

Dioxin Package

March 1993

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**Environmental Research Foundation
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Environmental Research Foundation (ERF) provides technical assistance to grass-roots environmental groups working to strengthen democracy at the local level. ERF's projects are designed to sustain a democratic movement of grass-roots groups, to curtail abuses of toxic and radioactive materials and to advocate reduction of the use of hazardous materials. Our weekly newsletter, *Rachel's Hazardous Waste News* and our on-line computer database RACHEL (Remote Access Chemical Hazards Electronic Library) provide timely information to citizens.

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Our DIOXIN package includes the following:

Incineration and Dioxin

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"Part 4: Scientists Study How 'Mass Burn' Incinerators Produce Dioxin, but Public's Fears May Not Disappear" from *Rachel's Hazardous Waste News* #45

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Incineration and Dioxin

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Reprint of:

RACHEL'S HAZARDOUS WASTE NEWS #31

Providing news and resources to the Movement for Environmental Justice -- June 29, 1987

WILL NEW 'MASS BURN' INCINERATORS PRODUCE ANY AIR POLLUTION THAT THREATENS TO HARM HUMAN HEALTH?

Are there valid reasons for people to be worried about air pollution from garbage incinerators?

The incineration of garbage (often called "mass burn," "resource recovery" or "waste-to-energy") seems to produce the family of toxic chemicals known as dioxins. The dioxins are partly released from the smoke stack of the incinerator, and they are partly retained in the ash, which is eventually sent to a landfill. No one seems to dispute that burning garbage produces dioxins. People argue about the amount of dioxins produced, and they argue about which particular components of garbage cause the production of dioxins. But no one seems to argue that you can burn garbage without producing dioxins.

Given that mass burn produces dioxins, whenever someone proposes a mass burn incinerator for garbage, we need to ask ourselves whether dioxins are really dangerous to humans. No one seems to doubt that dioxins are harmful to animals. The only unsettled question seems to be whether dioxins are harmful to humans.

Writing in the *Journal of the American Medical Association* April 18, 1986 (pgs. 2031-2038) a team of doctors and scientists reported on their study of 154 humans who had been exposed to dioxins during a two-year period (and their study of a control group of 155 people who had not been exposed to dioxins). The exposed group had lived for two years in a trailer park where dioxins had been mixed with waste oil and sprayed on roads to suppress dust.

The doctors reported no differences in the medical histories of the exposed and non-exposed groups; physical exams showed no differences; studies of blood and urine chemistry showed no differences; neurologic tests showed no differences. Yet the doctors reported that protective cells in the immune systems of the dioxin-exposed humans were reduced in number or impaired, not operating at peak levels, compared to the immune systems of the unexposed control group. The results were expressed in terms of impaired liver function, and in terms of impaired T-cell characteristics. It is the immune system in humans that fights off disease, so an impaired immune system would expose a person to risk of disease from non-dioxin-related causes.

For a free reprint of the medical article, write to Dr. Richard E. Hoffman, MD, Division of Environmental Hazards and Health Effects, Center for Environmental Health, Centers for Disease Control, U.S. Department of Health and Human Services, Atlanta, GA 30333; or phone (404) 329-3311 and ask for Dr. Hoffman. Request a copy of "Health Effects of Long-term Exposure to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin."

* * *

The California Air Resources Board (the state agency concerned with air pollution) in 1986 asked a Scientific Review Panel on Toxic Air Contaminants to look at available evidence on dioxins. Dr. Emil M. Mrak, Chancellor Emeritus of the University of California at Davis, on May 23, 1986, submitted the findings of the Scientific Review Panel, which he chaired. The report says, in part, "Dioxins are potent toxins and are known carcinogens and/or promoters of carcinogenesis in animals. Dioxins... are potential carcinogens or promoters of carcinogenesis in humans. The current and planned waste-to-energy facilities in California will provide a high potential for emissions of dioxins into air in the state. An exposure level [at] which no significant health effects will occur cannot be identified. For these reasons, we agree that dioxins should be listed by the ARB [Air Resources Board] as toxic air contaminants with no determined threshold below which adverse health effects will not occur."

The report goes on to say, "Dioxins and dibenzofurans are stable, lipophilic [fat-soluble] compounds that may be expected to accumulate up the food chain. Thus, airborne dioxins emitted during combustion may contribute to dioxin intake by humans not only via inhalation but by other routes such as ingestion of food. Assessment of dioxin intake only via inhalation may thus underestimate total intake."

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"Given that tissue levels of these compounds [in humans] are measurable, we expect measurable levels in air, and we feel that the expense involved in obtaining baseline data [on dioxins in air] is small compared to the potential risk. Monitoring of ambient dioxin levels should be commenced before any waste-to-energy facilities go on line....

"Since there is good evidence that dioxins can enhance the action of other carcinogens, the potential for the harmful interaction of dioxins with other environmental toxins could be important and must not be forgotten or underestimated when considering research reports on the actions of dioxins alone."

The two-page report, "Findings of the Scientific Review Panel on the Report on Chlorinated Dioxins and Dibenzofurans as Adopted at the April 16, 1986 Meeting" is available from Mr. William Lockett, California Air Resources Board, P.O. Box 2815, Sacramento, CA 95812; phone (916) 322-8168; or from Dr. Emil Mrak, Chancellor Emeritus, University of California at Davis, Davis, CA 95616; phone (916) 752-2442.

NEW STUDY SHOWS HOW ONE TOWN CAN RECYCLE AND CUT LANDFILL USE 70%

The town of East Hampton, New York, hired Barry Commoner's Center for the Biology of Natural Systems (CBNS) to devise a plan for handling the town's garbage without using an incinerator. The town has 15,000 winter time residents but it swells with tourists during summer. The resulting report recommends a combination of recycling and composting that would reduce by 70% the amount of garbage going to the landfill. Get *An Intensive Trash Separation and Recycling System for the Town of East Hampton* for \$10 from CBNS, Queens College, Flushing, NY 11367; phone (718) 670-4182.

[Dated announcement omitted from this reprint.]

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Part 4: SCIENTISTS STUDY HOW 'MASS BURN' INCINERATORS PRODUCE DIOXIN, BUT PUBLIC'S FEARS MAY NOT DISAPPEAR

When municipal garbage goes into a "mass burn" incinerator, one of the products that goes out the smokestack (or is trapped by a filter and ends up in an ash landfill) is the family of chemicals called dioxins. No one disputes that dioxins are produced by mass burn incineration, but scientists disagree on where the dioxins come from.

Just a few years ago, many scientists were saying that the dioxins was produced by low-temperature combustion (less than, say, 1400 degrees Fahrenheit). They advocated that incinerators should be designed to burn hotter to "burn up" the dioxins or perhaps not ever create the dioxins at all. Others scientists were saying that dioxins was produced by the burning of pvc plastic and if we would just keep the pvc out of the waste stream, all would be well.

Dr. Barry Commoner had a different idea. He thought that dioxins was being created inside the smokestack itself, after the smoke left the combustion chamber of the incinerator. If he was right, running the firebox hotter wouldn't prevent the formation of dioxins. He did some experiments, wrote up his results, and then began advocating his ideas. He's a persuasive speaker and a tough opponent in a debate. His effective arguments angered his opponents and they began to call him a charlatan and a "pseudo-scientist."

Recently, new research has confirmed Dr. Commoner's original ideas. In the *Journal of Chromatography*, Vol. 389 (1987), pgs. 127-137 and in *Science* Vol. 237 (August 14, 1987), pgs. 754-756, Canadian scientists report that they have found a major mechanism for creating dioxins in waste combustion: the fly ash (the particles that make up the smoke in the smokestack) acts as a catalyst (a chemical promoter) and causes the formation of dioxins. They reported experiments in which they created dioxins with chlorine-containing wastes but they also reported creation of dioxins from non-chlorinated compounds. *Science News* (Aug. 22, 1987, pgs. 118-119) quoted one of these researchers saying, "We found that [fly ash] is indeed a very strong catalyst which causes dioxins to form from almost anything." They are now hard at work trying to manipulate the composition of garbage to minimize the production of certain types of fly ash catalysts, especially those containing metals. Pigments in printing inks, for example, contain metals that may produce fly ash that produces dioxins. They think that getting the metal out of garbage might substantially reduce dioxin production.

Still other recent research throws additional light on the "dioxin from mass burn" problem. Experiments at an incinerator in Pittsfield, Massachusetts were aimed at discovering relationships between combustion conditions, garbage constituents, and dioxin production. The report, Results of the Combustion and Emissions Research Project at the Vicon Incinerator Facility in Pittsfield, Massachusetts appeared in June, 1987. Some of its more interesting conclusions were:

** There is no evidence that the amount of PVC plastic in the garbage affects the amount of dioxin produced (pg. 10-4);

** Wet garbage produces more dioxins than drier garbage (pg. 10-4);

** There is no consistent relationship between the amount of dioxin in the raw garbage and the amount of dioxin in the smoke stack gases (pg. 10-5);

** There is a relationship between the amount of carbon monoxide in the smokestack gas and the amount of dioxin (pgs. 10-1, 10-5); "However," the report says, "one must be cautioned that the specific relationships between operating temperature, carbon monoxide levels, and levels of [dioxins] found during this study cannot necessarily be generalized to other incinerators, particularly of a different design."

**** The location of measuring devices in the incinerator affects how much dioxin is found (pg. 10-1).**

What can we conclude from all this? Authors of the Pittsfield study say future research should focus on the effects of facility design and operation (especially carbon monoxide levels, operating temperature, and air pollution control systems) on the production of dioxins, instead of focusing on characteristics of the garbage.

On the other hand, the Canadian researchers believe that manipulating the contents of the garbage is the key to discouraging dioxin production.

From the citizen's perspective, faced with a machine that will be in operation for 20 to 30 years, during which time the chemical contents of American garbage are likely to change dramatically (if the last 20 or 30 years are any indication), trying to manipulate the contents of the garbage seems complicated, untrustworthy and doomed to fail. No government authority will ever be able to control the contents of the stream of garbage entering a mass burn incinerator.

On the other hand, trying to find and maintain the exact operating conditions that minimize dioxin formation (a narrow range of temperatures, for example, and 100 ppm of carbon monoxide in the stack) seems equally hopeless. Garbage incinerators are not going to be run under laboratory conditions by chemists. They're going to be run by garbage companies who have demonstrated time and time again that they'll cut any corner necessary to make a buck. The garbage industry, including the incineration industry, is dominated by organized crime or by people who competed with organized crime and won out. These are not the kind of people to whom you want to entrust an enormous, complex and dangerous machine, the careful operation of which will dramatically affect public health and safety.

In any case, knowing what minimizes dioxin production under controlled conditions cannot ease the public's fear that mass burn will pollute the air. The fly ash researchers writing in *Science* noted that "more than 600 organic compounds" have been identified on incinerator fly ash. That's 600 more than the public wants to breathe or dump into the local landfill. Dioxin is simply No. 601 on the public's list of unwanted pollutants from mass burn.

The Pittsfield study is NYSERDA Report 87-16, available from Department of Communications, NY State Energy R&D Authority, Two Rockefeller Plaza, Albany, NY 12223; phone (518) 465-6251.

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INCINERATION OF INFECTIOUS WASTE

In 1985, there were 6872 hospitals in the U.S. with 1.3 million beds. If beds are occupied 70% of the time and each occupied bed creates 13 pounds of waste per day, U.S. hospitals produce 2.2 million tons of waste per year. An estimated 15% of this is infectious waste containing human anatomical waste, plus garments, gauze pads, diapers, catheters, and so forth. But the infectious 15% cannot be segregated from the non-infectious, so it all gets mixed together, creating a larger mass that must all be considered infectious.

An estimated 90% of all hospitals run their own incinerators; this means about 6,200 neighborhoods are impacted by such machines. From the viewpoint of people living near hospitals, there are five items of concern: (1) the emission of bacteria or viruses that might make people sick; (2) the emission of low molecular weight organic molecules (trichloroethylene [a suspected carcinogen], and tetrachloroethylene, among others); (3) the emission of high molecular weight organic molecules, (so-called "products of incomplete combustion," benzopyrenes, PCBs, polynuclear aromatic hydrocarbons and other polycyclic organic matter, much of which is carcinogenic); (4) the emission of toxic particles small enough for a person to breathe deep into their lungs; (5) and the emission of dioxins and furans.

The U.S. Environmental Protection Agency (EPA) recently released a report completed under contract by the Radian Corporation. The aim was to gather what is known about pollution from hospital incinerators. The authors searched the scientific literature, and interviewed many knowledgeable experts: staff of regulatory agencies (EPA, state and local), the American Hospital Association, and incinerator vendors.

Of the estimated 6200 hospital incinerators in use, perhaps 1200 are large incinerators (burning more than 400 pounds per hour or 400 tons per year) and 5000 are smaller. Hospital incinerators fall into three types: rotary kiln, excess air and starved air. By far the commonest (especially among units installed during the last 15 years) is starved air (also called "controlled air," "two-stage" and "modular"). Excess air incinerators (also called "pyrolytic incinerators," and "multi-chamber incinerators") come in two types: large units, which are generally "in-line," and smaller units, which are generally "retort" types.

Radian could find no air emissions data for small excess-air incinerators, the kind that serve perhaps 75% or more of the nation's hospitals. Therefore, the remainder of this article describes air emissions from large controlled-air incinerators.

Even among these few incinerators, the lack of data is shocking. Let's look at the five categories mentioned above.

Bacteria and viruses. You might think emission of disease-causing organisms (pathogens) from hospital incinerators would be the subject of intense scrutiny. Not so. The Radian corporation could find only two studies in the scientific literature. One study found twice as many bacteria leaving an incinerator smoke stack as were found in normal outdoor air (though the results were not statistically significant; in other words, they may have occurred by chance). The second study showed that, in a two-chamber incinerator, the temperature in the first chamber has to be 1400 degrees Fahrenheit and in the second chamber had to be 1600 deg. F. to guarantee sterilization of bacteria and viruses. (Many incinerators are not designed to exceed 1400 deg. in their secondary chamber, so presumably do not effectively sterilize wastes they burn.) That ends the discussion of bacteria and viruses in the Radian study. The entire section on pathogens takes up 14 lines of text in a 151-page report.

Low molecular weight organic compounds: Radian says, "One large data gap in the current hospital waste incinerator emissions data base is for lower molecular weight organic compounds." End of discussion.

Products of incomplete combustion, benzopyrene, PCBs and other carcinogenic organic molecules: These pollutants are "important," says Radian, but are not included in the study "due to lack of emissions data."

Tiny toxic particles: A micron is a millionth of a meter (and a meter is about a yard). If you breathe particles (soot) in the size range of 2 to 10 microns in diameter, they are filtered out by the respiratory system (nose, esophagus, etc.) and are moved into the mouth, then swallowed. Particles smaller than 2 microns are considered "respirable" because they can enter the deep lungs. The smallest particles (1 micron or less) enter the deepest part of the lung, the alveoli, the 3 million little sacs that pass oxygen into the blood and let carbon dioxide pass back out. Particles that enter the alveoli may eventually be removed by natural cleansing mechanisms, or they may lodge there, contributing a grey color to the normally-pink lungs, or they may actually pass into the blood stream and go someplace else in the body.

As luck would have it, the respirable particles from an incinerator contain more than their fair share of toxic materials, especially toxic metals and the often-carcinogenic polycyclic organic molecules. The small particles have a larger surface area in relation to their bulk than do larger particles. (A physicist would say small particles have a larger surface-to-volume ratio.) The relatively large surface area of the smallest particles attracts toxic heavy metals during combustion. Thus, respirable particles end up loaded (Radian says "enriched") with lead, arsenic, cadmium, chromium and other dangerous metals. (Radian presents no data on enrichment by polycyclic organics, unfortunately.) Thus the particles that reach deepest into the lungs are the most toxic. Typical hospital incinerators emit 1.5 to 36 pounds of particles per ton of waste incinerated. (Incidentally, bacteria and viruses have diameters ranging from 0.4 microns down to 0.02 microns, so they are definitely respirable.)

Dioxins and furans: The Radian study describes in detail the combustion conditions under which dioxins and furans will be formed. They conclude that the primary chamber of a hospital incinerator is perfect for manufacturing dioxins and furans, and that safety depends upon the secondary chamber completely destroying the dioxins by maintaining ideal combustion conditions. They conclude that the average amount of total dioxins emitted from a 1000 pound-per-hour hospital incinerator operating 2000 hours per year would be 3.7 grams of dioxin per year. (There are 28 grams in an ounce.) This may not sound like much, but dioxin is considered extremely toxic, so it may be a lot, depending on where it goes once it is released into your neighborhood.

Conclusion: The nation's primary medical care institutions are almost all operating incinerators without knowing what they are doing, literally. The data are simply not available. What data there are, on respirable particles and dioxins, indicate the need for very tight air pollution control, which is not provided under federal law, nor under most state laws. In short, hospital incineration of infectious wastes is a scandal.

To get your free copy of Radian's *Hospital Waste Combustion Study, Data Gathering Phase, Final Draft Report*, write Ray Morrison, Pollutant Assessment Branch, Office of Air Quality Planning and Standards, EPA, Research Triangle, NC 27711; phone (919) 541-5332.

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MEDICAL INCINERATORS EMIT DANGEROUS METALS AND DIOXIN, NEW STUDY SAYS

Medical waste incinerators are a major source of toxic air emissions, especially dioxin and cadmium, according to a lengthy study just released by the California Air Resources Board (CARB), a state agency. [Page numbers in our text (below) refer to pages in this new study, which is cited in our next-to-last paragraph.] The study says the public health risks from breathing these emissions are unacceptably high. On May 23, the CARB will propose new air pollution controls on California's medical incinerators; CARB predicts the new rules will force 129 of the state's 142 incinerators to cease operation because compliance will be too costly.

Dioxin

The CARB studied dioxin in 1986 and declared it "highly toxic" in experimental animals. Toxic effects in animals include severe weight loss, liver necrosis [death of tissue] and hypertrophy [enlargement], skin lesions, immunosuppression, reproductive toxicity, teratogenesis [birth defects], carcinogenesis [cancer] and death. The CARB concluded that dioxin is an animal carcinogen and a "potential human carcinogen." They also reported that both acute (short-term) and chronic (long-term) exposure to dioxin has caused liver damage in humans (pg. 10).

Cadmium

Chronic exposure to cadmium, a toxic metal, causes a wide range of adverse effects in humans, including "cardiovascular [heart, arteries], endocrine [hormone], hepatic [liver], bone, hematological [blood], immunological [immune system], respiratory [lung], renal [kidney], reproductive and teratogenic [birth defect] effects." Furthermore, "There is strong evidence of an association between cadmium exposure and an increased risk of respiratory [lung] cancer," says the CARB report (pg. 18).

California has 142 medical waste incinerators operating today. Together, they burn 20,050 tons of wastes per year, so they are not large. (For example, a big municipal waste incinerator burns this much waste every 10 days.) Medical waste includes paper, cardboard, plastic, rubber, food wastes, cans, bottles, animal carcasses, sharps [hypodermic needles, scalpels, etc.], chemotherapy wastes, diapers, bandages, solvents, and pathological wastes [body parts, bed pan residues, and so forth]. Medical waste also contains metals such as lead, cadmium, mercury, and zinc; it can also contain low-level radioactivity (pg. 34).

Despite their relatively small size, California's 142 medwaste incinerators emit substantial quantities of dioxins and cadmium: 16 grams of dioxin each year (pg. 31) and 30 to 40 pounds of cadmium (pg. 33) each year.

To gather emissions data, the CARB monitored eight medwaste incinerators. The report notes that, "Although the emissions tests were not conducted for compliance purposes, we observed that many of the facilities tested could not meet the particulate limitations in their permits. In addition, the hydrochloric acid emissions were elevated...." (pg. 48)

The eight emissions tests revealed many other air contaminants emitted by one or more of the incinerators. Here is a list of pollutants emitted, followed by the number (1 to 8) of incinerators, out of 8 tested, found to be emitting each pollutant: arsenic (7); ammonia (1); benzene (8); bromodichloromethane (2); cadmium (8); carbon tetrachloride (6); chromium (7); chlorodibromomethane (3); chloroform (4); cumene (5); 1,2-dibromoethane (5); dichloromethane (6); dichloroethane (3); dioxins and furans (8); ethyl benzene (7); lead (8); mercury (5); mesitylene (7); nickel (3); particulate matter (8); naphthalene (5); tetrachloroethane (4); toluene (7); trichloroethane (3); 1,1,1-trichloroethane (6); trichloroethylene (6); trichloromethane (5); vinyl chloride (2); xylenes (7) (pg. 51).

The CARB study indicates that it will cost from \$383,000 to \$479,000 to put best available control air pollution control technology (BACT) (a spray dryer [also called a dry scrubber] and baghouse filter) on an existing large, regional medwaste incinerators (pg. 115), of which California has 12.

According to the CARB's data, small medwaste incinerators serving an individual institution will not be able to afford to install air pollution equipment that would allow continued operation of the incinerator after the new dioxin and cadmium regulations become effective this summer. For these 129 incinerators, the available alternatives include (a) shut down and use a large, regional medwaste incinerator; (b) shut down and use off-site steam sterilization; (c) shut down and use onsite steam sterilization.

Steam sterilization (also called autoclaving) means heating the wastes in a metal chamber by filling the chamber with saturated steam and holding the temperature at 250° Fahrenheit for 30 minutes. It effectively sterilizes wastes, killing viruses and bacteria. The wastes then go to a landfill. Of these three options, the cheapest is onsite sterilization; this option would increase medwaste disposal costs 10 cents per pound, and would increase patient costs 16 cents per hospital bed per day, compared to present costs of onsite incineration. The use of offsite, regional incineration would increase disposal costs 16 cents per pound and would increase average patient costs 41 cents per bed per day. Onsite steam sterilization would increase disposal costs 16 cents per pound and would increase patient costs 42 cents per bed per day. Thus, shutting down its incinerator and sending wastes out for regional steam sterilization is the cheapest way for a hospital to come into compliance with the proposed California air regulations for cadmium and dioxin (pg. 118).

A different study of medical wastes, by members of the Civil Engineering Department of University of California at Davis (cited below in our last paragraph), points out that, in a typical hospital, infectious waste makes up only about 15% of the total waste stream. Infectious waste by itself will burn, but the volume is usually not sufficient to allow heat recovery. Therefore, the decision to install an incinerator for infectious waste encourages the burning of other non-infectious wastes. In effect, a medwaste incinerator is a municipal solid waste incinerator with 15% of the waste being infectious.

There are, however, a few differences between municipal wastes and infectious wastes. Plastics make up 3% to 7% of municipal waste, but 14% to 30% of infectious waste. Because plastics are the major sources of cadmium and lead, medical waste incinerators emit more of these toxic metals, per pound of burned waste, than do municipal incinerators.

Lead is present as a pigment and as a stiffening and strengthening agent in many plastic items, especially those made of PVC (polyvinyl chloride). For example, the red bags themselves, in which "red bag" medical wastes are packaged, is colored red by a lead compound. Lead is also present in yellow bags for collecting soiled linens, yellow bedpans, and yellow tops of urine containers. In plastics, a bright color (yellow, orange, red, purple) is often an indication of a toxic metal (cadmium or lead).

The Davis researchers didn't say so, but *hospitals could refuse to purchase medical supplies made with toxic metals, thus minimizing problems.*

Get: *DRAFT Technical Support Document to Proposed Dioxins and Cadmium Control Measure for Medical Waste Incinerators* (Sacramento, CA: Air Resources Board [1102 Q St., P.O. Box 2815, Sacramento, CA 95812], 1990. Phone Information Officer Jerry Martin at (916) 322-2990. This document is about 500 pages long, contains a great deal of useful information on medical waste incineration, and is free while supplies last.

Also get: D.C. Hickman, D.P.Y. Chang, and H. Glasser, "Cadmium, and Lead in Bio-Medical Waste Incinerators," a paper presented at the 82nd Annual Meeting of the Air and Waste Management Association at Anaheim, CA, June 25-30, 1989; available free from Dan Chang, Department of Civil Engineering, Mail ID# 1855, University of California at Davis, Davis, CA 95616.

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RACHEL'S HAZARDOUS WASTE NEWS #280

Providing news and resources to the Movement for Environmental Justice -- April 7, 1992

ALL HAZARDOUS WASTE INCINERATORS FAIL TO MEET EPA REGULATIONS, EPA SAYS

Hazardous waste incinerators, and the web of regulations intended to make them operate safely, have come under withering criticism from government scientists, private researchers, and the *Wall Street Journal* during the last 60 days. Officials of EPA (U.S. Environmental Protection Agency) and private research scientists now admit that hazardous waste incinerators emit hundreds of times more dioxins and other toxic air pollutants than is allowed by EPA regulations, and the *Journal* revealed a record of malfunctions, including explosions and major releases of toxins, that incinerator operators have tried to cover up and that regulatory officials seem powerless to understand, much less curtail.

Scientists employed by U.S. EPA (Environmental Protection Agency) admitted last month that modern hazardous waste incinerators simply cannot comply with existing federal regulations because they cannot destroy all chemicals with 99.99% destruction/removal efficiency (DRE), which is the efficiency required by federal law. Federal law further requires that certain wastes of "special concern" such as dioxins, furans, and PCBs must be destroyed with 99.9999% DRE. EPA scientists said last month that they have known since at least 1985 that hazardous waste incinerators could not meet any of these regulatory requirements.

The story broke when Pat Costner, a chemist and research director for Greenpeace, published an independent analysis of dioxin emissions from the Jacksonville, Arkansas incinerator.¹ The Jacksonville incinerator has begun burning 16.5 million pounds of herbicides (2,4,5-T and 2,4-D) left over from the Vietnam war. These wastes are known to be contaminated with total dioxins and furans at concentrations ranging from 3 to 40 parts per million (ppm).

Costner's analysis revealed that the Jacksonville incinerator was only achieving 99.96% destruction of the dioxins entering the incinerator, thus emitting 400 times more dioxin into the community than the law allows. An official with the Arkansas Department of Pollution Control and Ecology (DPC&E) acknowledged in telephone interviews that Costner's calculations are correct. He also said the department had no intention of shutting down the incinerator despite its continuing emissions of dioxin directly into a residential community. He said the department did not know what the total dioxin emissions into the population of Jacksonville would be, but, he said, no matter what the total may be, it is safe.

The Jacksonville incinerator is a key demonstration project, established with the cooperation of EPA Administrator William Reilly and Arkansas Governor Bill Clinton to show that dioxin-containing wastes can be incinerated in a residential neighborhood over the objections of the community.² In a city-wide referendum in March, 1986, the people of Jacksonville voted two-to-one (1383 to 656) to stop the project but government officials simply ignored the vote and have overridden all objections ever since. Costner's analysis clearly showed that residents of Jacksonville are being exposed to levels of dioxin contamination that exceed federal health and safety standards by a wide margin. This is the first systematic dioxin experiment on humans using a residential population. Previous dioxin exposures of humans have occurred during industrial accidents and in the industrial manufacture of chemical-biological warfare agents. Dioxin is now known to cause cancer in humans and to disrupt normal growth and development of fetuses and infants at low levels of exposure.³

About 100 waste sites in the U.S. contain substantial quantities of dioxin,⁴ and the U.S. has stockpiles containing billions of pounds of chemical-biological warfare (CBW) agents the government has said it wants to incinerate. If the Jacksonville dioxin experiment can be maintained despite ethical and public health objections, government agencies will be able to claim they have a green light to incinerate just about anything just about anywhere.

However the Jacksonville experiment has brought to light information that could derail the entire U.S. incineration program. In preparing her analysis of dioxin exposure of the Jacksonville populace, Costner uncovered a government study showing that tests in 1984-85 by private researchers, under contract to EPA, revealed that hazardous waste incinerators cannot be expected to achieve 99.9999 percent destruction of wastes that occur in concentrations lower than 10,000 parts

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per million, and cannot be expected to achieve 99.99 percent destruction of wastes that occur in concentrations lower than 1000 ppm. EPA published the 1985 data in 1989.⁵

When this information came to light, a news reporter from the *Arkansas Democrat-Gazette*, Sandy Davis, interviewed Bob Hall, chief of the EPA's Combustion Research Branch in Research Triangle, North Carolina, and he confirmed what the EPA report had shown. "The fact is that you run into problems with your DRE [destruction/removal efficiency] when a low concentration of wastes is fed into the incinerator," Hall said. "Our data clearly shows that," he said. Davis asked Hall why EPA hasn't changed its regulations since it knows existing incinerators cannot comply with the regulations. Hall said, "I don't know why that hasn't been changed. It's a regulatory issue. I'm in research."⁶

Costner uncovered a second EPA report,⁷ published in 1984 but never widely circulated, showing that, among eight major hazardous waste incinerators studied, none could achieve 99.99% DRE. Sandy Davis interviewed the author of that report, Drew Trenholm of the Midwest Research Institute in Research Triangle, North Carolina, who said incinerators simply cannot achieve the DRE required by federal law. "The trend is very strong in the data that this is the case," Trenholm told Davis.

At public hearings over the past decade, dozens of EPA officials have stated for the record that incinerators can achieve the legally-required DREs, in what appears to be a coverup of public health information of astonishing proportions.

Many of the most dangerous toxins such as dioxins, furans, and PCBs occur in wastes at low concentrations. If low-concentration chemicals cannot be destroyed effectively, this means all sludge incinerators, all contaminated-soil burners, and all wood-treatment-waste incinerators will fail to meet federal regulations and will emit illegal quantities of potent toxins into surrounding air.

Ongoing failure to achieve the required destruction efficiencies is not all that plagues incinerators. *Wall Street Journal* reporter Jeff Bailey pointed out March 20 that federal, state and local regulatory officials pay close attention to hazardous waste incinerators but they can't be everywhere at the same time and they often learn about accidents, explosions and violations from tips phoned to them anonymously by insiders.⁸ For example, Chem Waste, the nation's largest hazardous waste hauler, is not considered a fly-by-night operator. According to Joan Bernstein, vice-president for environmental policy and ethical standards at Chem Waste, "Environmental compliance is what drives this company." If this is true, then Chem Waste's ongoing record of accidents, explosions, leaks, releases and coverups involving their incinerators must mean that even the wealthiest companies that have written down their best intentions on a piece of paper still cannot operate hazardous waste incinerators in a fashion that any reasonable person would call safe.

[To be continued.]

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[1] Pat Costner, *The Incineration of Dioxin in Jacksonville, Arkansas: A Review of Trial Burns and Related Air Monitoring at Vertac Site Contractors Incinerator, Jacksonville, AR* (Washington, DC: Greenpeace Toxics Campaign, January 29, 1992).

[2] Stephanie Arbanel and others, "Toxic Nightmare on Main Street," *Family Circle* August 14, 1990, pgs. 77-80, 120-128.

[3] Karen F. Schmidt, "Puzzling Over a Poison; On closer inspection, the ubiquitous pollutant dioxin appears more dangerous than ever," *U.S. News & World Report* April 6, 1992, pgs. 60-61.

[4] Peter A. Johnson and others, *Dioxin Treatment Technologies: Background Paper* [OTA-BP-O-93] (Washington, DC: U.S. Government Printing Office, 1991). 80 pages. \$4.00 from U.S. Government Printing Office, P.O. Box 371954, Pittsburgh, PA 15250-7954 or phone your order to (202) 783-3238.

[5] John C. Kramlich and others, *Experimental Investigation of Critical Fundamental Issues in Hazardous Waste Incineration* (Springfield, VA: National Technical Information Service [NTIS], September, 1989.) This is EPA document No. EPA/600/2-89/048 available from NTIS for \$26.00; phone (800) 553-6847 and request NTIS document No. PB90-108507.

[6] Sandy Davis, "Incinerator can't do job, engineer says," *Arkansas Democrat-Gazette* March 14, 1992, pgs. 1A, 15A.

[7] A. Trenholm and others, *Performance Evaluation of Full-Scale Hazardous Waste Incinerators. Vol. I. Executive Summary*. [EPA/600/2-84/181A]. (Washington, DC: U.S. Environmental Protection Agency, 1984). Available from NTIS as document No. PB 85-129500. \$17.00. Phone 800/553-6847.

[8] Jeff Bailey, "Concerns Mount Over Operating Methods Of Plants That Incinerate Toxic Waste," *Wall Street Journal* March 20, 1992, pgs. B1, B5.

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RACHEL'S HAZARDOUS WASTE NEWS #311

Providing news and resources to the Movement for Environmental Justice -- November 12, 1992

JACKSONVILLE

On Tuesday, October 27, one week before his election as President of the United States, Governor Bill Clinton of Arkansas gave the final order to start burning dioxin in an incinerator in a residential area of Jacksonville, Arkansas, a community of 29,000 people 15 miles northeast of Little Rock. The incinerator was built in the residential neighborhood with state funds for the purpose of burning 30,000 barrels of mixed hazardous wastes abandoned in Jacksonville by a company called Vertac Chemical, which manufactured pesticides and herbicides there from 1948 to 1986.

The *New York Times* (Nov. 2, 1992, pg. B11) reported that the Governor made the decision himself to start the burn: "Mr. Clinton, who has overseen the investigation and cleanup at Vertac most of the last 13 years, gave final approval on Tuesday to a plan to burn the chemical wastes in an incinerator...." The *Times* said (Oct. 28, 1992, pg. A14), "No other environmental issue in Arkansas has so dominated the public debate during Gov. Bill Clinton's 12 years in the Governor's Mansion as the chemical contamination in Jacksonville and what to do about it."

Over the years the Vertac site was used for manufacture of DDT, aldrin, dieldrin, toxaphene and the chemical warfare defoliants 2,4-D, Silvex, 2,4,5-T, and Agent Orange. In 1979 state and federal investigators discovered dioxin on the Vertac property and in the soil and water several hundred yards from the site. Official surveys subsequently found dioxin from the plant in Jacksonville's central city park, making its once-popular swimming and fishing lake off limits to the public.

Almost immediately, citizens began pressing for a thorough, safe cleanup of the Vertac site. In what has now become a familiar story, state and federal authorities delayed cleanup. According to the *New York Times*, "Since 1979... Vertac's toxic wastes and what to do about them have been the source of considerable strife in the community and have dominated the attention of Gov. Bill Clinton and his environmental advisers." (Oct. 30, 1992, pg. A16.)

In 1986, Vertac declared bankruptcy and willed its 93-acre site to the people of Arkansas. Vertac's executives abruptly left town and have never been successfully traced. The *New York Times* said, "Vertac abandoned the plant leaving behind roughly 30,000 barrels of chemical wastes, along with acres of contaminated soil, tanks filled with toxic materials, and miles of poisonous piping. The EPA [U.S. Environmental Protection Agency] considers the site one of the country's worst hazardous waste sites, not only because of [the] extent of the contamination but also because the plant is only a few blocks from a day care center, a hospital, and hundreds of houses." (Oct. 28, 1992, pg. A14.)

By 1989 state and federal officials had made firm plans to build an incinerator in a residential neighborhood of Jacksonville to burn some 22 million pounds of Vertac's dioxin-laced wastes.

On at least two occasions a majority of the citizens of Jacksonville expressed, through referendums and public meetings, that they did not want the incinerator built. Many local people considered it a dirty, dangerous way to "get rid of" the wastes. They pointed out that cleanup teams had already packed the 30,000 barrels in special drums, which were not an immediate threat. The real threat was the wastes already released into the community, the ground and the groundwater. Incinerating the 30,000 barrels would be a cosmetic gesture that did not address the residual problems. State and federal officials ignored these expressions of sentiment and forged ahead with their plan to burn the visible evidence, to make Jacksonville look clean again. The real cleanup of soil and water would have to wait for a later time.

In 1990, Marco Kaltofen, a chemical engineer with the National Toxics Campaign presented Governor Clinton with a White Paper outlining alternatives to incineration, including chemical destruction (dechlorination), and above-ground storage in steel-reinforced concrete buildings.¹ Subsequently in 1991 the U.S. Office of Technology Assessment (an arm of Congress) released a report called *Dioxin Treatment Technologies*, in which they reported the successful destruction of dioxin-containing wastes by chemical dechlorination processes. Chemical dechlorination occurs inside a closed container, releasing nothing to the surrounding environment. Jacksonville has two other Superfund dumps besides the Vertac site, and OTA reported that a chemical dechlorination technology called BCD had been shown to successfully detoxify soils from these other sites. "Test results confirmed that BCD is a candidate technology for the cleanup of halo-carbon-contaminated liquids and soils in an environmentally acceptable manner (closed system)," OTA said.² The term "halo-carbon" in this case referred specifically to 2,4-D, Silvex, 2,4,5-T, and dioxins, which were successfully destroyed by the BCD process. The U.S. Navy has selected BCD technology to clean up its contaminated sites, and has built a BCD decontamination machine which is in use now to clean up PCB-contaminated soils in Stockton, California. State and federal officials in Arkansas turned a blind eye to these alternative technologies.

At public meetings throughout 1989 and 1990 --some of which we attended--state and federal environmental officials insisted repeatedly that the Vertac site incinerator would emit zero dioxin into the

surrounding community.

A trial burn was conducted during October, 1991. State and federal officials examined the data and declared the incinerator a resounding success. But Greenpeace chemist Pat Costner analyzed the trial burn data and published her own analysis, showing that the incinerator had not achieved the required 99.9999% destruction of the wastes, but had in fact achieved only 99.96% destruction. This meant that the incinerator was releasing 400 times as much dioxin as the regulations intended.

State and federal officials studied Costner's analysis and subsequently admitted that she was right. Costner then calculated that the incinerator would release somewhere between 150 and 800 grams of dioxin into the community during the two-year burn. Is this a lot of dioxin? EPA has established a "safe" level of dioxin as 0.000001 micrograms of dioxin per kilogram of body weight per day. If you ate this much dioxin every day for a lifetime and retained it all in your body, you would accumulate a body burden of 1.79 micrograms of dioxin. (A microgram is a millionth of a gram, and there are 28 grams in an ounce.) So we'll call 1.79 micrograms an "EPA safe" dose.

If we say, somewhat arbitrarily, that 10 times this amount represents an "EPA unsafe" dose, we can calculate that the Vertac incinerator will emit somewhere between 8 million and 45 million "EPA unsafe" doses of dioxin into the community of Jacksonville during the two-year burn. State and federal officials say the proposed burn does not violate any state or federal laws and thus should be allowed to proceed. One Arkansas health department official excused the dumping of 150 to 800 grams of dioxin into the community saying, "You have to appreciate how much dioxin there is in this community already." Pat Costner points out that this will be the largest intentional release of dioxin that has ever been executed.

Officials of the Arkansas Health Department say they had nothing to do with choosing incineration to get rid of the Vertac wastes. However, they have made careful plans to take advantage of the experiment by gathering data about dioxin levels in human tissues in Jacksonville residents before and after the burn. The pre-burn study of dioxin in blood and urine of Jacksonville residents will be released some time during the next month or so. After the experimental burn is over, new samples will be taken and comparisons will be made to see what levels of toxins (if any) have lodged in the tissues of the human subjects of the Jacksonville dioxin experiment. "I can say without fear of contradiction, we will have the best database on [dioxin in tissues of] the general population of the U.S. that has ever been developed," Dr. Morris Kranmer, principal investigator of the study, told us. No long-term follow up of health effects in the community has been planned.

Despite massive pressure from national environmental groups and local community organizations, one week before the election, Bill Clinton gave the final order to begin the Jacksonville dioxin burn experiment. The *New York Times* noted, "Unfavorable publicity about the Vertac project contributed to Mr. Clinton's reputation as a leader with a less than sterling environmental record, his aides said. 'He's been beaten up pretty badly over this,' said Kenneth L. Smith, Mr. Clinton's top environmental advisor." (Nov. 2, 1992, pg. B11.)

The *Times* went on: "The Vertac project has become typical of toxic waste cleanups around the country in which costs escalate amid interminable delays caused by Federal rules aimed at gaining public trust. Mr. Smith said the Governor believed that unless changes were made in the rules and the public began to accept some degree of risk, fewer toxic-waste cleanup projects could proceed or ever larger sums of money would be siphoned from the Government's budget for all environmental programs."

No sooner had the Governor given the go-ahead than a coalition of five organizations—including the Washington-based Government Accountability Project—sued in court to stop the experiment. Judge Stephen M. Reasoner ruled October 29 that the experimental burn could go ahead for three days, during which the state must test the incinerator's ability to destroy dioxin with 99.9999% efficiency. If anything less than 99.9999% is achieved, "the Court orders that the burning be stopped immediately," said Judge Stephen M. Reasoner.

The experimental burn occurred over the weekend, but by that time Governor Clinton's attention had been swept up by other matters, namely a nation to which he has solemnly promised that things will now be different.

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[1] Marco Kaltofen and Sanford J. Lewis, *A White Paper on the Feasibility of Alternatives to Incineration of Wastes at the Vertac Site in Jacksonville, Arkansas* (Boston: National Toxics Campaign Fund, October 27, 1990).

[2] U.S. Congress, Office of Technology Assessment, *Dioxin Treatment Technologies--Background Paper* [OTA-BP-O-93] (Washington, D.C.: U.S. Government Printing Office, November, 1991).

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NEW EPA MEMO SAYS ALL HAZARDOUS WASTE INCINERATORS FAIL TO MEET REGULATIONS

An internal memo sent to all 10 regional offices of EPA [U.S. Environmental Protection Agency] by Sylvia Lowrance, EPA Director of Solid Waste, confirms that hazardous waste incinerators cannot meet EPA requirements for near-total destruction of hazardous wastes. (See *RHWN* #280.)

EPA's incinerator regulations require 99.99 percent destruction of all hazardous wastes and 99.9999 percent destruction of especially-hazardous wastes such as PCBs and dioxins. The Lowrance memo dated Sept. 22, 1992, and interviews with Sonya Sasseville of Ms. Lowrance's staff, confirm that the agency possessed scientific information as early as 1984 showing that hazardous waste incinerators cannot destroy some of the most dangerous wastes as completely as the regulations require.

EPA possessed this information but chose to ignore it when hazardous waste incinerator regulations were established for dioxin in 1985.

These new revelations cast doubt on the safety of all hazardous waste incinerators, and could conceivably lead to charges of criminal wrongdoing by some EPA officials. The agency has been touting incineration as "safe" for more than a decade. When asked, agency officials define "safe" as "in compliance with all regulations." In sum, the agency established regulations in 1985 knowing no incinerator could comply, and now the agency's own logic forces the conclusion that no hazardous waste incinerator can be operated safely. It would appear to expose the agency to liability claims by anyone believing they have been harmed by incinerator emissions.

EPA's acknowledgement of its malfeasance surfaced during an incinerator battle in Jacksonville, Arkansas. For the past decade Arkansas Governor Bill Clinton and the citizens of Jacksonville, have been battling each other over the Governor's plan to burn dioxin-contaminated chemical warfare agents in a residential neighborhood of Jacksonville. (See *RHWN* #311.) EPA officials in Region 6 (Dallas, Texas) supported the Governor's plan.

The Jacksonville wastes contain an estimated 75 pounds (34 kilograms) of pure dioxin, a poison that kills laboratory animals such as guinea pigs exposed to only a few micrograms, making it one of the most powerful poisons ever found. From 1988 onward, federal and state environmental officials in Jacksonville said publicly on numerous occasions that an incinerator could destroy dioxin with 99.9999 percent efficiency, thus eliminating all health threats to the surrounding community. The Lowrance memo makes it clear that Region 6 EPA officials were either lying or were kept ignorant by officials at EPA headquarters in Washington who knew the truth.

EPA's regulatory failure was discovered when an independent researcher, chemist Pat Costner of Greenpeace, analyzed government data from the Jacksonville incinerator as it was being tested before startup. In early 1992, Costner analyzed government data collected during an October, 1991, trial burn in Jacksonville. Her analysis revealed that instead of 99.9999 percent ("six nines") destruction, the Jacksonville incinerator had achieved only 99.96 percent destruction of dioxin. Federal and state officials confirmed her analysis. At that rate the Jacksonville incinerator would release 400 times as much dioxin as the regulations say it should.

How the Regulations Work

EPA's hazardous waste regulations require the owner/operator of a new incinerator to select several POHCs (principal organic hazardous constituents)—chemicals to be destroyed. The selected POHCs must be harder to burn than dioxin. The POHCs are "surrogates" for dioxin—they "stand for" dioxin or "represent" dioxin during the test. During a "trial burn," the POHC surrogates are fed into the incinerator in nearly pure form under ideal laboratory conditions, and the incinerator's ability to destroy them is measured. If a destruction/removal efficiency (DRE) of 99.9999 percent is achieved with the POHCs, then EPA allows the owner/operator to assume that 99.9999 percent of dioxin will also have been destroyed. It is this assumption that EPA has known since 1984 is false.

The trial burn procedure was followed precisely in the Jacksonville case, with one exception. The owner/operator inadvertently burned some actual dioxin along with the POHCs during the trial burn and dutifully reported the DRE for the POHCs, but did not analyze the data to establish a DRE

for dioxin. Costner did the calculation for dioxin and revealed that dioxin was not destroyed with an efficiency anywhere near six nines.

Since Region 6 officials had been promising for several years that the Jacksonville incinerator would destroy dioxin with six nines efficiency, Costner's analysis made them look like fools or liars or both. Region 6 called headquarters for guidance and on September 22, Sylvia Lowrance sent out a memo telling regional EPA offices how to handle this embarrassing situation.

The Lowrance memo says, in part, "The low dioxin DRE in this recent [Jacksonville] case was consistent with our current body of incinerator performance data, which show a very clear trend of decreasing DRE for hazardous constituents with decreasing incoming concentration of the constituents in the waste feed. (That is, the lower the constituent concentration in the waste, the lower the DRE.) The data show that a properly operating incinerator, which reached 99.99% DRE (four nines) on higher concentrations of POHCs, will often achieve less than four nines when the concentration of a POHC (principal organic hazardous constituent) in the waste is less than 1000 ppm [parts per million]. At this time we have not established a definitive scientific explanation for this phenomenon," the memo says.

The Lowrance memo goes on to point out that, in establishing regulations for incineration of dioxin-contaminated wastes, in 1985, EPA relied on risk assessments in which the agency assumed that 99.9999 percent destruction was routinely achieved. "For this reason, the risk assessment calculations performed in the course of the rulemaking may not be representative in some cases," the Lowrance memo says. In sum, the entire superstructure of regulations created for dioxin incineration in 1985 was based on assumptions that the agency knew at the time were false. Indeed, in an internal EPA memo dated October 24, 1985, Robert A. Olexsey, who was at the time an employee of EPA's Hazardous Waste Environmental Research Laboratory, wrote "We have a problem with the 'surrogate POHC' approach for the determination of the dioxin destruction efficiency. In our incinerator and boiler field tests, we found a consistent relationship.... In essence, across the entire test program, POHC DRE increased with increasing POHC concentration in the feed. If this relationship holds for dioxin (we see no reason why it would not), reporting the DRE for the dioxin material as being identical to that of the higher concentration surrogate will result in overstating the DRE for the dioxin waste." Olexsey went on to recommend that dioxin itself be measured during incinerator tests, to check the efficiency of destruction, rather than testing a POHC and assuming that it revealed something about dioxin. Olexsey's advice was not followed.

During 1984-1985, John C. Kramlich of the Energy and Environmental Research Corporation (Irvine, Cal.) completed a contract study for EPA, analyzing the failure of hazardous waste incinerators to destroy wastes. EPA did not publish the Kramlich study until 1989. Kramlich wrote, "[Our] results indicate that current technology has difficulty meeting the licensing regulations when the waste represents less than 1000 ppm [parts per million] of the feed stream. This finding has significance with respect to waste streams contaminated by low concentrations of extremely hazardous materials (e.g. dioxin or chlorophenol contaminated pesticides)."¹

EPA's data reveal that all incinerators fail in the same way, but the public health hazard seems especially great at sites burning wood-preserved wastes, pesticides, PCBs, pulp and paper mill sludges, or dioxins. All contaminated-soil incinerators, all Superfund cleanup incinerators, and all of the Army's proposed chemical weapons incinerators are also cast into doubt by EPA's recent admissions. Furthermore, all of the agency's risk assessments and rulemakings regarding hazardous waste incinerators are now known to have been based on false assumptions. In short, the entire regulatory structure intended to guarantee the protection of public health and safety from hazardous waste incinerators has now been thrown into grave question.

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[1] John C. Kramlich and others, *Experimental Investigation of Critical Fundamental Issues in Hazardous Waste Incineration* (Springfield, VA: National Technical Information Service [NTIS], September, 1989.) This is EPA document No. EPA/600/2-89/048 available from NTIS for \$26.00; phone (800) 553-6847 and request NTIS document No. PB90-108507. See pgs. 5-1, 5-2.

[Dated announcement omitted from this reprint.]

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Reprint of:

RACHEL'S HAZARDOUS WASTE NEWS #314

Providing news and resources to the Movement for Environmental Justice -- December 2, 1992

CEMENT AND KILN DUST CONTAIN DIOXINS

During routine preparation of a *Report to Congress on Cement Kilns*, the U.S. Environmental Protection Agency (EPA) has learned that cement and cement kiln dust contain dioxins and furans (both of which are powerful poisons in animals and humans), according to a briefing document dated October 8, 1992, prepared by EPA staff for EPA's Director of Solid Waste, Sylvia Lowrance. The October briefing document also says 20 percent of the cement kiln dust that EPA tested contains the non-natural radioactive elements plutonium-238, plutonium-239 and cesium-137. Dioxins are the most powerful carcinogens (cancer-causing agents) ever tested in laboratory animals; plutonium is the most potent carcinogen in humans ever discovered.

Cement is a principal component of pipe often used to distribute drinking water in many American cities. Cement kiln dust is a byproduct of cement manufacture and is routinely given or sold to farmers as a soil treatment, or is discarded into pits or is piled on the ground near cement kilns in an uncontrolled fashion. According to Bill Schoenborn, an EPA staff member working on the *Report to Congress*, about 6 million tons of kiln dust is disposed of each year by cement kilns, 5.1 million tons of it buried on-site, and 900,000 tons of it shipped off-site for use in stabilizing other wastes (such as sewage sludge) or as a soil additive on farms. Cement kiln dust has previously been reported to contaminate groundwater with the toxic metals lead and chromium,¹ but until now no one has reported dioxins, furans, plutonium or cesium-137 in cement or cement kiln dust.

The *Report to Congress* is required by the federal Resource Conservation and Recovery Act (RCRA), the nation's basic hazardous waste law. Like mine wastes, cement kiln dust was initially exempt from RCRA because it is a high-volume waste presumed to be low in toxicity. Cement clinker (that is to say, cement itself) is exempt from RCRA because it is a product, not a waste. Section 8002(o) of RCRA required EPA to study cement kiln dust and to write a report for Congress on its findings. For several years, EPA dragged its feet preparing the report. Then Environmental Defense Fund (EDF) brought a lawsuit, and now EPA is under a court order to finish the report by April, 1993.

In the course of preparing the *Report to Congress*, EPA randomly selected 15 cement manufacturing plants (called kilns) for sampling, out of the 114 such plants presently operating in the U.S. Of the 15 plants sampled, eight burn hazardous waste as fuel and seven do not. In recent years, cement kiln operators have increasingly been using hazardous waste as fuel, to reduce fuel costs and thus increase profitability. The practice has proved controversial. (See *RHWN* #174 and #243.) Opponents of the practice say they fear cement will become contaminated with industrial poisons. Cement is a key raw material in concrete pipe for water delivery systems, and in concrete block and other concrete materials used in construction of private homes, commercial dwellings, public buildings, bridges and highways. Seventy to 80 million tons of cement are produced in the U.S. each year, depending on market demand.

Sampling Results

EPA took 15 samples of "clinker" (the product of a kiln, from which cement is made), plus 28 samples of dust (the unwanted byproduct of a kiln). All samples were analyzed for metals, chloride, cyanide, fluoride, total sulfate, total organic carbon, moisture content, and radioactive elements.

Samples from six kilns (4 burning hazardous waste, 2 not burning hazardous waste) were tested for dioxins and furans, volatile organic compounds, semivolatile organic compounds, and pesticides. All chemical analyses were completed by EPA's National Air and Radiation Environmental Laboratory (NAREL) in Alabama.

Dioxins and furans were detected in all samples of "clinker" and all samples of kiln dust analyzed for these compounds. The October briefing document says that the dioxin molecule known as 2,3,7,8-TCDD, the most potent poison in the dioxin family, was only identified in samples from kilns burning hazardous waste. Other dioxins were found in samples from kilns not burning hazardous waste, but no 2,3,7,8-TCDD. However, the October briefing document says it is not possible to generalize these differences to the entire 114 operating cement kilns.

Samples of cement kiln "clinker" did not contain pesticides or semivolatile organics. Clinker was not analyzed for volatile organics. On the other hand, cement kiln dust contained amounts of the volatile organics benzene and acetonitrile that exceeded RCRA limits "in a number of the samples of hazardous waste burners" but not in samples from kilns not burning hazardous wastes. The dust from

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one kiln not burning hazardous waste proved to be high in methylene chloride, according to the October briefing document.

These findings lend support to the view that burning hazardous waste in a cement kiln increases the amount and potency of toxins in the resulting cement kiln dust and perhaps in the cement itself.

At three kilns (2 burning hazardous waste, one not burning hazardous waste) levels of naturally-occurring radioactive radium-226 exceeded the cleanup standard for uranium mine and mill wastes (the standard being 5 picoCuries per gram). Cesium-137, a non-natural radioactive element, was present in the dust of 26 percent of the kilns tested (4 out of 15)—one hazardous waste burner and three non-hazardous waste burners. Plutonium-238 and plutonium-239 were detected in kiln dust samples from 3 of the 15 kilns tested. Each of these 3 facilities is "located near a DOE [U.S. Department of Energy] nuclear weapons production/testing facility," according to EPA's October briefing document. Plutonium and cesium-137 do not occur in nature but are created by nuclear bomb explosions and in nuclear power reactors.

A second EPA briefing document dated November 24, 1992, contains additional information about the problem of potent toxins being found in cement and in cement kiln dust. The document is titled "OSW Office Briefing on Cement Kiln Dust Risk Screening" and it contains a summary of a risk assessment that is being conducted by the EPA's Communications and Budget Division within the Regulatory Analysis Branch, Office of Solid Waste.

The November briefing document outlines two risk assessment scenarios: one in which cement dust blows off-site and affects a person living 750 feet from an active waste pile, and a second in which an individual is presumed to be living on top of an abandoned waste pile. No risk assessment was reported for the case of a farmer growing crops in soil to which cement kiln dust has been added.

Furthermore, no risk assessment is reported for the dioxins and furans measured in cement clinker, which it to say, in cement itself.

Based on the two risk assessment scenarios, the November briefing document describes amounts of toxins in cement kiln dust that appear to be acceptable, which is to say will only give cancer to one in 100,000 individuals so exposed. The November document lists 22 instances in which one or more EPA tests of cement kiln dust exceeded the criteria developed in the risk assessments. Criteria that are exceeded by one or more samples include: 2,3,7,8-TCDD, total dioxins, total dioxins and furans, total hexachloro dioxins, arsenic, beryllium, cadmium, chromium, lead, and thallium, plus the following radioactive elements: bismuth-214, cesium-137, potassium-40, lead-212, lead-214, radium-226, radium-228 and thorium-227.

The purpose of the risk assessments reported in the November document is to help EPA decide whether the agency needs to regulate cement kiln dust as a legally hazardous waste or not. Declaring cement kiln dust a legally hazardous waste would greatly increase the cost of waste disposal for some cement kilns, and thus might reduce the profitability of some kilns.

EPA employee Hugh Kaufman has previously charged that the agency has been "accommodating the regulated cement kiln hazardous waste incineration industry with nonexistent, or at best loose, regulation..."²

Now that EPA has found dioxins in cement clinker, and dioxins and radioactive elements in cement kiln dust, the agency will likely come under considerable pressure to regulate all cement kiln wastes as hazardous wastes.

For their part, citizens seem likely to start asking themselves anew whether kilns can be good neighbors.

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[1] Jeffrey D. Smith, "Cement Kilns 1991," *EI Digest* (August, 1991), pgs. 20-32.

[2] Kaufman made his charge in a letter to EPA chief William Reilly dated Dec. 7, 1990; on February 21, 1991, cement kilns burning hazardous waste became regulated under the so-called "BIF" (boiler and industrial furnace) regulations, which can be found in the *Federal Register* February 21, 1991, pgs. 7134-7240. See also *Federal Register* July 17, 1991, pgs. 32688-32692; August 27, 1991, pgs. 42504-42517; September 5, 1991, pgs. 43874-43877; and August 25, 1992, pgs. 38558-38566.

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Reprint of:

RACHEL'S HAZARDOUS WASTE NEWS #325

Providing news and resources to the Movement for Environmental Justice -- February 17, 1993

A SEA OF TROUBLES ENGULFS INCINERATION

The incineration industry has suffered a series of major setbacks in recent weeks. The federal government's showcase dioxin-burning incinerator in Jacksonville, Arkansas--to which Bill Clinton personally gave a green light one week before he was elected President (see *RHWN* #311)--was shut down last week by a federal judge. Lawyers for EPA (U.S. Environmental Protection Agency) admitted to U.S. District Judge Stephen Reasoner in Little Rock that the Jacksonville incinerator could not destroy 99.9999% of the dioxin fed into it, as is required by EPA regulations, whereupon the judge ordered the plant shut.

It was a stunning victory for Greenpeace chemist Pat Costner, who had been the first to reveal that incinerators could not achieve 99.9999% destruction (see *RHWN* #312 and #280), and for local activist-citizens like Sharon Golgan in Jacksonville who had fought the incinerator proposal for at least six years.

The nation's other showcase incinerator--built by Waste Technologies Industries (WTI) on the banks of the Ohio River in East Liverpool, Ohio--was fighting for its life in federal court in Cleveland earlier this week. Local citizens there have been opposing the WTI incinerator proposal for 12 years. EPA's Region 5 office in Chicago and Ohio state officials bent the rules, and even broke a few, to give WTI a permit to start burning wastes (see *RHWN* #287). The state of West Virginia, local citizens, and Greenpeace filed a lawsuit January 13 asking a federal judge to prevent WTI from conducting a "test burn."¹ As soon as a test burn is completed, the WTI incinerator can begin commercial operation for up to a year while EPA evaluates the test results. (Vice-President Al Gore said December 7, 1992, that he and Mr. Clinton would stop WTI from proceeding until a study was completed of all health issues, and all legal issues surrounding EPA's issuance of a permit [see *RHWN* #315]--but it now appears that Mr. Gore has changed his mind.) Federal Judge Ann Aldrich in Cleveland issued a temporary restraining order against WTI January 15 and set February 16 as the day she would decide whether to issue a preliminary injunction, which would prevent WTI from conducting the trial burn until a full court trial could be held.²

As we go to press, it is not clear how this phase of the battle will come out. However, secret EPA documents that came to light during in court have raised serious health questions about every incinerator in the country, including solid waste incinerators.

Background

At issue in every instance is dioxin, which is created as an unwanted byproduct of incineration (and as a byproduct of other industrial processes, such as pulp and paper manufacture, and the manufacture of some pesticides). In April, 1991, EPA began a major "scientific reassessment" of dioxin and discovered that effects of dioxin can be observed in human cells at the levels of exposure now present in the environment. In other words, there's already sufficient dioxin in the environment to produce observable effects in humans. The Clinton/Gore administration knows this is a problem because a December, 1992, briefing document prepared by the EPA Transition Team says, "This [EPA's dioxin reassessment] is likely to be an extremely controversial document as there are new findings indicating adverse reproductive effects at existing environmental levels."

A Secret Risk Assessment and a Leaked Memo

EPA's position throughout the 1980s has been that incineration is safe, even though every incinerator is known to produce dioxin. To "prove" the safety of incinerators, EPA has used a technique called "risk assessment." A risk assessment estimates the amount of dioxin being released, estimates various pathways it might travel through the environment, and calculates the resulting exposures of humans. Finally, a risk assessment estimates the health effects resulting from the calculated exposures.

In the case of dioxin, over the years EPA's "standard" risk assessment has assumed that airborne dioxin only enters humans through their lungs. Dioxin that falls to the ground and is then incorporated into the food chain and eaten has always been ignored in EPA risk assessments.

However, the EPA's team of scientists conducting the official "reassessment" of dioxin's toxicity published a draft report last summer called, *Estimating Exposure to Dioxin-like Compounds* in which they clearly stated that a proper risk assessment for an incinerator must include all routes of exposure for dioxin, not merely via the lungs.³ It is well known that dioxins accumulate in the food chain, and that meat, milk and fish are the major sources of dioxin exposure for humans.⁴

When Greenpeace researcher Joe Thornton did his own risk assessment on the WTI incinerator, using the technique recommended in EPA's draft report, including dioxins in beef and milk, he found that WTI posed risks 10000 times higher than EPA had calculated. To counter Thornton, EPA did its own food-chain risk assessment, which was not released to the public, but which came to light in court.⁵ The EPA's secret risk assessment concludes that dioxin from WTI is 1000 times more dangerous than the "official" published EPA risk assessment says it is.

Leaving aside the serious ethical issue of EPA refusing to publish important health and safety information about WTI, an internal memo from Richard Guimond, acting chief of EPA's Office of Solid Waste and

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Emergency Response, dated January 22, 1993, leaked to Greenpeace, says, "There are very serious implications associated with adopting risk assessment procedures based on indirect exposure routes for air emission sources."⁶ Translation: if food-chain exposures are now to be counted in incinerator risk assessments, many incinerators will be found to be unacceptably dangerous.

The new understanding of dioxin--that it's already present in the environment at levels that affect humans--plus the inclusion of food-chain exposures in risk assessments, plus the failure of incinerators to achieve the destruction efficiencies required by regulations--all seem to add up to a mushrooming debacle for the incineration industry.

What are the alternatives to incineration?

Jacksonville holds the key

EPA scientists have known since 1985 that incinerators cannot achieve 99.9999% efficiency in destroying wastes present in low concentrations (see *RHWN* #280 and #312.), but EPA officials have stated at hundreds of public presentations since 1985 that 99.9999% could be achieved. Lying to the public carries no penalty, but lying to a judge is a different matter. At the hearing in Little Rock February 12, EPA was represented by U.S. Justice Department lawyer Ron Spritzer. Judge Reasoner said to Spritzer, "Indulge me for a moment. If I asked you to prove that you could achieve a six 9 DRE [99.9999% destruction and removal efficiency] on dioxin, could you physically produce technological data that shows that?"

"No sir, we could not," said Mr. Spritzer.⁷

That damaging admission was sufficient for the judge. He ordered the plant shut. EPA is considering appealing the judge's verdict, but they do not appear to have a strong case. In all likelihood, the Jacksonville incinerator is shut for good.

The Jacksonville incinerator was in considerable trouble even before Judge Reasoner's decision. On January 22, an Arkansas state official revealed that the incinerator was producing a larger volume of hazardous waste than it was destroying. The machine had been set up in a residential neighborhood of Jacksonville to "destroy" 30,000 drums of hazardous liquids left over from a defunct chemical factory that had made chemical warfare agents on the site for many years (most recently, herbicides for Vietnam). Local citizens had recommended moving the waste out of town by rail, or simply building a concrete mausoleum on the site to contain the waste safely until someone found a way to detoxify it. But Arkansas and federal EPA officials insisted that incineration was the safest, cheapest solution to the problem. They convinced then-Governor Bill Clinton to put up \$10.7 million of state money to build and operate the incinerator.

At the end of a year's burning, 9,600 drums of waste had been "destroyed" by the Jacksonville incinerator, but in the process the incinerator had created 12,000 drums of salt and another 1730 drums of ash (13,730 drums total) for a net gain of 43% in the volume of waste. Furthermore, the salt and the ash are so laced with dioxin that they are legally a "hazardous waste" and thus cannot be taken off the site.

"We did not anticipate this," said Doug Szenher, a spokesperson for the Arkansas state department of Pollution Control and Ecology. "The whole idea was that the salt and ash was to have been de-listed [declared non-hazardous] and taken to a landfill site. It just didn't work out that way," he said.⁸

So after spending \$7 million burning chemicals fruitlessly for a year, EPA is now spending \$400,000 to build a 30,000-square-foot building on the site, to store the hazardous salt and ash until someone can figure out how to detoxify it. So far as we know, this is the first above-ground mausoleum built to store hazardous waste. (See *RHWN* #260.) In Jacksonville, above-ground concrete storage will soon be a real, demonstrated alternative to incineration.

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[1] *Greenwire* staff, "WTI Incinerator: Foes File Suit in Effort to Block Test," *Greenwire* Vol. 2 No. 171 (January 14, 1993). *Greenwire* is "the daily executive briefing on the environment" available on-line; phone (703) 237-5130.

[2] *Greenwire* staff, "Incinerator: WTI Counters with Suit of its Own," *Greenwire* Vol. 2 No. 172 (January 15, 1993). *Greenwire* is "the daily executive briefing on the environment" available on-line; phone (703) 237-5130.

[3] U.S. Environmental Protection Agency, *Estimating Exposure to Dioxin-Like Compounds [EPA/600/6-88/005B] Workshop Review Draft* (Washington, D.C.: U.S. Environmental Protection Agency, August, 1992).

[4] U.S. Environmental Protection Agency, cited above, Appendix B.

[5] Memo from William Farland, director of EPA's Office of Health and Environmental Assessment, to Brian Grant, U.S. Department of Justice, "WTI Screening Level Analysis," dated Feb. 8, 1993, attaching a 21-page risk assessment called "Screening Level Analysis of Impacts From WTI Facility," dated Feb. 5, 1993.

[6] Memo from Richard Guimond, Acting Assistant Administrator, Office of Solid Waste and Emergency response, to EPA Administrator Carol Browner, "WTI Incinerator Issues," dated January 22, 1993. 2 pgs.

[7] Sandy Davis, "Judge Halts Dioxin Burn at Vertac," *Arkansas Democrat Gazette* February 13, 1993, pgs. 1A, 13A.

[8] Sandy Davis, "At vertac, more waste rolls out than in," *Arkansas Democrat Gazette* January 24, 1993, pg. 12A.

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Studies on Dioxin

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RACHEL'S **HAZARDOUS WASTE NEWS #73**

Providing news and resources to the Movement for Environmental Justice -- April 18, 1988

STUDY OF DIOXIN-EXPOSED HUMANS REVEALS CANCER, BIRTH DEFECTS, LIVER AND CARDIOVASCULAR DAMAGE

A new study of Vietnam veterans, conducted by Air Force physicians, links dioxin exposure to increases in cancer, birth defects, psychological damage, liver damage, cardiovascular deterioration, and degeneration of the endocrine system. The new work stops short of saying dioxin exposures caused the observable health damage among dioxin-exposed vets, but it explicitly reverses the conclusions of a 1984 Air Force study which said dioxin exposures had been shown to be harmless.

The study is continuing, and will begin using sensitive new blood tests to determine past exposures to dioxin; future study may be able to pin down, or rule out, direct causal effects. In the meantime, advocates of mass burn incinerators and other dioxin-producing facilities can no longer claim lack of evidence that dioxin harms humans. There is now tangible cause for concern about serious effects of dioxin on humans.

The new work is part of the Air Force's ongoing study of Vietnam veterans who participated in Operation Ranch Hand, which was a military program spraying herbicides on the jungles of Vietnam from 1961 to 1972, to defoliate and thus eradicate Viet Cong hiding places. In manufacture, the herbicides (Agents Orange, Pink, Green and Purple) were contaminated with dioxin (33 ppm to 66 ppm [parts per million]).

The study compares two groups of Vietnam veterans--one group of 1045 Ranch Hands who definitely had dioxin exposures and another group of 773 veterans not known to have been exposed to dioxin. In addition, 2708 wives and former wives of veterans in the two groups participated in the study.

The study team first reviewed more than 400 published articles on the effects of dioxin on animals and humans; from this they derived a profile of 190 different health effects that might be expected to be related to dioxin exposure. They examined the 1818 study participants for these 190 health effects.

Cancer

The study found that 4.59% of the Ranch Hands have some kind of cancer, compared to 2.33% of the unexposed group. Thus the overall risk of cancer among the dioxin-exposed group is doubled (risk increased by a factor of 1.97). The greatest risk increase is for skin cancers (where the risk is increased by a factor of 2.6), whereas the risk of "systemic cancers" (non-skin cancers) is increased by a factor of 1.2; in other words, the dioxin-exposed group has a 20% greater chance of getting a non-skin cancer.

Birth Defects

Analyzing for birth defects, the study looked at children born before the Vietnam experience and children born after Vietnam. Prior to Vietnam, the dioxin-exposed group had born 85% as many children with birth defects as the non-exposed group; after Vietnam, the exposed group bore 139% as many children with birth defects. The earlier Air Force study had said birth defects among dioxin-exposed families were limited to "minor skin lesions" but the new study reverses that conclusion; 32 children with severe defects were born to families in the exposed group, vs. 18 in the non-exposed group. The total number of birth defects in the two groups was: 80 with defects out of 917 total births in the exposed group vs. 48 with defects out of 744 total births in the non-exposed group.

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Psychological Damage

Psychological testing revealed significant increases in fatigue, anger, anxiety, and isolation among the dioxin-exposed group compared to the non-exposed group.

Liver Functions

The new study looked at nine chemical measures of liver function and in three categories the dioxin-exposed group showed reduced liver functions, compared to the non-exposed group. In addition, among the exposed group, 16 showed enlarged livers, vs. six among the non-exposed group. Furthermore, 13 among the exposed group had a verifiable medical history of liver disorder other than hepatitis, jaundice, or cirrhosis, vs. only two with such histories among the non-exposed group.

Cardiovascular system

Heart disease rates and heart attack rates did not differ among the two groups. However, during physical examination, 10 different heart pulse measurements were taken in the extremities (e.g., the ankle), and statistically significant abnormalities were found in one or more pulses in 12.8% of the exposed group vs. 9.4% of the non-exposed group. Abnormal pulses in the extremities are evidence of blood circulation problems.

Endocrine system

The endocrine system is a body control system composed of a group of glands that maintain a stable internal environment by producing chemical regulatory substances called hormones. Glands that participate in the endocrine system include the pituitary, thyroid, parathyroid and adrenal glands, as well as the pancreas, ovaries and testicles. The new study looked at five chemical measures of endocrine system functions. In three of the five measures, the dioxin-exposed group showed abnormal functioning of the endocrine system, compared to the non-exposed group. Functioning of the endocrine system reduces with age, but the new study showed that, among the dioxin-exposed group, functioning of the endocrine system is being reduced much faster than among the non-exposed group.

Thus the new study shows that, in six out of 11 areas of suspected dioxin effects, exposed Vietnam veterans have health problems in greater proportion than the comparison group.

The Ranch Hand Study (formally known as the Air Force Health Study [AFHS]) began in 1979 and is continuing. The most recent publication, reviewed above, is: Richard A. Albanese, *United States Air Force Personnel and Exposure to Herbicide Orange, Interim Report for Period March 1984 - February 1988* (United States Air Force: Brooks Air Force Base, TX, Feb., 1988). The study is 34 pages long; it is available from Dr. Albanese, USAF School of Aerospace Medicine, Human Systems Division (AFSC), Brooks Air Force Base, TX 78235-5301; phone (512) 536-3884. Our thanks to Laura Petrou, legislative assistant to U.S. Senator Thomas Daschle (South Dakota) who made the study available to us. Ms. Petrou can be reached at (202) 224-2321.

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Providing news and resources to the Movement for Environmental Justice -- March 14, 1989

DANGERS OF DIOXIN EXPOSURES: ABSORPTION THROUGH THE SKIN

Traditionally, people concerned about the toxicity of chemicals have mainly worried about the mouth and lungs as a means of entry into the human body. Now new evidence suggests that absorption through the skin may be an important way for some chemicals to enter the body. In addition, the skin of babies may allow more toxics to pass through it than the skin of older humans.

Researchers at the National Institute of Environmental Health Sciences studied the absorption of dioxins and furans in mice and rats. They discovered several new aspects of chemical absorption by the skin: (1) the skin presents a more effective barrier against some chemicals than against others; (2) mice absorb a greater percent of dioxin when lower doses are administered than when higher doses are administered; (3) young adult rats absorbed a greater percentage of the administered dose than did middle-aged rats.

In the past, the theory has been that the skin (which has a total area of 1.8 square meters in the adult human) has served as a passive barrier to chemicals. Now it is apparent that the skin is very active in metabolizing (biologically altering) chemicals and that these metabolic processes affect the way the body absorbs (or does not absorb) a particular chemical. Sweat glands, sebaceous glands (which produce oils), and hair follicles can all contribute to the way chemicals are absorbed through the skin.

Chemicals administered at low doses are more effectively absorbed through the skin than are chemicals administered at high doses. Mice receiving 0.3 micrograms of dioxin per kilogram of body weight absorbed 40% of the dose; mice receiving 32 to 320 micrograms of dioxin per kilogram of body weight absorbed less than 20% of the dose. This may be important for human exposures, which usually occur at low doses over long periods rather than in high doses over short periods.

Three month old rats (young adults) absorbed 16% of dioxin applied to their skin; nine-month-old rats (middle-aged) absorbed less than 5% of a similar dose.

Linda Birnbaum, who directed the research, says that her work shows that acute toxicity from skin exposure to dioxins and furans is "unlikely." Chronic (long-term) toxicity is a different matter: That's where "you're going to have the potential to build up a body burden" of the toxic chemicals, she says. Her work with young rats also concerns her because there is evidence that the skin of human babies is much more permeable than skin of adults.

The importance of this work for grass roots activists seems to be this: if someone is going to expose your community to small amounts of dioxins and furans for a long time, ask them to please consider absorption through the skin, and especially so in the case of babies and children. Any risk assessments that have been done without considering skin absorption should be redone in light of the new findings.

Get: David Brewster and others, "Comparative Dermal Absorption of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Three Polychlorinated Dibenzofurans." *Toxicology and Applied Pharmacology*, Vol. 97 (January, 1989), pgs. 156-166. Reprints are free from: Linda S. Birnbaum, Systemic Toxicology Branch, National Institute of Environmental Health Sciences, Research Triangle, North Carolina 27709; phone (919) 541-3212. Ask Ms. Birnbaum for a copy of her unpublished paper on absorption of dioxins by young rats, which she presented at a meeting of the Society of Toxicology in Atlanta, GA, the week of Feb. 27, 1989.

C00127

NIOSH ISSUES CANCER ALERT FOR DIESEL EXHAUST FUMES

One reason why people often oppose a new facility is increased truck traffic. Trucks are big and noisy and subject to accidents, and when accidents occur involving trucks, there's a high likelihood of fatalities.

However, the federal government has recently concluded officially that there is another good reason to be concerned about increased truck traffic in your neighborhood: five separate studies in the last 3 years have shown that diesel exhaust certainly causes cancer in laboratory animals, and two studies of railroad workers show that it causes cancer in humans as well. As a result of this determination, the National Institute for Occupational Safety and Health (NIOSH) has issued a special publication, *Carcinogenic Effects of Exposure to Diesel Exhaust*, offering this recommendation: "As prudent public health policy, employers should assess the conditions under which workers may be exposed to diesel exhaust and reduce exposures to the lowest feasible limits." Citizens may reasonably ask: if NIOSH believes workers should not be exposed to diesel exhaust because of the cancer hazard, can health officials in other parts of government believe that the general public should continue to be exposed to diesel exhaust? Taken in this light, risk assessments that discuss only the traffic hazards associated with a facility are missing the major point: diesel trucks can evidently kill innocent people even if no traffic accidents occur.

Diesel engines are more efficient than gasoline engines; they produce more horsepower per gallon of fuel, and they use a less-refined (thus cheaper and more plentiful) fuel. When diesel fuel burns in an engine's combustion chamber, the resulting exhaust contains gases and particles (soot). The gases include nitric oxide, nitrogen dioxide, oxides of sulfur, and hydrocarbons (e.g., ethylene, formaldehyde, methane, benzene, phenol, 1,3-butadiene, acrolein, and polynuclear aromatic hydrocarbons [PAHs], several of which are known carcinogens). Of the particles in diesel exhaust, 95% are less than 1 micron in diameter and thus they are respirable, which is to say they are easily taken into the deepest portions of the human lung where they may lodge forever. The core of each particle is made up of pure carbon, but as many as 18,000 different chemicals from the gaseous portion of the exhaust may be adsorbed (attached) onto the carbon core, and thus diesel exhaust can carry a whole host of exotic, toxic and carcinogenic chemicals into the deepest portions of your lung--down in the region where the transfer of gas occurs to put oxygen into your blood stream and to take carbon dioxide out.

As recently as 1986, NIOSH concluded that diesel exhaust did not cause cancer in laboratory animals. However, in the period 1986-1988, five long-term animal studies, and two epidemiologic studies of humans, all concluded that exposure to diesel exhaust causes lung cancer. As a result, NIOSH reversed itself and in August, 1988, issued a special "current intelligence bulletin" to get the word out that diesel fumes are dangerous. NIOSH estimates that 1.35 million American workers are routinely exposed to diesel exhausts.

Get: *Carcinogenic Effects of Exposure to Diesel Exhaust* [Current Intelligence Bulletin 50; DHHS (NIOSH) Publication No. 88-116]. Cincinnati, OH: Division of Standards Development and Technology Transfer, NIOSH, Robert A. Taft Laboratories [4676 Columbia Parkway, Cincinnati, OH 45226], August, 1988; phone (513) 533-8287. It's 30 pages and free.

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Reprint of:

RACHEL'S HAZARDOUS WASTE NEWS #171

Providing news and resources to the Movement for Environmental Justice -- March 7, 1990

Dioxin--Part 1 DIOXINS AND CANCER: FRAUDULENT STUDIES

For years, industry scientists have been claiming there's no evidence that dioxins cause cancer in humans. Now there is mounting evidence that such claims rely heavily on studies that are fraudulent. Two companies recently accused of producing fraudulent dioxin-and-health data are Monsanto and BASF.

Monsanto

A scientist with U.S. Environmental Protection Agency (EPA) says Monsanto falsified data in important studies that Monsanto used to support its claim that dioxin does not cause cancer in humans. Dr. Cate Jenkins, a chemist in EPA's Office of Solid Waste and Emergency Response, says EPA itself relied upon Monsanto's fraudulent data in setting health standards for dioxin, and Jenkins has asked the EPA's Science Advisory Board to reopen the matter of EPA's dioxin standards, to take a fresh look at available data.¹

Two important sources of dioxins in the environment are old chemical dumps and the incineration of municipal solid wastes,² which is why EPA is concerned about allowable levels of human exposures to dioxin.

BASF

The British technical journal, *New Scientist*, says, "A new analysis by a West German epidemiologist may have established the first clearcut evidence of a direct link between exposure to dioxins and cancer in humans. Friedemann Rohleder, an independent specialist, has produced a report detailing an unexpectedly high incidence of cancer among workers exposed to dioxins during an industrial accident at a chemicals plant in 1953."³

"The plant, operated by the West German company BASF, made trichlorophenol. Rohleder claims the company presented the data in a way that disguised the cancers," says *New Scientist*.

Background

Each of these claims of fraud relates to an industrial accident in which workers were exposed to dioxins; follow-up medical studies funded by the responsible companies have been published in mainstream scientific journals, claiming to show that no excess cancers have occurred in the dioxin-exposed workers. In fact, excess cancers *have* occurred, but it appears that the data have been manipulated to hide the facts.

The Monsanto Case

In 1949, an explosion occurred at a Monsanto chemical factory in Nitro, West Virginia; as a result, many workers in the plant were exposed to the herbicide 2,4,5-T, which was contaminated with dioxin. (This herbicide was later the principal component of Agent Orange, the chemical defoliant used by the U.S. in Viet Nam.) In subsequent years, two Monsanto scientists, J.A. Zack and R. W. Gaffey, studied the exposed workers, comparing their health against the health of a similar group of workers who were not exposed to dioxin or 2,4,5-T.⁴

According to court documents attached to the EPA memo, "Zack and Gaffey deliberately and knowingly omitted 5 deaths from the exposed group and took four workers who had been exposed and put these workers in the unexposed group, serving, of course, to decrease the death rate in the exposed group and increase the death rate in the unexposed group."

Other studies of this same accident were also fraudulent, according to the same court documents, including a study by R.R. Suskind published in the *Journal of the American Medical Association*.⁵ "This published study of the workers exposed in the 1949 accident reported only 14 cancers in the exposed group and 6 in the unexposed group (a smaller cohort). However, the medical records produced [by Monsanto] to the Plaintiffs

conclusively prove gross miscalculations and omissions... there were 28 cancers in the group that had been exposed to dioxins in 1949 as opposed to only 2 cancers in the unexposed group." Mr. Suskind published two other reports^{6,7} on the same accident, using his same data, to reach the conclusion that dioxin does not cause cancer.

The BASF Case

On the night of November 17, 1953, a runaway chemical reaction spewed dioxin-contaminated chemicals over workers and community residents of two small German cities, Mannheim and Ludwigshafen. Subsequently, an epidemiological study was used to deny workers any compensation for ailments they claimed they suffered as a result of exposure. In keeping with German law, the workers retained their own expert to review the data. Their expert, Friedemann Rohleder, received the data from the German government but found, to his surprise, that all the data actually came from the BASF company itself. He analyzed the data and found that some workers suffering from chloracne, which is universally acknowledged to be evidence of high exposure to dioxin, had been placed in the low-exposure or non-exposed group. He found evidence of "diluting" the exposed group with 20 plant supervisory staff who, Rohleder believes, were not exposed. When Rohleder omitted the 20 supervisory staff, his analysis revealed statistically significant increases in two groups of cancers: cancers of the respiratory organs (lungs, trachea, etc.), and cancers of the digestive tract. "This analysis adds further evidence to an association between dioxin exposure and human malignancy," Rohleder told *New Scientist*.

[To be continued.]

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[1] Cate Jenkins, "Memo to Raymond Loehr: Newly Revealed Fraud by Monsanto in an Epidemiological Study Used by EPA to Assess Human health Effects from Dioxins," dated February 23, 1990. Jenkins is a chemist with the Waste Characterization Branch (OS 332), Characterization and Assessment Division, U.S. EPA, 401 M St., SW, Washington, DC 20460. Loehr is Chairperson of the Executive Committee of the Science Advisory Board (A-101), Office of the Administrator, U.S. EPA, 401 M St., SW, Washington, DC 20460. The Jenkins memo has attached to it 25 pages of a brief filed in Case No. 5-88-0420, in the Appellate Court of Illinois, Fifth District by attorneys suing Monsanto on behalf of plaintiffs who say they were harmed when a Norfolk and Western railroad tank car derailed, spilling 19,000 gallons of a Monsanto chemical called "OCP-crude" into the community of Sturgeon, Missouri the night of January 10, 1979. Chief attorney for the plaintiffs is Rex Carr, 412 Missouri Avenue, East St. Louis, IL 62201; phone (618) 274-0434. Our thanks to Margo Blackwell, People Against the Incinerator (PATI), Bloomington, Indiana, and to EPA official William Sanjour, both of whom independently sent us the information about Monsanto. We can mail copies of the Jenkins memo and attachments (28 pages) for \$14.00, which covers our actual costs of photocopying, handling and mailing.

[2] Oak Ridge National Laboratory. *Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin* [ATSDR/TP-88/23; PB89-214522] (Springfield, VA: National Technical Information Service, June, 1989), pg. 22, identifies dumps and incinerators as "the important sources of 2,3,7,8-TCDD exposure to the general population." This is the Agency for Toxic Substances and Disease Registry [ATSDR] toxicological profile for dioxin (See *RHWN* #169.)

[3] Stephanie Wanchinski, "New Analysis links dioxin to cancer," *New Scientist* October 28, 1989, pg. 24. Thanks to Paul Connett of Work on Waste USA, we have a copy of Friedemann Rohleder's paper, which he presented in late September at a dioxin conference in Toronto, Ontario; Rohleder's paper is entitled "Dioxins and Cancer Mortality--Reanalysis of the BASF Cohort." We can mail you the 14-page Rohleder paper for \$7.00, or you can request a copy directly from the author: Friedemann Rohleder, Friedrich - Hebel Str. 13, 1712 Werne, West Germany.

[4] Zack, J.A., and W. R. Gaffey, "A Mortality Study Of Workers Employed At The Monsanto Company Plant In Nitro, West Virginia," *Environmental Science Research*, Vol. 26 (1983), pgs. 575-591.

[5] R.R. Suskind, and V.S. Hertzberg, "Human Health Effects Of 2,4,5-T And Its Toxic Contaminants," *Journal of the American Medical Association*, Vol. 251, No. 18 (1984), pgs. 2372-2380.

[6] R.R. Suskind, "Chloracne, The Hallmark Of Dioxin Intoxication," *Scandinavian Journal of Work, Environment and Health*, Vol. 11, No. 3 (1985), pgs. 165-171.

[7] R.R. Suskind, "Long-Term Health Effects Of Exposure To 2,4,5-T And/Or Its Contaminants," *Chemosphere*, Vol. 12, No. 4-5 (1983), pg. 769.

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Dioxin--Part 2 GAUGING THE TOXICITY OF DIOXIN

[Continuing our series on dioxin. Page numbers in parentheses refer to the ATSDR (the federal Agency for Toxic Substances and Disease Registry) *Toxicological Profile* for dioxin, cited in our last paragraph, below.]

It has become fashionable to pooh-pooh dioxin. We believe there are two reasons why this is occurring. First, some scientists have been publishing studies indicating that humans exposed to dioxin do not have an increased risk of cancer. As we saw in *RHWN* #171, some of the most important of these studies have now been exposed as fraudulent. The second reason is that dioxin is so toxic that it is difficult to express its potency in normal terms; therefore the media frequently print scary claims without offering much evidence, leading some people to conclude (incorrectly) that there isn't much substance to any claims about the extreme toxicity of dioxin.

In this series, we hope to lay the groundwork for an understanding of dioxin, to help people put dioxin into perspective. Some of what follows may seem a bit more technical than you are accustomed to reading in this newsletter; but stick with it, and you'll see why we have taken this approach.

The scientific and medical evidence presented by ATSDR forces us to conclude that dioxin deserves our greatest respect. It seems to be one of the two or three most toxic chemicals ever discovered, and it is produced as a byproduct of several different industrial processes. For years, industry has been dumping dioxin into the environment in large quantities without paying attention to the consequences. This does not mean there have been no consequences; it just means no one has made any systematic effort to tally them up.

Dioxin is a family of chemicals (75 in all) that does not occur naturally, nor is it intentionally manufactured by any industry (pg. 1). The most toxic dioxin is called 2,3,7,8-TCDD. Dioxins are produced as byproducts of the manufacture of some herbicides (for example, 2,4,5-T), wood preservatives made from trichlorophenols, and some germicides (for example, hexachlorophene). Dioxins are also produced by the manufacture of pulp and paper, by the combustion of wood in the presence of chlorine, by fires involving chlorinated benzenes and biphenyls (e.g., PCBs), by the exhaust of automobiles burning leaded fuel, and by municipal solid waste incinerators.

ATSDR says, "2,3,7,8-TCDD is highly toxic to all laboratory animals tested...." (pg. 11). Even the most conservative of toxicologists says, "TCDD has been called the most toxic synthetic chemical known to man. If its acute toxicity to the guinea pig, and even the rat and mouse, is the criterion, the statement is probably correct.... TCDD is unquestionably a chemical of supreme toxicity to experimental animals. Moreover, severe chronic effects from low dosages have also been demonstrated in experimental animals. Therefore, the concern about its effects on human health and the environment is understandable."¹

In cases of high exposure of humans through industrial accidents, 2,3,7,8-TCDD causes a severe acne (called chloracne) which is not just a skin ailment; chloracne is a systemic disease that is more disfiguring than teenage acne and its effects last for years (in some cases, decades) after exposure (pgs. 3, 39).

There is "suggestive evidence" that 2,3,7,8-TCDD causes liver damage in humans (pgs. 3, 52-53). It definitely causes severe liver damage in animals.

In animals, 2,3,7,8-TCDD is toxic to the immune system; such effects have not been proven in humans (pgs. 3, 40, 54-56). In animals, 2,3,7,8-TCDD causes reproductive disorders, including spontaneous abortions. Monkeys are particularly sensitive to reproductive effects from exposure to 2,3,7,8-TCDD. Such effects have not been proven in humans (pgs. 3, 17, 58-59). In animals, dioxin causes genetic damage (pgs. 60-61).

Both the U.S. Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) have concluded that dioxin is a "probable human carcinogen" (pgs. 7, 61-68, 94). As we saw in *RHWN* #171, scientists within EPA have asked that this question be reviewed again because some of the key studies of dioxin and cancer were fraudulent, and EPA has relied on these fraudulent studies to set current standards.

How can we judge the toxicity of dioxin (or of any chemical, for that matter)? One way is to look at the standards that have been set by regulatory agencies.

In the case of dioxin, EPA has calculated a "safe" dose, taking into consideration dioxin's ability to cause cancer. The "safe" dose is expressed in extremely small units: femtograms. There are 28 grams in an ounce, and one femtogram is 0.000,000,000,000,001 grams, or one quadrillionth of a gram, or 10^{-15} grams.

EPA believes that ingesting (eating) 6.4 femtograms (6.4×10^{-15} grams) of 2,3,7,8-TCDD per kilogram of body weight per day would cause cancer in one in a million people so exposed (pg. 95). Since an average adult weighs 62 kilograms or 137 pounds (average men weigh 70 kilograms [154 pounds] and average women weigh 55 kg [120 pounds]), the EPA is saying that 397 femtograms of 2,3,7,8-TCDD consumed in food each day would kill one-in-a-million humans so exposed. Over a year's time, 397 femtograms per day add up to 145,000 femtograms; over a 70-year lifetime, this would add up to 10.1 million femtograms, so 10.1 million femtograms (or 0.01 micrograms) is the maximum amount you could safely get into your body during your entire lifetime, EPA believes.

How can we express this in terms that people can grasp?

Let's compare it to one single aspirin tablet. One aspirin tablet weighs 5 grains (or 325 milligrams, or 325 trillion femtograms), so to express one "safe" lifetime dose of 2,3,7,8-TCDD, you would take a single aspirin tablet and divide it into 32 million (actually 32,172,218) minuscule pieces. Then one of those tiny pieces would represent one "safe" lifetime dose of 2,3,7,8-TCDD.

Another comparison: A single grain of table salt weighs approximately 0.1 milligrams or 100 billion femtograms, so to get an amount of table salt that weighs the same amount as one "safe" lifetime dose of 2,3,7,8-TCDD, you would divide a single grain of table salt into 9,900 microscopic pieces. One of those tiny pieces would represent a "safe" lifetime dose of dioxin.

The U.S. Food and Drug Administration (FDA) has its own way of calculating the same one-in-a-million cancer risk and they believe the EPA has overestimated the hazard by a factor of 10. In other words, FDA believes you could represent a "safe" dose of 2,3,7,8-TCDD by dividing a single grain of table salt into 990 pieces, with one of those pieces representing a safe lifetime dose. The federal Centers for Disease Control (CDC) in Atlanta has done its own calculation, concluding that the cancer hazard from dioxin is about half-way between the EPA's estimate and the FDA's estimate. EPA says 6.4 femtograms per kilogram of bodyweight per day is the safe dose; CDC says the correct number is 27.6; FDA says it's 57.2 (pg. 95). No matter which agency does the calculation, there's no escaping the fact that dioxin is considered supremely toxic.

One other way to understand the toxicity of dioxin is to compare the dioxin "reference dose" established by EPA to the "reference dose" they have set for other common toxic materials. The "reference dose" is the highest amount they believe you could eat regularly without incurring any disease (not considering cancer).

The reference dose for dioxin is 0.000,000,001 milligrams per kilogram of body weight per day (mg/kg/day) (pg. 94); the reference dose for the toxic metal cadmium² is 0.001 mg/kg/day and the "reference dose" for the toxic metal arsenic³ is the same as for cadmium. Thus we can see that EPA considers dioxin in food 1,000,000 times (one million times) more toxic than cadmium or arsenic, not counting the cancer hazard from dioxin. Yes, dioxin is toxic, no doubt about it.

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[1] Fred H. Tschirley, "Dioxin," *Scientific American* Vol. 254 (February, 1986), pg. 34.

[2] Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Cadmium* (Springfield, VA: National Technical Information Service [NTIS], 5285 Port Royal Rd., Springfield, VA 22161; phone (703) 487-4650), pg. 76.; NTIS number PB89-194476. \$21.95.

[3] Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Arsenic* (Springfield, VA: National Technical Information Service [NTIS], 5285 Port Royal Rd., Springfield, VA 22161; phone (703) 487-4650), pg. 92.; NTIS number PB89-185706. \$21.95.

Get: Agency for Toxic Substances and Disease Registry, *Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin* (Springfield, VA: National Technical Information Service [NTIS], 5285 Port Royal Rd., Springfield, VA 22161; phone (703) 487-4650); NTIS number PB89-214522. \$21.95.

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Dioxin--Part 3

NEW EVIDENCE THAT DIOXIN CAUSES HUMAN CANCERS AND OTHER DISEASES

Two recent studies, published in 1988 and 1989, have indicated a connection between exposure to dioxin and development of cancers in humans. Earlier studies had shown little evidence linking cancers to dioxins, but these earlier studies have now been challenged by U.S. government officials as fraudulent. (See *RHWN* #171.) One of the recent studies also reveals a connection between dioxin exposure and a variety of non-cancer health effects, including heart attacks (among men), and digestive diseases, cirrhosis of the liver, gall bladder and biliary tract diseases, and genitourinary diseases (among women).

Seveso, Italy

On July 10, 1976, an industrial disaster occurred at a chemical plant operated by the pharmaceutical giant, Hoffman-La Roche, in the town of Meda, Italy. A visible chemical cloud containing dioxin (2,3,7,8-TCDD and other dioxins) spread over several square miles of populated countryside; most heavily hit was the community of Seveso. More than 200,000 people aged 20 to 74 lived in towns near the accident. Nearly 31,000 people aged 20 to 74 lived in zones contaminated to some degree by the accident and about 200 individuals had such high exposures that they developed chloracne, an acne condition known to be caused by exposure to dioxins.

A team of Italian physicians and researchers has been studying health conditions, including death certificates, of 30,703 people aged 20 to 74 living in areas termed "exposed," comparing people's experience there against a larger population of 167,391 individuals aged 20 to 74 living nearby in non-exposed areas. A recent report in the *American Journal of Epidemiology*¹ reveals several elevated disease rates among the exposed group.

The report covers the decade 1976 to 1986, which is a short period in which to find cancer occurrences. All cancers exhibit a "latency period" (or delay period) between the time a cancer-causing exposure occurs and a cancer actually develops; the latency period varies from 7 to 40 or 50 years. Thus a study of cancers occurring 10 years after an exposure to cancer-causing chemicals could only reveal the earliest evidence of cancers and should be understood to be preliminary in nature.

The results of the study are reported for people living in three areas, labeled zones A, B and R. Zone A is closest to the accident site and zone R extends several miles distant; zone B is between the two. The assumption is that people's exposure varied with distance from the accident.

In zone A, women had elevated cancers of the gall bladder and biliary tract. They also had elevated occurrences of circulatory diseases and of chronic rheumatic heart disease. Men in zone A had elevated occurrence of cerebrovascular disease (such as stroke). In zone B, men had elevated melanomas (serious skin cancers) and cancers of the lining of the chest cavity (pleura); women in zone B had elevated incidence of soft tissue sarcomas. In zone R, men had elevated incidence of cancers of the lining of the chest cavity (pleura), and they had increased incidence of all blood diseases, and of cerebrovascular disease; women in zone R had increased incidence of cancer of the uterus, as well as hypertensive vascular disease.

The results reported above are averages for the entire decade. In the case of cancers, which would only begin to occur after a latency period, the time-period of interest is the second five years of the decade, and, accordingly, the Italian researchers reported results for each half of the decade.

During the second half of the decade: In zone A there were no elevated cancer levels. In zone B, men showed elevated incidence of cancers of the lung, cancers of the lining of the chest cavity (pleura), serious skin cancers (melanoma), Hodgkin's disease (cancer of the lymph nodes), and leukemia. In zone B, women showed increased incidence of soft tissue sarcomas and of the thyroid gland. In zone R, men showed elevated incidence of leukemia, and women

showed elevated incidence of cancer of the brain. It is perhaps relevant to note that two previous studies have implicated brain cancer with exposure to dioxins in weed killers.²³

This study of the people exposed to dioxin during the Seveso accident does not prove that dioxin exposure caused the cancers or the other serious ailments from which these people suffer in abnormally high numbers (mainly diseases of the heart, blood, and arteries). Nevertheless, this study confirms that it is definitely misleading and untrue when anyone says there is "no evidence" of cancer or other serious diseases among humans exposed to dioxins. (In addition, an earlier U.S. government study has shown that Vietnam veterans exposed to Agent Orange [a weed killer contaminated with dioxin] suffer from elevated incidence of cancers, liver damage, cardiovascular deterioration, and degeneration of the endocrine system. See *RHWN* #73.)

Sweden

Phenoxyacetic acids (for example, the weed killers known as 2,4,5-T and 2,4-D) are almost always contaminated with dioxins during manufacture. During the late 1970s, Swedish researchers studied workers who had been exposed to phenoxyacetic acids and found increased incidence of soft tissue sarcomas (rare cancers of the connective tissues).⁴⁵ During the mid-'80s, several additional studies confirmed the relationship between soft tissue sarcomas and exposure to phenoxy herbicides, while other studies failed to confirm such a relationship. Now a new study has once again shown a three-fold increase in soft tissue sarcomas among workers exposed to phenoxy herbicides.⁶ This is the tenth study that we know of showing a positive relationship between exposure to phenoxy herbicides and soft tissue sarcomas.⁷ Five studies have failed to confirm such a relationship.⁸

How can one make sense out of conflicting reports, when 10 studies show that certain chemicals cause cancer and 5 studies show that those same chemicals do not cause cancer? How can the public know what policies make sense to pursue?

In our experience, people who make money manufacturing, or using, such chemicals prefer to argue that "we just don't know," and "until all the facts are in, we should not make any changes." Unfortunately, all the facts will never be in. People, including consumers buying phenoxy herbicides at the lawn-care store and politicians making laws, will always have to make decisions based on incomplete information.

From our viewpoint, the key question is this: Is it more important to protect people and the environment from damage, or to protect chemicals from regulation, control and outright bans?

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[1] Pier Alberto Bertazzi and others, "Ten-year Mortality Study of the Population Involved in the Seveso Incident in 1976," *American Journal of Epidemiology*, Vol. 129 (1989), pgs. 1187-1200.

[2] A. Bair and others, "Lung Cancer and Other Causes of Death Among Licensed Pesticide Applicators," *Journal of the National Cancer Institute* Vol. 71 (1983), pgs. 31-37.

[3] D. Coggon and others, "Mortality of Workers Exposed to 2-methyl-4-chlorophenoxy-acetic acid," *Scandinavian Journal of Work, Environment and Health* Vol. 12 (1986), pgs. 448-454.

[4] Olav Axelson and others, "Herbicide Exposure and Cancer Mortality," *Scandinavian Journal of Work, Environment and Health* Vol. 6 (1980), pgs. 73-79.

[5] Lennart Hardell and others, "Malignant Lymphoma and Exposure to Chemicals, Especially Organic Solvents, Chlorophenols, and Phenoxy Acids: A Case-Control Study," *British Journal of Cancer* Vol. 43 (1981), pgs. 169-176.

[6] Lennart Hardell and others, "The Association Between Soft Tissue Sarcomas and Exposure to Phenoxyacetic Acids--A New Case-referent Study," *Cancer* Vol. 62 (1988), pgs. 652-656.

[7] Nine of the studies are listed in footnotes 1 through 9 of the Hardell study we cited in our footnote 6 (above), and the tenth study is the Hardell study itself cited in our footnote 6 (above).

[8] The five studies are listed in footnotes 10 through 14 of the Hardell study we cited in our footnote 6 (above).

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Providing news and resources to the Movement for Environmental Justice -- December 19, 1990

REPORT LINKS HERBICIDE EXPOSURE TO ILLNESSES AMONG VIETNAM VETS

For more than a decade Vietnam veterans have sought compensation for illnesses they believe were caused by their wartime exposure to herbicides, which were used heavily during the war to defoliate the jungle, to reduce available cover for enemy troops. U.S. soldiers and airmen who prepared, handled or sprayed the herbicides, and ground troops who were doused, have been routinely denied compensation by the federal Department of Veterans Affairs (VA) because the VA has taken the position that there is not enough scientific evidence linking herbicide exposure to disease.

Now an independent scientific review sponsored by the American Legion, the Vietnam Veterans of America, and the National Veterans Legal Services Project has concluded that there is a "significant statistical association" between exposure to chemical herbicides and several serious illnesses. According to the April, 1990, report of the Agent Orange Scientific Task Force, there is "a significant statistical association" between exposure to the herbicide Agent Orange and various cancers (non-Hodgkin's lymphoma and soft tissue sarcomas), serious skin disorders (chloracne), and liver disorders. The Task Force said, "The aggregate interpretation of several sound studies showing a statistically significant association for each of these conditions makes this conclusion inescapable." Agent Orange was the military code name for the chemical herbicide used most often in Vietnam to defoliate the jungle. Agent Orange was a chlorinated phenoxy herbicide made up of two common weed killers, 2,4,5-T and 2,4-D, both of which are routinely contaminated with dioxins during manufacture. Anyone exposed to Agent Orange is presumed to have been exposed to dioxins.

When a scientist says there is a "significant statistical association" between one event (such as exposure to an herbicide) and another event (such as the onset of illness), he or she means it is very unlikely that the two events occurred together by random chance; it is much more likely that the two events occurred together for a reason. (What is meant by "very unlikely" differs from study to study; often "very unlikely" means there is less than a 5% probability that the observed relationship occurred by random chance; sometimes "very unlikely" means there was less than a 1% probability that the observed relationship occurred by random chance. In each individual study, the author decides which definition of "very unlikely" he or she will use.)

The 1990 report is based on a review of 285 different published studies of human exposure to phenoxy herbicides and/or dioxins, all appearing in scientific journals from 1978 onward. The 285 studies are listed on pages 49-75 of the 1990 report; the list provides a unique resource for anyone seeking additional information on phenoxy herbicide effects on humans. The VA does not allow consideration of animal studies, so only human data were evaluated for the 1990 report. However, to make the point that animal studies are universally recognized as valid evidence for human cancer risk, the 1990 report contains an appendix in which various scientists and government agencies are quoted saying that animal studies provide valid evidence for those trying to understand human cancers in relation to chemical exposures. This appendix (pgs. 42-48 of the 1990 report) is a unique resource for citizens trying to make the case that animal studies should be heeded in public policy decisions involving human exposure to chemicals.

The 1990 report concludes further that three additional health effects "are at least as likely as not" to be associated with exposure to phenoxy herbicides: Hodgkin's disease (a cancerous enlargement of the lymph nodes, spleen, and general lymphoid tissues, which usually appears first in the neck), neurologic effects, and reproductive and developmental disorders.

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The observed reproductive and developmental disorders include (a) low sperm count among Vietnam vets compared to a control group of non-Vietnam veterans; (b) increased incidence of spontaneous abortion among wives of Vietnam vets; (c) increased incidence of birth defects in children of Vietnam vets, including skin defects, nerve defects, heart defects, kidney defects, and oral clefts (cleft lip and cleft palate).

The conclusion that these illnesses are "at least as likely as not" to occur from Agent Orange exposure is important within the VA because the VA's rules for compensation require a finding that a disease is "as likely as not" to occur from a chemical exposure before that disease becomes compensable.

The report concludes further that there is "sound scientific evidence of an association with exposure to agent orange, but the evidence does not reach the level of formal statistical significance for the following additional effects:" cancers of the kidney, testicles, stomach, prostate, colon, hepatobiliary tract (liver and related systems), brain, and blood-forming cells (leukemia); psychosocial effects; immune system disorders; gastrointestinal ulcers; and altered lipid metabolism (the body's ability to digest and handle fats and some oils).

Authors of the report (the Agent Orange Scientific Task Force) are seven prominent independent scientists and physicians: Richard W. Clapp, Barry Commoner, John D. Constable, Samuel S. Epstein, Peter C. Kahn, James R. Olson, and David M. Ozonoff.

This is an important report because the issue of compensation for Vietnam vets is merely the tip of an enormous iceberg. The components of agent orange (2,4,5-T and 2,4-D) are both still widely used in the United States for clearing rights of way beneath power lines and along highways; rain then carries the chemicals into water supplies. Many homeowners use these chemicals (knowingly or not) to kill lawn "pests" (broad-leaf plants such as crab grass and dandelions). Farmers use them extensively for weed control. Thus exposure to these chemicals is very widespread among the American people.

Since the 1990 report appeared (in April), the VA has reversed itself and declared that soft tissue sarcomas and non-Hodgkin's lymphomas in Vietnam vets are compensable. The VA's Advisory Committee on Environmental Hazards continues to study the relationship of other diseases to Agent Orange exposures among vets.

Get: *Human Health Effects Associated With Exposure to Herbicides And/Or Their Associated Contaminants - Chlorinated Dioxins* (Washington, DC: National Veterans Legal Services Project [2001 S Street, NW, Washington, DC 20009-1125; phone 202/265-8305], April, 1990); 41 pages, plus 33 pages of useful appendices; \$10.00 plus \$3.00 shipping.

[Dated announcement omitted from this reprint.]

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Dioxin--Part 4
NEW STUDY LINKS DIOXIN TO HUMAN CANCER

A new study published last month provides fresh evidence that dioxin [TCDD] causes cancer in humans. Dioxin is not a commercial product but is created as an unwanted byproduct of many industrial processes; significant quantities of dioxin are released from the smoke stacks (and the ash landfills) of incinerators that burn chlorine-containing items--such as medical wastes (*RHWN* #179), sewage sludge, and municipal solid wastes. Once it is released into the environment dioxin persists for a very long time, enters food chains, and accumulates; when humans eat dioxin-contaminated food, such as milk or fish, the humans themselves accumulate dioxin in their blood and fatty tissues.¹

Scientists have known since the mid-1960s that dioxin is an extremely powerful promoter of cancer in laboratory animals, but industry researchers have recently been claiming that humans somehow are exempt from the dioxin danger. The question of dioxin's hazard to humans took on real urgency in the early '80s when 15,000 veterans sued Dow chemical and other producers of Agent Orange (a dioxin-contaminated herbicide widely used to defoliate the jungle in Vietnam from 1962 to 1971); the vets sought money damages for health effects (cancer, defective offspring, and so forth) they said they were experiencing. Lawyers for the Vietnam vets offered documentary evidence that Dow chemists convened a private meeting of their competitors in 1965 to share new information that impurities [dioxins] in the herbicide 2,4,5-T (principal component of Agent Orange) caused severe liver damage in rabbits. According to court records, a chemist at Hercules Powder Company who attended the private Dow meeting in 1965, received a phone call from a Dow executive who "warned him to keep the findings away from the federal government," according to a reporter for *Nature*, the British science journal.² If this is true, it would not be the first time, nor the last, that money has influenced the outcomes, and the uses, of scientific studies.

In any case, as a result of these lawsuits, during the 1980s the question of dioxin's effects on humans became subject of bitter controversy--with enormous sums of money riding on the outcome of the debate. As the 1980s drew to a close and it became known that all incinerators create and release dioxin into the local environment, industry felt enormous pressure to "prove" that dioxin was harmless to humans. From 1980 onward, industry researchers published several studies of dioxin-exposed workers, claiming to show that they suffered no more cancer than the general public. Last year, however, evidence began to accumulate indicating that the industry-funded studies of dioxin dangers to humans were badly flawed or were simply fraudulent (see *RHWN* #171, #173, #175).

The latest study is not by industry researchers but by Dr. Marilyn Fingerhut of the federal National Institute for Occupational Safety and Health (NIOSH); Fingerhut looked at the health of 5172 workers at 12 chemical plants that manufacture (or formerly manufactured) products contaminated with dioxin such as the herbicides 2,4,5-T, Silvex, Ronnel, Erbon, and pentachlorophenol (which has also been used as a fungicide, algicide, and wood preservative for telephone poles and pilings), and the bacterial cleansing agent, hexachlorophene--until the 1970s, a leading bactericide in hospitals.

Of the 5172 exposed workers (all of whom were male), 1520 met two key conditions: they had been exposed for at least a year, and their exposure had begun at least 20 years previously. The onset of cancer is always delayed by 7 to 40 years (or more) between the time of initial exposure and the time disease appears; therefore, the "latency" period of at least 20 years is important in studying cancer that may be related to a particular chemical exposure. This group ("cohort," to use the language of medical researchers) had nine times (900%) the normal amount of soft tissue sarcoma--malignant cancer of the soft connective tissues. The same group also had 42% more cancers of the respiratory tract (trachea,

bronchus and lung) than would be expected among males in the general public; by various means, Dr. Fingerhut examined and tried to eliminate the possibility that tobacco smoking explained the increase in respiratory cancers.

Among the entire cohort of 5172 men, the occurrence of all cancers was significantly increased, by 15%; in the high-exposure group of 1520 men, the "all cancers combined" increase was even more pronounced--46%; furthermore "all cancers combined" were increased among workers at nine of the 12 plants studied. Even when cancers of the respiratory tract were omitted in an attempt to eliminate smoking as a possible cause, "all cancers combined" was increased among the 5172 and even more so among the high-exposure 1520.

Dr. Fingerhut says correctly that her results do not prove that dioxin causes cancer in exposed workers. The workers she studied were exposed to many other chemicals, in addition to dioxin, on the job, and these other chemicals could explain the cancer increases she observed.

Nevertheless, the Fingerhut study makes it ever more difficult for the purveyors of dioxin-creating machines (such as incinerators for solid waste, hazardous waste, or sewage sludge) to claim that their dioxin emissions are negligible or harmless. Because dioxin accumulates in the food chain, even small amounts can build up to significant levels as time passes.

An editorial in the *New England Journal of Medicine* tried to shed light on the meaning of the Fingerhut study the day it appeared.³ The well-known Canadian biostatistician, John Bailar, wrote, "This evidence is short of proof, as the authors explain, but it must be taken seriously as a flag of a probable human risk. If one accepts the best estimate of excess risk given here (3 deaths observed among the 1520 workers minus 0.3 expected deaths equals 2.7), the lifetime risk of death from TCDD-related soft-tissue sarcoma is already approaching 2 per 1000 workers, and it may increase with additional follow-up study. This estimate falls in a range that is widely considered unacceptable for occupational hazards, and it is far in excess of the usual limits for lifetime risk to the public of 1 per 100,000 or 1 per million.

"Despite the problems, which Fingerhut et al. carefully note, this work is a model of its kind. Occupational cohort studies are inherently difficult and uncertain, and we are likely to wait a long time for appreciably better or broader evidence of the effects of TCDD [dioxin] on human health....

"The hypothesis that low exposures [to dioxin] are entirely safe is distinctly less tenable now than before," Dr. Bailar said.

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[1] Bengt-Göran Svensson and others, "Exposure to Dioxins and Dibenzofurans Through the Consumption of Fish." *New England Journal of Medicine* Vol. 324 (Jan. 3, 1991), pgs. 8-12.

[2] Peter David, "Dioxin--When was the Danger Known?" *Nature* Vol. 303 (May 12, 1983), pg. 104.

[3] John C. Bailar III, "How Dangerous is Dioxin?" *New England Journal of Medicine* Vol. 324 (Jan. 24, 1991), pgs. 260-262.

And get: Marilyn A. Fingerhut and others, "Cancer Mortality in Workers Exposed to 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin," *New England Journal of Medicine* Vol. 324 (Jan. 24, 1991), pgs. 212-218. Reprints free from Dr. Fingerhut at: Industrywide Studies Branch, Division of Surveillance, National Institute for Occupational Safety and Health, Centers for Disease Control, 4676 Columbia Parkway, Cincinnati, OH 45226.

A more complete report of this research is available under the title *Mortality Among U.S. Workers Employed in the Production of Chemicals Contaminated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD)*. Springfield, VA: National Technical Information Service (NTIS), Dec., 1990. Available from NTIS, 5285 Port Royal Rd., Springfield, VA 22161; phone (703) 487-4650; NTIS number PB91-125971. \$15.00 + shipping.

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Reprint of:

RACHEL'S HAZARDOUS WASTE NEWS #263

Providing news and resources to the Movement for Environmental Justice -- December 11, 1991

The Wingspread Statement--Part 1

CHEMICALS IN ENVIRONMENT AFFECT SEXUAL DEVELOPMENT IN WILDLIFE. AND HUMANS?

Gradually during recent years a new body of knowledge has developed showing that some chemicals in food and water can mimic hormones and disrupt the development of living things like fish, birds, and mammals, including their sexual development.

In some cases, the effects on wildlife have been dramatic. For example, male herring gulls on Lake Ontario, exposed to DDT and other organochlorine compounds, developed female sex organs. Female-female pairing has been observed in herring gulls on Lake Michigan and on Santa Barbara Island, California. Because humans share the same basic mechanisms of growth and development as wildlife, an increasing number of scientists has become concerned that humans may already be affected without recognizing it.

In July of this year an international group of 21 scientists met at Wingspread in Racine, Wisconsin to assess what is known about these matters. They have now released a "consensus statement" containing information and opinions about the nature and possible causes of these problems. The five-page statement is called "Chemically Induced Alterations in Sexual Development: The Wildlife/Human Connection."

Background

Hormones are produced by the endocrine system--a bodily system consisting of specialized cells, tissues, and organs that create and secrete (usually into the blood) organic chemicals called hormones, which then regulate other kinds of cells in the body. Particular hormones only affect particular cells that contain "receptors" for those hormones. A small amount of a hormone attaches to a "receptor site" and the hormone-receptor pair then initiates a cascade of chemical changes, often with major and far-reaching consequences.

The endocrine system shares with the nervous system the job of adjusting the body's response to a changing external environment. The nervous system copes with environmental changes on an immediate basis, whereas the endocrine system copes with environmental changes on a sustaining basis. For example, when the body gets cold, the nervous system causes shivering, which raises the body's temperature. But each month it is the endocrine system that starts the human female menstrual cycle. In a developing fetus, it is the endocrine system that regulates cell division and organ differentiation. The endocrine system regulates pattern and timing of bird migration and of hibernation in mammals. Examples of endocrine glands in humans include the adrenal gland, pancreas, thyroid, pituitary, ovaries and testes.

The scientists gathered at Wingspread last July focused on the sex hormones--the androgens that make males look and act like males and the estrogens that make females look and act like females. The Wingspread Statement begins, "Many compounds introduced into the environment by human activity are capable of disrupting the endocrine system of animals, including fish, wildlife, and humans. The consequences of such disruption can be profound because of the crucial role hormones play in controlling development...."

"The following consensus was reached by participants at the workshop.

"1. We are certain of the following:

"A large number of man-made chemicals that have been released into the environment, as well as a few natural ones, have the potential to disrupt the endocrine system of animals, including humans. Among these are the persistent, bioaccumulative, organohalogen compounds that include some pesticides (fungicides, herbicides, and insecticides) and industrial chemicals, other synthetic products, and some metals.¹

"Many wildlife populations are already affected by these compounds. The impacts include thyroid dysfunction [impaired or abnormal functioning] in birds and fish; decreased fertility in birds, fish, shellfish, and mammals; decreased hatching success in birds, fish and turtles; gross birth deformities in birds, fish and turtles; metabolic abnormalities [impaired or abnormal use of energy, manufacture of tissue, or handling of resulting wastes] in birds, fish, and mammals; behavioral abnormalities in birds; demasculinization and feminization in male fish, birds, and mammals; defeminization and masculinization of female fish and birds; and compromised [impaired] immune systems in birds and mammals.

"The patterns of effects vary among species and among compounds. Four general points can nonetheless be made: (1) the chemicals of concern may have entirely different effects on the embryo, fetus, or perinatal [meaning "near the time of birth," from the 28th week of pregnancy through the first week of life, in humans] organism than on the adult; (2) the effects are most often manifested in offspring, not

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in the exposed parent; (3) the timing of exposure in the developing organism is crucial in determining its character and future potential; and (4) although critical exposure occurs during embryonic development [from conception through the end of the second month of pregnancy], obvious manifestations [effects] may not occur until maturity.

"Laboratory studies corroborate the abnormal sexual development observed in the field and provide biological mechanisms to explain the observations in wildlife.

"Humans have been affected by compounds of this nature, too. The effects of DES (diethylstilbesterol), a synthetic therapeutic agent, like many of the compounds mentioned [in footnote 1] are estrogenic [meaning they act like estrogen, a family of female sex hormones]. Daughters born to mothers who took DES now suffer increased rates of clear cell adenocarcinoma [cancer], various genital tract abnormalities, abnormal pregnancies, and some changes in immune responses. Both sons and daughters exposed in utero [while in the uterus] experience congenital anomalies of their reproductive system and reduced fertility. The effects seen in in utero DES-exposed humans parallel those found in contaminated wildlife and laboratory animals, suggesting that humans may be at risk to those same environmental hazards as wildlife."

The Wingspread Statement goes on:

"2. We estimate with confidence that:

"Some of the developmental impairments reported in humans today are seen in adult offspring of parents exposed to synthetic hormone disruptors (agonists and antagonists) released in the environment. The concentrations of a number of synthetic hormone agonists and antagonists measured in the U.S. human population today are well within the range and dosages at which effects are seen in wildlife populations. [An agonist is a chemical that is not a hormone but mimics a natural hormone; an antagonist interferes with a natural hormone.] In fact, experimental results [in animals] are being seen at the low end of current environmental concentrations [in humans].

"Unless the environmental load of synthetic hormone disruptors is abated and controlled, large scale dysfunction at the population level is possible. The scope and potential hazard to wildlife and humans are great because of the probability of repeated and/or constant exposure to numerous synthetic chemicals that are known to be endocrine disruptors...."

"3. Current models predict that:

"... Both exogenous (external source) and endogenous (internal source) androgens (male hormones) and estrogens (female hormones) can alter the development of brain function.

"Any perturbation [disturbance] of the endocrine system of a developing organism may alter the development of that organism: typically these effects are irreversible. For example, many sex-related characteristics are determined hormonally during a window of time in the early stages of development, and can be influenced by small changes in hormone balance. Evidence suggests that sex-related characteristics, once imprinted, may be irreversible."

The Wingspread statement then gives three reasons why these predictions are subject to "many uncertainties:" (1) effects of exposure of humans are not well understood, especially exposure of embryos; (2) data on reproductive problems in wildlife exist but data on behavior changes are not so readily available; (3) the potency of many synthetic [human-created] estrogenic chemicals is not well known.

The British publisher, Elsevier Applied Science, will publish a book on this subject by next fall. Until then, the best source of information is Theodora E. Colborn and others, *Great Lakes Great Legacy?* available for \$20.00 (plus \$2.00 shipping) from: World Wildlife Fund, P.O. Box 4866, Hampden Post Office, Baltimore, MD 21211; phone (301) 516-6951.

For \$3.00 plus stamped, self-addressed envelope, we can send you the Wingspread statement itself.

[More on this subject coming soon.]

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[1] A footnote on page 1 of the Wingspread Statement says, "Chemicals known to disrupt the endocrine system include: DDT and its degradation products, DEHP (di(2-ethylhexyl)phthalate), dicosol, HCB (hexachlorobenzene), kelthane, kepone, lindane and other hexachlorocyclohexane congeners, methoxychlor, octachlorostyrene, synthetic pyrethroids, triazine herbicides, EBDC fungicides, certain PCB congeners, 2,3,7,8-TCDD and other dioxins, 2,3,7,8-TCDF and other furans, cadmium, lead, mercury, tributyltin and other organo-tin compounds, alkyl phenols (non-biodegradable detergents and anti-oxidants present in modified polystyrene and PVCs), styrene dimers and trimers, soy products, and laboratory animal and pet food products."

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**The Wingspread Statement--Part 2
MAJOR CHALLENGE TO "BUSINESS AS USUAL"**

An international group of 21 scientists¹ met at Wingspread in Racine, Wisconsin in July, 1991 to discuss evidence that chemicals in the environment are causing changes in the sexual development of wildlife and conceivably in humans as well. (See *RHWN* #263.) The group produced a five-page "consensus statement."

Sexual development in wildlife, as in humans, is controlled by the endocrine system, a group of organs, tissues and cells that secrete hormones; the hormones interact with other cells, initiating chemical/biological reactions with far-reaching consequences. Male hormones are called androgens; female hormones are called estrogens.

It is now known that some chemicals disrupt the endocrine system. The Wingspread statement identifies the following: "Chemicals known to disrupt the endocrine system include: DDT and its degradation products [DDE and DDD], DEHP (di(2-ethylhexyl)phthalate), dicofol, HCB (hexachlorobenzene), kelthane, kepone, lindane and other hexachlorocyclohexane congeners [forms], methoxychlor, octachlorostyrene, synthetic pyrethroids, triazine herbicides, EBDC fungicides, certain PCB congeners [forms], 2,3,7,8-TCDD and other dioxins, 2,3,7,8-TCDF and other furans, cadmium, lead, mercury, tributyltin and other organo-tin compounds, alkyl phenols (non-biodegradable detergents and anti-oxidants present in modified polystyrene and PVCs), styrene dimers and trimers, soy products, and laboratory animal and pet food products."

In animals, and in humans, sexual characteristics are established at a particular point during development in the womb or egg. For example, Dr. Theodora Colborn of the World Wildlife Fund (Washington, DC) points out that a single dose of dioxin, administered to a pregnant rat at day 15 of the pregnancy--near the time when gender is established--results in demasculinization and feminization of male offspring.²

Samples of herring gulls from Lake Ontario collected in 1975 and 1976 showed cellular and anatomical changes in embryos and in newly hatched chicks that caused feminization of male chicks and overdevelopment of female reproductive organs. Elevated concentrations of DDE [a break down by product of DDT] and other residues were found in eggs from the same population. To test whether these sexual problems might be linked to the presence of the DDT, laboratory experiments were conducted in which Western gull eggs were injected with concentrations of DDT similar to those found in the Great Lakes environment. Male chicks became feminized, developing ovarian tissue and oviducts.³

What is becoming clear is that the older picture of chemical toxicity underestimates the number and kinds of effects that chemicals can have in fish, birds, and mammals. For decades, U.S. regulatory officials have focused their attention almost exclusively on cancer. Meanwhile, many of the chemicals that have been dumped into the environment, and are now coursing through food chains, cause many other detrimental effects besides cancer.

For example pesticides such as DDE, dieldrin, lindane, mirex, toxaphene, and PCBs, block communication between cells. Normal metabolism [energy use] and development of a cell may be disrupted because movement of nutrients, electrolytes, and hormones in and out of a cell is blocked by the presence of these poisons.⁴

Furans, benzo[a]pyrene, 2,3,7,8-TCDD [dioxin], DDE, dieldrin, HCB [hexachlorobenzene], lindane [beta-HCH], mirex, toxaphene, and PCBs induce enzyme activity. [Enzymes are large protein molecules that promote chemical activity in the body; the presence of particular enzymes makes possible particular chemical reactions that would not otherwise be possible.] When the enzyme activity is induced, normal products of the endocrine hormonal system can be released into the bloodstream. This can disrupt the role of steroid hormones, affecting growth and sexual maturation.⁴

The structure of DDT and DDE are, themselves, quite similar to estrogens and thus may

mimic female hormones. In addition, DDE induces enzymes that break down male hormones. Under different circumstances, dioxin acts like an estrogen, or it may act as an anti-estrogen (what causes the same chemical to have opposite effects is not understood).⁴

At one Superfund site where data are available, humans are experiencing abnormalities of sexual development. At the Brio site south of Houston, Texas, where a housing development was built atop a chemical dump, girls 4 to 5 years old have developed pubic hair and enlarged breasts. One child (now four years old) was born without any genital organs; chromosome tests revealed that this is a male child, though he has a birth canal. This information, and other data about abnormal sexual development of children at the Brio site, is contained in a unique database of information that resulted from a health survey of the community by a local group (HELP) and by the Environmental Health Network (EHN) in Harvey, Louisiana.⁵ Such surveys--providing an unusually valuable source of information about health problems near chemical dumps--are under way in several Superfund communities, looking for patterns of problems, including the kinds the Wingspread statement warns of.

The Wingspread statement presents a major new challenge to advocates of "business as usual." The participants in the conference represent 17 different fields of scientific inquiry. They have many thousands of data points on which they have based their conclusions. If they are right, we are all being exposed, on a daily basis, to chemicals that threaten our reproductive health, and the health of our offspring.

Because hormones and hormone disrupters do their work at extremely low concentrations, the only "safe" dose of an endocrine-system disrupter is zero. Thus any new sources of these chemicals should be aggressively discouraged while we figure out how to minimize exposure to the quantities of these chemicals already in the environment.

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[1] Participants in the Wingspread meeting included the following individuals (whose institutional affiliations are given for identification purposes only): Dr. Howard A. Bern, Professor of Integrative Biology (emeritus) and Research Endocrinologist, University of California-Berkeley; Dr. Phyllis Blair, Professor of Immunology, University of California-Berkeley; Sophie Brasseur, Marine Biologist, Research Institute for Nature Management, Texel, The Netherlands; Dr. Theo Colborn, Senior Fellow, World Wildlife Fund, Washington, DC; Dr. Gerald R. Cunha, Developmental Biologist, University of California-San Francisco; Dr. William Davis, Research Ecologist, Environmental Research Laboratory, U.S. Environmental Protection Agency, Sabine Island, FL; Dr. Klaus D. Dohler, Director, Research, Development & Production, Pharma Bissendorf Peptide GmbH, Hannover, Germany; Glen Fox, Contaminants Evaluator, National Wildlife Research Center, Environment Canada, Quebec, Canada; Dr. Michael Fry, Research Faculty, Department of Avian Sciences, University of California-Davis; Dr. Earl Gray, Section Chief, Developmental and Reproductive Toxicology Division, Health Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, NC; Dr. Richard Green, Professor of Psychiatry in Residence, School of Medicine, University of California-Los Angeles; Dr. Melissa Hines, Assistant Professor in Residence, School of Medicine, University of California-Los Angeles; Timothy J. Kubiak, U.S. Fish and Wildlife Service, East Lansing, MI; Dr. John McLachlan, Director, Division of Intramural Research, National Institute of Environmental Health Sciences, Research Triangle Park, NC; Dr. J.P. Myers, Director, W. Alton Jones Foundation, Charlottesville, VA; Dr. Richard E. Peterson, Professor of Toxicology and Pharmacology, School of Pharmacy, University of Wisconsin-Madison; Dr. P.J.H. Reijnders, Head, Section of Marine Mammalogy, Research Institute for Nature Management, Texel, The Netherlands; Dr. Ana Soto, Associate Professor, Tufts University School of Medicine, Boston, MA; Dr. Glen Van Der Kraak, Assistant Professor, University of Guelph, Ontario, Canada; Dr. Frederick vom Saal, Professor, Division of Biological Sciences, University of Missouri-Columbia; Dr. Pat Whitten, Assistant Professor, Department of Anthropology, Emory University, Atlanta, GA.

[2] David J. Hanson, "Dioxin Toxicity: New Studies Prompt Debate, Regulatory Action," *C&EN [Chemical & Engineering News]* August 12, 1991, pg. 13.

[3] Theodora E. Colborn and others, *Great Lakes Great Legacy?* (Washington, DC: Conservation Foundation, 1990), pg. 139.

[4] Theodora E. Colborn and others, cited above, pg. 142.

[5] For more information about the Brio site, contact HELP: 10904 Scarsdale Boulevard, M263, Houston, TX 77089; phone (713) 992-1867. For advice about health surveys, contact Linda King, Environmental Health Network, P.O. Box 1628, Harvey, LA 70058; phone (504) 362-6574.

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C00142

YOUNG MALE RATS ARE 'DEMASCULINIZED' AND 'FEMINIZED' BY LOW DOSES OF DIOXIN

Three new studies by researchers at University of Wisconsin reveal that very low doses of dioxin alter the sexual development of young male rats, causing demasculinization and feminization.^{1,2,3}

Dr. Linda S. Birnbaum, a scientist with U.S. EPA [Environmental Protection Agency] calls the new studies "highly significant."⁴ Birnbaum is one of the chief scientists conducting the EPA's formal reassessment of the toxicity of dioxin (see RHWN #269, #270, #275). As we reported earlier (RHWN #279), many scientists, including Birnbaum, now consider dioxin an "environmental hormone." The new Wisconsin studies support that view.

The Wisconsin researchers, led by Dr. Richard E. Peterson, showed that dioxin interferes with the sexual development of male rats exposed to dioxin before, and shortly after, birth. Pregnant female rats were given a single oral dose of dioxin on the 15th day of pregnancy; their male offspring showed reduced levels of male hormones in their blood and a variety of sexual aberrations that stayed with them as they matured. The young males are demasculinized and feminized by doses of dioxin too low to cause any measurable toxicity in the mother rat. The sexual changes in the young males are both physiological and behavioral, and last into adulthood.

Dioxin passes through the placenta and enters the fetus, so the rat fetuses received part of the mother's dose almost immediately. After birth, the baby rats continued to receive a small dose of dioxin through their mother's milk. Peterson says the baby rats received the bulk of their dose through milk. In rats and humans both, females rid their bodies of dioxin chiefly by excreting it in their milk. Dioxin is soluble in fats and oils, and milk is high in fat.

Dioxin is the common name for a family of 75 toxins, the most potent of which is TCDD [2,3,7,8-tetrachlorodibenzo-*p*-dioxin]. The Wisconsin researchers used TCDD in their experiments.

Dioxin is not made intentionally for any industrial purpose, but is produced as a byproduct of the combustion of chlorine-containing wastes, the bleaching of paper, and the manufacture of some pesticides. The burning of municipal solid waste, and of many hazardous wastes, releases dioxin into the environment, as does paper manufacture. Government officials responsible for the quality of the environment in the Great Lakes have called for a phase-out of chlorine, to reduce dioxin levels in wildlife and humans around the Lakes. (See RHWN #284.)

In the Wisconsin experiments, young males whose mothers were given as little as 0.064 micrograms of dioxin per kilogram of body weight showed consistently reduced levels of male hormones, plus a variety of physical and behavioral changes, including:

- reduced testosterone levels and probably a reduced response to testosterone. Testosterone is a powerful hormone controlling various aspects of sexual development in males.
- smaller accessory sex organs, including smaller testicles;
- slower sexual maturation;
- distinctly feminine-style regulation of one hormone related to testosterone production;
- greater willingness to assume a receptive-female posture when approached by a sexually stimulated male.

These effects "strongly suggest, though do not conclusively prove, that TCDD impairs sexual differentiation in the CNS [central nervous system]," according to Peterson and co-workers. They go on to say that, "The present study provides the first evidence that TCDD impairs sexual differentiation of the CNS." Sexual differentiation--the full development of a female instead of a male, or vice versa--is affected by hormones circulating in the blood before and after birth.

Furthermore, these studies "strongly suggest" that "the demasculinization and feminization caused by *in utero* and lactational TCDD exposure are irreversible," the Wisconsin researchers say. *In utero* means "in the womb" and lactational means "from milk."

Other effects revealed by these studies include:

- Even the lowest dose tested (0.064 micrograms of dioxin per kilogram of the mother's body weight), yielded consistent reductions in a male offspring's daily sperm production.
 - The developing male reproductive system is more sensitive to the effects of this hormone-like toxicant [dioxin] than any other organ or organ-system studied.
 - the unborn or newborn is about 100 times more sensitive to dioxin than the sexually mature animal.
- What do these studies mean for humans?

The Wisconsin researchers speculate, "Thus the findings from this study raise the possibility that TCDD could potentially affect sexually dimorphic behavior in man if exposure were to occur during fetal develop-

ment." "Sexually dimorphic behavior" refers to the bodily and behavioral differences between men and women.

Peterson and co-workers point out that male rats typically inseminate a female rat with up to 10 times as many sperm as are typically needed to ensure impregnation. Humans, by contrast, typically release only about as many sperm as would be required for fertilization. "As a result," Peterson and his co-workers write, human reductions in sperm production "similar in magnitude to that in rats would be expected to reduce fertility in man." In other words, rats can continue to reproduce despite a reduction in sperm count because they produce an excess of sperm, but humans do not produce excess sperm so a reduction in human sperm count would likely reduce humans' ability to reproduce.⁵

"The real question is how general these effects are," Birnbaum says. Her EPA lab will repeat the Peterson studies with another strain of rats and eventually other species. And if these effects occur in another species? "I would get very concerned [about the potential human-health implications]," Birnbaum told *Science News* reporter Janet Raloff.

At a public hearing on EPA's dioxin reassessment at EPA headquarters in Washington April 28, a representative of the American Paper Institute argued that only the study of humans can reveal anything meaningful about humans. Birnbaum responded somewhat testily, spelling out a dozen ways in which studies of rats and mice reveal useful information about dioxin's potential effects on humans.

June 10 at a Congressional hearing on dioxin in Washington, Assistant U.S. Surgeon General Barry L. Johnson, announced that a new study by the National Institute for Occupational Safety and Health (NIOSH) has found that workers exposed to high levels of dioxin have abnormally low levels of testosterone (male hormone) in their blood streams.⁶ This finding is consistent with the rat studies of Peterson and co-workers. We have learned that this new NIOSH study was presented at a scientific meeting on June 10, but NIOSH sources have so far not released details of the new study to the general public.

At the Congressional hearing June 10, under questioning from Representative Ted Weiss (D-NY), Barry Johnson said that if it were faced with the Times Beach, Missouri, situation today, the U.S. Public Health Service would do exactly what it did 10 years ago, which is to evacuate people from their homes. He said the Times Beach evacuation was the appropriate response and would be repeated under similar circumstances today. Another official of U.S. Public Health Service, Vernon L. Houk, made headlines 14 months ago saying if he had the decision to make over again, he would not evacuate people from Times Beach. Times Beach is a town near St. Louis where an unscrupulous waste hauler spread dioxin-contaminated oil around as a dust suppressant in the 1970s. Horses and other animals became sick and died, and the Public Health Service evacuated the town in the early 1980s.

During the Congressional hearing Dr. Houk's views were further contradicted by the testimony of Dr. Marilyn Fingerhut of NIOSH, who studied the health of 5172 workers exposed to dioxin on the job. (See *RHWN* #219.) Dr. Houk made headlines a year ago when he said that, if dioxin causes cancer in humans at all, it is only "a weak carcinogen." (See *RHWN* #249.) Dr. Fingerhut contradicted this view, reporting that, among workers who had been exposed to dioxin for at least a year at least 20 years ago, there was 46% more cancer than among average U.S. males. During the hearing, Representative Weiss characterized Dr. Houk's views on dioxin as "quirky" and "cockamamie."

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- [1] Thomas A. Mably and others, "In Utero and Lactational Exposure of Male Rats to 2,3,7,8-Tetrachlorodibenzo-p-dioxin. 1. Effects on Androgenic Status." *Toxicology and Applied Pharmacology* Vol. 114 (May, 1992), pgs. 97-107.
- [2] Thomas A. Mably and others, "In Utero and Lactational Exposure of Male Rats to 2,3,7,8-Tetrachlorodibenzo-p-dioxin. 2. Effects on Sexual Behavior and the Regulation of Luteinizing Hormone Secretion in Adulthood." *Toxicology and Applied Pharmacology* Vol. 114 (May, 1992), pgs. 108-117.
- [3] Thomas A. Mably and others, "In Utero and Lactational Exposure of Male Rats to 2,3,7,8-Tetrachlorodibenzo-p-dioxin. 3. Effects on Spermatogenesis and Reproductive Capability." *Toxicology and Applied Pharmacology* Vol. 114 (May, 1992), pgs. 118-126.
- [4] J. Raloff, "Perinatal dioxin feminizes male rats," *Science News* Vol. 141 (May 30, 1992), pg. 359.
- [5] In unrelated studies, Congress's Office of Technology Assessment (OTA) reported several years ago that Americans in their prime reproductive years (ages 20 to 24) have experienced an increase in infertility in recent years. See "Reproductive Dysfunction in the Population," in U.S. Congress, Office of Technology Assessment, *Reproductive Health Hazards in the Workplace [OTA-BA-266]* (Washington, DC: U.S. Government Printing Office, 1985), pgs. 341-364. At the time of this 1985 OTA report, low doses of dioxin were not known to interfere with reproductive systems of rats or humans.
- [6] Barry L. Johnson, "Testimony... Before the Subcommittee on Human Resources and Intergovernmental Relations, Committee on Government Operations, House of Representatives, June 10, 1992," pg. 8. Johnson is Assistant U.S. Surgeon General with the U.S. Public Health Service.

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C00144

Reassessment of Dioxin

C00145

A TALE OF SCIENCE AND INDUSTRY

For the last several years two debates have been swirling around the notorious chemical dioxin. One debate has engaged real scientists in the question, How dangerous is dioxin to humans and to wildlife? The second debate has occurred within industries that release dioxin into the environment and so will be directly affected by the outcome of the science debate.

According to *Chemistry & Industry* magazine (Feb. 18, 1991, pg. 112) the chlor-alkali industry (which produces chlorine, the use of which by many other industries creates dioxin) "has taken an active interest in the course of the scientific debate." In fact, the Chlorine Institute--a trade association--is coordinating a "public outreach program" to "capitalise [sic] on the outcome of a recent Banbury conference on dioxin."

Could such a "public outreach program" have any effect? The *New York Times* took a most unusual step August 15, 1991, when it announced on page one that the U.S. Environmental Protection Agency (EPA) was beginning a year-long review to develop "a new formal opinion on the risks of dioxin." The *Times* often reports that studies have been *completed*, but it rarely reports that studies have been *begun*. Furthermore, *Science* magazine had reported May 17, 1991 (pg. 911) that "(EPA) administrator William Reilly has launched a major new effort to reassess the toxicity" of dioxin--so the *Times*'s August 15th news was three months old.

In the *Times*'s August 15th story, EPA Administrator Reilly not only announced that his agency was undertaking a year-long review of dioxin toxicity, he also took the unusual step of suggesting to the *Times* how the study would come out: "I don't want to prejudge the issue, but we are seeing new information on dioxin that suggests a lower risk assessment for dioxin should be applied," Mr. Reilly told the *Times*.

The next day the *Times* ran an editorial praising federal officials for "sensibly considering new evidence that could lead to relaxation of the current strict and costly regulatory standards [for dioxin]." And three days later the *Times* ran a second front-page story which began, "Dioxin, once thought of as the most toxic chemical known, does not deserve that reputation, according to many scientists." The *Times* did not name any of the "many scientists."

What "new information" about dioxin has EPA Administrator Reilly found? According to *Science* May 17th, the "new information" is a "description [given to Mr. Reilly by two EPA scientists] of a meeting last November at the Banbury Center at Cold Spring Harbor Laboratory that Reilly says made the most compelling case for change [in EPA standards for dioxin]."

Science had written its own description of the Banbury Conference Feb. 8, 1991 (pg. 624): *Science* said 38 dioxin researchers from the U.S. and Europe met at Banbury and "reached an agreement that surprised almost everyone." *Science* went on to say, "And this unlikely agreement on how dioxin works at the molecular level--and some hurried calculations scribbled on a blackboard--could force a dramatic change in how the federal government assesses the risk of this and similar carcinogens [cancer-causing chemicals]."

However, two weeks later--February 22, 1991--*Science* reported something quite different about the Banbury meeting ("Flap Erupts Over Dioxin Meeting," pg. 866), revealing that the meeting had been sponsored by the Chlorine Institute and that the Institute had hired a public relations firm to circulate a summary of the meeting claiming a "consensus" had been reached when in fact nothing of the kind had happened. For example, one letter, from a PR firm to the North Carolina Environmental Management Commission, said, "The Banbury Conference was organized for the express purpose of developing a scientific

consensus concerning a biological basis for risk assessment.... They were able to achieve consensus."

However, independent scientists who had attended the Banbury meeting dispute this view. For example, according to *Science*, Dr. Ellen Silbergeld of the University of Maryland and "a number of other participants" at the conference felt "manipulated and misused." Silbergeld sent a letter to the Banbury Center's director saying, "I am in fact rather astounded by such a product from a Banbury Conference.... The press releases and statements imply that a major focus of the conference was a discussion of the regulatory risk assessments that have been applied to dioxins; this was not the focus of this meeting." "I did not expect to be manipulated by industry and government spokespeople (who are not dioxin researchers, incidentally) to be made into a supporter of their political views on dioxin and risk assessment," Silbergeld said.

According to *Science*, the Chlorine Institute initiated the Banbury Conference and paid for half of it. *Science* quoted The Chlorine Institute's head of communications, saying the Institute believed the meeting could be "beneficial to our interests, particularly our interest in the paper industry." The use of chlorine in industrial processes often leads to the creation of dioxin as a by-product; the paper industry is under great pressure to reduce its dioxin emissions. For example, the newsletter *Environment Week* reported Feb. 14, 1991:

A \$1 million punitive damages award by a Mississippi jury last October against Georgia Pacific [paper company] for alleged dioxin pollution of the Leaf River has touched off a stampede of similar suits against paper mills in Mississippi, Tennessee and several other states. The legal actions, which involve thousands of plaintiffs and billions of dollars in damage claims against major paper companies, have begun to resemble the avalanche of legal problems that hit the asbestos industry in the 1980s.

Enormous dioxin liabilities don't stop with the paper industry. On July 10, 1991, a Missouri jury awarded \$1.5 million to the family of Alvin J. Overmann, a St. Louis trucker who died of cancer; his workplace had been contaminated by dioxin. Several hundred similar lawsuits are pending in the Missouri courts now as a result of the contamination of the town of Times Beach.

Why might the *New York Times* participate in a public relations campaign to promote the view that dioxin is less dangerous than previously believed? James Ledbetter, a media columnist for New York's *Village Voice* said Aug. 27 (pg. 8) said, "For some reason, August is dioxin revisionism month.... Once again, the chemical industry is trying to bamboozle federal regulators and the public, with the eager cooperation of the mainstream press," he said, citing the *Times*. Ledbetter suggested that perhaps the *Times*'s "industrial-strength enthusiasm over eliminating 'strict and costly' standards can be traced to the fact that the New York Times Company has an 80 per cent interest in a Maine paper mill, and a 49 per cent interest in three Canadian paper mills. Indeed, on August 12, just four days before [the *Times*'s] editorial ran, two groups of Canadian Indians filed suit against Kimberly Clark and the Times Company for \$1.3 billion (Canadian), charging that one of the mills has polluted three rivers with dioxin and other toxins," Ledbetter said.

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C00147

DIOXIN DANGERS—WHAT'S GOING ON?

The recent flap in the press over dioxin can all be traced back to a single government scientist who announced in April that he believes dioxin is less able to cause cancer in humans, and is therefore less dangerous, than he believed 10 years ago. In late April, Vernon N. Houk, a 62-year-old government worker, was keynote speaker at a University of Missouri conference sponsored by Syntex, a company that was being sued in a Missouri court by the family of a truck driver who had died of cancer and who had worked for years in a truck terminal that was contaminated with dioxin. Syntex faces another 350 dioxin lawsuits from individuals who lived at Times Beach, Missouri—a town the U.S. government bought out when they found it was heavily contaminated with dioxin, 10 years ago. During his speech in April, Dr. Houk dropped a bombshell: he said he believed he himself had made a mistake 10 years ago when he urged the federal government to evacuate 2200 residents from Times Beach. In August, the *New York Times* turned Dr. Houk's reassessment into two front-page stories and an editorial, the main message of which was, "Many scientists now believe dioxin isn't as bad as we thought." At least 26 other newspapers in the U.S. and Canada jumped on the bandwagon.

As the summer wore on, Dr. Houk himself went even further. In August he was quoted in the *Seattle* (Washington) *Times* (August 18, pg. B3) saying he believed the pulp and paper industry in the northwestern U.S. "has reduced dioxin levels enough to protect public health." He said this was his personal view, not a government opinion. As we saw last week (*RHWN* #248), the paper industry is facing so many dioxin lawsuits that it is now being compared to the asbestos industry. In a story devoted to Dr. Houk himself, the *New York Times* noted that he is now being praised and acclaimed (the *Times* said "lionized") by industrial companies that produce dioxin as a waste product, because they say he has put an end to the "dioxin scare."

Dr. Houk's views on the dangers of dioxin reflect the narrow public health perspectives of the federal government. Standards for exposure to a chemical are generally based on the chemical's ability to cause cancer, and not on its ability to cause other health problems. On this basis, Dr. Houk justifies his change of heart: "If [dioxin's] a carcinogen, it's a very weak carcinogen and Federal policy needs to reflect that," Dr. Houk says.

In actual fact there is much new scientific evidence indicating that dioxin is more dangerous than anyone knew 10 years ago. Recent evidence (summarized in a long article in the *New York Times* May 15, 1991 [pg. C4], and in *Chemical & Engineering News* August 12, 1991 [pgs. 7-14]) reveals that dioxin has many toxic effects on wildlife and humans besides its ability to cause cancer. In one species of animal or another, dioxin causes wasting syndrome [progressive weight loss leading to death]; atrophy of the thymus (a blood-forming organ important in the immune system); atrophy of the spleen (another blood-forming organ important in the immune system); atrophy of the testicles; enlargement, deterioration and death of liver tissue; hyperplasia (excessive cell growth) in the urinary tract and bile ducts; birth defects; and suppression of the immune system. In addition, its ability to cause cancer in laboratory animals, wildlife and humans is now a matter of record (see *RHWN* #219.)

The present understanding of dioxin is that it functions like a steroid hormone. Steroid hormones are powerful chemicals that enter cells, bind to a "receptor" (a protein), form a "complex" that then attaches to the cell's chromosomes where it directly encounters the genetic material, turning on and off chemical switches that may then affect distant parts of the body in various important ways. It is not unusual for very small amounts of a steroid hormone to have major effects on the body.

In animal studies, dioxin is still the most potent poison ever seen. For example, it kills hamsters at one 64-thousandth of the fatal dose of sodium cyanide. The hamsters die of "wasting syndrome"—they lose weight and die. In rainbow trout, northern pike, and salmon, one-tenth of a part per trillion of dioxin in water causes growth retardation of young fish. The

U.S. Fish and Wildlife Service calls dioxin "the most toxic synthetic compound ever tested under laboratory conditions."¹ No new evidence has changed any of this. Back when Dr. Houk was recommending that people be evacuated from Times Beach, in 1981, the danger did not seem remote or theoretical. Seventy-five horses in the town had died. Dogs, rodents, chickens, cats and birds had died. No new evidence has been found in the past 10 years to change anyone's estimate of dioxin's toxicity to these species.

In rhesus monkeys, as little as 25 parts per trillion in the diet causes increased passivity and measurable reductions in the ability to learn.²

"With laboratory animals, it seemed as if dioxin caused just about any effect you can think of," says Dr. Steven Safe, a toxicologist at Texas A&M University. "You name it, it did it, and at extremely low doses," he says. Dr. Safe says the mystery of dioxin has largely been solved by confirmation that TCDD [dioxin] and other toxic agents interact directly with genetic material [the chromosomes that, taken together, comprise the DNA molecule], the way hormones do. It is not unusual for a hormone to have different effects and different potencies in different species of animals. This does not make dioxin any less toxic—it merely makes it better-understood.

Chemical & Engineering News says, "One of the most significant realizations of the past few years is that TCDD [dioxin] cannot be considered by itself... that there are a number of dioxin-like compounds that can all have toxic effects." Dioxins, dibenzofurans, PCBs, and some halogenated naphthalenes all mimic hormones; therefore scientists now believe they can all cause the range of toxic effects attributed to dioxin. This is not good news for anyone because dioxin alone may account for 7 ppt [parts per trillion] in the blood of average Americans but when these related compounds are taken into consideration, the average American may be carrying 100 ppt of dioxin equivalents in his or her blood stream. The meaning of these levels of contamination will become clearer in the next few years.

Even now, far-reaching effects in fish and birds in the Great Lakes are observable at 35 to 65 ppt. Philip M. Cook at the U.S. Environmental Protection Agency laboratory in Duluth, MN, says he believes Great Lakes fish are failing to reproduce already because of the dioxin burden they carry.

Researchers observing wildlife in the Great Lakes are seeing hermaphroditic offspring of birds—for example, male birds with partially developed female sex organs. They are seeing female-female bonding behavior. These potent dioxin-family toxins seem to act like, or interfere with, normal sex hormones like estrogen, producing "chemically castrated" males, and sexually-confused females.

Linda S. Birnbaum, director of the Environmental Toxicology Division of the EPA's Environmental Health Effects Research Laboratory has overall responsibility for the EPA's year-long reassessment of dioxin's toxicity (see *RHWN* #248). Unlike Vernon Houk, for whom "the issue is mostly decided" already (says *Chemical & Engineering News*), Dr. Birnbaum believes when all the evidence has been evaluated, EPA may not change its regulatory number for dioxin much. The number was initially established because of a great fear of dioxin as a carcinogen. Now a whole new set of toxic properties of dioxin, and dioxin-like chemicals, have become apparent, even as the carcinogenicity question has assumed less importance.

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[1] Ronard Eisler, *Dioxin Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*. [Biological Report 85(1.8); Contaminant Hazard Reviews Report No. 8]. Laurel, Maryland: U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, May, 1986.

[2] R. Bowman and others. "Behavioral Effects in Monkeys Exposed to 2,3,7,8-TCDD Transmitted Maternally During Gestation and for Four Months of Nursing," *Chemosphere* Vol. 18 (1989), pg. 235.

[Dated announcement omitted from this reprint.]

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RACHEL'S HAZARDOUS WASTE NEWS #269

Providing news and resources to the Movement for Environmental Justice -- January 22, 1992

EPA's Dioxin Reassessment--Part 1 NEW PICTURE OF DIOXIN'S TOXICITY EMERGES

U.S. Environmental Protection Agency [EPA] is pressing ahead with its "reassessment" of the toxicity of dioxin, aiming to produce a draft report by June. It has been widely rumored in newspapers that EPA's reassessment will downgrade the dangers of dioxin. But in fact it now appears that EPA may conclude in June that dioxin is at least as toxic as the agency now assumes it to be-- and it's even possible the agency will declare dioxin more toxic, not less.

EPA officials announced in April 1991 that "new scientific evidence" showed dioxin may be less dangerous than the agency had said back in 1985 when it first set stringent standards for dioxin exposure to humans. Based on the "new evidence" it was time to reassess the dangers of dioxin, agency chief William Reilly announced in April. Then, as the year-long reassessment got under way, Mr. Reilly jumped the gun and told the *New York Times* August 15 (pg. 1) what the conclusions of his agency's study would be: "We are now seeing new information on dioxin that suggests a lower risk assessment for dioxin should be applied." The *Times* ran the story on page one.

We have since learned that the whole idea of reassessing the toxicity of dioxin originated with executives of four major paper companies who visited Mr. Reilly's office January 23rd, 1991, and that Mr. Reilly's August statement to the *New York Times* reflected the paper industry's agenda, not the views of EPA scientists. At an EPA public hearing on dioxin in Washington November 15, Greenpeace and Alder-Hill Associates of Tidewater, Oregon [(503) 528-7151], released leaked documents including a January 25th, 1991, letter from the four paper company chief executive officers (John A. Georges, International Paper; T. Marshall Hahn, Jr., Georgia-Pacific Corp.; Furman C. Moseley, Simpson Paper; and Andrew C. Sigler, Champion International) thanking Mr. Reilly for his receptiveness to their ideas January 23: "We were also encouraged by what we perceived as your willingness to move expeditiously to re-examine the potency of dioxin and chloroform in light of the important new information that has been submitted with respect to those chemicals."

In their discussions with Mr. Reilly Jan. 23, these executives browbeat EPA for "failure to act on the emerging health science." They claimed there is now a "prevailing view that low-level dioxin exposures do not pose a serious health threat." "Despite this new reality," they said in their January 25 letter, "EPA has taken no tangible or timely steps to revisit its health criteria for dioxin, and has even failed to temper the Agency's zeal in acting on the worst risk estimates...." As a direct result of these EPA failures, the executives told Mr. Reilly, the paper industry is beleaguered by "public fears about risk associated with dioxin which bears no relationship to scientific evidence. A consequence of this atmosphere is that our companies are now the subject of groundless class action toxic tort suits seeking billions of dollars in damages."

In other words, it's entirely EPA's fault that the paper industry has now begun to resemble the asbestos industry--overwhelmed by lawsuits by citizens claiming harm from exposure. In no uncertain terms, these paper company executives told Mr. Reilly how important it was that his agency play down the toxicity of dioxin in public statements: "Reasoned public statements can help calm the needless public alarm that has, in turn, stimulated the proliferation of unjustified legal action against so many companies in our industry," the paper company officials said.

In August Mr. Reilly met the paper industry's need for "reasoned public statements" and the *New York Times* put it on page one: "U.S. Officials Say Dangers of Dioxin Were Exaggerated." At least 26 other major American newspapers picked up this story, and it is now "common knowledge" that dioxin is not as dangerous as officials used to think. William Reilly himself says so.

Meanwhile EPA's scientific staff was pursuing a very different line of reasoning. They had concluded that dioxin not only causes cancer in humans, but also that it interferes with human health in a broad range of ways. EPA scientists are now referring to dioxin as an "environmental hormone."

Based on concern about dioxin's potency as a carcinogen, EPA in 1985 established a very stringent allowable intake of dioxin for humans: 0.006 picograms (or 0.000000006 micrograms) per kilo-

gram of body weight per day. Since the "average" person is assumed to weigh 70 kilograms (154 pounds), this "average" person is allowed to eat no more than $0.006 \times 70 = 0.42$ picograms of dioxin each day. One microgram (μg) is one-millionth of one gram; a picogram (pg) is a millionth of a microgram, or a trillionth of a gram. There are 28 grams in an ounce.

Unfortunately, because of widespread dioxin contamination of the food chain by the paper industry and other sources, the average American eats dioxin in daily quantities ranging from 3 to 50 picograms, thus exceeding the EPA's "safe" dose of 0.42 picograms by anywhere from seven-fold to 120-fold, depending on whose data you accept regarding dioxin contamination of our food supply. [1, pgs. 94-95.].

Here is the basic problem: if the EPA's stringent 1985 standard is warranted, then the dioxin in our food supply represents a major failure of public health protection. Naturally, therefore, it is in the interests of EPA itself--which does not want to look like a failure--and of the dioxin dumpers in the paper industry (and others)--who do not want to look like thoughtless poisoners--to find "new" evidence making dioxin appear less potent. Unfortunately, all the new evidence seems to be pointing in the other direction.

Dioxin is never produced intentionally; dioxin serves no useful purpose and, so far as anyone knows, there are no benefits from dioxin in your body, only risks. Nevertheless, dioxin is created in major quantities as a byproduct: from chlorine bleaching of pulp and paper, from manufacture of certain herbicides (e.g., 2,4,5-T), from manufacture of many plastics (e.g., PVC), from manufacture of many chlorinated hydrocarbon chemicals, and from incineration of municipal solid wastes.

At the heart of the dioxin problem is chlorine. Chlorine is a highly reactive chemical (it tries to hook onto anything it touches, to form a new chlorinated molecule). Because it is so reactive, chlorine almost never occurs in a free state in nature; it is all bound up in the very stable molecule known as sodium chloride, or table salt.

Starting at the turn of this century Dow Chemical, and later other firms, began producing free chlorine as they split salt to get sodium, which they sold commercially as sodium hydroxide (caustic soda). Then they began to invent uses (to create markets) for all the waste chlorine, and thus the chemistry of "chlorinated hydrocarbons" was developed. Unfortunately, chlorinated hydrocarbons--solvents, pesticides, plastics, and a host of other chemicals--have turned out to be uniquely persistent, uniquely able to accumulate in food chains, and uniquely toxic in fish, birds and mammals, like humans.

Now, with EPA's dioxin reassessment chugging along, a new body of knowledge about the toxicity of dioxin is developing rapidly. A review article in *Science News* (Vol. 141, January 11, 1992, pgs. 24-27) summarizes current understanding of dioxin's toxicity--and there is nothing in the new picture that would be comforting to the paper industry or to its fellow dioxin polluters in chemicals, plastics, pesticides or solid waste incineration.

Dioxin has always baffled toxicologists because it produces different effects in different species, and different effects within the same species at various doses. But the new science of dioxin is beginning to explain these mysteries by viewing dioxin as a hormone. Hormones are chemical messengers that travel throughout the body turning on and off various chemical switches. Hormones need only be present in tiny quantities to set off a cascade of major changes in bodily systems, changes not limited to cancer.

Linda Birnbaum--a key member of EPA's team reassessing dioxin--told *Science News*, "If you think of TCDD as a hormone, it makes it easier to understand these very big differences [in dioxin's behavior]."

[Continued next week.]

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[1] Agency for Toxic Substances and Disease Registry, *Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin* [ATSDR/TP-88/23] (Springfield, VA: National Technical Information Service [NTIS], 1989); available from NTIS [phone (703) 487-4650] as publication number PB89-214522.

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Providing news and resources to the Movement for Environmental Justice -- January 29, 1992

EPA's Dioxin Reassessment--Part 2 DIOXIN DAMAGES HUMAN IMMUNE SYSTEM

U.S. Environmental Protection Agency [EPA] is presently reassessing the dangers of dioxin, one of the most toxic chemicals ever tested on laboratory animals. As a result of animal tests, EPA has declared only exceedingly small amounts of dioxin "safe" for the human food supply. Americans eat food routinely containing roughly 10 to 100 times more dioxin than EPA considers safe. [See *RHWN* #269.]

No one makes dioxin intentionally, but many industries create dioxin as a byproduct of their main activity. Industries that emit dioxin into the environment (paper, plastics, chemicals, and solid waste incinerators) are being sued for their emissions by citizens claiming harm from exposure, and one industry--paper--faces billions of dollars in lawsuits.

Partly to please the paper industry, and partly because there were new scientific findings worth considering, EPA chief William Reilly announced a year-long dioxin reassessment to begin in April, 1991.

Now, nine months into the year-long process, EPA scientists responsible for parts of the reassessment have begun talking openly about new findings that make dioxin seem as bad as, or worse than, EPA used to think.

For two decades dioxin has baffled toxicologists. They are used to seeing cancer-causing chemicals that predominantly cause cancer in one organ or another--like asbestos, which chiefly affects the lungs, or benzene, which chiefly affects the blood-forming cells, causing leukemia. But dioxin seems to cause cancer in many organs, raising the general level of cancer in a population without causing a huge increase in any one type of cancer. In addition, dioxin causes certain toxic effects in one species and other effects in another species. Likewise, dioxin at low doses causes one kind of illness, and at higher doses it causes different illnesses. Only recently have EPA scientists concluded that this puzzling pattern occurs because dioxin acts like an "environmental hormone." Hormones are potent natural chemicals that send messages via the bloodstream, turning on and off chemical switches throughout the body, creating an array of effects in different organs. Dioxin behaves this way. Hormones are present in the body in tiny amounts, yet they can trigger huge changes in various bodily systems. For example, it is hormones that trigger the different stages of growth in a fetus, and that cause young humans to go through puberty.

New Information

EPA chief William Reilly was right--there *is* new information about dioxin. But it won't be reassuring to the paper industry. On the contrary, two studies of workers exposed to dioxin, published during the past year, have shown unmistakable increases in cancers of several types. A study of 5172 American workers revealed a cancer rate 46% above the norm.¹ Likewise, a study of 1583 German workers revealed a cancer rate 39% above the norm; among German workers 20 years on the job, the rate was 82% above the norm, and among the most heavily exposed German workers, the cancer rate was three times the norm.² Notably, among female German workers, the risk of breast cancer was doubled. Whereas a year ago one might have argued whether dioxin had ever been shown to cause cancer in humans, now such arguments are only voiced by the kind of people who say it still isn't proven that cigarettes cause lung cancer.

Linda Birnbaum, one of the scientists conducting EPA's reassessment of dioxin, says these two studies have convinced her that dioxin causes cancer in humans, at least at relatively high exposures. But, she told *Science News* (January 11, 1992, pgs. 24-27.), she has an even greater concern about dioxin: "I'm very concerned that much lower exposure to dioxin may result in adverse health effects that are very subtle and difficult to detect." She was talking about dioxin's impact on the immune system.

The immune system is an exceedingly complex network of organs, cells, and chemical secretions (hormones) that react to preserve health in the face of a vast array of hostile microorganisms and toxicants that our bodies encounter every day. The immune system fights against common colds, influenza, and the body's own cells that go haywire and start to multiply uncontrollably (a definition of cancer).

A degraded immune system leaves the body less able to defend itself against hostile forces in the natural environment. Dioxin attacks the immune system.

EPA's dioxin reassessment will "focus much greater attention on toxicological data revealing TCDD's [dioxin's] reproductive, developmental, and immunotoxic effects," says *Science News*. Immunotoxic means toxic to the immune system. Furthermore, "This document [EPA's draft reassessment] will also establish TCDD as the first pollutant to be regulated on the basis of toxicity observed at the cellular level."

This is one reason why the dioxin controversy is being followed so carefully by industry and by environmentalists. It promises to set precedents in the way chemicals are regulated in the future. In the past, chemicals were considered harmless if they caused no "clinical" damage (damage your family doctor might detect). Now, with the dioxin reassessment, evidence of chemical changes inside individual cells is being considered important to a person's well being.

"So far, studies in mice suggest that dioxin's immunotoxic punch occurs in extremely low doses and may well be more important than cancer in determining dioxin's primary health risk," says Birnbaum."

To study TCDD's toxicity to the immune system, researchers use mice, whose immune systems model those of humans. For example, EPA researchers have measured how well TCDD-treated mice withstand the influenza virus. Mice pre-treated with TCDD readily die after exposure to a quantity of virus that rarely kills healthy mice.

Naturally, it would be very difficult to detect such effects in people. If people exposed to unusually high levels of dioxin, say from a solid waste incinerator, had damaged immune systems and consequently experienced various illnesses, no one might ever suspect dioxin as a cause.

People might question whether some of dioxin's low-level effects represent real harm to people, but "...few people will contend that suppression of the immune system is not an adverse health effect," Birnbaum told *Science News*.

Unlike hormones, which remain in the body only a few hours, dioxin has a half-life in the body of seven years. At the end of one half-life, half the initial dioxin remains. What this means is that dioxin has, relatively, a very long half-life in the body, unlike the hormones that it mimics, so it stays around to play havoc with the body's chemical systems year after year. "Thus one TCDD [dioxin] molecule can continuously disrupt normal cell physiology," says *Science News*, citing work by well-known dioxin researcher Thomas A. Gasiewicz at the University of Rochester (NY) Medical School. EPA's Birnbaum, and Michael Holsapple, a well-known dioxin researcher at the Medical College of Virginia, say studies of humans at Times Beach, Missouri, and of Vietnam veterans, were essentially bungled. Holsapple says, "If I were to take mice and ask the same [research] questions that are routinely asked of the populations of Times Beach or in the Ranch Hand study [of Vietnam vets exposed to dioxin-contaminated herbicide], I would come up with a very nebulous picture [of dioxin's immunotoxicity]," says Holsapple. "But when we ask different questions [in mice], we can certainly show very strong effects on the immune response," he says.

Is there a threshold for dioxin's damage to the human body? Is there a level of dioxin below which no effects can be observed? George Lucier of the National Institute of Environmental Health Sciences in Research Triangle, North Carolina, has been asking this question in his laboratory. His data show no evidence of any threshold. "My data might not prove that a threshold doesn't exist," he told *Science News*, "but there's also no evidence of any thresholds." In other words, any amount of dioxin does some damage, according to Lucier's findings. This means the only safe amount is zero.

This conclusion is not what the paper industry wanted to hear when its executives urged William Reilly to initiate EPA's dioxin reassessment. As the reassessment reaches its draft stages early this summer, we'll have new measures of the potency not only of dioxin, but also of industry's muscle in a contest with unwelcome scientific conclusions.

For all of us, much is riding on the outcome.

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[1] Marilyn Fingerhut and others, "Cancer Mortality in Workers Exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin," *New England Journal of Medicine* Vol. 324 (1991), pgs. 212-218.

[2] A. Manz and others, "Cancer Mortality Among Workers in Chemical Plant Contaminated With Dioxin," *The Lancet* Vol. 338 (October 19, 1991), pgs. 959-964.

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DIOXIN DETOXIFICATION CAMPAIGN EXPOSED

A remarkable front-page story in the *Wall Street Journal* February 20 confirmed that the paper and chlorine industries have waged a successful two-year campaign to bamboozle the nation's media about the toxicity of dioxin, and that U.S. Environmental Protection (EPA) fell for it too.

The point of the campaign was to salvage the paper industry, which uses 15% of all the chemical industry's chlorine output, and which is facing billions of dollars in lawsuits brought by citizens claiming damages from dioxin released from paper mills.

The *Journal's* story ("How Two Industries Created a Fresh Spin on the Dioxin Debate") by Chicago-based staffer Jeff Bailey, describes a bald-faced campaign by the American Paper Institute (API) and the Chlorine Institute to "revisit" the scientific evidence that dioxin is a potent carcinogen.

The *Journal* says, "The paper industry scored its first major public-relations success in 1990, when paper companies arranged to challenge the findings of the most influential dioxin study ever done. That study, reported in 1978 by Richard Kociba, a Dow Chemical Co. pathologist, was done on 485 white rats, whose food was spiked with dioxin. Dr. Kociba found a strong link to cancer: a daily dose of billionths of a gram led to tumors."

To counteract the Kociba study, API hired five pathologists and brought them to a Maryland Lab in March, 1990, where for two days they reviewed Dr. Kociba's rat slides under a microscope. The pathologists voted on each slide--were they looking at a cancer tumor or at a "benign" tumor? At the end of the two days, they had voted for 50% fewer cancer tumors than Dr. Kociba had observed 12 years earlier. Robert A. Squire, the pathologist who oversaw the recount, told the *Journal*, "There was't much unanimity. This was an uncertain finding."

Nevertheless API managed to ignore the uncertainties. Based on its "new evidence" that dioxin is less potent than previously believed, API wrote stern letters to the Food and Drug Administration (FDA), to President Bush's science adviser, and to William Reilly, chief of EPA. API told EPA, "All of the Agency's analyses are now out of date in light of the significant new evidence showing that the risks of dioxin has been overstated."

The *Journal* does not say so, but almost immediately the API's publicity machine began cranking out the "news" that dioxin was no longer considered very dangerous. May 31, 1990 the *Washington Post* (pg. A3) surprised the world with the headline, "Scientists Temper Views on Cancer-Causing Potential of Dioxin." The story, by Malcolm Gladwell, said, "Dioxin--the chemical that forced the evacuation of Love Canal, sparked a wave of lawsuits over Agent Orange and became notorious as the most potent carcinogen ever tested--may be far less dangerous than previously imagined, according to new scientific evidence." Gladwell went on, "Enough experts have joined the revisionist chorus that some scientists consider a softening of the government's stance toward the chemical inevitable." Gladwell's "chorus" consisted of quotations from four scientists. Gladwell neglected to mention that three of them were consultants paid by the paper industry.

With the *Washington Post* on board, the "detoxify dioxin" campaign was rolling.

The *Journal* goes on: "Next the Chlorine Institute... arranged to bring three dozen of the world's foremost experts on dioxin to a conference at the Banbury Center [on Long Island in October, 1990]." The *Journal* continues, "Also present was George L. Carlo, a scientist but not widely regarded as a dioxin expert...." "Carlo is not a scientist with a long history of dioxin credentials," Dr. George Lucier of the National Institutes of Environmental Health Sciences told the *Journal*. The *Journal* goes on: "Why was Carlo there? Though described as a 'conference participant' by the Chlorine Institute, he was actually the industry's \$150-an-hour observer. Based on his account, the institute would later circulate reports that the scientists had reached an important consensus...."

Carlo's account, which the Chlorine Institute immediately circulated widely to journalists and to state regulatory officials, said that the scientists at Banbury had reached consensus that dioxin does no harm until a certain threshold of exposure is reached. In other words, Carlo claimed--and the Chlorine Institute sent out press statements claiming--that the Banbury meeting had reached agreement that there is some amount of dioxin that is safe. The *Journal* continues, "The institute's statement, however, didn't accurately reflect what had happened at the conference.... A Chlorine Institute official concedes its representations about the conference were a 'botched publicity effort.' The institute now agrees there was no conference consensus on whether a dioxin threshold exists."

However, before the world had a chance to learn that the Chlorine Institute was playing fast and loose with the facts, the Institute's disinformation about Banbury was fed to William Reilly, chief of EPA, who fell for it. Citing the Banbury "consensus," in early 1991 Reilly ordered his scientific staff to officially "reassess" the toxicity of dioxin.

The paper industry got help from other friends in high places. In May, 1991, a highly-placed federal health official just three years shy of retirement announced that dioxin was much less toxic than previously believed. Dr. Vernon Houk, Director of the Center for Environmental Health and Injury Control, announced that he believed dioxin was only "a weak carcinogen." Houk's statements formed the "news hook" that allowed the *New York Times* to climb on board with its own page-one story August 15, 1991: "U.S. Officials Say Dangers of Dioxin Were Exaggerated." With the *Post*, the *Times*, Houk and Reilly all speaking with one voice, the "detoxify dioxin" campaign was clearly succeeding.

But with the publication of the *Wall Street Journal's* story Feb. 20, the campaign has come unraveled. The scientist in charge of EPA's reassessment, Peter Preuss, is quoted in the *Journal* saying that Vernon Houk's statements "misled" the public about the dangers of dioxin. Other scientists on EPA's reassessment team say dioxin seems to be just the tip of a nasty iceberg--that other chemicals in the environment seem to share dioxin's ability to interfere with the human reproductive and immune systems. If we all carry dioxin in our bodies at an average of 7 ppt [parts per trillion], when you add furans and PCBs [polychlorinated biphenyls], our average body burden of "dioxin equivalents" may be as high as 100 ppt.¹ This is not good news. And it means that any additional dioxins or furans added to the environment would worsen a situation that it already unacceptable from a public health perspective. Knowing this, anyone who intentionally emits dioxins into the environment seems like a logical target for a barrage of lawsuits.

It is now clear that dioxin lawsuits can devastate an industry. For example, the *Wall Street Journal* reported February 7, 1992 (pg. A5), that the Georgia-Pacific Co.--a major paper producer--recently lost two dioxin lawsuits in which juries awarded \$4.2 million to residents living downstream of its paper mill on the Leaf River in New Augusta, Mississippi. Georgia-Pacific has been named in 159 additional lawsuits filed by 8209 plaintiffs who claim they suffered emotional harm after eating fish contaminated with dioxins from the Georgia-Pacific Plant. Furthermore, according to the *Journal*, Georgia-Pacific's insurance carriers say their policies don't cover damages in lawsuits like these. Georgia-Pacific has now sued Aetna Life & Casualty and seven other insurance companies in Mississippi federal court asking a judge to force the insurance carriers to pay. No matter how that lawsuit comes out, someone is likely to have to pay tens, or perhaps hundreds, of millions of dollars--and this represents the problems of only one mill owned by one company.

As the *Journal* commented, "Other paper companies are likely taking note of Georgia-Pacific's setbacks. International Paper Co. and Champion International Corp. are among those who faced similar suits." Likely they are.

Other industries are also likely watching the paper's industry's dioxin troubles proliferate.

For example, cement kilns don't produce dioxin in substantial amounts until they start burning hazardous waste as a fuel, which many kilns have recently started doing. It is interesting to note reports that George Carlo has recently been hired as a consultant by a trade association representing the cement kiln industry.

When will dioxin lawsuits against cement kilns and against municipal solid waste incinerators begin? And how big will the jury awards be in these cases? When your community is deciding whether to install a solid waste incinerator,² or whether to tolerate a cement kiln seeking permission to burn hazardous waste, ask your community leaders, "Have you been reading the *Wall Street Journal* lately?"

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[1] David J. Hanson, "Dioxin Toxicity: New Studies Prompt Debate, Regulatory Action," *C&EN* [Chemical & Engineering News] Aug. 12, 1991, pgs. 7-14.

[2] A good discussion of dioxin emissions from solid waste incinerators appears in Donald J. Lisk, "Environmental Implications of Incineration of Municipal Solid Waste and Ash Disposal," *Science of the Total Environment* Vol. 74 (1988), pgs. 39-66, and in the articles Lisk cites in his footnotes.

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RACHEL'S HAZARDOUS WASTE NEWS #283

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WASTE WARS: THE ARMY OPENS A NEW FRONT

Here's the emerging picture of hazardous waste disposal in the U.S.:

Citizens have blocked construction of all new hazardous waste dumps. There are no new dumps on the drawing boards. EPA [U.S. Environmental Protection Agency] is aggressively promoting hazardous waste incinerators because the alternative--waste avoidance and pollution prevention--would inconvenience the generators of hazardous waste. Rather than inconvenience anyone with power and connections, EPA is promoting incinerators. It's really that simple.

Industry likes incinerators because liability for toxic waste disappears when the waste enters the incinerator. From the Superfund dump-cleanup program where they're spending billions defending themselves against lawsuits, industry knows the liability problem is serious. Incinerators solve this problem because after chemicals pass through an incinerator, there are no labels left on the barrels to identify responsible parties. Most of the waste goes up the smoke stack and can't be traced. The incinerator company--not the original waste generator--bears responsibility for the toxic ash, but in the scheme of things incinerator companies are relatively small and can declare bankruptcy when their ash dumps begin to leak, a few decades from now. EPA will then put the ash dumps on the Superfund list, and the taxpayer will bear all the costs. It's a practical system and it should work.

The only hitch is that citizens are blocking the siting of new incinerators. So EPA is cooperating with the U.S. Army and the Department of Energy (DOE) to solve this problem. The Department of Energy has announced plans to site a huge, privately-owned incinerator on the Hanford Reservation in Washington state. DOE has produced plutonium for A-bombs at Hanford for 40 years. DOE will deflect public outrage about siting the privately-owned incinerator, and Chem Waste will run it, despite Chem Waste's embarrassing record of failure running incinerators (see *RHWN* #280 and #281). For its part, the Army has announced plans to site eight huge chemical-waste incinerators at Aberdeen, Maryland; Richmond, Kentucky; Anniston, Alabama; Pine Bluff, Arkansas; Pueblo, Colo.; Newport, Indiana; Tooele, Utah; and Umatilla, Oregon. The Army's stated goal is to burn up old chemical warfare weapons that now aren't needed. At a public hearing at Aberdeen three weeks ago, the Army said with a straight face it will spend \$500 million building the incinerator, use it for 14 months to destroy mustard gas and then dismantle it, never burning any other defense wastes or civilian wastes. Local people don't believe it, and they fear military incinerators would be as dangerous as civilian incinerators.

But let's be fair. Maybe the private incinerator companies have bungled the job merely because they are careless and cut corners to increase profits. The army would have no such motives and could perhaps be relied upon to do a better job. What do we know about Army experience running incinerators?

The military has already built and operated a modern, state-of-the-art incinerator specifically designed to destroy left-over chemical warfare weapons. To avoid a hassle with local citizens, they built the incinerator on an island in the South Pacific--Johnston Atoll. Environmentalists and local native people opposed the facility but that did not deter the Army. During the period July 16, 1990 through February 27, 1991, the Army fed 7490 M55 rockets containing 75,000 pounds of the nerve agent GB (also known as Sarin), into the Johnston Atoll Chemical Disposal System (JACADS). GB is among the most powerful nerve poisons ever developed.

The operation of the JACADS incinerator was evaluated by MITRE Corporation (McLean, Virginia) under contract to the Army. The MITRE report has been scrutinized by chemist Pat Costner, research director for Greenpeace. Her paper, "Chemical Weapons Demilitarization and Disposal: The Army's Experience at Johnston Atoll Chemical Disposal System," contains valuable data and insights into what we can expect from the Army's path-breaking chemical-weapons incineration program.¹

Here are facts Costner gleaned from the book-length MITRE report:

** During the period of operation, the JACADS incinerator functioned for 500 hours but during the same period it experienced 929 hours of down-time because of malfunctions. The "mean (average) time between failures" throughout the period was 5.6 hours.

** During the 500-hour operating period when agent GB was being burned, the network of monitors for detecting GB releases triggered 776 major process alarms, an average of 22 per day. Major process alarms are "those that are so important that agent or spent decon[tamination] processing is stopped," according to the MITRE report.

A high level of carbon monoxide (CO) in smoke-stack gases is an indication of poor burning. Of the 776 major process alarms that occurred, "the majority" were for high carbon monoxide in the stack gas--a dead give away that the incinerator was experiencing "upsets."

During upset conditions, an incinerator emits large quantities of pollutants into the environment. The EPA Science Advisory Board says, "Even relatively short-term operation of incinerators in upset conditions can greatly increase the total incinerator-emitted loadings [pollution emissions] to the environment."

According to the MITRE report, the JACADS incinerator suffered 90 burner lockouts and 40 fuel flow shut-

downs during the GB-burning campaign, further evidence of upsets. As Costner said in her report, "In other words, the [incinerator] operated in a continuous upset condition during the GB campaign."

On 32 occasions the JACADS incinerator released GB nerve agent into corridors frequented by workers. Furthermore, during 500 hours of incineration, the JACADS workforce accumulated 1944 hours of lost-time accidents. In other words, every hour of operation was accompanied by 3.9 hours of injury-related lost-time among the workers. Costner documents other serious problems with the Army's JACADS incinerator as well.

This is not good news. It means the Army's 8 proposed state-side incinerators will do no better--perhaps considerably worse--than civilian hazardous waste incinerators, which are poor indeed.

Inside their furnaces, incinerators destroy some chemicals, but in the process they create many new toxic by-products that did not exist before, all of which they emit into the environment partly via the smoke stack and partly via the ash, which is buried temporarily in a landfill until it leaks out. Among the most toxic of those chemicals are dioxins and furans. On January 27, 1992, EPA staff presented their latest findings about dioxins and furans to EPA chief William Reilly. *RHW* has obtained a set of 24 acetate transparencies prepared by EPA staff for chief Reilly. They contain important new information about dioxin:

EPA's transparency 17 says:

Recent scientific results and conclusions:

* *Dioxin does cause cancer in humans.*

* *Cancer may not be the most sensitive toxic response resulting from dioxin exposure. Immunotoxicity and reproductive effects appear to occur at body burdens that are approximately 100 times lower than those associated with cancer.*

* *Recent data indicate that there may not be a threshold for certain responses to dioxin. However, the implications for specific risk assessments, such as for cancer, are not yet clear.*

* *Recent evidence has strengthened the conclusion that the sensitivity of humans is similar to that of experimental animals (cancer, immunotoxicity, Ah receptor binding, etc.).*

EPA's transparency 18 says:

New science and conclusions.....continued.

* *Current exposure levels to dioxin and related compounds appear to place people at or near the body burden when sensitive responses may occur, especially for subpopulations at high-end exposure, e.g., nursing infants, recreational and subsistence anglers.*

* *Continuing research into the risk from dioxin exposure should result in a continuing process of reassessment as new data become available and are incorporated into a new, more flexible model.*

* *ORD [EPA's Office of Research and Development] scientists have reached the tentative conclusion that dioxin exposure may have been responsible for the decline of Lake Trout in Lake Ontario as a result of the reproductive toxicity of dioxin.*

Let's review briefly what EPA has said here:

One. Dioxin causes cancer in humans, but cancer is not the most sensitive indicator of damage from dioxin; damage to the immune system, and reproductive damage, are the most sensitive indicators.

Two. Animals and humans are about equally sensitive to damage from dioxin.

Three. The amount of dioxin presently in our food ("current exposure levels," EPA calls it) puts humans at or near the body-burden of dioxin at which "sensitive responses" [immune system damage, and damage to the reproductive system] may occur. Those at greatest risk are infants who drink their mother's breast milk, or people who eat more-than-average amounts of fish because fish accumulate these poisons in their tissues.

Four. In fact, it appears that because fish accumulate these poisons in the fat tissues of their bodies, and because fish eggs have a high fat content and therefore a high dioxin content, Lake Trout in the Great Lakes are not able to thrive. Evidently humans are not the only species endangered by dioxin.

The logical conclusion from EPA's data is that Americans cannot afford to increase the amount of dioxins and furans in their bodies--they are already at or near the levels where immune system damage and reproductive system damage occurs, especially babies. Thus all plans to build new incinerators in the U.S. stand in direct conflict with public health priorities. New incinerators (and uses of chlorine) should be banned, old ones phased out.

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[1] Pat Costner, "Chemical Weapons Demilitarization and Disposal: The Army's Experience at Johnston Atoll Chemical Disposal System." (Washington, DC: Greenpeace, April 11, 1992). Available for \$5.00 from Greenpeace, 1436 U St., NW, Washington, DC 20009. Telephone (202) 462-1177 and ask for Sanjay.

There's a growing network of activists fighting all 9 of the Army's proposed incinerators; contact: Craig Williams, Kentucky Environmental Foundation, P.O. Box 467, Berea, KY 40403; phone (606) 986-7565.

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RACHEL'S HAZARDOUS WASTE NEWS #310

Providing news and resources to the Movement for Environmental Justice -- November 4, 1992

THE N.Y. TIMES DETOXIFIES DIOXIN (AGAIN)

During August and September U.S. Environmental Protection Agency (EPA) released drafts of 8 chapters of its long-awaited report on the dangers of dioxin. The agency then promptly convened a 4-day meeting of independent scientists September 22-25 to review and discuss the drafts.

The day after the meeting ended, the *New York Times* reported (Sept. 26, 1992, pg. 9) that "An independent panel of scientists concluded today that dioxin was not a large-scale cancer threat except to people exposed to unusually high levels of the toxic compound in chemical factories and from accidents."

The *Times* said, "The panel based its conclusions about the effects of dioxin on human health largely on four studies...."

The *Times* story said, "...the risk to average Americans exposed to dioxin, principally by eating beef, dairy products, chicken and fish, is lower than previously believed."

The *Times* reported that, "The risks, said several panelists, are largely confined to chemical workers and people exposed to high levels of dioxin from industrial accidents. They said the levels of dioxin ordinarily found in the environment had not been shown to be dangerous to people."

The *Times* went on, "Scientists have known for a long time that laboratory animals are apparently much more sensitive to the dioxin molecule than people for reasons that are not well understood, and newer studies have shown that trout, salmon, and some species of birds also are very sensitive."

The *Times* concluded, "In separate interviews, several panelists said today that they did not consider the levels of dioxin in most Americans, about 5 parts per trillion in fat, to be an important health hazard."

In sum, the *New York Times* led its readers to believe that a panel of independent scientists reviewed four studies and concluded, apparently by consensus, that dioxin is only a danger to the health of heavily-exposed chemical workers and victims of rare accidents, and even for them the only danger is cancer; that existing levels of dioxin in the environment are not dangerous to people; that the risks of dioxin are lower than previously believed; that laboratory animals and some wildlife may be at risk but this has little to do with humans because humans--for reasons unknown--seem to be uniquely unaffected by low doses of dioxin.

The *Times* put this comforting story on the wire and many U.S. newspapers picked it up. The result was a nation-wide press blitz saying dioxin is basically harmless to humans. For example, the *Detroit News* reprinted the *Times* story, then capped it off Sept. 29 with an editorial, "The End of Dioxin Hysteria?" which concluded, "But the fact that dioxin has turned out to be something of a non-issue where humans are concerned suggests the religious zeal with which some environmentalists are trying to close down the chemical industry deserves to be greeted with extreme skepticism."

Is this the last word on dioxin? It's not a threat to human health; levels found in the environment today are of little concern; the risks used to seem worse than they seem today; and the risks are limited to cancer among chemical factory employees. Is that what EPA spent 18 months and untold millions of dollars to find out?

It seems not. Other reporters and participants in the four-day meeting—including top EPA officials who called the meeting—came away with views and information almost 180 degrees out of sync with what the *New York Times* reported. On October 9, Erich W. Bretthauer, EPA's Assistant Administrator for Research and Development, sent a memo to EPA chief William Reilly giving Reilly his own views on what EPA has learned so far about dioxin. In his memo, Bretthauer made seven points, each of which was either contradicted, or missed entirely, by the *Times*:

1. To understand the risks of dioxin, we should consider a broad range of health effects, not just cancer, Bretthauer told Reilly.

2. Dioxin has been observed to cause certain non-cancer effects in animals by disrupting the body's endocrine system (glands and tissues that control bodily functions via chemical messengers called hormones). These endocrine effects include reproduction, behavior of offspring, and changes in the immune system. "Some data suggest that these effects may be occurring in people at body burden levels that can result from exposures at, or near, current background," Bretthauer told Reilly. In other words, the amount of dioxin already present in the environment, and in the bodies of Americans, is at, or close to, levels that, in animals, cause hormone shifts, reproductive disorders, changes in behavior, and immune system damage.

3. Recent studies indicate that dioxin causes cancer in humans; these studies need to be evaluated further and then EPA needs to form a new official position about the cancer hazard to humans.

4. Additional compounds besides dioxin (for example, some types of PCBs) have dioxin-like effects

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and should be included in EPA's reassessment of the hazards of dioxin.

5. There is insufficient data to develop a model that will allow us to predict the cancer hazards to humans from low-level exposure to dioxin. During the next 3 to 5 months, government studies may provide the needed data.

6. The available data seem to indicate that dioxin will cause cancer in humans in proportion to the exposure--high doses will cause many cancers, lower doses will cause fewer cancers, and the only dose that is risk-free is zero. In other words, the so-called "linear hypothesis" of cancer causation appears to hold true in the case of dioxin, though this is not certain.

7. Risks from existing background levels of dioxin in the general population need to be "carefully considered."

The *Wall Street Journal* (Oct. 16, 1992, pg. B9) summarized Bretthauer's memo this way: "Data reviewed by an independent scientific panel suggest that the danger from dioxin may be broader and more serious than previously thought, according to an internal Environmental Protection Agency memo."

The business journal, *Environment Reporter* [ER] (Oct. 2, 1992, pg. 1504), offered its own coverage of the Sept. 22-25 meeting. The basis of ER's story was an interview with William H. Farland, director of the EPA Office of Health and Environmental Assessment, the man in charge of the EPA's reassessment of dioxin. Farland summarized the four-day meeting by saying dioxin is "a major health threat," and that the draft report on dioxin is "unlikely to ease public concern over dioxin."

Farland said that "scientists at the recent meeting reported a host of non-carcinogenic effects at very low dose levels--near background levels--as well as the ability to cause cancer in humans at high doses," *Environment Reporter* said.

In sum, EPA's scientific reassessment of dioxin--which is based on several thousand studies of dioxin--isn't over yet, but so far the scientific evidence is showing dioxin to be a worse problem than formerly believed. The reassessment was initiated by EPA chief Reilly 18 months ago in response to complaints by the paper and chlorine industries, who charged that EPA's regulation of dioxin was too strict because low doses of dioxin are harmless. These industries argued that there is a "threshold," an amount of dioxin below which no effects will occur. EPA's scientific reassessment of dioxin has, so far, substantiated EPA's original view of dioxin, that it is a potent toxin for which there is no observable threshold. Furthermore, the reassessment has added a host of new concerns, which were discussed at length during the four-day meeting--concerns about disruption of the reproductive system, harm to the immune system, and behavioral changes in offspring of dioxin-exposed parents.

The really big news from the meeting was the revelation that these endocrine-system effects in animals are observable at body burdens similar to the body burdens in Americans today. If this view is upheld in the next few weeks as scientists continue to review the available data, it will mean that any addition of dioxin to the environment will be adding to an already-unacceptable situation. This would provide a scientific foundation for a demand that "zero discharge" be adopted as the basis for control of dioxin. William Farland nearly said as much in his interview with ER: "We have to be very cautious about any additions of dioxin to the environment. We must be very concerned about these high background levels of dioxin and what they may mean for human health."

Industries that emit dioxin into the environment--paper producers, waste incinerators, metal smelters, and herbicide producers and users--are feeling tremendous pressure to curtail emissions, and the pressure seems likely to increase.

Newspapers, of course, are dependent upon paper for their existence and they have a material interest in keeping paper prices low. According to the *Wall Street Journal*, the paper industry has already spent over a billion dollars trying to control dioxin.

The *New York Times*, which has a history of odd reporting on dioxin (see *RHWN* #248, #249, #275), received at least three letters to the editor complaining about its misleading coverage of the September meeting--two of them from scientists who participated in the meeting and who objected strongly to the *Times*'s reporting. The *Times* also received a letter from a consultant to the incineration industry who said it was "comforting" to learn that the dioxin found now in the bodies of Americans is "less toxic than previously assumed." The *Times* did not print either of the letters from the scientists but did print the letter from the incineration consultant, who argued that automobiles are the leading source of dioxin. This point of view, which is no doubt comforting to the incineration industry, and to the paper and newspaper industries, is almost certainly wrong. We will provide more details next week.

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