to those who sometimes assert that Bacon was naïvely optimistic about the advantages which would follow from the progress of discovery, he well understood that the advancement of knowledge could be put to good or evil purpose: speaking of the fruits of mechanical invention he said: "Out of that same fountain come instruments of lust and instruments of death. For, not to speak of the arts of procurers, the most subtle poisons, as well as

cannon and other instruments of destruction are the fruits of mechanical invention, and well we know how far in cruelty they do exceed the Minotaur himself". There could hardly be a better passage with which to illustrate the theme of this book.

Bacon was also clear about a point that is of great contemporary importance; that the advancement of knowledge was not to be judged only on the basis of its immediate utility. In the fable of Atalanta he makes the point very clearly. Atalanta lost the race because she stooped down to pick up the golden apple. She is typical of that short-sighted mechanical ingenuity which fails to see that the true aim is the widening of our basic understanding, so that useful applications may flow "not singly, but in knots and clusters".

It is unnecessary for me to stress the immense success of the developments which Bacon foreshadowed. The whole material basis of our civilization has been transformed, and surely it is a process to which there is no limit. This is not always firmly understood. It is easy to acknowledge that our whole society has been transformed by the advancement of science without really understanding that, if we don't destroy ourselves, the whole world in a hundred years' time will be as different from the present as our own times are from the world of 1800. Of course material prosperity is not the only thing for securing a happy life. But it certainly shouldn't be underrated. At least in the developed countries, technology has immensely improved the human condition; the most severe critics would hardly deny that the three-fold increase in the expectation of life in the United Kingdom is a good thing. What could be more important than an increase in good health or of the life-span?

But our powers have now reached the point where we are presented with the familiar dilemma where we have on the one hand the possibility of an indefinite extension of human well-being and, on the other, the growing danger of a fearful catastrophe. A number of clear responsibilities rest upon scientists in this situation, and one of the bright features of our time is the growing disposition of scientists to assume them.

The first is that they should take an active part in public affairs, especially in fields where they have special technical competence. At least in some scientific circles this idea has not, in the past, always been welcome. I can remember eminent colleagues of mine, less than twenty years ago, most vigorously asserting, and taking some pleasure in doing so, that their work had no relevance to prac-

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tical affairs. Of course, we have to make a clear distinction between the manifold aims which animate scientists in their work and why great public resources should be applied to their support. It is very dangerous to take too narrow a view here in formulating public policy. It is possible for a scientist to be passionately interested in his work and to be quite indifferent to the practical consequences which flow from it. But his indifference is not a reason for not supporting him if his work contributes to the advancement of science, or to the intellectual quality of the work and teaching of the institute in which he is employed, or to practical benefits in the short or the long term, or to all of these ends.

But the idea still persists in some circles that fundamental science merely provides a pleasant diversion, an agreeable life, for scientists, without any real advantage to society. An engineer from one of our great steel companies recently asserted to me, for example, that fundamental science had never contributed anything substantial to the advancement of industry. He was somewhat taken aback when I asked him if he attached any importance for industry to the discovery of electricity. Of course this was rather an extreme example, but we can find undercurrents in the same direction in many places.

Even among scientists scholastic attitudes still linger on, in the sense that there is an indifference to the practical advantages flowing from scientific discovery, and this indifference ought to be corrected by the close involvement of scientists in everything touching the consequences of their work. But the advantage of their involvement in public affairs does not depend only on their technical competence. They are the representatives of severe disciplines. They are used to challenging the basic premises of their subject, and it is not only in science that this process is of fundamental importance for the formulation of wise policies leading to successful action. It is easy for us to laugh at the flat-earth theories of the Middle Ages and to fall into the same kind of error in relation to the imaginative leap required to resolve the profound problems which we face in our sciences and our societies.

It is in the nature of the human condition that we find it difficult to escape from the limitation of our commonly accepted premises. We establish principles which are valid for the particular range of experience with which we are familiar in our local situation in history, in time, and in environment. And their success in allowing us to take successful action in our particular circumstances encourages us to assume for them a universal, eternal validity, until painful experience overthrows our pride. This process of rejecting

cherished assumptions in the face of a wider experience, although often painful, is familiar to scientists, and they can help with the resolution of some of the great problems of public affairs to which, with their expertise, they seriously apply themselves, through the critical and creative approach to problems which science encourages.

It is in such issues that it seems to me scientists have the responsibility to speak out, not least in order to ensure the continuing prestige of their calling. I remember a discussion with Joliot* in which he pointed out that it is an illusion to think that in order to influence people and opinion it is sufficient for a man to tell the truth as he sees it. An element of art is involved here. One can be so far ahead of one's audience as to be out of sight, so that one becomes isolated and ineffective. But to give any kind of leadership in the formation of opinion one has to be ahead and not dragging behind the march of events. And surely our standing is increased when through the march of events the soundness of the position we have taken early is demonstrated.

I have said that the public involvement of scientists is essential for the maintenance of the morale of science. Scientists too would be flat-earthists if they thought that they could count automatically upon the continuing esteem they have enjoyed in the past while doing nothing in the face of the fearful possibilities which have arisen as the result of their work. In this connection they ought to be deeply concerned with the attitudes of young people, for example, in Western countries. What do they see? They may know of the aspirations of the founders of modern science, but what is the present reality? They see not only the fearful perversions of modern science that we have been discussing, but they may reasonably be anxious that the balance between the good and the evil seems to be rapidly changing. Of all our resources on a world scale, about 70 parts in 1000 are devoted to armaments—and much of it today to scientifically based armaments; and 3 parts in 1000 to fundamental science.

They see that, by contrast with the immense resources going into armaments, relatively little is going into the developing countries, most of it ill-considered and therefore ineffective—and quite insufficient in the prevailing political situations in these states to enable them to take any serious steps to ensure the economic and social emancipation of their people. In this situation, the appraisal of science by the young people is such that they begin to prefer what they tend to regard as more innocent and less exacting occupations.

* The French physicist; one of the fathers of atomic science. *Ed.*

The sciences in our time are exacting disciplines and make great demands on young people. With our increasing penetration into nature, the language of science and its basic conceptions and ideas become more and more remote from those of our ordinary lives. Young people welcome challenges to their creative powers; they are prepared to submit to severe disciplines if they believe in their validity and moral standing, but they are not prepared to meet the personal sacrifices involved if they suspect the ends to which their work leads, or if they regard them as evil. I am anxious in that these considerations seems to me to have a bearing on the fact that in the United Kingdom the number of young people entering science is, in a growing population, beginning to decline; and this is at a time when science and its even wider applications is our indispensable resource for the resolution of the profound problems with which we are faced.

Of course, scientists have other responsibilities too, but I have tried to indicate some of the crucial ones. I may add perhaps the important battle for the recognition of the proper role of science, for the increase in its standing. The fact is that it is still true that science in our country is almost outside the common culture. In the organs of government there are many people who are entirely innocent of any acquaintance with science. In a scientific age it is almost impossible to understand fully the role and significance of fundamental science, its potentialities and the problems involved in its proper sustenance and application if one has acquaintance with it only at second-hand. It is for this reason that scientists must become more involved in government, must contribute more effectively to the popularization of science, must help to ensure that our whole culture becomes more and more infused with all that is best in science.

In conclusion, let me add, that throughout his working life, in all these issues Bernal, in whose honour this conference has been held, has been one of our great champions. Without him, not only would this meeting never have taken place, but the whole level of our discussions would have been much poorer, and our possibilities of solving our problems much less. We are all deeply in his debt.

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16. Some Conclusions

Steven Rose

The implications of CBW spread far beyond the issues of lethality versus non-lethality, or even legality versus non-legality. Firstly, any examination of CBW inevitably involves one in a study of its use in what has become the greatest political issue of our day—the war in Vietnam. Most of those who attended the Conference—as indeed most people throughout the world—had strong, though often divergent, views on this issue. That chemical weapons have been used in Vietnam is unquestioned. The scale of this use is outlined by Dr Kahn and Professor Galston in Chapters 5 and 7, and the profound effects on the population by Professor Mayer in Chapter 6. It is perhaps now doubtful whether the use of the weapons has or will alter the course of this war. If it is the case that chemical and biological weapons are seen by the military as in some sense ideally suited to anti-revolutionary, anti-guerilla warfare, in that they are primarily directed against the population as a whole, events in Vietnam are far from demonstrating the success of this strategy. And in terms of scale, conventional weapons, such as napalm, fragmentation bombs, etc., have certainly done more damage to the civilian population than has the—still relatively limited—use of CS or the defoliants. None the less the two issues, CBW and Vietnam, have, for better or worse, become inextricably intertwined.

But CBW research will continue even when the American expeditionary force finally leaves Vietnam to reconstruct itself. The research and development of these weapons has achieved a powerful, perhaps irreversible, momentum. And in this onward course, many hitherto untouched institutions of scientific research have been swept along. Attempts, as Elinor Langer describes, to assess the extent of CBW research in the United States revealed that substantial areas of American university life have become permeated by secret research on CBW and related defence topics. This classified research is now proceeding on an unprecedented scale; a considerable proportion of America's scientific talent is becoming

locked up in activities directly or indirectly of service to the U.S. Department of Defense.

Until recently it had been possible to regard this as a uniquely American problem, but at the beginning of 1968 a series of Parliamentary Questions revealed that in Britain, too, universities were undertaking a significant amount of classified research. No less than thirty-seven classified research projects were let to British universities by way of the Ministry of Technology in the latter half of 1967, and two directly by the Ministry of Defence. The exact fields of these research projects are not known at present, although most probably they are concerned with many topics other than CBW as well.

But these revelations, which have sprung directly out of concern by the scientific community about the extent of CBW research, also change, at least in part, the emphasis of the issues raised.

Classified research in the universities presents a new class of problems; the traditionally open teaching and researching activities of the university are seen to be in danger of becoming undermined, so that various fields of scientific research, ostensibly without military significance, are supported by military funds and shackled by the demands of secrecy and security. The need for secret research may be argued; the case against it being performed in the universities in peace-time, so distorting and warping their educative role, is overwhelming. Here is another theme with which, inevitably, an investigation into CBW becomes interlocked.

The problem of the potentially military significance of basic scientific research raises other issues, too, that need a more thorough investigation. At least since World War II, a substantial proportion of scientific research, in fields as diverse as linguistics and high-energy physics, has been supported by money from a variety of Defence budgets, both in Britain and the United States—and probably many other countries too. This research, unlike the classified research we have so far discussed, has been open and often without any apparent military significance. Yet the chapters by Dr Sidel and Professor Powell inevitably raise the question of how far one should continue to regard this money as manna from heaven. Does the source of such funds—and the fact that the donors perceive a military advantage in obtaining this knowledge, even if its military significance is obscure to the scientist actually performing the experiments—mean that, to adopt Dr Sidel's phrase, "the ethical scientist or physician should have no truck with them"? Such questions begin to open out from the confines of CBW itself to the very heart of

any exploration of the role of scientific research in contemporary society.

To return to CBW again, the third major issue raised is that exemplified in Chapter 13 by Dr Humphrey and Professor Meselson, and expanded in the discussion of the work of Pugwash and SIPRI which have not been included in this published text, of how far it is possible, by techniques of agreed international inspection and control, to limit the development of new weapons systems and the arms race that these imply. The endeavours of the arms controllers have so far met with some partial success in the field of nuclear weapons—the test ban treaty and the non-proliferation agreement are examples. CBW presents unique problems in that the relatively low-grade technologies required for the fabrication of the weapons are within the capacity of many countries, not merely the handful with nuclear capability, but it also presents unique possibilities for the achievement of control, because of both the legal position on the use of the weapons, discussed by Dr Brownlie, and the relatively cautious attitude to their development so far taken by most of the important powers with the possible exception of the United States. And, as Professor Meselson pointed out, the time to control a weapon is before it has been developed and put into the military arsenals.

Here perhaps lies our hope. The enormous repugnance that CBW arouses in most people and its present limited—though future enormous and enormously dangerous—potential, would seem to make both the possibility of and the need for some action imperative. This, certainly, was the message of our conference. We believe that this presentation of the record of our proceedings, by alerting as many as possible to the issues involved, will help in this aim.

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Glossary

This short glossary defines those technical words and phrases that appear in the text but which are *not* defined there. Formulae of chemicals, or names of microbial organisms, which are referred to only *passim* are also not included.

Adamsite. Yellow irritating smoke containing arsenic.

Aerosol. Dispersion of fine droplets of a liquid in the atmosphere.

Anthrax. Malignant fever of sheep or cattle, or of humans infected by them; often fatal.

Antibody. Molecule produced as part of the body's natural defence against infection; it has the property of binding onto the surface of foreign particles or cells.

Asphyxia. Extreme condition resulting from oxygen lack and excess carbon dioxide in the bloodstream.

Atropine. Poisonous crystalline alkaloid which interferes with certain types of nervous stimulation. Under some conditions, in low concentrations, it can serve as an *antidote* to the anticholinesterase nerve gases.

Auxins. Plant hormones responsible for growth and leaf retention.

Avitaminosis. Debilitated condition caused by insufficient vitamin intake—often results from prolonged starvation or undernutrition.

Beri-Beri. Disease of the peripheral nerves due to lack of Vitamin B_1, common in tropical countries. The symptoms are body swelling and pain in the extremities.

Botulism, botulinus toxin. Fatal poisoning of the nervous system caused by the toxic product of a bacteria, clostidium botulinum.

Brucellosis. Bacterial infection which can cause abortion in animals and remittent fever in man.

Ecology. Study of the relationship between organisms and their environment.

ED_{50}. Dose of an agent (generally non-lethal) which is effective against half the population exposed.

Emetic. An agent which induces vomiting.

Enzyme. A biochemical substance (protein) which acts as a catalyst, changing the speed of a chemical reaction without itself being used up in the process.

In vitro. Biological term implying an experiment conducted in an artificial environment ("in glass", *i.e.*, a test tube).

Lachrymator. Substance which causes shedding of tears.

Laterization. An almost irreversible process whereby tropical soil exposed to the sun becomes converted to a hard, brick-like rock.

Lethal Dose. Dose of an agent sufficient to kill.

Lethal $Dose_{(50)}$ or $LD_{(50)}$. Dose of an agent which is lethal against half the population exposed.

Lewisite. Chemical warfare agent with blistering action.

Mg/metre^3. Measurement of the concentration of an agent as milligrams (thousandths of a gram) per cubic metre.

Mg/metre³/min. Measurement of the exposure to an agent; as exposure of a given number of minutes to a given concentration.
Necrosis. Death of a piece of tissue or an organ.
Neuritis. Inflammation of the nerve causing pain.
Non-pathogenic (of a micro-organism). Not causing disease.
Pathogenic (of a micro-organism). Causing disease or death.
Percutaneous. Effected through the skin.
Prophylaxis. Prevention of disease.
Psychotomimetic. Substance whose effects are similar to those of mental illness, *e.g.*, schizophrenia.
Psychotropic. Substance which acts upon the central nervous system causing effects on behaviour.
Pulmonary oedema. Swelling of the tissue of the lungs with results usually associated with bad circulation.
Pyrotechnic charge. A munition containing smoke-producing chemicals.
Sequellae. Abnormal condition resulting from previous disease.
Subcutaneous. Introduced below skin.
Tolerance. Phenomenon whereby with repetitive doses of a drug increasingly large concentrations are required to produce the same result.
Cross-tolerance. When doses of one drug produce tolerance to another.
Toxic. Acting as or having the effects of a poison.
Toxicity. Measure of the dose of a toxic agent required to produce a toxic effect.
Ugr. Microgram, *i.e.*, millionth of a gram.
Vector. An animal, insect, or other organism that carries and transmits a virus or other micro-organism.

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NO.	DATE	DESCRIPTION	STAT
116		Laboratory Studies on the Immune Effects of Halogenated Aromatics By Michael L. Luster, Robert E. Faith and George Clark	Ir
117		The Effect of Perinatal Exposure to Tetrachlorodibenzo-p-Dioxin on the Immune Response of Young Mice, by Peter T. Thomas and Ronald D. Hinsdill	"
118		Increased Susceptibility to Bacterial Infection as a Sequela of Exposure to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin, J.E. Thigpen, et al.	"
119		Studies on 2,3,7,8-Tetrachlorodibenzo-p-Dioxin Induced Immune Suppression and Decreased Resistance to Infection: Endotoxin Hypersensitivity, Serum Zinc Concentrations and Effect of Thymosin Treatment, by J.G. Vos, et al.	"
120		Examination of Bone Marrow Immunologic Parameters and Host Suspectibility Following Pre- and Postnatal Exposure to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	"
121		Tissue Distribution of the Receptor for 2,3,7,8-Tetrachlorodibenzo-p-dioxin in the Rat, by Jan M.B. Carlstedt-Duke	"
122		Effects of Brief in Vitro Exposure to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) on Mouse Lymphocytes, by Michael I. Luster, et al.	"
123		Medical Problems Raised by the TCDD Contamination in Seveso, Italy G. Reggiani	"
124		Direct Testimony of Dr. Renate D. Kimbrough, w/Cirrculum Vitae	"
125		Map MISSOURI / ILLINOIS	"
126		Memorandum to Director, Center for Disease Control, from Bacterial Disease Branch, Epidemiology Program, Subject: Toxic Illness, Lincoln County, Missouri	"
127		Memorandum to Chief, Bacterial Diseases Branch, from Renate D. Kimbrough Subject: Rabbit Ear Test for the Presence of 2,3,7,8-Tetrachlorodibenzo dioxin, dtd July 30, 1974	"
128		Tetrachlorodibenzodioxin: An Accidental Poisoning Episode in Horse Arenas, Coleman D. Carter, etal.	"
129		Epidemiology and Pathology of a Tetrachlorodibenzodioxin Poisoning Episode by Renate D. Kimbrough, M.D., et al.	"
130		Memorandum to Director, Center for Disease Control, from Cancer and Birth Defects Div., Bureau of Epidemiology, and Clinical Chemistry Div., Bureau of Laboratories Subject: 2,3,7,8-Tetrachlorodibenzodioxin Contamination of Salvage Motor Oil Used in Dust Control Spraying in Missouri	"

EPA EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
131	4-22-80	Direct Testimony of Frederick W. Kutz	In
132	4-22-80	A Method For Determination of Low Levels of Exposure To 2,4-D and 2,4,5-T	In
133	"	Comparative Metabolism of Phenoxy Herbicides In Animals	"
134	"	Residual Properties of 2,4,5-T and Picloram in Sandy Rangeland Soils C.J. Scifres, H.G. McCall, R. Maxey, and H. Tai	"
135	"	Memorandum to Robert Brown, SPR dtd March 22, 1979 in re Summary of Federal Monitoring Program Data On 2,4,5-T,Silvex and TCDD	"
136	"	Human and Environmental Monitoring for Herbicides Used In Forestry	"
137	"	Multiresidue Procedure for Halo-and Nitrophenols. Measurement of Exposure to Biodegradable Pesticides Yielding these Compounds as Metabolites	"
138	"	National Pesticide Monitoring Programs	"

EPA EXHIBITS

NO.	DATE	DESCRIPTION	STATU
139	4-16-80	Direct Testimony of Dr. William M. Upholt	IN
140	"	Dioxin Implementation Plan	"
141	4-17-80	Direct Testimony of Dr. Morton Beroza	"
142	"	Exposure, Toxicity, and Risk Assessment of 2,4,5-T/TCDD (This Report Contains Information of Proprietary Nature and Should Be Disseminated Accordingly)	"
143	"	Table 1 - Summary of Environmental Chemistry Data Requirements By Intended Use Pattern (FR Reprint)	"
144	"	Persistence of 2,4,-D, 2,4,5-T, and Dicamba in Range Forage Grasses	"
145	"	The Fate of 2,4,5-Trichlorophonoxyacetic Acid (2,4,5-T) Following Oral Administration to Man - P. J. Gehring, CG. Kramer, B.A. Schwetz, J.Q. Rose, and V.K. Rowe	"
146	"	Environmental Degradation of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	"
147	"	Response of Dow Chemical U.S.A. To Notice of Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing 2,4,5-T	"
148	"	Environmental Distribution of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin	"
148 (A)	9-25-80	(TCDD) Applied With Silvex To Turf In Microagpecpsuste	
148 (b)	2-5-81	Corrections - A Comparison of Trace Element Contents of FL.&Brazil Orange	
149	"	Persistence and Metabolism of Chlorodioxins In Soils	"
150	"	Report OEHL TR-78-92 USAF OEHL TECHNICAL REPORT The Toxicology, Environmetal Fate, and Human Risk of Herbicide Orange and Its Associated Dioxin	"
150-1	"	The Toxicology, Environmental Fate, and Human Risk of Herbicide Orange and Its Associated Dioxin	"
151	"	Distribution of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) In Aquatic Model Ecosystem	"
152	"	Chlorodioxins in Pesticides, Soils, and Plants - C.S. Helling, A. R. Isensee, E.A. Woolson, P.D.J. Ensor, G.E. Jones, J.R. Plimmer, and P. C. Kearney	"
153	"	Photolysis of highly Chlorinated Dibenzo-p-dioxins by Sunlight	"
.54	"	Absorption and Translocation of Root and Foilage Applied, 2,4-Dichlorophenol, 2,7-Dichlorodibenzo-p-Dioxin, and 2,3,7,8-Tetrachlorodibenzo-p-dioxin - Allan R. Isense and Gerald E. Jones	"

EPA EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
155	4-17-80	Retention and Excretion of 2,3,7,8-Tetrachlorodibenzo-p-dioxin by Rats - George F. Fries and George S. Marrow	In
156	"	TCDD Analysis in Environmental Samples	"
157	"	Letter to EPA Document Control Officer dated June 28, 1980 from Dow Chemical Company, Environmental Services signed by John R. Cledhill	"
158	"	A Search for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) in an Environment Exposed An-ually to 2,4,5-Trichlorophenoxyacetic Acid Ester (2,4,5-T)Herbicides	"
159	"	A Residue Study of Beef Cattle Consuming 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD)	"
160	"	A Residue Study On Sheep Consuming 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD)	"
161	"	Secretion of TCDD In Milk and Cream Folowing The Feeding of TCDD To Lactating Dairy Cows	"
62	"	A Search for the Presense of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin Beef Fat - C.W. Kocher, N.H. Mahle, R.A. Hummel & L.A. Shadoff	"
163	"	Analysis For TCDD Residues In Rice Grain From Retail Stores and From Fields Treated with 2,4,5-T	"
164	"	Chlorinated Insecticides: Fate In Aqueous Suspension Containing Mosquito Larvae	"
165	4-18-80	Direct Testimony of Mr. Robert A. Maxey	"
166	"	Phenoxy Acid Herbicides In Water	"
167	"	Water Tables	"
168			
169	"	Memorandum in re Summary of Water Supply Performance Evaluation Study from John A. Winter to Dwight G. Ballinger	"
170	"	Direct Testimony of Mr. Thomas E. Dixon	"
171	"	Interagency Agreement Between The U.S. Geological Survey and The U.S. Environmental Protection Agency	"
172	"	National Surface Water Monitoring Program Routine Analytical List	"
173	"	MASQUAN Stations to be included in an Intensive Survey of U.S. Cotton Culture Pesticides	"

EPA EXHIBITS

).	DATE	DESCRIPTION	STATU
174	4-18-80	Letter to R.J. Pickering, U.S. Geological Survey dated Jan. 29, 1979	In
175	"	Memorandum to Mike Dellarco, EPA, SPR dated July 24, 1979 from Tom Dixon, Life Scientist Survey and Analysis Div.	"
176	"	Maps	"
176-1	"	Letter to John Zackrison, Esq. of Kirkland & Ellis dated April 17, 1980 w/attachment	"
177	"	Memorandum to Forest Supervisor, Williamette NF and Director Watershed dated Dec. 28, 1978 from Paul E. Buffam	"
178	"	Letter to Mr. Tuck Vath, North Coast Water Quality Control Board dated August 18, 1977 in re to Six River Falls 1975 Herbicide Spray Monitoring Program signed by Richard D. LaVEN	"
179	"	Letter to Mr. Tuck Vath dated Nov. 26, 1975 signed by W.D. Harlan	"
180	"	Memorandum dated Aug. 30, 1976 to Howard Steeley from John Bernhardt in re Aquatic Monitoring Results of ITT Rayonier 1976 Aerial Spray Project In Western Washington	"
81	"	Letter to Mr. Tim Backstrom dated Sept. 24, 1979 from John Bernhardt	"
182	"	2,4,5-T Persistence In A West Virginia Watershed	"
183	"	Forests and Rangelands As Sources of Chemical Pollutants, 1976 L.A. Norris and D.G. Moore	"
184	4-22-80	Direct Testimony of Robert W. Baughman	"
185	"	Tetrachlorodibenzo-P-Dioxins In The Environment High Resolution Mass Spectrometry At The Picogram Level - A thesis by Robert W. Baughman	"
186	"	An Improved Analysis For Tetrachlorodibenzo-p-Dioxins	"
187	"	An Analytical Method for Detecting TCDD (Dioxin): Levels of TCDD In Samples From Vietnam	"
188	"	Tetrachlorodibenzodioxin In The Environment: Sources, Fate, and Decontamination	"
189	"	N eutral Cleanup Procedure for 2,3,7,8-Tetrachlorodibenzo-p-dioxin Residues in Bovine Fat and Milk	"
190	"	Symposium On The Use of Herbicides In Forestry	"
191	"	TCDD Analysis In Environmental Samples (Final Report)	"

ENVIRONMENTAL PROTECTION AGENCY
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J.	DATE	DESCRIPTION	STATUS
192	4-22-80	Letter to Ms. Carolyn K. Offutt, Office of Special Pesticide Review, EPA signed by Matthew Meselson, Harvard University	In
193	"	Memorandum to Associate Director, RRP dated Feb. 13, 1979 from Chief, ECB in re TCDD In Human Milk Study	"
194	"	Memorandum to Mr. Karl E. Bremer, Toxic Substances Coordinator EPA, Region V dated Dec. 20, 1978 from Dr. Edward O. Olswald in re Summary of Results for Analyses of Samples of Fish From Michigan	"
195	"	Memorandum dated April 9, 1979 to Mr. Karl E. Bremer, TS,EPA Region, V signed by Edward O. Olswald, Ph.D.	"
196	"	Memo to A. Dupy , Harless, Meselson, Berosa, Baughman, Upholt & Gross dated March 20, 1980 signed by Donald G. Barnes, Health Effects & Science Policy, Office of Assistant Administrator for Pesticides & Toxic Substances	"
197	"	Tables (Beef Fat)	"
198	"	Virginia Right of Way Study	"
99	"	DMP Deer and Elk Study	"
00	4-23-80	Direct Testimony of Dr. Aubry E. Dupuy, Jr.	"
201	"	Sample Preparation and Gas Chromatography-Mass Spectrometry Determination of 2,3,7,8-Tetrachlorodibenzo-P-Dioxin	"
202	"	Direct Testimony of Gunter Zweig	"
203	"	Essays In Toxicology by Frank R. Blood	"
204	"	Wiley Award Address - The Vanishing Zero--Ten Years Later. A Status Report on Recent Advances in Pesticide Analysis, by Gunter Zweig	"
205	"	Estimation of Small Amounts of O,O-Diethyl - O,p-Nitrophenyl Thiophosphate by P. R. Averell and M.V. Norris	"
206	"	Colorimetrick Determination of DDT Color Test For Related Compounds	"
207	"	DDT Residues In Milk from Dairy Cows Fed Low Levels of DDT In Their Daily Rations	"
208	"	Separation and Identification of Chlorinated Organic Pesticides by Paper Chromatography. XI. A Study of 114 Pesticide Chemicals; Technical Grades Produced in 1957 and Reference Standards by Lloyd C. Mitchell, HEW	"

ENVIRONMENTAL PROTECTION AGENCY
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.O.	DATE	DESCRIPTION	STATU
209	4-23-80	Specific Detection of Halogens and Phosphorus by Flame Ionization by Arthur Karmen, John Hopkins University School of Medicine	In
210	"	Chromatographic Techniques for Pesticide Residue Analysis, Gunter Zweig	"
211	"	Residue Analysis Applications of Mass Spectrometry by James F. Ryan	"
212	4-24-80	Direct Testimony of Mr. Robert L. Harless	"
213	"	Dioxin Toxicological and Chemical Aspects, Edited by Flamingo Cattabeni , and Aldo Cavallaro and Giovanni Galli	"
214	"	26th Annual Conference On Mass Spectrometry and Allied Topics dated May 28-June2, 1978, St. Louis, Mo.	"
215	"	25th Annual Conference on Mass Spectrometry and Allied Topics dated May 29 - June 3, 1977, Washington, D.C.	"
216	"	Analysis of Environmental Samples for TCDD Residues	"
217	4-25-80	Gas Chromatography/Mass Spectrometric Methods of Analysis For 2,3,7,8-Tetrachlorodibenzo-P-Dioxin (TCDD) Residues	"
218	"	Memorandum to Mike Dellarco, SPR, from Robert Harless dated Feb. 26, 1980 in re Results of Capillary Column GC/HRMS Analysis Performed On Extracts of Human Adipose Tissue for 2,3,7,8-TCDD Residues (Neele Biopsy Feasibility Study)	"
219	"	Chart	"
220	"	Memorandum dated Feb. 11, 1980 to Mike Dellarco, SPR Div. from Robert Harless in re Results of Capillary Colum GC?HRMS Analysis Performed On Extracts of Sediment, Soil, and Fish for 2,3,7,8-TCDD Residues	"
221	"	Memorandum to Ms. Carolyn K. Offutt, SPR from Edward O. Oswald, Ph.D. dated April 4, 1979 in re Results of Capillary Column GC/HRMS Analyses Performed on Extract of Water and Sediment for 2,3,7,8-Tetrachlorodibenzo pardoxin (TCDD)	"
222	"	Direct Testimony of Dr. Thomas O. Tiernan	"
223	4-30-80	Direct Testimony of Dr. Michael L. Gross w/Cirrculum Vitae	"
223A	6-25-80	Direct Testimony of Dr. Michael L. Gross (SUPPLEMENT)	"
224	"	Final Report Analysis of Selected Environmental Samples for TCDD by GLC-MS Techniques	"
25	"	Memo dtd Feb 20, 79 Ms. Carolyn Offutt, Dioxin Project Manager, EPA w/attachments Data Report #1	"

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NO.	DATE	DESCRIPTION	STATUS
226	4-30-80	Herbicide Orange Site Treatment and Environmental Monitoring Summary Report and Recommendations for Naval Construction Battalion Center Gulfport Mississippi November 1979	In
227	"	Final Report Ultratrace Analysis of Tetrachlorodibenzo-p-dioxin in Samples of Deer Tissue by Gas Chromatography/High Resolution Mass Spectrometry July 10, 1979	"
228	"	Ltr dtd July 13, 79 to Dr. Hugh Black, U.S. Forest Service San Francisco, CA, w/attachment Final Report Ultratrace Analysis of Tetrachlorodibenzo-p-dioxin in Samples of Deer Tissue by Gas Chromatography/High Resolution Mass Spectrometry, from Dr. Michael L. Gross	"
229	5-1-80	Direct Testimony of Mr. James J. Boland	"
230	"	Pesticide Incident Monitoring System (PIMS)	"
231	"	Pesticide Incident Report #16228	"
232	"	Summary of Reported Pesticide Incidents Involving 2,4,5-T Pesticide Incident Monitoring System Report No. 245	"
233	"	Summary of Report Incidents Involving 2,4,5-TP Pesticide Incident Monitoring System Report No. 242	"
234	5-6-80	Direct Testimony of Mr. Michael J. Dellarco	"
235	"	FR dated April 21, 1978 - Pesticide Programs - Rebuttable Presumptions Agains Registration and Continued Registration of Pesticide Products Containing 2,4,5-T	"
236	"	FR dtd March 15, 1979 Decision and Emergency Order Suspending Regist ations for Certain Uses - Suspension Order - Notice of Intent To Cancel Suspended Uses (30 Days to Request Hearing) on 2, 4,5-T & Silvex	"
237	"	(FRL-1275-1 OPP-30000/26D) Preliminary Determination Concering the Rebuttable Presumption Against Registration of Certain Uses of Pesticide Products Containing 2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T); Hearing Availibity of Position Document	"
238	"	Final Determination Concering the Rebuttable Presumption Against Registration for Certain Uses of Pesticide Products Containing 2,4,5-T - Notice of Intent to Hold a Hearing To Determine Whether or Not Certain Uses of 2,4,5-T Should Be Cancelled - Publication of Final Position Document Concering all Non-Suspended Uses of 2,4,5-T	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STATUS
239	5-6-80	(FRL-1275-2-OPP-30000/3I) Preliminary Determination Concering A Rebuttable Presumption Against Registration of Pesticide Products Containing 2-(2,4,5-Trichlorophenoxy Propionic Acid (Silvex); Hearing: Availaibility of Position Document	In
240	"	Final Determination Concerning The Rebuttable Presumption Against Registration For Certain Uses of Pesticide Products Containing Silvex- Notice of Intent to Hold a Hearing To Determine Whether Or Not Certain Uses of Silvex Should be Cancelled- Publication of Final Position Document Concerning All Non-Suspended Uses of Silvex	"
241	"	Letters to EPA, Administrator commenting on the Suspension & Cancellation of 2,4,5-T & Silvex	"
242	5-9-80	Direct Testimony of Mr. Emil Regelman	"
243	"	Report of Analysis - Tobacco for 2,4-D & 2,4,5-T Check	"
244	"	Results of Arkansas Dept of Health resuls from Samples of Alfalfa, watermelon, pond water and well water collected July 21, 1978 and analyzed	"
245	"	Laboratory Analysis Reprot - State of Oregon Department of Forestry dated March 16, 1977 - Water Sample	"
246	"	Report of Loss Arising Out of Use of Pesticides Invistegation Report	"
247	"	Laboratory Service Sample Report - West Virginia Department of Agricultrue	"

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NO.	DATE	DESCRIPTION	STATU
248	5-7-80	Direct Testimony of Mr. Charles Lewis	In
249	"	Map - Location of Houston and Dickerson Counties, Tennessee	"
250	"	Map - Location of Treatment Area in Relation to Houston and Dickerson Counties, Tennessee	"
251	"	Affidavit of Mr. L. P. Denevan	"
252	"	Dickson Municipal Airport in Relation to Treatment Area	"
253	"	Report on Analysis	"
253a	"	Report of Analysis	"
253b	"	Report of Analysis	"
254	"	DED - WEED LV-4T Label	"
255	"	DED-WEED LV-4D Label	"
256	"	Pesticide Episode Report Form dated May 1, 1979	"
257	"	Pesticide Episode Report Form dated May 1, 1979	"
258	"	Dickson-Houston County Herbicide Incident	"
258a	"	Affidavit of William D. Parchman	"
258b	"	Affidavit of Douglas Adams	"
258c	"	Affidavit of Willie Ray Pate	"
258d	"	Affidavit of Mrs. John Spicer	"
258e	"	Affidavit of Mr. E. N. Stanfill	"
258f	"	Affidavit of Mr. Bob Dillard	"
258g	"	Affidavit of Mr. Pat Whitaker	"
258h	"	Affidavit of Mrs. Earlene Whitaker	"
258i	"	Affidavit of Mr. Lester Baker	"
258j	"	Affidavit of W. G. Vann	"
258k	"	Affidavit of Mrs. Dorothy Parchment	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
258L	5-7-80	Affidavit of Mr. J. L. Stanfill	In
258m	"	Affidavit of Mr. Charles Adkins	"
258n	"	Affidavit of Mr. Douglas Adams	"
258o	"	Affidavit of Mr. J. R. Anderson	"
259	"	Selected Damage Sites In Relation To Trenchmann Property, TN	"
260	"	Selected Damage Sites in Relation to Trenchman Property in Houston Co. TN	"
261	"	Photo	"
262	"	Photo	"
263	"	Photo	"
264	"	Photo	"
265	"	Map (Lester Baker Dwelling Area)	"
266	"	Photo	"
267	"	Letter to Mr. Frank Daviti in re Aerial application of defoilant over 3000 acre tract of timber in houston County, Tennessee from Joseph H. Rossman	"
268	"	Letter to Vernon R. Knight in re Aerial Herbicide applications and possible groundwater contamination in Houston County, Tennessee	"
269	"	Photo	"
270	"	Photo	"
271	"	Letter to Mr. E. H. Trenchmann dated July 25, 1978 in re Aerial Applicationof defoilant in Houston County	"
272	"	Photo - Aeiral view looking east showing Mr. Pate's farm	"
273	"	Report of Analysis (tobacco for 2,4-D & 2,4,5-T Check	"
274	"	Report of Analysis (Dark Fired Tobacco Leaves)	"
275	"	Photo - Color photograph taken , showing typical injury to tobacco damaged from herbicides applied to property owned by Mr. E.H. Trenckmann	"
276	"	Color photo taken July 20, 1978, showing typical injury to tobacco damaged from herbicides applied to property owned by Mr. E.H. Trenckmann	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STATU
276a	5-7-80	Color Photograph taken July 20, 1978 showing typical Injury to tobacco damaged from herbicides applied to property owned by Mr. E.H. Trenckmann	In
276b	"	Color photograph taken July 20, 1978 showing typical injury to tobacco damaged grom herbicides applied to property owned by Mr. E.H. Trenckmann	"
277	"	Report of Analysis (Tobacco for 2,4-D & 2,4,5-T check)	"
278	"	Report of Analysis (Tobacco Leaves)	"
279	"	Aerial view looking north, showing location of Mr. Vann's Tobacco field	"
280	"	View looking east (color photograph taken Sept. 27, 1979) showing home of Mr. Pate (center) and walnut tree (center right) damaged from herbicides being applied to a site approximately 4000 feet to the south on property owned by Mr. E.H. Trenckmann	"
281	"	View looking north (color photo taken Sept. 27, 1979) showing walnut trees damaged on Mr. Pate's farm from herbicides applied approximately 4000 feet to the south on property owned by Mr. E.H. Trenckmann	"
282	"	View looking north (color photo taken Sept. 27, 1979) showing five of six walnut trees damaged on Mr. Pate's farm from herbicides applied approximately 4000 feet to the south of Property owned by Mr. E.H. Trenckmann	"
283	"	Aerial view looking south (color infrared photograph taken Sept.26, 1979) of Mr. Pate's farm	"
284	"	Map - W.R. Pate Dwelling Area	"
284a	"	View looking north(photo taken July 20, 1978) showing walnut trees bordering Mr. Pate's farm damaged by herbicides applied apporximately 4000 feet to the south on property owned by Mr. E.H. Trenckmann	"
284b	"	View looking North (photo taken July 20, 1978) showing trees bordering Mr. Pate's farm. The yellowing in these trees was typical of the more susceptible species in the area (walnut, locust, and sassafrass)	"
284c	"	Rose bushes (photo taken July 20, 1978) on Mr. Pate's farm showing damage from herbicides applied approximately 4000 feet to the south on property owned by Mr. E.H. Trenckmann	"
284d	"	Tomato plant (Photo taken July 20, 1978) on Mr. Pate's farm showing damage from herbicides applied approximately 4000 feet to the south on property owned by Mr. E.H. Trenckmann	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STAT
285	5-7-80	Aerial photo looking east taken July 24 1978, of Mr. William's farm showing typical damage to trees in the vicinity of the area receiving the herbicide application. Spray area is approximately 4000 feet to the southwest.	In
286	"	Color photograph of relief model showing the Trenckmann property and five (5) selected damage sites.	"
286a	"	Relief Model - large map approximately 36"x24" (Not in the Folder)	"
287	"	Location of Topographic Profile	"
288	"	Profile (Graph)	"
289	"	Tennessee Valley Authority Meteorological Data	"
290	"	County Maps of Dickerson and Houston Counties	"
291	"	Color infrared photograph taken Sept. 26, 1979 showing a portion of the treated area	"
292	"	Letter to the property owners dated July 26, 1978 in re damage crops from Jack Milton, Assistant Extension Agent University of Tenn.	"
293	"	Direct Testimony of Mr. Michael E. Cooper	"
294	"	Direct Testimony of Mr. Lester Baker	"
295	"	Direct Testimony of Mr. William Edgar Matthews	"
296	5-9-80	Direct Testimony of Richard Petrie	"
297	"	Map - Pumphrey Farm Location Perry County, Arkansas	"
298	"	Map - Pumphrey Farm Location Perry County	"
299	"	Map - Pumphrey Farm and Adjacent Owners	"
300	"	Aerial Spray Agreement	"
301	"	Record of Custom-Application with 2,4-D,2,4,5-T Or Other Hormone-Type Herbicide	"
302	"	DED-WEED Label	"
303	"	Answers of Richard Black and Omniflight Helicopter, Inc. To Interrogatories of Plaintiff - Civ 78-47 - In The Circuit Court of Perry County, Arkansas	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STAT
304	5-9-80	Answers To Interrogatories - No. Civ 78-47 - In the Circuit Court of Perry County, Arkansas	In
305	"	Aircraft Inspection for Certificate To Apply 2,4,-D, 2,4,5-T Or Other Hormone-Type Herbicide	"
306	"	Color Infrared Aerial Photograph of Pumphrey Farm and Sprayed Area taken On October 1, 1979	"
307	"	Pumphrey Farm and Sprayed Areas - Flight Path of 6-15-78 - Arkansas Kraft Co. Sprayed Area	"
308	"	Arkansas Regulations of 2,4-D, 2,4,5-T and Other Hormone-Type Herbicides	"
309	"	Graphic Sc[illegible]	"
310	"	Pumphrey Farm Topographic Map	"
311	"	Color photograph of 2'x2' relief model showing 160 acre Arkansas Kraft owned timberland, 80 acre International Paper Company Timberland and 240 acre Pumphrey Farm.	"
312	"	Longitudinal Profile - Cross Section B	"
313	"	Local Climatological Data	"
314	"	Color Infrared Aerial Photograph of Pumphrey Farm and Arkansas Kraft Company Taken on Oct. 1, 1979	"
315	"	Color low Altitude Aerial Photo of 160 acre Arkansas Kraft Company treatment area and adjacent Pumphrey farm lookin toward the Southwest	"
316a	"	Blowup of Exhibit 314	"
316b	"	Blowup of Exhibit 314	"
317	"	Report of Inspection on 2,4-D, Etc. Complaint	"
318	"	Letter to Omniflight Helicopters, Inc and Arkansas Kraft Corp. dated July 25, 1978 sighed by Ralph Pay, Dept. of Commerce Arkansas State Plant Board	"
319			
320			
321	5-9-80	Pumphrey Farm Topographic Map - Health Department Sample Points	"
321a	"	Pumphrey Farm Topographic Map - Health Department Sample Points	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
322	5-9-80	Local Climatological Data	In
323	"	Letter to Mr. Allen Pumphrey dated July 21, 1978 from James T. Green, Jr., University of Arkansas Cooperative Extension Ser.	"
324	"	Plant Analysis Information Sheet - Agricultural Diagnostic Service Laboratory University of Arkansas	"
325	"	Agricultural Diagnostic Service Laboratory , University of Arkansas Soil Diagnosis and Plant Analysis Report	"
326	"	Letter to Mr. Allen Pumphrey dated July 31, 1978 from James T. Green	"
327	"	Agricultural Diagnostic Service Laboratory - University of Arkansas Diagnostic Soil Sample Information Sheet	"
328	"	Soil Testing and Research Laboratory Agriculture Experiment Station and Extension Service Cooperating Agronomy Department Fayetteville and Marianna, Arkansas - Test Results	"
329			
330			
331	5-9-80	Direct Testimony of Mr. Wibur D. Wise	"
332	"	Rejected	
333	5-13-8-	Direct Testimony of Dr. George W. Keitt	"
334	"	WEEDONE 2,4,5-T Label - Reg. No. 264-9	"
335			
336	"	Monongahela Power Company Contract Specification for Vegetation Management Aerial	"
337	"	Right-of-Way Maintenance Inspector Aerial Spray Check List	"
338	"	Appalachian Power Company's Accumulation 12-Point Dismissal System	"
339.1	"	Helicopter Spray Claim - 45H	"
339.2	"	Release In Full of All Claims	"
340.1	"	Vicinity Map, Annamoriah Area, Calhoun County, WV.	"
340.2	"	Location Map, Annamoriah Area	"
340.3	"	Detail Map, Annamoriah Area	"

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ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
340.4	5-13-80	Aerial Color Photograph, Site 2, Annamoria Area	In
340.5	"	Aerial Color Photograph, Site 3, Annamoria Area	"
340.6	"	Aerial Color Photograph, Site 4, Annamoria Area	"
340.7	"	Aerial Color Photograph, Site 5, Annamoria Area	"
340.8	"	Aerial Color Photograph, Site 6, Annamoria Area	"
340.9	"	Aerial Color Photograph, Site 7, Annamoria Area	:
340.10	"	Aerial Color Photograph, Site 8, Annamoria Area	"
340.11	"	Letter to Warnings from R.E. Frame to W.R. Jeffrey, Asplundh	"
340.12	"	Letter from John Perdue and Thomas Mason to Deward Offutt	"
340.13	"	An Aerial Spraying Investigation on the Property of Beward L. Offutt	"
341.	"	EPA Study In West Virginia - Detail Map-Calhoun County	"
342.1	"	Vicinity Map Showing Millstone Area in relation to West Virginia	"
342.2	"	Location Map showing Millstone site in relation to Calhoun County	"
342.3	"	Detail Map with Contours, showing Power Line and Oil and Gas Wells on Parsons' Property	"
342.4	"	Detail Map showing same features as 342.3 and With U.S. Geological Survey 20 ft. Contours added	"
342.5	"	Aerial Color Photograph of Parsons' Property and Vicinity	"
342.6	"	Aerial Color Photograph of Pasture Field showing water hole, under power line, within the right-of-way	"
342.7	"	Color Photograph of Topographic Relief Model Showing Parson's property and vicinity, Power lines, buildings , roads, oil and gas wells, and vegetation	"
342.8	"	Letter from John Perdue and Thomas Mason to Paul A. Parson dated Oct. 10, 1978	"
343	"	Model, Millstone Case (approximately 3'x3')	"
344	"	Film Millstone - Annamoriah Cases (No Duplicate)(Filed in Expando)	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
345	5-13-80	Letter from Robert Fram, Program leader, Pesticide Use and Application Plant Pest Control Div. West Virginia Department of Agriculture to W. R. Jeffrey, Chemical Supervisor, Asplundh Tree Expert Company, dated August 4, 1978.	In
346.1	"	Asplundh Accident Report	"
346.2	"	Letter from Harold Kahn to Mr. Bailey, Kentucky Utilities Company	"
346.3	"	Letter from John Kirch, Amchem, to Harold Kahn	"
346.4	"	Memo from Gaston Rose to Don Burrell, Asplundh	"
346.5	"	Memo from Bob Jeffrey to Don Burrell, Asplundh	"
346.6	"	Liability Accident Notice, Asplundh, describing nature of spray acitivity	"
346.7	"	Handwritten Note	"
346.8	"	Memo from Gaston Rose to Don Burrell dated 8/1/76	"
346.9	"	Letter from Lindsey Ingram to Herschel Taylor	"
346.10	"	Letter from Herschel Taylor to Harold Kahn	"
346.11	"	WEEDONE 2,4,5-T Label - EPA Reg. No. 264-9-AA	"
346a.1	"	Case Review by Arthur D. Baker, III	"
346a.2	"	Civil Peanlty Warning Citation from Robert A. Poss to King County Road District	"
346a.3	"	Letter from Mary Toohey to Vance Hitchcock covering Notice of Violation from WSDA	"
346a.4	"	Letter of Warning from Mary Toohey to Dewey Easter	"
346a.5	"	Map of Vicinity where the incident occured	"
346.6	"	Letter from James Seidel to EPA Federal Register Section in response to the RPAR Notice (FR#843/30,000/26)	"
347.1	"	Letter from T. Saylor to Asplundh	"
347.2	"	Letter from Donald Burrell, Asplundh, to T. Saylor, dated July 2, 1979	"
347.3	"	Letter from Donald Burrell, Asplundh, To T. Saylor, dated Aug. 11, 1979	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATI
348.1	5-13-80	Memo, Public Service Indiana, Aerial Spray Claim re Billie Faith, by S. E. Brandt	In
348.2	"	Map of location	"
348.3	"	Memo from J. McQueen to K. Weigand and L. Neidiffer dated Sept. 14, 1978, containing lab analysis data	"
348.4		Not Used	
348.5	"	Asplundh Daily Helicopter Spray report for Aug. 1, 1978	"
348.6	"	Asplundh Daily Helicopter Spray Report for August 8, 1978	"
348.7	"	Asplundh Daily Helicopter Spray Report for August 2, 1978	"
349.1	"	Helicopter Spray Claim re Thomas Young	"
349.2	"	Letter from W. R. Jeffrey, Asplundh, to Dr. J. Baniecki, WVU, requesting analysis of a plant sample	"
349.3	"	WVU Lab report, pest Specimen record, 9/4/74	"
349.4	"	Release in full of all Claims	"
350.1	"	Helicopter Spray Claim re Willie B. Greenleif	"
350.2	"	Monongahela Power R/W Maintenance Inquiries	"
350.3	"	Letter from E. Haines to R. Frame, WV DA, requesting sample Analysis	"
350.4	"	Release in full of all Claims	"
350.5	"	Letter from W. R. Jeffrey, Asplundh, to Greenleif acknowledging damage due to spraying	"
350.6	"	Daily Helicopter Spray Reports for July 6-8, 1978	"
351.1	"	Helicopter Spray Claim, re: W. Cottrell	"
351.2	"	Monongahela Power R/W Maintenance Inquiry re: W. Cottrell	"
351.3	"	Letter from E. Haines to R. Frame requesting Sample Analysis	"
351.4	"	Release In Full of All Claims	"
351.5	"	Letter from W. R. Jeffrey, Asplundh, to W. Cottrell acknowledging damages from spray and enclosing check	"
351.6	"	Copy of Check from Asplundh to Clarence W. Cottrell	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
352.1	5-13-80	Helicopter Spray Claim re: Martyn Lee Krupa	In
352.2	"	Helicopter Spray Claim re: Wilma Krupa	"
352.3	"	Memo on the Krupa Claim by Ed Haines, Asplundh	"
352.4	"	Memo by Bob Jeffrey on Krupa Claim	"
352.5	"	Ohio Department of Agriculture Laboratory Report	"
352.6	"	Memo on Plant Damages	"
352.7	"	Letter from W. R. Jeffrey to R. Wulfhorst, Ohio Department of Agriculture	"
352.8	"	Release In Full of All Claims	"
353.1	"	Public Service Indiana Claim memo re Meredith Lee, by S. E. Brandt	"
353.2	"	Location Map, attached to Claim Memo	"
353.3	"	Helicopter Daily Spray Report for August 24, 1978	"
353.4	"	Helicopter Daily Spray Report for August 25, 1978	"
353.5	"	Letter from W.R. Jeffrey, Asplundh, to Meredith Lee acknowledging damages	"
353.6	"	Release In Full of All Claims	"
353.7	"	Helicopter Daily Spray Report for August 23, 1978	"
354.1	"	Aerial Spray Claim re: Cecil Rowlett, by S.E. Brandt	"
354.2	"	Daily Helicopter Spray Report for August 1, 1975 A.M.	"
354.3	"	Daily Helicopter Spray Report for August 1, 1975 P.M.	"
354.4	"	Letter to Cecil Rowlett from W.R. Jeffrey, Asplundh, acknowledging damage	"
354.5	"	Release In Full of All Claims	"
355.1	"	Aerial Spray Claim re: Howard Mallory, by S.E. Brandt	"
355.2	"	Daily Helicopter Spray Report for 8/1/75, A.M.	"
355.3	"	Daily Helicopter Spray Report for 8/1/75, P.M.	"
355.4	"	INA Report of Claim Settlement, from R. Christian	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATI
356.1	5-13-80	Helicopter Spray Claim re: Jerry Elkins	In
356.2	"	Daily Helicopter Spray Report for May 25, 1976	"
356.3	"	Daily Helicopter Spray Report for May 26, 1976, A.M.	"
356.4	"	Daily Helicopter Spray Report for May 26, 1976, P.M.	"
356.5	"	Daily Helicopter Spray Report for June 11, 1976	"
356.6	"	Release In Full of All Claims	"
357.1	"	Spray Claim Report re: Raymond King	"
357.2	"	Asplundh Spray Claim Report re: Raymond King	"
357.3	"	Daily Helicopter Spray Report for July 27, 1976	"
357.4	"	Memo from W. R. Jeffrey to Bob Christian	"
357.5	"	Handwritten Note	"
357.7	"	Release In Full of All Claims	"
357.8	"	Daily Helicopter Spray Report for July 26, 1976	"
357.9	"	Daily Helicopter Spray Report for July 26, 1976	"
357.10	"	Daily Helicopter Spray Report for July 28, 1976	"
358.1	"	Helicopter Spray Claim re: Anthony Stevens	"
358.2	"	Daily Helicopter Spray Report for July 27, 1976	"
358.3	"	Copy of a Check to Anthony Stephens from Asplundh	"
359.1	"	Letter from Simon Herold to SWEPCO	"
359.2	"	Letter from L. Bango to S. Herold	"
359.3	"	Helicopter Spray Claim re: Simon Herold	"
359.4	"	Daily Helicopter Spray Report for May 22, 1976	"
359.5	"	Memo from H. Daniel to Bob Jeffrey	"
359.6	"	File Note indicating May ww was last spray date on that utility	"
359.7	"	Release In Full of All Claims	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STAT
360.1	5-13-80	Helicopter Spray Claim re: Theodore Karpinski	IN
360.2	"	Helicopter Spray Claim re: Alexander Karpinski	"
360.3	"	R/W Maintenance inquiry re: Theodore Karpinski	"
360.4	"	R/W Maintenance inquiry re: Alexander Karpinski	"
360.5	"	Letter from Ed Haines to Bob Frame Requesting Lab Analyses	"
360.6	"	West Virginia Department of Agriculture Lab Service sample Report	"
360.7	"	Daily Helicopter Spray Report of June 23, 1977	"
360.8	"	Daily Helicopter Spray Report of June 23, 1977	"
360.9	"	Daily Helicopter Spray Report of June 23, 1977	"
360.10	"	Letter to Mr. Theodore Karpinski dated July 18, 1978 from W.R. Jeffrey	"
360.11	"	Release In Full of All Claims	"
360.12	"	Letter to Alexander Karpinski dtd July 18, 1978 from W.R. Jeffrey	"
360.13	"	Release In Full of All Claims	"
361.1	"	Helicopter Spray Claim re: Thomas Slaughter	"
361.2	"	Daily Helicopter Spray Report for May 19, 1977	"
361.3	"	Daily Helicopter Spray Report for May 23, 1977	"
361.4	"	Letter from K. Lawrence to R. Bates	"
361.5	"	Release In Full of All Claims	"
362.1	"	Aerial Spray Claim Report and Map re: C.H. Eckert, by S.E. Brandt	"
362.2	"	Helicopter Spray Claim re: C. H. Eckert	"
362.3	"	Daily Helicopter Spray Report for Aug. 1, 1978	"
362.4	"	Memo from W.R. Jeffrey to C.H. Eckert	"
362.5	"	Letter from W.R. Jeffrey to C. H. Eckert	"
362.6	"	Release In Full of All Claims	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STAT
363.1	5-13-80	Letter from Mabel Stephens to Jim Martindale, including tally of destroyed timber	IN
363.2	"	Letter from John Gaughan II to Jim Martindale, with map	"
363.3	"	Letter from David Hunter to Steve Bodie	"
363.4	"	Letter from John Gaughan II to David Hunter	"
363.5	"	Letter and table of damaged trees from Jim Neil to John Gaughan, including invoice to Marvin Morgan Estate	"
363.6	"	Letter from David Hunter to Bob Jeffrey	"
363.7	"	Letter from W.R. Jeffrey to David Hunter	"
363.8	"	Release of All Claims, Elsie Morgan	"
363.9	"	Release of All Claims, Mabel Stephens	"
363.10	"	Letter to Mr. Bob Jeffrey in re Claim of Elsie C. Morgan dated July 9, 1976 from David E. Hunter Arkansas Power & Light Company	"
364	"	Report entitled "Chemical Control of Brush and Environmental Safety of Roadside Vegetation Management Chemicals," by D. James Moore, Purdue University, 1976. (Joint Highway Research Project JHRP-76-32), pp. 86-90	"
365	"	Direct Testimony of Dr. William Loy	"
366	"	EPA Study Areas in Oregon - Map	"
367	"	Benton County - Alsea Study Area - Map	"
368	"	North Half Lincoln County - Rose Lodge Study Area - Map	"
369	"	Alsea Vegetation Distribution - Map	"
370	"	Alsea Settlement Distribution - Map	"
371	"	Alsea Hydrology - Map	"
372	"	Alsea Land Ownership - Map	"
373	"	Alsea, Three Dimensional Contour Model (only 1) O&C Took 12/9/80	"
374	"	Rose Lodge, Three Dimensional Contour Model (only 1) 12/9/80	"

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ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
375	5-13-80	Alsea Area all Herbicide Applications - Maps	In
376	"	Rose Lodge, Herbicide Applications - Maps	"
377	"	Rose Losge, Vegetation Distribution - Map	"
378	"	Rose Lodge, Settlement Distribution -Map	"
379	"	Rose Lodge, Hydrology - Map	"
380	"	Rose Lodge, Land Ownership - Map	"
381b	"	Rose Lodge #1 - Low oblique aerial view facing South. Showing the Salmon River in the background with Bear Creek Road on the right. Houses and proximate clear cuts shown	"
382b	"	Rose Lodge #2 - Low oblique Aerial view facing North. Showing the Salmon River in the background with Bear Creek Road in the center ground. Relationship between clear cuts and proximity of settlement	"
383b	"	Rose Lodge #3 - Low oblique aerial view facing South looking at Bear Creek Road area. Relationship of settlement and proximity of clear cut operations	"
384a	"	Alsea#1- Photograph of low oblique aerial view of Honey Grove Valley area facing west. Showing settlement patterns and relationships of roads and creeks	"
384b	"	Alsea#1- Tracing of Low oblique aerial view of Honey Grove Creek Valley area facing west. Showing settlement patterns and relationship of roads and creeks	"
385a	"	Alsea #2 - Photograph of low oblique aerial view of Honey Grove Creek Valley facing north east, showing surrounding hills and past logging operations	"
385b	"	Alsea #2 - Tracing of low oblique aerial view facing N.E. Showing Honey Grove Creek Valley and surrounding hills and past logging operations	"
386a	"	Alsea #3 - Photograph of high oblique aerial view looking east showing general relationships of settlement patterns, roads and creeks in a forest milieu	"
386b	"	Alsea #3 - Tracing of high oblique aerial view looking east. General relationships of settlement patterns, roads, and creeks in forest milieu	

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STAT
387	5-14-80	Direct Testimony of Mr. John Anderson	In
388	"	Results of the Stream Monitoring Program Conducted during FY 1977 Herbicide Spray Project by John J. Cameron and John W. Anderson	"
389	"	Memorandum to District Manager from Assistant District Fisheries Biologist dated June 24, 1978 in re Follow-up on the Results of the Stream Monitoring Program Conducted During the FY1977 Aerial Application of Herbicides to the Bud-break Stage of Vegetation Growth	"
390a	"	High elevation photo of water monitoring study area - Class II stream with buffer	"
390b	"	Helicopter application of Herbicide spray at Upper Smith River Unit	"
390c	"	Typical watercourse monitored in study	"
390d	"	White paint strip marking buffered zone for aerial application	"
391		Direct Testimony of Mrs. Christina Hutchison	"
392		Oregon Department of Forestry Map showing location of spray unit and monitoring points	"
393a		Letter from Susan Page to Logan A. Norris, dated April 7, 1977	"
393b		Letter from Logan A. Norris to Susan Page, dated April 26, 1977	"
394		Letter from Cliff Mann, Oregon Department of Forestry, to Mr. Ernest Hutchison, dated May 2, 1977	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
395	5-14-80	Direct Testimony of Dr. Bernard Smale	In
396	"	USDA Forest Service map of Pacific Northwest Region	"
397	"	Aerial infrared color point of Publishers' Paper Company Forest spray unit after broadcast burn (looking east). McMillin Creek and tree buffer seperate the two sprated sites. The broadcast burn (black in photo) of the herbicide dessicated trees and brush indirectly defines the size and location of sprayed areas. Manual or mechanical tree and brush removal was applied to sensitive areas such as canyon walls of McMillin Creek	"
398	"	Rose Lodge Settlement and Land Ownership Map. Combination of 378 and 380	"
399	"	Rose Lodge Land Ownership and Hydrology Map	"
400	"	Aerial infrared color print of the Bear Creek Road section of Rose Lodge, Oregon and portion of ajoining Publishers' Paper Company forest clear cut. Photo taken due east after broad cast burn of July 31, 1978. Green of trees, grass appears red, sunlighted areas yellow and black recorded unchanged in infrared photographs	"
401	"	Aerial infrared color print of McMillin Property and adjoining Publishers' land	"
402	"	Oregon Forest Practices Act Chemical Rules and Guidelines, 1978 State of Oregon, Department of Forestry	"
403	"	Notification of Operations filed by Publisher Paper Company of intent to apply herbicides (Feb. 4, 1977)	"
404	"	Pilot applicator record of spray operation	"
405	"	WEEDONE 170 Product Label	"
406	"	DOWPON (M) Grass Killer Label	"
407	"	Portland State University, Oct. 3, 1978 Newspaper account of Rose Lodge Incident	"
408	"	State of Oregon, Department of Agriculture Memorandum by T. Harrison summarizing Rose Lodge Incident, 1978	"
409	"	Color Pring of malformed leaves of artichoke plant from McMillin's garden	"
410	"	Color pring of malformed leaves (formative effects) on artichoke plant from McMillin's garden	"
411	"	Report of Loss Arising out of Use of Pesticides Investigation Report dated Sept. 19 & Oct. 6 1978, claimant Norma McMillin	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
412	5-14-80	Request for Analysis - Pesticide residue	In
413	"	Color print of damaged squash plant from McMillin vegetable garden	"
414	"	Color print of of branch of damaged rose of McMillin property	"
415	"	Color print of Phenoxy symptoms on grape in McMillin garden	"
416		Not Used	
417	"	Map - Relationship of Publishers Spray Site To Major Streams water Sampleing Locations on Streams and Following Rose Lodge Dwellings	"
418	"	NOAA Weather records from Pacific City, Salem and Depoe Bay Oregon for June 21 through July 20, 1978	"
419	"	Memorandum dated Nov.17, 1978 to Jeff Handy, Oregon State Dept. of Forestry from Kent A. Smith, OSDA Dept of Agriculture, Oregon	"
420	"	Request for Analysis - Pesticide Residue by Oregon State Forestry	"
421	"	Oregon State Dept. of Forestry's Memo dated Dec. 5, 1978 to M. Templeton signed by J. Handy in re spray contamination at Rose Lodge, Oregon	"
422	"	Common Sense Steps to Take In Avoiding Drift	"
423	"	Reducing Airborne Drift Losses with Nalco-Trol by Norman B. Akesson, and Wesley E. Yates	"
424 thru 430		Not Used	
431	5-15-80	Direct Testimony of Mr. Thomas Harrison	"
432	"	Summary Sheets on Pesticide Investigations made by the Oregon State Department of Agriculture in 1976, 1977 and 1978	"
433		Direct Testimony of Mr. William H. Kosean	"
434	"	Direct Testimony of Dr. Thomas Ellwanger	"
435	"	Herbicide Project 1976, Performance Summary, from: Matthew Kowalewski (Alsea area Silviculturist), to File: -YA 514-CT6-132	"
436	"	Bureau of Land Management (BLM) Project Map for Project No. S-A-HT-76-1d	"
437	"	Location of Alsea Area within Benton County, Oregon	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBIT

NO.	DATE	DESCRIPTION	STAT
438	5-15-80	Topographic Map of Alsea Area	In
439	"	Log book of Mr. Charles H. McKeen (Evergreen pilot) indicating activities from April 1 thu April 6, 1976	"
440	"	Observation of Mr. Mell Killman (BLM) dated April 3, 1976	"
441	"	Aerial black and white photo showing the Greens' property viewed from directly overhead. Property lines are indicated along with locations of pictures and sampling points of April 7, 1976 BLM inspection	"
442	"	Deposition of Mr. Charles H. McKeen, dated January 21, 1977	"
443	"	Statement of Mr. Gary Green to Bruce L. Engel (Counsel for the Greens)	"
444	"	Statement of Mrs. Giselä Green to Bruce L. Engel	"
445	"	Statement of Mr. Merrill Maloney to Bruce L. Engel	"
446	"	Overlay of Alsea settlements over BLM-treated portions of Sections 33, 3, 27, and 5	"
447	"	Letter dated May 25, 1979 from Mr. Neil Skill, Office of State Forester, to Mr. Tom Ellwanger, EPA	"
448	"	Overlay of Alsea land ownership over Alsea settlements	"
448b	"	Same as exhibit 448 with markings	"
449	"	BLM Report Number 1 (Form 9100-1a) by Mr. Matthew Kowalewski, dated April 3, 1976	"
450	"	Letter dated May 29, 1979, from Mr. Robert Thompson of Evergreen Helicopters to Mr. Tom Ellwanger of EPA	"
451	"	Memorandum dated April 9, 1976, from Mr. Joe Patton of BLM to District Manager and Files, subject: Investigation of possible Herbicide Spray Damage - Honeygrove Road Area	"
452	"	Plot map indicating location of samples taken on April 7, 1976 by BLM personnel	"
453	"	Memorandum dated April 19, 1976, from Mr. Joe Patton of BLM to District Manager and Files, in re Possible Herbicide Spray Damage - Gary Green Property	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBIT

NO.	DATE	DESCRIPTION	STAT
454	5-15-80	Editorial on Spray damage from Corvallis Gazette Times, dated August 12, 1976, by Beverly anc Charles Chance	In
455	"	Letter dated June 29, 1977, from Mr. Lloyd B. Ericsson (Attorney representing Evergreen Helicopters) to Bruce L. Engel (Counsel for the Greens)	"
456	"	Direct Testimony of Gisela Green	"
457	5-16-80	Direct Testimony of Daniel Elam	"
458	"	Letter to Daniel Elam dated August 24, 1979 from Martin Thingvold	"
459	"	Letter to Mr. Daniel W. Elam dated January 31, 1977 from Arnold P. Appleby, Professor of Crop Science, Oregon State University	"
460a	"	Letter to Daniel Elam dated September 16, 1976 from Martin Thingvold Extension Agent, Oregon State University	"
460b	"	Letter to Mr. Martin Thingvold dated September 13, 1976 from Homer M. Hepworth, Extension Agronomist -- Weed Control, Oregon State University	"
461a	"	Letter to James Wakefield, Chief Meterologist Weather Forecasing National Weather Service, Portland Oregon dated September 20, 1976 from Daniel W. Elam	"
461b	"	Letter to Daniel W. Elam dated September 22, 1976 from James D. Wakefield, Meteorologist U.S. Department of Commerce, National Oceanic and Atmospheric Administration	"
462	6-5-80	Direct Testimony of Mr. Neil T. Skill	"
463	"	Field Guide to Oregon Forest Practice Rules - State of Oregon Department of Forestry	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
464	5-20-80	Direct Testimony of Mr. Ugo F. Pezzi	In
465	"	Map of Siuslaw National Forest	"
466	"	Laboratory Analysis Report, dated June 14, 1971 from Oregon Department of Agriculture, Purchase Order, U.S.D.A, dated May 25, 1971, Sample analyses and tags.	"
467	"	Laboratory Analysis Report, dated September 19, 1972 from Oregon Department of Agriculture, and letter from Perske to Hiatt, dated August 22, 1972	"
468	5-22-80	Direct Testimony of Dr. Jack D. Griffith	"
469	"	Letter from Bonnie Hill of Alsea, Oregon dated April 11, 1978 to EPA	"
470	"	Report of the Consultative Council on Congential Abnormaltities in Yarram District, by J. E. Alfred March, 1978 & 2,4,5-T and Human Birth Defects, Department of Health June 1977	"
471	"	Field, Barbara and Kerr, Charles. 1979. Herbicide Use and Incidence of Neural-Tube Defects. Lancet, June 23, 1979, p. 1341	"
472	"	Report of Assessment of A Field Investigation of six-year Spontaneous Abortion Rates In Three Oregon Areas in Relation To Forest 2,4,5-T Spray Practices, EPA 1979	"
473		Direct Testimony of Dr. Thomas J. Keefe	
474		Protocol for Second Phase of Spontaneous Abortion Study - Colorado Epidemiologic Pesticide Studeis Center Colorado State University, Fort Collins, Coloracdo, October 1978	
475		Investigation of Six-Year Spontaneous Abortion Rates in Three Oregon Areas In Relation to 2,4,5-T Spray Areas - Colorado Epidemiologic Pesticide Studies Center - January 1979	
476		Direct Testimony of Dr. Robert C. Duncan	
477		Letter from Thomas J. Keefe, Ph.D. to Dr. Robert Duncan dated June 27, 1979	
478		Computer file - Spontaneous abortions in Study Area and Control Areas by hospital, zip code, date, and gestational age (Feb. 14, 1980)	
479		Direct Testimony of Dr. Renee Stringham	In
480		Resolution No. 16- Oregon Medical Association House of Delegates April 22-24, 1977	(By Ord
481		Notice - Physicians Reaffirm call for Herbicide Moratorium, Oregon	"
482 a		Ballot Title - Lincoln County Watershed Protection Ordinance filed 4/10/80	"
482b		Ballot Title - Lincoln County Roadside Spraying Ordinance filed 4/10/80	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STA
483	5-30-80	Direct Testimony of Mrs. Bonnie Hill	In
484	"	The Protection and Purification of Rural Water Supplies - Div. Of Sanitation Oregon State Borard of Health - 1971	"
485	"	Letter to Federal Register Section dated July 27, 1980 from Wilbur P. McNulty, M.D., Oregon Regional Primate Research Center	"
486	"	Oregon Blue Book 1979-1980	"
487	"	Index to Cities and Towns with 1975 Populations - From Oregon Official Highway Map, prepared by Oregon State Transportation Commission	"
488	"	Letter to Ms. Bonnie Hill dated July 12, 1977 transmitting copies of Alsea Area aerial herbicide spraying in 1975 and 1976	"
488b	"	Bureau of Land Management Use of 2,4,5-T, Silvex and 2,4-D (chart)	"
489	"	Map - Willamette Industries	"
490a	"	Letter to Mr. Jay Giles EPA dated Oct. 1, 1979 from Neil T. Skill Forest Practices Director, Forestry Dept. Oregon transmitting copy of Computer Printout	"
490b	"	Computer Print Out	"
491	"	Letter to Bonnie Hill dated March 31, 1980 in re to data requested from Laurence R. Foster, M.D., Oregon Department of Human Resources Health Division	"
492a	"	Letter to Bonnie Hill dated April 8, 1980 in re to request for for Information from Edward G. Stauber, U.S. Dept of Interior	"
492b(1)	"	Map - Salem District Transportation Plan	"
492b(2)	"	Map - Alsea Ranger District Siuslaw National Forest	"
493a(1)	"	Map - Alsea Quadrangle	"
493a(2)	"	Map - Alsea Oregon	"
493a(3)	"	Map - Alsea Oregon	"
493a(4)	"	Map - Alsea Oregon	"
493a(5)	"	Map - Alsea Oregon	"
93a(6)	"	Map - Alsea Oregon	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	ST
493a(6)	5-30-80	Map - Alsea Oregon	I
493b	"	Letter to Mrs. Bonnie Hill dated March 10, 1980 from Ned H. Davis District Ranger, U.S. Department of Agriculture	"
493c	"	Chart (showing Year, Tri Number, Acres, Formulation of 2,4,5-T Sprayed)	"
493d	"	Bureau of Land Management Spray Data	"
493e(1)	"	Map - Alsea	"
493e(2)	"	Map - Salem District Transportation Plan	"
494	5-29-80	Direct Testimony of Dr. Ian C.T. Nisbet	"
495	6-6-80	Direct Testimony Of Mr. James R. Yowell	"
496	"	20"x20" enlargement of aerial photo taken April 19, 1980, with overlay indicating the locations of the application site, soybean damage, and adjacent crops	"
497	"	Photo report prepared for EPA by the Remote Sensing Laboratory, LockheedEngineering and Management Services Company	"
197a	"	Photo taken April 19, 1980, with overlay indicating property and crop Ownership	"
497b	"	Photo taken April 19, 1980, with overlay indicating areas of herbicide application and crop damage	"
497c	"	Photo taken April 19, 1980, with overlay indicating selected physical and Cultural features	"
497d	"	Photo taken April 19, 1980, with overlay indicating rice field irrigation and drainage flow	"
498a	"	Photo taken March 10, 1980 looking east across Casey rice field	"
498b	"	Photo taken March 10, 1980 looking northeast from Beasley soybean field	"
498c	"	Photo taken March 10, 1980 looking west across Beasley soybean field	"
499	"	Record of Custom Application with 2,4-D, 2,4,5-T, or other Hormone-type herbicide, Don Spurlock, Heth Flying Service, August 7, 1979	"

ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATU
500a	6-6-80	Record of Communication Memoranda Prepared by Jim Yowell dated April 10, 1980	I
500b	"	Record of Communication Memoranda prepared by Jim Yowell April 24, 1980	"
500c	"	Record of Communication Memoranda prepared by Jim Yowell dated May 15, 1980	"
501a	"	Transvaal Brush-Rhap A-4T Label, EPA Reg. No. 11687-13, accepted Feb. 14, 1972	"
501b	"	Letter from A. E. Sidwell, Director of Research, Transvaal, Inc. to EPA Fungicide-Herbicide Branch, and attachment, November 20, 1972	"
501c	"	Memo from T. E. Adamczyk, Acting Chief, Fungicide-Herbicide Branch to files, Dec. 20, 1972	"
501d	"	Letter from James L. Skaptason, Chief, Products Control Branch to Vertac, Inc., concerning transfer of Pesticide Registerations from Transvaal, Inc., to Vertac, Inc., Dec. 8, 1976	"
501e	"	Vertac Brush-Rhap A-4T Label, EPA Reg. No. 39511-17, accepted Sept. 27, 1979	"
501f	"	Application for Supplemental Registration of Distributor No. 39511-17-1386, submitted by Universal Cooperatives, Inc. approved Sept. 26, 1978	"
502	"	Dow DMA 4 Herbicide Label, EPA Reg. No. 464-196, accepted May 16, 1979	"
503	"	Regulations of 2,4-D, 2,4,5-T, and other hormone-type herbicides adopted under Acts 389 and 410 of 1975 by the Arkansas State Plant Board on Dec. 5, 1975 and amended Dec. 9, 1977, Circular 9-A, Arkansas State Plant Board	"
504	"	Aircraft Inspection for Certificate to Apply 2,4-D, 2,4,5-T, or other hormone-type Herbicide, Mr. Ernest Hill, March 23, 1979	"
505	"	Form 1004, Arkansas State Plant Board, 2,4-D, etc., Complaint submitted by Mr. Thomas Beasley, August 27, 1979	"
506	"	Report of Inspection on 2,4-D, etc., Complaint, filed by Mr. Ernest Hill, August 27, 1979	"

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STATU
507	6-6-80	Soybean Diseases Atlas, Published by the Cooperative Extension Service, University of Arkansas, copy of page 18 concerning "Phenoxy Herbicide Injury" (The entire Soybean Disease Atlas is listed as Reference 1)	In
508	"	Letter from Ralph Pay, Arkansas State Plant Board, to Heth Flying Service, September 7, 1979	"
509	"	Photo taken March 10, 1980 of access road, rice field, and soybean field	"
510a	"	Photo taken March 10, 1980 of soybean stubble showing adventitous root formation	"
510b	"	Photo taken March 10, 1980 of soybean stubble showing adventnetious root formation	"
511a	"	Close-up photo of sample of soybean stubble taken March 10, 1980	"
511b	"	Close-up photo of sample of soybean stubble taken March 10, 1980	"
12	"	Samples of Soybean stubble collected by Jim Yowell from Beasley soybean field on March 10, 1980. (brown Box filed in Expando)	"
513	6-12-80	Direct Testimony of Dr. Connie J. Stone	"
514	"	Lavy, T. L. Project Completion Report to National Forest Products Association. Measurement of 2,4,5-T Exposure of Forest Workers. August 30 to Oct. 3, 1978	"
515	"	Ramsey, J.C., Lavy. T. L., and Braun, W.H. Exposure of Forest Workers to 2,4,5-T: Calculated Dose Levels.	"
516	"	Simpson, G.R., Higgins, V., Chapman, J., and Bermingerham, S. 1978. Exposure of Council and Forestry Workers to 2,4,5-T. Med. J. Aust 2: 536-537	"
517a	"	Kolmodin-Hedman, B., Erne, K., Hakansson, M., and Egqvist, A. 1979. Control of Occupational Exposure to Phenoxy Acids (2,4-D and 2,4,5-T)	"
517b	"	Vetenskaplig Skriftserie 1979:17 Kontroll av yrkesmassig exponering for Fenoxisyror (2,4,-D och 2,4,5-T - Birgitta Kolmodin-hedman, Kurt Erne, Marianne Hakansson och Anita Engqvist	"

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O.	DATE	DESCRIPTION	ST
518	6-12-80	Direct Testimony of Mr. Frank P. Beck Jr.	Ir
519	"	General Highway Map, Washington County, Mississippi, marked to Indicate location of the site	
520	"	Mississippi Dept. of Agriculture and Commerce, Div. of Plant Industry, Applicator's Report of the Application of Hormone-Type Herbicides by Airplane (2,4-D, 2,4,5-T, etc.) July 17, 1979	"
521a	"	Letter from Philip D. Lewis, Lewis Bros. Flying Service, dtd May 19, 1980	"
521b	"	Letter from W. G. Boykin, Triple B. Farms, to Frank Beck, dtd May 20, 1980	"
522	"	Label for Amchem Amine 2,4,5-T for Rice, EPA Reg. No. 264-86	"
523	"	Test of Mississippi Code, Sections 69-21-1 to 69-21-27, concerning Regulation of Crop Spraying by Aircraft	"
524	"	Mississippi Department of Agriculture and Commerce, Div. of Plant Industry, Regulations Governing the Application of Hormone-Type Herbicides by Aircraft, as amended, Dec. 12, 1974	"
525	"	Mississippi Department of Agriculture and Commerce, Division of Plant Industry, Requirements for Aerial Applicators to Obtain a License to Apply Hormone-Type Herbicides	"
526	"	Mississippi Department of Agriculture and Commerce, Division of Plant Industry, Aerial Application of Hormone Herbicides: Study Guide.	"
527	"	Mississippi Department of Agriculture and Commerce, Division of Plant Industry, Certificate of Aircraft Inspection for Lewis Bros. Flying Service, April 6, 1979	"
528	"	Letter from Roy Reid, Area Pest Management Specialist, Mississippi Cooperative Extension Service, Stoneville, Mississippi, to Mike Peeples, Division of Plant Industry, Mississippi Department of Agriculture, May 19, 1980	"
529	"	Magazine artice entitled "Drop Boom" boosts accuracy and improves Coverage," in Custom Applicators, Februrary, 1980, p. 111-115	"
529b-1 thru b5		5 Photos of Lewis Bros. Flying Service Aircraft	"

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NO.	DATE	DESCRIPTION	ST
530	6-12-80	Sketch of application Site and damaged crops by Mike Peeples, Division of Plant Industry Mississippi Department of Agriculture and Commerce, April 16, 1980	I
531	"	Mississippi Department of Agriculture and Commerce, Division of Plant Industry, Report of the Inspection of Crops for the presence of injury resulting from Aerial Application of Hormone-Type Herbicides, August 1, 1979	"
532	"	Color Photograph taken August 1, 1979 looking south across Mr. Wood's soybean and cotton fields.	"
533a-c	"	Color photographs taken August 1, 1979 of 2,4,5-T damage in Mr. Wood's soybean field	"
533d-f	"	Color photographs taken August 1, 1979 of Mr. Woods' cotton field and damaged cotton.	"
534	"	Color photograph taken August 1, 1979 looking north across Mr. Woods' cotton and soybean fields.	"
535	6-17-80	Direct Testimony of Dr. Rainer Frentzel-Beyme	"
36a	"	Professor Alfred M. Thiess & Dr. R. Fretzel-Beyme, Department of Occupational Medicine & Health Protection, BASF Aktiengesellschaft, Ludwigshafen AM Rhein, West Germany. Mortality Study of Persons Exposed to Dioxin Following an Accident Which Occurred in the BASF on 13 November 1953	
536b	"	Table - Cancer deaths of the BASF Dioxin study, by site and morphology of malignancy	
537	"	Mortality of Persons Exposed to Dioxin during an Accident which Occurred in the BASF AG on November 17, 1953	
538	"	Tables: Additional Data from follow-up of BASF dioxin study	
539a	"	Schwerste akute Chloracne, eine Massenintoxikation durch 2,3,6,7-Tetrachlordibenzodioxin, by Paul J. Goldmann	
539b	"	Severe Acute Chloracne. A Mass Intoxication by 2,3,6,7-Tetrachlorobenzodioxin, by Paul J. Goldmann	
540a	"	Follow-up Report uber das Trichlorphenol-Dioxin-Unfallgeschehen in der BASF AG vom 13. November 1953	
540b	"	Follow-up Report on the Trichlorophenol-Dioxin Accident at BASF AG on 13 November 1953, by A.M. Thiess and P. Goldmann	

ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STA
541	6-18-80	Direct Testimony of Dr. Kirk T. Kitchin	I
542	"	Induction of Multiple Forms of Mouse Liver Cytochrome P-450	"
543	"	2,3,7,8-Tetrachlorodibenzo-p-Dioxin Induction of Aryl Hydrocarbon Hydroxylase in Femlae Rat Liver. Evidence for De Novo Synthesis of Cytochrome P-448, by Kirk T. Kitchin and James S. Woods	"
544	"	Induction and Suppression of Hepatic and Extrahepatic Microsomal Foreign-Compound-Metabolizing Enzyme Systems by 2,3,7,8-Tetrachlorodibenzo-p-Dioxin, by G.F.R. Hook, J.K, Haseman and G.W. Lucier	"
545	"	Induction of Aryl Hydrocarbon Hydroxylase Activity in Various Cell Cultures by 2,3,7,8-Tetrachlorodibenzo-p-dioxin	"
546	"	Comparison of 2,3,7,8-Tetrachlorodibenzo-p-dioxin, a Potent Inducer of Aryl Hudrocarbon Hydroxylase, with 3-Methylcholanthrene	"
547	"	Transplacental Induction of Mixed-Function Oxygenases in Extra-Hepatic Tissues by 2,3,7,8-Tetrachorordibenzo-p-Dioxin	"
548	"	2,3,7,8-Tetrachlorodibenxo-p-Dioxin (TCDD) Effects on Hepatic Microsomal Cytochrome P-448-Mediated Enzyme Activities [1,2]	"
549	"	TCDD- Induced Changes in Rat Liver Microsomal Enzymes, by G.W. Lucier, et al.	"
550	"	Sex-Dependent Regulation of Benzo[a]Pyrene and Zoxazolamine Metabolism in Rat Tissues, Hira L. Gurtoo and Natalie Bejba Parker	"
551	"	Transplacental Inducution of Carcinogen-Hydroxylating Systems with 2,3,7,8-Tetrachlorodibenxo-p-dioxin [1;2], by David L. Berry, et al.	"
552	"	Organ Specific Induction of Drug Metabolizing Enzymes by 2,3,7,8-Tetrachlorodibenzo-p-dioxin in the Rat	"
553	"	Quantitative and Qualtative Changes in the Metabolism of Benzo(a) pyrene in Rat Tissues after Intragastric Administration of TCDD	"
554	"	Postnatal Stimulation of Hepatic Microsomal Enzymes Following Administration of TCDD to Pregnant Rats	"
555	"	Studies on the Mechanism of Toxicity of the Chlorinated Dibenzo-p-dioxins, by Alan Poland and Edward Glover	"
556	"	Selection of Inducers: An Important Factor in Characterizing Genetic Differences to Induction of Aryl Hyrdocarbon Hydroxylase in Strains of Mice, Rajendra S. Chhabra, et al.	"

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NO.	DATE	DESCRIPTION	STATI
557	6-18-80	Chlorinated Dibenzo-p-dioxins: Potent Inducers of o-Aminolevulinic Acid Synthetase and ARyl Hydrocarbon Hydroxylase II. A Study of the Structure-Activity Relationship, Alan Poland & Edward Glover	In
558	"	Effects of Microsomal Enzyme Inducers IN VIVO and Inhibitors IN VITRO on the Covalent Binding of Benzo[a]Pyrene Metabolites to DNA Catalyzed by Liver Microsomes from Genetically Responsive and Nonresponsive Mice, Alan R. Boobis, et al.	"
559	6-24-80	Direct Testimony of Dr. Joseph Franklin Holson	"
560	"	Utility of Pharmacokinetics in Designing Toxicological Protocols and Improving Interspecies Extrapolation, John F. Young and Joseph F. Holson	"
561	"	Data Tables from NCTR Teratology Study of Technical and Purified Grades of 2,4,5-T	"
562	6-25-80	Report to Veterans Administration c/o Lawrence B. Hobson, M.D. Ph.D., Trace Analysis of Tetrachlorodibenzo-p-dioxin (TCDD) in Human Adipose by Michael L. Gross, Professor and Director 2-13-80	"
563	"	TCDD (2,3,7,8-Tetrachlordibenzo-p-dioxin) in Body Fat of Vietnam Veteran and Others	"
564	6-27-80	Direct Testimony of Dr. George Streisinger	"
564A	"	Direct Testimony of Dr. George Streisinger (ADDENDUM)	"
565	"	Ltr dtd 17 Oct 1977 to Mr. Jerry MacLeod, Biologist from Michael Watson, Ph.D.	"
566	"	Ltr dtd 6/4/79 to Mr. David Graham, USDA, Forest Service from Aubry E. Dupuy, Jr., Ph.D., Chemist	"
567	"	Memo to Hugh Black, U.S. Forest Service, from Dave Zeiner dtd December 8,1978 w/Deer Tissue-Phenoxy Herbicide Study	"
568	"	Animal History	"
569	"	Ltr dtd January 28, 1974 Sioxin Meeting - December 13, 1973	"
570	"	Ltr dtd March 13, 74 Mr. Carroll W. Collier "Dioxin Meeting, Washington, D.C. December 13, 1973,	"
571	"	Beef Fat Phase II	
572	"	Three-Generation Reproduction Study of Rats Given 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) in the Diet, F.J. Murray, et al.	"

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NO.	DATE	DESCRIPTION	ST
573	6-27-80	2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD): Results of a 13-Week Oral Toxicity Study in Rats', R.J. Kocira, et al.	
574	9-23-80	Ltr to Dr. C.B. Hiremath transmitting a copy of Promoting Action of TCDD In Hepatocarcinogenesis	
575	"	2,3,7,8-Tetrachlorodibenzo-p-dioxin as Cocarcinogen Causing 3-Metycholanthrene-initiated Subcutaneous Tumors in Mice Genetically "Nonresponsive" at Ah Locus	
576	"	Historical Control Data - Study No. 4	
577	"	Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin for Possible Carcinogenicity (Gavage Study)	
578	"	Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin for Possible Carcinogenicity (Dermal Study)	
579	7-1-80	Direct Testimony of Dr. Arthur W. Galston	"
580	"	Photodecomposition of 2,4,5-Trichlorophenoxyacetic Acid 2,4,5-T in Water	"
581	"	Residues of Chlorophenoxy Acid Herbicides and Their Phenolic Metabolites in Tissues of Sheep and Cattle	"
582	"	Degradation of 2,4,5-Trichlorophenoxyacetic Acid in Woody Plants by Charles H. Fitzgerald, et al.	"
583	"	Ecological Considerations Regarding Massive Environmental Contamination with 2,3,7,8-Tetrachlorodibenzo-para-Dioxin	"
584	7-3-80	Direct Testimony of Dr. Matthew Meselson	"
585	"	Memorandum of Telephone Conversation between Dr. Patrick O'Keefe Department of Chemistry, Harvard University and Dr. David Firestone, Analytical Chemistry and Physics Branch in re Analysis of Low Levels (parts per trillion and sub parts per trillion) of TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) in Human Milk	"
586	"	Light and Electron Microscopic Observations in Macaca Mulatta Monkeys fed Toxic Fat, Jr. R. Allen, D.V.M., Ph.D., and L.A. Carstens, B.S.	
587	7-7-80	Direct Testimony of Dr. Olav Axelson	"

NO.	DATE	DESCRIPTION	STATUS
588	7-7-80	Herbicide Exposure, Mortality and Tumor Incidence. An Epidemiological Investigation On Swedish Railroad Workers	In
589	"	Aspects On Confounding In Occupational Health Epidemiology	"
590a	"	Fenoxisyror Och Cancer	"
590b	"	Phenoxy Acids and Cancer	"
591a	"	Herbicide Exposure and Tumor Mortality	"
591b	"	Herbicide Exposure and Tumor Mortality; an Updated Epidemiological Investigation On Swedish Railroad Workers	"
592	"	Analysis of Some Older Scandinavian Formulations of 2,4-Dichlorophenoxy Acetic Acid and 2,4,5-Trichlorophenoxy Acetic Acid For Contents of Chlorinated Dibenzo-p-dioxins and Dibenzofurans	"
593a	"	Epidemiologisk metodik och bekampningsmedelsstudier	"
593b	"	Status: MSG: 202 (Listing from 1 to 173)	
593c	"	Epidemiologic Methodology And Pesticide Studies	"
593d	"	Epidemiologisk Metodik - Synpunkter PA Kritik Over Bekampningsmedelsstudier	"
593e	"	Epidemiologic Methodology - Views On Criticism Of Pesticide Studies	"
594	"	Case-Control Study: Soft-Tissue Sarcomas and Exposure to Phenoxyacetic Acids Or Chlorophenols	"
595a	"	Case-Control-studie Over maligna mesenkymala mjukdelstumorer och exposition for kemiska substanser	"
595b	"	Case-Control Study on Malignant Mesenchymal Tumors of the Soft Tissue and Exposure To Chemical Substances	"
596a	"	Maligna lymfom och exposition for kemiska substanser, sarskilt organiska losnigsmedel, klorfenoler och fenoxisyror	"
596b	"	Malignant Lymphoma and Exposure To Chemical Substances, Especially Organic Solvents, Chlorophenols and Phenoxy Acids	"
597a	"	A Note on Observational Bias In Case Referent Studies In Occupational Health Epidemiology	"
597b	"	A Note on Observational Bias In Case-Referent Studies In Occupational Health Epidemiology	"
598	"	Symptomatology, Morbidity and Mortality Experience of Chlorinated Phenoxyacid Herbicide (2,4-D; 2,4,5-T) sprayers in Finland. A Clinical and epidemiological Study	"
	"		

NO.	DATE	DESCRIPTION	STA
617	7-9-80	Letter to Stevens Van Strum concerning lack of spray records for Lane County for roadside spraying, 1974 thru 1976	In
618	"	University of Oregon Medical School records of Irene Durbin's delivery of youngest son	"
619	"	University of Oregon Medical Records concerning her youngest son surrounding his birth (Lyle George Durbin)	"
620	"	Resume of Office Visits of Lyle George Durbin, Dr. Reed McNeal, M.D.	"
621	"	Medical Records Concerning James Patrick and Kim Kerstin Durbin, 1974 and 1975, Dr. Reed McNeal, M.D.	"
622	7-10-80	Direct Testimony of Gail Slentz	"
623	"	BlM, Coos Bay District, Smith-Umpqua Resource Area Map	"
624	"	U.S. Geological Survey Topographical Map of relevant portion of Roman Nose Quadrangle	"
625	"	Spray Data from Bureau of Land Management for 22-acre tract in question, for 1975 and 1977	"
626	"	Medical Records for Gail Slentz, Sacred Heart Hospital, Feb. 13, 1978	"
627	"	Medical Records for Gail Slentz, Dr. Alan Cohn, Feb. 16, 1978	"
628	"	Laboratory Analysis Report, GHT Laboratories of Imperial Valley, California D-10831	"
629	"	Laboratory Analysis Report, GHT Laboratories of Imperial Valley, California 000169-E	"
630	"	Medical Records of Gail Slentz, Dr. Sarah Hendriskson, Apr.5, 1978	"
631	"	Ltr to NCAP dtd April 11, 1980 in re to ltr dated April 8, 1980 from Robert W. Bright, U.S. Department of Interior	"
632	"	Direct Testimony of Phissis Cribby	"
633	"	Transcript of BLM spray operation by Chetco ranger district inspector, 4-9-75	"
634	"	Contractor Performance Rating Sheet for Forest Service Contract 11-10428-8	"
635	"	Report to Files, Inspector Robert Turner, 3-14-75, Gold Beach Ranger District	"

NO.	DATE	DESCRIPTION	STATUS
599a	7-7-80	XXXVI Lakaresallskapets Riksstamma anordnad av Svenska Lakaresallskapet pa Stockholmsmassa, Alvsjo, Stockholm 5-8-dec 1979	In
599b	"	XXXVI National Conference Of The Medical Society Sponsored By The Swedish Medical Society At Stockholmsmassan, Alvsjo, Stockholm, 5-8 December 1979	"
600	7-9-80	Direct Testimony of Pat Clary	"
601	"	Bureau of Land Management, Roseburg District Map (Stouts Creek watershed area)	"
602	"	U.S. Geological Survey topographical map of Stouts Creek watershed area	"
603	"	Spray data from BLM, Stouts Creek, April, 1976	"
604	"	Affidavit from Judith Kahle concerning response of Roseburg Lumber to request for spray information	"
605	"	Medical Records for Patricia Clary, spring of 1976, 4-4-76, Dr. Monson	"
606	"	Medical Records for Patricia Clary 5-12-76 and 5-28-76 Dr. Grosso	"
607	"	Oregon Department of Agriculture Laboratory Analysis Report, Lab. Number 10653	"
608	"	Medical Records for Aisha Candrian, November, 1977, Douglas Comm. Hospital	"
609	"	Medical Records for Patricia Clary, June of 1978, Dr. Falk	"
610	"	Direct Testimony of Irene Durbin	"
611	"	See Map, (Exhibit No. 616)	"
612	"	Letter of March, 1979, from David White, Lane County Health Department to Irene Durbin	"
613	"	EPA Exhibit No. 223, p. 16,17,18, Site #9 Sample analyzed by Research Triangle Park, and University of Nebraska Laboratories	"
614	"	Responses by the U.S. Forest Service, Alsea District to Interrogatory #25, C.A.T.S. v. Bergland (D.Or. 1977) 428 F.S. 908	"
615	"	Aerial Maps	"
616	"	Alsea District map with overlays of roadside and areal spray sites 1974 through 1976	"

NO.	DATE	DESCRIPTION	STA
636	7-10-80	Contract Daily Diary, 8-30-75, 3-31-75, 9-10-75	In
637	"	Handwritten report on Contract 11-10466-S by Suskiyou Contracting Officer	"
638	"	Report on Herbicide Spill, Gold Beach District 4-28-76 by Forest Pesticide Use Coordinator	"
639	"	Handwritten report of spill described in PHC-7	"
640	"	Contract Daily Diary, April 20-27, 1976 on Contract 11-10485-S	"
641	"	Determination of Non-Responsibility, 1977, U.S. Forest Service, Region 6 Office	"
642	"	Letter from Siskiyou Contracting Officer, 4-19-76 find Corvallis Aero Service Non-Responsible	"
643	"	Contract 53-0401-9-12 Chetco Ranger District and Corvallis Aero Services, Inc.	"
644	"	Payment Invoice for Contract in PHC-12	"
645	"	Incident Report, Contract 53-101-8-6, Siskiyou National Forest, 4-27-78	"
646	"	Contract Daily Diary, 4-29-78 on Contract 11-1-485-S	"
647	"	Direct Testimony of Norma Grier	"
648	"	U.S. Geological Survey Topographical Map of French Creek watershed area	"
649	"	Oregon Department of Agriculture Water Sample Laboratory Analysis Report #05427 through 05430, PARC 3	"

NO.	DATE	DESCRIPTION	STA
650	7-15-80	Direct Testimony of Mr. Jere Christner	In
651	"	Map (water sampling site) Lowell Ranger District Willamette National Forest Oregon	"
652	"	Water Sample Analysis Results	"
653	"	Samples After Spraying On Day of Spraying	"
654	7-17-80	Direct Testimony of Dr. Logan Norris	"
654A	"	Corrections or Clarifications	"
655	"	The Biologic and Economic Assessment of 2,4,5-T- A Report of the USDA-States-EPA 2,4,5-T RPAR Assessment Team, Feb. 15, 1979 (Expando)	"
656	"	Herbicide Residues in Air, Alsea Basin - 1977	"
657	"	Impact of 2,4,5-T on Blodgett Forest - Description of an Experimental Aerial Application of 2,4,5-T	"
658	"	The Persistence of 2,4,5-T in a Pacific Northwest Forest	"
659	"	Persistence of 2,4-D and 2,4,5-T in Chaparral Soil and Vegetation	"
660	"	Update on Data From 2,4,5-T Study In A West Virginia Watershed dated June 16, 1980	"
661	"	TCDD Persistence In Low Light Intensities - Office Report 7/1/80	"
662	"	TCDD In Fish From Oregon Forest Streams - Office Report 7/1/80	"
663	"	The Response of Coho Salmon and Guppies to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in Water	"
664	7/21/80	Direct Testimony of Mr. Lavell O. Stranger	"
665	"	Siskiyou National Forest - Herbicide Applicators Handbook	"
666	"	Herbicide Methods	"
667	"	Division 100 - General Specifications - Contractor furnished Property and Services	
668	7-24-80	Direct Testimony of Alvin L. Young, Major, USAF	"
668-1	"	Portions of Direct Testimony and Exhibits of Major Alvin Young Subject to Motion To Exclude	"
668-2		Prints of 41 slides shown by Dr. Young July 22, 1980	"
669	"	Persistence, Bioaccumulation and Toxicology of TCDD In an Ecosystem Treated With Massive Quanities of 2,4,5-T Herbicide	"
670	"	Ecological Studies On A Herbicide-Equipment Test Area (TA C-52A) Eglin AFB Reservation, Florida, Jan. 1974	"

NO.	DATE	DESCRIPTION	STA
671	7-24-80	Field Studies of Wildlife Exposed to TCDD Contaminated Soils	IN
672	"	Studies of the Ecological Impact of Repetitive Aerial Applications Of Herbicides On The Ecosystem of Test Area C-52A, Elgin AFB, Florida	"
673	"	Fate of 2,3,7,8-Tetrachlorodibenso-p-Dioxin (TCDD) In the Environment Summary and Decontamination Recommendations	"
674	"	Residual Levels of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) Near Herbicide Storage and Loading Areas at Elgin AFB, Florida	"
675	"	Histopathological and Ultrastructual Studies of Liver Tissue From TCDD Exposed Beach Mice (Peromyscus Polionotus)	"
676	7/25/80	Direct Testimony of Dr. Rodney w. Bovey	"
678	"	Control of Live Oak by Herbicides Applied at Various Rates and Dates	
679	"	Control of Huisache and Associated Woody Species In South Texas	
680	"	Herbicide Combinations for Woody Plant Control	
681	"	The Use of A Woody Plant Nursery In Herbicide Research	
682	"	Hormone-like Herbicides In Weed Control	
683	"	Control of Yaupon and Associated Species	
684	"	Daily and Seasonal Response of Huisache and Macartney Rose to Herbicides	
685	"	Response of Selected Woody Plants In The United States to Herbicides	In
686	"	Effect of Paraquat and 2,4,5-T on the Uptake and Transport of Picloram in Woody Plants	"
687	"	Persistence of 2,4,5-T in Grasslands of Texas	"
688	"	Occurrence of 2,4,5-T and Picloram in Surface Runoff Water In the Blacklands of Texas	"
689	"	Occrrence of 2,4,5-T and Picloram in Subsurface Water In the Blacklands of Texas	"
690	"	Chemical Control of Mesquite	
691	9/4/80	Direct Testimony of Mr. David A. Graham	"
692	"	Memorandum of Understanding between the U.S. Department of Agriculture and the U.S. Environmental Protection Agency dtd Dec. 2, 1976, and Supplement dated Oct 14/18, 1977	"

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NO.	DATE	DESCRIPTION	STATU
693	9/4/80	USDA-Forest Service, June 11, 1980, letter to the Record "2,4,5_T Use on National Forest System Lands."	In
694	"	Report of the Forest Service, Fiscal Year 1977, USDA, Feb. 15, 1978, Part III E. Pesticide Use.	"
695	"	Summary of Phenoxy Herbicides in Water. Forest Service lter from Region 6 to Chief, June 23, 1980	"
696	"	Progress Report - Biological Assessment of Herbicide Treatment on a Pine-hardwood Ecosystem, James R. Dickson, USDA-Forest Service, 1978	"
697	"	Feeding Preferences of Deer for Selected Herbicide Treated Vegetation, Campbell, Dan L. et. al., 1980, USDA-Fish and Wildlife Service, Forest-Animal Damage Control Research Project, Olympia, Washington	"
698	"	USDA-Forest Service, June 17, 1980, letter to the Record, "1978 California 2,4,5-T/TCDD Deer Study."	"
698a	10-9-80	Memo dtd Oct. 3, 1980 to the record in re 2150 Pesticide Management and Coordination - 1978 Calif. 2,4,5-T/ TCDD Deer Study	"
699	"	Environmental Protection Agency may 4, 1977, memorandum from Edwin L. Johnson to the Dioxin Implementation Task Force with attached Dioxin: Position Document, Draft April 26, 1977, Dioxin Working Group, U.S. Environmental Protection Agency	"
699-A	"	State of California, Department of Health, Memorandum to the Record from Dwayne Reed, MD et al on "Epidemiologic Investigation of Reports of Abnormal Outcome of Pregnancies in Denny, CA," March 21, 1979	"
699B	"	University of Iowa, College of Medicine, letter from D.P. Morgan to Lee Leiserson, Feb. 20, 1979, with attached report entitled "Report on Investigation of Alleged Phenoxy Herbicide Effects On Persons Residing in Six Rivers and Trinity Forest Areas of Calif."	"
700	"	Environmental Protection Agency National Interim Primary Drinking Water Regulations 40 CFR 141; 40 CFR 59565, 12/24/75; Amended by 41 FR 28402, July 9, 1976; 44 FR 68641, Nov. 29, 1979	"
701	"	Department of Agriculture Federal Agency Plan: Certification of Applicators of Restricted-Use Pesticides. 45 FR 21703, Apr. 2, 1980	"
702	"	Forest Service Manual, Chapters 2140 and 2150, Oct. 1979	"
703	"	Forest Pest Management-Washington Office, Pesticide Incidents Report-ed Fiscal Year 1980, USDA-Forest Service	"
704	"	Forest Service letter to Chief from Region 5, "Pesticides Incidents" with enclosure, March 14, 1980	"

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NO.	DATE	DESCRIPTION	STATU
705	9-4-80	Forest Service letter from Zane Smith, Regional Forester to Chief, January 18, 1980, relating to Tordon 101R incident on the Six Rivers National Forest (2) Claim of Richard C. Overholt against Humbolt County, California with appended 12/5/79 report by Jon Resenberg, M.D. filed by Harland and Gromala, attorneys. Recieved by County Clerk on Jan. 11, 1980. (3) Forest Service ltr from Zane Smith to Chief, June 9, 1980, fowarding report on the Six Rivers National Forest Tordon 101R incident	In "
706	"	Forest Service Nov. 13, 1978, letter to Douglas M. Costle, EPA EPA's Nov. 29, 1978 response to John R. McGuire, Chief, Forest Service signed by A. E. Conroy II. Forest Service Jan. 12, 1979 letter to Douglas M. Costle, EPA.	"
707	"	Forest Service Health and Safety Code (FSH 6709.11) USDA Forest Service, June 1979, pp. 339-343.	"
708	"	Forest Service Manual, Chapter 1950, Feb. 1980	"
709	"	Forest Service Handbood 1909.15, "The NEPA Process Handbook	"
710	"	Forest Service Advisory Memorandum No. 206 Summary of correspondence received on 2,4,5-T issue, Oct. 24, 1978	"
711	"	Example letter, Private individual - H.S. Lovestead to USDA Secretary Bob Bergland, May 24, 1978	"
712	"	Example letter, non-Forest industry - Richard A. McDougal, President National Cattlemen's Association, to USDA Secretary Bob Bergland, May 18, 1978	"
713	"	Example letter, Forest Industry - Phillip D. Chadsey for the Industrial Forestry Association to USDA Secretary Bob Bergland, Apr. 27, 1978	"
714	"	Example letter, local governments - Board of County Commissioners, Douglas County, Oregon, to Rupert Cutler, assistant Director (Sec.) of Agriculture	"
715	"	Forest Service letter to the record, D.R. Hamel, June 9, 1980- Summarizes correspondence received as a result of CATH and NCAP campaign	"
716	"	National Coalition for Alternatives to Pesticides (NCAP) News, Fall 1979, page 9	"
717	"	Citizens Against Toxic Herbicides (CATH), Issue Alert (undated)	"
718	"	CATH letter to Bill Bitsas, NCAP, Oct. 10, 1979	"

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NO.	DATE	DESCRIPTION	STATU
719	9-4-80	Example letter from CATH and NCAP campaign, Jim Davis to Stuart Eizenstat, Dec. 26, 1979	"
720	"	Example reply, Forest Service ltr to Jim Davis, Feb. 7, 1980	"
721	"	Example ltr to CATH and NCAP campaign, Gary Reiss to Mr. Eizenstat received Nov. 29, 1979	"
722	"	Example reply, Forest Service letter to Gary Reiss, Dec. 13, 1979	"
723	"	Publication of final Rule, Federal, Vol. 43, NO. 175 Sept. 8, 1978" "Reservations in Authority to Apporve Herbicides." (43 FR 39953)	"
724	"	"Apply Pesticides Correctly - A guide for commercial applicators," USDA and EPA (undated)	"
725	"	Forest Service Manual, Chapter 2140, March 1978	"
726	"	Forest Service Manual, Interim Directive No. I, 2140 - Pesticide Use Management, 4/27/78	"
727	"	Forest Service Manual, Interim Directive NO. II, 2140 - Pesticide Use Management, 5/4/78	"
728	"	Forest Service Manual, Interim Directive No. III, 2140- Pesticide Use Management, 6/29/78	"
729	"	Forest Service Manual, Interim Directive No. IV, 2140-Pesticide Use Management, 9/14/78	"
730	"	Forest Service Manual, Chapter 2140, Feb. 1979	"
731	"	Forest Service Policy on Pesticide-Use and Criteria for Use of 2,4,5-T and 2,4-D, 45 FR 1650, Jan. 8, 1980, and correction, 45 FR 10824, Feb. 18, 1980	"
732	"	Forest Service letter to Regional Foresters, Station Directors, and Area Directors, Aug. 21, 1978 2,4,5-T and Related Compound Risk Information - EPA's RPAR Position Document No. I	"
733	"	Forest Service ltr to Regional Foresters, Station Directors, and Area Directors, Advisory Memorandum No. 196, July 24, 1978, 2,4,5-T and related Compound Risk Information - assessment of Hazards by Dr. Frank Dost	"
734	"	Pesticide-Use Management Activity Review, Pacific Northwest Region USDA-Forest Service, 1978	"
735	"	Forest Service Manual Chapter 1410, July 1978	

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NO.	DATE	DESCRIPTION	STATU
736	9-5-80	Direct Testimony of Dr. Roy J. Smith, Jr.	In
736-1	"		
737	"	Pay, R. Letter dated July 23, 1980, to Roy J. Smith, Jr., USDA, Stuttgart, Arkansas. In re Use of 2,4,5-T for weed control in rice in Arkansas	"
738	"	Rice Miller's Association, Rice Acreage in the United States (5-Page report presenting rice acreage statistics for 1979, published by the Rice Millers' Association, Arlington, Virginia). 1979	"
739	"	McCarty, R. letter dated July 23, 1980, to Roy J. Smith, Jr., USDA Stuttgart,Arkansas. Personal communication regarding use of 2,4,5-T for weed control in rice in Mississippi	"
740	"	Coley, J.D. letter dated August 7, 1979, to Document Control Officer EPA, Washington, D.C. Use of 2,4,5-T for weed control in rice in Mississippi	"
741	"	Coley, J.D. letter dated January 10, 1980, to Hearing Clerk, EPA Washington, D.C. Use of 2,4,5-T on rice in Mississippi	"
742	"	Coley, J.D. letter dated January 28, 1980 to Hearing Clerk, EPA Washington, D.C. Use of 2,4,5-T on rice in Mississippi	"
743	"	Ross, J.B. letter dated August 10, 1979 to Document Control Officer EPA, Washington, D.C. in re Use of 2,4,5-T for weed control in rice in Mississippi	"
744	"	Baldwin, F.L., letter dated July 23, 1980, to R.J. Smith, Jr., USDA Stuttgart, Arkansas, in re Use of 2,4,5-T for weed control in rice in Arkansas	"
745	"	Houston, D.W., letter dated July 28, 1980, to Roy J. Smith, Jr., USDA, Stuttgart, Arkansas.	"
746	"	Huey, B.A., letter dated July 21, 1980, to Roy J. Smith, Jr., USDA Stuttgart, Arkansas, in re Use of 2,4,5-T for weed control in Arkansas	"
747	"	Smith, J.E. letter dated July 25, 1980, to Roy J. Smith, Jr., USDA Stuttgart, Arkansas in re Use of 2,4,5-T for weed control in rice	"
748	"	Smith, R.J., Jr., W.T. Flinchum and D.E. Seaman in re Weed Control in U.S. Rice Production, USDA Agriculture Handbook 497 (Repository No. 442) 1977, pages 14-15, 36-42, and 45-48.	"
749	"	Arkansas Cooperative Extension Service. Recommended Chemicals for Weed and Brush Control. MP-44. Arkansas, 1980, pages 42-46.	"

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NO.	DATE	DESCRIPTION	STATI
750-	9-5-80	October 17, 1978 Memo to Charles Lewis, EPA from Roy J. Smith, Jr. Subject: Size of rice farms in 2,4,5-T use area	In
751	9-10-80	Direct Testimony of Dr. Louis F. Bouse	"
752	"	Information for 2,4,5-T Assessment Team, 1978, L.F. Bouse	"
753	"	Aerial-Spray Penetration Through Foliage Canopies, Trans. of the ASAE 12(1): 86-89, 1969, L.F. Bouse	"
754	"	Drift Comparisons of Low-Expansion Foams and Conventional Sprays. Weed Science 21(5): 405-409, 1973, L. F. Bouse and R. E. Leerskov.	"
755	"	Comparison of Recoveries from a Tower-Mounted Dual Spray. Trans. of the ASAE 18(3): 467-470, 1975, L.F. Bouse and M. G. Merkle.	"
756	"	Spray Recovery From Nozzels Designed to Reduce Drift. Weed Science 24(4): 361-365, 1976 L. F. Bouse, J.B. Carlton and M.G. Merkle. (Repository USDA No. 70)	"
757		Direct Testimony of Dr. Philip C. Kearney	
758		Pesticide Mobility In Soils II. Applications of Soil Thin-Layer Chromatography by Charles S. Helling	
759	9-15-80	Direct Testimony of Dr. Robert Alfred Squire	"
760	"	Dow TCDD Chronic Toxicity Study In Rats Tumor Incidence Summary Table	"
761	"	Histologic Typing of Liver Tumors of the Rat	"
762	9-30-80	Direct Testimony of Dr. Lennart Hardell	"
763a	"	Soft-Tissue Sarcomas and Exposure To Chemical Substances: A Case Referent Study	"
763b	"	Table: Type and duration of exposure for cases and referents exposed to Phenoxyacetic acids in Eriksson et al. 1980	"
764	"	Malignant lymphoma and exposure to chemical substances, especially organic solvents, chlorophenols and phenoxy acids. A case-control study	"
765a	"	(In Swedish) Maligna mesenkymala tumorer och exposition for fenoxisyror - en klinisk observation, Lakartidningen 74: 2753-4.	"
765b	"	Soft-Tissue Sarcomas and Exposure to Phenoxy Acids: A Clinical Observation by L. Hardell (English translation of 765a)	"

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NO	DATE	DESCRIPTION	STATUS
766	9-30-80	Malignant lymphoma of histiocytic type and exposure to phenoxyacetic acids or chlorophenols, Lancet 1 (8106): 55-56	In
767	"	"Case-Control Studies,: Chapter XII in Epidemiology: Principles and Methods, Little Brown and Co., at 256-265.	"
768a	"	Questionnaire for living subjects in Eriksson, et al.	"
768b	"	EPA translation of Eriksson et al. questionnaire	"
769	"	The Use of phenoxy herbicides in Swedish Forestry: Amounts, types and modes of application. In Ramel, C. (ed.), Chlorinated Phenoxy Acids and Their Dioxins, Ecol. Bull. (Stockholm) 27:219-230	"
770	"	Granstrom, B. 1978. The Use of phenoxy acid herbicides In Swedish agriculture. In Ramel, C. (ed.), Chlorinated Phenoxy Acids and Their Dioxins, Ecol. Bull. (Stockholm) 27:231-234	"
771	"	Zack, J.A. and R.R. Suskind 1980. The mortality Experience of workers exposed to Tetrachlorodibenzodioxin in a trichlorophenol process accident. J. Occup. Med. 22: 11.	"
772	"	Cook, R.R., J.C. Townsend, M.G. Ott, and L.G. Silverstein 1980. Mortality experience of employees exposed to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD). J. Occup. Med. (in press).	"
773	10-1-80	Direct Testimony of Dr. Richard R. Monson	"
774	"	A Mortality Analysis of Employees Engaged In The Manufacture of 2,4,5-Trichlorophenoxyacetic Acid	"
775	"	In Camera	"
776	10-7/80	Memo dtd Sept. 12, 1980 to Elizabeth Anderson, CAG from Acting, Chief Environmental Fate Branch, HED in re Exposure Assessment for 2,4,5-T, Silvex and TCDD	"
777	10-8-80	CV of William D. Schutte	"

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NO.	DATE	DESCRIPTION	STATUS
00	10-9-80	Direct Testimony of Kenneth V. Thimann	In
801	"	Molecular Model of Methane (Photo)	"
802	"	Molecular Model of Benzene (Photo)	"
803	"	Molecular Model of Acetic Acid (Photo)	"
804	"	Molecular Model of Indole-3-Acetic Acid (Auxin) (Photo)	"
805	"	Molecular Model of 2,4,5-T (Modifiable to Depict Silvex and 2,4,-D)	"
805a	"	Molecular Model of Silvex (Photo)	"
805b	"	Molecular Model of 2,4-D (Photo)	"
806	10-10-80	Direct Testimony of Dr. Robert R. Bumb	"
806A		Townsend Ratios	"
807	"	"Evaluation of the Analytical Chemical Procedures of The Dow Chemical Company for Determination of Polychlorinated Dibenzo-p-dioxins" (June 1979)	"
808	"	Molecular Model of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (Photo)	"
809	"	"The Trace Chemistries of Fire -- A Source of the Routes for the Entry of Chlorinated Dioxins into the Environment" by the Chlorinated Dioxin Task Force (With Appendices) (1978)	"
810	10-14-80	Direct Testimony of David T. Buzzelli	"
811	"	Memorandum from P. E. McGrath To Director, SPRD, in re Identification of Pesticides Possibly Contaminated With Chlorodioxins, dated Feb. 22, 1980	"
812	"	Dow 2,4,5-T & Silvex Labels	"
813	10-15-80	Direct Testimony of Professor Robert Kilpatrick, Cbe, Md, FRCP	"
814	"	"Review of the Safety for Use In The U.K. of the Herbicide 2,4,5-T" Advisory Committe on Pesticides, submitted to Ministry of Agriculture, Fisheries and Food (March 1979)	"
815	"	Herbicide 2,4,5-T, House of Commons Official Report 971 (48) at 434-35 (July 26, 1979)	"
816	"	Composition of the Advisory Committee on Pesticides (Jan. 1, 1980)	"
817	"	Government Departments, etc. Represented on the Advisory Committee on Pesticides	"

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NO.	DATE	DESCRIPTION	STAT
818	10-15-80	"The Control Scheme: The Pesticides Safety Precautions Scheme (PSPS)," extract from the Royal Commission on Environmental Pollution Report at 61 (Sept. 1979)	"
819	"	United Kingdom Official Recommendations for Sale of 2,4,5-T and Fenoprop (Silvex):	"
819a	"	Ministry of Agriculture, Fisheries and Food, Pesticides Branch, Chemical Compounds Used In Agriculture and Food Storage: Recommendations for Safe Use In Great Britain, 2,4,5-T" Sheet 168 (Jan. 24, 1967)	"
819b	"	Ministry of Agriculture, Fisheries and Food, Pesticides Branch "Chemical Compounds Used as Pesticides: Recommendations for Safe Use In the U.K., 2,4,5-T" Sheet 1430 (March 14, 1980)	"
819c	"	Ministry of Agriculture, Fisheries and Food, Pesticides Branch, "Chemical Compounds Used As Pesticides (Use In Forestry): Recommendations for Safe Use In the U.K., 2,4,5-T," Sheet 1443 March 14, 1980	"
819d	"	Ministry of Agriculture, Fisheries and Food, Pesticides Branch, "Chemical Compounds Used In Agriculture and Food Storage (Home Garden Use)" Recommendations for Safe Use In the U.K, Fenoprop," Sheet 804 (June 1, 1971)	"
819e	"	Ministry of Agriculture, Fisheries and Food, Pesticides Branch "Chemical Compounds Used in Agriculture and Food Storage (Agricultural and Horticultural Use): Recommendations for Safe Use in the U.K., Fenoprop," Sheet 972 (Dec. 1, 1972)	"
820	"	Anonymous, "2,4,5-T-Where Next? The Lancet at 1114-15 (Nov. 24, 1979)	"
821	"	The Safety of Herbicides 2,4-D and 2,4,5-T, Turner, D.J., Forestry Commission Bulletin 57 (1977)	"
822	10-16-80	Direct Testimony of Dr. Richard J. Kociba	"
823	"	Historical Control Data	"
824	"	Letter to Mr. J. Guy Gwenne dtd Jan. 17, 1980 from Professor Dr. F. Leuschner	"
825	"	2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD): Results of a 13-week Oral Toxicity Study in Rats	"
826	"	Evaluation of Carcinogenic, Teragenic, and Mutagenic Activities of Selected Pesticides and Industrial Chemicals, Vol. 1, Carcinogenic Study, Bionetic Research Labs., Inc., Prepared for NCI - Aug. 1968	"

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NO.	DATE	DESCRIPTION	STATU
827a	10-16-80	Chronic Oral Toxicity of 2,4,5-T, Batch No. 503, Control No. 15374 b - Called for Short '2,4,5-T' - In Sprague-Dawley (Siv 50) Rats - Repeated dosage over 130 weeks) (With Special attention to Carcinogenic Properties) Apr. 9, 1979 (Expando)	In
827b	"	Chronic Oral Toxicity of 2,4,5-T, Batch No. 503, Control No. 15374 b - Called for short '2,4,5-T - In Sprague-Dawley (Siv 50) Rats - Repeated dosage over 130 weeks)(With special attention to carcinogenic properties) Apr. 9, 1979 (Expando)	"
827c	"	Chronic Oral Toxicity of 2,4,5-T, Batch No. 503, Control No. 153574 b - Called for short '2,4,5-T - In Sprague-Dawley (Siv 50) Rats - Repeated dosage over 130 weeks - (With special attention to carcinogenic properties) Apr. 9, 1979 (Expando)	"
828	"	Results of Two Year Dietary Feeding Study In Rats of Kurosal Sl (Potassium Salt of Silvex)	"
829	10-17-80	Direct Testimony of Dr. Francis J.C. Roe	"
830	"	Strategy for detection of cancer hazards to Man , Sir Richard Doll	"
831	"	Mutagenic Principle(s) in Tryptophan and Phenylalanine Pyrolysis Products	"
832	"	Mutagen-carcinogens in Food, with Special Reference to Highly Mutagenic Pyrolytic Products In Broiled Foods	"
833	"	Isolation and Structure Determination of Mutagenic Substances in L. Glutamic Acid Pyrolysate	"
834	"	Cancer and Environment: Higginson Speaks Out	"
835	"	Recent Developments In the Design of Carcinogenicity Tests on Laboratory Animals	"
836	"	Effect of Quality and Quanity of Diet On Survival and Tumour Incidence in Outbred Swiss Mice	"
837	"	The Effect of Long-Term Food Restriction On Tumors In Rodents	"
838	"	Aetiology of Breast Cancer: A Brief Review	"
839	"	Food and Cancer	"
840	"	Are Nutritionist worries about the epidemic of Tumours In Laboratory Animals? F.J.C. Roe	"

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NO.	DATE	DESCRIPTION	STATU
841	10-17-80	Effects of Pretreatment with 2,3,7,8-Tetrachlorodibenzo-p-Dioxin on the Capacity of Hepatic and Extrahepatic Mouse Tissues to Convert Procarcinogens to Mutagens for Salmonella Typhimurium Auxotrops	In
842	10-22-80	Direct Testimony of Dr. James G. Wilson	"
842a	"	Chart (Response)	"
843	"	Report of the Advisory Committe on 2,4,5-T to The Administrator of the Environmental Protection Agency, submitted May 7, 1971	"
844	"	Report on the Treatment of Pregnant Rhesus Monkeys with 2,4,5-T Acid	"
845	10-28-80	Direct Testimony of Dr. Bernard A. Schwetz	"
846	"	Toxicol. Appl. Pharmocol. 22:317 (Abstract) Teratogenic Effects of 2,4,5-T in Mice	"
847	"	Investigations on the Embryotic Effect of 2,4,5-T In NMRI-Mice	"
848	"	Nonteratogenic Effects of 2,4,5-Trichlorophenoxyacetic Acid and 2,4,50T propylene glycol butal esters herbicides in sheep	"
849	"	A Quality Assurance Review of Dr. Allen's 500 PPT TCDD Monkey Study dated May 8, 1980	"
850	"	Evaluation of 2,4-dichlorophenoxyacetic acid (2,4-D), 2,4,5-Trichlorophenoxyacetic acid (2,4,5-T), and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity in C57BL/6 mice	"
851	10-30-80	Direct Testimony of Dr. Colin N. Park	"
852	"	Direct Testimony of Dr. E. Marshall Johnson	"
853	11-26-80	Direct Testimony of Dr. Steven H. Lamm	"
854	10-29-80	Direct Testimony of Dr. E. G. McQueen	"
855	10-31-80	Direct Testimony of Dr. Sheldon L. Wagner	"
856	"	Direct Testimony of Dr. Alan Agresti	"
857	11-4-80	Direct Testimony of Michel A. Ibrahim, M.D., Ph.D.	"
858	"	The Case-Control Study Consensus and Controversy	"

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NO.	DATE	DESCRIPTION	STA
859	11-5-80	Direct Testimony of Dr. Ralph R. Cook	I
860	11-7-80	Direct Testimony of Dr. Philip Cole	"
860a & b		Chart	
861	11-10-80	Direct Testimony of Dr. G. Reggiani	"
861a	"	Review Article - Medical Problems Raised by the TCDD Contamination in Seveso, Italy, G. Reggiani	"
861b	"	Valutazione Quantitativa Della Esposizione Umana Della TCDD Nelle Zone Di Seveso	"
861c	"	Map - Location of Contaminated Area	"
862		Letter to The Health Officer of Seveso and Meda	"
863		Acute Human Exposure To TCDD In Seveso, Italy, by G. Reggiani	"
864	11-13-80	Direct Testimony of Dr. H. Tuchmann-Duplessis	"
864a	"	Frequency of Abortions Broken Down by Degree of Contamination	"
864b	"	Hand drawn document (diagram)	
865	"	Direct Testimony of Dr. V.K. Rowe	"
866	"	Research on Chemical Acnegens Using Human Subjects	"
867	11-17-80	Direct Testimony of Dr. Kenneth D. Crow	"
	"		"
867a	"	Ltr to K.D. Crow, FRCP dtd June 21, 1978 from P.J. Gehring	"
868	"	Chloraae: The Chemical Disease	"
869	"	Chloracne from the accidental production of tetrachloridebenzodioxin	"
870	11-18-80	Direct Testiomony of Dr. Otto Hutzinger	"
871	"	Workshop - Impact of Chlorinated Dioxins and Related Compounds On The Environment, Oct. 22-24, 1980	"
872	11-19-80	Direct Testimony of Dr. Ralph T. Ross	"
873	"	Preliminary Dioxin Implementation Plan	"
874	"	Critique of Segments of Letters Whick Relate To The Adequacy of Analytical Methods for TCDD In Environmental Samples	"

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O.	DATE	DESCRIPTION	STA
875	11-20-80	Direct Testimony of Dr. Ford L. Baldwin	In
876a	"	Photo - View of Flooded Rice Field and Levees at the Early Tillering Stage	"
876b	"	Photo- View of Rice Field with Early Season Weed Problem	"
876c	"	Photo- Draining of Rice Fiel in Preparation for Early Season Application of 2,4,5-T	"
876d	"	Photo- View of Rice Field Following Early Season Application of 2,4,5-T	"
876e	"	Photo - View of Rice Field at Midseason, Just Prior to Time of Midseason Application of 2,4,5-T.	"
877f	"	Photo - Typical Broadleaf Weed Problem--Curly Indigo	"
876g	"	Photo - Typical Broadleaf Weed Problem -- Morning Glory	"
°76h	"	Photo - Base of Rice Stems Showing Panicle Initiation and Internode Elongation	"
76i	"	Photo - Typical Computer Print-Out Showing Appropriate 2,4,5-T Midseason Application Dates	"
876j	"	2,4,5-T Treatment of Levees.	"
876k	"	View of Levee Following Application of 2,4,5-T	"
876L	"	Photo - Aerial Application of 2,4,5-T	"
876m	"	Photo - View of Rice Plant Showing Phenoxy Herbicide Damage	"
876n	"	Photo - View of Rice Field Showing "Heading" Stage	"
876o	"	Photo - Rice Field Containing Mature Rice	"
876p	"	Photo - Rice Panicle Containing Rice Hulls	"
876q	"	Photo - Rough Rice	"
876r	"	Photo - Rice Elevator, and Part of Rice Drying and Storage Facility	"
876s	"	Photo - Dumping Harvested Rice at the Drying and Storage Facility	"
76t	"	Photo - Final, Polished White Rice Kernals	"

U.S. ENVIRONMENTAL PROTECTION AGENCY
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	DATE	DESCRIPTION	STAT
877	11-21-80	Direct Testimony of John L. Merrill	In
878	"	Livestock and Meat Situation - Aug. 1980	"
879	"	Bovine Growth and Composition by H.B. Hedrick	"
880	"	Technical Standard and Specifications for Deferred Grazing	"
881	"	Livestock Slaughter Annual Summary 1979	"
882	"	Meat Animals Production Disposition, Income 1978-1979	"
883	"	Money Value of Food Used By Households In The United States, Spring 1977	"
884	"	Increasing Forage on Ozark Wooded Range, John H. Ehrenreich & Robert F. Butter	"
885a	"	Photo- Cow with Newborn Calf	"
885b	"	Photo- Cows with Calves Near Weaning Time	"
885c	"	Photo - Weaned Calves on Wheat Pasture	"
885d	"	Photo - Yearlings in Feedlot	"
885e	"	Photo - Cattle On Dormant Range Receiving Supplemental Feed	"
885f	"	Photo - Cattle Receiving Hay When Snow Covers Grass	"
885g	"	Photo - Nutrient Uses of Cattle	"
885h	"	Photo - Body Composition of Cattle	"
885i	"	Photo - Brush Root Growth	"
885j	"	Photo - Brush Use of Moisture	"
885k	"	Photo - Brush Competition With Grass	"
885L	"	Photo - Mesquite Infestation in Texas	"
885m	"	Photo - Untreated Mesquite in Willacy County, Texas	"
885n	"	Photo - Untreated Mesquite in DeWitt-Gonzales County Area, Texas	"

U.S. ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STA
885o	11-21-80	Photo - Treated Mesquite in Oldham County, Texas	In
885p	"	" Treated Running Mesquite In McMullen County, Texas	"
885q	"	" Untreated Mesquite In the rolling Plains of Texas	"
885r	"	" Regrowth Mesquite in Knox County, Texas	"
885s	"	" Untreated Running Mesquite in McMullen County, Texas	"
885t	"	" Non-infested Rangeland in Wheeler County, Texas	"
885u	"	" Post and Blackjack Oak Infestation in Texas	"
885v	"	" Untreated (left and Treated (Right) Post Oak and Blackjack Oak Infestation Near Bryan, Texas	"
885w	"	" Sand Shinnery Oak Infestation in Texas	"
885x	"	" Untreated Sand Shinnery Oak Infestation in the Eastern Panhandle of Texas	"
885y	"	" Cactus Infestation in Texas	"
5z	"	" Cactus and Mesquite Competition with Grass on Texas Range	"
886	11-23-80	Direct Testimony of Dr. Lewis A. Shadoff	"
887	"	Interlaboratory Method Validation Study for Dioxin an interim Report	"
888	"	Environmental Samples of Selected Methods Development	"
889	12-3-80	Direct Testimony of Dr. Henry Freiser	"
890	12-4-80	Direct Testimony of Hyland R. Johns	"
891	"	Asplundh Chemical Foreman's Manual	"
892	12-8-80	Direct Testimony of Dr. John C. Ramsey	"
893	"	The Fate of 2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T) Following Oral Administration to Rats and Dogs	"
894	"	Pharamacokinetics of 2,4,5-T PGBE Ester Applied Dermally to Rats	"
895	"	Determination of 2,4,-D Exposure Received by Forestry Applicators Spring 1980	"

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U.S. ENVIRONMENTAL PROTECTION AGENCY EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
896	12-9-80	Direct Testimony of Dr. Richard Wilson	In
896a	"	Interspecies Comparison of Carcinogenic Potency, Edmund Crouch, & Richard Wilson	"
897	12-11-80	Direct Testimony of Dr. Michael Newton	"
898	"	The Phenoxy Herbicides (Second Edition) Aug. 1978	"
899	"	Polution Control In The Forest Products Industry	"
900	"	Letter to Arvid C. Ellson, Portland,Oregon dtd Nov. 17, 1978 from William D. Clothier	"
901	"	Exposure of Forest Herbivores to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) In Areas Sprayed with 2,4,5-T	"
902a		Chart	
902	12-12-80	Direct Testimony of Werner H. Braun	"
903a	"	Photograph of 2,4,5-T Applicator Study - Backpack Crew	"
903b	"	" - Backpack Loading	"
903c	"	" - Backpack Sprayer's Truck	"
903d	"	" - Backpack Sprayer	"
903e	"	" - Backpack Sprayer	"
903f	"	" - Tractor Sprayer	"
903g	"	" - Mixing for Tractors	"
903h.	"	" - Loading the Tractor	"
903i	"	" - Flagmen	"
903j	"	" - Loading the Helicopter	"
903k	"	" - Flagman's View	"
904a	"	Photographs of 2,4-D Applicator Study - 2,4-D Application Area	"
904b	"	" - Aerial Crew	"
904c	"	" - Batchman Loader	"
904d	"	" - Pilot's Boot	"
904e	"	" - Leather Flying Gloves	"
904f	"	" - Protective Clothing	"

U.S. ENVIRONMENTAL PROTECTION AGENCY
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NO.	DATE	DESCRIPTION	STA
905	12-16-80	Direct Testimony of Philip G. Watanabe	In
905a		Chart	
905b		Chart (reversible)	
906	"	"The Application of Fundamentals In Risk Assessment", Paper delivered at American Chemical Society Symposium of Pesticide Chemist and Modern Toxicology, (June, 1980)	"
907	"	Cornfield, J., "Carcinogenic Risk Assessment," Science, 198 at 693-699 (Nov. 18, 1977).	"
908	"	Gehring, P.J. and Blau, G.E., "Mechanisms of Carcinogenesis: Dose Response," J. Environ. Pathol. Toxicol., 1 at 163-179 (1977)	"
909	"	Squire, Robert A., "Human Risk Assessment from Animal Data," Paper Delivered at American Chemical Society Symposium on Pesticide Chemist and Modern Toxicology, (June, 1980)	"
910	"	Pitot, H.C., et al., "Quantitative Evaluation of the Promotion by 2,3,7,8-Tetrachlorodibenzo-p-dioxin of Hepatocarcinogenesis from Diethylnitrosamine,"Cancer Research, 40 at 3613-3620 (Oct., 1980)	"
911	"	Poland, A. and Glover, C., "An Estimate of the Maximum in Vivo Covalent Binding of 2,3,7,8-Tetrachloro-dibenzo-p-dioxin to Rat Liver Protein, Ribosomal RNA, and DNA," Cancer Research, 39 at 3341-3344 (Sept. 1979)	"
912	12-17-80	Direct Testimony of Dr. Perry J. Gehring	"
913	"	General Electric Comments on IRLG Report	"
914	"	FDA, 21 C.F.R. 170.22	"
915	"	FDA, "Caffeine/ Deletion of Gras Status, Proposed Declaration That No Prior Sanction Exists, and Use on an Interim Basis Pending Additional Study," 45 Fed. Reg. 69817-38 (Oct. 21, 1980)	"
916	"	Collins, T.F.X., et al., " A Comprehensive Study of the Teratogenic	"
917	"	FDA, "Selenium in Animal Feed," 38 Fed. Reg. 10458-60 (Apr. 27, 1973)	"
918	"	Sauerhoff, M.W., et al., "Dose-Dependent Pharmacokinetic Profile of Silvex Following Intravenous Administration in Rats," J. Toxicol. Environ. Health 2:605-618 (1977)	"

U.S. ENVIRONMENTAL PROTECTION AGENCY
EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
920			
921			
922			
923			
924			
925	2-11-81	Testimony of the American Council on Science & Health	In
926	"	American Council on Science and Health Preliminary Report on 2,4,5-T	"

U.S. ENVIRONMENTAL PROTECTION AGENCY EXHIBITS

NO.	DATE	DESCRIPTION	STATUS
930	1-27-81	Direct Testimony of Dr. Michael L. Gross	In
931	"	Letter to Mr. Michael Dellarco dated Oct. 2, 1980 from Michael L. Gross enclosing Data Report XIII	"
932	"	Memo to Mike Dellarco dated Sept. 18, 1980 from Robert Harless in re Results of Capillary Colum GC-HRMS Analyses Performed on Extracts of Human Adipose for 2,3,7,8-TCDD Residues	"
933			
934		** Direct Testimony of Dr. Renate D. Kimbrough	"
934a		** Centers For Disease Control's Human Health Assessment Approach Involving Improperly Disposed Chemical Wastes	"
935	1-30-81	Direct Testimony of Joseph S. Carra	"
936	2-4-81	Direct Testimony of Dr. John C. Bailar, III	"
937	"	Memo to Diana Reisa, Ph.D. dated Jan. 26, 1979 from H.W. Spencer, Ph.D. in re Report of TDA review of TCDD 2, year rat feeding study by Dow Chemical Company	"
938	2-3-81	Direct Testimony of Mr. Robert G. Heath	"
939	"	Edwin L. Johnson, Letter to Blue Ball Construction Company, dated November 27, 1979 (w/responses)	"
940	"	Lincoln County Road Department, Maintenance Division, Daily Spray Report dtd 1974	"
941	"	Lincoln County Road Department, Maintenance Division, Daily Spray Reports (1975)	"
942	"	Amchem Products, Inc., Label for Weedon 2,4,5-T	"
943	"	Township and Range Map of Lincoln County, Oregon (with Zip Code Zones)	"
944		**Direct Testimony of Mr. Gerald Hall	
945a		**Annual Precipitation Summary - 1963-1979, Source: Department of Commerce, N.O.A.A. National Climatic Center, Asheville, N.C.	"
945b		**Daily Precipitation Summary - Tidewater, Oregon (Lincoln) July 1, 1979 to June 30, 1980	"
945c		**Isohyetal map of Lincoln County - Source: Frank and Laenen; Water Resources of Lincoln County Coastal Area, Oregon, U.S.G.S. 1977	"

**/Admitted by Order Admitting Exhibits dated Feb. 26, 1981

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U.S. ENVIRONMENTAL PROTECTION AGENCY EXHIBITS

.O.	DATE		DESCRIPTION	STATI
45d		**	Mean monthly discharge of Siletz and Alsea Rivers. Source: IBID	In
945e		**	Relationship between precipitation and water well levels,Source Ibid	"
945f		**	Selected USGS gaging stations and average monthly flows, Source Ibid	"
946a		**	Lincoln County, Oregon - base map 1"= 1 mile - problem area	"
946b		**	Appendix- Index of River Basins and Sub Basins, Lincoln County, 1979	"
946c		**	Goal 6 - Air, Water and Land Resources Quality - Inventory Maps	"
947a		**	Lincoln County, Oregon - two Cross Sections from aerial Photo soils mapping	"
947b		**	USDA Soil Conservation Service or Soils - 1 Index of Soils from Cross Sections	"
947c		**	Identification Legend - Mapping symbols for mapping units	"
948		**	High Altitude (U-2) aerial photo of Alsea Bay, southern portion of Lincoln County and Northwesternmost Lane County,Oregon 1:32,500	"
949		**	Photo enlarged and spliced U.S.G.S. quadrangle Map of southeastern Lincoln County, Oregon 4" - 1 Mile (1 copy in the Original File)	"
950		**	Air Photo of unicorporated Lincoln County, Oregon, south of Lincoln City and Siletz Bay 1" = 400 miles (1 Copy in the Original file)	"
951		**	Location map for map exhibits, Lincoln County, Oregon	"
952a	2-3-81		Map of principal domestic water rights by approximate point diversion. Lincoln County Applications(large copy not in folders)	"
952b		**	Appendix to water rights map number 952 above	"
952c		**	Water rights filings for all Lincoln County Applications	"
952d		**	Township and Range mapping of approximate points of diversion for all Lincoln County water rights applications	"
952e		**	Index of valid water rights by Township, range and Section for all Lincoln County water rights	"

**/ Admitted by Order Admitting Exhibits dated Feb. 26, 1981

Ex. E-2

RESULTS OF A TWO-YEAR CHRONIC TOXICITY AND ONCOGENIC STUDY OF RATS INGESTING DIETS CONTAINING 2,4,5-TRICHLOROPHENOXY-ACETIC ACID (2,4,5-T)

RECEIVED
NOV 30 1978

By

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September 27, 1978

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#16 L [30000/26]
attachments

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SUMMARY

Groups of Sprague-Dawley rats were maintained on diets supplying 30, 10 or 3 mg of 2,4,5-T/kg body weight/day for up to 2 years, with an interim necropsy after 118-119 days. The highest dose level of 30 mg of 2,4,5-T/kg/day was associated with some degree of toxicity. This included a decreased body weight gain, increased relative kidney weights, increased urinary excretion of total urine volume, coproporphyrin and uroporphyrin plus slight morphologic alterations of the kidney, liver and lungs. In the kidney, this involved primarily the presence of mineralized deposits in the renal pelvis. Liver effects associated with the highest dose level of 30 mg of 2,4,5-T/kg/day included an increased incidence of focal biliary hyperplasia and inflammatory changes plus decreased incidence of hepatocellular degenerative changes. The incidence of focal pulmonary inflammatory changes, primarily accumulation of alveolar macrophages, was also increased at this highest dose level.

Parameters unaffected by treatment with this highest dose level of 30 mg 2,4,5-T/kg/day included the rate of mortality, food consumption, palpable masses, hematology (red blood cell count, hemoglobin, packed cell volume, total and differential white blood cell count, thrombocytes and reticulocytes), routine urinalyses, urinary excretion of creatinine or delta-aminolevulinic acid, serum clinical chemistry (urea nitrogen, glutamic pyruvic transaminase and alkaline phosphatase activities, bilirubin, total protein, albumin and globulin), organ weights (except

for kidneys as described above), tumor incidence as well as gross and microscopic morphology of all organ systems of the body (except as described above).

At the intermediate dose level of 10 mg of 2,4,5-T/kg/day, there were only minimal effects, limited primarily to an increased incidence of mineralized deposits in the renal pelvis; an increase in urinary excretion of coproporphyrin was noted in males only during the early phase of the study. At the lower dose level of 3 mg of 2,4,5-T/kg/day, there were no alterations in any parameter considered related to treatment over the course of this 2-year study.

Thus, the results of this study revealed no oncogenic response in rats, even when administered a sufficiently high dosage of 2,4,5-T to induce toxicity.

INTRODUCTION

2,4,5-Trichlorophenoxyacetic acid (or 2,4,5-T) has been used as a selective herbicide for weed control on certain crops, pastures, grazelands and forests. In the production of 2,4,5-T, the starting material for the aromatic portion of the compound is tetrachlorobenzene. Tetrachlorobenzene is hydrolyzed to 2,4,5-trichlorophenol, which is subsequently reacted with chloroacetic acid to form 2,4,5-T. In the production of the trichlorophenol, several impurities may be formed under certain unfavorable conditions. One of these impurities is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) which may be found at low levels in the trichlorophenol. In 1966 The Dow Chemical Company imposed upon itself a manufacturing specification of less than 1 ppm TCDD in 2,4,5-T. This has been subsequently lowered to 0.1 ppm maximum level of TCDD in 2,4,5-T.

Various toxicologic studies have been conducted with 2,4,5-T over the past 25 years. The single oral dose LD50 of 2,4,5-T has been reported to be 389 mg/kg in mice, 381 mg/kg in guinea pigs, 500 mg/kg in rats and 310 mg/kg in chicks (Rowe and Hymas, 1954). In dogs, the single oral LD50 of 100 mg/kg reported by Drill and Hiratzka (1953) is probably due to the slower rate of urinary elimination of organic acids such as 2,4,5-T in dogs (Piper et al., 1973).

The compound 2,4,5-T is readily absorbed and rapidly excreted by animals, including man. The half-life values for the clearance of carbon-14 activity from the plasma of rats given single oral doses of labeled

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2,4,5-T at doses of 4, 50, 100 and 200 mg/kg were 4.7, 4.2, 19.4 and 25.2 hours, respectively (Piper et al., 1973). The half-lives for elimination from the body of those rats were 13.6, 13.1, 19.3 and 28.9 hours, respectively. In dogs given C^{14} labeled 2,4,5-T at 5 mg/kg, half-life values for clearance from the plasma and clearance from the body were 77.0 and 86.6 hours, respectively. This lower rate of clearance may explain the greater toxicity of 2,4,5-T in dogs than in rats. Five human volunteers ingested 5 mg/kg of 2,4,5-T containing less than 0.05 ppm TCDD (Gehring et al., 1973). Essentially all the 2,4,5-T was absorbed and excreted unchanged in urine, with a half-life of 23.1 hours. This rate of excretion more closely resembles that observed in the rat than the dog.

In a previous unpublished subchronic toxicity study of 2,4,5-T in rats conducted by this laboratory, no evidence of adverse effects was observed in rats fed dose levels of 3 or 10 mg/kg/day for 90 days. At higher dose levels of 30 or 100 mg/kg/day, there were increases in liver and kidney weights, elevations in serum enzymes, and other indications of slight toxicity. Drill and Hiratzka (1953) found no adverse effects in dogs given 2.5 or 10 mg/kg of 2,4,5-T for 5 days/week for 3 months; four dogs could not tolerate a higher dose level of 20 mg/kg and died during the experiment.

Highman et al. (1976) reported results of short-term toxicity studies in mice using samples of 2,4,5-T with varying content of TCDD. These

studies, which were in conjunction with teratology studies, reported multiple toxicologic effects due to treatment with high doses of 120 or 60 mg of 2,4,5-T/kg; lower dose levels were not studied.

Smith et al. (1978) recently have reported on a 3-generation reproduction study of rats ingesting 2,4,5-T. No adverse effects were seen at a dose level of 3 mg/kg/day. Dose levels of 2,4,5-T which were sufficiently high to cause signs of toxicity had no effect on reproductive capacity of rats except for a tendency toward a reduction in neonatal survival at dose levels of 10 and 30 mg/kg/day.

There have been several studies conducted over the past 10 years in mice to assess the potential oncogenicity of 2,4,5-T. Innes et al. (1969) reported no increase in the incidence of tumors in 2 strains of mice given the maximum tolerated dose (21.5 mg/kg/day) of 2,4,5-T (containing approximately 30 ppm of TCDD) via stomach tube from day 7 to day 28 of age. After weaning at 28 days of age, 60 ppm of 2,4,5-T was mixed directly in the diet for approximately 18 months. In a National Cancer Institute study conducted at Bionetics Research Laboratories (1968) but never published, weanling mice (2 strains) were given by single subcutaneous injection a dose of 215 mg/kg, with subsequent observation for 18 months. The authors concluded there was no increase in the incidence of tumors due to 2,4,5-T.

In 1976, Muranyi-Kovacs et al. published the results of some of their studies of 2,4,5-T in 2 strains of mice. This initial publication

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reported results of their study of 2 inbred strains of mice (C3Hf and XVIIG) given 2,4,5-T (100 mg/l) in their drinking water for 2 months beginning at 6 weeks of age. The mice were subsequently fed diets containing 80 ppm (approximately 12 mg/kg/day) of 2,4,5-T for the duration of the lifetime study. Based on their method of combining tumor types, the authors reported a statistically significant increase in tumor incidence of C3Hf mice. No significant differences in tumor incidence were found in the mice of the XVIIG strain. In a subsequent communication, Muranyi-Kovacs et al. (1977) reported on the other studies they had conducted in which the same 2 strains of mice as used in their diet study were given a series of neonatal subcutaneous injections of 2,4,5-T with no resultant increase in tumors.

In order to evaluate the long-term toxicity and oncogenic potential of 2,4,5-T in rats, the study reported herein was conducted. This study involved the dietary administration of 30, 10 or 3 mg of 2,4,5-T/kg/day to rats for up to 2 years.

MATERIALS AND METHODS

Experimental Design. Two groups of male and female Sprague-Dawley rats, Spartan substrain[1], 7-8 weeks old, were randomly placed (2/cage) in suspended wire-bottomed cages to be used for a 118-119 day and a 2-year chronic toxicity study. Animals were randomized using a table of random numbers. Individual rats were identified by metal ear tags; rats of each dose level were also identified by toe-clipping. Food[2] and water were available *ad libitum*. On March 5, 1975, the rats were placed on diets providing 2,4,5-T according to the following:

2,4,5-T mg/kg/day	118-119 Day Study Initial No. of Rats		2-Year Study Initial No. of Rats	
	Male	Female	Male	Female
0	10	10	86	86
30	10	10	50	50
10	10	10	50	50
3	10	10	50	50

Test Material. The purified sample of 2,4,5-T used for this two-year study was supplied by M. Getzendaner of The Dow Chemical Company and was designated as Sample AGR133711. The identity of the 2,4,5-T acid material was verified prior to study initiation by elemental analysis, gas chromatography, solid probe mass spectrometry, gas chromatography-mass spectrometry, nuclear magnetic resonance spectrometry and infrared spectrometry. Within the limits of precision of each of these techniques, the material was essentially that of 2,4,5-trichlorophenoxyacetic acid. Assay via titration

[1]Spartan Research Animals, Haslett, MI.

[2]Purina Laboratory Chow, Ralston-Purina Co., St. Louis, MO.

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indicated approximately 99% 2,4,5-trichlorophenoxyacetic acid. Other phenoxy acid impurities amounted to 1.3 weight percent as determined by gas chromatography. Using gas chromatography-mass spectrometry, 2,3,7,8-tetrachlorodibenzo-p-dioxin, hexachlorodibenzo-p-dioxin, heptachlorodibenzo-p-dioxin and octachlorodibenzo-p-dioxin were not detected at limits of detection of 0.33 ppb, 0.12 ppb, 0.40 ppb and 0.40 ppb, respectively. The recrystallization solvent, tetrachloroethylene, was present at 0.22%. Table 1 lists the specific analytical data.

Diet Preparation. This sample was used to prepare the premixes and test diets according to the following general procedure:

> A 1% premix was prepared by dissolving an appropriate amount of 2,4,5-T in reagent grade acetone and thoroughly mixing it with ground laboratory chow. This premix, after drying, was mixed with rat chow to prepare the diets that supplied 30, 10 and 3 mg 2,4,5-T/kg body weight/day. The body weights and food consumption of 20 rats/sex/group were used as the basis for preparing these diets. Control rats were supplied with ground laboratory chow treated with an equivalent amount of acetone. Portions of each prepared diet from each dose level collected during months 5, 7, 12, 19 and 23 were submitted for 2,4,5-T analysis.

Clinical Observations. Rats were observed for general health status and possible toxicologic response during the study, with observations

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recorded at the time of each monthly weighing of the rats. Beginning at 6 months, observations were also recorded at the time of each monthly palpation for the detection of palpable masses.

Body weights were recorded at approximate weekly intervals for all 10 rats/sex/group killed after 118-119 days and also for 20 rats/sex/group during the first 3 months of the two-year portion of the study. Thereafter, body weights of rats used in the 2-year study were recorded at approximate monthly intervals.

Food consumption was recorded at approximate semi-weekly intervals for all 10 rats/sex/group scheduled for the interim kill after 118-119 days and also for 20 rats/sex/group during the first 4 months of the two-year portion of the study. Thereafter, food consumption was recorded for up to 20 rats/sex/group for 1 week/month during the rest of the two-year study.

Blood samples for hematological determinations were collected from the tail veins of 8 rats/sex/group after 82-83 days of treatment from the group scheduled for the interim kill after 118-119 days. From the rats on the two-year study, blood samples were also collected from 8 rats/sex/group after 89-90, 364-365, and 726-727 days of treatment. The total erythrocyte count (RBC), total and differential leukocyte counts (WBC), thrombocyte and reticulocyte counts, packed cell volume (PCV), and hemoglobin (Hgb) concentration were determined using automated[3] or manual procedures.

[3]Coulter Counter Model ZBI, Coulter Electronics, Hileah, Florida.

Urine samples were collected from 8 rats/sex/group after 82-83 days of treatment of the group scheduled for the interim kill at 118-119 days. The same number of rats from the two-year study were sampled after 89-90, 364-365, and 726-727 days of treatment (when group sizes permitted).

Urine specific gravity, pH, and the presence or absence of glucose, protein, ketones, bilirubin and occult blood were determined[4] at each of these times, and urinary urobilinogen[4] was also evaluated on days 364-365 and 726-727 of treatment.

Urinary levels of creatinine, coproporphyrin, uroporphyrin with or without delta-aminolevulinic acid (delta-ALA) were determined by a consulting laboratory[5]. Rats were housed individually in metabolism cages for collection of 24 or 48 hour urine samples in glass jars wrapped in aluminum foil and packed in dry ice. Urine samples of 5 rats/sex/group were collected from the group scheduled for the interim kill at 118-119 days on three occasions before day 106 of treatment and once just prior to the interim kill. Urine samples of 4-5 rats/sex/group were also collected after 95-97, 188-190, 361-363, 564-566, and 698-700 days of the two-year period of treatment. Urine volumes were recorded for each individual rat.

At both the interim necropsy after 118-119 days and the terminal necropsy after 2 years, serum samples were collected from a maximum of 10 rats/sex/group for the determination of blood urea nitrogen (BUN), serum glutamic

[4]Ames Bililabstix or Multistix, Ames Co., Elkhart, IN, and TS Meter, AO Optical, Buffalo, NY.

[5]Bio-Science Laboratories, Van Nuys, CA.

pyruvic transaminase (SGPT), alkaline phosphatase (AP), total direct and indirect bilirubin, total protein, albumin, and globulin. Automated procedures were used for these determinations[6].

Necropsy Examination. An interim necropsy examination was conducted after 118-119 days of treatment of 10 rats/sex/group that had been pre-designated for this purpose. All rats were deprived of food overnight prior to killing by decapitation. The eyes of all rats were examined by gently pressing a glass slide against the cornea under bright fluorescent illumination. Any observations on the eyes were recorded as part of the gross necropsy observation records. The eyes for a maximum of 5 rats/sex/group were preserved in Zenker's fixative. Eyes from the remaining rats were fixed in 10% buffered formalin. A complete gross pathologic examination was performed by a veterinary pathologist. Representative sections of all major organs and tissues were preserved in formalin fixative. These tissues included liver, kidneys, heart, pancreas, spleen, brain (cerebrum, cerebellum and brain stem), spinal cord, peripheral (sciatic) nerve, pituitary gland, stomach, small intestine, large intestine, mesenteric lymph node(s), skeletal (thigh) muscle, salivary gland, testes, epididymides, accessory male sex glands, urinary bladder, uterus, ovary, trachea, esophagus, aorta, thoracic lymph node(s), thymus, lungs, bronchi, integument, thyroid gland, parathyroid glands, adipose tissue, eyes, adrenal gland(s), and any other grossly observed lesion. The weights of the liver, kidneys, brain, heart, thymus, spleen, testes and ovaries/uterus (as a unit) were recorded on all 10 rats/sex/group.

[6]Technicon AutoAnalyzer, Technicon Corporation, Rye, NY.

All rats dying or culled during the course of the 2-year study were also subjected to a gross pathologic examination. Representative portions of all major organs and tissues as listed above, plus tongue, mandible, skull (including nasal turbinates, ear canal, etc.) along with any gross lesions suggestive of a significant pathologic process or tumor formation were collected from each rat and preserved in formalin fixative.

Terminal necropsy examination was conducted on all survivors at the end of 2 years of treatment, (March 8-11, 1977) using procedures similar to that described for the interim necropsy to weigh and preserve in fixative all those organs and tissues listed above, including those tissues preserved from rats dying or culled during the study. At the time of terminal necropsy, a peripheral blood and femoral bone marrow smear was prepared from most rats, and filed for future reference, if indicated. Portions of fat, liver and kidney from a maximum of 5 rats/sex/group were frozen for possible 2,4,5-T analysis at the time of both the interim and terminal necropsy examinations.

Histologic Examination of Tissues. Histologic examination was conducted on paraffin embedded sections of tissues which were stained with hematoxylin and eosin (H&E). All rats from the control and high dose groups killed after 118-119 days were subjected to histologic examination of an extensive list of tissues listed in Table 39. Additional sections of liver and kidney from most control and top dose rats killed after 118-119 days were stained for lipid content using Oil Red O Stain. Tissues

collected at the interim kill (after 118-119 days) from the lower dose groups were not examined due to the absence of any definite treatment-related effects in the high dose group. Rats from all treatment and control groups of the 2-year study, regardless of whether they died, or were culled during the study, or were killed at the termination, were subjected to histologic examination of H&E stained sections of an extensive list of tissues from organ systems of the body. These tissues are listed in Tables 40 and 41. Additional sections of kidneys from 5-7 female rats from the control and high dose groups of the terminal kill were stained with Oil Red O, Mallory's reaction for iron, Ziehl-Neelson stain for acid fastness, Dahl's Method for Calcium and the periodic-acid-Schiff reaction (Luna, 1968).

Statistical Evaluation of Data. Hematology, urinary and clinical chemistry parameters, body weights, organ weights, and organ/body weight ratio data were statistically analyzed by a one-way analysis of variance followed by Dunnett's Test, $p<0.05$, (Steel and Torrie, 1960). Data on mortality, palpable masses, gross pathology, histopathology and tumor incidences of the rats of the 2-year study were analyzed using Fisher's Exact Probability Test, $p<0.05$, one sided test (Siegel, 1956).

The statistical evaluation of the gross and histopathologic data from the 2-year study was conducted on the following basis:

a) Gross Pathology Observations: Statistical evaluation of the cumulative data for the entire study compared the data of each

of the treatment groups against the data of the control group of that sex. The data were visually inspected, and those cases suggestive of a statistical difference from control were analyzed.

b) Histopatholbgy Observations and Tumor Incidences: This statistical evaluation compared the cumulative data of each dose group against the data of the control group of that sex. The exact number of tissues examined was used as the total group size in each analysis performed. The data were visually inspected and those cases suggestive of a statistical difference from control were analyzed.

RESULTS

Dietary Content of 2,4,5-T. Analyses of feed samples indicated the dosage levels of 30, 10 and 3 mg 2,4,5-T/kg/day equated with approximately 677 ppm, 230 ppm and 85 ppm of 2,4,5-T in the diet (Table 2 and Appendix 1). Repeated analyses of the feed samples indicated generally good agreement between the intended content of 2,4,5-T and the results of analysis for 2,4,5-T content. On one occasion, analysis of the diet for the group of rats given 3 mg 2,4,5-T/kg/day indicated a somewhat higher than expected content of 2,4,5-T, but this was considered to have minimal impact on the study.

Body Weight Data. Body weight data are included in Table 3 and Appendices 2 and 3 (118-119 day study), and Table 4 and Appendices 4 and 5 (two-year study). Mean body weights of all treated male and female rats in the 118-119 day study showed no statistically significant differences from control means. Mean body weights of females given 30 mg 2,4,5-T/kg/day were statistically decreased at four time periods during the course of the two-year study. The mean body weights of males given 30 mg 2,4,5-T/kg/day had a trend towards slightly lower values during most of the two-year study. Males and females given 10 or 3 mg 2,4,5-T/kg/day had no statistical differences from control values, except for 1 time period when the mean body weights of males given 10 mg 2,4,5-T/kg/day were statistically increased. This isolated observation was considered of no toxicologic significance.

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Food Consumption Data. Food consumption data are listed in Table 5 and Appendices 6 and 7 (118-119 day study) and Table 6 and Appendices 8 and 9 (2-year study). There were no consistent deviations in the food consumption of males or females at any of the 3 dose levels during the course of the 118-119 day study or the 2-year study. The few sporadic cases in which there was a statistical increase or decrease between the control and various treatment groups followed no consistent trend, and were considered of no toxicological significance.

Mortality: Cumulative mortality data from the two-year study are included in Tables 7 and 8 and Figures 1 and 2. The group of male rats given 30 mg 2,4,5-T/kg/day had a statistically significant decreased rate of mortality during the latter portion of the study, possibly secondary to a lesser degree of chronic renal disease noted in these male rats. Females at this dose level showed no difference from the control mortality rate. Males and females given 10 mg 2,4,5-T/kg/day had statistical increases in mortality rate during 4-5 time periods. Males given 3 mg 2,4,5-T/kg/day had statistically decreased mortality rates during 4 time periods. Females at this low dose level showed no differences from control values. These differences in mortality rates in rats given 10 or 3 mg 2,4,5-T/kg/day showed no dose-related effects and were considered of no toxicological significance.

Palpable Masses and Animal Observations: The cumulative numbers of rats of the two-year study with palpable masses detected at monthly intervals

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are given in Table 9. The first palpable mass was noted in a male rat of the control group. There were no statistically significant differences between control and treated male rats except during months 13 and 14, when male rats given 10 mg 2,4,5-T/kg/day had an increased incidence of palpable masses. This was considered of no toxicological significance due to its isolated occurrence and lack of a dose or temporal response.

There were no differences in the number of treated female rats with palpable masses except for a statistically increased number of rats with palpable masses in the group receiving 30 mg of 2,4,5-T/kg/day during months 9 and 10 only; the female group receiving 10 mg 2,4,5-T/kg/day showed a statistically significantly increased incidence of palpable masses during month 14 only. These observations were considered of no toxicological significance due to these isolated occurrences and absence during the latter phases of the study. Examination of the rats during the course of the 2-year study revealed no clinical evidence of toxicity attributable to these levels of treatment with 2,4,5-T.

Hematology. The hematology data from the 118-119 day study are listed in Table 10 and Appendices 10 and 11. The hematology data from the 2-year study are listed in Tables 11-13 and Appendices 12-17. There were no statistically significant differences from control values at any dose level at any time during the course of the subchronic or chronic portions of the study.

Urinalysis. Urinalysis data for the 118-119 day study are listed in Table 14 and Appendices 18-19. Examination of urinary specific gravity, pH, glucose, protein, ketones, bilirubin and occult blood showed no changes considered related to treatment. Urinalysis data from the two-year study are listed in Tables 15-17 and Appendices 20-25. Repeated examination of the above listed parameters plus urobilinogen at the last two time periods revealed no changes considered related to treatment. On one occasion there was a statistical decrease in the specific gravity of male rats given 10 mg 2,4,5-T/kg/day; however, this was considered of no toxicological significance due to its isolated occurrence and lack of dose response.

Urinary Volume, Creatinine, Porphyrins and delta-ALA. These data for the 118-119 day study are summarized in Tables 18 and 19 and individually listed in Appendices 26-29. The data from rats used in the two-year study are summarized in Tables 20-24 and listed individually in Appendices 30-34.

In males receiving 30 mg 2,4,5-T/kg/day, there were statistically significant increases in total urine volume and excretion of coproporphyrin and uroporphyrin. In males receiving 10 mg 2,4,5-T/kg/day, there was a statistically significant increase in coproporphyrin excretion in the early phase of the study only. In females receiving 30 mg 2,4,5-T/kg/day, there was increased excretion of coproporphyrin at most of the sampling times during the study; the excretion of uroporphyrin was statistically decreased after 119 days and statistically increased after 566 days.

In male rats receiving 3 mg of 2,4,5-T/kg/day and in female rats receiving 10 or 3 mg 2,4,5-T/kg/day there were no statistically significant changes in total urinary volume, or excretion of creatinine, coproporphyrin, uroporphyrin, or delta-ALA at any time during the study.

Clinical Chemistry. The data from rats killed at the interim necropsy after 118-119 days on study are listed in Table 25 and Appendices 35-36.

The data from rats killed at the termination of the two-year study are listed in Table 26 and Appendices 37-38. Analyses of serum samples collected by decapitation showed no alterations considered related to treatment with any of the dose levels in regard to BUN, SGPT, AP, bilirubin (total, direct, indirect), total protein, albumin, and globulin. SGPT was statistically increased in females given 10 mg 2,4,5-T/kg/day for 118-119 days; however, this was considered of no toxicological significance due to its isolated nature, lack of dose response and absence at time of the terminal necropsy.

Organ Weights. The data from rats killed at the interim necropsy after 118-119 days are listed in Table 27 and Appendices 39-40. A statistically significant increase in relative weight of the kidneys of male rats given 30 mg 2,4,5-T/kg/day was considered to be related to treatment.

The data from rats killed at the termination of the two-year study are listed in Tables 28 and 29 and Appendices 41-42. The only statistical

difference was a decrease in the absolute heart weight of males given 30 mg 2,4,5-T/kg/day; this was considered to be of highly questionable toxicological significance due to the absence of any discernible gross or histopathologic changes in these hearts. There were no alterations in weights of brain, liver, testes, spleen, thymus or uterus/ovaries at any of the dose levels which were considered related to treatment.

Gross and Histopathologic Observations. The gross necropsy and histopathologic observations for rats killed after 118-119 days are tabulated in Tables 30 and 31, respectively. There were no gross or histopathologic observations that were considered to be the result of treatment with any of these dose levels for 118-119 days.

The gross necropsy and histopathologic observations for rats from the two-year study are included in Tables 32-33 and 34-35, respectively. This includes data on all rats that died, were culled or survived to the terminal kill after 2 years. In view of the voluminous nature of the pathology data included in Tables 30-35, examples of the major treatment-related observations have been selected for the attached Summary Table. These pathology results are discussed below.

The kidneys of female rats receiving 30 mg 2,4,5-T/kg/day had gross and microscopic evidence of an increased incidence of mineralized deposits in the renal pelvis; these mineralized deposits were sometimes accompanied by a localized inflammatory reaction of the adjacent renal pelvis

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epithelium. Other renal effects attributed to ingestion of 30 mg 2,4,5-T/kg/day included an increased content of pigment within the cytoplasm of proximal convoluted tubular epithelial cells (females), and a decrease in the severity of the chronic nephropathy that normally occurs as a spontaneous entity, especially in the males of this strain of rat. The special stains applied to sections of kidneys of the control and high dose group indicated similar staining reactions for both groups, except that the increased pigment noted in the high dose group gave an increased positive reaction with Mallory's stain for iron. The 3 cases of petechial hemorrhage of the urinary bladder noted in the group of male rats given this high dose level were not considered to be related to treatment due to the fact these cases of hemorrhage occurred secondarily to geriatric inflammatory processes originating in adjacent structures. At the intermediate dose level of 10 mg 2,4,5-T/kg/day, there was an increased incidence of mineralized deposits in the renal pelvis of female rats. Statistical increases in the incidence of focal renal tubular atrophy and renal aggregations of lymphoid cells noted in this group of female rats were considered to be a secondary result of an overall decrease in the severity of the geriatric chronic renal disease noted in this group. A statistical decrease in the incidence of mineralized deposits in the renal pelvis of kidneys of male rats given 10 mg 2,4,5-T/kg/day may or may not have been the result of treatment. At the lower dose level of 3 mg 2,4,5-T/kg/day there were no renal lesions of any type in either males or females that were considered related to treatment.

The decreased severity of the spontaneous chronic nephropathy noted in male rats receiving 30 mg 2,4,5-T/kg/day, was accompanied by a secondary decrease in the incidence of mineralization of pulmonary alveoli, myocardium, myocardial blood vessels and gastric mucosa and muscularis.

Although the kidney was the primary target organ, the liver was also affected slightly by lifetime treatment with 30 mg 2,4,5-T/kg/day. The group of male rats given 30 mg 2,4,5-T/kg/day had a decreased incidence of liver enlargement and an increased incidence of focal biliary hyperplasia and periportal inflammation. Livers of females at this high dose level had an increased incidence of focal aggregation of reticuloendothelial cells adjacent to degenerate or necrotic hepatocytes and a decreased incidence of multiple foci of hepatocellular alteration (swollen hepatocytes). The incidence of focal hepatocellular cytoplasmic vacuolization was decreased in livers of males given 30 or 10 mg 2,4,5-T/kg/day. Although the incidence of grossly visible liver nodules was higher in the control group of females than in the treatment groups, this was assumed to be due to a somewhat higher incidence rate of this observation in this control group as compared to our historical experience (~6%). Livers of rats given 3 mg 2,4,5-T/kg/day had no observations attributed to the lifetime ingestion of this dose level.

The lungs of rats given 30 mg 2,4,5-T/kg/day had some observations that didirectly or indirectly may have been the result of this level of treatment. The group of males given this high dose level had an increased incidence

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of focal pulmonary interstitial inflammation, focal accumulations of alveolar macrophages and cholesterol clefts. A few females given this high dose level had increased incidence of focal accumulations of secretory material in the alveoli. At the intermediate or lower dose levels of 10 or 3 mg 2,4,5-T/ kg/day there were no pulmonary changes attributed to treatment, with the possible exception of an increased incidence of focal interstitial fibrosis in lungs of females of the intermediate dose level. However, as this observation was not seen at the higher dosage level, it is of highly questionable toxicologic significance.

Examination of the tissues of the cardiovascular system revealed no lesions that could be definitely attributed to the ingestion of any of these dose levels of 2,4,5-T. In the group of rats given 30 mg 2,4,5-T/kg/day, the females had a statistical increase in the incidence of hearts with a dilated, flaccid ventricle; however, the opposite trend occurred in the heart of males given this same high level. In males given either 30 or 10 mg of 2,4,5-T/kg/day, there was an apparent trend toward decreased incidence of both periarteritis and left atrial thrombosis; however, these observations may not have been the result of treatment with these dose levels of 2,4,5-T.

There were no other gross or histopathologic observations on the various organs that were considered to be the result of ingesting 30, 10 or 3 mg of 2,4,5-T/kg/day for up to 2 years. The other observations not discussed above but listed in Tables 32-35 which were statistically different from

the controls were not considered to be the result of treatment usually due to (1) lack of a dose response or (2) abnormal incidence in the control group when compared to historical control data.

Tumor incidence. Tumor incidence rates for male and female rats are tabulated in Tables 36 and 37, respectively. These data are listed for each of the sequential 6-month periods as well as the terminal kill: the cumulative results for each category of tumor type are also included. The spectrum of tumors that have been historically observed in rats of this strain were noted in the liver, nasal turbinates/hard palate, lungs, pancreas, kidney, urinary bladder, testes, ovary, uterus, musculoskeletal tissue, oral cavity, tongue, salivary glands, stomach, small intestine, large intestine, subcutaneous tissues, integument, mammary gland, ear canal, brain, peripheral nerves, pituitary gland, cranial cavity, adrenal glands, eye, lymph nodes, thymus, spleen, mesentery, thyroid and parathyroid glands. Upon statistical analyses of these tumor data, the incidence rates for all categories of tumor types occurring in any of the groups given 30, 10 or 3 mg 2,4,5-T/kg/day were comparable to the control group, with the exception that the incidence rate of interfollicular C-cell adenoma of the thyroid was statistically increased in females given the low dose level of 3 mg 2,4,5-T/kg/day. This observation was considered not related to treatment based on the lack of a dose response and an unusually lower incidence of thyroid adenomas in this control group of females when compared to historical control data (up to 17%). Thus, none of the tumors listed in Tables 36 and 37 were considered

related to treatment with any of these dose levels of 2,4,5-T. Table 38 summarizes the total numbers of tumors per group, the average number of tumors per rat, and the time interval listing of rats with tumors for each group. None of these parameters were affected by any of these dose levels of 2,4,5-T with these data comparable to the control groups.

DISCUSSION

During the course of this two-year study, parameters found to be unaffected by treatment with any of these dose levels of 30, 10 or 3 mg of 2,4,5-T/kg/day included the rate of mortality, food consumption, palpable masses, hematology (RBC count, Hgb, PCV, total and differential WBC, thrombocytes and reticulocytes), routine urinalysis, urinary excretion of creatinine or delta-ALA, serum clinical chemistry (BUN, SGPT, AP, bilirubin, total protein, albumin and globulin), organ weights (except kidney at highest dose level), tumor incidence as well as gross and microscopic morphology of all organ systems of the body (except for kidney, liver and possibly lung lesions described below.

The highest dose level of 30 mg of 2,4,5-T/kg/day predictably caused some degree of toxicity. This included a decrease in body weight gain, an increased urinary excretion of total urine volume, coproporphyrin and uroporphyrin, increased relative kidney weights as well as morphologic alterations of the kidney, liver and possibly lung. In the kidney, this involved primarily the presence of mineralized deposits in the renal pelvis, sometimes accompanied by a resultant localized reaction of the adjacent renal pelvis epithelium. The chronic nephropathy normally encountered as a geriatric occurrence in aged rats was decreased in rats given 30 mg of 2,4,5-Tkg/day. Morphologic alterations in the liver considered to be the result of treatment with this high dose level of 30 mg 2,4,5-T/kg/day included an increased incidence of periportal inflammation, focal biliary hyperplasia and

inflammatory cell aggregations adjacent to degenerate hepatocytes; this was accompanied by a decreased incidence of multiple foci of swollen hepatocytes and focal cytoplasmic vacuolization. Lungs of rats given 30 mg 2,4,5-T/kg/day had indications of focal pulmonary inflammation, including focal accumulations of alveolar macrophages and cholesterol clefts.

At the intermediate dose level of 10 mg of 2,4,5-T/kg/day there was only minimal toxicity, limited primarily to an increased incidence of mineralized deposits in the renal pelvis; an increase in urinary excretion of coproporphyrin was noted in males of this dose level only during the early phase of the study. At the lower dose level of 3 mg of 2,4,5-T/kg/day there were no alterations in any parameter considered related to treatment over the course of this two-year study.

In this study, there were no tumors considered to be the result of treatment with any of the dose levels, including the maximum tolerated dose of 30 mg 2,4,5-T/kg/day given for 2 years. The absence of any oncogenic response noted in this study concurs with the results of most of the previous oncogenic studies on 2,4,5-T that have been reported.

The 1969 report of Innes et al. found no oncogenic response in either of 2 strains of mice given the maximum tolerated dose of 21.5 mg 2,4,5-T/kg/day. The 1968 study by the Bionetics Research Laboratory noted no increase in tumors in mice of 2 strains given a single subcutaneous injection of 215 mg 2,4,5-T/kg.

The only published oncogenic study which reports an increase in tumors allegedly due to treatment with 2,4,5-T is one of the four studies conducted by Muranyi-Kovacs et al. and published in 1976 and 1977. In these studies, mice of the C3Hf strain that were given a single dose level of 80 ppm (approximately 12 mg/kg/day) of 2,4,5-T had a statistical increase in the so-called "non-incidental" types of tumors. Mice of the XVIIG strain that were given the identical regimen of treatment with 2,4,5-T did not have any increase in tumor incidence. When Muranyi-Kovacs et al. (1977) gave neonatal mice of these same 2 strains (C3Hf and XVIIG) a series of subcutaneous injections of 2,4,5-T there was no resultant increase in tumor incidence in either strain of mouse. In the one oncogenic study out of four conducted by Muranyi-Kovacs et al. in which there was reported to be an oncogenic response due to 2,4,5-T, it is interesting to note that there was no specific "target tissue" that was showing the alleged oncogenic response. This is contrary to the vast majority of cases wherein a true positive oncogenic response typically affects specific target tissue(s), with the resultant increase in tumors originating from the target tissue(s).

Overall, the results of this study concur with the results of all but one of the previous oncogenic studies which have been reported on 2,4,5-T. In the study reported herein, rats given 30 or 10 mg of 2,4,5-T/kg/day for 2 years had some dose-related toxicity (primarily renal) but no oncogenic response; rats given 3 mg of 2,4,5-T/kg/day had no adverse effects related to treatment.

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SUMMARY TABLE

SUMMARY OF MAJOR OBSERVATIONS IN RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO 2 YEARS

(Including Selected Data Extracted from Full Data Base for Purpose of Depicting Effects Considered Related to Treatment)

Sex	Males				Females			
Dose (mg/kg/day)	0	30	10	3	0	30	10	3
Number of Rats in Group (2-Year + 118-Day Studies)	86+10	50+10	50+10	50+10	86+10	50+10	50+10	50+10
PARAMETER								
Body Weight (grams) (Mean±S.D.)	385±21	375±21	386±25	385±25	253±14	244±12[a]	253±15	254±12
Food Consumption		----	----	----		----	----	----
Mortality		----	----	----		----	----	----
Palpable Masses and Clinical Observations		----	----	----		----	----	----
Hematology (RBC, PCV, Hgb, WBC, differential WBC, platelets, reticulocytes)		----	----	----		----	----	----
Urinalysis (specific gravity, pH, protein, glucose, ketones, occult blood, bilirubin, urobilinogen)		----	----	----		----	----	----
Urinary Excretion - (118-119 Day Study)								
Total urine volume (48 hr. volume in ml) (Mean±S.D.)	38±8	49±11[a]	36±14	40±10		----	----	----
Coproporphyrin (μg/48 hr.) (Mean±S.D.)	17.5±4.9	43.6±9.3[a]	24.6±9.6[a]	23.1±4.2	9.6±1.9	16.4±7.0[a]	9.2±0.8	10.3±3.5
Uroporphyrin (μg/48 hr.) (Mean±S.D.)	4.1±1.8	7.7±4.7[a]	5.0±3.1	4.5±2.0		----	----	----
Creatinine		----	----	----		----	----	----
Delta-aminolevulinic acid		----	----	----		----	----	----
Urinary Excretion - (Termination of Two-Year Study)								
Coproporphyrin (μg/48 hr.)	14.6±6.0	22.8±13.0	9.7±4.4	15.2±3.6	5.8±1.9	8.0±9.8	9.8±2.3	6.5±2.3
Uroporphyrin (μg/48 hr.)	4.7±1.8	4.0±1.5	3.0±1.3	3.0±0.9		----	----	----
Clinical Chemistry (SGPT, BUN, AP, bilirubin, albumin, globulin, total protein)		----	----	----		----	----	----
Organ Weights (brain, liver, testes, heart, thymus, spleen, ovaries/uterus) - (118-119 Day and 2-Year Studies)		----	----	----		----	----	----
Kidneys - (118-Day Study only) (gm/100 gm B.W.) (Mean±S.D.)	0.70±0.09	0.79±0.06[a]	0.73±0.05	0.72±0.05		----	----	----
Gross and Histopathology Examination (All organ systems):								
Kidney -								
Mineralized deposits in pelvis		----	----	----	1/86	8/50[a]	5/50[a]	1/50
Localized reaction adjacent to mineralized deposits		----	----	----	25/86	37/50[a]	22/50	19/50
Moderate to severe chronic renal disease	56/86	22/50[a]	30/50	28/50	6/86	1/50	2/50	6/50
Increased pigment in renal tubular cytoplasm		----	----	----	0/86	11/50[a]	0/50	0/50
Mineralization secondary to renal disease seen in -								
pulmonary alveoli	13/86	0/50[a]	4/50	8/50		----	----	----
myocardium	5/86	0/50	2/50	3/50		----	----	----
myocardial blood vessels	12/86	0/50[a]	5/50	6/50		----	----	----
gastric mucosa and muscularis	25/85	1/50[a]	9/49	13/49		----	----	----

[a]Statistically different from control value(s) when analyzed using analysis of variance and Dunnett's Test, $p<0.05$ or Fisher's Exact Probability Test, $p<0.05$.

---- Indicates parameter was considered unaffected at any level of treatment.

For parameters with voluminous or multiple observations, only selected representative values are listed in summary table.

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SUMMARY TABLE (Continued)

SUMMARY OF MAJOR OBSERVATIONS IN RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO 2 YEARS

(Including Selected Data Extracted from Full Data Base for Purpose of Depicting Effects Considered Related to Treatment)

Sex	Males				Females			
Dose (mg/kg/day)	0	30	10	3	0	30	10	3
Number of Rats in Group (2-Year + 118-Day Studies)	86+10	50+10	50+10	50+10	86+10	50+10	50+10	50+10
PARAMETER (cont'd)								
Gross and Histopathology Examination (cont'd)								
Liver -								
Enlargement	11/86	1/50[a]	7/50	3/50		----	----	----
Focal biliary hyperplasia	9/86	14/50[a]	8/50	6/50		----	----	----
Periportal inflammation	29/86	28/50[a]	9/50	7/50[a]		----	----	----
Focal aggregates of reticuloendothelial cells adjacent to degenerate or necrotic hepatocytes		----	----	----	36/86	34/50[a]	21/50	17/50
Multiple foci of hepatocellular alteration (swollen hepatocytes)		----	----	----	15/86	3/50[a]	6/50	4/50
Focal hepatocellular cytoplasmic vacuolization	34/86	8/50[a]	12/50[a]	20/50		----	----	----
Lungs -								
Focal interstitial inflammation	21/86	27/50[a]	5/50[a]	5/50[a]		----	----	----
Focal accumulations of alveolar macrophages in alveoli	28/86	29/50[a]	13/50	16/50		----	----	----
Focal cholesterol clefts	6/86	9/50[a]	5/50	4/50		----	----	----
Focal accumulations of secretory material in alveoli		----	----	----	0/86	4/50[a]	2/50	2/50
Cardiovascular -								
Dilated/flaccid ventricle	28/86	9/50	7/50	15/50	0/86	3/50[a]	2/50	1/50
Periarteritis	20/86	8/50	8/50	12/50		----	----	----
Left atrial thrombosis	12/86	3/50	4/50	6/50		----	----	----
Tumors (all organ systems) (See attached supplemental tumor data)		----	----	----		----	----	----

[a]Statistically different from control value(s) when analyzed using analysis of variance and Dunnett's Test, $p<0.05$ or Fisher's Exact Probability Test, $p<0.05$.

---- Indicates parameter was considered unaffected at any level of treatment.

For parameters with voluminous or multiple observations, only selected representative values are listed in summary table.

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SUMMARY TABLE (Continued)

SUPPLEMENTAL TUMOR INCIDENCE DATA IN RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T

Sex	Males				Females			
Dose (mg/kg/day)	0	30	10	3	0	30	10	3
Number of Rats in Group	86	50	50	50	86	50	50	50
Total Number of Tumors in Group	172	97	71	89	308	150	147	140
Average Number of Tumors per Rat	2.0	1.9	1.4	1.8	3.6	3.0	2.9	2.8
Number of Tumor-bearing Rats/Number of Rats in Group								
These Data Listed in Time Intervals in Months:								
1-6	1/1	0/0	0/0	0/0	0/0	0/0	0/1	1/1
7-12	1/2	0/0	1/1	0/1	1/2	2/2	1/2	0/1
13-18	8/14	6/9	15/22	9/12	16/17	9/9	11/11	10/11
19-24	60/63	24/25	20/23	26/28	30/31	18/18	25/25	18/18
Terminal Kill	6/6	14/16	4/4	9/9	36/36	21/21	11/11	19/19
Total[a]	76/86 (88%)	44/50 (88%)	40/50 (80%)	44/50 (88%)	83/86 (97%)	50/50 (100%)	48/50 (96%)	48/50 (96%)

[a]No statistical differences from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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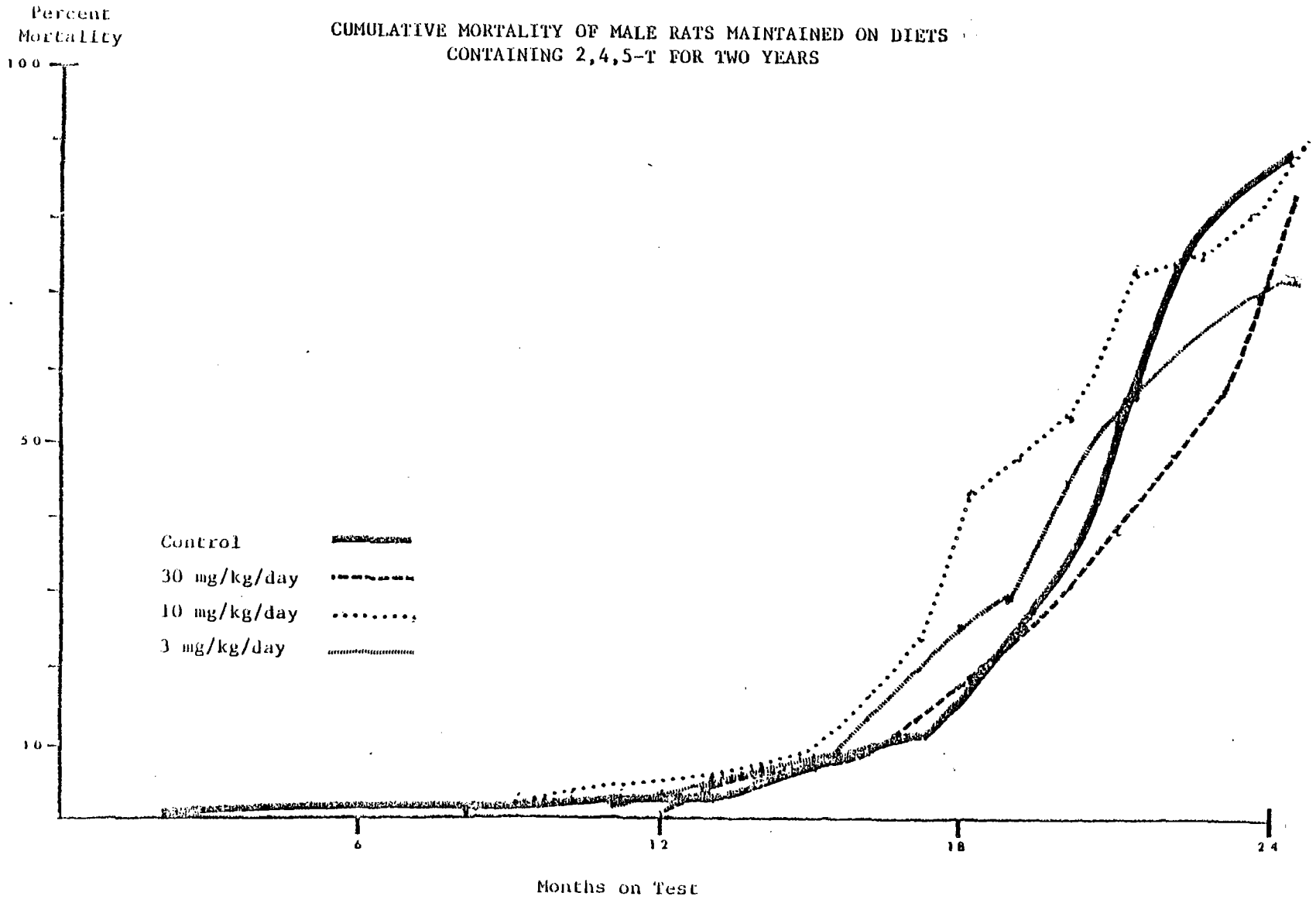
FIGURE 1

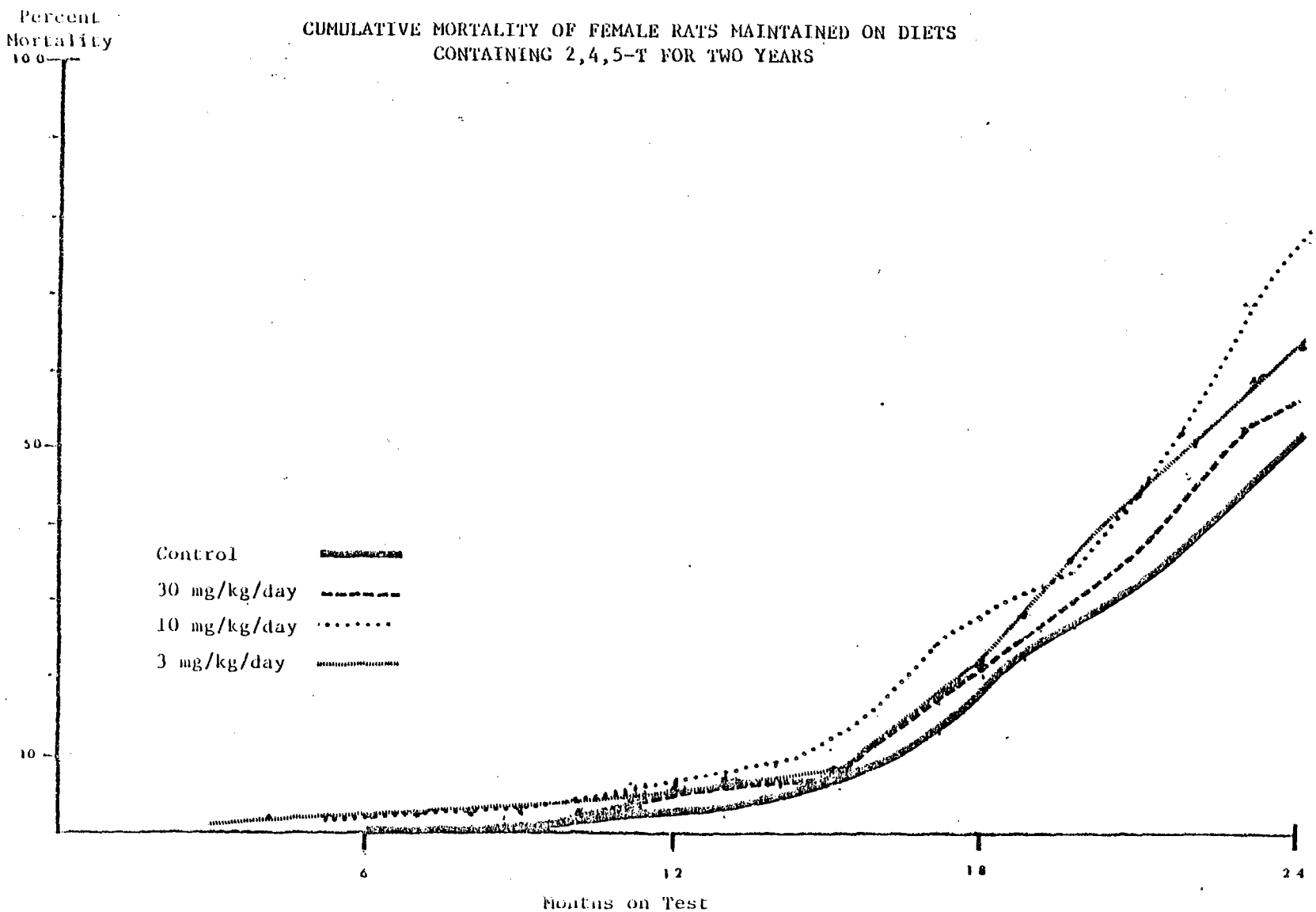
CUMULATIVE MORTALITY OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR TWO YEARS



21292

FIGURE 2

CUMULATIVE MORTALITY OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR TWO YEARS



212 83

TABLE 1

ANALYSIS OF SAMPLE OF 2,4,5-T
USED IN TWO-YEAR DIETARY STUDY

2,4,5-Trichlorophenoxyacetic Acid by titration - 99.1%

Other Phenoxy Components	GLC Condition No.[a]	Weight Percent
2,6-dichlorophenol	1,3	N.D.[b]
2,4-dichlorophenol	1,3	N.D.
2,3,6-trichlorophenol	1,3	N.D.
2,4,6-trichlorophenol	1,3	N.D.
2,4,5-trichlorophenol	1,3	N.D.
2-chlorophenoxyacetic acid	1,3	N.D.
4-chlorophenoxyacetic acid	1,3	N.D.
2,5-dichlorohydroquinone mono methyl ether	1,3	N.D.
4,6-dichlororesorcinol mono methyl ether	1,3	N.D.
4,5-dichlorocatechol mono methyl ether	1,3	N.D.
2,6-dichlorophenoxyacetic acid	1,3	N.D.
2,5-dichlorophenoxyacetic acid	1,3	0.12
2,4-dichlorophenoxyacetic acid	1,3	0.02
3,4-dichlorophenoxyacetic acid	1,3	N.D.
2,4,6-trichlorophenoxyacetic acid	1,3	N.D.
2,3,6-trichlorophenoxyacetic acid	1	N.D.
4,5-dichloro-2-methoxyphenoxyacetic acid	1	0.34
2,5-dichloro-4-methoxyphenoxyacetic acid	1	0.67
2,4-dichloro-5-methoxyphenoxyacetic acid	1	
bis(2,4,5-trichlorophenoxy)acetic acid	2	N.D.
Unknown A[c]	3	0.08
Unknown B[d]	3	0.06

[a]The following gas-liquid chromatography conditions were used to determine the impurities: Flame ionization detectors were used in all cases.

GLC Conditions #1
Column: 3' x 1/8" 25% LAC 2R446, 2% H_3PO_4 on 80/100 mesh Chrom W
Column Temp. 215°C, Injector Temp. 235°C, Detector Temp. 230°C

GLC Conditions #2
Column: (glass) 10' x 1/8" 5% OV-101 on 80/100 mesh Chrom Q
Column Temp. 280°C, Injector Temp. 305°C, Detector Temp. 325°C

GLC Conditions #3
Column: (glass) 10' x 1/8" 5% OV-101 on 80/100 mesh Chrom Q
Column Temp. 235°C, Injector Temp. 230°C, Detector Temp. 215°C

[b]N.D. is not detected. The limit of detection is estimated as 0.02 %.
[c]Unknown A has the proposed structure of bromodichlorophenoxyacetic acid.
[d]Unknown B has the proposed structure of tetrachlorophenoxyacetic acid.
Data from Dow reports ML-AL 87-561 and AL 83-607.

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TABLE 1 (Continued)

ANALYSIS OF SAMPLE OF 2,4,5-T
USED IN TWO-YEAR DIETARY STUDY

Dioxin	Found ppb	Limit of Detection ppb
2,3,7,8-Tetrachlorodibenzo-p-dioxin	N.D.	0.33
Hexachlorobenzo-p-dioxin	N.D.	0.12
Heptachlorodibenzo-p-dioxin	N.D.	0.40
Octachlorodibenzo-p-dioxin	N.D.	0.40

N.D. = None Detected

Data from Dow Report AL-78-70077.

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TABLE 2

MEAN ANALYTICAL CONTENT OF 2,4,5-T IN DIETS OF RATS
USED IN A TWO-YEAR TOXICITY STUDY

Time Interval, Months	Anticipated Dose Level of 2,4,5-T (mg/kg/day)		
	30	10	3
5	580 ppm	180 ppm	63 ppm
7	660 ppm	240 ppm	63 ppm
12	750 ppm	250 ppm	93 ppm
19 (males)	760 ppm	250 ppm	65 ppm
19 (females)	630 ppm	200 ppm	64 ppm
23 (males)	700 ppm	250 ppm	150 ppm
23 (females)	660 ppm	240 ppm	94 ppm
Mean dietary content of 2,4,5-T	677 ppm	230 ppm	85 ppm

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TABLE 3

MEAN BODY WEIGHTS[a] OF MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 AND 119 DAYS

Dose mg/kg/day	Sex	Days on Test 6	13	20	26	32	39	46	53	60	68	81	88	95	102	117
0	M	247±9	337±15	367±18	384±19	401±22	412±35	437±29	450±30	462±30	454±31	481±35	494±34	499±32	507±33	490±39
30	M	243±10	329±18	357±22	377±24	391±38	408±27	426±30	440±31	447±31	462±36	474±34	480±35	484±42	492±36	494±42
10	M	248±10	334±19	364±26	386±31	402±35	419±38	437±43	451±46	463±51	463±56	495±61	500±63	510±66	517±65	512±66
3	M	243±12	336±19	366±22	385±26	402±28	421±30	436±34	450±36	462±40	458±46	486±42	494±42	502±42	508±45	508±44
0	F	200±7	240±10	251±8	259±11	270±12	274±12	282±13	288±14	289±12	299±14	302±15	306±15	310±15	316±18	318±18
30	F	205±7	242±8	255±10	265±11	274±10	277±12	282±11	289±15	290±13	296±13	302±15	306±16	311±16	312±15	319±18
10	F	199±6	240±14	254±15	261±16	269±20	277±18	283±20	288±21	294±24	303±22	306±23	310±24	313±26	316±22	326±27
3	F	201±4	240±7	256±10	263±12	268±8	273±13	284±13	290±12	290±14	299±13	305±14	307±14	312±14	314±13	318±24

[a]All weights are in grams ± one standard deviation. (10 rats/group housed 2 rats/cage).
No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

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TABLE 4

MEAN BODY WEIGHTS[a] FOR MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Days on Test	Dose Level mg/kg/day							
	Males				Females			
	0	30	10	3	0	30	10	3
0	244±12	243±10	247±11	244±13	195±7	194±9	197±10	197±9
6	290±13	290±10	297±15	287±14	217±10	214±11	217±10	220±11
13	334±17	329±12	337±19	331±19	234±9	230±12	235±10	237±11
20	362±21	356±16	367±24	361±23	246±14	240±12	249±11	249±13
26	385±21	375±21	386±25	385±25	253±14	244±12[b]	253±15	254±12
32	396±26	393±20	406±30	397±28	261±15	254±12	261±14	263±13
39	414±30	408±20	423±32	411±30	266±12	259±13	266±13	272±14
46	430±33	424±22	439±35	428±30	272±15	264±12	270±14	278±18
53	444±30	437±30	449±36	447±34	278±16	266±15[b]	273±15	279±15
60	455±36	452±23	464±40	454±33	281±16	271±14	281±14	284±15
68	472±38	467±27	481±40	472±37	289±16	279±15	288±16	293±15
75	479±39	472±28	485±48	479±37	290±16	280±16	290±15	294±17
81	488±39	477±27	497±45	484±36	293±18	282±14	294±15	297±18
88	493±43	487±29	510±47	493±37	299±16	287±15	299±16	303±17
95	498±36	492±38	508±43	503±41	301±17	288±17[b]	299±17	306±17
123	521±46	508±32	526±53	517±40	310±19	303±17	305±23	317±20
152	536±42	527±42	546±50	546±48	315±21	307±18	318±22	320±17
175	560±49	550±34	572±60	556±42	330±22	319±19	326±24	338±24
180	561±57	557±35	577±60	560±44	330±25	320±18	330±17	339±24
203	567±46	554±45	575±54	570±51	328±26	322±22	333±20	336±21
232	586±50	578±46	598±56	593±54	340±28	331±23	340±21	347±20
258	596±49	585±47	609±57	605±55	345±26	335±26	346±25	353±23
291	617±52	606±47	625±55	623±56	354±33	347±26	358±25	359±26
314	627±53	616±50	635±59	631±59	361±28	347±27[b]	365±28	363±26
349	622±54	609±53	628±59	625±57	364±31	354±29	370±33	367±28
377	635±55	620±62	641±62	638±64	372±35	359±33	379±38	378±35
405	641±58	626±60	639±68	643±61	373±36	360±39	382±44	380±40
433	656±60	643±55	649±81	660±64	393±42	381±42	396±50	407±53
468	654±61	639±60	651±80	654±72	400±50	389±45	417±65	411±56
501	647±61	641±61	647±80	654±65	403±47	399±55	408±49	420±53
531	632±59	636±66	640±93	650±52	406±51	395±45	405±43	422±57
559	620±62	628±70	649±81	639±58	418±58	404±44	420±52	424±56
587	617±65	636±66	650±79	619±65	421±63	411±50	434±52	427±61
622	591±64	611±85	643±89[b]	621±56	420±67	420±61	449±38	425±69
650	578±72	607±77	580±106	612±55	420±79	432±71	427±79	427±65
685	562±61	583±99	564±80	596±64	425±69	433±69	439±99	436±69
714	548±50	578±110	516±125	589±79	451±92	451±96	419±110	433±75
732	538±51	580±65	559±72	587±102	458±112	447±83	418±59	432±101

[a] grams.

[b] Statistically significant deviation from control mean using analysis of variance and Dunnett's test, $p<0.05$.

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TABLE 5.

MEAN FOOD CONSUMPTION[a] OF MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 AND 119 DAYS

Dose mg/kg/day	Sex	Days on Test 0-3	8-10	11-13	14-17	18-20	21-24	25-27	28-31	32-34	35-38	39-41	42-45	46-48	49-52	53-55
0	M	25±2	26±2	28±2	29±2	28±2	26±2	23±3	26±1	26±2	27±2	28±2	28±1	27±2	26±2	26±2
30	M	25±1	26±1	27±1	26±1[b]	27±3	26±2	25±1	25±1	26±1	26±1	26±1	27±3	26±2	27±3	26±2
10	M	25±1	26±1	27±1	26±1[b]	26±1	24±4	26±3	26±2	26±2	26±2	25±2	26±3	26±2	26±3	26±3
3	M	26±1	27±1	27±1	27±1	26±1	26±1	26±1	27±1	26±1	26±1	26±1	28±1	26±1	27±1	27±1
0	F	20±3	19±1	20±1	21±3	19±1	26±1	20±1	20±1	19±2	20±2	20±2	20±2	19±1	20±1	19±2
30	F	19±0	19±2	19±1	20±1	20±2	25±3	18±1	19±1	19±1	18±1	19±1	18±1	18±2	18±1	18±1
10	F	20±1	19±2	20±1	20±2	23±7	26±1	19±1	20±1	22±3	20±1	20±2	23±5	20±3	20±1	20±2
3	F	20±1	19±1	19±2	20±2	20±2	25±2	19±1	20±3	19±1	19±1	19±1	21±1	19±2	19±2	18±2

[a]All data in grams as mean ± one standard deviation (10 rats/group housed 2 rats/cage).

[b]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

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TABLE 5 (Continued)

MEAN FOOD CONSUMPTION[a] OF MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 AND 119 DAYS

Dose mg/kg/day	Sex	Days on Test 56-59	60-62	63-66	67-69	70-73	77-80	81-83	84-87	88-90	91-94	95-97	102-105	106-108	109-111
0	M	26±2	27±2	25±2	24±1	26±1	27±2	25±2	26±1	25±2	26±4	25±2	25±2	24±2	23±2
30	M	22±2	25±2	26±2	25±2	24±2	27±2	24±2	25±1	25±2	26±1	26±3	25±1	28±5	24±1
10	M	25±4	26±2	25±2	25±3	25±2	26±1	25±2	26±2	25±2	26±2	26±3	27±1	24±2	24±3
3	M	25±2	26±1	26±1	25±1	25±1	27±1	26±1	26±0	27±5	27±1	26±1	26±1	26±4	24±2
0	F	20±1	22±3	19±1	20±4	19±1	20±0	20±3	20±1	20±3	20±1	19±1	20±1	19±5	19±1
30	F	17±1[b]	18±2[b]	18±1	17±2	17±1	19±2	18±1	20±1	18±2	18±1	18±1	20±0	20±3	22±8
10	F	19±1	19±1	19±1	17±2	17±1	20±2	19±1	20±1	19±1	20±1	19±1	19±1	21±6	18±2
3	F	17±0[b]	19±1	19±2	17±2	18±2	26±2[b]	19±1	20±1	19±2	20±2	18±2	20±1	20±3	21±2

[a]All data in grams as mean ± one standard deviation (10 rats/group housed 2 rats/cage).

[b]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

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TABLE 6

MEAN FOOD CONSUMPTION[a] FOR MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Days on Test	Dose Level mg/kg/day							
	Males				Females			
	0	30	10	3	0	30	10	3
0-3	24±1	24±1	24±1	24±1	18±2	19±1	20±2	20±2
4-6	27±1	27±1	27±1	26±2	19±2	20±2	20±2	20±1
7-10	26±2	25±1	26±2	26±2	18±2	18±2	19±1	19±1
11-13	27±1	26±2	26±2	26±2	19±1	18±1	19±1	19±1
14-17	28±2	26±1	27±2	27±2	21±2	18±1[b]	21±3	19±2
18-20	26±2	25±1	26±1	26±2	21±3	20±5	20±2	19±2
21-24	25±1	24±1	25±1	26±2	20±3	17±1[b]	18±1	18±1
25-27	27±2	25±1	27±2	25±2	20±2	19±1[b]	18±1[b]	18±2[b]
28-31	25±1	25±1	25±1	25±2	18±2	20±3	22±4	20±1
32-34	26±2	24±1	25±1	25±2	19±2	19±2	21±3	19±1
35-38	27±2	25±1	25±2	25±2	19±1	18±1	18±1	19±1
39-41	26±1	25±1	25±1	25±2	19±1	18±1	21±5	19±2
42-45	27±1	27±2	27±1	28±3	21±3	19±2	19±4	20±2
46-48	26±1	26±1	26±2	26±3	19±2	18±2	18±3	18±1
49-52	26±2	27±3	26±1	26±2	19±2	17±1	20±4	19±3
53-55	26±2	25±1	25±1	24±2	18±1	17±1	18±1	18±3
56-59	26±1	25±1	25±3	25±2	18±1	16±1	18±3	19±2
60-62	25±2	24±1	24±2	24±2	18±1	18±1	17±1	17±1
63-66	26±2	25±1	26±1	26±2	19±2	18±1	20±1	19±1
67-69	23±2	23±1	25±2	24±3	18±2	16±1	18±2	17±2
70-73	26±2	25±1	26±2	26±4	19±3	18±3	20±3	20±3
77-80	26±1	24±1[b]	26±1	26±2	18±1	18±1	19±1	19±1
81-83	25±1	24±2	26±1	25±2	18±2	17±1	19±2	18±1
84-87	26±1	25±1	26±1	26±2	19±2	18±1	20±3	19±1
88-90	25±2	25±1	26±1	25±2	18±3	17±2	19±3	18±1
91-94	25±1	25±1	25±1	25±2	18±1	17±1	18±1	18±2
119-122	24±5	26±1	26±2	24±2	19±1	19±2	19±4	19±2
123-125	26±2	26±1	25±2	25±2	20±2	19±2	19±4	19±1
147-150	25±2	25±1	25±1	25±2	19±2	18±1	19±2	18±2
151-153	24±3	23±1	24±1	24±2	17±2	17±2	18±2	17±2
180-181	29±5	26±1	27±1	26±2	20±1	19±3	21±5	20±2
182-185	25±3	25±1	24±3	25±4	18±1	18±1	19±2	19±1
203-206	26±3	25±4	24±1	24±3	17±1	20±3	19±2	19±2
207-209	25±4	25±4	27±1	26±5	20±2	23±4	20±2	22±3
231-234	26±2	27±3	26±2	25±2	20±2	21±3	22±4	20±2
235-237	25±2	26±1	26±1	26±2	19±2	20±3	22±5	20±2
259-262	25±1	26±2	26±1	25±2	18±2	19±2	20±3	19±2
263-265	26±2	26±2	25±1	26±2	19±2	19±3	21±2	20±2
287-290	25±2	25±1	25±1	25±2	20±5	19±2	19±2	19±2
291-293	26±2	26±2	26±2	26±2	19±2	19±2	19±2	20±2
315-318	24±1	25±1	25±1	25±2	18±2	19±2	20±2	19±2
319-321	27±3	27±1	27±1	26±3	21±2	20±2	21±4	21±2
350-353	25±3	26±3	28±3	24±4	19±3	21±3	20±2	20±2
378-381	25±4	26±4	27±5	26±3	18±6	21±4	21±3	19±2
406-409	25±2	25±2	24±2	25±2	19±3	19±2	19±2	20±2
434-437	25±6	26±2	23±4	24±4	19±2	19±1	19±1	21±2
469-472	20±5	24±6	22±5	24±3	18±3	19±5	16±6	17±4
497-500	25±2	24±4	26±3	25±1	20±2	21±5	23±6	20±2
532-535	24±2	25±3	24±1	24±3	20±3	18±2	19±3	19±3
560-563	26±4	27±6	29±6	25±2	24±5	24±4	25±4	24±6
588-591	23±3	24±2	23±3	24±2	21±3	19±2	21±4	21±2
623-626	26±3	22±4[b]	24±1	24±2	21±3	20±2	21±3	23±4
651-654	21±6	23±3	21±5	22±5	20±4	19±3	19±2	21±4
686-689	22±2	26±7	22±3	23±6	20±3	20±6	21±5	20±2
714-717	21±2	20±4	21±5	22±3	21±5	21±3	19±3	20±4

[a] grams/rat/day.

[b] Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21301

TABLE 7

CUMULATIVE MORTALITY DATA OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR TWO YEARS

	Dose Level (mg/kg/day)			
	0	30	10	3
	No. Dead (% Dead)	No. Dead (% Dead)	No. Dead (% Dead)	No. Dead (% Dead)
Original No. in Group	86	50	50	50
Days on Test				
0-30	0	0	0	0
31-60	0	0	0	0
61-90	1(1.2)	0	0	0
91-120	1(1.2)	0	0	0
121-150	1(1.2)	0	0	0
151-180	1(1.2)	0	0	0
181-210	1(1.2)	0	0	0
211-240	1(1.2)	0	0	0
241-270	1(1.2)	0	1(2.0)	0
271-300	2(2.3)	0	1(2.0)	0
301-330	2(2.3)	0	1(2.0)	0
331-360	2(2.3)	0	1(2.0)	1(2.0)
361-390	2(2.3)	2(4.0)	2(4.0)	2(4.0)
391-420	5(5.8)	2(4.0)	2(4.0)	3(6.0)
421-450	6(7.0)	2(4.0)	4(8.0)	4(8.0)
451-480	9(10.5)	4(8.0)	9(18.0)	6(12.0)
481-510	10(11.6)	6(12.0)	12(24.0)[a]	10(20.0)
511-540	16(18.6)	8(16.0)	22(44.0)[a]	12(24.0)
541-570	23(26.7)	11(22.6)	24(48.0)[a]	14(28.0)
571-600	32(37.2)	16(32.0)	29(58.0)[a]	23(46.0)
601-630	47(54.6)	19(38.0)	37(74.0)[a]	30(60.0)
631-660	67(77.9)	24(48.0)[a]	38(76.0)	32(64.0)[a]
661-690	74(86.0)	27(54.0)[a]	42(84.0)	34(68.0)[a]
691-720	77(89.5)	32(64.0)[a]	45(90.0)	38(76.0)[a]
721-728	79(91.7)	39(78.0)[a]	46(92.0)	40(80.0)[a]
Total No. of Rats on Study	86	50	50	50

[a]Statistically significant difference from control values by Fisher's Exact Probability Test, $p<0.05$.

21302

TABLE 8

CUMULATIVE MORTALITY DATA OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR TWO YEARS

	Dose Level (mg/kg/day)			
	0	30	10	3
	No. Dead (% Dead)	No. Dead (% Dead)	No. Dead (% Dead)	No. Dead (% Dead)
Original No. in Group	86	50	50	50
Days on Test				
0-30	0	0	0	0
31-60	0	0	0	0
61-90	0	0	0	0
91-120	0	0	0	1(2.0)
121-150	0	0	0	1(2.0)
151-180	0	0	1(2.0)	1(2.0)
181-210	1(1.2)	0	1(2.0)	1(2.0)
211-240	1(1.2)	0	1(2.0)	1(2.0)
241-270	1(1.2)	0	1(2.0)	1(2.0)
271-300	1(1.2)	1(2.0)	1(2.0)	1(2.0)
301-330	2(2.3)	1(2.0)	3(6.0)	2(4.0)
331-360	2(2.3)	2(4.0)	3(6.0)	2(4.0)
361-390	2(2.3)	3(6.0)	3(6.0)	3(6.0)
391-420	4(4.6)	3(6.0)	4(8.0)	3(6.0)
421-450	5(5.8)	3(6.0)	5(10.0)	3(6.0)
451-480	9(10.5)	7(14.0)	8(16.0)	5(10.0)
481-510	12(14.0)	9(18.0)	13(26.0)[a]	7(14.0)
511-540	18(21.0)	11(22.6)	14(28.0)	10(20.0)
541-570	20(23.2)	12(24.0)	15(30.0)	12(24.0)
571-600	25(29.1)	15(30.0)	16(32.0)	16(32.0)
601-630	29(33.7)	18(36.0)	21(42.0)	20(40.0)
631-660	34(39.5)	24(48.0)	25(50.0)	23(46.0)
661-690	41(47.7)	26(52.0)	33(66.0)[a]	26(52.0)
691-720	46(53.5)	27(54.0)	35(70.0)[a]	29(58.0)
721-732	46(53.5)	28(56.0)	38(76.0)[a]	29(58.0)
Total No. of Rats on Study	86	50	50	50

[a]Statistically significant difference from control values by Fisher's Exact Probability Test, $p<0.05$.

21303

TABLE 9

RATS WITH PALPABLE MASSES MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR TWO YEARS

Dose (mg/kg/day)	Original Number of Animals	Month of Study and Cumulative Number of Rats with Palpable Masses																							
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Males																									
0	86	0	0	1	1	1	1	2	1	0	1	1	1	1	2	5	8	8	12	13	14	15	15	15	15
30	50	0	0	0	0	0	0	0	1	1	0	0	0	1	2	2	2	4	4	5	9	7	7	8	8
10	50	0	0	0	0	0	0	1	1	1	1	3	3	6[a]	6[a]	6	7	9	10	11	12	13	13	13	13
3	50	0	0	0	0	0	0	0	0	0	0	0	0	1	2	3	8	9	11	11	12	12	14	14	14
Females																									
0	86	0	0	0	0	0	0	0	0	1	2	6	9	14	18	27	37	45	53	55	59	60	67	69	69
30	50	0	0	0	0	0	0	1	1	6[a]	6[a]	6	7	7	12	14	16	23	28	31	35	38	41	42	44
10	50	0	0	0	0	0	0	0	2	2	2	4	6	14	19[a]	25	27	29	31	32	38	39	40	42	42
3	50	0	0	0	0	0	0	0	0	1	1	4	7	10	12	17	23	29	31	33	36	37	40	40	41

Palpable masses may include tumors, preputial gland abscesses, etc. which sometimes undergo drainage and subsequent regression.

[a]Statistically significant difference from control values by Fisher's Exact Probability Test, $p<0.05$.

21304

TABLE 10

MEAN HEMATOLOGIC VALUES OF MALE AND FEMALE RATS ON DAYS 82 AND 83
OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex		PCV Percent	RBC x $10^6/mm^3$	Hgb g/100 ml	WBC x $10^3/mm^3$	WBC Differential Count (%) Neut	Lymph	Mono	Eosin	Baso	Thrombocytes x$10^6/mm^3$	Reticulocytes %
0	M	Mean	54.2	9.21	17.0	13.7	12	84	3	1	0	1.393	1.2
		±S.D.	±1.3	±0.34	±0.7	±2.2						±.265	
30	M	Mean	54.0	9.22	17.1	14.1	10	88	2	0	0	1.298	1.2
		±S.D.	±1.4	±0.20	±0.5	±1.8						±.128	
10	M	Mean	53.2	9.18	17.3	15.3	11	86	2	1	0	1.202	1.2
		±S.D.	±1.8	±0.52	±0.5	±2.0						±.182	
3	M	Mean	52.5	8.91	17.3	14.5	11	87	1	1	0	1.295	0.6
		±S.D.	±2.2	±0.35	±0.8	±2.3						±.278	
0	F	Mean	50.1	8.19	16.2	12.4	10	88	1	1	0	1.175	1.6
		±S.D.	±1.8	±0.43	±0.6	±1.6						±.145	
30	F	Mean	52.5	8.56	16.4	12.0	11	87	1	1	0	1.183	1.3
		±S.D.	±5.4	±0.63	±1.2	±2.8						±.203	
10	F	Mean	50.6	8.12	15.9	11.5	7	91	1	1	0	0.978	1.4
		±S.D.	±2.3	±0.28	±0.4	±2.3						±.088	
3	F	Mean	50.1	8.15	16.0	11.4	8	91	0	1	0	1.144	1.1
		±S.D.	±2.9	±0.45	±0.9	±2.2						±.187	

No statistically significant differences from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21305

TABLE 11

MEAN HEMATOLOGIC VALUES FOR MALE AND FEMALE RATS
ON DAYS 89 AND 90 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group		PCV Percent	RBC x $10^6/mm^3$	HgB g/100 ml	WBC x $10^3/mm^3$	WBC Differential Count (%) Neut	Lymph	Mono	Eosin	Baso	Throm-bocytes x$10^6/mm^3$	Reticulo-cytes %
0	M	8	MEAN	55.7	9.22	17.4	12.3	11	87	2	0	0	1.204	1.1
			±S.D.	±1.4	±.41	±.8	±2.9						±.194	
30	M	8	MEAN	54.8	9.29	17.5	12.8	11	87	1	1	0	1.276	.9
			±S.D.	±.8	±.32	±.3	±2.4						±.169	
10	M	8	MEAN	55.6	9.41	17.7	12.9	12	86	1	1	0	1.292	1.1
			±S.D.	±3.1	±.36	±1.0	±1.3						±.287	
3	M	8	MEAN	55.4	9.44	17.8	12.7	11	87	1	1	0	1.084	1.1
			±S.D.	±2.4	±.39	±.8	±3.2						±.131	
0	F	8	MEAN	49.3	8.17	15.8	9.4	9	90	1	0	0	1.268	1.4
			±S.D.	±1.1	±.38	±.6	±1.8						±.241	
30	F	8	MEAN	48.8	8.24	16.2	12.0	9	89	1	1	0	1.073	1.7
			±S.D.	±2.9	±.39	±.7	±2.4						±.134	
10	F	8	MEAN	50.3	8.27	16.2	11.5	12	86	1	1	0	1.253	1.1
			±S.D.	±2.2	±.52	±.9	±3.5						±.121	
3	F	8	MEAN	50.7	8.11	16.3	11.3	8	90	1	1	0	1.171	1.4
			±S.D.	±2.0	±.46	±.7	±1.8						±.125	

No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

213906

TABLE 12

MEAN HEMATOLOGIC VALUES FOR MALE AND FEMALE RATS
ON DAYS 364 AND 365 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group		PCV Percent	RBC x 10^6/mm^3	HgB g/100 ml	WBC x 10^3/mm^3	WBC Differential Count (%) Neut	Lymph	Mono	Eosin	Baso	Thrombocytes x10^6/mm^3	Reticulocytes %
0	M	8	MEAN	50.4	8.34	16.4	12.4	17	79	3	1	0	1.121	1.4
			±S.D.	±4.2	±.71	±1.3	±5.2						±.206	
30	M	8	MEAN	49.9	8.24	16.4	13.5	15	81	2	2	0	1.161	.9
			±S.D.	±2.2	±.24	±.7	±2.3						±.299	
10	M	8	MEAN	49.9	8.10	16.2	12.5	17	79	2	2	0	1.101	1.1
			±S.D.	±3.5	±.68	±.8	±1.0						±.195	
3	M	8	MEAN	49.6	8.30	16.4	14.3	18	78	2	2	0	1.028	1.2
			±S.D.	±5.2	±.92	±1.6	±3.9						±.156	
0	F	8	MEAN	47.1	7.58	15.2	10.7	16	81	1	2	0	.938	1.1
			±S.D.	±1.4	±.34	±.4	±3.5						±.098	
30	F	8	MEAN	47.6	7.72	15.7	11.3	13	84	1	2	0	.903	1.2
			±S.D.	±2.2	±.49	±.9	±2.8						±.125	
10	F	8	MEAN	46.4	7.79	15.3	8.6	17	80	1	2	0	1.052	1.1
			±S.D.	±2.1	±.45	±.7	±2.2						±.170	
3	F	8	MEAN	46.3	7.79	15.3	8.9	15	80	2	3	0	.951	1.0
			±S.D.	±2.7	±.51	±1.0	±2.9						±.089	

No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

213307

TABLE 13

MEAN HEMATOLOGIC VALUES FOR MALE AND FEMALE RATS
ON DAYS 726 AND 727 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group		PCV Percent	RBC x $10^6/mm^3$	HgB g/100 ml	WBC x $10^3/mm^3$	WBC Differential Count (%) Neut	Lymph	Mono	Eosin	Baso	Thrombocytes x$10^6/mm^3$	Reticulocytes %
0	M	8	MEAN	44.3	6.79	14.8	14.8	42	49	8	1	0	1.066	1.7
			±S.D.	±5.4	±1.01	±1.7	±6.5						±.262	
30	M	8	MEAN	47.7	7.62	15.4	14.4	33	59	7	1	0	.878	2.1
			±S.D.	±3.4	±.55	±1.0	±4.8						±.107	
10	M	4	MEAN	47.3	7.50	15.6	17.6	42	48	8	2	0	.840	1.8
			±S.D.	±3.4	±.43	±.9	±5.2						±.055	
3	M	8	MEAN	44.2	7.05	14.9	12.9	31	61	7	1	0	.932	1.1
			±S.D.	±4.3	±.66	±1.2	±4.2						±.193	
0	F	8	MEAN	47.3	6.91	14.9	8.0	25	66	7	2	0	1.200	1.6
			±S.D.	±3.8	±.55	±1.1	±1.8						±.156	
30	F	8	MEAN	44.4	6.04	14.3	8.1	27	66	6	1	0	1.134	3.7
			±S.D.	±5.6	±1.20	±1.9	±1.8						±.279	
10	F	8	MEAN	44.7	6.40	14.9	8.2	26	65	7	2	0	.991	2.3
			±S.D.	±4.2	±1.03	±1.8	±2.9						±.105	
3	F	8	MEAN	45.2	6.33	15.1	9.3	32	60	7	1	0	1.222	1.5
			±S.D.	±5.0	±.64	±1.8	±2.0						±.218	

No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

21308

TABLE 14

SUMMARY OF URINALYSES OF MALE AND FEMALE RATS ON DAYS 82 AND 83
OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	Specific Gravity[a]	pH	Sugar	Protein	Ketones	Occult Blood	Bilirubin
0	M	1.030±0.006	6.5-7.0	neg.(8)	1+(1),2+(3) 3+(3),4+(1)	neg.(8)	neg.(7),mod.(1)	neg.(8)
30	M	1.027±0.007	6.5-7.0	neg.(8)	1+(1),2+(4) 3+(3),4+(0)	neg.(8)	neg.(8)	neg.(8)
10	M	1.035±0.005	6.0-7.0	neg.(8)	1+(2),2+(1) 3+(5),4+(0)	neg.(8)	neg.(6),small(1) mod.(1)	neg.(8)
3	M	1.038±0.010	6.0-7.0	neg.(8)	1+(1),2+(2) 3+(4),4+(1)	neg.(8)	neg.(7),small(1)	neg.(8)
0	F	1.043±0.006	6.5-7.0	neg.(8)	trace(3),1+(3) 2+(1),3+(1)	neg.(8)	neg.(8)	neg.(8)
30	F	1.042±0.008	6.0-7.0	neg.(8)	trace(6),1+(1) 2+(1)	neg.(8)	neg.(8)	neg.(8)
10	F	1.044±0.013	6.0-7.5	neg.(8)	1+(5),2+(2) 3+(1)	neg.(8)	neg.(8)	neg.(8)
3	F	1.050±0.010	6.0-8.0	neg.(8)	trace(1),1+(2) 2+(2),3+(3)	neg.(8)	neg.(8)	neg.(8)

pH listed as range of values.
Protein content quantitated on basis of trace to 4+.

[a]No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

21309

TABLE 15

SUMMARY OF URINALYSES OF MALE AND FEMALE RATS
ON DAY 89 AND 90 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group	Specific Gravity[a]	pH	Glucose	Protein	Ketones	Occult Blood	Bilirubin
0	M	8	1.044±.006	(6.0-7.5)	Neg(8)	1+(1) 2+(6) 3+(1)	Neg(8)	Neg(8)	Neg(8)
30	M	8	1.044±.008	(6.5-7.0)	Neg(8)	1+(2) 2+(3) 3+(3)	Neg(8)	Neg(8)	Neg(8)
10	M	8	1.041±.009	(6.0-6.5)	Neg(8)	1+(2) 2+(3) 3+(3)	Neg(8)	Neg(8)	Neg(8)
3	M	8	1.042±.007	(6.0-7.5)	Neg(8)	1+(4) 2+(2) 3+(2)	Neg(8)	Neg(8)	Neg(8)
0	F	8	1.030±.008	(6.0-7.0)	Neg(8)	Trace(6) 3+(2)	Neg(8)	Neg(8)	Neg(8)
30	F	8	1.026±.004	(6.0-7.0)	Neg(8)	Trace(7) 2+(1)	Neg(8)	Neg(8)	Neg(8)
10	F	8	1.025±.007	(6.0-7.0)	Neg(8)	Trace(3) 1±(3) 2+(1) 3+(1)	Neg(8)	Neg(7) Small(1)	Neg(8)
3	F	8	1.029±.006	(6.0-7.5)	Neg(8)	Trace(3) 1+(2) 2+(2) 3+(1)	Neg(8)	Neg(8)	Neg(8)

[a] No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.
pH listed as range of values.
Protein content quantitated on basis of trace to 4+.

21310

TABLE 16

SUMMARY OF URINALYSES VALUES OF MALE AND FEMALE RATS
ON DAY 364 AND 365 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group	Specific Gravity	pH	Glucose	Protein	Ketones	Occult Blood	Bilirubin	Urobilinogen Ehrlich Units/dl
0	M	8	1.041±.008	(5.5-6.0)	Neg(8)	3+(8)	Neg(8)	Neg(7) Small(1)	Neg(8)	0.1(8)
30	M	8	1.039±.009	(6.0-7.0)	Neg(8)	3+(8)	Neg(8)	Neg(8)	Neg(8)	0.1(8)
10	M	8	1.031[a]±.009	(5.5-6.5)	Neg(8)	3+(8)	Neg(8)	Neg(7) Small(1)	Neg(8)	0.1(8)
3	M	8	1.041±.007	(6.0-6.5)	Neg(8)	3+(7)4+(1)	Neg(8)	Neg(8)	Neg(8)	0.1(8)
0	F	8	1.025±.009	(5.5-6.5)	Neg(8)	1+(1)2+(4) 3+(3)	Neg(8)	Neg(8)	Neg(8)	0.0(1)0.1(7)
30	F	8	1.022±.006	(5.5-7.0)	Neg(8)	1+(3)2+(2) 3+(3)	Neg(8)	Neg(8)	Neg(8)	0.0(1)0.1(7)
10	F	8	1.029±.009	(6.0-6.5)	Neg(8)	2+(3)3+(5)	Neg(8)	Neg(7) Small(1)	Neg(8)	0.1(8)
3	F	8	1.031±.010	(5.5-6.0)	Neg(8)	1+(1)2+(1) 3+(6)	Neg(8)	Neg(8)	Neg(8)	0.1(8)

[a]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.
pH listed as range of values.
Protein content quantitated on basis of trace to 4+.

21311

TABLE 17

SUMMARY OF URINALYSES OF MALE AND FEMALE RATS
ON DAYS 726 AND 727 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group	Specific Gravity[a]	pH	Glucose	Protein	Ketones	Occult Blood	Bilirubin	Urobilinogen Ehrlich Units/dl
0	M	7	1.023±.008	(6.0-7.0)	Neg(7)	3+(7)	Neg(7)	Neg(5) Small(1) Moderate(1)	Neg(7)	0.1(6)1.0(1)
30	M	8	1.027±.009	(6.0-6.5)	Neg(8)	3+(8)	Neg(8)	Neg(8)	Neg(8)	0.1(7)1.0(1)
10	M	4	1.022±.003	(6.0)	Neg(4)	3+(4)	Neg(4)	Neg(4)	Neg(4)	0.1(4)
3	M	8	1.024±.007	(6.0-6.5)	Neg(8)	3+(7)2+(1)	Neg(8)	Neg(8)	Neg(8)	0.1(8)
0	F	8	1.029±.009	(6.0-7.0)	Neg(8)	2+(2)3+(6)	Neg(8)	Neg(6) Trace(1) Small(1)	Neg(8)	0.1(8)
30	F	8	1.025±.007	(6.0-7.0)	Neg(8)	1+(1)2+(1) 3+(6)	Neg(8)	Neg(5) Trace(1) Small(1) Large(1)	Neg(8)	0.1(8)
10	F	7	1.030±.014	(6.0-7.0)	Neg(7)	2+(3)3+(3) 4+(1)	Neg(7)	Neg(5) Trace(1) Large(1)	Neg(7)	0.1(7)
3	F	8	1.032±.010	(6.0-7.0)	Neg(8)	2+(1)3+(7)	Neg(8)	Neg(6) Small(2)	Neg(8)	0.1(8)

[a]No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.
pH listed as range of values.
Protein content quantitated on basis of trace to 4+.

21312

3	M	15	40 ±10	30.5 ±2.8	23.1 ±4.2	0.76 ±0.11	4.5 ±2.0	0.15 ±0.07	113 ±35	3.7 ±1.1
0	F	15	40 ±12	20.0 ±3.1	7.7 ±2.0	0.39 ±0.11	1.8 ±0.9	0.09 ±0.06	74 ±36	3.6 ±1.4
30	F	15	48 ±17	17.2 ±3.1	8.6 ±2.6	0.49 ±0.13	1.7 ±0.7	0.10 ±0.05	88 ±56	4.8 ±3.3
10	F	15	50 ±16	20.3 ±2.7	9.1 ±2.2	0.45 ±0.11	1.9 ±1.2	0.10 ±0.08	86 ±27	4.3 ±1.4
3	F	15	39 ±15	18.6 ±3.3	8.3 ±1.8	0.46 ±0.14	2.0 ±1.3	0.11 ±0.07	60 ±33	3.1 ±1.6

[a]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21313

TABLE 18

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN, UROPORPHYRIN AND DELTA-ALA (DELTA-AMINOLEVULINIC ACID) OF MALE AND FEMALE RATS FOR UP TO DAY 106 OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	Number Samples	Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin / mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin / mg Creatinine	delta-ALA µg/48 hr.	µg delta-ALA / mg Creatinine
0	M	15	38 ±8	29.4 ±5.4	17.5 ±4.9	0.60 ±0.16	4.1 ±1.8	0.15 ±0.09	116 ±22	4.1 ±1.1
30	M	15	49[a] ±11	28.8 ±3.2	43.6[a] ±9.3	1.48[a] ±0.30	7.7[a] ±4.7	0.28[a] ±0.17	94 ±33	3.3 ±1.3
10	M	15	36 ±14	29.1 ±7.6	24.6[a] ±9.6	0.84[a] ±0.22	5.0 ±3.1	0.19 ±0.16	102 ±36	3.7 ±1.9

3	M	5	14.9 ±7.3	10.5 ±4.5	12.6 ±7.8	1.15 ±0.38	0.8 ±0.6	0.08 ±0.04
0	F	5	18.4 ±4.5	8.5 ±0.9	4.4 ±1.0	0.52 ±0.11	0.4 ±0.1	0.05 ±0.01
30	F	5	21.1 ±9.1	7.3 ±1.4	6.5 ±2.8	0.89 ±0.36	0.1[a] ±0.1	0.02 ±0.02
10	F	5	15.0 ±4.2	8.1 ±0.7	5.1 ±0.5	0.64 ±0.10	0.2 ±0.1	0.03 ±0.01
3	F	5	17.7 ±8.3	8.2 ±1.3	4.3 ±0.6	0.53 ±0.07	0.4 ±0.1	0.04 ±0.02

[a]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

TABLE 19

URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN AND UROPORPHYRIN OF MALE AND FEMALE RATS AT TERMINATION OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	Number Samples	Total Urine Vol. 24 hr. (ml)	Creatinine mg/24 hr.	Coproporphyrin µg/24 hr.	µg Coproporphyrin / mg Creatinine	Uroporphyrin µg/24 hr.	µg Uroporphyrin / mg Creatinine
0	M	5	12.8 ±2.2	12.0 ±1.6	13.7 ±3.6	1.13 ±0.21	0.9 ±0.2	0.07 ±0.02
30	M	5	11.3 ±5.4	10.6 ±3.3	20.4 ±7.2	1.92[a] ±0.22	1.2 ±0.5	0.12 ±0.05
10	M	5	17.8 ±6.9	13.3 ±2.2	18.8 ±4.9	1.47 ±0.50	0.9 ±0.5	0.07 ±0.05

3	M	5	MEAN	36	27.4	22.4	.81	2.5	.09	166	6.1
			±S.D.	±4	±2.2	±5.8	±.17	±0.4	±.02	±60	±2.2
0	F	5	MEAN	45	17.7	6.6	.37	1.5	.10	156	8.3
			±S.D.	±23	±6.0	±2.7	±.08	±0.1	±.05	±82	±4.0
30	F	5	MEAN	31	14.9	9.7	.65[a]	1.1	.08	116	7.9
			±S.D.	±13	±1.7	±2.1	±.14	±0.2	±.02	±66	±4.6
10	F	5	MEAN	41	17.5	8.3	.47	1.4	.08	152	9.6
			±S.D.	±19	±5.5	±3.5	±.12	±0.8	±.04	±29	±3.9
3	F	5	MEAN	49	19.4	9.7	.50	1.8	.09	140	7.4
			±S.D.	±11	±2.5	±3.7	±.17	±1.0	±.05	±31	±2.1

[a]Statistically significant increase from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21315

TABLE 20

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN, UROPORPHYRIN AND DELTA-ALA (DELTA-AMINO-LEVULINIC ACID) FOR MALE AND FEMALE RATS ON DAYS 95-97 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	# of Animals/ Group		Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin/ mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin/ mg Creatinine	Delta-ALA µg/48 hr.	µg Delta-ALA/ mg Creatinine
0	M	5	MEAN	20	27.5	20.0	.75	2.9	.11	152	5.5
			±S.D.	±10	±4.5	±4.9	±.27	±1.1	±.06	±26	±0.8
30	M	5	MEAN	35	29.1	39.6[a]	1.37[a]	2.9	.10	116	3.9
			±S.D.	±9	±1.6	±4.4	±.22	±0.9	±.04	±34	±1.1
10	M	5	MEAN	34	27.1	31.0[a]	1.14[a]	2.5	.09	142	5.2
			±S.D.	±14	±6.5	±8.7	±.07	±0.8	±.03	±66	±1.8

0	F	5	MEAN	41.0	20.0	9.6	.49	1.5	.077	90	4.4
			±S.D.	±17.9	±4.1	±1.9	±.11	±.2	±.015	±30	±0.8
30	F	5	MEAN	45.2	19.0	16.4[a]	.85[a]	1.7	.089	80	4.2
			±S.D.	±9.4	±3.0	±7.0	±.26	±.2	±.019	±20	±0.2
10	F	5	MEAN	42.2	17.8	9.2	.52	1.3	.076	80	4.8
			±S.D.	±6.8	±2.2	±.8	±.07	±.3	±.020	±10	±0.7
3	F	5	MEAN	51.4	18.6	10.3	.56	1.2	.064	90	4.8
			±S.D.	±19.2	±3.3	±3.5	±.17	±.2	±.009	±30	±0.8

[a]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21316

TABLE 21

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN, UROPORPHYRIN AND DELTA-ALA (DELTA-AMINO-LEVULINIC ACID) FOR MALE AND FEMALE RATS ON DAYS 188-190 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	# of Animals/ Group		Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin/ mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin/ mg Creatinine	Delta-ALA µg/48 hr.	µg Delta-ALA/ mg Creatinine
0	M	5	MEAN	33.5	30.2	19.2	.63	2.2	.072	84	2.8
			±S.D.	±10.3	±4.5	±6.4	±.14	±0.7	±.012	±24	±0.6
30	M	5	MEAN	39.6	30.6	47.4[a]	1.55	3.4	.111	110	3.6
			±S.D.	±9.9	±1.5	±10.3	±.36	±1.5	±.048	±16	±0.6
10	M	5	MEAN	42.0	31.2	29.8	.97	2.4	.075	120	3.9
			±S.D.	±8.1	±4.3	±4.8	±.17	±1.1	±.025	±34	±1.1
3	M	5	MEAN	38.8	30.2	25.6	.87	2.3	.075	110	3.6
			±S.D.	±10.4	±4.5	±3.1	±.19	±0.6	±.016	±26	±0.8

0	F	5	MEAN	60.8	23.8	13.5	.56	1.8	.075	70	3.0
			±S.D.	±26.0	±7.1	±4.2	±.07	±.7	±.013	±20	±0.3
30	F	5	MEAN	58.8	23.2	19.8[a]	.86[a]	1.4	.060	80	3.4
			±S.D.	±11.9	±1.8	±3.6	±.14	±.6	±.024	±30	±1.2
10	F	5	MEAN	68.0	23.0	15.4	.67	2.2	.097	80	3.3
			±S.D.	±12.1	±2.9	±1.7	±.07	±.8	±.033	±10	±0.3
3	F	5	MEAN	56.4	23.6	13.8	.58	1.5	.064	70	2.9
			±S.D.	±16.2	±2.9	±3.6	±.09	±.2	±.004	±10	±0.4

[a]Statistically significant increase from control means by analysis of variance and Dunnett's test, $p<0.05$.

21317

TABLE 22

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN, UROPORPHYRIN AND DELTA-ALA (DELTA-AMINO-LEVULINIC ACID) FOR MALE AND FEMALE RATS ON DAYS 361-363 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	# of Animals/ Group		Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin/ mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin/ mg Creatinine	Delta-ALA µg/48 hr.	µg Delta-ALA/ mg Creatinine
0	M	4	MEAN	39.5	36.5	19.3	.53	2.2	.061	110	2.8
			±S.D.	±7.9	±4.0	±2.6	±.03	±1.1	±.031	±20	±0.3
30	M	5	MEAN	41.0	34.8	44.0[a]	1.26[a]	2.1	.059	80	2.3
			±S.D.	±11.4	±2.6	±8.3	±.16	±.8	±.022	±10	±0.4
10	M	5	MEAN	53.0	36.4	24.2	.67	3.1	.083	110	3.1
			±S.D.	±24.6	±3.5	±10.0	±.26	±1.2	±.031	±30	±0.7
3	M	5	MEAN	41.8	36.0	24.3	.71	2.4	.069	110	3.0
			±S.D.	±5.4	±5.0	±5.9	±.13	±1.0	±.023	±10	±0.5

0	F	5	MEAN	53.6	18.2	10.7	.60	5.3	.30
			±S.D.	±31.5	±6.0	±3.7	±.17	±1.6	±.05
30	F	5	MEAN	79.0	17.2	19.3[a]	1.14[a]	8.9	.52[a]
			±S.D.	±32.9	±4.8	±5.7	±.23	±3.2	±.14
10	F	5	MEAN	65.8	21.2	16.4	.79	7.8	.38
			±S.D.	±16.0	±3.6	±3.8	±.27	±2.5	±.17
3	F	5	MEAN	72.4	21.2	16.3	.81	6.8	.34
			±S.D.	±20.2	±5.1	±1.5	±.22	±.9	±.11

[a]Statistically significant increase from control mean by analysis of variance and Dunnett's test, $p<0.05$.

21318

TABLE 23

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN AND UROPORPHYRIN FOR MALE AND FEMALE RATS ON DAYS 564-566 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	# of Animals/ Group		Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin/ mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin/ mg Creatinine
0	M	4	MEAN	48.2	30.0	28.5	.98	11.0	.38
			±S.D.	±3.3	±6.4	±6.4	±.29	±.6	±.06
30	M	5	MEAN	93.8[a]	27.8	36.4	1.42	7.8	.31
			±S.D.	±40.1	±9.9	±16.0	±.73	±1.6	±.15
10	M	5	MEAN	71.4	30.0	32.8	1.09	12.8	.43
			±S.D.	±20.7	±3.3	±14.5	±.45	±2.9	±.08
3	M	5	MEAN	67.6	27.2	39.4	1.47	12.5	.46
			±S.D.	±28.8	±6.1	±15.3	±.52	±3.4	±.11

3	M	5	MEAN	95.2	24.2	15.2	.66	3.0	.13	180	7.8
			±S.D.	±22.9	±4.9	±3.6	±.24	±.9	±.05	±60	±2.4
0	F	5	MEAN	57.6	19.0	5.8	.30	1.7	.09	130	7.0
			±S.D.	±5.6	±2.5	±1.9	±.09	±.6	±.04	±10	±1.1
30	F	5	MEAN	54.8	16.0	8.0	.50	2.5	.16	130	8.4
			±S.D.	±21.7	±4.2	±9.8	±.33	±1.8	±.13	±50	±3.6
10	F	4	MEAN	60.8	18.5	9.8	.59	2.5	.15	140	9.1
			±S.D.	±14.2	±7.0	±2.3	±.25	±1.0	±.07	±20	±4.5
3	F	5	MEAN	71.0	18.6	6.5	.36	3.4	.18	170	9.4
			±S.D.	±21.2	±2.9	±2.3	±.12	±1.5	±.07	±30	±2.8

[a]Statistically significant difference from control mean by analysis of variance and Dunnett's test, $p<0.05$.

21319

TABLE 24

MEAN URINARY EXCRETION VALUES OF CREATININE, COPROPORPHYRIN, UROPORPHYRIN AND DELTA-ALA (DELTA-AMINO-LEVULINIC ACID) FOR MALE AND FEMALE RATS ON DAYS 698-700 OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose 2,4,5-T mg/kg/day	Sex	# of Animals/ Group		Total Urine Vol. 48 hr. (ml)	Creatinine mg/48 hr.	Coproporphyrin µg/48 hr.	µg Coproporphyrin/ mg Creatinine	Uroporphyrin µg/48 hr.	µg Uroporphyrin mg Creatinine	Delta-ALA µg/48 hr.	µg Delta-ALA/ mg Creatinine
0	M	5	MEAN	90.2	24.8	14.6	.60	4.7	.20	120	5.1
			±S.D.	±27.4	±5.4	±6.0	±.24	±1.8	±.10	±20	±1.3
30	M	5	MEAN	99.6	19.6	22.8	1.15[a]	4.0	.28	130	9.6
			±S.D.	±40.9	±9.9	±13.0	±.17	±1.5	±.20	±20	±8.7
10	M	5	MEAN	64.4	24.8	9.7	.38	3.0	.12	100	4.0
			±S.D.	±39.4	±4.5	±4.4	±.16	±1.3	±.05	±20	±0.8

TABLE 25

MEAN CLINICAL CHEMISTRY VALUES OF MALE AND FEMALE RATS AT TERMINATION OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex		BUN mg/100 ml	SGPT mU/ml	AP mU/ml	Bilirubin Total mg/100 ml	Bilirubin Direct mg/100 ml	Bilirubin Indirect mg/100 ml	Protein Total gm/100 ml	Albumin gm/100 ml	Globulin gm/100 ml
0	M	Mean	21	54	111	.1	0	.1	6.4	2.2	4.2
		±S.D.	±5	±9	±25	0	0	0	0.2	0.2	0.2
30	M	Mean	19	57	117	.1	0	.1	6.2	2.2	4.0
		±S.D.	±2	±10	±36	0	0	0	0.2	0.2	0.3
10	M	Mean	20	53	100	.1	0	.1	6.2	2.2	4.1
		±S.D.	±2	±10	±18	0	0	0	0.3	0.3	0.3
3	M	Mean	20	50	114	.1	0	.1	6.2	2.2	4.1
		±S.D.	±3	±5	±24	0	0	0	0.3	0.2	0.3
0	F	Mean	19	44	102	.1	.1	0	6.6	2.6	4.0
		±S.D.	±2	±3	±21	0	0	0	0.3	0.2	0.2
30	F	Mean	21	46	115	.1	0	.1	6.8	2.8	4.1
		±S.D.	±3	±5	±18	0	0	0	0.4	0.1	0.3
10	F	Mean	22	56[a]	111	.1	.1	0	6.8	2.6	4.2
		±S.D.	±3	±12	±35	0	0	0	0.3	0.3	0.3
3	F	Mean	20	52	90	.1	.1	0	6.8	2.8	4.0
		±S.D.	±3	±4	±16	0	0	0	0.3	0.2	0.3

[a]Statistically significant increase from control mean using analysis of variance and Dunnett's test, $p<0.05$.

0132C

TABLE 26

MEAN CLINICAL CHEMISTRY VALUES FOR MALE AND FEMALE RATS AT TERMINATION OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex	# of Animals/ Group		BUN mg/100 ml	SGPT mU/ml	AP mU/ml	Total Bilirubin mg/100 ml	Direct Bilirubin mg/100 ml	Indirect Bilirubin mg/100 ml	Total Protein gm/100 ml	Albumin gm/100 ml	Globulin gm/100 ml
0	M	6	MEAN	56	40	90	.2	.1	.1	5.7	1.6	4.0
			±S.D.	58	8	17	0	0	0	0.4	0.2	0.2
30	M	10	MEAN	40	48	108	.2	.1	.1	6.0	1.9	4.1
			±S.D.	47	11	61	0	0	0	0.3	0.3	0.4
10	M	4	MEAN	36	35	96	.2	.1	.1	5.6	1.6	4.0
			±S.D.	24	6	6	0	0	0	0.1	0.0	0.6
3	M	9	MEAN	25	38	74	.2	.1	.1	6.0	1.9	4.1
			±S.D.	20	5	24	0	0	0	0.4	0.2	0.5
0	F	10	MEAN	17	42	68	.2	.1	.2	6.5	2.6	3.9
			±S.D.	6	10	47	.1	0	.1	0.6	0.4	0.4
30	F	10	MEAN	19	37	59	.2	.1	.1	6.3	2.6	3.7
			±S.D.	6	6	19	.1	0	.1	0.2	0.5	0.5
10	F	9	MEAN	16	40	56	.2	.1	.1	6.4	2.6	3.7
			±S.D.	3	9	18	.1	.1	.1	0.3	0.5	0.7
3	F	10	MEAN	56	42	63	.2	.1	.2	6.2	2.2	4.0
			±S.D.	101	20	28	.1	0	.1	0.5	0.7	0.7

No statistically significant differences from control means using analysis of variance and Dunnett's test, $p<0.05$.

21321

TABLE 27

MEAN BODY WEIGHTS, ORGAN WEIGHTS, AND ORGAN/BODY WEIGHT RATIOS OF MALE AND FEMALE RATS AT TERMINATION OF A 118-119 DAY DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day	Sex		Fasted Body Weight g	Organ Weights (g and g/100 g Body Weights) Brain g	Brain g/100g	Heart g	Heart g/100g	Liver g	Liver g/100g	Kidneys g	Kidneys g/100g	Testes g	Testes g/100g	Spleen g	Spleen g/100g	Thymus g	Thymus g/100g
0	M	Mean	466	1.85	0.40	1.40	0.30	12.55	2.69	3.28	0.70	4.05	0.87	0.85	0.18	0.35	0.08
		±S.D.	±27	±0.09	±0.02	±0.11	±0.02	±1.87	±0.39	±0.44	±0.09	±0.31	±0.06	±0.12	±0.03	±0.08	±0.02
30	M	Mean	474	1.86	0.39	1.41	0.30	13.45	2.83	3.74	0.79[a]	4.01	0.85	0.89	0.19	0.37	0.08
		±S.D.	±36	±0.08	±0.02	±0.15	±0.03	±1.94	±0.26	±0.42	±0.06	±0.33	±0.09	±0.14	±0.03	±0.06	±0.01
10	M	Mean	489	1.85	0.38	1.39	0.29	13.25	2.69	3.57	0.73	4.00	0.83	0.92	0.19	0.40	0.08
		±S.D.	±61	±0.08	±0.05	±0.12	±0.02	±2.32	±0.17	±0.46	±0.05	±0.32	±0.11	±0.07	±0.02	±0.11	±0.02
3	M	Mean	418	1.85	0.39	1.38	0.29	13.57	2.81	3.42	0.72	4.14	0.87	0.87	0.18	0.38	0.08
		±S.D.	±43	±0.07	±0.04	±0.07	±0.02	±1.95	±0.23	±0.37	±0.05	±0.31	±0.10	±0.10	±0.02	±0.10	±0.02
												Ovaries/Uterus					
0	F	Mean	298	1.81	0.61	0.93	0.31	7.84	2.63	1.94	0.65	0.78	0.27	0.75	0.25	0.32	0.11
		±S.D.	±14	±0.08	±0.03	±0.12	±0.04	±0.55	±0.11	±0.39	±0.11	±0.25	±0.09	±0.10	±0.04	±0.07	±0.03
30	F	Mean	290	1.80	0.62	0.85	0.29	7.72	2.66	1.98	0.68	0.78	0.27	0.64	0.22	0.32	0.11
		±S.D.	±17	±0.09	±0.03	±0.18	±0.06	±0.70	±0.17	±0.17	±0.03	±0.22	±0.08	±0.15	±0.05	±0.10	±0.04
10	F	Mean	292	1.78	0.61	0.94	0.32	7.72	2.64	1.89	0.65	0.68	0.24	0.67	0.23	0.31	0.11
		±S.D.	±24	±0.06	±0.06	±0.06	±0.02	±0.91	±0.17	±0.19	±0.05	±0.19	±0.08	±0.18	±0.06	±0.07	±0.03
3	F	Mean	292	1.80	0.62	1.02	0.35	8.13	2.78	2.06	0.70	0.87	0.30	0.72	0.25	0.33	0.11
		±S.D.	±12	±0.05	±0.03	±0.07	±0.02	±0.74	±0.19	±0.20	±0.06	±0.25	±0.09	±0.06	±0.02	±0.09	±0.03

[a]Statistically significant increase from mean using analysis of variance and Dunnett's test, $p<0.05$.

21322

TABLE 28

MEAN ORGAN WEIGHTS AND ORGAN TO BODY WEIGHT RATIOS FOR MALE RATS AT TERMINATION OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day		Fasted Body Weight g	Organ Weights (g and g/100 g Body Weights)													
			Brain		Heart		Liver		Kidneys		Testes		Spleen		Thymus	
			g	g/100g	g	g/100g	g	g/100g	g	g/100g	g	g/100g	g	g/100g	g	g/100g
0	MEAN	508	2.00	0.40	2.07	0.41	17.72	3.49	5.86	1.14	3.93	0.77	1.29	0.25	0.10	0.02
	±S.D.	±54	±0.06	±0.04	±0.28	±0.07	±2.27	±0.35	±0.49	±0.11	±0.65	±0.11	±0.19	±0.02	±0.03	±0.01
30	MEAN	534	2.01	0.39	1.82[a]	0.35	15.52	2.96	5.03	0.97	3.78	0.70	1.20	0.23	0.11	0.02
	±S.D.	±92	±0.10	±0.07	±0.15	±0.07	±1.81	±0.61	±1.05	±0.26	±0.99	±0.15	±0.30	±0.05	±0.03	±0.01
10	MEAN	532	2.01	0.38	2.08	0.40	18.09	3.45	5.72	1.10	4.18	0.80	1.38	0.27	0.11	0.02
	±S.D.	±71	±0.01	±0.05	±0.19	±0.08	±0.36	±0.49	±0.68	±0.24	±0.05	±0.12	±0.21	±0.08	±0.04	±0.01
3	MEAN	554	2.04	0.38	1.87	0.35	17.59	3.12	5.42	1.02	3.62	0.66	1.24	0.23	0.13	0.02
	±S.D.	±100	±0.09	±0.05	±0.12	±0.05	±2.74	±0.31	±1.23	±0.33	±1.03	±0.20	±0.18	±0.04	±0.05	±0.01

[a]Statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21323

TABLE 29

MEAN ORGAN WEIGHTS AND ORGAN TO BODY WEIGHT RATIOS FOR FEMALE RATS AT TERMINATION OF A TWO-YEAR DIETARY STUDY OF 2,4,5-T

Dose mg/kg/day		Fasted Body Weight g	Organ Weights (g and g/100 g Body Weights) Brain g	Brain g/100g	Heart g	Heart g/100g	Liver g	Liver g/100g	Kidneys g	Kidneys g/100g	Ovaries/Uterus g	Ovaries/Uterus g/100g	Spleen g	Spleen g/100g	Thymus g	Thymus g/100g
0	MEAN	419	1.82	0.46	1.28	0.32	13.07	3.12	3.00	0.75	0.99	0.25	0.86	0.21	0.09	0.02
	±S.D.	±93	±0.07	±0.11	±0.20	±0.07	±3.92	±0.64	±0.47	±0.20	±0.48	±0.14	±0.36	±0.00	±0.04	±0.01
30	MEAN	411	1.83	0.45	1.26	0.32	12.55	3.10	3.00	0.75	1.11	0.29	0.85	0.21	0.09	0.02
	±S.D.	±80	±0.06	±0.09	±0.10	±0.07	±2.47	±0.57	±0.35	±0.19	±0.51	±0.16	±0.18	±0.06	±0.04	±0.01
10	MEAN	389	1.84	0.48	1.27	0.33	11.07	2.86	2.96	0.78	1.09	0.29	0.85	0.22	0.11	0.03
	±S.D.	±58	±0.04	±0.07	±0.10	±0.04	±1.73	±0.38	±0.39	±0.15	±0.31	±0.11	±0.13	±0.03	±0.03	±0.01
3	MEAN	414	1.86	0.47	1.30	0.32	13.82	3.31	3.12	0.79	0.25	0.24	0.88	0.22	0.09	0.02
	±S.D.	±92	±0.05	±0.09	±0.15	±0.06	±4.48	±0.62	±0.58	±0.22	±0.42	±0.11	±0.43	±0.12	±0.04	±0.01

No statistically significant difference from control mean using analysis of variance and Dunnett's test, $p<0.05$.

21324

TABLE 30

GROSS OBSERVATIONS OF MALE AND FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118-119 DAYS

	Dose Level (mg/kg/day)							
	Male				Female			
Observations	0	30	10	3	0	30	10	3
Congestion of thymus (secondary to decapitation)	0/10	1/10	0/10	0/10	0/10	0/10	0/10	0/10
Dilatation of renal pelvis with mineralized deposits	0/10	1/10	0/10	0/10	0/10	0/10	0/10	0/10
Hemorrhage within mediastinum (secondary to decapitation)	1/10	0/10	0/10	0/10	0/10	0/10	0/10	0/10
Circumscribed red foci in lung, ∿1 mm diameter)	1/10	0/10	0/10	0/10	0/10	0/10	0/10	0/10
Mineralized deposits in renal pelvis	0/10	1/10	1/10	0/10	0/10	2/10	0/10	1/10
Organized plug within lumen of urinary bladder	4/10	2/10	0/10	4/10	0/10	0/10	0/10	0/10
Organized plug within lumen of urethral orifice	0/10	1/10	1/10	0/10	0/10	0/10	0/10	0/10
Congenital anomaly of pectoral limb	0/10	0/10	1/10	0/10	0/10	0/10	0/10	0/10
Distended uterus due to estrus cycle	-	-	-	-	1/10	2/10	1/10	1/10
Focal corneal cloudiness								
- unilateral	0/10	0/10	0/10	0/10	1/10	0/10	0/10	1/10
- bilateral	0/10	0/10	0/10	0/10	0/10	1/10	1/10	0/10
Diffuse corneal cloudiness - unilateral	0/10	0/10	0/10	0/10	0/10	0/10	1/10	0/10

Data listed as number of rats with observation/number of rats in group.

21325

TABLE 31

MICROSCOPIC OBSERVATIONS ON TISSUES OF RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118-119 DAYS

	Dose (mg/kg/day)			
	Male		Female	
Observations	0	30	0	30
Increased hematogenous pigment in thoracic lymph node	0/6	0/8	0/10	1/8
Focal tracheitis	2/10	2/10	0/10	1/10
Peribronchiolar lymphoid aggregates in lung				
- slight	7/10	9/10	8/10	9/10
- slight to moderate	3/10	1/10	2/10	1/10
Perivascular lymphoid aggregates in lung				
- slight	2/10	1/10	0/10	1/10
- moderate	1/10	0/10	0/10	0/10
Focal accumulation of inflammatory exudate within bronchiole	1/10	1/10	0/10	0/10
Focal interstitial pneumonia	1/10	1/10	1/10	1/10
Alveolar histiocytosis	0/10	0/10	1/10	0/10
Focal alveolar epithelialization	0/10	0/10	1/10	0/10
Focal congestion of thymus	0/10	0/10	2/10	1/10
Renal tubular cast formation				
- slight or ungraded	6/10	8/10	6/10	5/10
- moderate	1/10	0/10	0/10	1/10
Aggregates of brown pigment in kidney	3/10	0/10	2/10	0/10
Focal renal tubular atrophy	4/10	3/10	1/10	1/10
Renal glomerular adhesions	1/10	1/10	0/10	0/10
Dilatation of renal tubules	2/10	2/10	0/10	0/10
Proteinaceous plug in lumen of bladder	4/10	1/10	0/7	0/10
Lymphoid aggregates in interstitium of kidney	1/10	0/10	0/10	0/10

Data listed as number of rats with observation/number of tissues examined.

21326

TABLE 31 (Continued)

MICROSCOPIC OBSERVATIONS ON TISSUES OF RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118-119 DAYS

	Dose (mg/kg/day)			
	Male		Female	
Observations	0	30	0	30
Mineralized deposits within kidney	1/10	2/10	3/10	2/10
Dilated renal pelvis	0/10	1/10	1/10	0/10
Kidney - fat content (oil red O)				
- absent	6/10	8/10	6/10	8/10
- absent to very slight	3/10	2/10	2/10	1/10
- absent to slight	1/10	0/10	2/10	1/10
Focal pancreatic acinar atrophy and fibrosis	2/9	0/10	1/8	1/10
Increase in size of single islet in pancreas	1/10	0/10	0/10	0/10
Sialoadenitis, sometimes with involvement of submandibular lymph node	2/8	2/10	4/10	2/9
Focal myocarditis	1/10	1/10	0/10	0/10
Intestinal nematodiasis	1/10	3/10	3/10	4/10
Focal edema of gastric wall	0/10	1/10	0/10	0/10
Focal aggregation of mononuclear cells in skeletal muscle	0/10	1/10	0/10	0/10
Slight increase in hematogenous pigment in spleen	0/10	0/10	5/10	4/10
Aggregations of inflammatory cells in lumen of uterus	-	-	1/10	0/10
Keratinized epithelial whorl(s) in thyroid	0/10	0/9	1/9	1/10
Focal periportal aggregation of reticuloendothelial cells in liver	0/10	0/10	1/10	0/10

Data listed as number of rats with observation/number of tissues examined.

TABLE 31 (Continued)

MICROSCOPIC OBSERVATIONS ON TISSUES OF RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118-119 DAYS

	Dose (mg/kg/day)			
	Male		Female	
Observations	0	30	0	30
Focal aggregation of reticulo-endothelial cells sometimes adjacent to degenerate-appearing hepatocytes	8/10	8/10	10/10	8/10
Cytoplasmic vacuolation of isolated hepatocytes	2/10	1/10	1/10	0/10
Liver - fat content (oil red O)				
- absent	2/9	6/9	8/10	6/9
- absent to very slight	4/9	1/9	1/10	3/9
- absent to slight	3/9	2/9	1/10	0/9

Data listed as number of rats with observation/number of tissues examined.

21328

TABLE 32

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	ADRENAL GLANDS							
A-1	Enlarged adrenal(s)	0 mg/kg/day	0	1	1	18	4	24
		30 mg/kg/day	0	0	0	8	6	14
		10 mg/kg/day	0	0	4	6	1	11
		3 mg/kg/day	0	0	0	5	4	9
A-2	Mottled adrenal(s)	0 mg/kg/day	0	0	1	6	0	7
		30 mg/kg/day	0	0	1	3	0	4
		10 mg/kg/day	0	0	2	5	0	7
		3 mg/kg/day	0	0	0	2	1	3
A-3	Pinpoint dark foci on adrenal(s)	0 mg/kg/day	0	1	3	7	2	13
		30 mg/kg/day	0	0	0	3	6	9
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	3	2	3	8
A-4	Pinpoint pale foci on adrenal(s)	0 mg/kg/day	0	1	3	7	0	11
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	1	0	1[a]
		3 mg/kg/day	0	0	2	1	0	3
	Darkened and/or congested appearance of adrenals	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	1	0	1	2
		3 mg/kg/day	0	0	0	1	1	2
A-6	Decreased size of adrenal(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-7	Hemorrhagic appearance of adrenal	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	2	4
A-9	Cyst(s) within adrenal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-10	Gray appearance of adrenals	0 mg/kg/day	0	0	0	1	3	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-11	Hemorrhagic material associated with adrenal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	CARDIOVASCULAR SYSTEM							
B-1	Enlarged heart	0 mg/kg/day	0	0	0	7	0	7
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	2	5	0	7

[a] Listed as number of rats for which the listed observation was present.

Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

21329

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	CARDIOVASCULAR SYSTEM (Cont'd)							
B-2	Dilatation and inelasticity of aorta and large vessels	0 mg/kg/day	0	0	4	13	2	19
		30 mg/kg/day	0	0	0	5	1	6
		10 mg/kg/day	0	0	3	3	0	6
		3 mg/kg/day	0	0	3	4	0	7
B-3	Mesenteric periarteritis with or without thrombosis (dilated, tortuous, sclerotic etc...)	0 mg/kg/day	0	0	1	18	1	20
		30 mg/kg/day	0	0	0	6	2	8
		10 mg/kg/day	0	0	1	6	1	8
		3 mg/kg/day	0	0	3	9	0	12
B-4	Thrombosis of left atrium	0 mg/kg/day	0	1	1	9	1	12
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	2	2	4
		3 mg/kg/day	0	0	0	6	0	6
B-5	Dilated and/or flaccid ventricular myocardium	0 mg/kg/day	0	1	4	22	1	28
		30 mg/kg/day	0	0	1	6	2	9
		10 mg/kg/day	0	0	4	3	0	7
		3 mg/kg/day	0	0	3	12	0	15

B-6	Mineralization of myocardium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	0	0	1
B-8	Thickening of mesenteric vessel	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
B-9	Aneurysm of mesenteric blood vessel	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
B-12	Pale foci or areas in myocardium	0 mg/kg/day	0	0	0	6	1	7
		30 mg/kg/day	0	0	0	1	3	4
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	3	1	5
B-13	Thickening and reddening of atrium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
B-15	Edema of mesenteric blood vessels	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	CENTRAL NERVOUS SYSTEM							
C-1	Posterior paralysis with or without atrophy of posterior musculature	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	CENTRAL NERVOUS SYSTEM (Cont'd)							
C-2	Compression on brain with or without malacia from pituitary mass	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
C-4	Focal hemorrhage in region of pineal gland	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-5	Cranial mass involving brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	0	2
C-6	Area of malacia in brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
C-8	Cranial mass involving cranial nerve with compression on brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	EYES							
D-1	Cloudiness of cornea(s) with or without dryness	0 mg/kg/day	0	1	2	26	4	33
		30 mg/kg/day	0	0	0	12	13	25
		10 mg/kg/day	0	0	7	8	4	19
		3 mg/kg/day	0	0	3	9	6	18
D-2	Cloudiness of lens (lenses)	0 mg/kg/day	0	0	0	4	1	5
		30 mg/kg/day	0	0	0	4	2	6
		10 mg/kg/day	0	0	2	0	1	3
		3 mg/kg/day	0	0	1	1	3	5
D-3	Hemorrhage and/or folding of retina in one eye	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
D-4	Erosion of cornea	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
D-5	Mass involving eye with or without exophthalmus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
D-6	Ulceration and protrusion of eye from orbit	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	EYES (Cont'd)							
D-7	Protrusion of eye with or without purulent necrotic debris extending from the oral cavity	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
D-8	Shrunken appearance of one eye	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	GENERAL							
E-1	Decreased amount of adipose tissue in abdominal cavity	0 mg/kg/day	0	1	4	31	0	36
		30 mg/kg/day	0	0	3	10	2	15
		10 mg/kg/day	0	0	5	7	1	13[a]
		3 mg/kg/day	0	0	6	11	0	17
E-2	Postmortem autolysis	0 mg/kg/day	0	0	6	5	0	11
		30 mg/kg/day	0	0	0	4	0	4
		10 mg/kg/day	0	0	5	5	0	10
		3 mg/kg/day	0	1	1	2	0	4
E-3	Loss of body condition (unkempt appearance, roughness of haircoat and/or decrease in size)	0 mg/kg/day	0	1	3	27	0	31
		30 mg/kg/day	0	0	1	11	0	12
		10 mg/kg/day	0	0	11	7	0	18
		3 mg/kg/day	0	0	6	9	0	15
E-5	Exudate accumulation lateral to external nares and eyes (bloody material)	0 mg/kg/day	0	1	2	8	0	11
		30 mg/kg/day	0	0	0	4	0	4
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	2	0	3
E-6	Excess adipose tissue	0 mg/kg/day	0	0	2	4	0	6
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	3	1	0	4
		3 mg/kg/day	0	0	0	2	0	2
E-7	Perineal soiling or matting	0 mg/kg/day	0	0	2	5	0	7
		30 mg/kg/day	0	0	1	4	0	5
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	5	0	5
E-8	Diffuse paleness of tissues (anemia) - other causes than subcutaneous mass formation	0 mg/kg/day	1	0	2	3	0	6
		30 mg/kg/day	0	0	1	4	0	5
		10 mg/kg/day	0	0	3	2	0	5
		3 mg/kg/day	0	0	1	1	0	2
E-9	Hemorrhage in abdominal cavity	0 mg/kg/day	1	0	1	3	0	5
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-10	Emaciated appearance (inanition)	0 mg/kg/day	0	0	0	6	0	6
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	3	0	3

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GENERAL (Cont'd)							
E-11	Diffuse paleness of tissues (anemia) - secondary to subcutaneous mass(es) formation	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-12	Cannibalism	0 mg/kg/day	0	0	2	2	0	4
		30 mg/kg/day	0	0	1	3	0	4
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-14	Reddened appearance of feet, pinna, scrotum, and abdominal muscle	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-15	Strangulated omental fat (focal fat necrosis)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
E-16	Excess fluid in abdomen	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	2	0	2
E-17	Edematous and/or atrophic appearance of omental fat or mesentery	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	2	0	2
E-18	Malocclusion of dental arches	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-19	Hemorrhage of mouth, tongue, hard palate, and nasal passages (unknown cause)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-22	Nodule of adipose type tissue in abdomen	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-25	Focal thinning of the haircoat	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-26	Accumulation of exudate around genital orifice	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GENERAL (Cont'd)							
E-27	Abdominal distention	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-30	Uremic odor	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-31	Blood along ventral aspect of body	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
E-32	Darkened external nares	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	GASTROINTESTINAL SYSTEM							
F-2	Ulceration or erosion of gastric mucosa	0 mg/kg/day	0	0	0	9	0	9
		30 mg/kg/day	0	0	1	2	1	4
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	2	3	0	5
F-3	Mineralization of gastric wall (pale linear striations)	0 mg/kg/day	0	0	4	20	1	25
		30 mg/kg/day	0	0	1	4	1	6[a]
		10 mg/kg/day	0	0	6	2	0	8
		3 mg/kg/day	0	0	7	6	0	13
F-4	Intestinal mass(es) or nodule(s)	0 mg/kg/day	0	0	0	4	1	5
		30 mg/kg/day	0	0	1	1	1	3
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
F-5	Hemorrhage into gastrointestinal tract	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	2	0	2
F-6	Thickening, edema, and/or hemorrhage in wall of cecum sometimes due to uremia	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	2	5	0	7
F-7	Dark material in gastrointestinal tract (or darkened ingesta or feces)	0 mg/kg/day	0	0	2	4	0	6
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	4	2	0	6
		3 mg/kg/day	0	0	2	1	0	3
F-9	Petechial hemorrhage on gastric mucosa with or without associated hemolyzed blood	0 mg/kg/day	0	0	4	4	0	8
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	2	2	0	4

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
F-10	Decrease of normal ingesta in gastrointestinal tract	0 mg/kg/day	0	0	3	9	0	12
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	3	2	0	5
		3 mg/kg/day	0	0	1	4	0	5
F-11	Thickening and/or edematous appearance of mucosa of stomach	0 mg/kg/day	0	0	2	6	1	9
		30 mg/kg/day	0	0	1	4	1	6
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	4	6	1	11
F-12	Firm white focus in stomach	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-13	Nonglandular mucosa of stomach showing roughening and/or possible focal proliferation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-14	Dilatation of stomach or gastrointestinal tract with gas (cause other than mouth breathing)	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-15	Focal gastric hemorrhage or hemolyzed blood	0 mg/kg/day	0	0	0	18	1	19
		30 mg/kg/day	0	0	1	8	3	12
		10 mg/kg/day	0	0	7	7	0	14
		3 mg/kg/day	0	0	1	6	2	9
F-16	Hemorrhagic enteritis	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-17	Nodule or polyp on stomach mucosa	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
F-18	Distention of stomach and gastrointestinal tract due to mouth breathing	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-19	Edematous appearance of mesentery	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-20	Diverticulum of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
F-21	Roughened appearance of nonglandular mucosa	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-22	Hyperemic appearance of gastric mucosa	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-23	Ulceration of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-24	Intussusception of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-25	Necrotic enteritis of small intestine extending into a peritonitis involving liver and omentum	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-26	Focal thickening of cecum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-27	Infarction of small intestine due to vasculitis and thrombosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-28	Hyperemia of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-29	Nodule in mesentery	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-30	Enlarged Peyer's Patch along small intestine	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-32	Yellow bile-staining material in gastrointestinal tract	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
F-33	Mucoid, loose, and/or watery-like material in gastrointestinal tract	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	0	0
F-34	Change in texture of stool - softened, moistened	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	2	1	0	3[a]
		3 mg/kg/day	0	0	0	0	0	0
F-35	Hemolyzed blood in gastrointestinal tract due to self-ingestion	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	1	0	1
F-37	Reddish mucoid fluid in intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-41	White film over stomach surface	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-42	White granular appearance of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-43	Increased ingesta in gastrointestinal tract	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-44	Gaseous distention of portions of intestinal tract	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-45	Overdistention of intestine with ingesta secondary to intestinal mass	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-46	Erosions of cecum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-47	Edema of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
F-48	Dilatation of stomach with ingesta	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-49	Friable gastrointestinal tract	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-50	Catarrhal exudate in cecum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	LIVER							
G-1	Enlarged liver	0 mg/kg/day	1	0	3	7	0	11
		30 mg/kg/day	0	0	1	0	0	1[a]
		10 mg/kg/day	0	0	5	2	0	7
		3 mg/kg/day	0	0	1	2	0	3
G-2	Darkened or congested liver	0 mg/kg/day	0	0	4	8	0	12
		30 mg/kg/day	0	0	2	2	1	5
		10 mg/kg/day	0	0	7	3	0	10
		3 mg/kg/day	0	0	1	3	0	4
G-3	Paleness of liver - (anemia) - probably related to subcutaneous mass(es) formation	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-13	Paleness of liver - (anemia) - other causes	0 mg/kg/day	0	0	4	12	1	17
		30 mg/kg/day	0	0	1	4	1	6
		10 mg/kg/day	0	0	3	0	0	3[a]
		3 mg/kg/day	0	0	4	3	0	7
G-4	Atrophied liver	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-5	Small pale foci in liver	0 mg/kg/day	0	0	2	10	5	17
		30 mg/kg/day	0	0	3	1	1	5
		10 mg/kg/day	0	0	0	2	3	5
		3 mg/kg/day	0	0	0	5	4	9
G-6	Red or darkened foci in liver	0 mg/kg/day	0	0	0	11	5	16
		30 mg/kg/day	0	0	0	4	5	9
		10 mg/kg/day	0	0	0	5	1	6
		3 mg/kg/day	0	0	0	4	5	9
G-7	Nodule(s) in liver	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LIVER (Cont'd)							
G-9	Accentuated lobular pattern of liver	0 mg/kg/day	0	0	2	4	0	6
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	1	1	0	2
G-10	Biliary cysts in liver	0 mg/kg/day	0	0	0	7	0	7
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	1	0	1
G-11	Nodular mass(es) in liver (probably metastatic)	0 mg/kg/day	1	0	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-12	Decreased size of liver (atrophy)	0 mg/kg/day	0	0	1	5	0	6
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	3	0	3
G-14	Linear, fibrotic reaction or capsular thickening on liver due to distortion with or without necrosis	0 mg/kg/day	0	0	1	1	2	4
		30 mg/kg/day	0	0	0	1	5	6
		10 mg/kg/day	0	0	1	0	1	2
		3 mg/kg/day	0	0	0	1	1	2
G-15	Pale area(s) in liver	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-16	Strangulation of small portion of liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-26	Fibrotic adhesions between liver and kidney	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-17	Cyst in liver containing blood	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-18	Linear pale areas (3/4 cm) in liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
G-19	Icterus	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LIVER (Cont'd)							
G-21	Focal darkened area(s) in liver	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-22	Friable texture of liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-23	Mottled appearance of liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	LYMPHORETICULAR SYSTEM							
H-1	Enlarged cervical lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-2	Enlarged thoracic lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	0	0	0	0
H-3	Enlarged mesenteric lymph nodes	0 mg/kg/day	1	0	0	5	2	8
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	1	0	1
H-4	Moist, edematous and/or hyperemic appearance of mesenteric lymph nodes	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	0	2	2	4
		3 mg/kg/day	0	0	0	3	0	3
H-5	Edema and/or hyperemia of thoracic lymph nodes and/or congestion	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	4	0	4
H-6	Enlarged thymus	0 mg/kg/day	1	0	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-7	Mass involving thymus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-8	Enlarged and/or moist appearance of hemorrhagic axillary lymph node(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LYMPHORETICULAR SYSTEM (Cont'd)							
H-9	Darkened, hemorrhagic appearance of mesenteric lymph node and/or hematogenous pigment	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	0	0	5	5
		10 mg/kg/day	0	0	0	1	2	3
		3 mg/kg/day	0	0	0	2	0	2
H-11	Mass involving mesenteric lymph nodes, thymus, renal lymph node, and mesenteric tissues	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
H-12	Congestion of mesenteric lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	1	0	2
H-14	Darkened and/or congestion of thoracic lymph nodes	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	2	0	2
H-15	Edema with or without hemorrhagic pigment in sublumbar lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-16	Hematogenous pigment in mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-17	Hyperplastic appearance of lymph nodule in small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-18	Enlarged retropharyngeal lymph node with edema and purulent exudate	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-19	Darkened, hemorrhagic appearance of thoracic lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-20	Darkened appearance of submandibular lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-21	Enlarged and/or darkened periaortal lymph nodes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	6[illegible]	6	36
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	[illegible]	50
		3 mg/kg/day	0	1	12	28	9	50*
	LYMPHORETICULAR SYSTEM (Cont'd)							
H-22	Decreased size of mesenteric lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-23	Enlarged submandibular lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-24	Congestion of iliac lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-28	Hematogenous pigment in thoracic lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-29	Gelatinous appearance of mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-30	Mottled appearance of mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	MUSCULOSKELETAL SYSTEM							
I-1	Demineralization of bones	0 mg/kg/day	0	0	2	3	0	5
		30 mg/kg/day	0	0	0	5	1	6
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	2	0	3
I-3	Atrophy of hind limb musculature	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
I-4	Mass or nodule involving rib	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
I-5	Generalized atrophy of musculature	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	PANCREAS							
J-1	Nodule(s) in region of pancreas	0 mg/kg/day	0	1	6	20	5	32
		30 mg/kg/day	0	0	1	4	11	16
		10 mg/kg/day	0	0	3	7	1	11
		3 mg/kg/day	0	0	2	10	5	18

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	PANCREAS (Cont'd)							
J-4	Edematous appearance of pancreas	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
J-5	Pale appearance of pancreas	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	PITUITARY GLAND							
K-1	Enlarged pituitary	0 mg/kg/day	0	0	1	7	0	8
		30 mg/kg/day	0	0	1	2	1	4
		10 mg/kg/day	0	0	1	5	0	6
		3 mg/kg/day	0	0	0	2	1	3
K-2	Dark red focus(foci) on pituitary	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	3	3
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	1	1	2	4
K-3	Hemorrhagic or darkened pituitary	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	0	2
K-5	Mass involving pituitary with extension into brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
K-6	Cyst within pituitary	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
L-0	Decreased size of testis (unilateral) (atrophy)	0 mg/kg/day	0	0	1	8	1	10
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	0	1	1[a]
L-1	Decreased size of testes (bilateral) (atrophy)	0 mg/kg/day	0	0	2	22	0	24
		30 mg/kg/day	0	0	4	12	1	17
		10 mg/kg/day	0	0	3	7	0	10
		3 mg/kg/day	0	0	1	10	1	12
L-2	Flaccid testis (unilateral)	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	1	1
L-2A	Flaccid testes (bilateral)	0 mg/kg/day	0	0	4	14	0	18
		30 mg/kg/day	0	0	1	5	1	7
		10 mg/kg/day	0	0	1	6	1	8
		3 mg/kg/day	0	0	1	3	1	5

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	REPRODUCTIVE SYSTEM (Cont'd)							
L-3	Decreased size of accessory sex glands (atrophy)	0 mg/kg/day	0	1	3	16	1	21
		30 mg/kg/day	0	0	3	10	1	14
		10 mg/kg/day	0	0	3	5	0	8
		3 mg/kg/day	0	0	0	9	1	10
L-4	Flaccid accessory sex glands	0 mg/kg/day	0	0	3	8	0	11
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	1	1	2
L-5	Accessory sex glands impacted with secretory material	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-6	Focal pale appearance of seminiferous tubules	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	0	0
L-7	Mass involving prostate	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
L-8	Enlarged and/or edematous accessory sex gland(s) with or without exudative material	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
L-9	Decreased size of epididymides	0 mg/kg/day	0	0	0	6	1	7[a]
		30 mg/kg/day	0	0	0	0	0	0[a]
		10 mg/kg/day	0	0	0	0	0	0[a]
		3 mg/kg/day	0	0	0	0	0	0
L-10	Edematous appearance of testes	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
L-11	Yellow discoloration of epididymis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-12	Flaccid appearance of epididymis	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-13	Friable texture of testes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	REPRODUCTIVE SYSTEM (Cont'd)							
L-14	Inflammation and protrusion of penis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-15	Area of discoloration adjacent to pale area in testes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-16	Hemorrhagic area on testis (50%)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
L-17	Yellowish material in orifice of penis (probably agonal)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-18	Tan color of testis(es)	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
L-19	Dark discoloration of prepuce and penis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-20	Congenital absence of one testis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	RESPIRATORY SYSTEM							
M-12	Clear liquid in nasal passages	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	3	0	3
M-16	Mass within dorsal nasal passages	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
M-10	Keratinized abscess-like lesion located above the hard palate in the ventral portion of the nasal turbinates, blocking airway	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
M-10A	Keratinized debris in nasal turbinates	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	RESPIRATORY SYSTEM (Cont'd)							
M-1	Pulmonary congestion, consolidation, and/or edema	0 mg/kg/day	0	2	2	9	1	14
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	2	0	2[a]
		3 mg/kg/day	0	0	0	4	0	4
M-2	Pleural effusion (hydrothorax)	0 mg/kg/day	1	1	2	18	1	23
		30 mg/kg/day	0	0	1	8	1	10
		10 mg/kg/day	0	0	2	2	0	4[a]
		3 mg/kg/day	0	0	1	8	0	9
M-3	Subpleural pale foci in lungs (suggestive of alveolar histiocytosis)	0 mg/kg/day	0	0	1	6	0	7
		30 mg/kg/day	0	0	0	2	3	5
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	0	0	1	1
M-4	Red or dark foci in lungs	0 mg/kg/day	0	0	2	8	0	10
		30 mg/kg/day	0	0	0	2	3	5
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	1	4	1	6
M-5	Darkened and/or congested color in lung tissue	0 mg/kg/day	0	0	3	7	0	10
		30 mg/kg/day	0	0	1	3	0	4
		10 mg/kg/day	0	0	5	0	0	5
		3 mg/kg/day	0	0	1	5	0	6
M-6	Pulmonary atelectasis	0 mg/kg/day	0	0	1	13	1	15
		30 mg/kg/day	0	0	0	1	2	3[a]
		10 mg/kg/day	0	0	1	3	1	5
		3 mg/kg/day	0	0	0	7	0	7
M-8	Resistance to collapse or subpleural aggregates suggestive of mineralization	0 mg/kg/day	0	0	1	5	0	6
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	1	0	2
M-9	Hematogenous pigment in lungs	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	0	1	0	1
M-13	Pale and/or dark areas in lungs (mottled)	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	3	0	3
M-14	Frothy fluid in bronchioles	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
M-17	Nodule(s) in lung	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	RESPIRATORY SYSTEM (Cont'd)							
M-11	Nodule(s) in lung (possible metastasis)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
M-12	Respiratory distress	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
	SPLEEN							
N-1	Enlarged spleen	0 mg/kg/day	0	0	3	4	0	7
		30 mg/kg/day	0	0	0	0	0	0[a]
		10 mg/kg/day	0	1	0	1	0	2
		3 mg/kg/day	0	0	0	1	0	1
N-2	Mass(es) involving spleen	0 mg/kg/day	1	0	0	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-5	Decreased size of spleen	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-6	Cyst on spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	THYROID AND PARATHYROID GLANDS							
O-1	Enlarged thyroid or parathyroid	0 mg/kg/day	0	0	1	4	0	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	0	0	0
O-2	Enlarged parathyroid(s)	0 mg/kg/day	0	0	4	19	1	24
		30 mg/kg/day	0	0	2	9	0	11
		10 mg/kg/day	0	0	3	6	1	10
		3 mg/kg/day	0	0	3	6	0	9
	URINARY SYSTEM							
P-1	Moderate to severe chronic renal disease (moderate to severely mottled, enlarged, pale, and/or roughened kidneys)	0 mg/kg/day	0	1	7	47	4	59
		30 mg/kg/day	0	0	4	17	5	26[a]
		10 mg/kg/day	0	0	17	13	2	32
		3 mg/kg/day	0	0	10	15	2	27
P-2	Slight chronic renal disease (slight enlarged, pale, mottled, and/or roughened kidneys)	0 mg/kg/day	0	1	5	6	1	13
		30 mg/kg/day	0	0	3	4	7	14
		10 mg/kg/day	0	0	2	4	2	8
		3 mg/kg/day	0	0	0	12	5	17[a]

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	URINARY SYSTEM (Cont'd)							
P-3	Mineralization of renal cortex or pelvis	0 mg/kg/day	0	0	1	5	1	7
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	2	2	0	4
		3 mg/kg/day	0	0	2	2	2	6
P-4	Dilated renal pelvis	0 mg/kg/day	0	0	2	5	1	8
		30 mg/kg/day	0	0	1	3	0	4
		10 mg/kg/day	0	0	4	3	0	7
		3 mg/kg/day	0	0	1	2	0	3
P-5	Cyst(s) in kidney	0 mg/kg/day	0	0	4	27	4	35
		30 mg/kg/day	0	0	4	10	4	18
		10 mg/kg/day	0	0	9	7	2	18
		3 mg/kg/day	0	0	4	11	2	17
P-6	Subcapsular white foci in kidney	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-7& P-20	Mass or nodule in kidney	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-10	Reddened color of urine in urinary bladder	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-12	Organized plug within lumen of urinary bladder	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	0	1	0	1
P-13& P-16	Dark and congested appearance of kidneys	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	3	1	0	4
		3 mg/kg/day	0	0	0	1	0	1
P-14	Several calculi in urinary bladder with distension of bladder with urine with or without epithelial congestion or hemorrhagic appearance	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-15	Renal calculi	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-17	Slight pale appearance of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	URINARY SYSTEM (Cont'd)							
P-18	Slight roughened appearance of kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	0	0
P-19	Urolithiasis with distension of urinary bladder	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-21	Thickening and/or hyperemia of urinary bladder	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-22	Ascending urinary tract infection	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-23	Petechial hemorrhages on urinary bladder mucosa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	3	0	3[a]
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-24	Friable texture of kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-25	Cystic mass extending off both kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
P-26	Pale focal area in kidney	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-27	Urethral plug causing distension of urinary bladder	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-28	Mass within urinary bladder	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-33	Overdistension of urinary bladder	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probabiltiy Test, $p<0.05$.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	URINARY SYSTEM (Cont'd)							
P-34	Enlarged kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-35	Edema of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-36	Pale to greenish color of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-37	Granular appearance of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-38	Darkened focus in wall of urinary bladder	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-39	Hemorrhagic material in pelvis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	INTEGUMENT AND SUBCUTANEOUS							
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 1	0 mg/kg/day	0	0	0	6	1	7
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	1	1	0	0	2
		3 mg/kg/day	0	0	1	4	1	6
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 2	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
Q-2	Subcutaneous mass - eye, ear, head region Number of masses per animal: 1	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
Q-3	Subcutaneous mass - body or limb region Number of masses per animal: 1	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
Q-3	Subcutaneous mass - body or limb region Number of masses per animal: 2	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	[illegible]
		3 mg/kg/day	0	0	0	0	0	[illegible]

Data listed as number of rats for which the listed observation was present.

TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	INTEGUMENT AND SUBCUTANEOUS (Cont'd)							
Q-3	Subcutaneous mass - body or limb region Number of masses per animal: 3	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
Q-4	Hyperplasia of mammary tissue	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-5	Mammary gland galactocele formation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-6	Subcutaneous abscess	0 mg/kg/day	0	1	0	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
Q-7	Cutaneous nodule or mass formation Number of masses or nodules per rat: 1	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	2	1	4
		3 mg/kg/day	0	0	0	1	0	1
Q-8	Abscess of Zymbal gland and causing otitis	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-9	Cystic structure adjacent to salivary glands	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-10	Epidermal inclusion cyst	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	2	1	0	3[a]
		3 mg/kg/day	0	0	0	1	0	1
Q-12	Subcutaneous edema	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-13	Abscess of preputial gland	0 mg/kg/day	0	0	0	6	1	7
		30 mg/kg/day	0	0	0	2	4	6
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	4	0	4
Q-14	Necrotic debris in ear canal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	INTEGUMENT AND SUBCUTANEOUS (Cont'd)							
Q-7	Cutaneous nodule or mass formation Number of nodules per rat: 2	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-15	Cyst formation on lateral aspect of thorax	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	SALIVARY GLANDS							
R-1	Edematous appearance of salivary glands	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
R-2	Enlarged and firm appearance of salivary glands	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	TONGUE							
S-1	Rostral lesion on tongue (apparent tearing) possibly agonal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
S-2	Mass or nodule involving tongue with or without adjacent abscessation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	ORAL CAVITY							
T-1	Softening and necrosis of gums	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
T-2	Necrotic and inflammatory process in oral cavity	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
T-3	Mass within oral cavity involving hard palate	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
T-4	Accumulation of keratinized debris on hard palate	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 32 (Continued)

GROSS OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	THORACIC CAVITY							
U-1	Cystic mass within thoracic cavity	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 33

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	ADRENAL GLANDS							
A-1	Enlarged adrenal(s)	0 mg/kg/day	0	0	4	7	13	24
		30 mg/kg/day	0	0	0	4	9	13
		10 mg/kg/day	0	0	0	6	0	6[a]
		3 mg/kg/day	0	0	0	1	2	3[a]
A-2	Mottled adrenal(s)	0 mg/kg/day	0	0	2	5	0	7
		30 mg/kg/day	0	0	2	5	1	8
		10 mg/kg/day	0	0	1	8	0	9
		3 mg/kg/day	0	0	0	3	0	3
A-3	Pinpoint dark foci of adrenal(s)	0 mg/kg/day	0	0	4	3	23	30
		30 mg/kg/day	0	0	2	2	15	19
		10 mg/kg/day	0	0	0	3	5	8[a]
		3 mg/kg/day	0	0	3	6	11	20
A-4	Pinpoint pale foci on adrenal(s)	0 mg/kg/day	0	0	2	3	18	23
		30 mg/kg/day	0	0	1	2	12	15
		10 mg/kg/day	0	0	0	2	2	4[a]
		3 mg/kg/day	0	0	1	5	8	15
A-5	Darkened and/or congested appearance of adrenals	0 mg/kg/day	0	0	1	1	5	7
		30 mg/kg/day	0	0	0	1	4	5
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	0	0	0	0
A-6	Decreased size of adrenal(s)	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-7	Hemorrhagic appearance of adrenal(s)	0 mg/kg/day	0	0	0	1	5	6
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
A-8	Brown discoloration of adrenal(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-9	Cyst(s) within adrenal	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-10	Gray appearance of adrenals	0 mg/kg/day	0	0	0	0	3	3
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
	CARDIOVASCULAR SYSTEM							
B-3	Mesenteric periarteritis with or without thrombosis (dilated, tortuous, sclerotic, etc...)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	CARDIOVASCULAR SYSTEM (Cont'd)							
B-4	Thrombosis of left atrium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	0	2
B-5	Dilated and/or flaccid ventricular myocardium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	2	0	1	3[a]
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	1	0	1
B-7	Dilatation of pericardial sac with hemorrhage and apparent infiltration of neoplastic cells (thoracic mass)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1
B-10	Abdominal mass with cystic spaces (possible vascular origin)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
B-11	Hematoma at base of tail	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
B-14	Axillary vessels enlarged (adjacent to subcutaneous mass)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	CENTRAL NERVOUS SYSTEM							
C-2	Compression on brain with or without malacia from pituitary mass	0 mg/kg/day	0	0	1	4	2	7
		30 mg/kg/day	0	0	0	4	0	4
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	5	1	6
C-3	Paravertebral mass causing lordosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-7	Focal hemorrhage in brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-8	Cranial mass involving cranial nerve with compression on brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	EYES							
D-1	Cloudiness of cornea(s) with or without dryness	0 mg/kg/day	0	0	1	10	20	31
		30 mg/kg/day	0	0	1	5	14	20
		10 mg/kg/day	0	0	0	10	7	17
		3 mg/kg/day	0	0	0	5	11	16
D-2	Cloudiness of lens (lenses)	0 mg/kg/day	0	0	1	3	15	19
		30 mg/kg/day	0	0	0	1	5	6
		10 mg/kg/day	0	0	0	0	3	3[a]
		3 mg/kg/day	0	0	1	0	10	11
	GENERAL							
E-1	Decreased amount of adipose tissue in abdominal cavity	0 mg/kg/day	0	1	2	11	2	16
		30 mg/kg/day	0	1	1	7	2	11
		10 mg/kg/day	1	0	2	13	0	16
		3 mg/kg/day	0	0	1	2	3	6
E-2	Postmortem autolysis	0 mg/kg/day	0	1	0	4	0	5
		30 mg/kg/day	0	1	0	1	0	2
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	1	1	2	0	4
E-3	Loss of body condition (unkempt appearance, roughness of haircoat and/or decrease in size)	0 mg/kg/day	0	1	3	6	0	10
		30 mg/kg/day	0	1	1	5	0	7
		10 mg/kg/day	1	0	0	9	0	10
		3 mg/kg/day	0	0	2	3	1	6
E-4	Overgrowth of incisor teeth (with or without ulceration or inflammation of hard palate or gum line) or other dental abnormalities (teeth broken off, etc.)	0 mg/kg/day	0	1	0	0	3	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	1	2
		3 mg/kg/day	0	0	0	0	0	0
E-5	Exudate accumulation lateral to external nares and eyes (bloody material)	0 mg/kg/day	0	0	0	4	1	5
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	3	0	4
		3 mg/kg/day	0	0	0	0	1	1
E-6	Excess adipose tissue	0 mg/kg/day	0	0	2	2	0	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-7	Perineal soiling or matting	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	1	0	1
E-8	Diffuse paleness of tissues (anemia) - other causes than subcutaneous mass formation	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	GENERAL (Cont'd)							
E-10	Emaciated appearance (inanition)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	1	0	0	2	0	3[a]
		3 mg/kg/day	0	0	0	0	0	0
E-11	Diffuse paleness of tissues (anemia) - secondary to subcutaneous mass(es) formation	0 mg/kg/day	0	1	0	4	1	6
		30 mg/kg/day	0	1	1	1	0	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	2	1	4
E-12	Cannibalism	0 mg/kg/day	0	1	0	1	0	2
		30 mg/kg/day	0	0	2	1	0	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-13	Abdominal organs decreased in size due to starvation	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-15	Strangulated omental fat (focal fat necrosis)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	1	1	3
E-16	Excess fluid in abdomen	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-17	Edematous and/or atrophic appearance of omental fat or mesentery	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-19	Hemorrhage of mouth, tongue, hard palate, and nasal passages (unknown cause)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-20	Abdominal mass with adhesions to multiple organs	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-21	Inflammation or necrosis of mesenteric fat sometimes with yellow coloration	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
E-23	Nodule involving mesenteric fat	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	GENERAL (Cont'd)							
E-24	Thickened gingiva surrounding molar teeth	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-26	Accumulation of exudate around genital orifice	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
E-27	Abdominal distention	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	0	0	0	0
E-28	Firm and granular appearance of mesenteric fat	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-29	Hemorrhage from tail	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	GASTROINTESTINAL SYSTEM							
F-2	Ulceration or erosion of gastric mucosa	0 mg/kg/day	0	0	1	3	2	6
		30 mg/kg/day	0	1	0	2	1	4
		10 mg/kg/day	0	0	1	4	0	5
		3 mg/kg/day	1	0	0	3	0	4
F-3	Mineralization of gastric wall (pale linear striations)	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
F-4	Intestinal mass(es) or nodule(s)	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
F-5	Hemorrhage into gastrointestinal tract	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-6	Thickening, edema, and/or hemorrhage in wall of cecum sometimes due to uremia	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-7	Dark material in gastrointestinal tract (or darkened ingesta or feces)	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	1	0	2	3

Data listed as number of rats for which the listed observation was present.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	GENERAL (Cont'd)							
F-9	Petechial hemorrhage on gastric mucosa with or without associated hemolyzed blood	0 mg/kg/day	0	1	0	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-10	Decrease of normal ingesta in gastrointestinal tract	0 mg/kg/day	0	1	0	3	0	4
		30 mg/kg/day	0	1	0	2	0	3
		10 mg/kg/day	1	0	1	7	0	9[a]
		3 mg/kg/day	0	0	1	1	0	2
F-11	Thickening and/or edematous appearance of mucosa of stomach	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
F-13	Nonglandular mucosa of stomach showing roughening and/or possible focal proliferation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	1	0	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-14	Dilatation of stomach or gastrointestinal tract with gas (cause other than mouth breathing)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
F-15	Focal gastric hemorrhage or hemolyzed blood	0 mg/kg/day	0	0	1	3	6	10
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	1	5	1	7
		3 mg/kg/day	0	0	0	5	2	7
F-17	Nodule or polyp on stomach mucosa	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-18	Distention of stomach and gastrointestinal tract due to mouth breathing	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-20	Diverticulum of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-30	Enlarged Peyer's Patch along small intestine	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-31	Thickened wall and overdistended appearance of small intestine	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	GENERAL (Cont'd)							
F-32	Yellow bile-staining material in gastrointestinal tract	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	1	0	1
F-33	Mucoid, loose, and/or watery-like material in gastrointestinal tract	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-34	Change in texture of stool - softened, moistened	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
F-35	Hemolyzed blood in gastrointestinal tract due to self-ingestion	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	1	2
F-36	Intestinal nematodes	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-37	Reddish mucoid fluid in intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
F-38	Decreased fluid in gastrointestinal tract	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-39	Overdistention of esophagus with ingesta	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-40	Implants from intestinal neoplasm throughout abdominal viscera	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
F-41	White film over stomach surface	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LIVER							
G-1	Enlarged liver	0 mg/kg/day	0	0	1	2	4	7
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kd/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	1	3
G-2	Darkened or congested liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
G-3	Paleness of liver - (anemia) - probably related to subcutaneous mass(es) formation	0 mg/kg/day	0	0	2	4	2	8
		30 mg/kg/day	0	1	3	2	0	6
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	2	1	1	4
G-13	Paleness of liver - (anemia) - other causes	0 mg/kg/day	0	0	3	0	3	6
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	2	0	1	3
		3 mg/kg/day	0	0	1	1	1	3
G-5	Small pale foci in liver	0 mg/kg/day	0	0	0	4	13	17
		30 mg/kg/day	0	0	0	0	4	4
		10 mg/kg/day	0	0	1	1	1	3[a]
		3 mg/kg/day	0	0	0	1	3	4
G-6	Red or darkened foci in liver	0 mg/kg/day	0	0	6	8	30	44
		30 mg/kg/day	0	1	1	1	17	20
		10 mg/kg/day	0	0	0	3	6	9[a]
		3 mg/kg/day	0	0	1	0	13	14
G-7	Nodule(s) in liver	0 mg/kg/day	0	0	1	1	11	13
		30 mg/kg/day	0	0	0	0	1	1[a]
		10 mg/kg/day	0	0	0	0	0	0[a]
		3 mg/kg/day	0	0	0	1	0	1[a]
G-9	Accentuated lobular pattern of liver	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
G-10	Biliary cysts in liver	0 mg/kg/day	0	0	1	1	5	7
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	0	2	3
G-12	Decreased size of liver (atrophy)	0 mg/kg/day	0	0	0	3	3	6
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	1	0	0	3	0	4
		3 mg/kg/day	0	0	0	0	1	1
G-14	Linear, fibrotic reaction or capsular thickening on liver due to distortion with or without necrosis	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LIVER (Cont'd)							
G-15	Pale area(s) in liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-25	Diaphragmatic herniation of liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-28	Yellow appearance of liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-29	White discoloration of one lobe of liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-19	Icterus	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-20	Subcapsular depressed foci in liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-21	Focal darkened area(s) in liver	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	2	3
		3 mg/kg/day	0	0	0	0	4	4
G-22	Friable texture of liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-23	Mottled appearance of liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-24	Discoloration and diffuse infiltrative enlargement of liver	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	LYMPHORETICULAR SYSTEM							
H-0	Generalized enlargement of many lymphoid tissues (lymphadenopathy) sometimes with a reddish appearance)	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LYMPHORETICULAR SYSTEM (Cont'd)							
H-2	Enlarged thoracic lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-3	Enlarged mesenteric lymph nodes	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1
H-4	Moist, edematous and/or hyperemic appearance of mesenteric lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1
H-6	Enlarged thymus	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-7	Mass involving thymus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1
H-8	Enlarged and/or moist appearance of hemorrhagic axillary lymph node(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	1	0	0	1	1	3
H-9	Darkened, hemorrhagic appearance of mesenteric lymph node and/or hematogenous pigment	0 mg/kg/day	0	0	0	1	19	20
		30 mg/kg/day	0	0	0	0	11	11
		10 mg/kg/day	0	0	0	0	8	8
		3 mg/kg/day	0	0	0	4	12	16
H-12	Congestion of mesenteric lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-13	Enlarged and congested or hemorrhagic iliac lymph nodes	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-14	Darkened and/or congestion of thoracic lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-21	Enlarged and/or darkened periaortal lymph nodes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LYMPHORETICULAR SYSTEM (Cont'd)							
H-25	Mass involving mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-26	Mass(es) involving thoracic or cervical lymph nodes (probable metastasis from thyroid)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-27	Enlarged iliac and renal lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	MUSCULOSKELETAL SYSTEM							
I-1	Demineralization of bones	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	PANCREAS							
J-1	Nodule(s) in region in pancreas	0 mg/kg/day	0	0	0	0	8	8
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	3	1	4
J-3	Interlobular edema	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	PITUITARY GLAND							
K-1	Enlarged pituitary	0 mg/kg/day	0	0	4	12	15	31
		30 mg/kg/day	0	0	2	9	6	17
		10 mg/kg/day	0	0	0	11	2	13
		3 mg/kg/day	0	0	1	13	8	22
K-2	Dark red focus(foci) on pituitary	0 mg/kg/day	0	0	5	4	8	17
		30 mg/kg/day	0	0	0	4	4	8
		10 mg/kg/day	0	0	0	3	2	5
		3 mg/kg/day	0	0	0	4	3	7
K-3	Hemorrhagic or darkened pituitary	0 mg/kg/day	0	0	1	5	7	13
		30 mg/kg/day	0	0	1	5	3	9
		10 mg/kg/day	0	0	0	5	1	6
		3 mg/kg/day	0	0	1	6	8	15[a]
K-7	Irregular shape	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	PITUITARY GLAND (Cont'd)							
K-8	Mottled	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
K-9	Pale area in pituitary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
LL-1	Mass or nodule involving ovary and/or oviduct	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
LL-2	Cyst in ovary	0 mg/kg/day	0	0	2	1	6	9
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	1	2	1	4
		3 mg/kg/day	0	0	0	0	3	3
LL-4	Endometrial polyp(s)	0 mg/kg/day	0	0	2	3	8	13
		30 mg/kg/day	0	0	1	0	6	7
		10 mg/kg/day	0	1	1	3	2	7
		3 mg/kg/day	0	0	0	2	1	3
LL-5	Endometrial hyperplasia (thickened)	0 mg/kg/day	0	0	0	2	3	5
		30 mg/kg/day	0	0	1	0	3	4
		10 mg/kg/day	0	0	0	2	3	5
		3 mg/kg/day	0	0	1	0	3	4
LL-6	Cystic endometrial hyperplasia (thickened, cystic)	0 mg/kg/day	0	0	5	0	13	18
		30 mg/kg/day	0	0	3	3	6	12
		10 mg/kg/day	0	0	0	1	2	3[a]
		3 mg/kg/day	0	0	1	2	3	6
LL-8	Distension of uterus with dark fluid (altered blood and soft debris)	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
LL-9	Slight roughened appearance of uterine endometrium	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
LL-10	Hemorrhagic cyst-like structure adjacent to ovary and nodules within mesometrium attached to uterus	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-11	Nodule or mass involving uterus	0 mg/kg/day	0	0	0	2	3	5
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	1	1	0	2

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	REPRODUCTIVE SYSTEM (Cont'd)							
LL-12	Distention of uterus	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-13	Hyperemia of uterus	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-14	Decreased size of ovaries with a brown discoloration	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
LL-15	Clear fluid in uterus	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-16	Red focus in uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-17	Mass involving vagina	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-18	Decreased size of uterus	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
LL-19	Hemorrhagic material in uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
LL-20	Thickened area in uterus	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	RESPIRATORY SYSTEM							
M-12	Clear liquid in nasal passages	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
M-15	Mucoid material in nasal turbinates	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	RESPIRATORY SYSTEM (Cont'd)							
M-1	Pulmonary congestion, consolidation, and/or edema	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	1	0	3	0	4
		3 mg/kg/day	0	0	0	0	1	1
M-3	Subpleural pale foci in lungs (suggestive of alveolar histiocytosis)	0 mg/kg/day	0	0	0	7	12	19
		30 mg/kg/day	0	0	1	2	4	7
		10 mg/kg/day	0	0	1	6	2	9
		3 mg/kg/day	0	0	1	1	4	6
M-4	Red or dark foci in lungs	0 mg/kg/day	0	0	0	4	9	13
		30 mg/kg/day	0	0	1	1	5	7
		10 mg/kg/day	0	0	1	0	1	2[a]
		3 mg/kg/day	0	0	1	3	2	6
M-5	Darkened and/or congested color in lung tissue	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	1	0	2
M-6	Pulmonary atelectasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	2	0	3
		3 mg/kg/day	1	0	0	0	0	1
M-9	Hematogenous pigment in lungs	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
M-13	Pale and/or dark areas in lungs (mottled)	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
M-17	Nodule(s) in lung	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
M-11	Nodule(s) in lung (possible metastasis)	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	2	0	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	SPLEEN							
N-1	Enlarged spleen	0 mg/kg/day	0	0	2	4	2	8
		30 mg/kg/day	0	0	4	2	0	6
		10 mg/kg/day	0	0	3	2	0	5
		3 mg/kg/day	1	0	3	6	1	11[a]

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	SPLEEN (Cont'd)							
N-3	Congested appearance of spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-4	Pale focus in spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	1	0	1
	THYROID AND PARATHYROID GLANDS							
O-1	Enlarged thyroid or parathyroid	0 mg/kg/day	0	0	0	0	3	3
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	2	2
O-2	Enlarged parathyroid(s)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	URINARY SYSTEM							
P-1	Moderate to severe chronic renal disease (moderate to severely mottled, enlarged, pale, and/or roughened kidneys)	0 mg/kg/day	0	0	3	0	3	6
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	1	4	6
P-2	Slight chronic renal disease (slight enlarged, pale, mottled, and/or roughened kidneys)	0 mg/kg/day	0	0	1	3	11	15
		30 mg/kg/day	0	0	0	0	8	8
		10 mg/kg/day	0	0	1	2	2	5
		3 mg/kg/day	0	0	0	2	4	6
P-3	Mineralization of renal pelvis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	2	6	8[a]
		10 mg/kg/day	0	0	3	2	0	5[a]
		3 mg/kg/day	0	0	0	0	1	1
P-4	Dilated renal pelvis	0 mg/kg/day	0	0	1	3	3	7
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	1	2	1	4
		3 mg/kg/day	0	0	1	0	1	2
P-5	Cyst(s) in kidney	0 mg/kg/day	0	0	1	1	4	6
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	1	1	4	6
P-6	Subcapsular white foci in kidney	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	URINARY SYSTEM (Cont'd)							
P-7 &P-20	Mass or nodule in kidney	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-8	Atrophy of kidney(s) (decreased size)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	1	0	2
		3 mg/kg/day	0	0	0	0	0	0
P-9	Small intestine adherent to capsular surface of enlarged kidney, and the cortex showing multiple pale focal areas	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
P-13 &P-16	Dark and congested appearance of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
P-15	Renal calculi	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
P-17	Slight pale appearance of kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
P-18	Slight roughened appearance of kidneys	0 mg/kg/day	0	0	3	1	0	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	1	0	1
P-19	Urolithiasis with distention of urinary bladder	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-22	Ascending urinary tract infection	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
P-24	Friable texture of kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-26	Pale focal area in kidney	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	URINARY SYSTEM (Cont'd)							
P-29	Slight enlargement and flaccid kidney secondary to dilated pelvis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-30	Pale, anemic appearance of kidneys	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-31	Dilatation of urinary bladder and compression of ureter secondary to uterine mass	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-32	Dark greenish discoloration of kidneys	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	INTEGUMENT AND SUBCUTANEOUS							
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 1	0 mg/kg/day	0	1	5	11	8	25
		30 mg/kg/day	0	1	4	6	7	18
		10 mg/kg/day	0	1	7	10	3	21
		3 mg/kg/day	0	0	6	8	8	22
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 2	0 mg/kg/day	0	0	1	8	9	18
		30 mg/kg/day	0	0	1	7	7	15
		10 mg/kg/day	0	0	1	7	5	13
		3 mg/kg/day	0	0	0	4	6	10
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 3	0 mg/kg/day	0	0	3	4	10	17
		30 mg/kg/day	0	0	3	1	4	8
		10 mg/kg/day	0	0	1	1	1	3[a]
		3 mg/kg/day	0	0	2	1	3	6
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 4	0 mg/kg/day	0	0	2	2	4	8
		30 mg/kg/day	0	0	2	0	1	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	0	2
Q-1	Subcutaneous mass - mammary and midcervical region Number of masses per animal: 5	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	0	0	0
Q-2	Subcutaneous mass - eye, ear, head region Number of masses per animal: 1	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	1	0	2

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	INTEGUMENT AND SUBCUTANEOUS (Cont'd)							
Q-3	Subcutaneous mass - body or limb region Number of masses per animal 1	0 mg/kg/day	0	0	3	5	3	11
		30 mg/kg/day	0	1	0	1	3	5
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	3	2	5
Q-3	Subcutaneous mass - body or limb region Number of masses per animal: 2	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
Q-3	Subcutaneous mass - body or limb region Number of masses per animal: 3	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-4	Hyperplasia of mammary tissue	0 mg/kg/day	0	0	3	6	12	21
		30 mg/kg/day	0	0	2	2	9	13
		10 mg/kg/day	0	0	1	3	3	7
		3 mg/kg/day	0	0	2	3	5	10
Q-5	Mammary gland galactocele formation	0 mg/kg/day	0	0	2	3	6	11
		30 mg/kg/day	0	0	1	2	5	8
		10 mg/kg/day	0	0	0	4	2	6
		3 mg/kg/day	0	0	1	2	3	6
Q-7	Cutaneous nodule or mass formation Number of nodules per rat: 1	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-11	Abscess of clitoral gland	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Q-12	Subcutaneous edema	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	SALIVARY GLANDS							
R-3	Cystic structure involving salivary gland	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	TONGUE							
S-1	Rostral lesion on tongue (apparent tearing) possibly agonal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
S-2	Mass or nodule involving tongue with or without adjacent abscessation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 33 (Continued)

GROSS OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	THORACIC CAVITY							
U-2	Fluid in thoracic cavity	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	2	0	2

Data listed as number of rats for which the listed observation was present.

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TABLE 34

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LIVER							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Hepatocellular Changes							
H-01	Necrosis with accompanying inflammatory reaction - single focus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	1	0	0	2
		3 mg/kg/day	0	0	0	0	0	0
H-02	Necrosis with accompanying inflammatory reaction - focal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
H-03	Necrosis with accompanying inflammatory reaction - multifocal	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	1	0	1	0	2
H-04	Necrosis with accompanying inflammatory reaction - disseminated	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-05	Cytoplasmic vacuolization - single focus	0 mg/kg/day	0	0	2	2	0	4
		30 mg/kg/day	0	0	1	3	3	7
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	1	1	0	2
H-06	Cytoplasmic vacuolization - focal	0 mg/kg/day	0	1	4	24	5	34
		30 mg/kg/day	0	0	0	3	5	8[a]
		10 mg/kg/day	0	0	2	8	2	12[a]
		3 mg/kg/day	0	0	0	14	6	20
H-07	Cytoplasmic vacuolization - multifocal	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-08	Cytoplasmic vacuolization - disseminated	0 mg/kg/day	0	1	0	5	0	6
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	0	0	1
H-10	Accentuated lobular pattern due to centrilobular atrophy	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
H-11	Hepatocellular alteration (swollen hepatocytes) - single focus	0 mg/kg/day	0	1	1	8	1	11
		30 mg/kg/day	0	0	0	2	2	4
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	1	3	1	5

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months
is	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14
		30 mg/kg/day	0	0	9
		10 mg/kg/day	0	1	22
		3 mg/kg/day	0	1	12
	Hepatocellular Changes (Cont'd)				
12	Hepatocellular alteration (swollen hepatocytes) - focal (2-3)	0 mg/kg/day	0	1	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	1
17	Hepatocellular alteration (swollen hepatocytes) - several foci (>2)	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	2
		3 mg/kg/day	0	0	0
13	Hepatocellular alteration (swollen hepatocytes) - single area	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	0
14	Hepatocellular alteration (swollen hepatocytes) - several areas	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
[illegible]	Hepatocellular carcinoma(s)	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
[illegible]	Focal vacuole formation with or without mineralized debris	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
[illegible]	Focal confluent vacuole formation with or without mineralized debris	0 mg/kg/day	0	0	1
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
[illegible]	Inflammatory Cell Changes				
[illegible]	Focal accumulation of inflammatory cells, primarily mononuclear cells within liver	0 mg/kg/day	0	0	2
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	1
[illegible]	Focal aggregates of reticulo-endothelial cells frequently adjacent to degenerate or necrotic hepatocytes	0 mg/kg/day	0	1	3
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	1
[illegible]	Fibrosis within hepatic tissue - focal	0 mg/kg/day	0	0	1
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
[illegible]	Fibrosis - subcapsular - focal	0 mg/kg/day	0	0	1
		30 mg/kg/day	0	0	1
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	0

[illegible] listed as [illegible] d as number of rats for which the listed observation was present.

[illegible] ically [illegible] ally different from control data when analyzed using Fisher's Exact Probability

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Inflammatory Cell Changes (Cont'd)							
H-48	Fibrosis - subcapsular - multifocal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-49	Increased pigment aggregates in liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-50	Inflammatory adhesions to capsule	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Bile Ducts and Vascular Channels							
H-70	Biliary hyperplasia - focal	0 mg/kg/day	0	0	1	6	2	9
		30 mg/kg/day	0	0	1	7	6	14[a]
		10 mg/kg/day	0	0	0	7	1	8
		3 mg/kg/day	0	0	0	4	2	6
H-71	Biliary hyperplasia - multifocal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-72	Biliary cyst formation	0 mg/kg/day	0	0	0	7	0	7
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	1	2	1	4
		3 mg/kg/day	0	0	0	3	1	4
H-74	Periportal inflammation	0 mg/kg/day	0	1	2	23	3	29
		30 mg/kg/day	0	0	5	11	12	28[a]
		10 mg/kg/day	0	0	4	4	1	9
		3 mg/kg/day	0	0	0	3	4	7[a]
H-75	Focal periportal fibrosis	0 mg/kg/day	0	1	1	14	4	20
		30 mg/kg/day	0	0	2	9	7	18
		10 mg/kg/day	0	0	1	1	1	3[a]
		3 mg/kg/day	0	0	0	8	3	11
H-85	Ectasia of vascular channels	0 mg/kg/day	0	0	0	7	1	8
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	1	4	1	6
H-86	Congestion of hepatic sinusoids	0 mg/kg/day	1	1	1	4	1	8
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	1	1	2
H-89	Perivasculitis - focal	0 mg/kg/day	0	0	3	2	0	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Bile Ducts and Vascular Channels (Cont.)							
H-91	Focal extramedullary hematopoiesis	0 mg/kg/day	0	0	2	6	0	8
		30 mg/kg/day	0	0	2	4	0	6
		10 mg/kg/day	0	1	5	1	0	7
		3 mg/kg/day	0	0	3	2	0	5
H-92	Retention cyst formation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	RESPIRATORY SYSTEM							
	Nasal Turbinates/Hard Palate							
	Number of tissues examined	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	2	0	2
R-04	Keratoacanthoma of nasal turbinates /hard palate	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-05	Squamous cell carcinoma of hard palate	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
R-06	Acute hemorrhage (trauma) and area of inflammation of nasal turbinates	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-07	Mixed adenoma of hard palate	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-08	Squamous metaplasia of nasal turbinate epithelium and glands of hard palate	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Trachea							
	Number of tissues examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	9	24	16	49
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
R-20	Focal tracheitis	0 mg/kg/day	0	0	2	16	3	21
		30 mg/kg/day	0	0	2	4	7	13
		10 mg/kg/day	0	0	2	4	4	10
		3 mg/kg/day	0	0	2	6	2	10

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Trachea (Cont'd)							
R-21	Diffuse tracheitis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	2	0	2
R-23	Focal epithelial hyperplasia of tracheitis	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
R-24	Diffuse epithelial hyperplasia of trachea	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
R-25	Secretory material in trachea	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Lungs							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
R-30	Focal peribronchiolar aggregates of lymphocytes	0 mg/kg/day	0	1	9	57	5	72
		30 mg/kg/day	0	0	7	21	14	42
		10 mg/kg/day	0	1	17	13	4	35
		3 mg/kg/day	0	0	7	17	8	32
R-31	Focal interstitial inflammation	0 mg/kg/day	0	0	0	18	3	21
		30 mg/kg/day	0	0	3	16	8	27[a]
		10 mg/kg/day	0	0	3	2	0	5[a]
		3 mg/kg/day	0	0	1	3	1	5[a]
R-32	Focal accumulation of inflammatory cells in alveoli	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-33	Focal accumulation of alveolar macrophages in alveoli	0 mg/kg/day	0	1	2	24	1	28
		30 mg/kg/day	0	0	5	14	10	29[a]
		10 mg/kg/day	0	0	4	8	1	13
		3 mg/kg/day	0	0	5	7	4	16
R-34	Alveolar proteinosis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
R-35	Cholesterol clefts in lung	0 mg/kg/day	0	1	0	5	0	6
		30 mg/kg/day	0	0	1	5	3	9[a]
		10 mg/kg/day	0	0	0	5	0	5
		3 mg/kg/day	0	0	1	1	2	4

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED [illegible]
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-[illegible] Months
		0 mg/kg/day	1	2	[illegible]
		30 mg/kg/day	0	0	[illegible]
		10 mg/kg/day	0	1	[illegible]
		3 mg/kg/day	0	1	[illegible]
	Lungs (Cont'd)				
R-36	Pulmonary edema, congestion, cellular exudation, with or without focal fibrosis secondary to heart failure, renal disease and/or vascular disease	0 mg/kg/day	0	0	1
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	1
R-64	Cellular exfoliation secondary to renal failure or heart failure	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	2
		3 mg/kg/day	0	0	0
R-37	Focal alveolar fibrosis secondary to heart failure or renal failure	0 mg/kg/day	0	1	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	2
		3 mg/kg/day	0	0	0
R-38	Pulmonary atelectasis	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
R-39	Alveolar mineralization	0 mg/kg/day	0	0	2
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	1
		3 mg/kg/day	0	0	5
R-40	Focal pulmonary aggregates of hematogenous pigment	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
R-41	Focal alveolar hyperplasia	0 mg/kg/day	0	1	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	2
R-42	Pulmonary edema	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	0
		10 mg/kg/day	0	0	0
		3 mg/kg/day	0	0	0
R-43	Focal interstitial fibrosis	0 mg/kg/day	0	0	0
		30 mg/kg/day	0	0	[illegible]
		10 mg/kg/day	0	0	[illegible]
		3 mg/kg/day	0	0	3
R-44	Focal granulomatous inflammation with or without foreign body present	0 mg/kg/day	0	0	[illegible]
		30 mg/kg/day	0	0	[illegible]
		10 mg/kg/day	0	0	[illegible]
		3 mg/kg/day	0	0	[illegible]
R-45	Focal pulmonary hemorrhage	0 mg/kg/day	0	0	[illegible]
		30 mg/kg/day	0	0	[illegible]
		10 mg/kg/day	0	0	[illegible]
		3 mg/kg/day	0	0	[illegible]

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact [illegible]

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Lungs (Cont'd)							
R-48	Focal hyperplasia of bronchiolar epithelium	0 mg/kg/day	0	0	0	8	1	9
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	0	0	0	0[a]
		3 mg/kg/day	0	0	0	2	0	2
R-49	Aspirated particulate matter	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
R-51	Multifocal exudative pneumonia	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-52	Pulmonary congestion	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-53	Increased perivascular aggregates of lymphocytes	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	1	0	1
R-54	Secretory material in bronchus or bronchiole	0 mg/kg/day	0	0	0	8	0	8
		30 mg/kg/day	0	0	1	3	0	4
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0[a]
R-55	Focal inflammation of bronchus	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-56	Focal area of interstitial inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-59	Inflammatory cells within lumen and/or around bronchiole(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	3	0	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-60	Focal secretory material in alveoli	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	0	0	1
R-61	Multifocal alveolar hyperplasia, squamous metaplasia and focal necrosis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Lungs (Cont'd)							
R-62	Multifocal alveolar hyperplasia	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-63	Area of adenomatous alveolar hyperplasia and interstitial inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
R-70	Pulmonary adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
R-65	Area of necrosis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Pleura							
R-90	Focal pleuritis	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
R-92	Focal fibrotic pleuritis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-94	Focal fibrosis of pleura	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	CARDIOVASCULAR SYSTEM							
	Heart							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
C-01	Focal myocardial degeneration and inflammation with or without fibrosis - slight	0 mg/kg/day	0	1	7	37	4	49
		30 mg/kg/day	0	0	5	15	12	32
		10 mg/kg/day	0	1	15	15	2	33
		3 mg/kg/day	0	1	9	18	5	33
C-02	Focal myocardial degeneration and inflammation with or without fibrosis - moderate	0 mg/kg/day	0	1	6	15	1	23
		30 mg/kg/day	0	0	0	5	4	9
		10 mg/kg/day	0	0	4	4	1	9
		3 mg/kg/day	0	0	1	6	4	11

Data listed as number of rats for which the listed observation was present.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Heart (Cont'd)							
C-03	Focal myocardial degeneration and inflammation with or without fibrosis - pronounced	0 mg/kg/day	0	0	0	6	1	7
		30 mg/kg/day	0	0	1	2	0	3
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	1	0	0	1
C-04	Myocardial mineralization	0 mg/kg/day	0	0	0	4	1	5
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	2	1	0	3
C-05	Atrial thrombosis	0 mg/kg/day	0	1	0	10	1	12
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	0	1	2	3
		3 mg/kg/day	0	0	0	6	0	6
C-06	Subendocardial fibroelastosis of myocardium	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	1	0	1
C-07	Area of myocardial necrosis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-08	Congestion of heart	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
C-09	Focal hemorrhage	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-10	Focal myocarditis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-11	Focal pericarditis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Aorta and/or Large Anterior Mediastinal Artery(ies)							
	Number of tissues examined	0 mg/kg/day	1	2	14	60	6	83
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	21	4	48
		3 mg/kg/day	0	1	11	27	9	48
C-20	Focal mural mineralization	0 mg/kg/day	0	1	6	34	2	43
		30 mg/kg/day	0	0	3	10	11	24
		10 mg/kg/day	0	0	6	9	2	17
		3 mg/kg/day	0	0	7	14	3	24

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Aorta and/or Large Anterior Mediastinal Artery(ies) (Cont'd)							
C-21	Focal thickening of endothelial lining	0 mg/kg/day	0	1	2	25	5	33
		30 mg/kg/day	0	0	3	9	14	26
		10 mg/kg/day	0	0	1	8	2	11
		3 mg/kg/day	0	0	0	6	5	11
C-22	Thrombosis of thoracic arteries	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
C-23	Inflammatory reaction around thoracic artery	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
C-24	Aortic plaque formation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Blood Vessels							
C-30	Degeneration of myocardial blood vessels	0 mg/kg/day	0	2	11	48	4	65
		30 mg/kg/day	0	0	6	19	12	37
		10 mg/kg/day	0	0	16	14	3	33
		3 mg/kg/day	0	1	11	15	9	36
C-31	Mineralization of myocardial blood vessels	0 mg/kg/day	0	0	1	11	0	12
		30 mg/kg/day	0	0	0	0	0	0[a]
		10 mg/kg/day	0	0	3	2	0	5
		3 mg/kg/day	0	0	3	3	0	6
C-32	Periarteritis and sclerosis of mesenteric and/or other blood vessels - very slight	0 mg/kg/day	0	1	2	16	3	22
		30 mg/kg/day	0	0	1	5	1	7
		10 mg/kg/day	0	0	7	9	2	18
		3 mg/kg/day	0	0	3	4	5	12
C-33	Periarteritis and sclerosis of mesenteric and/or other blood vessels - slight	0 mg/kg/day	0	0	2	11	2	15
		30 mg/kg/day	0	0	1	5	3	9
		10 mg/kg/day	0	0	4	3	0	7
		3 mg/kg/day	0	0	4	4	1	9
C-34	Periarteritis and sclerosis of mesenteric and/or other blood vessels - moderate	0 mg/kg/day	0	0	1	11	0	12
		30 mg/kg/day	0	0	0	4	0	4
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	7	0	8
C-35	Periarteritis and sclerosis of mesenteric and/or other blood vessels - pronounced	0 mg/kg/day	0	0	2	7	0	9
		30 mg/kg/day	0	0	0	1	3	4
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Blood Vessels (Cont'd)							
C-36	Thrombosis secondary to periarteritis and sclerosis	0 mg/kg/day	0	0	1	9	0	10
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	3	0	3
C-37	Mineralization of vascular channels - isolated or focal	0 mg/kg/day	0	0	1	8	0	9
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	2	1	0	3
C-38	Mineralization of vascular channels - disseminated	0 mg/kg/day	0	0	1	5	0	6
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	2	0	2
C-39	Hemorrhagic rupture of mesenteric vessel with severe periarteritis	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	PANCREAS							
	Acinar							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
P-01	Focal atrophy of pancreatic acinar tissue	0 mg/kg/day	0	2	6	28	2	38
		30 mg/kg/day	0	0	3	12	13	28
		10 mg/kg/day	0	0	7	7	1	15
		3 mg/kg/day	0	0	2	14	5	21
P-02	Focal fibrosis of atrophic pancreatic acinar tissue	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	3	3
P-03	Focal inflammation of pancreas	0 mg/kg/day	0	0	5	5	1	11
		30 mg/kg/day	0	0	0	3	5	8
		10 mg/kg/day	0	0	3	0	0	3
		3 mg/kg/day	0	0	1	0	2	3
P-04	Hyperplastic focus(foci), nodule(s) of pancreatic acini	0 mg/kg/day	0	0	1	14	0	15[a]
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	2	4	1	7
		3 mg/kg/day	0	0	2	0	0	2[a]
P-10	Pancreatic acinar adenoma	0 mg/kg/day	0	1	2	19	4	26
		30 mg/kg/day	0	0	3	9	8	20
		10 mg/kg/day	0	0	2	4	3	9
		3 mg/kg/day	0	0	2	12	3	17
P-11	Pancreatic acinar adenocarcinoma	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Acinar (Cont'd)							
P-12	Multifocal atrophy of acinar tissue	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-13	Vacuole formation in acinar tissue	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	0	0	0	0
P-14	Focal necrosis of pancreas	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Islets of Langerhans							
P-20	Enlarged pancreatic islet	0 mg/kg/day	0	1	0	1	0	2
		30 mg/kg/day	0	0	1	1	1	3
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	1	0	1	2
P-25	Pancreatic islet cell adenoma	0 mg/kg/day	0	1	0	10	2	13
		30 mg/kg/day	0	0	0	2	5	7
		10 mg/kg/day	0	0	1	5	1	7
		3 mg/kg/day	0	0	2	5	3	10
P-26	Pancreatic islet cell adenocarcinoma	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	URINARY SYSTEM							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Kidney							
U-01	Dilated renal tubules with eosinophilic cast formation - slight	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-04	Focal atrophy of renal tubules	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-08	Mineralized deposits in renal parenchyma	0 mg/kg/day	0	0	3	14	0	17
		30 mg/kg/day	0	0	0	1	0	1[a]
		10 mg/kg/day	0	0	6	1	0	7
		3 mg/kg/day	0	0	5	5	0	10
U-09	Mineralized deposits in renal pelvis	0 mg/kg/day	0	0	5	16	1	22
		30 mg/kg/day	0	0	1	5	2	8
		10 mg/kg/day	0	0	1	2	0	3[a]
		3 mg/kg/day	0	0	4	7	4	15

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Kidney (Cont'd)							
U-10	Hematocyst in renal pelvis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-11	Cyst formation in kidney not due to chronic nephropathy	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	2	1	0	3
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	1	1	2
U-12	Inflammation of renal pelvis	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-13	Progressive chronic nephropathy - very slight	0 mg/kg/day	0	0	2	2	0	4
		30 mg/kg/day	0	0	1	1	4	6
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	2	0	1	3
U-14	Progressive chronic nephropathy - slight	0 mg/kg/day	0	1	3	4	1	9
		30 mg/kg/day	0	0	1	2	5	8
		10 mg/kg/day	0	0	2	5	0	7
		3 mg/kg/day	0	0	0	3	3	6
U-15	Progressive chronic nephropathy - moderate	0 mg/kg/day	0	1	1	11	2	15
		30 mg/kg/day	0	0	3	6	5	14
		10 mg/kg/day	0	0	4	5	3	12
		3 mg/kg/day	0	0	2	7	3	12
U-16	Progressive chronic nephropathy - pronounced	0 mg/kg/day	0	0	7	46	3	56
		30 mg/kg/day	0	0	4	16	2	22[a]
		10 mg/kg/day	0	0	16	13	1	30
		3 mg/kg/day	0	0	8	18	2	28
U-18	Focal tubular hyperplasia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-19	Dilated renal pelvis	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	1	0	1
U-22	Increased hematogenous pigment associated with chronic renal disease	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-23	Nodular proliferation of renal pelvis epithelium	0 mg//kg/day	0	0	3	32	6	41
		30 mg/kg/day	0	0	1	14	7	22
		10 mg/kg/day	0	0	3	9	1	13[a]
		3 mg/kg/day	0	0	6	8	6	20

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Kidney (Cont'd)							
U-24	Focal hyperplasia of renal pelvis epithelium	0 mg/kg/day	0	0	3	15	1	19
		30 mg/kg/day	0	0	4	5	1	10
		10 mg/kg/day	0	0	2	4	0	6
		3 mg/kg/day	0	0	4	4	0	8
U-25	Multifocal suppurative nephritis, hyperplasia of renal pelvis epithelium, with inflammation and a fibrotic area in renal pelvis	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-26	Focal purulent nephritis not associated with nephropathy	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-27	Eosinophilic material within epithelium of renal tubules	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-28	Area of interstitial fibrosis and hyperplasia of renal tubules	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-29	Anomalous focus of columnar mucosal epithelium in cortex of kidney	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-51	Renal pelvis transitional cell adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-33	Renal tubular adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-55	Papillary adenoma of renal pelvis (unilateral)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-37	Perirenal steatitis (inflammation)	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-38	Lipidosis (fatty change)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Kidney (Cont'd)							
U-39	Thrombosed renal vessel	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-40	Focal granulomatous inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-41	Fibrosis, lipidosis, hematocyst, organized hematoma with secondary fibrosis, lipidosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-42	Accumulation of lipid laden macrophages	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-43	Single subcapsular focus of fibrosis and inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-58	Renal carcinoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Urogenital Tract							
	Number of urinary bladders examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	8	25	16	49
		10 mg/kg/day	0	1	20	22	4	47
		3 mg/kg/day	0	1	12	28	9	50
U-70	Diffuse urocystitis	0 mg/kg/day	0	0	1	3	1	5
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
U-71	Focal urocystitis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	1	1	3
U-72	Focal hyperplasia of urinary bladder mucosa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2
U-73	Diffuse hyperplasia of urinary bladder mucosa	0 mg/kg/day	0	0	1	3	1	5
		30 mg/kg/day	0	0	0	3	1	4
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Urogenital Tract (Cont'd)							
U-74	Organized plug within lumen of urinary bladder	0 mg/kg/day	0	0	3	3	0	6
		30 mg/kg/day	0	0	2	2	1	5
		10 mg/kg/day	0	0	2	0	1	3
		3 mg/kg/day	0	0	1	0	1	2
U-78	Transitional cell carcinoma of urinary bladder with metastasis to kidney	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-79	Diffuse epithelial hyperplasia of ureter secondary to calculi	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-80	Organized plug of secretory material in urethra	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-81	Focal hemorrhage of bladder secondary to inflammation of accessory sex glands	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-83	Mineralized deposits in lumen of urinary bladder	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-84	Diffuse inflammation of urethra	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-85	Single submucosal lymphoid aggregate	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-86	Inflammation of accessory sex glands with extension into muscular layer of urinary bladder	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
	Testes							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
X-01	Unilateral slight decrease in spermatogenesis	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Testes (Cont'd)							
X-02	Bilateral slight decrease in spermatogenesis	0 mg/kg/day	0	0	1	4	3	8
		30 mg/kg/day	0	0	2	0	1	3
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	0	2	0	2
X-03	Unilateral moderate to pronounced decrease in spermatogenesis (atrophy) - focal	0 mg/kg/day	0	0	1	4	0	5
		30 mg/kg/day	0	0	1	4	1	6
		10 mg/kg/day	0	0	4	2	1	7
		3 mg/kg/day	0	0	4	4	3	11[a]
X-04	Unilateral moderate to pronounced decrease in spermatogenesis (atrophy) - diffuse	0 mg/kg/day	0	0	1	7	1	9
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	2	2	4
X-05	Bilateral moderate to pronounced decrease in spermatogenesis (atrophy) - focal	0 mg/kg/day	0	0	2	18	0	20
		30 mg/kg/day	0	0	0	3	2	5[a]
		10 mg/kg/day	0	0	1	7	2	10
		3 mg/kg/day	0	0	0	7	0	7
X-06	Bilateral moderate to pronounced decrease in spermatogenesis (atrophy) - diffuse	0 mg/kg/day	0	0	1	5	0	6
		30 mg/kg/day	0	0	1	8	1	10[a]
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	1	1
X-07	Mineralization of atrophic seminiferous tubules	0 mg/kg/day	0	0	2	9	0	11
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	1	1	3	5
X-08	Interstitial edema	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-09	Focal hyperplasia of interstitial cells	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
X-20	Interstitial cell adenoma of testis	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	1	0	2	3
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	1	1
X-10	Sperm granuloma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-21	Mesothelioma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-22	Sertoli cell tumor	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Accessory Sex Glands							
	Number of accessory sex glands examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	8	25	16	49
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Number of epididymal tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	27	9	49
X-30	Slight decrease in secretory content of accessory sex glands	0 mg/kg/day	0	0	7	31	2	40
		30 mg/kg/day	0	0	3	10	7	20
		10 mg/kg/day	0	1	9	17	3	30
		3 mg/kg/day	0	0	6	19	4	29
X-31	Moderate to pronounced decrease in secretory content of accessory sex glands (atrophy)	0 mg/kg/day	1	2	4	10	1	18
		30 mg/kg/day	0	0	4	3	6	13
		10 mg/kg/day	0	0	6	2	0	8
		3 mg/kg/day	0	1	1	3	1	6
X-32	Inflammation of accessory sex glands with or without edema	0 mg/kg/day	0	0	1	7	1	9
		30 mg/kg/day	0	0	0	4	1	5
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	3	0	4
X-33	Focal prostatic epithelial hyperplasia	0 mg/kg/day	0	1	0	5	0	6
		30 mg/kg/day	0	0	1	0	2	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
X-40	Decreased (and/or degenerate appearing) spermatogenic elements in epididymis	0 mg/kg/day	0	0	5	25	0	30
		30 mg/kg/day	0	0	4	15	1	20
		10 mg/kg/day	0	0	4	7	0	11
		3 mg/kg/day	0	0	1	7	3	11
X-41	Focal interstitial inflammation, edema and/or atrophy of epididymis	0 mg/kg/day	0	0	1	16	0	17
		30 mg/kg/day	0	0	2	8	1	11
		10 mg/kg/day	0	0	2	2	0	4
		3 mg/kg/day	0	0	0	3	0	3[a]
X-42	Cystic dilatation of seminal vesicle	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-43	Corpora amylacea in accessory sex glands	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	MUSCULOSKELETAL SYSTEM							
	Number of tissues examined	0 mg/kg/day	1	1	14	62	6	84
		30 mg/kg/day	0	0	8	25	15	48
		10 mg/kg/day	0	1	20	22	4	47
		3 mg/kg/day	0	1	11	25	9	46

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	MUSCULOSKELETAL SYSTEM (Cont'd)							
M-01	Focal degeneration and inflammation of skeletal muscle	0 mg/kg/day	0	1	0	23	5	29
		30 mg/kg/day	0	0	2	10	5	17
		10 mg/kg/day	0	0	4	6	2	12
		3 mg/kg/day	0	0	1	9	5	15
M-04	Multifocal degeneration, inflammation, with or without atrophy of skeletal muscle	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
M-06	Chondrosarcoma of rib	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
M-07	Vertebral healing reaction to fracture	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	GASTROINTESTINAL SYSTEM							
	Oral Cavity							
	Number of gingival tissues examined	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-06	Oral papilloma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-08	Focal inflammation and keratinization of epithelium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Tongue							
	Number of tissues examined	0 mg/kg/day	0	1	13	63	6	83
		30 mg/kg/day	0	0	8	25	16	49
		10 mg/kg/day	0	0	19	23	4	46
		3 mg/kg/day	0	1	12	28	9	50
G-10	Vasculitis and vascular degeneration	0 mg/kg/day	0	0	3	11	2	16
		30 mg/kg/day	0	0	1	3	5	9
		10 mg/kg/day	0	0	5	2	0	7
		3 mg/kg/day	0	0	2	4	3	9
G-11	Inflammation and/or hyperplasia of submucosal glands	0 mg/kg/day	0	1	7	44	3	55
		30 mg/kg/day	0	0	4	15	7	26
		10 mg/kg/day	0	0	6	9	3	18
		3 mg/kg/day	0	0	7	10	4	21

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p \leq 0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Tongue (Cont'd)							
G-13	Degeneration and inflammation of muscles	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-15	Squamous metaplasia of tongue	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	2	1	1	4
		3 mg/kg/day	0	0	0	0	1	1
G-16	Hyperplasia and/or hyperkeratosis of squamous epithelium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-17	Focal hemorrhage	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-20	Stratified squamous cell carcinoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	2	1	4
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Salivary Glands							
	Number of tissues examined	0 mg/kg/day	1	2	12	59	6	80
		30 mg/kg/day	0	0	7	23	15	45
		10 mg/kg/day	0	1	17	23	4	45
		3 mg/kg/day	0	1	10	25	8	44
G-30	Interstitial edema of salivary gland	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-31	Sialoadenitis	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-33	Hypertrophy of glandular components of salivary gland	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-35	Interstitial inflammation	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	2	2	4
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	2	1	4
G-36	Atrophy of acinar tissue	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	3	2	5
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	1	1	2

Data listed as number of rats for which the listed observation was present.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Salivary Glands (Cont'd)							
G-37	Cystic dilatation of ductular structures	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-38	Adenoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-39	Increased numbers of mast cells	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Stomach							
	Number of tissues examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	22	4	49
		3 mg/kg/day	0	1	12	27	9	49
G-45	Focal gastric ulceration	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
G-46	Dilatation of gastric pits	0 mg/kg/day	0	1	4	28	3	36
		30 mg/kg/day	0	0	5	9	4	18
		10 mg/kg/day	0	0	10	8	1	19
		3 mg/kg/day	0	0	5	9	4	18
G-47	Mineralization of gastric muscularis and/or gastric mucosa	0 mg/kg/day	0	0	6	19	0	25
		30 mg/kg/day	0	0	0	1	0	1[a]
		10 mg/kg/day	0	0	6	3	0	9
		3 mg/kg/day	0	0	7	6	0	13
G-48	Submucosal edema of stomach	0 mg/kg/day	0	0	1	4	0	5
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	0	1	1
G-49	Hyperplasia of nonglandular mucosal epithelium	0 mg/kg/day	0	1	2	4	0	7
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	3	1	5
G-50	Focal area of inflammation of stomach wall	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	0	0	0	0
G-51	Hyperkeratosis of nonglandular mucosa of stomach	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	3	1	5

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Stomach (Cont'd)							
G-52	Focal erosion of gastric mucosa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-53	Diffuse inflammation of stomach wall	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	0	1	2
G-55	Keratinized cyst formation - wall of stomach	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
G-59	Stratified squamous papilloma of gastric mucosa	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-61	Anomalous epithelial cyst formation in gastric wall	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	Small Intestine							
	Number of tissues examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	25	9	47
G-71	Focal ulceration and/or inflammation of small intestine	0 mg/kg/day	0	0	1	3	0	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-74	Reactive lymphoid hyperplasia of small intestine	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	1	2	0	3
		3 mg/kg/day	0	0	0	1	2	3
G-76	Mucocystadenocarcinoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-77	Adenocarcinoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2
G-78	Fibrosarcoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Small Intestine (Cont'd)							
G-79	Lymphosarcoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-80	Multifocal hemorrhage in small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-81	Hyperemia of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-87	Necrotic enteritis	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-88	Undifferentiated sarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89	Lymphosarcoma of small intestine with metastasis to mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89A	Mucocystadenoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89B	Segmental necrosis due to intussusception	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89C	Papillary adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Large Intestine							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	21	23	4	49
		3 mg/kg/day	0	1	12	27	8	48
G-90	Intestinal nematodiasis	0 mg/kg/day	0	0	1	4	1	6
		30 mg/kg/day	0	0	1	3	4	8
		10 mg/kg/day	0	0	2	0	1	3
		3 mg/kg/day	0	0	0	4	4	8

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Large Intestine (Cont'd)							
G-91	Diffuse inflammation of large intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-92	Focal inflammation of large intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-93	Edema and/or inflammation of wall of large intestine and/or cecum	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	4	0	5[a]
G-94	Edema and/or ulceration with or without inflammation of cecum due to uremia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-95	Reactive lymphoid hyperplasia of large intestine	0 mg/kg/day	0	0	0	6	0	6
		30 mg/kg/day	0	0	0	4	1	5
		10 mg/kg/day	0	0	0	3	1	4
		3 mg/kg/day	0	0	0	1	0	1
G-99	Reactive lymphoid hyperplasia of cecum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-100	Lymphosarcoma of large intestine with metastasis to mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Peritoneal Cavity							
G-110	Peritonitis secondary to perforated ulcer	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND							
	Subcutaneous (nontumorous)							
S-01	Subcutaneous abscess	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	2	0	2
	Subcutaneous (tumorous)[b]							
	Total number of subcutaneous tumors/number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	0/0	1/1	3/3	0/0	9/9
		30 mg/kg/day	0/0	0/0	1/1	3/3	3/3	7/7
		10 mg/kg/day	0/0	1/1	1/1	1/1	0/0	3/3
		3 mg/kg/day	0/0	0/0	1/1	6/6	5/3	12/10

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

[b]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Subcutaneous (tumorous)[a] (Cont'd)							
S-12	Subcutaneous fibroma	0 mg/kg/day	0/0	0/0	1/1	3/3	0/0	4/4
		30 mg/kg/day	0/0	0/0	0/0	1/1	2/2	3/3
		10 mg/kg/day	0/0	1/1	0/0	1/1	0/0	2/2
		3 mg/kg/day	0/0	0/0	1/1	4/4	3/1	8/6
S-17	Subcutaneous adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-20	Malignant fibrous histiocytoma with metastasis to abdominal cavity	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-22	Malignant fibrous histiocytoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-23	Subcutaneous liposarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-24	Subcutaneous myxoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-24A	Subcutaneous carcinoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Mammary Gland (nontumorous)							
	Number of tissues examined	0 mg/kg/day	0	0	0	4	1	5
		30 mg/kg/day	0	0	1	0	2	3
		10 mg/kg/day	0	0	8	3	0	11
		3 mg/kg/day	0	0	4	6	2	12
S-25	Mammary gland hyperplasia	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	2	1	0	3
S-26	Mammary gland galactocele formation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Mammary (tumorous)[a]							
	Total number of subcutaneous tumors /number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	0/0	1/1	8/8	0/0	9/9
		30 mg/kg/day	0/0	0/0	1/1	3/3	3/3	7/7
		10 mg/kg/day	0/0	1/1	1/1	1/1	0/0	3/3
		3 mg/kg/day	0/0	0/0	1/1	6/6	5/3	12/10
S-30	Mammary gland fibroadenoma/ adenofibroma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	2/2	2/2
S-32	Mammary gland adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-33	Mammary gland adenocarcinoma with metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-35	Mammary gland carcinosarcoma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Integument (nontumorous)							
	Number of tissues examined	0 mg/kg/day	0	1	9	59	6	75
		30 mg/kg/day	0	0	5	23	16	44
		10 mg/kg/day	0	0	16	19	4	39
		3 mg/kg/day	0	0	7	27	9	43
S-60	Epidermal inclusion cyst(s) with or without inflammation/rats bearing epidermal cysts	0 mg/kg/day	0/0	1/1	0/0	3/3	3/2	7/6
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	2/2	3/3	0/0	5/5
		3 mg/kg/day	0/0	0/0	0/0	1/1	1/1	2/2
S-61	Abscess of preputial gland	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	3	4
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
S-63	Superficial necrotic and inflammatory debris on integument	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-64	Epidermal hyperplasia and diffuse inflammation of debris adjacent to subcutaneous mass or abscess	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
S-68	Keratinizing hyperkeratosis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-69	Area of inflammation of subcutaneous adnexa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Integument (tumorous)[1]							
	Total number of cutaneous tumors/ number of rats bearing cutaneous tumors	0 mg/kg/day	0/0	0/0	0/0	2/2	0/0	2/2
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	2/2	0/0	1/1	3/3
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-72	Squamous papilloma of integument	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-75A	Adnexal carcinoma with metastasis to lungs	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76A	Fibrosarcoma adjacent to ear tag	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76C	Adnexal cystadenoma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Ear Canal[a]							
	Total number of ear canal tumors/ number of rats bearing ear canal tumors	0 mg/kg/day	0/0	0/0	1/1	4/4	0/0	5/5
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76	Zymbal gland carcinoma of ear canal	0 mg/kg/day	0/0	0/0	1/1	3/3	0/0	4/4
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-79	Subauricular undifferentiated sarcoma with metastasis to lungs	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	NERVOUS SYSTEM							
	Brain							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
N-01	Focal malacia in brain	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1

[1]Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	NERVOUS SYSTEM (Cont'd)							
N-02	Focal gliosis of brain	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
N-03	Focal inflammation in brain	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-04	Vacuolization of white matter of brain which may or may not be artifact	0 mg/kg/day	0	1	0	18	0	19
		30 mg/kg/day	0	0	0	6	0	6
		10 mg/kg/day	0	0	2	4	1	7
		3 mg/kg/day	0	0	8	4	0	12
N-05	Mineralized deposits in brain	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-06	Compression malacia or hemorrhage, and/or hematogenous pigment in brain adjacent to pituitary adenocarcinoma	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-08	Hemorrhage in brain	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-12	Hemorrhage surrounding pineal gland	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-22	Astrocytoma within cerebrum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-23	Astrocytoma within cerebellum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
N-25	Granular cell myoblastoma of brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
N-19	Astrocytoma or mixed glial neoplasm of cerebellum	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Spinal Cord							
	Number of tissues examined	0 mg/kg/day	1	1	14	63	6	85
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	21	23	4	49
		3 mg/kg/day	0	1	12	27	9	49
N-40	Vacuolization of spinal cord which may or may not be artifact	0 mg/kg/day	0	0	0	11	0	11
		30 mg/kg/day	0	0	0	4	0	4
		10 mg/kg/day	0	1	5	8	2	16[a]
		3 mg/kg/day	0	1	8	4	0	13[a]
N-41	Increased number of glial cells	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
N-42	Focal hemorrhage in spinal cord	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	Peripheral Nerves							
	Number of tissues examined	0 mg/kg/day	1	1	13	61	6	82
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	20	23	4	48
		3 mg/kg/day	0	1	11	24	9	45
N-60	Vacuolization of peripheral nerves	0 mg/kg/day	0	0	6	38	6	50
		30 mg/kg/day	0	0	0	14	13	27
		10 mg/kg/day	0	0	2	4	23	29
		3 mg/kg/day	0	0	4	12	7	23
N-62	Mediastinal malignant schwannoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-64	Schwannoma of cranial nerve	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-66	Focal inflammation of peripheral nerve	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-67	Mineralization of peripheral nerve	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-68	Malignant schwannoma of cranial nerve with involvement of cerebrum and nasal turbinates	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Pituitary							
	Number of tissues examined	0 mg/kg/day	1	2	13	59	6	82
		30 mg/kg/day	0	0	9	24	16	49
		10 mg/kg/day	0	1	21	22	4	48
		3 mg/kg/day	0	1	11	26	9	47
N-80	Retention cyst(s) in pituitary	0 mg/kg/day	0	0	3	3	1	7
		30 mg/kg/day	0	0	0	1	5	6
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	4	1	5
N-81	Pituitary hematocyst	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
N-82	Increased hematogenous pigment in pituitary	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-83	Hemangiectasis of pituitary	0 mg/kg/day	0	0	2	4	1	7
		30 mg/kg/day	0	0	0	0	4	4
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	3	3
N-85	Hyperplastic focus in pituitary	0 mg/kg/day	0	1	4	10	2	17
		30 mg/kg/day	0	0	0	4	1	5
		10 mg/kg/day	0	0	3	2	0	5
		3 mg/kg/day	0	0	3	1	2	6
N-86	Pituitary adenoma formation(s)	0 mg/kg/day	0	0	2	17	3	22
		30 mg/kg/day	0	0	0	5	5	10
		10 mg/kg/day	0	0	3	9	1	13
		3 mg/kg/day	0	0	1	4	5	10
N-87	Pituitary adenocarcinoma without extension or invasion of brain	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	2	0	2
N-88	Pituitary adenocarcinoma with invasion of brain	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-89	Mineralized debris in pituitary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-92	Hemorrhage adjacent to pituitary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Cranial Cavity							
N-71	Neurofibroma adjacent to cranial nerve	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	ADRENAL GLAND							
	Number of tissues examined	0 mg/kg/day	1	2	12	63	6	84
		30 mg/kg/day	0	0	9	25	15	50
		10 mg/kg/day	0	0	22	23	4	49
		3 mg/kg/day	0	1	12	28	9	50
	General							
A-01	Adrenal gland hematocyst formation	0 mg/kg/day	0	1	0	15	1	17
		30 mg/kg/day	0	0	0	6	3	9
		10 mg/kg/day	0	0	2	7	2	11
		3 mg/kg/day	0	0	0	6	2	8
A-02	Hemangiectasis of adrenal gland(s)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-03	Isolated focus of purulent inflammation in adrenal glands	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
A-05	Congestion	0 mg/kg/day	0	1	1	0	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	3	0	0	3
		3 mg/kg/day	0	0	0	1	0	1
	Cortex							
A-20	Adrenal cortical necrosis with or without hemorrhage and/or purulent inflammation	0 mg/kg/day	0	0	0	5	0	5
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	1	0	1
A-21	Vacuolization of adrenal cortical cells	0 mg/kg/day	0	1	9	48	4	62
		30 mg/kg/day	0	0	5	17	14	36
		10 mg/kg/day	0	0	13	20	4	37
		3 mg/kg/day	0	0	5	25	5	35
A-22	Mineralization of adrenal cortex	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-23	Atrophy of adrenal cortex	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2
A-26	Aggregates of hematogenous pigment in adrenal cortex	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Cortex (Cont'd)							
A-30	Hyperplastic focus(foci), nodule(s) of adrenal cortex	0 mg/kg/day	0	0	3	16	0	19
		30 mg/kg/day	0	0	3	5	2	10
		10 mg/kg/day	0	0	6	4	2	12
		3 mg/kg/day	0	1	4	9	4	18
A-31	Adenoma of adrenal cortex	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	3	0	3
A-32	Adenocarcinoma of adrenal cortex	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	Medulla							
A-40	Hyperplastic nodule of adrenal medulla(s)	0 mg/kg/day	0	0	3	17	2	22
		30 mg/kg/day	0	0	3	5	2	10
		10 mg/kg/day	0	0	7	7	1	15
		3 mg/kg/day	0	0	4	6	4	14
A-41	Adrenal pheochromocytoma, unilateral	0 mg/kg/day	0	0	2	29	1	32
		30 mg/kg/day	0	0	1	9	4	14
		10 mg/kg/day	0	0	4	8	3	15
		3 mg/kg/day	0	0	2	5	4	11
A-42	Adrenal pheochromocytoma, bilateral	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	4	2	6
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	1	0	1
A-44	Adrenal pheochromocytoma, malignant, no metastasis - unilateral	0 mg/kg/day	0	0	0	5	1	6
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	2	1	3
A-45	Adrenal pheochromocytoma, malignant - unilateral	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-46	Adrenal ganglioneuroma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-47	Adrenal pheochromocytoma, malignant - no metastasis - bilateral	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	EYE							
	Number of tissues examined	0 mg/kg/day	0	2	13	61	6	82
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	20	23	4	48
		3 mg/kg/day	0	1	11	28	9	49

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	EYE (Cont'd)							
E-01	Focal keratitis of eye	0 mg/kg/day	0	0	1	7	1	9
		30 mg/kg/day	0	0	2	5	3	10
		10 mg/kg/day	0	0	0	5	1	6
		3 mg/kg/day	0	0	1	4	1	6
E-02	Diffuse keratitis of eye	0 mg/kg/day	0	1	0	24	0	25
		30 mg/kg/day	0	0	0	3	5	8
		10 mg/kg/day	0	0	4	1	1	6
		3 mg/kg/day	0	0	2	5	1	8
E-03	Focal or diffuse hyperplasia of corneal epithelium	0 mg/kg/day	0	1	0	18	1	20
		30 mg/kg/day	0	0	1	2	5	8
		10 mg/kg/day	0	0	3	2	2	7
		3 mg/kg/day	0	0	1	3	1	5
E-07	Focal mineralization of eye	0 mg/kg/day	0	0	1	3	0	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	2	0	3
E-08	Retrobulbar keratinizing squamous cell carcinoma with extension into cranium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-09	Intraorbital undifferentiated sarcoma with invasion of cranial cavity	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
E-10	Phthisis bulbi with chronic inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
E-11	Focal inflammation of ciliary body	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-12	Bilateral retinal atrophy and degeneration	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	MISCELLANEOUS							
Z-02	Postmortem autolytic changes in all organs	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-03	Inflammation of mesenteric fat	0 mg/kg/day	0	0	1	5	0	6
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	0	2	[illegible]	2

Data listed as number of rats for which the listed observation was present.

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TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	MISCELLANEOUS (Cont'd)							
Z-04	Focal mesenteric steatitis and/or necrosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
Z-07	Mediastinal fat inflammation	0 mg/kg/day	0	0	0	6	0	6
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	0	0	1
Z-08	Generalized increased number of mast cells	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-11	Mesenteric lipoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-12	Purulent abscessation of mesentery	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-13	Granulomatous mediastinal inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	LYMPHORETICULAR							
	Lymph Nodes - General							
L-01	Generalized lymphosarcoma with widespread metastasis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
L-03	Generalized lymphosarcoma (reticular cell type) with widespread metastasis	0 mg/kg/day	1	0	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thoracic Lymph Node(s)							
	Number of tissues examined	0 mg/kg/day	1	2	13	59	6	81
		30 mg/kg/day	0	0	7	21	16	44
		10 mg/kg/day	0	1	22	22	3	48
		3 mg/kg/day	0	1	11	27	7	46
L-20	Pooling of edema, pigment, and/or red blood cells in sinusoids of thoracic lymph node	0 mg/kg/day	1	1	3	48	4	57
		30 mg/kg/day	0	0	3	15	8	26
		10 mg/kg/day	0	0	9	18	3	30
		3 mg/kg/day	0	0	6	17	5	28

Data listed as number of rats for which the listed observation was present.

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Thoracic Lymph Node(s) (Cont'd)							
L-22	Reactive lymphoid hyperplasia of thoracic lymph node(s)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-23	Congestion of thoracic lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	3	0	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-24	Cystic dilatation of lymphatics of thoracic lymph node(s)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-25	Reticuloendothelial cell hyperplasia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
	Mesenteric Lymph Node(s)							
	Number of tissues examined	0 mg/kg/day	1	2	12	60	6	81
		30 mg/kg/day	0	0	9	23	16	48
		10 mg/kg/day	0	1	19	20	4	44
		3 mg/kg/day	0	0	9	25	8	42
L-30	Pooling of edema, pigment, and/or red blood cells in sinusoids of mesenteric lymph node	0 mg/kg/day	0	1	7	39	4	51
		30 mg/kg/day	0	0	6	16	12	34
		10 mg/kg/day	0	0	9	9	3	21
		3 mg/kg/day	0	0	1	15	4	20
L-31	Inflammatory process in mesenteric lymph node	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-32	Reactive lymphoid hyperplasia of mesenteric lymph node	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	1	3
L-33	Cystic distention of lymphatics of mesenteric lymph node	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-34	Fibrotic tissue in mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-35	Increased numbers of histiocytes in mesenteric lymph node	0 mg/kg/day	0	1	1	1	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
L-36	Congestion of mesenteric lymph node	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

20407

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	36
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Subcutaneous Lymph Nodes							
	Number of tissues examined	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	1	3	4	8
		10 mg/kg/day	0	0	0	3	1	4
		3 mg/kg/day	0	0	1	4	1	6
L-42	Reactive hyperplasia of subcutaneous lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-46	Fibrotic tissue in subcutaneous lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Periaortal Lymph Node							
	Number of tissues examined	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-90	Slight reactive lymphoid hyperplasia, edema, and pooling of red blood cells	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	Lymph Nodes (unknown location)							
	Number of tissues examined	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
L-95	Edema, pooling of red blood cells, and cystic dilatation	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thymus							
	Number of tissues examined	0 mg/kg/day	1	2	12	43	1	59
		30 mg/kg/day	0	0	4	22	12	38
		10 mg/kg/day	0	1	14	17	3	35
		3 mg/kg/day	0	1	10	19	7	37
L-52	Epithelial hyperplasia of thymus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-56	Thymic lymphosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
L-59	Focal hemorrhage (probably agonal)	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

20408

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	SPLEEN							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
L-60	Splenic atrophy with or without increased hemosiderin pigment	0 mg/kg/day	0	0	5	4	0	9
		30 mg/kg/day	0	0	0	3	0	3
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	1	2	2	0	5
L-61	Increased hematogenous pigment in spleen	0 mg/kg/day	0	1	5	42	3	51
		30 mg/kg/day	0	0	6	18	13	37
		10 mg/kg/day	0	0	18	13	1	32
		3 mg/kg/day	0	0	9	22	6	37
L-62	Increased extramedullary hematopoiesis in spleen	0 mg/kg/day	0	0	5	45	5	55
		30 mg/kg/day	0	0	3	16	9	28
		10 mg/kg/day	0	1	14	13	4	32
		3 mg/kg/day	0	0	9	22	8	39
L-63	Reticuloendothelial or lymphoid hyperplasia of spleen	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
L-70	Lymphosarcoma of spleen	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-64	Congestion of spleen	0 mg/kg/day	0	2	0	0	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
L-68	Capsular cyst formation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	THYROID AND PARATHYROID							
	Thyroid							
	Number of tissues examined	0 mg/kg/day	0	2	13	63	6	84
		30 mg/kg/day	0	0	8	24	16	48
		10 mg/kg/day	0	1	22	23	3	49
		3 mg/kg/day	0	1	12	28	8	49
T-01	Thyroid interfollicular C-cell adenoma	0 mg/kg/day	0	0	0	3	3	6
		30 mg/kg/day	0	0	1	2	4	7
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	1	3
T-03	Thyroid interfollicular C-cell adenocarcinoma with metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

20409

TABLE 34 (Continued)

MICROSCOPIC OBSERVATIONS ON MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Thyroid (Cont'd)							
T-04	Hyperplastic focus of interfollicular C cells within thyroid	0 mg/kg/day	0	0	1	13	1	15
		30 mg/kg/day	0	0	0	5	6	11
		10 mg/kg/day	0	0	0	4	0	4
		3 mg/kg/day	0	0	2	7	2	11
T-05	Thyroid hyperplasia (follicular)	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
T-06	Retention cyst in thyroid	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	0	0	2	2
T-10	Mineralized deposits in thyroid	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
T-11	Focal follicular overdistention	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	Parathyroid							
	Number of tissues examined	0 mg/kg/day	0	1	12	60	6	79
		30 mg/kg/day	0	0	7	24	12	43
		10 mg/kg/day	0	0	19	22	3	44
		3 mg/kg/day	0	1	10	25	6	42
T-20	Secondary parathyroid hyperplasia (renal)	0 mg/kg/day	0	0	8	38	4	50
		30 mg/kg/day	0	0	4	17	4	25
		10 mg/kg/day	0	0	14	14	1	29
		3 mg/kg/day	0	0	8	12	3	23
T-22	Hyperplastic focus in parathyroid	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
T-23	Interstitial fibrosis of parathyroid	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
T-24	Hematocyst in parathyroid	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

20410

TABLE 35

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LIVER							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Hepatocellular Changes							
H-01	Necrosis with accompanying inflammatory reaction - single focus	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	1	0	3	4
H-02	Necrosis with accompanying inflammatory reaction - focal	0 mg/kg/day	0	0	2	1	0	3
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	1	1	0	2
H-03	Necrosis with accompanying inflammatory reaction - multifocal	0 mg/kg/day	0	1	1	3	0	5
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	2	0	0	2
		3 mg/kg/day	0	0	0	2	0	2
H-05	Cytoplasmic vacuolization - single focus	0 mg/kg/day	0	0	1	1	1	3
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	0	0	0
H-06	Cytoplasmic vacuolization - focal	0 mg/kg/day	0	0	4	7	12	23
		30 mg/kg/day	0	0	0	2	5	7
		10 mg/kg/day	0	0	1	5	1	7
		3 mg/kg/day	0	0	0	2	4	6
H-07	Cytoplasmic vacuolization - multifocal	0 mg/kg/day	0	0	0	2	4	6
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	2	3
		3 mg/kg/day	0	0	0	1	2	3
H-08	Cytoplasmic vacuolization - disseminated	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	2	0	3
H-09	Cytoplasmic vacuolization - suggestive of fatty metamorphosis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	1	0	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	2	0	2
H-10	Accentuated lobular pattern due to centrilobular atrophy	0 mg/kg/day	0	1	0	4	2	7
		30 mg/kg/day	0	0	0	2	2	4
		10 mg/kg/day	0	0	2	3	0	5
		3 mg/kg/day	0	0	1	2	0	3
H-11	Hepatocellular alteration (swollen hepatocytes) - single focus	0 mg/kg/day	0	0	4	7	9	20
		30 mg/kg/day	0	0	2	5	6	13
		10 mg/kg/day	0	0	1	3	2	6
		3 mg/kg/day	0	0	3	1	10	14

Data listed as number of rats for which the listed observation was present.

20411

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Hepatocellular Changes (Cont'd)							
H-12	Hepatocellular alteration (swollen hepatocytes) - focal (2-3)	0 mg/kg/day	0	0	0	3	11	14
		30 mg/kg/day	0	0	0	2	8	10
		10 mg/kg/day	0	0	1	2	5	8
		3 mg/kg/day	0	0	1	2	1	4
H-17	Hepatocellular alteration (swollen hepatocytes) - several foci (>3)	0 mg/kg/day	0	0	3	4	8	15
		30 mg/kg/day	0	0	1	0	2	3[a]
		10 mg/kg/day	0	0	0	6	0	6
		3 mg/kg/day	0	0	0	1	3	4
H-13	Hepatocellular alteration (swollen hepatocytes) - single area	0 mg/kg/day	0	0	4	3	8	15
		30 mg/kg/day	0	0	1	3	6	10
		10 mg/kg/day	0	0	0	5	2	7
		3 mg/kg/day	0	0	0	0	5	5
H-14	Hepatocellular alteration (swollen hepatocytes) - several areas	0 mg/kg/day	0	0	1	3	7	11
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	0	0	3	3
H-15	Hyperplastic nodule(s)	0 mg/kg/day	0	0	1	0	3	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	0	0	2	2
H-16	Hepatocellular carcinoma(s)	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-18	Focal vacuole formation with or without mineralized debris	0 mg/kg/day	0	0	2	1	0	3
		30 mg/kg/day	0	0	2	2	0	4
		10 mg/kg/day	0	0	5	4	0	9[a]
		3 mg/kg/day	0	0	0	4	0	4
H-19	Focal confluent vacuole formation with or without mineralized debris	0 mg/kg/day	0	0	0	0	27	27
		30 mg/kg/day	0	0	0	1	15	16
		10 mg/kg/day	0	0	1	8	9	18
		3 mg/kg/day	0	0	0	5	15	20
H-20	Multiple multinucleated cells in liver (megakaryocytes)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	1	0	0	0	0	1
H-21	Swelling of individual hepatocytes	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
H-22	Distortion of lobular pattern	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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H-43	Fibrosis within hepatic tissue - focal	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-45	Fibrosis within hepatic tissue - disseminated	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-47	Fibrosis - subcapsular - focal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	0	0
H-49	Increased pigment aggregates in liver	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-50	Inflammatory adhesions to capsule	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-51	Thickened capsule	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a] Statistically different from control data when analyzed using Fisher's Exact Probability Test, p<0.05.

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Hepatocellular Changes (Cont'd)							
H-23	Increased mitotic figures	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Inflammatory Cell Changes							
H-40	Focal accumulation of inflammatory cells, primarily mononuclear cells within liver	0 mg/kg/day	0	1	2	4	4	11
		30 mg/kg/day	0	0	1	3	1	5
		10 mg/kg/day	0	1	1	1	2	5
		3 mg/kg/day	0	0	1	2	6	9
H-41	Focal aggregates of reticulo-endothelial cells frequently adjacent to degenerate or necrotic hepatocytes	0 mg/kg/day	0	0	8	9	19	36
		30 mg/kg/day	0	0	9	13	12	34[a]
		10 mg/kg/day	0	0	4	10	7	21
		3 mg/kg/day	0	0	2	8	7	17
H-42	Fibrosis within hepatic tissue - single focus	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Bile Ducts and Vascular Channels							
H-70	Biliary hyperplasia - focal	0 mg/kg/day	0	0	1	1	8	10
		30 mg/kg/day	0	0	0	1	3	4
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	2	2
H-71	Biliary hyperplasia - multifocal	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-72	Biliary cyst formation	0 mg/kg/day	0	0	2	2	7	11
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	1	0	2	3
H-74	Periportal inflammation	0 mg/kg/day	0	0	3	0	7	10
		30 mg/kg/day	0	0	0	2	3	5
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	1	0	1[a]
H-75	Focal periportal fibrosis	0 mg/kg/day	0	0	1	1	1	3
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	1	1
H-85	Ectasia of vascular channels	0 mg/kg/day	0	0	4	5	9	18
		30 mg/kg/day	0	0	0	1	5	6
		10 mg/kg/day	0	0	1	3	2	6
		3 mg/kg/day	0	0	1	3	6	10
H-86	Congestion of hepatic sinusoids	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	2	0	2
H-89	Perivasculitis - focal	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
H-91	Focal extramedullary hematopoiesis	0 mg/kg/day	0	0	6	16	13	35
		30 mg/kg/day	0	0	5	12	3	20
		10 mg/kg/day	0	0	8	10	1	19
		3 mg/kg/day	0	0	7	5	1	13
	RESPIRATORY SYSTEM							
	Trachea							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
R-20	Focal tracheitis	0 mg/kg/day	0	0	4	3	6	13
		30 mg/kg/day	0	0	0	1	5	6
		10 mg/kg/day	0	1	2	5	1	9
		3 mg/kg/day	0	0	4	1	4	9

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Trachea (Cont'd)							
R-23	Focal epithelial hyperplasia of trachea	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-24	Diffuse epithelial hyperplasia of trachea	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Lungs							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
R-30	Focal peribronchiolar aggregates of lymphocytes	0 mg/kg/day	0	1	15	24	30	70
		30 mg/kg/day	0	0	8	16	20	44
		10 mg/kg/day	0	1	11	18	9	39
		3 mg/kg/day	0	1	10	12	15	38
R-31	Focal interstitial inflammation	0 mg/kg/day	0	0	6	11	16	33
		30 mg/kg/day	0	0	5	9	11	25
		10 mg/kg/day	0	1	2	5	3	11[a]
		3 mg/kg/day	0	0	4	4	3	11[a]
R-32	Focal accumulation of inflammatory cells in alveoli	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
R-33	Focal accumulation of alveolar macrophages in alveoli	0 mg/kg/day	0	2	9	21	21	53
		30 mg/kg/day	0	0	7	13	10	30
		10 mg/kg/day	0	2	5	11	5	23
		3 mg/kg/day	0	0	5	8	10	23
R-34	Alveolar proteinosis	0 mg/kg/day	0	0	4	7	9	20
		30 mg/kg/day	0	0	0	5	4	9
		10 mg/kg/day	0	0	1	4	2	7
		3 mg/kg/day	0	0	2	1	7	10
R-35	Cholesterol clefts in lung	0 mg/kg/day	0	1	0	6	6	13
		30 mg/kg/day	0	0	3	6	4	13
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	1	1	1	3
R-36	Pulmonary edema, congestion, cellular exudation, with or without focal fibrosis secondary to heart failure, renal disease and/or vascular disease	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Lungs (Cont'd)							
R-38	Pulmonary atelectasis	0 mg/kg/day	0	1	0	0	1	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	1	1	0	1	0	3
		3 mg/kg/day	1	0	0	0	0	1
R-39	Alveolar mineralization	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
R-40	Focal pulmonary aggregates of hematogenous pigment	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	0	0
R-41	Focal alveolar hyperplasia	0 mg/kg/day	0	0	2	3	4	9
		30 mg/kg/day	0	1	1	5	4	11
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	2	4
R-42	Pulmonary edema	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
R-43	Focal interstitial fibrosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	1	1	1	1	4[a]
		3 mg/kg/day	0	0	0	0	0	0
R-44	Focal granulomatous inflammation with or without foreign body present	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	2	0	2	4
		10 mg/kg/day	0	1	0	1	0	2
		3 mg/kg/day	0	0	0	0	2	2
R-45	Focal pulmonary hemorrhage	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
R-48	Focal hyperplasia of bronchiolar epithelium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	2	0	0	2
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	0	0
R-49	Aspirated particulate matter	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-51	Pulmonary congestion	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Lungs (Cont'd)							
R-53	Increased perivascular aggregates of lymphocytes	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	3	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
R-54	Secretory material in bronchus or bronchiole	0 mg/kg/day	0	0	0	1	3	4
		30 mg/kg/day	0	0	1	1	2	4
		10 mg/kg/day	0	0	2	1	0	3
		3 mg/kg/day	0	0	0	1	0	1
R-59	Inflammatory cells within lumen and/or around bronchiole(s)	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	2	2	0	4
		10 mg/kg/day	0	0	1	0	1	2
		3 mg/kg/day	0	0	0	0	0	0
R-60	Focal secretory material in alveoli	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	2	2	0	4[a]
		10 mg/kg/day	0	1	1	0	0	2
		3 mg/kg/day	0	0	0	2	0	2
R-62	Multifocal alveolar hyperplasia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
R-64	Cellular exfoliation secondary to renal failure or heart failure	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
	CARDIOVASCULAR SYSTEM							
	Heart							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
C-01	Focal myocardial degeneration and inflammation with or without fibrosis - slight	0 mg/kg/day	0	0	9	5	17	31
		30 mg/kg/day	0	0	1	5	13	19
		10 mg/kg/day	0	0	2	10	6	18
		3 mg/kg/day	0	0	4	9	13	26
C-02	Focal myocardial degeneration and inflammation with or without fibrosis - moderate	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
C-03	Atrial thrombosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Heart (Cont'd)							
C-06	Subendocardial fibroelastosis of myocardium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	2	0	3[a]
C-10	Focal myocarditis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-12	Ventricular thrombosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Aorta and/or Large Anterior Mediastinal Artery(ies)							
	Number of tissues examined	0 mg/kg/day	0	1	16	30	36	83
		30 mg/kg/day	0	2	7	16	21	46
		10 mg/kg/day	1	2	10	23	11	47
		3 mg/kg/day	0	1	11	16	19	47
C-20	Focal mural mineralization	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	3	3
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	2	2	2	6[a]
C-21	Focal thickening of endothelial lining	0 mg/kg/day	0	0	3	2	7	12
		30 mg/kg/day	0	0	1	3	7	11
		10 mg/kg/day	0	0	1	5	1	7
		3 mg/kg/day	0	0	1	2	2	5
C-24	Aortic plaque formation	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Blood Vessels							
C-30	Degeneration of myocardial blood vessels	0 mg/kg/day	0	0	9	10	13	32
		30 mg/kg/day	0	1	0	7	5	13
		10 mg/kg/day	0	1	5	8	4	18
		3 mg/kg/day	0	0	4	5	6	15
C-31	Mineralization of myocardial blood vessels	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
C-32	Periarteritis and sclerosis of mesenteric and/or other blood vessels - very slight	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	1	0	3	4

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	13	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Blood Vessels (Cont'd)							
C-33	Periarteritis and sclerosis of mesenteric and/or other blood vessels - slight	0 mg/kg/day	0	0	1	1	3	5
		30 mg/kg/day	0	0	0	0	4	4
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
C-34	Periarteritis and sclerosis of mesenteric and/or other blood vessels - moderate	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
C-35	Periarteritis and sclerosis of mesenteric and/or other blood vessels - pronounced	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
C-36	Thrombosis secondary to periarteritis and sclerosis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
-37	Mineralization of vascular channels - isolated or focal	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2
	PANCREAS							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Acinar							
P-01	Focal atrophy of pancreatic acinar tissue	0 mg/kg/day	0	0	6	6	16	28
		30 mg/kg/day	0	0	1	4	8	13
		10 mg/kg/day	0	1	1	3	7	12
		3 mg/kg/day	0	0	4	4	7	15
P-02	Focal fibrosis of atrophic pancreatic acinar tissue	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
P-03	Focal inflammation of pancreas	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
P-04	Hyperplastic focus (foci), nodule(s) of pancreatic acini	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Acinar (Cont'd)							
P-10	Pancreatic acinar adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
P-13	Vacuole formation in acinar tissue	0 mg/kg/day	0	0	1	2	10	13
		30 mg/kg/day	0	0	0	0	7	7
		10 mg/kg/day	0	0	0	2	0	2[a]
		3 mg/kg/day	0	0	1	1	5	7
	Islets of Langerhans							
P-20	Enlarged pancreatic islet	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	1	2
P-25	Pancreatic islet cell adenoma	0 mg/kg/day	0	0	1	1	6	8
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	1	0	1
P-26	Pancreatic islet cell adenocarcinoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	URINARY SYSTEM							
	Kidney							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
U-01	Dilated renal tubules with eosinophilic cast formation - slight	0 mg/kg/day	0	1	4	9	4	18
		30 mg/kg/day	0	0	5	7	5	17
		10 mg/kg/day	0	2	6	7	2	17
		3 mg/kg/day	1	0	7	7	2	17
U-02	Dilated renal tubules with eosinophilic cast formation - moderate	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
U-03	Dilated renal tubules - pronounced	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-04	Focal atrophy of renal tubules	0 mg/kg/day	0	0	3	9	4	16
		30 mg/kg/day	0	0	5	7	4	16
		10 mg/kg/day	0	1	7	7	2	18[a]
		3 mg/kg/day	1	0	4	0	2	7

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Kidney (Cont'd)							
U-05	Focal glomerular adhesions	0 mg/kg/day	0	0	1	6	1	8
		30 mg/kg/day	0	0	1	5	2	8
		10 mg/kg/day	0	0	1	3	1	5
		3 mg/kg/day	0	0	2	3	1	6
U-06	Focal interstitial fibrosis	0 mg/kg/day	0	0	0	3	2	5
		30 mg/kg/day	0	0	0	1	2	3
		10 mg/kg/day	0	1	2	0	0	3
		3 mg/kg/day	0	0	0	1	0	1
U-07	Focal interstitial aggregates of lymphoid cells	0 mg/kg/day	0	0	1	3	1	5
		30 mg/kg/day	0	0	2	3	3	8
		10 mg/kg/day	0	1	5	4	0	10[a]
		3 mg/kg/day	0	0	4	2	1	7
U-08	Mineralized deposits in renal parenchyma	0 mg/kg/day	0	0	4	7	10	21
		30 mg/kg/day	0	0	2	1	3	6
		10 mg/kg/day	0	0	0	6	2	8
		3 mg/kg/day	0	0	2	3	7	12
09	Mineralized deposits in renal pelvis	0 mg/kg/day	0	2	4	16	22	44
		30 mg/kg/day	0	1	5	11	17	34[a]
		10 mg/kg/day	0	1	7	11	7	26
		3 mg/kg/day	0	1	4	9	7	21
U-11	Cyst formation in kidney not due to chronic nephropathy	0 mg/kg/day	0	0	1	1	3	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	1	1	3
U-12	Inflammation of renal pelvis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
U-13	Progressive chronic nephropathy - very slight	0 mg/kg/day	0	0	6	13	16	35
		30 mg/kg/day	0	0	2	6	10	18
		10 mg/kg/day	0	0	2	8	7	17
		3 mg/kg/day	0	0	3	6	9	18
U-14	Progressive chronic nephropathy - slight	0 mg/kg/day	0	0	2	4	10	16
		30 mg/kg/day	0	0	0	0	4	4
		10 mg/kg/day	0	0	0	2	1	3[a]
		3 mg/kg/day	0	0	0	1	4	5
U-15	Progressive chronic nephropathy - moderate	0 mg/kg/day	0	0	2	3	6	11
		30 mg/kg/day	0	0	0	0	0	0[a]
		10 mg/kg/day	0	0	0	3	1	4
		3 mg/kg/day	0	0	0	2	2	4
U-16	Progressive chronic nephropathy - pronounced	0 mg/kg/day	0	0	2	1	0	3
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	4	0	4
		3 mg/kg/day	0	0	1	1	2	4

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Kidney (Cont'd)							
U-17	Congestion of kidney	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	1	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-18	Focal tubular hyperplasia	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
U-19	Dilated renal pelvis	0 mg/kg/day	0	0	1	2	0	3
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	1	2	1	4
		3 mg/kg/day	0	0	1	0	0	1
U-20	Focal fibrotic area in cortex or pelvis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-22	Increased hematogenous pigment associated with chronic renal disease	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-23	Nodular proliferation of renal pelvis epithelium	0 mg/kg/day	0	0	1	10	14	25
		30 mg/kg/day	0	0	5	14	18	37[a]
		10 mg/kg/day	0	0	6	9	7	22
		3 mg/kg/day	0	0	4	8	7	19
U-24	Focal hyperplasia of renal pelvis epithelium	0 mg/kg/day	0	0	2	5	4	11
		30 mg/kg/day	0	0	0	1	0	1[a]
		10 mg/kg/day	0	0	3	1	0	4
		3 mg/kg/day	0	0	0	1	2	3
U-25	Multifocal suppurative nephritis, hyperplasia of renal pelvis epithelium, with inflammation and a fibrotic area in renal pelvis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
U-26	Focal purulent nephritis not associated with nephropathy	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-27	Eosinophilic material within epithelium of renal tubules	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-29	Anomalous focus of columnar mucosal epithelium in cortex of kidney	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Kidney (Cont'd)							
U-30	Diffuse suppurative nephritis with necrosis and congestion in one kidney	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-31	Increased vacuolization of tubular epithelium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	0	1
		3 mg/kg/day	0	0	0	1	0	1
U-32 and U-34	Epithelial hyperplasia of renal pelvis secondary to urolithiasis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	1	1
U-33	Perirenal inflammation	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
35	Area of lipomatosis, fibrosis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-36	Increased pigment within renal tubular cytoplasm	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	10	11[a]
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-37	Renal hemangioma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Urogenital Tract							
	Urinary Bladder							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	32	81
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	10	24	10	47
		3 mg/kg/day	1	1	10	18	17	47
U-70	Diffuse urocysticis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
U-71	Focal urocysticis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1

Data listed as number of rats for which the listed observation was present.

[a] Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Urinary Bladder (Cont'd)							
U-72	Focal hyperplasia of urinary bladder mucosa	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
U-73	Diffuse hyperplasia of urinary bladder mucosa	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	2	2
U-82	Diffuse epithelial hyperplasia of urinary bladder secondary to urolithiasis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
	Ovary							
	Number of tissues examined	0 mg/kg/day	0	2	13	29	33	77
		30 mg/kg/day	0	2	9	17	20	48
		10 mg/kg/day	1	2	8	20	11	42
		3 mg/kg/day	1	1	8	17	15	42
X-50	Fluid-filled ovarian cyst	0 mg/kg/day	0	0	3	4	14	21
		30 mg/kg/day	0	0	0	5	9	14
		10 mg/kg/day	0	0	2	5	2	9
		3 mg/kg/day	0	0	1	1	5	7
X-51	Atrophy of ovary	0 mg/kg/day	0	0	0	6	3	9
		30 mg/kg/day	0	0	0	6	1	7
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	0	3	0	3
X-52	Focal inflammation of ovary	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-53	Increased hematogenous pigment in ovary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
X-54	Focal fibrosis in ovary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-55	Hematocyst in ovary	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
X-56	Fibrosis of oviducts	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Ovary (Cont'd)							
X-57	Cystic dilatation of oviduct	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-58	Diffuse purulent and granulomatous inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
X-59	Inflammatory adhesions	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-60	Hyperplasia of interstitial cells	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
67	Granulosal cell neoplasm of ovary	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
	Uterus							
	Number of uterine tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	24	11	49
		3 mg/kg/day	1	1	11	18	19	50
X-70	Area(s) of cystic endometrial hyperplasia	0 mg/kg/day	0	0	7	9	16	32
		30 mg/kg/day	0	0	5	5	10	20
		10 mg/kg/day	0	0	3	2	5	10
		3 mg/kg/day	0	0	3	5	5	13
X-72	Squamous metaplasia with or without keratinization of endometrium	0 mg/kg/day	0	0	4	2	2	8
		30 mg/kg/day	0	0	1	1	3	5
		10 mg/kg/day	0	1	3	1	2	7
		3 mg/kg/day	0	0	1	2	1	4
X-74	Uterine inflammation	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	0	0	0
X-75	Hydrometra of uterus	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-77	Stromal polyp formation in uterus[a]	0 mg/kg/day	0	0	1/1	3/3	10/10	14/14
		30 mg/kg/day	0	0	1/1	3/3	3/7	10/11
		10 mg/kg/day	0	1/1	2/1	5/4	3/3	11/9
		3 mg/kg/day	0	0	0	1/2	2/2	4/4

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Uterus (Cont'd)							
	Number of vaginal tissues examined	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-81	Malignant schwannoma of the vagina	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-85	Malignant schwannoma of uterus with metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-87	Adenocarcinoma of uterus without metastasis	0 mg/kg/day	0	0	0	1	3	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
X-93	Area(s) of endometrial hyperplasia	0 mg/kg/day	0	0	3	3	8	14
		30 mg/kg/day	0	0	1	2	1	4
		10 mg/kg/day	0	1	1	5	1	8
		3 mg/kg/day	0	0	0	3	2	5
X-95	Large cyst formation in uterus	0 mg/kg/day	0	0	0	2	4	6
		30 mg/kg/day	0	0	0	2	2	4
		10 mg/kg/day	0	0	0	1	3	4
		3 mg/kg/day	0	0	0	0	1	1
X-96	Myxosarcoma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
X-97	Schwannoma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	1	0	0	1
X-98	Leiomyosarcoma of uterus with metastasis to lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
X-99	Fibroma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
X-100	Fibroma of vagina	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-101	Sclerosis of uterine vessels	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	MUSCULOSKELETAL SYSTEM							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	1	9	15	21	46
		10 mg/kg/day	1	2	11	24	10	48
		3 mg/kg/day	1	0	11	18	18	48
M-01	Focal degeneration and inflammation of skeletal muscle	0 mg/kg/day	0	0	3	4	16	23
		30 mg/kg/day	0	0	1	1	5	7
		10 mg/kg/day	0	0	2	9	2	13
		3 mg/kg/day	0	0	0	3	6	9
M-04	Multifocal degeneration, inflammation, with or without atrophy of skeletal muscle	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	1	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
M-05	Rhabdomyosarcoma surrounding vertebral column with metastasis to lungs and subcutaneous tissues	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	GASTROINTESTINAL SYSTEM							
	Oral Cavity							
	Number of gingival tissues examined	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-07	Fibroma of gingiva	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Tongue							
	Number of tissues examined	0 mg/kg/day	0	1	17	30	35	83
		30 mg/kg/day	0	1	9	18	21	49
		10 mg/kg/day	0	1	11	25	11	48
		3 mg/kg/day	0	0	11	18	19	48
G-10	Vasculitis and vascular degeneration	0 mg/kg/day	0	0	2	1	5	8
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
G-11	Inflammation and/or hyperplasia of submucosal glands	0 mg/kg/day	0	0	12	23	21	56
		30 mg/kg/day	0	0	7	13	10	30
		10 mg/kg/day	0	0	4	7	7	18
		3 mg/kg/day	0	0	4	6	10	20
G-13	Degeneration and inflammation of muscles	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	13	19	50
	Tongue (Cont'd)							
G-15	Squamous metaplasia of tongue	0 mg/kg/day	0	0	5	4	5	14
		30 mg/kg/day	0	0	2	5	2	9
		10 mg/kg/day	0	0	2	3	1	6
		3 mg/kg/day	0	0	1	0	2	3
G-20	Stratified squamous cell carcinoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-18	Adenoma of glandular components of tongue	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		0 mg/kg/day	0	0	0	0	0	0
	Stomach							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	17	19	49
G-46	Dilatation of gastric pits	0 mg/kg/day	0	0	5	3	9	17
		30 mg/kg/day	0	0	2	5	9	16
		10 mg/kg/day	0	0	3	4	1	8
		3 mg/kg/day	0	0	1	3	5	9
G-47	Mineralization of gastric muscularis and/or gastric mucosa	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2
G-48	Submucosal edema of stomach	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-49	Hyperplasia of nonglandular mucosal epithelium	0 mg/kg/day	0	1	0	1	1	3
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	0	0
G-50	Focal area of inflammation of stomach wall	0 mg/kg/day	0	1	0	2	2	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-51	Hyperkeratosis of nonglandular mucosa of stomach	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-52	Focal erosion of gastric mucosa	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Stomach (Cont'd)							
G-55	Keratinized cyst formation - wall of stomach	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-57	Squamous polyp of stomach mucosa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-59	Stratified squamous papilloma of gastric mucosa	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Small Intestine							
	Number of tissues examined	0 mg/kg/day	0	2	17	29	36	84
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
G-71	Focal ulceration and/or inflammation of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	1	0	2
		3 mg/kg/day	1	0	0	0	0	1
G-74	Reactive lymphoid hyperplasia of small intestine	0 mg/kg/day	0	0	0	0	11	11
		30 mg/kg/day	0	0	0	2	6	8
		10 mg/kg/day	0	0	0	2	2	4
		3 mg/kg/day	0	0	0	3	4	7
G-76	Mucocystadenocarcinoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-77	Adenocarcinoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
G-79	Lymphosarcoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-82	Diffuse inflammation of small intestine	0 mg/kg/day	0	0	0	0	2	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
G-83	Persisting Meckel's diverticulum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Small Intestine (Cont'd)							
G-84	Leiomyoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-85	Leiomyosarcoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-86	Focal mucosal hyperplasia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Large Intestine							
	Number of tissues examined	0 mg/kg/day	0	2	16	31	36	85
		30 mg/kg/day	0	2	9	16	21	48
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
G-90	Intestinal nematodiasis	0 mg/kg/day	0	0	4	4	4	12
		30 mg/kg/day	0	0	3	0	1	4
		10 mg/kg/day	0	0	2	1	1	4
		3 mg/kg/day	0	0	2	1	0	3
G-92	Focal inflammation of large intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
G-93	Edema and/or inflammation of wall of large intestine and/or cecum	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-95	Reactive lymphoid hyperplasia of large intestine	0 mg/kg/day	0	0	0	1	3	4
		30 mg/kg/day	0	0	1	1	3	5
		10 mg/kg/day	0	0	1	0	2	3
		3 mg/kg/day	0	0	3	0	2	5
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND							
	Subcutaneous (nonumorous)							
	Number of integumentary tissues examined	0 mg/kg/day	0	1	8	27	36	72
		30 mg/kg/day	0	0	6	16	21	43
		10 mg/kg/day	0	0	7	25	11	43
		3 mg/kg/day	0	0	10	18	19	47
S-01	Subcutaneous abscess	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Subcutaneous (nontumorous) (Cont'd)							
S-04	Subcutaneous trauma secondary to subcutaneous mass	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
S-05	Subcutaneous purulent inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Subcutaneous (tumorous)[a]							
	Total number of subcutaneous tumors/number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	1/1	32/14	69/30	79/31	181/76
		30 mg/kg/day	0/0	2/2	15/7	30/16	41/19	88/44
		10 mg/kg/day	0/0	1/1	12/9	50/23	22/10	85/43
		3 mg/kg/day	0/0	0/0	12/8	31/15	33/17	76/40
S-11	Subcutaneous lipoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
S-12	Subcutaneous fibroma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-14	Fibrosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-16	Subcutaneous carcinosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-21	Subcutaneous myxosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
	Mammary Gland (nontumorous)							
	Number of tissues examined	0 mg/kg/day	0	0	7	16	26	49
		30 mg/kg/day	0	0	1	3	18	22
		10 mg/kg/day	0	0	4	20	8	32
		3 mg/kg/day	0	0	9	10	11	30
S-25	Mammary gland hyperplasia	0 mg/kg/day	0	0	5	16	20	41
		30 mg/kg/day	0	0	1	3	17	21
		10 mg/kg/day	0	0	2	18	7	27
		3 mg/kg/day	0	0	3	6	7	16[b]

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

[b]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Mammary Gland (nontumorous) (Cont'd)							
S-26	Mammary gland galactocele formation	0 mg/kg/day	0	0	5	9	16	30
		30 mg/kg/day	0	0	1	2	6	9
		10 mg/kg/day	0	0	0	14	5	19
		3 mg/kg/day	0	0	2	6	6	14
S-27	Mammary gland granulomatous reaction	0 mg/kg/day	0	0	0	3	5	8
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0[a]
		3 mg/kg/day	0	0	0	0	1	1
	Mammary (tumorous)[a]							
	Total number of subcutaneous tumors /number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	1/1	32/14	69/30	79/31	181/76
		30 mg/kg/day	0/0	2/2	15/7	30/16	41/19	88/44
		10 mg/kg/day	0/0	1/1	12/9	50/23	22/10	85/43
		3 mg/kg/day	0/0	0/0	12/8	31/15	33/17	76/40
S-30	Mammary gland fibroadenoma/ adenofibroma	0 mg/kg/day	0/0	0/0	25/13	51/25	62/27	138/65
		30 mg/kg/day	0/0	1/1	11/7	15/10	28/15	55/33
		10 mg/kg/day	0/0	1/1	12/9	35/18	14/10	62/38
		3 mg/kg/day	0/0	0/0	9/7	14/9	24/15	47/31
S-31	Mammary gland adenoma	0 mg/kg/day	0/0	0/0	3/2	10/6	2/2	15/10
		30 mg/kg/day	0/0	0/0	1/1	3/2	5/5	9/8
		10 mg/kg/day	0/0	0/0	0/0	3/2	1/1	4/3
		3 mg/kg/day	0/0	0/0	1/1	4/4	2/1	7/6
S-32	Mammary gland adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	3/2	1/1	3/3	7/6
		30 mg/kg/day	0/0	0/0	1/1	2/2	3/3	6/6
		10 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		3 mg/kg/day	0/0	0/0	0/0	2/2	2/2	4/4
S-33	Mammary gland adenocarcinoma with metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	1/1	1/1	2/2	0/0	4/4
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	1/1	1/1	0/0	2/2
S-34	Mammary gland cystadenoma	0 mg/kg/day	0/0	0/0	0/0	2/2	0/0	2/2
		30 mg/kg/day	0/0	0/0	0/0	0/0	3/3	3/3
		10 mg/kg/day	0/0	0/0	0/0	2/2	4/2	6/4
		3 mg/kg/day	0/0	0/0	1/1	1/1	0/0	2/2
S-36	Mammary gland fibroma	0 mg/kg/day	0/0	0/0	1/1	0/0	3/3	4/4
		30 mg/kg/day	0/0	0/0	0/0	3/2	0/0	3/2
		10 mg/kg/day	0/0	0/0	0/0	3/3	0/0	3/3
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-37	Mammary gland cystfibroadenoma/ cystadenofibroma	0 mg/kg/day	0/0	0/0	0/0	4/4	9/7	13/11
		30 mg/kg/day	0/0	0/0	0/0	4/4	1/1	5/5
		10 mg/kg/day	0/0	0/0	0/0	7/6	2/1	9/7
		3 mg/kg/day	0/0	0/0	0/0	6/4	4/3	10/7
S-41	Mammary gland unclassified malignant neoplasm (carcinoma with accompanying fibrous proliferation)	0 mg/kg/day	0/0	1/1	0/0	0/0	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Mammary (tumorous)[a] (Cont'd)							
S-42	Mammary gland cystadenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Integument (nontumorous)							
	Number of tissues examined	0 mg/kg/day	0	1	8	27	36	72
		30 mg/kg/day	0	0	6	16	21	43
		10 mg/kg/day	0	0	7	25	11	43
		3 mg/kg/day	0	0	10	18	19	47
S-60	Epidermal inclusion cyst with or without inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-62	Abscess of clitoral gland	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-65	Focal epidermal thickening and inflammation of dermis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-66	Focal inflammation of dermis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
S-67	Chronic inflammation secondary to tumor	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Integument (tumorous)[a]							
	Total number of cutaneous tumors/number of rats bearing cutaneous tumors	0 mg/kg/day	0/0	0/0	0/0	0/0	2/2	2/2
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-72	Squamous papilloma of integument	0 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-73B	Keratoacanthoma	0 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Ear Canal[a]							
	Total number of ear canal tumors/ number of rats bearing ear canal tumors	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-76	Zymbal gland carcinoma of ear canal	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-78	Adenoma (sebaceous) of Zymbal gland	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
	NERVOUS SYSTEM							
	Brain							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
N-01	Focal malacia in brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
N-02	Focal gliosis of brain	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-03	Focal inflammation in brain	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-04	Vacuolization of white matter of brain which may or may not be artifact	0 mg/kg/day	0	0	3	5	12	20
		30 mg/kg/day	0	0	1	2	6	9
		10 mg/kg/day	0	1	1	10	3	15
		3 mg/kg/day	1	0	4	6	6	17
N-06	Compression malacia and/or hemorrhage, and/or hematogenous pigment in brain adjacent to pituitary adenocarcinoma	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	3	0	3
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-13	Neovascularization secondary to cranial neoplasm	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-14	Focal aggregate of mononuclear cells	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Spinal Cord							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	0	11	18	19	49
N-40	Vacuolization of spinal cord which may or may not be artifact	0 mg/kg/day	0	0	0	6	17	13
		30 mg/kg/day	0	0	0	4	6	10
		10 mg/kg/day	0	2	4	9	4	19[a]
		3 mg/kg/day	1	0	7	7	8	23[a]
	Peripheral Nerves							
	Number of tissues examined	0 mg/kg/day	0	2	16	30	35	83
		30 mg/kg/day	0	1	8	17	21	47
		10 mg/kg/day	1	2	10	23	10	46
		3 mg/kg/day	1	0	11	15	18	45
N-60	Vacuolization of peripheral nerves	0 mg/kg/day	0	0	5	9	24	38
		30 mg/kg/day	0	1	3	5	16	24
		10 mg/kg/day	1	2	2	6	5	16
		3 mg/kg/day	0	0	5	6	10	21
N-66	Focal inflammation of peripheral nerve	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Pituitary							
	Number of tissues examined	0 mg/kg/day	0	1	16	29	36	82
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	1	9	24	9	44
		3 mg/kg/day	0	0	10	17	18	45
N-80	Retention cyst(s) in pituitary	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-81	Pituitary hematocyst	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
N-83	Hemangiectasis of pituitary	0 mg/kg/day	0	0	1	6	12	19
		30 mg/kg/day	0	0	1	4	7	12
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	1	5	6	12
N-85	Hyperplastic focus in pituitary	0 mg/kg/day	0	0	0	7	8	15
		30 mg/kg/day	0	0	3	2	3	8
		10 mg/kg/day	0	0	0	0	0	0[a]
		3 mg/kg/day	0	0	1	1	0	3

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Pituitary (Cont'd)							
N-86	Pituitary adenoma formation(s)	0 mg/kg/day	0	0	8	10	24	42
		30 mg/kg/day	0	0	3	8	8	19
		10 mg/kg/day	0	0	1	13	6	20
		3 mg/kg/day	0	0	3	9	11	23
N-87	Pituitary adenocarcinoma without extension or invasion of brain	0 mg/kg/day	0	0	2	1	3	6
		30 mg/kg/day	0	0	0	2	3	5
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	4	2	6
N-88	Pituitary adenocarcinoma with invasion of brain	0 mg/kg/day	0	0	1	3	1	5
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	2	2
N-90	Pituitary adenoma arising from pars intermedia	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-91	Hyperplasia of pituitary	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	1	0	1	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Cranial Cavity							
N-70	Neurofibrosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	ADRENAL GLAND							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	General							
A-01	Adrenal gland hematocyst formation	0 mg/kg/day	0	1	6	20	20	47
		30 mg/kg/day	0	1	3	8	12	24
		10 mg/kg/day	0	0	2	19	7	28
		3 mg/kg/day	0	0	1	9	12	22
A-02	Hemangiectasis of adrenal gland	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-03	Isolated focus of purulent inflammation in adrenal glands	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-05	Congestion	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	2	0	0	2

Data listed as number of rats for which the listed observation was present.

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Cortex							
A-20	Adrenal cortical necrosis with or without hemorrhage and/or purulent inflammation	0 mg/kg/day	0	0	0	1	1	2
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	1	0	1	2
A-21	Vacuolization of adrenal cortical cells	0 mg/kg/day	0	1	11	23	26	61
		30 mg/kg/day	0	0	5	14	16	35
		10 mg/kg/day	0	0	5	14	8	27
		3 mg/kg/day	0	0	4	13	14	31
A-22	Mineralization of adrenal cortex	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	0	1	1
A-23	Atrophy of adrenal cortex	0 mg/kg/day	0	0	1	1	1	3
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-24	Focal areas of extramedullary hematopoiesis in adrenal glands	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	1	0	0	1
A-25	Focal fibrotic reaction within adrenal cortex	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-30	Hyperplastic focus(foci), nodule(s) of adrenal cortex	0 mg/kg/day	0	0	1	11	15	31
		30 mg/kg/day	0	0	3	6	10	19
		10 mg/kg/day	0	0	3	9	3	15
		3 mg/kg/day	0	0	4	6	11	21
A-31	Adenoma of adrenal cortex	0 mg/kg/day	0	0	2	0	4	6
		30 mg/kg/day	0	0	2	1	0	3
		10 mg/kg/day	0	0	0	2	2	4
		3 mg/kg/day	0	0	0	0	2	2
	Medulla							
A-40	Hyperplastic nodule of adrenal medulla(s)	0 mg/kg/day	0	0	2	2	2	6
		30 mg/kg/day	0	0	1	2	4	7
		10 mg/kg/day	0	0	1	3	0	4
		3 mg/kg/day	0	0	0	3	5	8
A-41	Adrenal pheochromocytoma, unilateral	0 mg/kg/day	0	0	1	1	4	6
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	0	1	2	3
A-44	Adrenal pheochromocytoma, malignant, no metastasis - unilateral	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	EYE							
	Number of tissues examined	0 mg/kg/day	0	1	17	31	36	85
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	10	25	11	49
		3 mg/kg/day	1	1	11	18	19	50
E-01	Focal keraticis of eye	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	2	2
		3 mg/kg/day	0	0	0	0	1	1
E-02	Diffuse keraciti of eye	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-03	Focal or diffuse hyperplasia of corneal epithelium	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-07	Focal mineralization of eye	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
E-10	Phthisis bulbi with chronic inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
	MISCELLANEOUS							
Z-02	Postmortem autolytic changes in all organs	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	1	0	0	0	1
Z-03	Inflammation of mesenteric fat	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-04	Focal mesenteric steatitis and/or necrosis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	2	1	4
Z-07	Mediastinal fat inflammation	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	2	0	3[a]
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	MISCELLANEOUS (Cont'd)							
Z-10	Multifocal mesenteric granulomatous inflammation with inflammatory adhesions within the abdominal cavity	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-11	Mesenteric lipoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-14	Hemorrhage into mesenteric tissue	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-15	Abdominal hemangiosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-16	Granulocytic leukemia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-17	Generalized leukocytic response due to purulent inflammation of subcutaneous mass	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
Z-18	Generalized monocytic leukemia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	LYMPHORETICULAR							
	Lymph Nodes - General							
L-01	Generalized lymphosarcoma with widespread metastasis	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	1	0	0	0	0	1
L-02	Intraabdominal lymphosarcoma or carcinosarcoma of unspecified origin with widespread metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thoracic Lymph Node(s)							
	Number of tissues examined	0 mg/kg/day	0	1	14	26	33	74
		30 mg/kg/day	0	2	8	13	19	42
		10 mg/kg/day	1	2	9	21	11	44
		3 mg/kg/day	1	1	10	17	17	46

ata listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Thoracic Lymph Node(s) (Cont'd)							
L-20	Pooling of edema, pigment, and/or red blood cells in sinusoids of thoracic lymph node	0 mg/kg/day	0	0	8	16	9	33
		30 mg/kg/day	0	0	3	7	5	15
		10 mg/kg/day	1	1	4	8	5	19
		3 mg/kg/day	0	0	6	7	9	22
L-22	Reactive lymphoid hyperplasia of thoracic lymph node(s)	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	2	0	1	0	3
		3 mg/kg/day	0	0	0	0	0	0
L-23	Congestion of thoracic lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	Mesenteric Lymph Node(s)							
	Number of tissues examined	0 mg/kg/day	0	2	16	30	36	84
		30 mg/kg/day	0	1	9	17	21	48
		10 mg/kg/day	1	1	9	25	11	47
		3 mg/kg/day	0	0	8	16	15	39
L-30	Pooling of edema, pigment, and/or red blood cells in sinusoids of mesenteric lymph node	0 mg/kg/day	0	0	10	17	16	43
		30 mg/kg/day	0	0	6	13	10	29
		10 mg/kg/day	0	0	2	12	2	16
		3 mg/kg/day	0	0	2	9	6	17
L-31	Inflammatory process in mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
L-32	Reactive lymphoid hyperplasia of mesenteric lymph node	0 mg/kg/day	0	1	2	4	4	11
		30 mg/kg/day	0	0	1	4	2	7
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	2	3	1	6
L-33	Cystic distention of lymphatics of mesenteric lymph node	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-35	Increased number's of histiocytes in mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-37	Lymphangioma of mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Subcutaneous Lymph Nodes							
	Number of tissues examined	0 mg/kg/day	0	0	2	3	6	11
		30 mg/kg/day	0	0	1	2	4	7
		10 mg/kg/day	0	0	2	9	1	12
		3 mg/kg/day	0	0	5	6	3	14
L-40	Inflammation of subcutaneous lymph node(s)	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-41	Pooling of red blood cells and serum within subcutaneous lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-42	Reactive hyperplasia of subcutaneous lymph node	0 mg/kg/day	0	0	2	1	2	5
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-45	Cystic dilatation of lymphatics	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
L-46	Fibrotic tissue in subcutaneous lymph node	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thymus							
	Number of tissues examined	0 mg/kg/day	0	1	9	24	36	70
		30 mg/kg/day	0	2	6	15	20	43
		10 mg/kg/day	1	2	8	22	10	43
		3 mg/kg/day	0	1	8	16	18	43
L-50	Lymphoid hyperplasia of thymus	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-56	Thymic lymphosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-57	Congestion of thymus	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-58	Fibrotic area in thymus	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	SPLEEN							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
L-60	Splenic atrophy with or without increased hemosiderin pigment	0 mg/kg/day	0	1	0	2	0	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	1	0	0	2	0	3
		3 mg/kg/day	0	1	0	3	1	5
L-61	Increased hematogenous pigment in spleen	0 mg/kg/day	0	1	9	15	16	41
		30 mg/kg/day	0	1	6	9	14	30
		10 mg/kg/day	0	2	6	8	7	23
		3 mg/kg/day	0	0	8	4	9	21
L-62	Increased extramedullary hematopoiesis in spleen	0 mg/kg/day	0	1	14	22	23	60
		30 mg/kg/day	0	2	7	12	15	36
		10 mg/kg/day	0	2	10	18	7	37
		3 mg/kg/day	0	0	9	12	11	32
L-63	Reticuloendothelial or lymphoid hyperplasia of spleen	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-65	Focal area of necrosis and hemorrhage in spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
L-66	Focal acellular deposits	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
L-67	Generalized myeloid leukemia apparently originating in spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	THYROID AND PARATHYROID							
	Thyroid							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	18	20	49
		10 mg/kg/day	1	2	11	23	11	48
		3 mg/kg/day	1	1	11	18	19	50
T-01	Thyroid interfollicular C-cell adenoma	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	1	2	3	6
		10 mg/kg/day	0	0	0	0	2	2
		3 mg/kg/day	0	0	1	2	7	10[a]

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 35 (Continued)

MICROSCOPIC OBSERVATIONS ON FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	36
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	Thyroid (Cont'd)							
T-02	Thyroid interfollicular C-cell adenocarcinoma without metastasis	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
T-03	Thyroid interfollicular C-cell adenocarcinoma with metastasis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
T-04	Hyperplastic focus of interfollicular C cells within thyroid	0 mg/kg/day	0	0	2	9	13	24
		30 mg/kg/day	0	0	1	4	8	13
		10 mg/kg/day	0	0	1	8	4	13
		3 mg/kg/day	0	0	1	3	4	8
T-06	Retention cyst in thyroid	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	3	0	3
T-09	Focal or diffuse inflammation - thyroiditis	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
	Parathyroid							
	Number of tissues examined	0 mg/kg/day	0	2	17	29	27	75
		30 mg/kg/day	0	2	9	17	13	41
		10 mg/kg/day	1	2	10	12	5	30
		3 mg/kg/day	0	1	9	10	14	34
T-20	Secondary parathyroid hyperplasia (renal)	0 mg/kg/day	0	0	2	2	0	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	2	1	2	5
T-21	Parathyroid adenoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
T-23	Interstitial fibrosis of parathyroid	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 36

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LIVER							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Hepatocellular Changes							
H-16	Hepatocellular carcinoma(s)	0 mg/kg/day	0	0	0	3	0	3
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	RESPIRATORY SYSTEM							
	Nasal Turbinates/Hard Palate							
	Number of tissues examined	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	2	0	2
R-04	Keratoacanthoma of nasal turbinates /hard palate	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
R-05	Squamous cell carcinoma of hard palate	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
R-07	Mixed adenoma of hard palate	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Lungs							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
R-70	Pulmonary adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
	PANCREAS							
	Acinar							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50

Data listed as number of rats for which the listed observation was present.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	PANCREAS (Cont'd)							
	Acinar (Cont'd)							
P-10	Pancreatic acinar adenoma	0 mg/kg/day	0	1	2	19	4	26
		30 mg/kg/day	0	0	3	9	8	20
		10 mg/kg/day	0	0	2	4	3	9
		3 mg/kg/day	0	0	2	12	3	17
P-11	Pancreatic acinar adenocarcinoma	0 mg/kg/day	0	0	0	2	0	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Islets of Langerhans							
P-25	Pancreatic islet cell adenoma	0 mg/kg/day	0	1	0	10	2	13
		30 mg/kg/day	0	0	0	2	5	7
		10 mg/kg/day	0	0	1	5	1	7
		3 mg/kg/day	0	0	2	5	3	10
P-26	Pancreatic islet cell adenocarcinoma	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	URINARY SYSTEM							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	Kidney							
U-51	Renal pelvis transitional cell adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
U-53	Renal tubular adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-55	Papillary adenoma of renal pelvis (unilateral)	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
U-58	Renal carcinoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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X-20	Interstitial cell adenoma of testis	0 mg/kg/day	0	0	0	3	1	4
		30 mg/kg/day	0	0	1	0	2	3
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	1	1
X-21	Mesothelioma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-22	Sertoli cell tumor	0 mg/kg/day	0	0	1	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	MUSCULOSKELETAL SYSTEM							
	Number of tissues examined	0 mg/kg/day	1	1	14	62	6	84
		30 mg/kg/day	0	0	8	25	15	48
		10 mg/kg/day	0	1	20	22	4	47
		3 mg/kg/day	0	1	11	25	9	46
M-06	Chondrosarcoma of rib	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	GASTROINTESTINAL SYSTEM							
	Oral Cavity							
	Number of gingival tissues examined	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-06	Oral papilloma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	URINARY SYSTEM (Cont'd)							
	Urogenital Tract							
	Number of urinary bladders, examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	8	25	16	49
		10 mg/kg/day	0	1	20	22	4	47
		0 mg/kg/day	0	1	12	28	9	50
U-78	Transitional cell carcinoma of urinary bladder with metastasis to kidney	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		0 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
	Testes							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
	Tongue							
	Number of tissues examined	0 mg/kg/day	0	1	13	63	6	83
		30 mg/kg/day	0	0	8	25	16	49
		10 mg/kg/day	0	0	19	23	4	46
		3 mg/kg/day	0	1	12	28	9	50
G-20	Stratified squamous cell carcinoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	2	1	4
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Salivary Glands							
	Number of tissues examined	0 mg/kg/day	1	2	12	59	6	80
		30 mg/kg/day	0	0	7	23	15	45
		10 mg/kg/day	0	1	17	23	4	45
		3 mg/kg/day	0	1	10	25	8	44
G-38	Adenoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Stomach							
	Number of tissues examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	22	4	49
		3 mg/kg/day	0	1	12	27	9	49
G-59	Stratified squamous papilloma of gastric mucosa	0 mg/kg/day	0	0	0	2	1	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Small Intestine							
	Number of tissues examined	0 mg/kg/day	1	2	14	62	6	85
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	25	9	47
G-76	Mucocystadenocarcinoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-77	Adenocarcinoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	1	2

Data listed as number of rats for which the listed observation was present.

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G-89A	Mucocystadenoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89C	Papillary adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Large Intestine							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	21	23	4	49
		3 mg/kg/day	0	1	12	27	8	48
G-100	Lymphosarcoma of large intestine with metastasis to mesenteric lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND							
	Subcutaneous (tumorous)[a]							
	Total number of subcutaneous tumors/number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	0/0	1/1	8/8	0/0	9/9
		30 mg/kg/day	0/0	0/0	1/1	3/3	3/3	7/7
		10 mg/kg/day	0/0	1/1	1/1	1/1	0/0	3/3
		3 mg/kg/day	0/0	0/0	1/1	6/6	5/5	12/12

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
	Small Intestine (Cont'd)							
G-78	Fibrosarcoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-79	Lymphosarcoma of small intestine	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-88	Undifferentiated sarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-89	Lymphosarcoma of small intestine with metastasis to mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

S-23	Subcutaneous liposarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-24	Subcutaneous myxoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-24A	Subcutaneous carcinoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Mammary (tumorous)[a]							
	Total number of subcutaneous tumors /number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	0/0	1/1	8/8	0/0	9/9
		30 mg/kg/day	0/0	0/0	1/1	3/3	3/3	7/7
		10 mg/kg/day	0/0	1/1	1/1	1/1	0/0	3/3
		3 mg/kg/day	0/0	0/0	1/1	6/6	5/3	12/10
S-30	Mammary gland fibroadenoma/ adenofibroma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	2/2	2/2
S-32	Mammary gland adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-33	Mammary gland adenocarcinoma with metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND (Cont'd)							
	Subcutaneous (tumorous)[a] (Cont'd)							
S-12	Subcutaneous fibroma	0 mg/kg/day	0/0	0/0	1/1	3/3	0/0	4/4
		30 mg/kg/day	0/0	0/0	0/0	1/1	2/2	3/3
		10 mg/kg/day	0/0	1/1	0/0	1/1	0/0	2/2
		3 mg/kg/day	0/0	0/0	1/1	4/4	3/1	8/6
S-17	Subcutaneous adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-20	Malignant fibrous histiocytoma with metastasis to abdominal cavity	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-22	Malignant fibrous histiocytoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

S-76A	Fibrosarcoma adjacent to ear tag	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76C	Adnexal cystadenoma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Ear Canal[a]							
	Total number of ear canal tumors/ number of rats bearing ear canal tumors	0 mg/kg/day	0/0	0/0	1/1	4/4	0/0	5/5
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76	Zymbal gland carcinoma of ear canal	0 mg/kg/day	0/0	0/0	1/1	3/3	0/0	4/4
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-79	Subauricular undifferentiated sarcoma with metastasis to lungs	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND (Cont'd)							
	Mammary (tumorous)[a] (Cont'd)							
S-35	Mammary gland carcinosarcoma	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Integument (tumorous)[a]							
	Total number of cutaneous tumors/ number of rats bearing cutaneous tumors	0 mg/kg/day	0/0	0/0	0/0	2/2	0/0	2/2
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	2/2	0/0	1/1	3/3
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-72	Squamous papilloma of integument	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-75A	Adnexal carcinoma with metastasis to lungs	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	NERVOUS SYSTEM							
	Brain							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
N-22	Astrocytoma within cerebrum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-23	Astrocytoma within cerebellum	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1
N-25	Granular cell myoblastoma of brain	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	1	0	1
N-19	Astrocytoma or mixed glial neoplasm of cerebellum	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Peripheral Nerves							
	Number of tissues examined	0 mg/kg/day	1	1	13	61	6	82
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	20	23	4	48
		3 mg/kg/day	0	1	11	24	9	45
N-62	Mediastinal malignant schwannoma	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
N-64	Schwannoma of cranial nerve	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
N-68	Malignant schwannoma of cranial nerve with involvement of cerebrum and nasal turbinates	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	Pituitary							
	Number of tissues examined	0 mg/kg/day	1	2	13	59	6	82
		30 mg/kg/day	0	0	9	24	16	49
		10 mg/kg/day	0	1	21	22	4	48
		3 mg/kg/day	0	1	11	26	9	47

ata listed as number of rats for which the listed observation was present.

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	ADRENAL GLAND							
	Number of tissues examined	0 mg/kg/day	1	2	12	63	6	84
		30 mg/kg/day	0	0	9	25	15	50
		10 mg/kg/day	0	0	22	23	4	49
		3 mg/kg/day	0	1	12	28	9	50
	Cortex							
A-31	Adenoma of adrenal cortex	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	3	0	3
A-32	Adenocarcinoma of adrenal cortex	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
	Medulla							
A-41	Adrenal pheochromocytoma, uni-lateral	0 mg/kg/day	0	0	2	29	1	32
		30 mg/kg/day	0	0	1	9	4	14
		10 mg/kg/day	0	0	4	8	3	15
		3 mg/kg/day	0	0	2	5	4	11

Data listed as number of rats for which the listed observation was present.

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	NERVOUS SYSTEM (Cont'd)							
	Pituitary (Cont'd)							
N-86	Pituitary adenoma formation(s)	0 mg/kg/day	0	0	2	17	3	22
		30 mg/kg/day	0	0	0	5	5	10
		10 mg/kg/day	0	0	3	9	1	13
		3 mg/kg/day	0	0	1	4	5	10
N-87	Pituitary adenocarcinoma without extension or invasion of brain	0 mg/kg/day	0	0	0	4	0	4
		30 mg/kg/day	0	0	0	2	1	3
		10 mg/kg/day	0	0	1	1	0	2
		3 mg/kg/day	0	0	0	2	0	2
N-88	Pituitary adenocarcinoma with invasion of brain	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Cranial Cavity							
N-71	Neurofibroma adjacent to cranial nerve	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	36
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	ADRENAL GLAND (Cont'd)							
	Medulla (Cont'd)							
A-42	Adrenal pheochromocytoma, bilateral	0 mg/kg/day	0	0	1	2	1	4
		30 mg/kg/day	0	0	0	4	2	6
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	1	0	1
A-44	Adrenal pheochromocytoma, malignant, no metastasis - unilateral	0 mg/kg/day	0	0	0	5	1	6
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	2	1	3
A-45	Adrenal pheochromocytoma, malignant, metastasis, unilateral	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-46	Adrenal ganglioneuroma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
A-47	Adrenal pheochromocytoma, malignant - no metastasis - bilateral	0 mg/kg/day	0	1	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	EYE							
	Number of tissues examined	0 mg/kg/day	0	2	13	61	6	82
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	20	23	4	48
		3 mg/kg/day	0	1	11	28	9	49
E-08	Retrobulbar keratinizing squamous cell carcinoma with extension into cranium	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
E-09	Intraorbital undifferentiated sarcoma with invasion of cranial cavity	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	MISCELLANEOUS							
Z-11	Mesenteric lipoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	LYMPHORETICULAR							
	Lymph Nodes - General							
L-01	Generalized lymphosarcoma with widespread metastasis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	1	0	0	1

Data listed as number of rats for which the listed observation was present.

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TABLE 36 (Continued)

TUMOR INCIDENCE IN MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50
	LYMPHORETICULAR (Cont'd)							
	Lymph Nodes - General (Cont'd)							
L-03	Generalized lymphosarcoma (reticular cell type) with widespread metastasis	0 mg/kg/day	1	0	0	0	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thymus							
	Number of tissues examined	0 mg/kg/day	1	2	12	43	1	59
		30 mg/kg/day	0	0	4	22	12	38
		10 mg/kg/day	0	1	14	17	3	35
		3 mg/kg/day	0	1	10	19	7	37
L-36	Thymic lymphosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	SPLEEN							
	Number of tissues examined	0 mg/kg/day	1	2	14	63	6	86
		30 mg/kg/day	0	0	9	25	16	50
		10 mg/kg/day	0	1	22	23	4	50
		3 mg/kg/day	0	1	12	28	9	50

L-70	Lymphosarcoma of spleen	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	THYROID AND PARATHYROID							
	Thyroid							
	Number of tissues examined	0 mg/kg/day	0	2	13	63	6	84
		30 mg/kg/day	0	0	3	24	16	48
		10 mg/kg/day	0	1	22	23	3	49
		3 mg/kg/day	0	1	12	28	8	49
T-01	Thyroid interfollicular C-cell adenoma	0 mg/kg/day	0	0	0	3	3	6
		30 mg/kg/day	0	0	1	2	4	7
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	2	1	3
T-03	Thyroid interfollicular C-cell adenocarcinoma with metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1

Data listed as number of rats for which the listed observation was present.

TABLE 37

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LIVER							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
H-15	Hyperplastic nodule(s)	0 mg/kg/day	0	0	1	0	3	4
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	1	1	1	3
		3 mg/kg/day	0	0	0	0	2	2
H-16	Hepatocellular carcinoma(s)	0 mg/kg/day	0	0	1	0	1	2
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	PANCREAS							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
P-10	Pancreatic acinar adenoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
P-25	Pancreatic islet cell adenoma	0 mg/kg/day	0	0	1	1	6	8
		30 mg/kg/day	0	0	0	1	1	2
		10 mg/kg/day	0	0	0	2	1	3
		3 mg/kg/day	0	0	0	1	0	1
P-26	Pancreatic islet cell adenocarcinoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	URINARY SYSTEM							
	Kidney							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
U-57	Renal hemangioma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	REPRODUCTIVE SYSTEM							
	Ovary							
	Number of tissues examined	0 mg/kg/day	0	2	13	19	33	77
		30 mg/kg/day	0	2	9	17	20	48
		10 mg/kg/day	1	2	8	20	11	42
		3 mg/kg/day	1	1	8	17	15	42

Data listed as number of rats for which the listed observation was present.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	REPRODUCTIVE SYSTEM (Cont'd)							
	Ovary (Cont'd)							
X-67	Granulosal cell neoplasm of ovary	0 mg/kg/day	0	0	0	0	4	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	1	1
	Uterus							
	Number of uterine tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	24	11	49
		3 mg/kg/day	1	1	11	18	19	50
X-77	Stromal polyp formation in uterus[a]	0 mg/kg/day	0	0	1/1	3/3	10/10	14/14
		30 mg/kg/day	0	0	1/1	3/3	8/7	12/11
		10 mg/kg/day	0	1/1	2/1	5/4	3/3	11/9
		3 mg/kg/day	0	0	0	2/2	2/2	4/4
	Number of vaginal tissues examined	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

X-81	Malignant schwannoma of the vagina	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-85	Malignant schwannoma of uterus with metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
X-87	Adenocarcinoma of uterus without metastasis	0 mg/kg/day	0	0	0	1	3	4
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	0	0	0	0
X-96	Myxosarcoma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
X-97	Schwannoma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	1	1
		3 mg/kg/day	0	0	1	0	0	1
X-98	Leiomyosarcoma of uterus with metastasis to lymph node	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type neoplasm.

TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	REPRODUCTIVE SYSTEM (Cont'd)							
	Uterus (Cont'd)							
X-99	Fibroma of uterus	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
X-100	Fibroma of vagina	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	MUSCULOSKELETAL SYSTEM							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	1	9	15	21	46
		10 mg/kg/day	1	2	11	24	10	48
		3 mg/kg/day	1	0	11	18	18	48
M-05	Rhabdomyosarcoma surrounding vertebral column with metastasis to lungs and subcutaneous tissues	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	1	0	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	GASTROINTESTINAL SYSTEM							
	Oral Cavity							
	Number of gingival tissues examined	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-07	Fibroma of gingiva	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Tongue							
	Number of tissues examined	0 mg/kg/day	0	1	17	30	35	83
		30 mg/kg/day	0	1	9	18	21	49
		10 mg/kg/day	0	1	11	25	11	48
		3 mg/kg/day	0	0	11	18	19	48
G-20	Stratified squamous cell carcinoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-18	Adenoma of glandular components of tongue	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated.	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	GASTROINTESTINAL SYSTEM (Cont'd)							
	Stomach							
	Number of tissues examined	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	17	19	49
G-57	Squamous polyp of stomach mucosa	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
G-59	Stratified squamous papilloma of gastric mucosa	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Small Intestine							
	Number of tissues examined	0 mg/kg/day	0	2	17	29	36	84
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
G-76	Mucocystadenocarcinoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-77	Adenocarcinoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	1	1
G-79	Lymphosarcoma of small intestine	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
G-84	Leiomyoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
G-85	Leiomyosarcoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND							
	Subcutaneous (tumorous)[a]							
	Total number of subcutaneous tumors/number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	1/1	32/14	69/30	79/31	181/76
		30 mg/kg/day	0/0	2/2	15/7	30/16	41/19	88/44
		10 mg/kg/day	0/0	1/1	12/9	50/23	22/10	85/43
		3 mg/kg/day	0/0	0/0	12/8	31/15	33/17	76/40
S-11	Subcutaneous lipoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
S-12	Subcutaneous fibroma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	1/1	0/0	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-14	Fibrosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-16	Subcutaneous carcinosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-21	Subcutaneous myxosarcoma	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
	Mammary (tumorous)[a]							
	Total number of subcutaneous tumors /number of rats bearing subcutaneous tumors	0 mg/kg/day	0/0	1/1	32/14	69/30	79/31	181/76
		30 mg/kg/day	0/0	2/2	15/7	30/16	41/19	88/44
		10 mg/kg/day	0/0	1/1	12/9	50/23	22/10	85/43
		3 mg/kg/day	0/0	0/0	12/8	31/15	33/17	76/40
S-30	Mammary gland fibroadenoma/ adenofibroma	0 mg/kg/day	0/0	0/0	25/13	51/25	62/27	138/65
		30 mg/kg/day	0/0	1/1	11/7	15/10	28/15	55/33
		10 mg/kg/day	0/0	1/1	12/9	35/18	14/10	62/38
		3 mg/kg/day	0/0	0/0	9/7	14/9	24/15	47/31
S-31	Mammary gland adenoma	0 mg/kg/day	0/0	0/0	3/2	10/6	2/2	15/10
		30 mg/kg/day	0/0	0/0	1/1	3/2	5/5	9/8
		10 mg/kg/day	0/0	0/0	0/0	3/2	1/1	4/3
		3 mg/kg/day	0/0	0/0	1/1	4/4	2/1	7/6
S-32	Mammary gland adenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	3/2	1/1	3/3	7/6
		30 mg/kg/day	0/0	0/0	1/1	2/2	3/3	6/6
		10 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		3 mg/kg/day	0/0	0/0	0/0	2/2	2/2	4/4

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND (Cont'd)							
	Mammary (Tumorous)[a] (Cont'd)							
S-33	Mammary gland adenocarcinoma with metastasis	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	1/1	1/1	2/2	0/0	4/4
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	1/1	1/1	0/0	2/2
S-34	Mammary gland cystadenoma	0 mg/kg/day	0/0	0/0	0/0	2/2	0/0	2/2
		30 mg/kg/day	0/0	0/0	0/0	0/0	3/3	3/3
		10 mg/kg/day	0/0	0/0	0/0	2/2	4/2	6/4
		3 mg/kg/day	0/0	0/0	1/1	1/1	0/0	2/2
S-36	Mammary gland fibroma	0 mg/kg/day	0/0	0/0	1/1	0/0	3/3	4/4
		30 mg/kg/day	0/0	0/0	0/0	3/2	0/0	3/2
		10 mg/kg/day	0/0	0/0	0/0	3/3	0/0	3/3
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
S-37	Mammary gland cystfibroadenoma/ cystadenofibroma	0 mg/kg/day	0/0	0/0	0/0	4/4	9/7	13/11
		30 mg/kg/day	0/0	0/0	0/0	4/4	1/1	5/5
		10 mg/kg/day	0/0	0/0	0/0	7/6	2/1	9/7
		3 mg/kg/day	0/0	0/0	0/0	6/4	4/3	10/7
S-41	Mammary gland unclassified malignant neoplasm (carcinoma with accompanying fibrous proliferation)	0 mg/kg/day	0/0	1/1	0/0	0/0	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-42	Mammary gland cystadenocarcinoma without metastasis	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Integument (tumorous)[a]							
	Total number of cutaneous tumors/ number of rats bearing cutaneous tumors	0 mg/kg/day	0/0	0/0	0/0	0/0	2/2	2/2
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-72	Squamous papilloma of integument	0 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-76B	Keratoacanthoma	0 mg/kg/day	0/0	0/0	0/0	0/0	1/1	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
	Ear Canal[a]							
	Total number of ear canal tumors/ number of rats bearing ear canal tumors	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	SUBCUTANEOUS TISSUES, INTEGUMENT, AND MAMMARY GLAND (Cont'd)							
	Ear Canal[a] (Cont'd)							
S-76	Zymbal gland carcinoma of ear canal	0 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
S-78	Adenoma (sebaceous) of Zymbal gland	0 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		30 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		10 mg/kg/day	0/0	0/0	0/0	0/0	0/0	0/0
		3 mg/kg/day	0/0	0/0	0/0	1/1	0/0	1/1
	NERVOUS SYSTEM							
	Pituitary							
	Number of tissues examined	0 mg/kg/day	0	1	16	29	36	82
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	1	9	24	9	44
		3 mg/kg/day	0	0	10	17	18	45
N-86	Pituitary adenoma formation(s)	0 mg/kg/day	0	0	8	10	24	42
		30 mg/kg/day	0	0	3	8	8	19
		10 mg/kg/day	0	0	1	13	6	20
		3 mg/kg/day	0	0	3	9	11	23
N-87	Pituitary adenocarcinoma without extension or invasion of brain	0 mg/kg/day	0	0	2	1	3	6
		30 mg/kg/day	0	0	0	2	3	5
		10 mg/kg/day	0	0	0	3	0	3
		3 mg/kg/day	0	0	0	4	2	6
N-88	Pituitary adenocarcinoma with invasion of brain	0 mg/kg/day	0	0	1	3	1	5
		30 mg/kg/day	0	0	0	2	0	2
		10 mg/kg/day	0	0	0	2	0	2
		3 mg/kg/day	0	0	0	0	2	2
N-90	Pituitary adenoma arising from pars intermedia	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Cranial Cavity							
N-70	Neurofibrosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
	ADRENAL GLAND							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50

Data listed as number of rats for which the listed observation was present.

[a]Data listed as total number of this type neoplasm/number of rats bearing this type of neoplasm.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS
CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code	Number of rats necropsied during the time period indicated		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
		0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	ADRENAL GLAND (Cont'd)							
	Cortex							
A-31	Adenoma of adrenal cortex	0 mg/kg/day	0	0	2	0	4	6
		30 mg/kg/day	0	0	2	1	0	3
		10 mg/kg/day	0	0	0	2	2	4
		3 mg/kg/day	0	0	0	0	2	2
	Medulla							
A-41	Adrenal pheochromocytoma, unilateral	0 mg/kg/day	0	0	1	1	4	6
		30 mg/kg/day	0	0	0	0	2	2
		10 mg/kg/day	0	0	0	1	1	2
		3 mg/kg/day	0	0	0	1	2	3
A-44	Adrenal pheochromocytoma, malignant, no metastasis - unilateral	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	MISCELLANEOUS (Cont'd)							
Z-11	Mesenteric lipoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
Z-15	Abdominal hemangiosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-16	Granulocytic leukemia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0
Z-18	Generalized monocytic leukemia	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	LYMPHORETICULAR							
	Lymph Nodes - General							
L-01	Generalized lymphosarcoma with widespread metastasis	0 mg/kg/day	0	0	1	1	0	2
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	1	0	0	1
		3 mg/kg/day	1	0	0	0	0	1
L-02	Intraabdominal lymphosarcoma or carcinosarcoma of unspecified origin with widespread metastasis	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	LYMPHORETICULAR (Cont'd)							
	Mesenteric Lymph Node(s)							
	Number of tissues examined	0 mg/kg/day	0	2	16	30	36	84
		30 mg/kg/day	0	1	9	17	21	48
		10 mg/kg/day	1	1	9	25	11	47
		3 mg/kg/day	0	0	8	16	15	39
L-37	Lymphangioma of mesenteric lymph node	0 mg/kg/day	0	0	0	1	0	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	Thymus							
	Number of tissues examined	0 mg/kg/day	0	1	9	24	36	70
		30 mg/kg/day	0	2	6	15	20	43
		10 mg/kg/day	1	2	8	22	10	43
		3 mg/kg/day	0	1	8	16	18	43
[illegible]-56	Thymic lymphosarcoma	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	1	0	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0
	SPLEEN							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	17	21	49
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
L-67	Generalized myeloid leukemia apparently originating in spleen	0 mg/kg/day	0	0	0	0	0	0
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	1	0	1
	THYROID AND PARATHYROID							
	Thyroid							
	Number of tissues examined	0 mg/kg/day	0	2	17	30	36	85
		30 mg/kg/day	0	2	9	18	20	49
		10 mg/kg/day	1	2	11	23	11	48
		3 mg/kg/day	1	1	11	18	19	50
T-01	Thyroid interfollicular C-cell adenoma	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	1	2	3	6
		10 mg/kg/day	0	0	0	0	2	2
		3 mg/kg/day	0	0	1	2	7	10[a]
T-02	Thyroid interfollicular C-cell adenocarcinoma without metastasis	0 mg/kg/day	0	0	0	1	2	3
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	2	2
T-03	Thyroid interfollicular C-cell adenocarcinoma with metastasis	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	0	0
		10 mg/kg/day	0	0	0	1	0	1
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

[a]Statistically different from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 37 (Continued)

TUMOR INCIDENCE IN FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

Diagnosis Code			1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
	Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
		30 mg/kg/day	0	2	9	18	21	50
		10 mg/kg/day	1	2	11	25	11	50
		3 mg/kg/day	1	1	11	18	19	50
	THYROID AND PARATHYROID							
	Parathyroid							
	Number of tissues examined	0 mg/kg/day	0	2	17	29	27	75
		30 mg/kg/day	0	2	9	17	13	41
		10 mg/kg/day	1	2	10	12	5	30
		3 mg/kg/day	0	1	9	10	14	34
T-21	Parathyroid adenoma	0 mg/kg/day	0	0	0	0	1	1
		30 mg/kg/day	0	0	0	0	1	1
		10 mg/kg/day	0	0	0	0	0	0
		3 mg/kg/day	0	0	0	0	0	0

Data listed as number of rats for which the listed observation was present.

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TABLE 38

SUPPLEMENTAL TUMOR INCIDENCE DATA IN RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T

Sex	Males				Females			
Dose (mg/kg/day)	0	30	10	3	0	30	10	3
Number of Rats in Group	86	50	50	50	86	50	50	50
Total Number of Tumors in Group	172	97	71	89	308	150	147	140
Average Number of Tumors per Rat	2.0	1.9	1.4	1.8	3.6	3.0	2.9	2.8
Number of Tumor-bearing Rats/Number of Rats in Group								
These Data Listed in Time Intervals in Months:								
1-6	1/1	0/0	0/0	0/0	0/0	0/0	0/1	1/1
7-12	1/2	0/0	1/1	0/1	1/2	2/2	1/2	0/1
13-18	8/14	6/9	15/22	9/12	16/17	9/9	11/11	10/11
19-24	60/63	24/25	20/23	26/28	30/31	18/18	25/25	18/18
Terminal Kill	6/6	14/16	4/4	9/9	36/36	21/21	11/11	19/19
Total[a]	76/86 (88%)	44/50 (88%)	40/50 (80%)	44/50 (88%)	83/86 (97%)	50/50 (100%)	48/50 (96%)	48/50 (96%)

[a]No statistical differences from control data when analyzed using Fisher's Exact Probability Test, $p<0.05$.

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TABLE 39

TISSUES EXAMINED MICROSCOPICALLY FROM MALE AND FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118-119 DAYS

	Dose (mg/kg/day)			
	Male		Female	
Tissues	0	30	0	30
Liver	10	10	10	10
Liver (oil red O)	9	9	10	9
Kidneys	10	10	10	10
Kidneys (oil red O)	10	10	10	10
Heart	10	10	10	10
Pancreas	9	10	8	10
Spleen	10	10	10	10
Brain	10	10	10	10
Spinal cord	10	10	9	10
Peripheral nerve	10	9	7	8
Pituitary	9	10	5	9
Stomach	10	10	10	10
Small intestine	10	10	10	10
Large intestine	10	10	10	10
Mesenteric lymph node(s)	10	9	7	10
Skeletal muscle	10	10	10	10
Salivary gland	8	10	10	9
Testes	10	10	--	--
Epididymis	10	10	--	--
Accessory sex glands	10	10	--	--
Urinary bladder	10	10	7	10
Uterus	--	--	10	10
Ovary	--	--	10	10
Vagina	--	--	0	3
Trachea	10	10	10	10
Esophagus	8	10	10	10
Aorta	9	10	9	9
Thoracic lymph node(s)	6	8	10	8
Thymus	10	10	10	10
Lungs	10	10	10	10
Integument	7	7	9	10
Thyroid gland	10	9	9	10
Parathyroid gland(s)	5	6	7	8
Adipose tissue	8	10	10	10
Eyes	5	5	5	5
Adrenal(s)	10	10	9	10
Peyer's patch	4	4	2	4
Submandibular lymph node(s)	3	2	1	1
Subcutaneous lymph node(s)	3	1	4	2
Unidentified lymph node(s)	0	3	0	2
Nictitating membrane	0	0	1	1

Data listed as number of rats for which the listed tissue was examined microscopically.

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TABLE 40

TISSUES EXAMINED MICROSCOPICALLY FROM MALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Liver	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Kidneys	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Adrenals	0 mg/kg/day	1	2	12	63	6	84
	30 mg/kg/day	0	0	9	25	15	50
	10 mg/kg/day	0	0	22	23	4	49
	3 mg/kg/day	0	1	12	28	9	50
Heart	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Pancreas	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Spleen	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Peripheral Nerve	0 mg/kg/day	1	1	13	61	6	82
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	20	23	4	48
	3 mg/kg/day	0	1	11	24	9	45
Spinal Cord	0 mg/kg/day	1	1	14	63	6	85
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	21	23	4	49
	3 mg/kg/day	0	1	12	27	9	49
Pituitary	0 mg/kg/day	1	2	13	59	6	82
	30 mg/kg/day	0	0	9	24	16	49
	10 mg/kg/day	0	1	21	22	4	48
	3 mg/kg/day	0	1	11	26	9	47
Brain	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Mesenteric Lymph Node	0 mg/kg/day	1	2	12	60	6	81
	30 mg/kg/day	0	0	9	23	16	48
	10 mg/kg/day	0	1	19	20	4	44
	3 mg/kg/day	0	0	9	25	8	42

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TABLE 40 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM MALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Small Intestine	0 mg/kg/day	1	2	14	62	6	85
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	25	9	47
Large Intestine	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	21	23	4	49
	3 mg/kg/day	0	1	12	27	8	48
Stomach	0 mg/kg/day	1	2	14	62	6	85
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	22	4	49
	3 mg/kg/day	0	1	12	27	9	49
Testes	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Epididymis	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	27	9	49
Urinary Bladder	0 mg/kg/day	1	2	14	62	6	85
	30 mg/kg/day	0	0	8	25	16	49
	10 mg/kg/day	0	1	20	22	4	47
	3 mg/kg/day	0	1	12	28	9	50
Accessory Sex Glands	0 mg/kg/day	1	2	14	62	6	85
	30 mg/kg/day	0	0	8	25	16	49
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Lungs	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Skeletal Muscle	0 mg/kg/day	1	1	14	62	6	84
	30 mg/kg/day	0	0	8	25	15	48
	10 mg/kg/day	0	1	20	22	4	47
	3 mg/kg/day	0	1	11	25	9	46
Salivary Gland	0 mg/kg/day	1	2	12	59	6	80
	30 mg/kg/day	0	0	7	23	15	45
	10 mg/kg/day	0	1	17	23	4	45
	3 mg/kg/day	0	1	10	25	8	44
Aorta or Large Mediastinal Artery	0 mg/kg/day	1	2	14	60	6	83
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	24	4	48
	3 mg/kg/day	0	1	11	27	9	48

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TABLE 40 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM MALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Thyroid	0 mg/kg/day	0	2	13	63	6	84
	30 mg/kg/day	0	0	8	24	16	48
	10 mg/kg/day	0	1	22	23	3	49
	3 mg/kg/day	0	1	12	28	8	49
Parathyroid	0 mg/kg/day	0	1	12	60	6	79
	30 mg/kg/day	0	0	7	24	12	43
	10 mg/kg/day	0	0	19	22	3	44
	3 mg/kg/day	0	1	10	25	6	42
Esophagus	0 mg/kg/day	1	1	14	62	6	84
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	21	21	4	47
	3 mg/kg/day	0	0	12	28	9	49
Trachea	0 mg/kg/day	1	2	14	62	6	85
	30 mg/kg/day	0	0	9	24	16	49
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Thoracic Lymph Node	0 mg/kg/day	1	2	13	59	6	81
	30 mg/kg/day	0	0	7	21	16	44
	10 mg/kg/day	0	1	22	23	3	48
	3 mg/kg/day	0	1	11	27	7	46
Thymus	0 mg/kg/day	1	2	12	43	1	59
	30 mg/kg/day	0	0	4	22	12	38
	10 mg/kg/day	0	1	14	17	3	35
	3 mg/kg/day	0	1	10	19	7	37
Integument	0 mg/kg/day	0	1	9	59	6	75
	30 mg/kg/day	0	0	5	23	16	44
	10 mg/kg/day	0	0	16	19	4	39
	3 mg/kg/day	0	0	7	27	9	43
Tongue	0 mg/kg/day	0	1	13	63	6	83
	30 mg/kg/day	0	0	8	25	16	49
	10 mg/kg/day	0	0	19	23	4	46
	3 mg/kg/day	0	1	12	28	9	50
Eyes	0 mg/kg/day	0	2	13	61	6	82
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	20	23	4	48
	3 mg/kg/day	0	1	11	28	9	49
Remarkable Mesenteric Blood Vessels	0 mg/kg/day	0	1	6	35	3	45
	30 mg/kg/day	0	0	1	11	5	17
	10 mg/kg/day	0	0	8	10	2	20
	3 mg/kg/day	0	0	7	12	5	24
Mammary Gland	0 mg/kg/day	0	0	0	4	1	5
	30 mg/kg/day	0	0	1	0	2	3
	10 mg/kg/day	0	0	8	3	0	11
	3 mg/kg/day	0	0	4	6	2	12

In addition to the standard list of tissues presented above, certain other tissues were examined microscopically, most often due to a grossly-recognized lesion. These tissues are listed below.

Subcutaneous Lymph Node	0 mg/kg/day	0	0	0	3	1	4
	30 mg/kg/day	0	0	1	3	4	8
	10 mg/kg/day	0	0	0	3	1	4
	3 mg/kg/day	0	0	1	4	1	6

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TABLE 40 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM MALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Pineal Gland	0 mg/kg/day	0	0	1	2	2	5
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Remarkable Thoracic Blood Vessels	0 mg/kg/day	0	0	1	0	0	1
	30 mg/kg/day	0	0	0	2	2	4
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	1	0	1
Subauricular Mass	0 mg/kg/day	0	0	1	4	0	5
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	1	1	0	2
	3 mg/kg/day	0	0	0	0	0	0
Ureter/Urethra	0 mg/kg/day	0	0	1	0	0	1
	30 mg/kg/day	0	0	1	0	0	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	1	0	1
Cervical Lymph Node	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	1	0	0	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Hard Palate/Nasal Turbinates	0 mg/kg/day	0	0	1	2	1	4
	30 mg/kg/day	0	0	0	1	0	1
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	2	0	2
Mesentery	0 mg/kg/day	0	0	0	6	0	6
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	1	2
	3 mg/kg/day	0	0	1	3	0	4
Remarkable Mediastinal TIssue	0 mg/kg/day	0	0	0	7	0	7
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	2	0	0	2
	3 mg/kg/day	0	0	2	0	0	2
Preputial Gland	0 mg/kg/day	0	0	0	3	1	4
	30 mg/kg/day	0	0	0	1	3	4
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Cecum	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	1	0	1
Oral Cavity	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0

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TABLE 40 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM MALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	1	2	14	63	6	86
	30 mg/kg/day	0	0	9	25	16	50
	10 mg/kg/day	0	1	22	23	4	50
	3 mg/kg/day	0	1	12	28	9	50
Lymph Nodes (unknown origin)	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	1	0	0	1
Rib	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Cranial Nerve or Cranial Cavity	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Retropharyngeal Lymph Node	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Retrobulbar Mass	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	1	0	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Lip	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Periaortal Lymph Node	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0

Data on tumorous and nontumorous grossly-recognized subcutaneous and cutaneous nodules or masses are listed in Table 34.

TABLE 41

TISSUES EXAMINED MICROSCOPICALLY FROM FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Liver	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Kidneys	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Adrenals	0 mg/kg/day	0	2	17	30	36	85
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Heart	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Pancreas	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Spleen	0 mg/kg/day	0	2	17	30	36	85
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Peripheral Nerve	0 mg/kg/day	0	2	16	30	35	83
	30 mg/kg/day	0	1	8	17	21	47
	10 mg/kg/day	1	2	10	23	10	46
	3 mg/kg/day	1	0	11	15	18	45
Spinal Cord	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	0	11	18	19	49
Pituitary	0 mg/kg/day	0	1	16	29	36	82
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	1	9	24	9	44
	3 mg/kg/day	0	0	10	17	18	45
Brain	0 mg/kg/day	0	2	17	31	36	85
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Mesenteric Lymph Node	0 mg/kg/day	0	2	16	30	36	84
	30 mg/kg/day	0	1	9	17	21	48
	10 mg/kg/day	1	1	9	25	11	47
	3 mg/kg/day	0	0	8	16	15	39

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TABLE 41 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Small Intestine	0 mg/kg/day	0	2	17	29	36	84
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Large Intestine	0 mg/kg/day	0	2	16	31	36	85
	30 mg/kg/day	0	2	9	16	21	48
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Stomach	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	17	19	49
Ovary(ies)	0 mg/kg/day	0	2	16	31	36	85
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	11	24	11	49
	3 mg/kg/day	1	1	11	18	19	50
Oviduct(s)	0 mg/kg/day	0	2	15	29	33	77
	30 mg/kg/day	0	2	9	17	20	48
	10 mg/kg/day	1	2	8	20	11	42
	3 mg/kg/day	1	1	8	17	15	42
Urinary Bladder	0 mg/kg/day	0	2	17	30	32	81
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	10	24	10	47
	3 mg/kg/day	1	1	10	18	17	47
Uterus	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	11	24	11	49
	3 mg/kg/day	1	1	11	18	19	50
Lungs	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Skeletal Muscle	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	1	9	15	21	46
	10 mg/kg/day	1	2	11	24	10	48
	3 mg/kg/day	1	0	11	18	18	48
Salivary Gland	0 mg/kg/day	0	2	15	29	36	82
	30 mg/kg/day	0	2	9	15	20	46
	10 mg/kg/day	1	2	9	21	11	44
	3 mg/kg/day	1	1	11	17	18	48
Aorta or Large Mediastinal Artery	0 mg/kg/day	0	1	16	30	36	83
	30 mg/kg/day	0	2	7	16	21	46
	10 mg/kg/day	1	2	10	23	11	47
	3 mg/kg/day	0	1	11	16	19	47

TABLE 41 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Thyroid	0 mg/kg/day	0	2	17	30	36	85
	30 mg/kg/day	0	2	9	18	20	49
	10 mg/kg/day	1	2	11	23	11	48
	3 mg/kg/day	1	1	11	18	19	50
Parathyroid	0 mg/kg/day	0	2	17	29	27	75
	30 mg/kg/day	0	2	9	17	13	41
	10 mg/kg/day	1	2	10	12	5	30
	3 mg/kg/day	0	1	9	10	14	34
Esophagus	0 mg/kg/day	0	2	17	31	33	86
	30 mg/kg/day	0	2	9	17	21	49
	10 mg/kg/day	1	2	8	25	11	47
	3 mg/kg/day	1	1	11	18	19	50
Trachea	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Thoracic Lymph Node	0 mg/kg/day	0	1	14	26	33	74
	30 mg/kg/day	0	2	8	13	19	42
	10 mg/kg/day	1	2	9	21	11	44
	3 mg/kg/day	1	1	10	17	17	46
Thymus	0 mg/kg/day	0	1	9	24	36	70
	30 mg/kg/day	0	2	6	15	20	43
	10 mg/kg/day	1	2	8	22	10	43
	3 mg/kg/day	0	1	8	16	18	43
Integument	0 mg/kg/day	0	1	8	27	36	72
	30 mg/kg/day	0	0	6	16	21	43
	10 mg/kg/day	0	0	7	25	11	43
	3 mg/kg/day	0	0	10	18	19	47
Tongue	0 mg/kg/day	0	1	17	30	35	83
	30 mg/kg/day	0	1	9	18	21	49
	10 mg/kg/day	0	1	11	25	11	48
	3 mg/kg/day	0	0	11	18	19	48
Eye(s)	0 mg/kg/day	0	1	17	31	36	85
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	10	25	11	49
	3 mg/kg/day	1	1	11	18	19	50
Remarkable Mesenteric Blood Vessels	0 mg/kg/day	0	0	1	1	9	11
	30 mg/kg/day	0	0	0	0	5	5
	10 mg/kg/day	0	0	1	3	1	5
	3 mg/kg/day	0	0	1	2	5	8
Mammary Gland	0 mg/kg/day	0	0	7	16	26	49
	30 mg/kg/day	0	0	1	3	18	22
	10 mg/kg/day	0	0	4	20	8	32
	3 mg/kg/day	0	0	9	10	11	30

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TABLE 41 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50

In addition to the standard list of tissues presented above, additional tissues were examined microscopically, most often resulting from grossly-recognized lesions, and these tissues are listed below.

Subcutaneous Lymph Node	0 mg/kg/day	0	0	2	3	6	11
	30 mg/kg/day	0	0	1	2	4	7
	10 mg/kg/day	0	0	2	9	1	12
	3 mg/kg/day	0	0	5	6	3	14
Pineal Gland	0 mg/kg/day	0	6	0	0	0	6
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	1	0	0	1
Abdominal Mass	0 mg/kg/day	0	0	0	1	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	1	0	0	1
	3 mg/kg/day	0	0	0	0	0	0
Cervical Lymph Node	0 mg/kg/day	0	0	0	0	1	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Paravertebral Mass	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	1	0	0	0	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Remarkable Mesenteric Tissue	0 mg/kg/day	0	0	0	3	2	5
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	1	3	1	5
Remarkable Mediastinal Tissue	0 mg/kg/day	0	1	0	0	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	1	2	0	3
	3 mg/kg/day	0	0	0	1	0	1
Clitoral Gland	0 mg/kg/day	0	1	2	0	0	3
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	1	1
	3 mg/kg/day	0	0	0	0	0	0
Vagina/Vaginal Mass	0 mg/kg/day	0	0	0	0	1	1
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Gingiva	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	0	1	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0

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Data on grossly-recognized subcutaneous and cutaneous masses and nodules are listed in Table 35.

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TABLE 41 (Continued)

TISSUES EXAMINED MICROSCOPICALLY FROM FEMALE RATS
MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR UP TO TWO YEARS

		1-6 Months	7-12 Months	13-18 Months	19-24 Months	Terminal Kill	Cumulative Results
Number of rats necropsied during the time period indicated	0 mg/kg/day	0	2	17	31	36	86
	30 mg/kg/day	0	2	9	18	21	50
	10 mg/kg/day	1	2	11	25	11	50
	3 mg/kg/day	1	1	11	18	19	50
Lymph Nodes (unknown origin)	0 mg/kg/day	0	1	0	0	0	1
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0
Cranial Mass	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Bone Marrow	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	0	0	0
	10 mg/kg/day	0	0	0	1	0	1
	3 mg/kg/day	0	0	0	0	0	0
Periaortal Lymph Node	0 mg/kg/day	0	0	0	0	0	0
	30 mg/kg/day	0	0	0	1	0	1
	10 mg/kg/day	0	0	0	0	0	0
	3 mg/kg/day	0	0	0	0	0	0

APPENDIX 1

ANALYTICAL CONTENT OF 2,4,5-T IN DIET
USED IN A TWO-YEAR TOXICITY STUDY IN RATS

Month of Study When Sample Collected	Content of 2,4,5-T in Diet Samples: Intended	Content of 2,4,5-T in Diet Samples: Analytical Results	Dietary Dose Level mg/kg/day
5	10,000 ppm	9000 ppm	Premix
5	591 ppm	190[a] 580 ppm	30
5	199 ppm	180 ppm	10
5	57.4 ppm	63 ppm	3
5	00.0 ppm	2 ppm	0
7	10,000 ppm	9500 ppm	Premix
7	660 ppm	660 ppm	30
7	219 ppm	170[a] 240 ppm	10
7	65.4 ppm	63 ppm	3
7	00.0 ppm	N.D.[b]	0
12	10,000 ppm	10,700 ppm	Premix
12	698 ppm	740-760 ppm	30
12	223 ppm	250 ppm	10
12	77.7 ppm	89-97 ppm	3
12	00.0 ppm	N.D.[b]	0
19	10,000 ppm	10,000 ppm	Premix
19	760 ppm	760 ppm	30 Males
19	276 ppm	250 ppm	10 males
19	81 ppm	62-67 ppm	3 Males
19	654 ppm	590-670 ppm	30 Females
19	192 ppm	200 ppm	10 Females
19	66.8 ppm	64 ppm	3 Females
19	00.0 ppm	N.D.[b]	0
23	10,000 ppm	9300 ppm	Premix
23	714 ppm	700 ppm	30 Males
23	253 ppm	250 ppm	10 Males
23	76.7 ppm	150 ppm	3 Males
23	650 ppm	660 ppm	30 Females
23	212 ppm	240 ppm	10 Females
23	60.4 ppm	94 ppm	3 Females
23	00.0 ppm	15 ppm	0

[a]Lower value excluded from calculation of average dietary content.
[b]None Detected.

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APPENDIX 2

INDIVIDUAL AND MEAN BODY WEIGHTS FOR MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 DAYS

Dose 2,4,5-T mg/kg/day	Animal Number	Days on Test 5	13	20	26	32	39	46	53	60	68	81	88	95	102	116
0	75-594	238	328	361	366	389	361	410	417	425	434	457	463	465	468	431
	75-595	238	333	365	380	396	368	414	433	443	440	469	471	474	485	464
	75-596	252	320	342	367	374	394	414	432	449	402	466	483	488	497	501
	75-597	248	347	378	389	406	427	444	463	467	448	472	487	498	498	477
	75-598	243	336	370	391	407	432	457	466	482	489	506	525	534	535	494
	75-599	251	342	365	389	399	421	441	450	464	460	484	498	498	515	510
	75-600	270	374	409	427	455	482	506	521	536	518	570	578	574	586	540
	75-601	244	330	355	373	391	406	424	437	448	453	467	485	495	501	508
	75-602	248	341	373	394	414	428	440	452	459	442	464	482	488	496	491
	75-603	242	326	357	366	382	402	415	427	447	455	455	473	474	490	486
	MEAN	247	337	367	384	401	412	437	450	462	454	481	494	499	507	490
	±S.D.	±9	±15	±18	±19	±22	±35	±29	±30	±30	±31	±35	±34	±32	±33	±29
30	75-624	253	327	359	380	388	405	420	436	443	445	462	470	476	455	477
	75-625	235	314	347	369	379	396	416	425	428	425	451	446	454	471	457
	75-626	250	346	368	395	411	427	443	463	470	494	506	508	516	526	537
	75-627	245	325	348	350	365	378	396	408	424	442	451	462	471	484	491
	75-628	248	362	399	422	447	457	484	493	502	529	531	462	550	543	558
	75-629	237	316	339	363	373	392	421	422	425	439	457	541	464	471	468
	75-630	256	353	389	407	423	446	470	483	492	508	525	531	543	552	561
	75-631	234	316	340	359	369	387	420	414	421	434	439	442	416	455	456
	75-632	223	308	332	351	366	386	395	409	414	437	448	451	434	465	458
	75-633	245	327	352	371	388	402	423	446	454	471	476	486	498	501	483
	MEAN	243	329	357	377	391	408	426	440	447	462	474	480	484	492	494
	±S.D.	±10	±18	±22	±24	±28	±27	±30	±31	±31	±36	±34	±35	±42	±36	±42
10	75-614	247	324	352	371	380	395	406	421	429	436	459	458	391	478	478
	75-615	229	297	311	325	336	347	357	361	372	356	386	389	476	405	409
	75-616	233	313	340	357	369	383	400	417	415	417	443	433	456	465	435
	75-617	250	341	375	401	415	441	455	476	490	502	530	546	552	543	558
	75-618	249	345	388	418	441	460	484	509	523	534	563	570	587	592	595
	75-619	261	358	387	410	434	451	470	484	502	489	533	543	547	558	566
	75-620	252	330	357	379	395	409	431	443	457	459	496	497	503	513	489
	75-621	254	349	378	411	424	438	456	476	484	490	520	522	542	546	547
	75-622	255	352	392	419	442	464	492	503	527	527	578	584	593	612	581
	75-623	252	329	356	366	383	398	414	423	432	424	443	441	450	457	460
	MEAN	248	334	364	386	402	419	437	451	463	463	495	500	510	517	512
	±S.D.	±10	±19	±26	±31	±35	±38	±43	±46	±51	±56	±61	±63	±66	±65	±66
3	75-604	256	353	387	407	419	444	461	475	493	490	510	515	524	521	529
	75-605	237	325	357	383	392	415	430	445	457	456	471	476	474	476	477
	75-606	244	339	380	399	423	453	473	496	512	497	460	532	564	569	569
	75-607	242	333	351	370	385	400	408	419	433	436	547	463	472	475	482
	75-608	222	302	328	334	345	358	365	378	377	346	397	405	420	421	429
	75-609	262	370	403	426	446	462	480	496	509	513	533	540	556	569	576
	75-610	241	325	355	379	402	413	431	437	451	452	483	489	499	501	495
	75-611	254	350	383	405	420	436	451	468	478	476	491	504	499	512	514
	75-612	243	336	364	381	398	416	439	455	468	469	496	513	523	534	522
	75-613	234	326	355	367	387	409	424	436	446	448	475	483	490	501	439
	MEAN	243	336	366	385	402	421	436	450	462	458	486	494	502	508	508
	±S.D.	±12	±19	±22	±26	±28	±30	±33	±36	±40	±46	±42	±42	±42	±45	±44

No statistically significant differences from control means using Dunnett's test, $p<0.05$.

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APPENDIX 3

INDIVIDUAL AND MEAN BODY WEIGHTS FOR FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 119 DAYS

Dose 2,4,5-T mg/kg/day	Animal Number	Days on Test 6	13	20	26	32	39	46	53	60	68	81	88	95	102	116
0	75-634	196	227	244	245	266	260	263	274	270	282	283	286	290	291	298
	75-635	201	239	247	252	260	266	274	281	290	294	289	297	300	304	315
	75-636	209	251	260	268	279	281	292	297	298	309	311	320	318	330	321
	75-637	191	225	243	245	260	258	273	271	276	284	292	297	300	305	288
	75-638	197	235	243	254	262	279	277	292	283	291	295	290	298	299	311
	75-639	202	247	254	259	269	266	281	289	289	302	299	312	309	316	326
	75-640	206	241	264	268	285	292	288	299	299	308	314	313	320	332	327
	75-641	188	237	243	250	255	266	273	279	278	284	292	295	299	304	312
	75-642	205	245	251	266	275	284	291	299	310	308	314	316	328	331	337
	75-643	208	255	258	278	293	295	309	313	295	326	333	334	335	345	350
	MEAN	200	240	251	259	270	274	282	288	289	299	302	306	310	316	318
	±S.D.	±7	±10	±8	±11	±12	±12	±13	±13	±12	±14	±15	±15	±15	±18	±18
30	75-664	208	246	252	263	269	281	276	285	290	294	292	303	309	312	320
	75-665	200	237	245	252	260	262	262	271	272	280	283	284	291	290	291
	75-666	195	233	248	255	266	265	279	288	282	284	294	291	295	297	306
	75-667	204	240	256	264	272	278	286	289	298	298	302	300	307	309	311
	75-668	208	247	261	270	275	284	281	280	284	286	294	295	307	301	312
	75-669	194	237	251	269	284	282	291	297	299	307	323	329	326	331	338
	75-670	206	235	247	258	273	265	278	276	282	290	301	301	300	311	319
	75-671	215	243	255	268	271	279	283	295	290	302	303	310	314	318	328
	75-672	212	261	278	292	298	301	307	324	318	325	332	335	345	341	357
	75-673	208	242	253	261	270	270	284	286	283	294	298	311	315	314	308
	MEAN	205	242	255	265	274	277	282	289	290	296	302	306	311	312	319
	±S.D.	±7	±8	±10	±11	±10	±12	±11	±15	±13	±13	±15	±16	±16	±15	±18
10	75-654	192	223	239	243	252	263	266	274	272	286	287	293	298	295	305
	75-655	199	249	262	269	279	287	295	295	300	311	310	314	314	312	348
	75-656	205	258	270	286	310	309	319	329	343	339	343	348	360	331	365
	75-657	195	229	242	248	256	258	265	268	272	280	282	283	288	290	298
	75-658	196	237	254	262	270	278	276	287	299	318	310	313	320	325	309
	75-659	202	237	252	259	260	276	285	286	293	296	310	324	315	323	331
	75-660	210	263	283	290	298	305	313	321	327	335	347	344	349	352	367
	75-661	194	229	236	246	252	256	262	266	270	279	281	278	276	292	301
	75-662	197	246	254	261	260	270	277	281	288	297	302	299	312	313	338
	75-663	197	230	246	250	258	266	272	275	277	287	292	300	301	308	299
	MEAN	199	240	254	261	269	277	283	288	294	303	306	310	313	316	326
	±S.D.	±6	±14	±15	±16	±20	±18	±20	±21	±24	±22	±23	±24	±26	±22	±27
3	75-644	194	231	245	253	263	266	283	286	293	293	295	297	299	303	308
	75-645	206	243	265	275	282	298	309	309	318	317	328	331	338	330	343
	75-646	199	233	252	253	265	266	274	283	281	292	296	298	307	320	316
	75-647	203	239	243	252	260	257	271	277	272	288	288	291	303	299	312
	75-648	199	237	257	264	272	264	278	286	285	294	303	306	312	316	334
	75-649	196	238	252	265	263	266	275	278	281	290	294	294	302	307	334
	75-650	198	239	248	253	257	271	271	280	274	285	296	296	294	308	299
	75-651	204	242	261	266	273	279	292	298	294	304	310	318	316	321	264
	75-652	208	250	256	260	268	271	283	295	296	308	313	312	316	322	331
	75-653	204	253	276	290	275	290	297	308	320	321	327	324	333	337	342
	MEAN	201	240	256	263	268	273	284	290	290	299	305	307	312	314	318
	±S.D.	±5	±7	±10	±12	±8	±13	±13	±12	±14	±13	±14	±14	±14	±13	±24

No statistically significant differences from control means using Dunnett's test, $p<0.05$.

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APPENDIX 4

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 73-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
0	674	228	289	340	365	383	397	411	424	433	451	462	469	476
	675	223	284	331	367	387	406	423	444	459	471	493	504	512
	676	246	303	348	372	395	400	425	448	456	460	474	476	486
	677	245	310	354	396	412	434	466	472	497	503	521	527	537
	678	227	272	308	330	351	362	383	392	403	422	435	440	448
	679	226	280	319	345	366	379	401	417	430	439	457	463	472
	680	244	297	343	377	396	406	435	448	470	478	498	497	513
	681	249	288	330	353	369	381	399	417	424	445	460	462	472
	682	255	304	352	379	392	409	430	446	459	467	492	498	513
	683	225	278	325	354	379	395	415	439	449	456	475	482	489
	684	225	293	333	364	387	407	418	439	456	467	476	483	489
	685	231	293	331	365	386	401	416	434	444	454	470	480	486
	686	251	308	363	395	405	424	442	461	482	495	522	524	527
	687	250	300	352	381	411	425	443	461	469	489	506	527	534
	688	243	301	344	376	395	416	436	454	476	487	506	513	524
	689	238	296	341	375	405	430	448	470	484	500	516	525	535
	690	242	285	325	347	360	374	394	410	428	435	448	454	464
	691	233	271	303	320	362	340	347	357	363	373	385	389	396
	692	216	262	304	326	342	344	348	355	366	369	392	401	410
	693	248	294	336	358	375	391	408	422	434	443	458	467	479
	694	261				432				506				
	695	224				362				415				
	696	249				392				435				
	697	266				402				455				
	698	259				431				514				
	699	252				384				443				
	700	257				412				485				
	701	277				427				495				
	702	254				394				414				
	703	228				362				463				
	704	247				393				447				
	705	239				377				446				
	706	245				392				442				
	707	247				392				452				
	708	252				386				459				
	709	240				393				457				
	710	254				381				426				
	711	236				360				412				
	712	245				363				483				
	713	232				411				418				
	714	250				383				433				
	715	255				400				465				
	716	263				388				437				
	717	247				385				454				
	718	232				346				386				
	719	243				397				460				
	720	258				394				466				
	721	253				401				469				
	722	239				363				413				
	723	243				412				468				
	724	256				416				438				
	725	228				401				455				
	726	237				355				404				
	727	243				374				429				
	728	241				371				422				
	729	241				375				427				
	730	241				364				426				
	731	224				372				431				
	732	234				386				440				
	733	237				364				412				
	734	252				386				455				
	735	254				381				441				
	736	226				328				375				
	737	243				409				473				
	738	256				411				476				
	739	249				382				440				
	740	243				392				438				
	741	241				397				452				
	742	260				377				436				
	743	259				415				475				
	744	240				373				428				
	745	246				334				447				
	746	251				407				481				
	747	242				368				419				
	748	225				341				398				
	749	252				396				448				
	750	230				385				451				
	751	230				369				420				
	752	233				362				415				
	753	229				348				395				
	754	254				379				430				
	755	250				379				428				
	756	261				434				506				
	757	252				393				448				
	758	258				386				441				
	759	257				411				468				
	MEAN±SD	244±12	290±13	334±17	362±21	385±21	396±26	414±30	430±33	444±30	455±36	472±38	479±39	488±39

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	123	152	175	180	203	232	258	291	314	349	377
0	674	480	487	509	513	530	545	540	552	581	589	605	602	600
	675	520	524	559	581	591	600	609	636	652	670	687	682	689
	676	494	497	482	539	561	559	571	594	605	630	644	647	653
	677	547	536	590	591	603	620	615	628	630	652	659	630	635
	678	453	463	479	490	506	515	519	532	529	552	555	551	561
	679	485	482	516	525	550	555	567	579	588	606	619	616	622
	680	520	524	535	538	589	577	607	617	619	654	677	663	671
	681	427	482	503	514	530	546	548	561	567	590	586	583	599
	682	510	516	539	565	574	586	600	612	637	631	655	655	661
	683	497	509	536	549	567	580	576	589	610	621	630	625	630
	684	302	507	525	530	553	548	566	571	580	605	616	626	642
	685	496	502	507	532	553	549	564	588	605	617	627	634	641
	686	545	544	595	635	643	655	658	688	686	705	678	701	720
	687	532	538	562	576	593	599	600	613	623	647	654	663	680
	688	543	549	566	606	628	633	649	695	684	710	732	715	733
	689	542	551	564	584	623	610	605	637	657	674	686	676	679
	690	470	470	484	509	520	482	530	552	562	580	589	595	601
	691	402	402	419	429	436	404	452	476	480	491	507	500	508
	692	415	526	441	478	501	503	519	531	549	563	567	569	575
	693	484	484	512	526	550	550	555	566	580	598	613	608	614
	694		584		657			709	722	734	749	768	774	788
	695		466		501			530	544	554	575	571	573	589
	696		520		555			607	631	644	680	705	716	740
	697		495		507			558	562	574	595	600	593	615
	698		579		643			684	709	725	743	755	755	761
	699		489		524			553	580	570	594	611	592	617
	700		553		591			645	671	677	688	704	697	705
	701		559		594			644	657	672	700	715	714	729
	702		521		561			588	601	608	634	650	630	654
	703		454		488			511	535	542	572	571	554	568
	704		485		504			540	546	556	570	580	563	573
	705		495		531			577	585	603	618	633	612	626
	706		491		542			577	592	606	633	636	626	657
	707		510		543			569	588	588	605	609	622	625
	708		513		553			580	596	612	639	648	628	642
	709		513		558			595	621	625	663	678	678	681
	710		481		516			559	575	590	606	620	610	625
	711		440		464			491	500	516	532	534	532	545
	712		547		588			627	646	653	672	685	678	683
	713		462		487			517	535	546	566	572	562	577
	714		474		487			524	536	549	566	584	573	587
	715		525		587			616	638	647	658	678	663	674
	716		470		504			533	556	553	588	595	593	601
	717		513		549			570	587	606	616	627	620	635
	718	Dead												
	719		537		587			620	641	660	685	692	695	717
	720		527		575			516	548	545	694	699	675	707
	721		513		529			542	547	543	Dead			
	722		434		474			500	515	522	544	560	551	560
	723		523		534			586	603	614	630	658	645	665
	724		522		571			610	624	632	654	658	627	652
	725		515		570			599	627	635	675	687	686	704
	726		467		513			543	569	557	593	613	594	610
	727		477		511			545	562	579	602	607	614	645
	728		467		506			540	553	569	584	595	591	605
	729		461		507			533	548	571	584	588	587	600
	730		461		514			550	563	566	606	603	599	606
	731		468		504			523	541	546	566	576	587	601
	732		492		525			557	567	597	612	625	614	629
	733		453		482			508	531	529	548	563	546	563
	734		498		541			574	605	597	619	623	634	638
	735		490		523			564	583	598	611	622	619	620
	736		427		469			491	518	507	537	543	558	550
	737		534		550			577	597	603	620	619	614	632
	738		534		573			602	631	643	655	669	665	682
	739		498		528			550	577	602	616	619	610	626
	740		499		537			575	594	615	634	641	626	635
	741		491		517			551	571	571	582	583	572	569
	742		479		525			545	564	576	585	589	586	595
	743		520		572			584	601	607	620	635	631	647
	744		480		521			539	562	574	602	619	600	611
	745		498		535			565	578	580	607	614	607	619
	746		526		572			601	631	634	661	680	676	697
	747		472		506			533	546	557	582	595	585	596
	748		426		468			499	510	522	532	540	553	563
	749		501		545			580	599	625	639	670	683	706
	750		521		579			626	663	620	659	680	673	692
	751		480		512			537	554	560	567	555	574	553
	752		451		478			505	524	544	534	539	554	577
	753		444		486			508	532	550	577	573	581	590
	754		477		493			522	542	557	552	568	561	550
	755		464		497			520	538	543	562	569	562	579
	756		574		609			664	686	700	723	733	745	744
	757		512		540			568	594	608	634	647	644	651
	758		487		528			545	564	573	586	613	608	622
	759		518		557			572	580	615	615	621	605	619
	MEAN±SD	493±43	498±36	521±46	536±42	560±49	561±57	567±46	586±50	596±49	617±52	627±53	622±54	635±55

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		-05	-33	468	501	531	559	587	622	650	665	714	732
0	674	615	641	652	646	640	646	650			Dead		
	675	688	701	693	691	429	614	606	441			Dead	
	676	674					Dead						
	677	621	599					Dead					
	678	569	561	568	559	552	546	534	511	510	492	477	434
	679	653	657	651	646	639	669	650	634	624	616	586	558
	680	690	708	712	713	714	704	696	664	660	656	616	615
	681	616	613	631	632	635	619	611	588	586	576	547	529
	682	686	703	709	698	686	687	659			Dead		
	683	640	659	653	659	634	639	618	601			Dead	
	684	648	663	675	663	676	666	665	650	620	602	570	546
	685	654	669	663	637	578	590	581	558	461		Dead	
	686	745	760	778	753	711	687	643	579	549		Dead	
	687	693	704	723	695	690	693	708	691	686	651		Dead
	688	757	762	775	770				Dead				
	689	695	704	699	677	684	666	610			Dead		
	690	623	627	646	642	640				Dead			
	691	514	537	545	535	533	544	533	512	500		Dead	
	692	572	584	577	564	543	440				Dead		
	693	625	647	649	643	542	529				Dead		
	694	800	825	820	823	784	760	757	694			Dead	
	695	593	602	585	577	572	546	564			Dead		
	696	743	784	750	726	677	535				Dead		
	697	613	643	640	641	634	620	622	587	488		Dead	
	698	746						Dead					
	699	638	659	675	663	644	653	639	613	583	553		Dead
	700	703	709	698	666	660	615				Dead		
	701	735	748	744	756	754	738	724	711	743		Dead	
	702	642	668	659	667	631	624	607	546			Dead	
	703	580	598	576	554	532				Dead			
	704	580	590	584	452				Dead				
	705	624	673	676	670				Dead				
	706	671	681	690	671	663	646	626			Dead		
	707	634	656	639	618	583				Dead			
	708	654	670	659	676	635	619	591	533			Dead	
	709	691	714	695	684	676	659	649	584			Dead	
	710	632						Dead					
	711	560	586	583	575	569	576	579	517			Dead	
	712	694	704	700	696	595	685	684	654	631		Dead	
	713	586	590	588	586	597	595	585	590	570	576		Dead
	714	598	617	615	697	585	573	571	516			Dead	
	715	693	695	698	659	659	633	631	602	560		Dead	
	716	621	629	637	627	642	628	620	585	376	468		Dead
	717	643	660	650	650	630	633	609			Dead		
	718						Dead						
	719	693	702	670	667	631	630	613			Dead		
	720	703	737	719					Dead				
	721						Dead						
	722	566	588	579	579	576	575	567	547	530	465		Dead
	723	673	698	690	689	662	659	643	656	671		Dead	
	724	646	747	476					Dead				
	725	729	669	758	777	781	791	779	733	637		Dead	
	726	623	683	642	612				Dead				
	727	656	649	689	688	690				Dead			
	728	608	611	633	604	568	504				Dead		
	729	605	617	616	634	619	619	622	609	599	571	481	Dead
	730	611	632	615	610	600	594	585			Dead		
	731	615	624	647	642	636	525				Dead		
	732	542	652	656	652	640	632	639	604	396	537	528	503
	733	519	587	585	573	539	525				Dead		
	734	644	657	650	644	616	624	595	563			Dead	
	735	637	647	665	664	668	347				Dead		
	736	551	578	575	539				Dead				
	737	644	672	671	666	646	647	619			Dead		
	738	672	702	691	678	605	658	634	453			Dead	
	739	639	644	657	642	565	618	601	546	415		Dead	
	740	635	644	660	613				Dead				
	741	556	493					Dead					
	742	599	605	612	605	521	573	493			Dead		
	743	647	666	647	642	636	617	578			Dead		
	744	633	652	638	630	611	612				Dead		
	745	629	648	650	642	627	620	588			Dead		
	746	703	727	715	686	629	575				Dead		
	747	604	624	624	620	610	618	604	576			Dead	
	748	580	583	599	595	593	610	604	595	562	581	575	562
	749	726	758	759	731	731				Dead			
	750	701	714	712	678	638	631	638	613	533		Dead	
	751	597	610	611	615	616	605	602	587	569	521		Dead
	752	579	610	598	606	599	612	602			Dead		
	753	600	620	619	619	626	625	625	612	586	579		Dead
	754	580	594	573	531	513				Dead			
	755	586	585	590	587	590	581	566	542			Dead	
	756	773	732	773	772	741	760	742	635			Dead	
	757	634	639	599					Dead				
	758	637	635	630	637	623	642	631	596	574		Dead	
	759	623	622	619	613	578	575	503			Dead		
MEAN±SD		641±58	656±60	654±61	647±61	632±59	620±62	617±65	591±64	578±72	562±61	548±50	538±51

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 73-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
30	860	244	296	332	370	385	413	429	443	462	473	498	503	512
	861	233	284	317	339	353	371	390	400	413	432	445	447	452
	862	237	281	312	333	361	381	393	407	421	434	445	454	461
	863	238	299	345	369	387	406	425	441	460	471	488	494	499
	864	244	304	348	378	401	427	443	459	475	492	503	511	518
	865	248	291	318	339	359	369	383	398	419	424	443	447	450
	866	240	288	324	347	368	389	402	416	433	447	460	469	471
	867	230	282	325	344	370	384	404	410	424	441	446	437	450
	868	243	287	329	346	365	374	386	404	412	426	443	449	458
	869	240	276	315	334	347	359	373	387	396	406	413	417	429
	870	263	318	358	395	414	442	453	473	490	499	525	533	542
	871	252	297	339	366	384	401	416	437	447	456	464	476	478
	872	246	296	329	358	372	394	403	419	429	444	449	455	467
	873	242	288	331	361	385	412	424	439	457	473	484	486	482
	874	244	290	324	355	372	389	406	416	431	445	460	464	469
	875	241	237	319	343	361	377	388	406	426	439	452	459	464
	876	231	290	325	360	381	396	411	424	442	456	466	475	477
	877	226	280	325	356	379	397	405	429	442	451	466	474	483
	878	225	278	329	361	382	396	416	438	452	464	493	489	438
	879	235	238	328	360	375	392	412	427	452	474	489	492	492
	880	257				405				482				
	881	238				359				408				
	882	231				358				428				
	883	232				381				449				
	884	249				364				413				
	885	234				359				429				
	886	238				358				419				
	887	242				327				373				
	888	261				408				475				
	889	240				347				391				
	890	244				402				489				
	891	254				379				440				
	892	262				404				465				
	893	244				376				427				
	894	230				360				423				
	895	253				377				425				
	896	249				370				426				
	897	265				410				486				
	898	244				370				426				
	899	239				364				423				
	900	256				431				518				
	901	244				359				412				
	902	243				373				427				
	903	257				407				473				
	904	239				357				595				
	905	248				388				459				
	906	246				398				468				
	907	247				371				424				
	908	227				344				384				
	909	236				343				417				
	MEAN±SD	243±10	290±10	329±12	356±16	375±21	393±20	408±20	424±22	437±30	452±23	467±27	472±28	477±27

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	120	152	175	230	203	232	258	291	314	349	377
10	360	522	530	510	564	579	587	597	619	631	669	681	682	725
	361	438	459	473	497	519	527	528	540	548	568	583	580	589
	362	476	475	495	515	530	536	535	563	576	601	607	610	619
	363	516	518	514	555	569	567	572	599	614	633	644	622	634
	364	525	463	558	584	601	608	611	644	637	667	681	697	709
	365	453	541	482	503	522	525	527	532	566	592	605	607	613
	366	430	436	510	537	546	553	546	567	584	610	614	597	597
	367	457	464	467	483	511	513	504	532	511	562	559	582	596
	368	460	466	473	499	525	526	525	567	563	582	594	600	611
	369	428	435	449	465	484	492	486	504	514	536	545	549	553
	370	551	559	570	612	628	635	645	668	679	695	724	712	717
	371	487	489	503	514	537	545	550	564	577	603	612	622	639
	372	484	483	502	530	553	568	557	581	594	597	615	609	631
	373	495	510	550	554	573	588	575	595	585	607	601	600	603
	374	480	486	501	530	539	539	557	574	583	591	599	579	584
	375	479	487	503	523	530	536	545	570	580	595	617	594	615
	376	483	497	501	517	535	539	538	594	574	589	587	602	608
	377	495	501	524	535	556	568	563	587	599	622	635	635	639
	378	502	520	536	532	564	578	592	607	607	647	635	644	667
	379	511	528	547	579	590	597	598	619	614	644	652	478	Dead
	380		543		589			617	667	661	687	688	687	700
	381		460		489			512	532	547	562	565	563	567
	382		485		527			570	599	607	640	655	662	673
	383		506		547			578	586	593	610	609	581	574
	384		450		470			486	505	518	524	528	539	552
	385		490		517			556	563	582	607	623	613	628
	386		443		489			509	564	547	562	571	571	593
	387		418		454			474	504	508	527	538	544	549
	388		548		573			625	669	672	688	726	731	753
	389		437		459			482	503	512	528	554	549	564
	390		552		592			632	669	678	704	717	711	758
	391		496		532			565	593	605	624	632	639	643
	392		514		551			595	617	629	658	654	662	673
	393		485		524			538	544	553	575	576	560	560
	394		481		516			533	549	563	574	586	579	584
	395		483		504			563	569	576	584	599	601	607
	396		479		526			561	578	592	613	626	536	656
	397		555		597			625	628	641	644	666	625	645
	398		477		507			532	562	570	588	609	597	620
	399		467		493			506	527	535	555	537	555	560
	400		580		627			525	548	544	652	652	616	418
	401		461		506			631	637	643	574	577	577	589
	402		484		520			606	576	576	600	613	601	585
	403		525		571			552	517	643	655	684	672	682
	404		440		462			499	517	517	541	545	545	564
	405		528		552			531	509	522	541	553	542	549
	406		516		563			589	635	623	633	639	648	653
	407		481		522			564	588	616	635	654	651	686
	408		425		444			459	491	489	506	512	510	521
	409		452		489			528	547	564	579	596	583	604
	MEAN±SD	487±29	492±38	508±32	527±42	550±34	557±35	554±45	578±46	585±47	606±47	616±50	609±53	620±62

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	685	714	732
10	360	715	735	740	743	741	738	743	698	626	583	Dead	
	361	583	599	608	601	603	595	590	574	543	515	488	Dead
	362	624	633	642	633	641	642	636	658	617	628	632	Dead
	363	640	646	638	622	620	588	574	536	492	441	381	Dead
	364	722	754	761	761	768	760	754	750	732	732	707	Dead
	365	631	648	638	631	603	577	548	Dead				
	366	606	612	597	594	577	556	561	445	Dead			
	367	603	625	635	639	636	637	635	621	606	562	516	Dead
	368	605	634	646	642	652	639	631	560	524	404	Dead	
	369	556	575	580	578	552	Dead						
	370	706	726	721	674	599	Dead						
	371	639	662	670	672	666	667	678	693	701	704	695	Dead
	372	639	659	645	661	657	656	643	570	Dead			
	373	581	613	604	Dead								
	374	567	572	576	607	565	538	Dead					
	375	621	627	626	612	Dead							
	376	616	640	643	634	622	624	613	560	Dead			
	377	661	678	671	672	681	685	682	658	635	642	Dead	
	378	636	687	685	677	684	633	Dead					
	379	Dead											
	380	703	672	692	682	683	677	676	628	601	532	Dead	
	381	561	575	565	553	540	529	524	362	Dead			
	382	670	692	705	715	718	723	728	726	721	707	688	672
	383	573	583	561	517	Dead							
	384	548	570	568	577	583	581	581	576	575	584	593	585
	385	634	660	657	648	648	624	625	606	588	566	518	484
	386	522	503	607	637	602	589	Dead					
	387	562	592	592	598	600	601	608	618	619	635	739	Dead
	388	782	786	792	792	798	770	757	708	661	453	402	Dead
	389	565	573	577	571	530	583	582	566	530	527	512	500
	390	725	728	719	711	589	632	Dead					
	391	679	672	681	673	670	676	691	677	671	671	635	636
	392	672	700	678	679	664	663	677	662	639	557	434	Dead
	393	592	577	531	Dead								
	394	591	620	609	603	608	606	614	593	571	577	565	557
	395	624	644	621	624	621	598	611	Dead				
	396	671	662	585	507	483	444	Dead					
	397	634	641	633	628	593	Dead						
	398	633	671	682	678	694	682	691	617	439	Dead		
	399	560	583	565	557	567	544	542	511	483	361	Dead	
	900	Dead											
	901	592	620	627	642	642	646	651	643	646	629	Dead	
	902	588	598	584	Dead								
	903	711	717	710	710	699	694	682	647	559	Dead		
	904	536	578	585	592	578	579	577	566	576	556	540	533
	905	648	659	653	658	628	599	577	498	Dead			
	906	666	692	695	682	683	679	686	669	654	640	629	626
	907	688	713	730	745	749	751	751	752	753	758	666	650
	908	511	538	533	575	514	500	520	Dead				
	909	601	626	623	624	629	626	631	612	620	589	570	555
	MEAN±SD	626±60	643±55	639±60	641±61	636±66	628±70	636±66	611±85	607±77	583±99	578±110	580±65

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
10	810	257	309	354	383	414	433	453	479	487	505	525	534	547
	811	247	297	333	367	385	409	416	431	443	456	476	479	489
	812	237	280	312	343	362	376	381	399	410	417	436	444	451
	813	237	282	318	347	368	387	397	414	432	438	459	462	472
	814	272	329	375	412	432	454	463	489	504	519	521	524	551
	815	240	308	353	383	412	427	443	453	463	479	496	504	511
	816	234	284	318	350	363	376	392	414	425	438	452	456	466
	817	253	308	345	377	390	403	413	435	447	438	489	483	502
	818	250	298	332	360	377	395	409	430	443	456	476	484	483
	819	259	305	338	359	375	395	415	430	451	461	476	479	491
	820	212	274	330	370	383	414	421	437	453	460	476	484	496
	821	232	300	352	390	410	432	452	481	495	512	530	541	553
	822	247	301	346	375	399	423	442	449	461	464	487	495	501
	823	239	281	308	326	343	356	372	378	386	393	398	396	402
	824	252	302	347	384	414	436	447	462	476	493	514	530	533
	825	263	324	374	419	449	474	504	526	548	567	585	589	609
	826	249	303	337	358	375	397	419	429	442	453	471	470	483
	827	242	285	325	343	363	377	398	408	417	427	449	446	465
	828	238	289	325	352	366	380	409	411	423	437	454	457	467
	829	252	287	322	348	363	387	409	419	433	442	457	456	466
	830	238				335				368				
	831	259				393				459				
	832	259				394				443				
	833	254				368				430				
	834	243				404				479				
	835	234				366				422				
	836	254				418				474				
	837	242				396				473				
	838	246				381				426				
	839	258				403				463				
	840	228				316				358				
	841	260				401				487				
	842	247				378				432				
	843	256				416				480				
	844	246				381				444				
	845	248				388				462				
	846	250				407				497				
	847	263				412				489				
	848	252				416				454				
	849	258				393				487				
	850	238				361				413				
	851	240				369				427				
	852	252				390				439				
	853	243				372				423				
	854	250				373				429				
	855	230				366				414				
	856	253				427				516				
	857	246				391				451				
	858	243				362				425				
	859	244				386				436				
	MEAN±SD	247±11	297±15	337±19	367±24	386±25	406±30	423±32	439±35	449±36	464±40	481±40	485±48	497±45

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 73-	Days of Study and Body Weights in Grams												
		38	93	123	152	175	230	293	332	358	391	414	349	377
10	810	562	570	588	617	645	644	647	670	671	694	712	702	718
	811	503	511	519	548	562	566	572	583	606	620	634	636	643
	812	458	467	477	499	524	529	529	544	561	581	571	552	548
	813	483	490	499	521	534	545	545	561	573	596	599	597	621
	814	570	580	578	611	637	647	646	668	680	701	710	719	733
	815	531	532	548	570	606	608	610	642	667	672	666	705	706
	816	476	483	496	519	545	543	554	564	575	591	609	614	625
	817	512	511	535	555	568	583	576	596	613	623	616	577	577
	818	494	495	511	543	566	573	588	591	605	620	624	612	629
	819	500	499	524	544	557	563	570	598	609	614	619	632	653
	820	505	504	519	526	536	565	560	572	589	588	601	577	593
	821	577	572	595	624	642	640	645	665	581	682	700	703	712
	822	515	511	509	544	573	578	570	599	593	593	602	575	579
	823	416	418	430	442	458	455	451	462	489	500	513	507	515
	824	545	552	562	599	622	629	639	658	671	687	705	693	706
	825	623	634	672	706	732	734	749	783	800	787	830	801	824
	826	491	497	482	514	530	537	541	561	573	581	592	606	610
	827	477	477	500	505	527	536	535	561	572	575	581	588	592
	828	475	479	495	506	525	534	539	554	567	584	595	589	596
	829	481	485	481	521	542	539	540	565	577	583	580	566	547
	830		413		432			468	487	498	522	534	530	Dead
	831		527		576			619	646	663	685	700	702	738
	832		488		539			555	569	581	605	604	596	618
	833		490		530			561	583	597	623	636	630	652
	834		529		547			579	617	629	642	663	655	677
	835		479		520			549	566	590	601	612	593	596
	836		551		578			612	643	648	662	681	674	694
	837		532		583			597	622	620	637	632	623	609
	838		479		512			558	576	588	598	609	615	626
	839		529		564			594	606	615	629	636	591	591
	840		391		415			444	462	470	494	503	508	532
	841		536		599			649	672	685	708	711	698	714
	842		490		538			581	598	627	640	662	673	694
	843		526		571			610	623	643	648	670	639	663
	844		508		544			573	588	603	626	633	642	660
	845		534		571			599	623	637	657	669	662	684
	846		545		589			627	645	651	679	684	683	697
	847		552		603			646	669	678	685	698	664	674
	848		523		562			595	617	625	633	645	646	653
	849		556		606			627	646	668	685	693	697	706
	850		486		507			539	589	571	590	598	607	620
	851		469		525			563	592	586	621	625	638	634
	852		485		511			506	536	Dead				
	853		473		503			525	539	539	568	575	556	569
	854		482		515			533	568	550	562	576	587	590
	855		485		527			560	580	592	623	635	643	658
	856		562		611			632	669	671	677	697	681	700
	857		499		545			572	589	605	634	631	636	644
	858		488		511			537	580	559	584	594	569	562
	859		507		544			580	595	594	610	613	601	590
	MEAN±SD	510±47	508±43	526±53	546±50	572±60	577±60	575±54	598±56	609±57	625±55	635±59	628±59	641±62

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	685	714	732
10	310	726	749	728	730	724	696	685	556	Dead			
	311	645	669	664	668	662	664	698	325	359	Dead		
	312	545	576	Dead									
	313	621	640	641	677	641	632	635	602	530	Dead		
	314	735	767	779	773	768	765	752	720	645	673	643	633
	315	711	747	743	738	741	729	734	721	629	637	636	Dead
	316	590	622	625	606	587	580	Dead					
	317	545	432	Dead									
	318	613	638	636	632	649	646	634	630	549	527	338	Dead
	319	649	657	661	638	670	663	633	617	567	553	513	472
	320	568	474	Dead									
	321	725	734	726	731	710	683	667	620	489	Dead		
	322	564	573	Dead									
	323	516	542	543	532	539	529	541	517	451	446	309	Dead
	324	711	727	701	711	689	649	596	Dead				
	325	824	842	846	832	825	814	780	756	Dead			
	326	610	625	627	551	451	Dead						
	327	593	601	590	583	570	575	550	Dead				
	328	601	621	621	617	582	Dead						
	329	530	520	488	494	410	378	521	505	Dead			
	330	Dead											
	331	756	764	749	Dead								
	332	620	639	640	624	636	629	Dead					
	333	638	671	637	636	470	Dead						
	334	680	674	611	516	Dead							
	335	603	540	419	Dead								
	336	744	736	732	716	750	753	767	720	Dead			
	337	593	588	541	Dead								
	338	619	637	617	601	Dead							
	339	590	595	Dead									
	340	527	548	557	569	559	Dead						
	341	703	708	671	633	Dead							
	342	693	728	725	739	732	745	740	712	643	Dead		
	343	644	665	645	627	Dead							
	344	658	680	690	685	672	682	681	671	644	636	634	600
	345	682	700	690	672	657	647	618	Dead				
	346	699	720	735	759	Dead							
	347	668	673	646	500	Dead							
	348	642	675	661	641	634	618	614	585	511	Dead		
	349	702	705	689	675	625	438	Dead					
	350	617	640	638	643	630	618	612	567	532	555	556	531
	351	663	664	676	677	670	660	641	607	497	483	447	Dead
	352	Dead											
	353	560	580	553	501	Dead							
	354	572	591	581	560	542	519	Dead					
	355	667	696	694	693	671	643	514	Dead				
	356	691	718	722	711	690	598	Dead					
	357	654	670	680	679	679	677	Dead					
	358	565	565	552	Dead								
	359	590	604	595	573	Dead							
	MEAN±SD	639±68	649±81	651±80	647±80	640±93	649±81	650±79	643±89[3]	580±106	564±80	516±125	539±71

[3]Statistically significantly different from the control mean by Dunnett's test, $p<0.05$.

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		0	6	13	20	25	32	39	46	53	60	68	75	81
3	760	227	276	324	359	377	395	417	431	448	459	485	492	497
	761	256	308	346	386	406	414	436	444	460	465	479	492	488
	762	238	276	313	342	356	364	376	394	405	409	422	431	425
	763	219	268	300	325	339	343	359	374	398	407	412	434	436
	764	248	299	352	381	405	428	443	455	471	484	502	506	511
	765	227	263	299	320	339	349	349	380	392	399	415	419	423
	766	251	292	330	354	372	380	400	417	429	438	451	458	465
	767	248	291	329	361	384	407	422	439	452	462	484	486	494
	768	260	307	334	392	408	421	438	459	476	489	513	518	522
	769	233	301	361	402	434	454	460	487	503	522	541	546	552
	770	246	286	325	349	375	394	410	420	429	440	452	461	467
	771	227	270	311	342	365	391	398	416	425	438	459	472	472
	772	257	300	345	381	397	410	427	443	473	477	499	499	507
	773	233	274	317	341	360	376	390	405	425	430	444	452	460
	774	235	271	310	338	363	368	373	387	398	408	428	428	437
	775	252	290	343	368	388	399	409	430	451	456	473	479	490
	776	230	278	321	352	371	388	404	422	454	450	471	474	475
	777	229	276	329	360	391	411	431	451	474	484	505	515	523
	778	256	309	362	389	412	429	437	456	467	482	498	503	511
	779	247	299	344	374	404	410	433	460	472	489	518	525	527
	780	257				343				399				
	781	236				420				502				
	782	269				415				478				
	783	251				403				470				
	784	225				342				381				
	785	250				395				458				
	786	264				437				516				
	787	259				415				475				
	788	258				388				452				
	789	239				383				433				
	790	228				348				384				
	791	253				404				472				
	792	268				413				486				
	793	255				390				439				
	794	235				339				381				
	795	228				358				403				
	796	230				363				420				
	797	253				394				464				
	798	225				382				430				
	799	222				346				392				
	800	248				384				432				
	801	237				393				463				
	802	256				384				432				
	803	243				383				450				
	804	257				414				481				
	805	240				392				453				
	806	247				381				444				
	807	241				390				449				
	808	254				412				485				
	809	238				408				483				
	MEAN±SD	244±13	287±14	331±19	361±23	385±25	397±28	411±30	428±30	447±34	454±33	472±37	479±37	484±36

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	123	152	175	230	203	232	258	291	314	349	377
3	760	504	515	534	561	582	590	593	625	630	662	678	677	680
	761	502	512	545	561	587	588	584	609	614	623	636	599	593
	762	433	437	443	469	484	481	482	503	503	523	527	526	532
	763	440	452	478	515	534	537	541	556	562	587	604	600	612
	764	528	524	532	544	534	561	560	583	596	621	625	624	640
	765	437	445	437	473	489	490	497	510	523	542	546	547	556
	766	471	474	473	512	524	534	533	546	558	582	585	578	588
	767	504	504	524	547	552	554	550	576	592	612	586	610	619
	768	533	538	557	604	624	622	639	704	676	697	724	706	731
	769	563	562	585	612	633	643	545	668	678	676	703	646	679
	770	471	485	516	526	559	567	569	586	602	627	635	634	650
	771	483	490	512	524	538	541	545	537	566	571	581	581	583
	772	520	523	550	569	584	589	606	632	621	651	646	653	651
	773	464	471	482	501	512	517	520	533	550	574	581	582	600
	774	446	450	458	484	497	501	512	529	541	578	599	612	635
	775	492	508	513	538	560	558	567	585	580	597	601	574	591
	776	487	493	521	539	549	546	558	566	586	610	619	611	Dead
	777	528	534	572	587	601	619	607	620	629	644	642	616	625
	778	517	517	526	552	564	566	571	600	623	622	635	635	639
	779	535	537	563	580	603	603	613	619	645	664	668	669	672
	780		454		485			509	527	538	553	562	556	575
	781		574		642			680	726	736	754	778	771	809
	782		547		596			622	646	654	682	696	684	689
	783		534		573			519	611	628	656	664	659	672
	784		431		468			489	525	524	541	544	544	561
	785		529		582			622	643	656	684	701	679	703
	786		583		645			672	690	712	727	752	745	771
	787		549		597			622	639	653	678	682	673	663
	788		511		568			597	613	628	663	665	668	687
	789		487		533			552	572	589	596	622	618	639
	790		426		462			484	509	519	531	538	536	549
	791		522		549			580	604	618	628	646	633	622
	792		560		623			639	667	707	717	722	704	732
	793		496		545			557	583	595	612	604	607	629
	794		418		462			481	503	514	524	531	533	553
	795		458		492			510	538	551	560	568	568	584
	796		479		516			539	562	573	598	602	631	616
	797		529		598			637	658	679	695	710	724	731
	798		506		549			574	588	606	618	617	556	595
	799		453		485			508	517	532	554	564	599	561
	800		471		490			527	547	574	575	599	608	627
	801		523		557			586	615	634	643	646	635	651
	802		477		505			526	543	558	570	579	573	586
	803		509		575			608	627	642	672	686	675	697
	804		545		581			583	599	593	588	574	542	496
	805		508		553			530	599	626	632	639	648	667
	806		499		548			564	597	590	603	620	606	613
	807		500		548			580	607	617	666	648	644	679
	808		544		584			621	633	661	668	682	669	695
	809		563		615			638	661	658	686	696	695	709
	MEAN±SD	493±37	503±41	517±40	546±48	556±42	560±44	570±51	593±54	605±53	623±56	631±59	625±57	638±64

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APPENDIX 4 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	683	714	732
3	760	675	690	681	666	648	638	613	568	525	Dead		
	761	570	569	428	Dead								
	762	550	558	575	565	569	574	560	566	558	556	522	508
	763	636	697	607	Dead								
	764	663	684	686	677	681	692	681	679	682	682	662	Dead
	765	563	590	584	583	572	549	Dead					
	766	594	626	622	624	583	573	512	Dead				
	767	630	644	633	634	597	Dead						
	768	754	732	709	704	694	680	542	Dead				
	769	Dead											
	770	663	687	690	688	667	672	621	615	607	586	555	Dead
	771	595	632	625	617	623	630	530	613	613	608	597	593
	772	673	693	686	677	656	570	Dead					
	773	603	618	622	608	610	614	618	605	581	570	561	544
	774	650	681	709	721	728	745	723	721	708	678	619	468
	775	585	584	522	Dead								
	776	Dead											
	777	614	616	571	Dead								
	778	639	658	655	620	622	608	547	Dead				
	779	676	684	686	664	672	641	611	Dead				
	780	584	553	579	564	583	571	556	Dead				
	781	314	339	333	343	328	788	Dead					
	782	677	690	671	662	633	615	541	Dead				
	783	675	700	693	669	Dead							
	784	566	589	579	576	580	590	575	592	597	599	617	610
	785	717	733	712	685	653	632	Dead					
	786	777	784	790	780	793	Dead						
	787	663	682	668	619	Dead							
	788	690	712	715	705	705	713	673	647	640	558	429	Dead
	789	652	674	670	631	640	629	594	511	Dead			
	790	552	580	607	569	569	567	570	543	508	Dead		
	791	626	634	594	502	Dead							
	792	695	702	704	663	588	Dead						
	793	627	632	650	652	641	647	639	611	605	572	Dead	
	794	555	580	583	578	582	589	585	572	576	564	554	547
	795	579	609	598	606	618	607	605	601	590	586	581	565
	796	619	647	660	658	653	643	632	615	606	521	Dead	
	797	741	740	749	735	714	718	690	Dead				
	798	601	552	Dead									
	799	560	577	592	591	599	603	623	652	Dead			
	800	630	658	667	671	673	681	691	676	676	701	751	825
	801	646	660	647	639	620	578	Dead					
	802	580	601	601	582	590	593	578	559	541	Dead		
	803	689	708	698	696	683	686	673	Dead				
	804	Dead											
	805	676	708	724	726	696	666	693	667	635	609	542	Dead
	806	630	634	639	625	614	587	483	Dead				
	807	691	709	714	713	715	699	716	694	689	690	668	625
	808	709	735	736	720	716	699	693	671	660	602	Dead	
	809	673	729	742	725	730	716	716	691	635	451	Dead	
	MEAN±SD	643±61	660±64	654±72	654±65	650±62	639±58	619±65	621±56	612±55	596±64	589±79	587±102

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APPENDIX 5

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		0	5	13	20	26	32	39	46	53	60	68	75	81
0	910	203	239	246	263	283	282	278	282	288	289	300	306	304
	911	209	239	247	259	270	280	278	289	293	297	304	300	310
	912	195	218	234	250	249	266	272	281	282	291	299	296	301
	913	192	204	222	232	233	245	255	251	258	259	262	272	264
	914	201	214	232	243	244	257	258	263	277	277	284	286	283
	915	202	213	235	244	252	260	269	273	282	283	293	292	292
	916	200	223	239	259	269	282	282	301	295	311	315	320	323
	917	204	225	253	274	272	286	288	297	309	309	320	322	324
	918	192	214	232	241	248	257	257	273	277	276	287	286	286
	919	194	216	232	249	259	276	273	280	275	279	292	293	302
	920	202	224	232	252	254	265	270	255	300	283	294	290	291
	921	187	208	219	229	229	238	246	274	254	260	266	265	267
	922	198	219	238	253	250	260	269	271	298	298	299	295	304
	923	199	208	230	242	240	250	258	255	283	255	273	275	279
	924	189	212	224	238	237	247	252	259	260	267	274	272	277
	925	198	210	231	221	235	248	259	257	266	266	272	274	277
	926	187	206	220	226	229	236	249	273	258	260	266	263	267
	927	195	218	235	259	250	253	272	246	278	283	290	288	292
	928	190	205	231	241	244	252	256	230	283	288	294	300	306
	929	200	233	246	244	255	262	278	280	298	294	300	298	308
	930	217				270				303				
	931	197				253				278				
	932	192				249				273				
	933	195				244				270				
	934	201				257				281				
	935	201				249				269				
	936	197				254				279				
	937	190				250				278				
	938	190				242				272				
	939	189				235				249				
	940	197				237				275				
	941	195				249				275				
	942	193				270				270				
	943	209				256				283				
	944	198				253				277				
	945	193				249				268				
	946	180				228				243				
	947	185				240				269				
	948	191				250				272				
	949	193				258				299				
	950	203				278				283				
	951	188				221				229				
	952	199				262				296				
	953	192				250				267				
	954	195				252				273				
	955	190				229				257				
	956	193				241				265				
	957	191				253				267				
	958	197				244				260				
	959	190				249				275				
	960	187				241				285				
	961	190				252				273				
	962	184				230				243				
	963	196				258				297				
	964	204				296				304				
	965	194				262				279				
	966	200				260				291				
	967	192				270				286				
	968	197				253				273				
	969	203				264				284				
	970	195				257				300				
	971	191				255				284				
	972	198				244				261				
	973	188				230				263				
	974	197				257				273				
	975	207				272				296				
	976	196				249				275				
	977	189				250				288				
	978	201				270				298				
	979	192				240				263				
	980	206				236				317				
	981	201				255				273				
	982	211				258				292				
	983	185				238				282				
	984	193				253				281				
	985	188				248				284				
	986	202				282				296				
	987	200				276				300				
	988	200				272				297				
	989	200				251				270				
	990	192				245				286				
	991	188				249				273				
	992	186				242				263				
	993	194				260				255				
	994	190				250				296				
	995	182				243				269				
	MEAN±SD	195±7	217±10	234±9	246±14	253±14	261±15	266±12	272±15	278±16	281±16	289±16	290±16	293±18

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	123	152	175	150	203	232	258	291	314	349	377
0	910	305	310	321	331	350	360	353	372	384	404	402	418	416
	911	313	306	322	329	345	343	339	355	360	385	383	364	366
	912	299	308	316	320	334	342	343	351	359	358	367	369	378
	913	270	266	280	283	300	295	302	310	320	321	326	333	339
	914	292	298	308	312	333	336	338	353	367	369	367	362	376
	915	303	299	305	311	323	321	321	332	332	346	350	351	364
	916	321	323	337	347	360	360	346	361	367	374	377	380	392
	917	329	331	342	377	370	373	388	386	406	441	444	449	459
	918	296	295	292	307	311	312	310	322	326	326	329	332	338
	919	289	311	324	322	343	340	358	358	379	393	390	386	399
	920	305	302	314	318	332	333	328	340	343	354	349	349	353
	921	275	273	291	288	300	297	308	307	301	313	321	323	334
	922	316	304	334	330	361	363	361	382	388	397	407	398	405
	923	290	282	293	298	311	310	305	321	317	328	337	322	338
	924	283	296	291	296	314	307	309	320	314	322	325	324	323
	925	289	285	287	301	311	311	305	324	324	336	369	380	395
	926	277	278	285	278	289	286	303	305	300	309	312	325	321
	927	303	297	306	309	324	327	328	337	342	346	350	340	358
	928	309	312	311	319	337	338	333	344	339	354	356	352	361
	929	310	318	333	331	345	349	341	355	353	376	367	393	395
	930		325		342			324	376	369	384	397	385	407
	931		301		310			369	346	336	361	358	357	362
	932		295		317			330	345	346	358	366	365	367
	933		297		307			328	342	336	346	365	355	357
	934		304		313			327	338	348	363	369	373	388
	935		292		306			293	318	321	330	337	331	334
	936		315		320			340	337	347	359	361	363	368
	937		306		314			333	353	344	360	353	371	375
	938		297		310			327	339	330	347	348	351	353
	939		256		265			286	300	308	319	326	326	342
	940		279		301			314	332	346	344	348	361	381
	941		302		316			323	334	341	201	Dead		
	942		299		324			324	340	343	393	396	390	398
	943		308		342			340	377	373	384	381	402	434
	944		300		314			322	340	344	348	349	354	354
	945		292		301			308	317	312	323	326	325	333
	946		265		277			290	297	297	307	318	316	322
	947		289		302			313	324	323	335	331	327	327
	948		295		314			335	345	354	362	370	381	393
	949		325		352			377	403	387	398	400	412	443
	950		316		310			340	338	350	361	368	385	387
	951		255		270			279	289	292	315	319	321	327
	952		311		329			343	349	350	361	362	376	386
	953		297		312			331	328	322	342	337	342	342
	954		299		311			325	328	333	341	340	345	350
	955		297		300			309	327	314	325	320	322	328
	956		293		308			316	318	318	329	333	335	336
	957		294		310			317	328	321	337	341	337	346
	958		293		300			305	320	328	323	337	328	334
	959		304		315			322	333	335	347	363	386	422
	960		294		343			329	371	383	370	372	377	389
	961		322		309			366	335	343	381	387	388	398
	962		263		279			291	305	310	314	317	320	326
	963		313		326			361	385	397	416	430	434	454
	964		319		346			Dead						
	965		307		325			343	355	351	363	362	364	366
	966		313		319			336	340	359	375	360	362	371
	967		318		338			344	354	355	366	362	364	360
	968		313		337			360	371	392	398	409	398	430
	969		313		327			341	352	356	368	380	369	370
	970		315		341			352	373	384	399	394	408	435
	971		310		324			332	342	345	382	380	387	397
	972		290		303			299	314	331	335	343	335	336
	973		285		296			305	311	315	324	326	326	328
	974		301		317			327	327	333	355	334	367	370
	975		322		341			350	366	370	381	380	384	393
	976		300		312			315	324	333	329	339	326	338
	977		315		338			354	368	363	389	392	402	413
	978		333		343			346	364	350	363	371	368	377
	979		290		305			314	326	334	338	347	340	343
	980		346		358			374	380	388	387	356	339	336
	981		303		312			324	325	329	340	339	346	343
	982		317		332			363	388	371	385	384	379	385
	983		279		297			305	317	314	324	325	321	323
	984		307		317			323	351	353	357	367	333	363
	985		287		296			301	312	321	329	337	336	343
	986		329		333			331	385	390	392	416	433	434
	987		324		347			379	377	381	393	392	392	401
	988		317		334			350	353	353	331	380	401	409
	989		288		306			321	327	327	340	347	362	365
	990		294		311			294	330	332	343	349	372	335
	991		305		323			325	339	362	375	396	405	392
	992		286		293			288	305	316	317	312	330	323
	993		309		253			232	204	325	234	343	346	362
	994		303		321			311	350	369	373	387	410	416
	995		299		317			317	355	360	373	377	382	397
	MEAN±SD	299±16	301±17	310±19	315±21	330±22	330±25	328±26	340±28	343±26	354±33	361±28	364±31	372±35

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APPENDIX 5 (Concinued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	685	714	732
0	910	422	476	479	520	567	Dead						
	911	359	377	379	366	373	392	385	378	381	Dead		
	912	376	392	414	420	407	413	419	417	418	418	406	398
	913	371	351	360	370	371	392	380	378	399	397	392	385
	914	382	395	392	390	393	407	399	397	413	436	434	431
	915	364	383	388	397	412	446	463	477	467	461	442	382
	916	384	408	409	419	Dead							
	917	474	498	494	516	523	567	601	Dead				
	918	335	347	350	353	357	357	349	352	356	360	367	362
	919	403	423	447	453	482	515	569	Dead				
	920	342	353	361	357	352	361	355	354	374	404	423	430
	921	354	376	420	Dead								
	922	418	426	444	473	484	541	Dead					
	923	340	352	359	356	363	375	374	Dead				
	924	327	341	353	360	359	327	291	Dead				
	925	406	418	429	453	463	485	513	586	695	Dead		
	926	311	331	339	319	322	328	327	326	318	317	307	292
	927	377	415	428	422	432	448	450	443	438	426	408	387
	928	364	368	351	349	323	Dead						
	929	397	419	425	439	446	445	440	426	430	433	425	412
	930	417	432	440	437	440	439	472	475	503	542	683	788
	931	358	376	372	371	369	384	396	405	426	506	Dead	
	932	359	402	413	403	405	423	433	467	489	582	610	568
	933	377	410	409	418	436	446	455	457	444	458	476	Dead
	934	408	445	Dead									
	935	349	360	355	356	348	363	363	363	373	420	488	490
	936	368	388	391	365	391	406	415	416	419	413	422	431
	937	385	399	390	392	426	419	458	548	Dead			
	938	347	367	391	449	497	Dead						
	939	341	359	385	384	389	405	409	398	404	436	475	455
	940	372	405	369	405	414	439	435	425	Dead			
	941	Dead											
	942	405	416	424	450	429	454	469	455	447	439	432	399
	943	426	446	441	457	457	475	460	487	483	515	539	563
	944	356	366	382	397	371	381	389	392	395	409	401	Dead
	945	337	352	388	427	Dead							
	946	316	336	351	350	356	375	383	379	385	415	446	473
	947	330	338	338	340	339	349	343	339	334	325	340	328
	948	397	426	443	463	464	487	495	488	504	499	596	587
	949	Dead											
	950	409	431	444	468	470	531	Dead					
	951	336	355	362	370	381	381	381	401	394	395	407	408
	952	388	394	393	425	438	420	439	435	407	407	460	462
	953	345	366	367	371	389	391	395	429	497	Dead		
	954	346	359	364	376	398	417	427	449	451	497	577	619
	955	Dead											
	956	341	355	382	373	386	470	587	Dead				
	957	336	346	346	357	350	Dead						
	958	333	371	392	407	398	399	425	425	440	470	Dead	
	959	463	512	383	Dead								
	960	388	410	453	464	470	466	424	Dead				
	961	396	408	412	422	417	414	423	425	451	561	629	667
	962	327	353	360	372	350	363	365	367	362	320	Dead	
	963	462	499	513	516	507	515	523	Dead				
	964	Dead											
	965	365	374	374	375	378	378	386	375	372	373	371	362
	966	379	400	416	413	418	436	437	448	441	454	524	578
	967	364	383	380	367	Dead							
	968	416	452	440	450	447	455	447	438	426	420	Dead	
	969	371	380	395	424	477	553	Dead					
	970	436	491	545	Dead								
	971	391	409	422	423	422	420	437	433	427	439	439	442
	972	325	347	348	356	352	349	424	398	401	398	413	448
	973	325	351	348	350	344	335	334	316	306	300	297	297
	974	355	385	392	405	420	489	Dead					
	975	388	409	413	411	408	421	427	390	297	Dead		
	976	335	338	322	315	Dead							
	977	425	460	454	461	468	492	537	612	Dead			
	978	360	376	335	Dead								
	979	340	352	352	339	341	347	352	347	356	377	423	Dead
	980	390	405	406	401	410	421	445	469	526	567	563	374
	981	346	363	366	373	364	368	373	377	376	369	350	352
	982	382	398	395	391	381	394	390	386	363	381	Dead	
	983	317	338	335	331	322	329	333	333	349	397	Dead	
	984	367	390	413	397	393	405	406	430	409	312	Dead	
	985	347	360	372	370	373	383	378	387	405	472	550	381
	986	401	406	416	405	392	367	384	389	385	388	433	445
	987	403	410	417	415	414	390	407	403	414	431	430	461
	988	407	423	451	480	461	507	536	633	746	Dead		
	989	362	379	382	389	391	401	401	418	415	431	489	534
	990	385	451	513	Dead								
	991	390	412	424	453	431	448	438	442	432	436	489	504
	992	325	336	344	359	346	352	363	341	356	338	305	291
	993	382	384	355	351	357	342	350	303	305	299	288	287
	994	413	445	462	493	Dead							
	995	392	419	432	426	427	435	445	463	459	493	520	541
	MEAN±SD	373±36	393±42	400±50	403±47	406±51	418±58	421±63	420±67	420±79	425±69	451±92	458±112

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
10	1096	178	200	217	232	233	240	241	248	249	253	259	261	263
	1097	199	222	236	249	253	263	268	271	282	279	285	285	285
	1098	192	210	229	226	245	249	259	264	265	257	272	272	276
	1099	204	224	242	248	251	265	269	273	284	283	298	298	298
	1100	204	227	240	247	255	257	265	264	271	279	288	281	285
	1101	206	216	236	248	256	263	275	277	285	286	302	294	290
	1102	192	213	232	244	247	257	261	271	275	279	287	288	287
	1103	186	206	211	232	232	239	243	250	251	252	264	263	266
	1104	185	197	216	225	229	241	242	248	251	249	261	262	258
	1105	191	218	232	248	250	259	264	265	267	271	283	289	287
	1106	200	216	228	233	236	248	254	249	255	257	265	265	278
	1107	197	214	225	240	243	253	263	273	275	275	281	284	285
	1108	182	199	217	235	238	238	239	250	253	257	263	266	267
	1109	191	210	236	247	250	267	272	279	284	291	296	301	300
	1110	197	216	233	242	248	257	260	270	269	276	284	285	264
	1111	187	204	219	227	230	237	243	244	248	261	260	257	283
	1112	200	221	241	255	264	266	273	281	292	295	303	307	306
	1113	204	230	251	254	255	265	271	270	272	276	281	293	295
	1114	218	238	248	261	262	276	275	275	284	284	291	289	296
	1115	185	201	214	218	231	237	245	257	255	256	264	261	270
	1116	188				241				259				
	1117	190				236				265				
	1118	213				267				300				
	1119	190				241				257				
	1120	188				230				253				
	1121	202				245				270				
	1122	200				245				260				
	1123	193				226				257				
	1124	200				248				270				
	1125	196				249				283				
	1126	195				234				250				
	1127	189				231				247				
	1128	193				244				263				
	1129	207				261				282				
	1130	207				261				291				
	1131	196				236				249				
	1132	186				231				251				
	1133	193				260				286				
	1134	184				251				274				
	1135	176				222				238				
	1136	196				242				255				
	1137	184				232				253				
	1138	184				228				246				
	1139	195				242				268				
	1140	184				235				261				
	1141	200				258				230				
	1142	196				241				258				
	1143	190				238				265				
	1144	214				265				238				
	1145	189				239				258				
	MEAN±SD	194±9	214±11	230±11	240±12	244±12[a]	254±12	259±13	264±12	266±15[a]	271±14	279±15	280±16	282±14

[a]Statistically significant deviation from control mean using Dunnett's test, $p<0.05$.

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	123	152	175	180	203	232	258	291	314	349	377
30	1096	258	268	279	279	296	297	293	300	323	Dead			
	1097	293	294	316	320	342	333	342	357	362	382	379	352	408
	1098	281	275	293	303	314	323	323	327	341	337	338	344	342
	1099	302	309	318	331	344	354	355	376	365	375	376	382	379
	1100	290	289	296	307	322	312	320	329	320	339	339	347	336
	1101	300	305	315	323	334	333	327	344	339	353	359	360	367
	1102	294	302	314	315	324	330	327	335	330	338	333	339	342
	1103	275	270	284	287	302	304	294	312	314	319	337	335	358
	1104	268	265	288	285	294	294	293	287	283	297	298	282	299
	1105	290	290	301	312	325	323	315	348	327	355	334	331	342
	1106	282	278	287	291	307	304	314	330	332	332	333	345	351
	1107	287	287	303	306	312	319	318	329	333	344	342	331	346
	1108	271	276	288	308	310	306	314	343	332	346	341	364	366
	1109	304	315	318	328	332	341	347	347	357	365	366	367	375
	1110	290	291	302	309	319	314	323	327	327	335	327	338	322
	1111	264	265	273	283	295	294	298	311	317	325	333	334	336
	1112	313	316	323	332	354	350	345	363	372	385	384	394	410
	1113	302	302	321	317	326	331	328	336	338	352	344	354	360
	1114	302	300	326	329	345	342	332	348	394	381	389	398	402
	1115	275	270	322	290	291	306	313	308	319	320	319	330	331
	1116		277		296			301	311	313	313	324	340	333
	1117		289		311			328	332	344	345	350	357	356
	1118		333		355			384	400	425	434	436	455	480
	1119		277		299			316	321	323	341	347	352	353
	1120		270		299			301	314	305	320	295	330	324
	1121		292		305			330	344	344	361	362	373	397
	1122		281		288			304	307	319	321	323	313	310
	1123		279		297			303	320	328	344	350	356	367
	1124		292		306			321	324	331	339	338	344	341
	1125		306		330			335	342	355	384	387	392	397
	1126		271		289			297	312	314	334	328	334	Dead
	1127		269		291			308	314	322	324	331	339	334
	1128		287		302			321	323	341	350	338	345	358
	1129		303		324			353	343	344	366	357	359	361
	1130		311		330			341	377	380	378	378	395	393
	1131		276		296			312	326	344	352	359	376	389
	1132		277		289			349	311	314	331	324	335	330
	1133		320		341			302	372	369	395	391	389	396
	1134		304		310			325	328	334	343	345	344	359
	1135		250		270			282	286	287	303	299	304	308
	1136		281		295			333	313	308	319	318	322	317
	1137		280		307			322	322	322	344	352	355	352
	1138		265		270			301	311	326	352	350	381	383
	1139		292		312			326	343	363	377	373	Dead	
	1140		281		301			327	336	329	329	339	363	381
	1141		312		334			371	364	354	374	388	395	388
	1142		282		296			299	309	304	322	325	326	334
	1143		293		315			365	334	342	347	350	359	359
	1144		306		331			341	349	347	365	378	394	385
	1145		269		292			304	307	317	320	317	333	335
	MEAN±SD	287±15	288±17[a]	303±17	307±18	319±19	320±18	322±22	331±23	335±26	347±26	347±27[a]	354±29	359±33

[a]Statistically significant deviation from control mean using Dunnett's test, $p<0.05$.

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	685	714	732
30	1096						Dead						
	1097	415	433	448	456	467	471	474	490	511	529	511	536
	1098	361	361	358	374	360	353	358	348	353	349	353	359
	1099	385	407	411	424	411	399	343			Dead		
	1100	336	348	345	344	352	345	350	343	351	370	382	395
	1101	370	403	469					Dead				
	1102	342	357	364	374	366	370	386	398	448	504	583	544
	1103	339	364	371	380	399	434			Dead			
	1104	299	311	324	341	326	327	338	340	361	371	396	407
	1105	334	349	355	347	350	366	389	375			Dead	
	1106	333	353	362	356	384	407	448	534	634		Dead	
	1107	343	378	399	442				Dead				
	1108	375	400	401	403	396	414	425	421	421	420	429	431
	1109	379	399	394	397	404	415	437	486			Dead	
	1110	332	343	346	348	300	355	365	358	367	397	363	Dead
	1111	336	354	373	378	393	393	364			Dead		
	1112	410	412	432	454	438	454	464	458	467	435	465	457
	1113	351	357	355	356	366	373	378			Dead		
	1114	399	435	429	367	445	458	468	376			Dead	
	1115	333	365	359	435	373	384	390	377	386	405	441	471
	1116	335	375	375	407	397	386	415	396	412	431	441	440
	1117	356	373	372	373	374	380	383	387	386	394	399	415
	1118	493	525	548	631				Dead				
	1119	356	374	373	365	364	378	373	374	366	363	372	364
	1120	326	335	311					Dead				
	1121	393	412	411	460	442	452	459	457	443	468	474	479
	1122	292	340	399					Dead				
	1123	368	389	382	385	396	414	437	464	502	580	627	657
	1124	340	358	354	356	365	351	359	357	356	351	350	329
	1125	412	441	448	453	468	477	486	495	460	449		Dead
	1126							Dead					
	1127	336	359	360	366	366	367	380	371	383		Dead	
	1128	361	389	421	400	408	426	438	469	490	584	738	Dead
	1129	306	299						Dead				
	1130	399	409	429	423	428	423	436	434	448	473	510	512
	1131	428	443	443	470	486	497	530	577	576		Dead	
	1132	341	357	362	365	375	382	381	381	382	363	365	362
	1133	399	412	428	428	439	430	450	454	458	458	496	494
	1134	355	361	381	350	371	368	369	362	362	351	346	339
	1135	310	315	318	325	329	348	353	415			Dead	
	1136	356	331	326	339	329				Dead			
	1137	315	376	381	415	467				Dead			
	1138	398	429	426	440	459	481	482	493	493	480	447	471
	1139							Dead					
	1140	405	457						Dead				
	1141	384	398	427	432	443	440	443			Dead		
	1142	344	372	394	456	408	439			Dead			
	1143	335	389	389	398	413	422	428	427	437	451	466	454
	1144	383	406	407	410	415	422	427	434	467	465	509	561
	1145	326	350	352	342	346	349	370	337	364	359	371	365
	MEAN±SD	360±39	381±42	389±45	399±55	395±45	404±44	411±50	420±61	432±71	433±69	431±96	447±83

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 73-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
10	1046	216	232	247	258	263	270	275	281	282	287	299	299	299
	1047	182	208	223	238	241	245	256	255	252	259	262	269	272
	1048	203	219	234	247	256	264	264	276	275	278	287	289	292
	1049	198	226	251	266	273	285	289	296	300	308	315	320	323
	1050	203	222	242	255	260	265	263	273	278	280	290	286	293
	1051	200	216	238	253	254	258	267	268	270	273	289	289	295
	1052	197	211	237	253	251	263	269	262	278	288	299	297	314
	1053	198	219	232	250	257	266	263	270	266	274	272	281	280
	1054	216	233	252	268	278	286	296	299	303	317	323	322	330
	1055	179	194	216	234	243	252	266	267	272	276	284	285	297
	1056	192	205	223	235	241	247	248	259	270	274	278	278	285
	1057	199	213	239	252	250	258	267	272	281	285	290	294	296
	1058	198	217	236	248	258	261	264	275	279	283	291	289	295
	1059	209	227	241	251	253	260	265	272	273	279	287	287	283
	1060	215	226	246	254	266	273	273	277	286	287	293	299	303
	1061	200	212	228	239	238	239	249	250	253	260	263	272	273
	1062	204	226	242	261	265	268	271	278	275	290	296	300	299
	1063	191	216	230	245	252	256	263	263	266	275	284	281	288
	1064	191	207	217	221	228	231	239	240	251	260	262	269	274
	1065	191	213	230	255	257	265	267	274	275	285	295	304	301
	1066	203				269				282				
	1067	186				244				265				
	1068	219				272				291				
	1069	201				249				267				
	1070	208				268				284				
	1071	191				238				266				
	1072	197				229				243				
	1073	197				254				280				
	1074	204				258				279				
	1075	205				273				286				
	1076	197				258				275				
	1077	193				257				276				
	1078	199				267				288				
	1079	183				239				253				
	1080	187				238				266				
	1081	193				267				292				
	1082	184				232				253				
	1083	194				249				263				
	1084	222				282				299				
	1085	178				230				241				
	1086	184				240				257				
	1087	186				225				246				
	1088	189				257				282				
	1089	201				261				278				
	1090	193				234				255				
	1091	201				243				267				
	1092	204				268				298				
	1093	186				247				270				
	1094	201				279				305				
	1095	195				249				267				
	MEAN±SD	197±10	217±10	235±10	249±11	253±15	261±14	266±13	270±14	273±15	281±14	288±16	290±15	294±15

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		38	95	123	152	175	180	203	232	258	291	314	349	377
10	1046	306	308	324	327	338	334	345	358	360	365	376	372	378
	1047	277	280	285	297	299	352	305	310	320	328	328	349	362
	1048	295	303	303	316	322	316	331	332	347	353	362	351	362
	1049	326	331	343	359	366	354	364	379	379	379	391	386	386
	1050	292	296	312	322	328	346	338	342	358	356	380	382	378
	1051	301	301	317	319	335	323	336	359	356	380	380	393	400
	1052	318	314	236	Dead									
	1053	286	289	312	305	318	320	327	339	337	336	345	333	339
	1054	333	335	325	358	368	340	363	363	368	377	379	374	379
	1055	299	297	309	324	326	349	332	332	343	348	365	358	367
	1056	290	289	300	308	310	313	308	312	330	325	330	327	333
	1057	303	301	312	313	332	333	336	350	353	368	393	369	408
	1058	302	300	308	318	344	339	330	343	346	354	365	374*	376
	1059	293	291	277	253	268	296	315	306	309	321	324	325	323
	1960	306	314	317	331	344	343	351	363	364	377	381	389	402
	1061	273	277	283	300	304	302	308	319	313	324	339	347	360
	1062	305	307	315	324	329	337	337	339	338	361	367	390	422
	1063	296	291	311	317	320	331	330	336	332	342	348	347	336
	1064	268	270	285	295	299	300	294	305	312	322	322	331	335
	1065	303	310	319	330	343	344	364	362	367	406	409	429	441
	1066		301		322			334	338	336	349	353	352	365
	1067		293		314			325	325	320	338	347	349	358
	1068		312		321			345	351	353	369	377	381	396
	1069		290		305			315	328	335	336	352	348	358
	1070		312		329			347	360	371	380	389	408	408
	1071		294		313			339	360	369	416	430	459	514
	1072		266		287			293	298	305	312	311	312	317
	1073		313		327			335	347	346	358	357	380	365
	1074		308		325			333	343	350	371	377	388	403
	1075		313		322			338	339	352	352	354	359	372
	1076		301		327			337	350	350	386	Dead		
	1077		301		322			333	338	340	344	356	351	356
	1078		318		384			358	365	377	385	419	431	432
	1079		269		310			322	336	296	332	339	342	348
	1080		391		316			337	334	333	343	351	354	372
	1081		322		340			365	373	388	392	402	425	436
	1082		282		302			314	320	344	342	344	362	377
	1083		292		305			312	315	319	362	404	Dead	
	1084		314		337			374	366	388	397	404	417	426
	1085		276		299			308	314	324	337	339	324	336
	1086		291		317			325	336	346	346	356	363	366
	1087		270		283			295	304	305	313	318	324	324
	1088		307		330			347	350	360	386	381	408	406
	1089		312		327			357	367	383	382	388	400	406
	1090		286		289			316	316	336	350	373	377	389
	1091		290		303			318	324	333	336	341	338	340
	1092		320		349			341	354	367	355	364	379	374
	1093		293		310			331	346	374	379	378	420	432
	1094		336		359			379	382	411	408	409	393	402
	1095		292		310			324	331	338	346	349	342	356
	MEAN±SD	299±16	299±17	305±23	318±22	326±24	330±17	333±20	340±21	346±25	358±25	365±23	370±33	379±38

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	685	714	732
10	1046	389	398	418	421	431	446	472	471	469	499	460	477
	1047	361	384	386	388	400	408	414	404	409	402	413	418
	1048	378	410	439	Dead								
	1049	384	390	404	403	406	413	414	403	411	435	439	424
	1050	385	396	436	453	465	504	522	575	Dead			
	1051	406	403	422	Dead								
	1052	Dead											
	1053	326	337	357	368	376	381	407	441	466	Dead		
	1054	370	386	381	395	383	390	383	Dead				
	1055	366	373	403	395	413	421	391	404	370	Dead		
	1056	327	344	358	367	361	366	373	368	349	342	343	332
	1057	382	404	393	394	415	424	431	Dead				
	1058	379	370	332	384	386	395	388	389	397	344	Dead	
	1059	320	319	386	315	327	332	328	336	332	336	343	341
	1060	409	414	437	424	433	466	513	Dead				
	1061	351	369	384	394	393	411	417	422	415	442	Dead	
	1062	478	Dead										
	1063	336	348	355	351	356	358	367	361	356	370	396	421
	1064	344	355	377	380	389	388	415	417	413	405	341	Dead
	1065	436	459	497	562	Dead							
	1066	361	371	386	381	386	404	407	437	476	581	Dead	
	1067	361	411	517	Dead								
	1068	407	488	Dead									
	1069	359	371	404	410	424	459	517	668	Dead			
	1070	398	416	408	423	414	451	443	441	422	464	461	451
	1071	536	576	603	Dead								
	1072	316	331	339	369	338	344	350	348	345	345	342	344
	1073	359	375	387	391	396	409	442	507	621	Dead		
	1074	430	516	662	Dead								
	1075	368	370	391	392	399	407	406	384	343	Dead		
	1076	Dead											
	1077	357	376	385	388	407	426	460	524	580	711	Dead	
	1078	450	471	505	506	511	547	575	640	Dead			
	1079	344	360	364	368	377	390	421	Dead				
	1080	357	373	395	397	406	422	436	474	485	527	Dead	
	1081	433	453	455	449	424	Dead						
	1082	374	385	412	431	451	485	514	568	Dead			
	1083	Dead											
	1084	416	442	467	467	Dead							
	1085	334	343	347	347	339	339	334	327	320	322	246	Dead
	1086	369	395	423	419	421	437	436	439	434	431	442	447
	1087	326	336	349	339	353	359	375	391	417	557	737	Dead
	1088	401	416	484	Dead								
	1089	446	412	435	450	447	479	509	543	583	Dead		
	1090	392	394	435	474	427	462	490	553	Dead			
	1091	344	355	365	365	366	373	373	376	363	370	375	383
	1092	373	390	396	420	425	447	439	474	468	483	506	529
	1093	434	457	462	493	524	534	565	Dead				
	1094	409	Dead										
	1095	375	393	391	423	397	412	417	408	421	420	438	445
	MEAN±SD	382±44	396±50	417±65	408±49	405±43	420±52	434±62	449±88	427±79	439±99	419±110	418±59

21500

APPENDIX 5 (Concinued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		0	6	13	20	26	32	39	46	53	60	68	75	81
3	996	200	220	237	240	255	262	269	276	276	279	292	290	297
	997	178	197	212	226	237	244	256	244	249	256	267	265	274
	998	202	228	245	263	260	269	279	281	289	297	301	304	304
	999	206	225	247	259	259	276	283	288	294	298	311	311	319
	1000	197	224	249	258	272	280	286	303	308	308	316	323	329
	1001	187	205	230	227	229	240	255	256	257	272	275	267	273
	1002	189	208	222	231	237	241	248	253	251	252	271	271	258
	1003	192	212	230	248	256	263	271	277	287	291	297	305	300
	1004	200	218	231	236	243	252	258	265	270	267	278	274	281
	1005	219	235	250	259	256	267	280	299	294	291	296	301	298
	1006	195	219	237	257	263	275	288	294	302	296	306	308	313
	1007	201	226	247	257	265	276	287	309	302	298	305	299	310
	1008	213	238	247	266	267	273	285	292	287	292	300	302	301
	1009	198	218	228	242	249	258	255	261	260	265	269	275	277
	1010	216	236	249	257	266	273	282	288	294	297	299	306	311
	1011	197	219	235	245	252	264	270	273	284	289	300	303	301
	1012	187	210	218	242	243	249	254	265	270	267	283	284	282
	1013	206	237	251	269	272	285	295	293	305	303	317	321	328
	1014	186	211	235	249	257	253	258	264	269	274	283	285	285
	1015	200	217	241	249	253	267	274	274	280	285	291	295	294
	1016	189				236				268				
	1017	208				251				269				
	1018	196				252				270				
	1019	192				250				284				
	1020	181				248				268				
	1021	197				284				314				
	1022	191				259				283				
	1023	138				240				258				
	1024	199				266				292				
	1025	201				250				273				
	1026	197				252				278				
	1027	193				256				291				
	1028	196				249				264				
	1029	197				276				300				
	1030	188				261				285				
	1031	195				255				273				
	1032	188				249				274				
	1033	196				250				274				
	1034	206				261				282				
	1035	218				275				299				
	1036	205				262				283				
	1037	201				260				286				
	1038	195				245				283				
	1039	198				257				281				
	1040	190				230				247				
	1041	195				249				267				
	1042	183				249				271				
	1043	138				236				258				
	1044	193				244				267				
	1045	204				269				296				
	MEAN±SD	197±9	220±11	237±11	249±13	254±12	263±13	272±14	278±13	279±15	284±15	293±13	294±17	297±18

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APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams												
		88	95	123	152	175	180	203	232	258	291	314	349	377
3	996	304	310	322	327	332	330	333	370	349	349	361	358	362
	997	292	279	287	298	317	321	313	326	350	339	360	374	381
	998	305	312	327	326	340	343	346	353	361	375	373	384	395
	999	321	325	336	350	360	363	354	368	376	377	381	375	375
	1000	329	333	336	342	351	344	351	358	364	362	378	369	378
	1001	278	282	287	324	314	318	315	347	349	361	368	373	414
	1002	278	278	284	296	302	308	310	318	314	331	336	334	344
	1003	310	311	316	326	352	348	344	339	363	368	372	374	370
	1004	286	285	300	312	313	315	314	366	327	328	336	344	343
	1005	311	319	330	339	365	363	361	359	384	395	403	384	439
	1006	314	333	350	345	360	374	370	382	392	406	408	428	437
	1007	320	325	328	338	371	381	350	364	373	390	406	404	433
	1008	302	305	318	320	340	338	351	345	341	358	358	362	371
	1009	283	286	303	286	296	296	298	313	303	305	307	308	311
	1010	324	323	339	339	372	362	382	385	409	420	429	427	454
	1011	304	311	324	330	340	343	337	340	351	365	364	364	372
	1012	287	291	299	310	317	309	325	354	338	336	332	328	346
	1013	333	333	347	350	368	371	373	381	385	401	398	404	420
	1014	285	296	300	309	325	321	338	330	360	351	353	379	367
	1015	299	309	314	313	329	332	329	338	363	361	367	361	386
	1016		285		310			327	346	351	345	Dead		
	1017		291		305			316	323	326	341	342	349	341
	1018		301		317			318	341	343	346	352	355	364
	1019		301		312			336	346	364	357	365	378	386
	1020		285		304			304	310	310	316	313	319	316
	1021		344		349			377	385	398	397	404	410	420
	1022		316		332			346	365	362	370	365	373	375
	1023		284		299			309	332	333	337	343	341	346
	1024		317		335			343	360	355	369	363	359	358
	1025		300		309			333	337	337	354	350	352	354
	1026		310		315			335	341	338	353	353	351	362
	1027		315		335			366	360	361	377	385	397	419
	1028		292		296			332	324	343	339	341	335	Dead
	1029		325		340			349	365	370	387	383	385	391
	1030		312		338			364	369	374	374	398	404	415
	1031		290		314			319	326	328	335	353	335	342
	1032		320		313			319	342	352	339	340	341	352
	1033		296		314			321	328	337	347	342	344	345
	1034		315		320			342	357	346	360	357	374	397
	1035		317		332			344	357	360	388	379	399	399
	1036		315		326			339	349	346	337	359	364	371
	1037		309		325			334	345	345	348	348	352	349
	1038		Dead											
	1039		307		327			343	360	375	385	388	402	417
	1040		278		312			298	310	312	319	327	324	333
	1041		298		292			327	325	338	344	352	349	363
	1042		300		306			321	332	354	355	356	364	365
	1043		283		280			303	311	321	316	327	333	336
	1044		298		315			329	337	355	375	357	396	396
	1045		324		334			358	365	392	398	406	398	416
	MEAN±SD	303±17	306±17	317±20	320±17	338±24	339±24	336±21	347±20	353±23	359±26	363±26	367±28	378±35

21502

APPENDIX 5 (Continued)

INDIVIDUAL AND MEAN BODY WEIGHTS OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days of Study and Body Weights in Grams											
		405	433	468	501	531	559	587	622	650	683	714	732
3	996	382	427	460	Dead								
	997	376	409	409	424	441	453	463	460	468	475	473	460
	998	395	416	419	418	Dead							
	999	374	395	395	402	398	405	407	408	410	417	455	454
	1000	374	394	410	416	418	441	513	Dead				
	1001	415	448	495	556	Dead							
	1002	367	377	385	445	Dead							
	1003	378	399	403	414	407	418	429	477	577	Dead		
	1004	351	380	Dead									
	1005	491	634	Dead									
	1006	457	494	534	543	592	Dead						
	1007	429	436	474	479	481	499	508	526	530	600	610	544
	1008	363	390	388	407	407	425	425	425	445	463	475	465
	1009	309	331	327	333	324	331	347	332	332	340	339	344
	1010	460	484	479	481	492	514	512	479	524	452	459	460
	1011	362	384	388	404	400	397	358	381	424	439	435	432
	1012	335	351	364	329	361	369	376	371	375	386	346	292
	1013	423	446	447	483	461	462	475	492	Dead			
	1014	368	387	428	418	436	443	449	474	512	575	Dead	
	1015	392	423	434	463	476	484	504	496	514	560	581	560
	1016	Dead											
	1017	343	368	361	374	366	373	376	370	382	368	378	381
	1018	368	387	390	400	396	412	414	413	414	404	408	413
	1019	378	416	412	426	432	436	439	443	432	444	450	394
	1020	309	326	322	322	321	323	330	328	334	351	385	404
	1021	422	436	435	443	423	380	406	319	Dead			
	1022	370	389	399	435	404	362	341	Dead				
	1023	336	363	383	375	382	407	377	386	406	434	497	563
	1024	333	350	290	Dead								
	1025	342	369	371	378	378	380	373	372	373	368	394	393
	1026	358	384	380	386	381	386	354	365	374	363	369	364
	1027	413	425	442	459	491	503	549	581	677	Dead		
	1028	Dead											
	1029	397	427	445	455	471	550	Dead					
	1030	423	442	433	435	445	433	Dead					
	1031	424	377	352	367	386	383	385	389	398	417	440	442
	1032	338	376	387	418	417	433	437	427	400	404	Dead	
	1033	343	368	359	360	363	384	419	Dead				
	1034	389	422	415	432	434	439	436	374	Dead			
	1035	399	430	450	450	449	461	457	442	466	461	457	460
	1036	401	473	595	Dead								
	1037	348	362	356	349	359	360	370	359	350	357	354	355
	1038	Dead											
	1039	428	455	462	472	478	487	489	468	465	495	Dead	
	1040	331	353	356	361	364	383	384	395	412	401	318	231
	1041	364	378	397	431	500	Dead						
	1042	362	375	382	389	391	376	Dead					
	1043	324	345	347	353	350	359	379	378	367	449	Dead	
	1044	407	440	448	479	487	510	550	600	Dead			
	1045	417	468	477	476	491	494	481	455	439	470	471	469
	MEAN±SD	380±40	407±53	411±56	420±53	422±57	424±56	427±61	425±69	427±65	436±69	433±75	432±101

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APPENDIX 6

INDIVIDUAL AND MEAN FOOD CONSUMPTION[a] OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 DAYS

Dose mg/kg/day	Animal Number	Days on Test 0-3	8-10	11-13	14-17	18-20	21-24	25-27	28-31	32-34	35-38	39-41	42-45	46-48	49-52	53-55
0	75-594 75-595	24	28	29	29	29	27	25	25	25	—	31	27	25	24	24
	75-596 75-597	24	25	26	27	26	24	25	25	24	26	27	27	27	25	24
	75-598 75-599	25	25	26	28	26	25	25	25	25	26	26	26	24	25	24
	75-600 75-601	29	29	29	31	29	28	18	28	28	31	29	29	30	29	28
	75-602 75-603	26	—	31	30	30	—	—	27	28	27	27	28	27	27	28
	MEAN±S.D.	25±2	26±2	28±2	29±2	28±2	26±2	23±3	26±1	26±2	27±2	28±2	28±1	27±2	26±2	26±2
30	75-624 75-625	25	25	25	25	26	25	25	24	24	26	25	26	25	26	25
	75-626 75-627	24	26	26	26	25	24	25	24	25	25	25	25	25	26	25
	75-628 75-629	27	28	29	26	32	28	27	27	27	27	27	31	28	29	28
	75-630 75-631	25	26	27	27	26	25	24	24	26	26	25	26	26	25	25
	75-632 75-633	26	26	27	27	—	26	26	27	28	27	27	28	28	31	27
	MEAN±S.D.	25±1	26±1	27±1	26±1[b]	27±3	26±2	25±1	25±1	26±1	26±1	26±1	27±3	26±2	27±3	26±2
10	75-614 75-615	24	24	25	25	25	17	22	24	24	23	22	23	22	22	21
	75-616 75-617	25	16	26	24	26	25	25	25	24	25	25	26	26	25	25
	75-618 75-619	26	27	28	28	27	25	26	27	27	27	26	28	29	29	28
	75-620 75-621	25	26	26	27	27	27	26	26	25	26	26	27	26	26	26
	75-622 75-623	25	27	28	28	26	28	31	27	27	27	27	29	27	28	28
	MEAN±S.D.	25±1	26±1	27±1	26±1[b]	26±1	24±4	26±3	26±2	26±2	26±2	25±2	26±3	26±2	26±3	26±3
3	75-604 75-605	26	27	27	27	27	26	28	27	27	28	27	28	27	27	27
	75-606 75-607	25	27	29	27	26	26	27	27	26	27	26	30	28	28	27
	75-608 75-609	27	27	27	27	26	26	26	26	25	26	25	26	25	25	25
	75-610 75-611	26	29	28	28	27	27	27	28	27	26	26	28	26	27	27
	75-612 75-613	25	25	26	26	26	25	24	26	26	26	26	28	26	26	27
	MEAN±S.D.	26±1	27±1	27±1	27±1	26±1	26±1	25±1	27±1	26±1	26±1	26±1	28±1	26±1	27±1	27±1

Each value represents the mean daily food consumption for 2 rats housed in the same cage.

[a]All weights are in grams with mean ± one standard deviation for each dose level.

[b]Statistically different from control mean using Dunnett's test, $p<0.05$.

21504

APPENDIX 6 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION[a] OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 118 DAYS

Dose mg/kg/day	Animal Number	Days on Test													
		56-59	60-62	63-66	67-69	70-73	77-80	81-83	84-87	88-90	91-94	95-97	102-105	106-108	109-111
0	75-594 75-595	24	25	23	23	26	24	23	24	22	21	23	24	22	20
	75-596 75-597	24	24	24	23	24	26	25	26	25	—	25	23	23	22
	75-598 75-599	25	27	25	24	26	29	26	25	25	26	25	24	24	24
	75-600 75-601	28	29	28	26	28	28	26	28	27	29	27	28	27	22
	75-602 75-603	27	27	26	25	27	26	26	26	26	28	27	27	26	25
	MEAN±S.D.	26±2	27±2	25±2	24±1	26±1	27±2	25±2	26±1	25±2	26±4	25±2	25±2	24±2	23±2
30	75-624 75-625	19	23	24	23	22	25	23	25	23	24	24	25	24	23
	75-626 75-627	21	25	25	26	24	27	25	24	24	25	30	24	24	25
	75-628 75-629	23	28	30	28	27	30	26	27	27	28	27	27	29	24
	75-630 75-631	25	24	25	23	23	26	23	25	23	25	24	24	24	25
	75-632 75-633	24	26	27	26	26	28	27	27	26	27	27	26	37	22
	MEAN±S.D.	22±2	25±2	26±2	25±2	24±2	27±2	24±2	25±1	25±2	26±1	26±3	25±1	28±5	24±1
10	75-614 75-615	19	22	22	21	21	24	23	22	22	23	22	27	22	23
	75-616 75-617	25	25	25	27	24	26	25	25	24	27	24	25	—	23
	75-618 75-619	27	27	27	27	27	25	26	27	27	27	29	27	26	28
	75-620 75-621	26	27	27	25	25	26	25	26	25	27	25	27	24	21
	75-622 75-623	28	28	27	27	27	27	27	27	27	28	28	28	23	22
	MEAN±S.D.	25±4	26±2	25±2	25±3	25±2	26±1	25±2	26±2	25±2	26±2	26±3	27±1	24±2	24±3
3	75-604 75-605	27	26	27	25	24	27	26	26	35	27	26	26	25	23
	75-606 75-607	27	27	27	26	25	28	28	27	25	28	27	27	33	26
	75-608 75-609	21	23	24	23	23	26	24	26	24	27	26	26	25	24
	75-610 75-611	27	25	27	25	25	27	25	26	25	26	25	26	26	24
	75-612 75-613	26	26	26	25	25	26	25	27	24	26	26	—	24	21
	MEAN±S.D.	25±2	26±1	26±1	25±1	25±1	27±1	26±1	26±0	27±5	27±1	26±1	26±1	26±4	24±2

Each value represents the mean daily food consumption for 2 rats housed in the same cage.

[a]All weights are in grams with mean ± one standard deviation for each dose level.

[b]Statistically significantly difference from control mean using Dunnett's test, $p<0.05$.

21505

APPENDIX 7

INDIVIDUAL AND MEAN FOOD CONSUMPTION[a] OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 119 DAYS

Dose mg/kg/day	Animal Number	Days on Test 0-3	8-10	11-13	14-17	18-20	21-24	25-27	28-31	32-34	35-38	39-41	42-45	46-48	49-52	53-55
0	75-634 75-635	17	19	19	19	21	27	19	20	20	18	19	18	18	20	18
	75-636 75-637	19	19	19	20	18	26	21	21	17	19	18	18	19	18	17
	75-638 75-639	20	20	21	21	18	25	19	17	19	20	20	20	19	19	18
	75-640 75-641	21	20	20	22	21	26	19	20	21	19	25	20	18	21	21
	75-642 75-643	24	20	22	25	19	27	22	20	20	22	21	22	22	21	19
	MEAN±S.D.	20±3	19±1	20±1	21±3	19±1	26±1	20±1	20±1	19±2	20±2	20±2	20±2	19±1	20±1	19±2
30	75-664 75-665	19	18	19	18	19	24	18	18	18	18	18	17	17	17	18
	75-666 75-667	18	18	20	20	19	25	18	19	19	18	19	20	21	19	19
	75-668 75-669	19	20	20	21	22	23	19	20	19	18	19	18	18	17	18
	75-670 75-671	19	18	19	19	19	24	18	18	18	17	19	17	16	17	18
	75-672 75-673	19	21	18	20	22	29	20	19	20	20	20	20	19	19	19
	MEAN±S.D.	19±0	19±2	19±1	20±1	20±2	25±3	18±1	19±1	19±1	18±1	19±1	18±1	18±2	18±1	18±1
10	75-654 75-655	21	19	19	19	19	25	19	19	20	20	18	19	18	18	18
	75-656 75-657	20	20	20	21	19	26	19	20	20	18	19	19	16	21	20
	75-658 75-659	18	17	22	18	20	25	18	19	22	21	21	23	21	22	21
	75-660 75-661	22	21	20	23	20	27	21	21	20	20	19	21	19	19	19
	75-662 75-663	20	19	19	22	36	28	20	22	27	20	23	31	23	20	23
	MEAN±S.D.	20±1	19±2	20±1	20±2	23±7	26±1	19±1	20±1	22±3	20±1	20±2	23±5	20±3	20±1	20±2
3	75-644 75-645	19	19	18	18	19	24	20	19	19	19	20	20	17	19	18
	75-646 75-647	20	17	19	20	19	24	19	27	19	18	18	22	18	17	16
	75-648 75-649	20	18	18	21	21	25	19	19	19	18	19	20	18	19	18
	75-650 75-651	19	19	18	18	20	24	17	18	20	18	17	19	18	18	18
	75-652 75-653	21	20	22	22	23	28	18	19	21	21	20	22	23	22	21
	MEAN±S.D.	20±1	19±1	19±2	20±2	20±2	25±2	19±1	20±3	19±1	19±1	19±1	21±1	19±2	19±2	18±2

Each value represents the mean daily food consumption for 2 rats housed in the same cage.

[a]All weights are in grams with mean ± one standard deviation for each dose level.

21506

APPENDIX 7 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION[a] OF FEMALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 119 DAYS

Dose mg/kg/day	Animal Number	Days on Test 56-59	60-62	63-66	67-69	70-73	77-80	81-83	84-87	88-90	91-94	95-97	102-105	106-108	109-111
0	75-634 75-635	21	20	18	19	19	20	17	22	20	19	19	21	19	21
	75-636 75-637	17	20	18	17	17	20	18	20	18	20	18	21	27	20
	75-638 75-639	19	22	19	20	18	20	20	19	18	20	18	19	18	18
	75-640 75-641	19	23	18	18	19	20	25	19	18	19	18	20	12	13
	75-642 75-643	21	21	21	27	21	20	20	21	24	20	20	20	20	18
	MEAN±S.D.	20±1	22±3	19±1	20±4	19±1	20±0	20±3	20±1	20±3	20±1	19±1	20±1	19±5	19±1
30	75-664 75-665	17	16	17	16	16	21	17	18	17	17	18	19	16	16
	75-666 75-667	16	17	18	17	18	17	18	20	19	19	19	20	19	20
	75-668 75-669	16	17	18	16	17	21	20	20	17	18	16	20	20	19
	75-670 75-671	16	18	13	16	17	13	17	13	16	18	20	20	24	37
	75-672 75-673	19	20	20	20	18	19	19	21	20	20	18	20	19	20
	MEAN±S.D.	17±1[b]	18±2[b]	18±1	17±2	17±1	19±2	18±1	20±1	18±2	18±1	18±1	20±0	20±3	22±8
10	75-654 75-655	18	18	18	16	17	19	18	19	17	20	19	19	17	19
	75-656 75-657	20	19	18	15	17	20	19	20	21	21	19	18	16	15
	75-658 75-659	19	21	19	17	17	20	20	21	18	19	19	20	19	17
	75-660 75-661	19	18	20	20	19	19	17	21	18	19	20	18	30	20
	75-662 75-663	19	18	18	19	18	23	19	21	19	20	18	20	21	18
	MEAN±S.D.	19±1	19±1	19±1	17±2	17±1	20±2	19±1	20±1	19±1	20±1	19±1	19±1	21±6	18±2
3	75-644 75-645	17	19	21	15	20	—	18	20	18	18	17	21	22	18
	75-646 75-647	17	19	17	16	16	27	19	20	13	13	17	19	20	21
	75-648 75-649	18	19	—	18	18	24	18	21	20	20	21	20	18	23
	75-650 75-651	—	19	18	17	18	24	18	18	16	20	17	19	23	19
	75-652 75-653	17	22	21	20	21	28	21	22	20	23	20	22	17	23
	MEAN±S.D.	17±0[b]	19±1	19±2	17±2	18±2	26±2[b]	19±1	20±1	19±2	20±2	18±2	20±1	20±3	21±2

Each value represents the mean daily food consumption for 2 rats housed in the same cage.

[a]All weights are in grams with mean ± one standard deviation for each dose level.

[b]Significantly different from control mean by Dunnett's test, $p<0.05$.

21507

APPENDIX 8

INDIVIDUAL AND MEAN FOOD CONSUMPTION OF MALE RATS MAINTAINED OF DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days on Test and Grams/Rat/Day																					
		0-4	4-7	7-11	11-14	14-18	18-21	21-25	25-28	28-32	32-35	35-39	39-42	42-46	46-49	49-53	53-56	56-60	60-63	63-67	67-70	70-74	77-81
0	674 675	24	27	25	27	31	28	26	ND	25	25	26	26	27	25	26	26	27	25	ND	24	27	27
	676 677	26	29	27	28	29	27	27	29	27	28	28	ND	28	28	28	28	27	27	27	24	25	28
	678 679	23	26	24	26	27	24	25	26	24	24	25	27	25	24	24	25	25	23	24	21	24	27
	680 681	24	26	24	26	27	26	25	26	24	27	27	26	26	26	25	26	26	25	26	22	26	ND
	682 683	26	29	29	28	28	27	26	28	26	27	28	28	28	26	27	26	27	25	27	24	27	27
	684 685	23	26	24	26	27	24	25	29	24	25	26	25	26	24	25	25	25	23	25	22	24	24
	686 687	25	29	28	29	29	29	26	28	26	27	29	27	27	27	28	29	28	28	29	25	28	27
	688 689	24	26	25	27	27	26	26	28	26	28	28	28	28	28	28	28	27	26	28	25	27	26
	690 691	23	27	25	26	26	24	23	24	23	23	24	26	25	24	23	25	26	22	24	21	23	24
	692 693	23	26	25	26	27	26	25	27	24	24	25	26	27	26	25	25	25	25	26	24	26	27
	694																						
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	MEAN±SD	24±1	27±1	26±2	27±1	28±2	26±2	25±1	27±2	25±1	25±2	27±2	26±1	27±1	26±1	26±2	26±2	26±1	25±2	25±2	23±2	26±2	26±1

ND = No data available.

21508

APPENDIX 3 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days on Test and Grams/Rat/Day																
		81-84	84-88	88-91	91-95	119-123	123-126	147-151	151-154	180-182	182-186	203-207	207-210	231-235	235-238	259-263	263-266	287-291
0	674 675	25	25	24	26	25	25	24	23	26	25	24	26	25	25	25	27	24
	676 677	26	27	26	25	27	27	27	26	27	27	29	19	27	27	25	27	25
	678 679	25	24	22	23	28	25	23	22	38[a]	16	24	25	23	23	ND	24	23
	680 681	24	25	25	25	26	26	25	24	26	25	24	26	25	23	ND	24	25
	682 683	27	27	28	27	27	28	26	29	28	27	31	27	27	28	ND	28	26
	684 685	24	25	25	25	24	24	23	21	39[a]	25	24	26	24	24	24	24	24
	686 687	28	27	27	27	26	29	27	26	27	27	27	28	28	29	26	27	28
	688 689	26	26	26	26	14	27	27	25	27	26	26	28	28	27	27	28	28
	690 691	24	24	23	23	18	24	22	21	ND	28	23	27	23	23	22	25	22
	692 693	26	26	27	27	28	29	23	24	25	24	32	18	25	25	24	25	24
	694 695 696 697 698 699 700 701 702 703 704 705 706 707 708 709 710 711 712 713 714 715 716 717																	
	718	Dead																
	719 720 721 722 723 724 725 726 727 728 729 730 731 732 733 734 735 736 737 738 739 740 741 742 743 744 745 746 747 748 749 750 751 752 753 754 755 756 757 758 759																	Dead
	MEAN±SD	25±1	26±1	25±2	25±1	24±5	26±2	25±2	24±3	29±5	25±3	26±3	25±4	26±2	25±2	25±1	26±2	25±2

ND = No data available.

[a]Values excluded from statistical analysis calculations.

21509

APPENDIX 8 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days on Test and Grams/Rat/Day 291-294	315-319	319-322	350-354	378-382	406-410	434-438	469-473	497-501	532-536	560-564	588-592	623-627	651-655	686-690	714-718
0	674	26	24	26	25	26	23	25	24	24	23	23	22			Dead	
	675														Dead		
	676	26	25	26	25	25	23					Dead					
	677							14					Dead				
	678	23	23	16	23	23	22	ND	21	24	23	23	22	23	21	20	19
	679																
	680	26	23	26	26	21	25	15	23	24	24	25	26	26	23	24	23
	681																
	682	28	25	28	27	28	27	27	24	27	26	27	25			Dead	
	683													25		Dead	
	684	25	23	25	23	21	23	25	13	23	21	23	21	22	12	21	Dead
	685															Dead	
	686	31	25	29	28	21	26	25	26	28	27	24	26	26	23	Dead	
	687															ND	Dead
	688	29	26	28	26	31	25	27	17	26				Dead			
	689										27	27	17			Dead	
	690	24	23	24	23	23	23	23	ND	24	24			Dead			
	691											37	24	28	25		Dead
	692	26	25	26	22	29	30	27	15	25	20	29			Dead		
	693														Dead		
	694									24	24	23	29	31		Dead	
	695														Dead		
	696														Dead		
	697												20	24	ND		Dead
	698											Dead					
	699													26	23	25	Dead
	700														Dead		
	701													30	28		Dead
	702															Dead	
	703														Dead		
	704												Dead				
	705													Dead			
	706															Dead	
	707														Dead		
	708															Dead	
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	710											Dead					
	711															Dead	
	712														21		Dead
	713															21	Dead
	714															Dead	
	715														10		Dead
	716																Dead
	717															Dead	
	718								Dead								
	719															Dead	
	720												Dead				
	721								Dead								
	722															ND	Dead
	723																Dead
	724												Dead				
	725																Dead
	726												Dead				
	727													Dead			
	728														Dead		
	729															20	ND
	730															Dead	
	731														Dead		
	732															25	21
	733														Dead		
	734															Dead	
	735														Dead		
	736													Dead			
	737														Dead		
	738															Dead	
	739															Dead	
	740													Dead			
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	746														Dead		
	747															Dead	
	748																22
	749													Dead			
	750															Dead	
	751																Dead
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	753																Dead
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	758																Dead
	759															Dead	
	MEAN±SD	26±2	24±1	27±3	25±3	25±4	25±2	25±6	20±5	25±2	24±2	25±4	23±3	26±3	21±6	22±2	21±2

ND = No data available.

21510

APPENDIX 8 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days on Test and Grams/Rat/Day																					
		0-4	4-7	7-11	11-14	14-18	18-21	21-25	25-28	28-32	32-35	35-39	39-42	42-46	46-49	49-53	53-56	56-60	60-63	63-67	67-70	70-74	77-81
30	360 361	24	26	25	25	25	24	24	26	25	24	25	25	25	24	25	25	25	23	26	24	25	25
	362 363	25	27	26	27	26	24	24	25	26	25	26	27	29	25	26	25	25	24	26	24	25	25
	364 365	25	27	25	25	25	25	24	25	25	25	25	25	27	26	26	26	25	24	25	23	25	24
	366 367	25	27	26	28	26	24	23	24	25	23	26	25	27	25	29	24	24	24	24	23	25	23
	368 369	23	25	25	25	26	24	24	24	24	23	25	24	26	26	34	25	25	24	24	24	24	25
	870 871	24	27	25	30	26	29	25	25	25	24	25	24	27	25	27	26	25	24	25	23	25	24
	872 873	23	25	24	25	25	24	24	25	25	24	24	23	25	24	25	24	24	22	23	21	23	24
	874 875	23	27	24	24	25	24	24	24	23	24	24	24	25	24	25	25	24	24	25	23	25	24
	876 877	24	27	26	26	26	26	25	25	26	26	24	25	25	28	25	27	25	24	25	23	24	25
	878 879	24	27	26	27	27	27	26	27	26	26	27	27	29	27	28	28	27	27	27	26	27	ND
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	MEAN±SD	24±1	27±1	25±1	26±2	26±1	25±1	24±1	25±1	25±1	24±1	25±1	25±1	27±2	26±1	27±3	25±1	25±1	24±1	25±1	23±1	25±1	24±1[a]

ND = No data available.

[a]Statistically significant difference from control mean using Dunnett's test, $p<0.05$.

21511

APPENDIX 3 (Continued)

INDIVIDUAL AND MEAN FOOD CONSUMPTION OF MALE RATS MAINTAINED ON DIETS CONTAINING 2,4,5-T FOR 2 YEARS

Dose mg/kg/day	Animal Number 75-	Days on Test and Grams/Rat/Day																
		31-34	34-38	38-91	91-95	119-123	123-126	147-151	151-154	180-182	132-136	203-207	207-210	231-235	235-238	259-263	261-266	287-291
30	360 361	25	25	24	24	27	27	26	23	27	26	24	28	28	28	27	28	26
	362 363	24	26	25	24	26	25	26	24	27	26	24	27	27	26	25	26	24
	364 365	24	24	25	25	26	26	26	25	28	26	32	20	27	26	26	27	27
	366 367	23	24	24	24	28	28	26	21	25	25	23	26	26	26	25	27	25
	368 369	24	25	25	24	25	25	26	22	24	23	23	26	27	26	23	22	26
	370 371	23	25	24	24	25	25	25	21	26	24	23	26	26	25	26	27	24
	372 373	23	24	26	25	27	25	25	24	25	24	23	27	26	26	26	26	24
	374 375	25	25	25	25	25	26	24	24	27	25	33	17	27	25	25	25	26
	376 377	24	25	25	25	25	25	24	23	27	26	24	27	26	25	25	24	24
	378 379	30	28	28	28	29	28	26	24	27	ND	25	28	35	27	30	28	26
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	MEAN±SD	24±2	25±1	25±1	25±1	26±1	26±1	25±1	23±1	26±1	25±1	25±4	25±4	27±3	26±1	26±2	26±2	25±1

ND = No data available.

21512