

FROM THE DESK OF
DON DANLY

JUN 29 1979

~~Harry~~ ~~Don~~

Since we are
not involved in
chlorination in any
NI plants, COD
is not at all a
likely impurity.

Don

C07408

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~~campbell~~
 Can you conceive of
 any way we might
 produce CDD in NT

FROM
 (NAME-LOCATION-PHONE)

H. M. Keating - G4WA (4-8171)

DATE

June 22, 1979

cc: J. T. Garrett - A2SG

S. L. Hunter - G4WA

G. E. Paget - A2SC

W. B. Papageorge - G4WA

SUBJECT

CHLORINATED DIBENZO DIOXINS (CDD)

REFERENCE

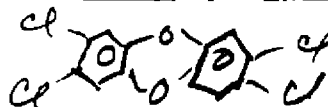
TO

W. G. Juhl - 1890

W. R. Richard - R2E

COMPANY CONFIDENTIAL

NO -



11 mis-

Common only. Since Cl^- / Cl_2 not used, it doesn't
 seem possible. (Can see Krummrich on chlorinating
 phenols.)

This is a request to identify all processes that in the light of feasible chemistry might give rise to CDD's in the workplace, product, by-product, or effluent. The request stems from an OSHA visit to the W. G. Krummrich plant, and a subsequent allegation by OSHA that CDD's were detected in measurable quantities in one product sample and one wipe sample. A citation followed, alleging violation of the OSH Act General Duty Clause.

To be certain that we are using a common base of understanding, I am including below excerpts from a draft prepared by Dr. G. E. Paget, DMEH.

"Nomenclature"

It is common for the class of compounds under discussion to be referred to as "dioxins" (c.f. Krummrich citation). This is incorrect. We are specifically considering chlorinated dibenzo dioxins, and to avoid falling into the trap of loose nomenclature, this whole class will be referred to here as CDD's. Of particular concern within that generic class are the tetrachlorinated species and this group (of 22 isomers) will be referred to as TCDD's. Of this group of 22 isomers, one is of particular concern. This is the 2,3,7,8. isomer and this will be referred to as 2,3,7,8. TCDD.

Background Biology

Several CDD's are extremely toxic experimentally and 2,3,7,8. TCDD is especially so. In various species, single doses of 2,3,7,8. TCDD of the order of 1 - 6 μ g/kg. produce serious and irreversible changes, ranging from death through teratogenesis and carcinogenesis to long-lasting biochemical anomalies.

Knowledge of the toxicology of CDD's and 2,3,7,8. TCDD in particular is still far from comprehensive.

Even more incomplete is knowledge of human response to these materials. It is known that the most obvious response is chloracne, and that CDD's are powerful causes of this condition. Other effects of exposure are less well established. However, evidence

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Background Biology (continued)

suggests inconclusively that humans may be less susceptible to CDD toxicity than most laboratory species. In the experimental animal, the half-life of TCDD's is of the order of 30 days. In the environment TCDD's are more stable, with a half-life of circa one year. They are firmly bound to soil and do not readily migrate.

In the absence of precise knowledge of human susceptibility, and in view of the extreme sensitivity of some species, it is obvious that very small amounts of TCDD in the human environment, in certain circumstances of intimate exposure, or of abuse or misuse, or casual worker procedures, may (we repeat may), have significance.

Background Chemistry

It appears that in the very small concentrations of which we speak of CDD's (p.p.b. and ppt, or $\mu\text{g}/\text{m}^2$) CDD's can be formed under a wide variety of conditions. They appear to be very frequently formed by combustion of many materials and may, therefore, be of ubiquitous appearance at low levels in the environment. Of importance to Monsanto is the fact that it appears that they may be formed when any aromatic substance is chlorinated under industrial conditions where, for example, some water usually contaminates so-called dry intermediates although some chemical processes (chlorination of phenols under alkaline conditions) are more likely to produce CDD's than others. Because of Monsanto's involvement with such processes at several locations, CDD's are of particular concern to us.

Analytical Background

It is important to distinguish between detection of CDD's generically, the detection of TCDD's generically, and the specific identification of 2.3.7.8. TCDD.

Many laboratories, including Monsanto, can perform the first two identifications at high levels of sensitivity. Few can perform specific 2.3.7.8. TCDD analysis at high sensitivity. Some that claim to be able to do so are mistaken. It is probable that Dow can perform this analysis, although they do not do so routinely.

It is also important to bear in mind the matrix in which the CDD of interest is found. Some matrices - water for example - present fewer problems than others and Monsanto analysts are already capable of performing analysis of TCDD in such matrices at very high levels of sensitivity. Other matrices, particularly products, present very many more difficulties, and we have to accept that in some circumstances the state of the art will not permit low level CDD analysis, and particularly 2.3.7.8. TCDD identification.

Additionally, achieving the ultimate in analytical sensitivity for TCDD's is extremely demanding of time and skilled labor. It cannot be undertaken on a routine "quality assurance" basis with any acceptable commitment of resources, but must be reserved for special circumstances."

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To give guidance on what might be considered "presence of CDD's", OSHA alleged having found 300 ppb of total CDD's in a product sample, and 642 nanograms of CDD's in a wipe sample probably taken on a surface of 100 sq. CM. It is probable they would have made the same allegations even if the quantities they report were much lower.

As further background information, attached is a copy of a Dow article from C & E News.

Response by July 13 is requested.

H.M. Keating
H. M. Keating *rl*

HMK:ku
Attachment

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Dow finds support, doubt for dioxin ideas

Scientists and regulators examine Dow Chemical's claim that most dioxins enter the environment from combustion, not pesticide manufacture

Rebecca L. Rawls
C&EN, Washington

According to the ancient Greeks, Prometheus stole fire from the gods and brought it to mankind, thereby bringing civilization, culture, and benign technology.

But the effects of this deed were not all beneficial. Zeus, angered that humans now had fire as the gods did, sent Pandora with a box, containing all the plagues and disasters of mankind, to take away some of the advantage that having fire had brought to humans.

There may be some chemical truth in this Greek myth. In a report it calls "The Trace Chemistries of Fire," Dow Chemical recently has said that several chlorinated organic compounds that have been causing increased concern as environmental pollutants may not be exclusively the products of the synthetic chemical industry. They are also being formed in trace amounts in all forms of combustion. Among the chemicals they find in the emissions of everything from power plant stacks to home fireplaces to automobile exhausts are polychlorinated biphenyls, polychlorinated dibenzofurans, and dioxins.

It is the possibility that dioxins—and particularly 2,3,7,8-tetrachlorodibenzo-p-dioxin (or simply 2,3,7,8-TCDD)—might be entering the environment from a wide variety of combustion sites that is focusing the attention of scientists, environmentalists, and government officials on the Dow report.

2,3,7,8-TCDD is one of the most toxic substances known for some laboratory animals, causing death at levels as low as 0.6 microgram per kg body weight in certain species. Its presence as a contaminant in Agent Orange, an herbicide used by the Air Force to defoliate sections of Vietnam in the late 1960's, raised ethical questions that eventually resulted in discontinuation of the herbicide spraying program in 1970.

These questions form the basis of a class action suit brought this winter by Vietnam veterans who claim their health

has been jeopardized by exposure to this chemical.

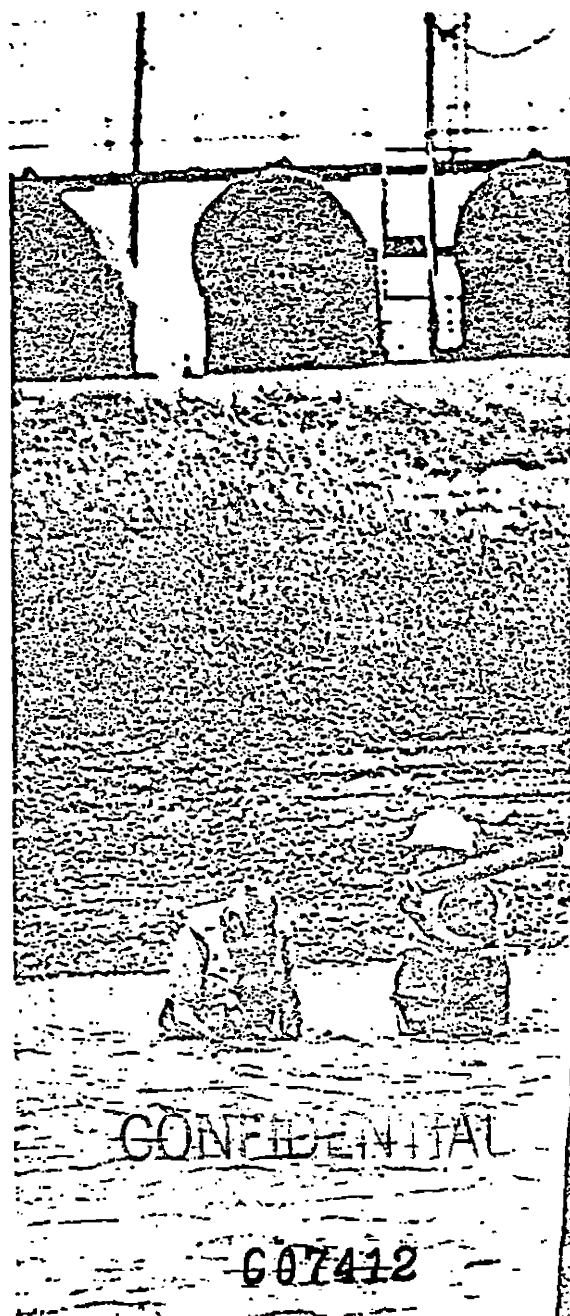
2,3,7,8-TCDD is also present in several herbicides used in the U.S. The question of whether these herbicides with 2,3,7,8-TCDD cause unacceptable risk to human health and the environment has been a matter of concern to the Environmental Protection Agency practically since it was established.

EPA is currently in the Rebuttable Presumption Against Registration (RPAR) stage of its review of the safety of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), the first pesticide containing 2,3,7,8-TCDD to come up for review. EPA requires 2,3,7,8-TCDD concentrations in 2,4,5-T to be less than 0.1 ppm. Current manufacturing practices result in about one tenth this level. The RPAR process involves a year-long period of scientific evaluation and public comment on the benefits and risks associated with use of the pesticide. A decision based on this evaluation is expected from EPA in April.

Dow's interest in this area, too, centers on dioxins. The company is one of three major producers of 2,4,5-T in the U.S. (the other two are Thompson-Hayward Chemical Co. and Transvaal Inc.) and the only U.S. producer of 2,4,5-trichlorophenol, the chemical intermediate whose production inevitably results in some formation of 2,3,7,8-TCDD. It is 2,3,7,8-TCDD from this source that eventually is carried over into 2,4,5-T and other products.

Dow began the four-and-a-half-month study that resulted in its report as an effort to find the source of parts-per-billion levels of "a compound that satisfies the analytical criteria of 2,3,7,8-TCDD" in fish taken from the Tittabawassee River on which Dow's Midland, Mich., chemical complex is located. This is the plant where Dow makes 2,4,5-T, 2,4,5-trichlorophenol, and another dioxin-containing compound, pentachlorophenol.

"If you were standing outside Dow Chemical Co., looking in at us, you'd say, 'Aha! You've got dioxins in the Tittabawassee River, and therefore, they are



Dow researchers take samples from the Tittabawassee River near Dow's plant



Bumb: our motives were suspect

coming from 2,4,5-T, trichlorophenol, and pentachlorophenol. The way they are getting into the river is you are discharging them in the waste streams, they are going through your collection pond, and they are coming out into the river," says Dr. Robert Bumb, director of research for Dow's production unit, called the Michigan division. "And, I've got to admit, that

makes good sense, if you are standing outside and looking in. If you are inside Dow, you wouldn't say that. We monitor those plants very, very closely, and we didn't think at the time the dioxins were coming from those plants," he continues.

Dow reported its findings of 2,3,7,8-TCDD in fish to Michigan's Department of Natural Resources last June. The company made a commitment to the state authorities that it would find the source of the dioxins in the fish. A task force was put together, headed by Bumb, which averaged about 30 members. This group worked full-time on the project from early July until mid-November, when the "Trace Chemistries of Fire" report was released. Altogether, Dow spent about \$1.8 million on the project, primarily to collect samples and analyze them.

Dow announced its findings at a press conference instead of taking the more conventional route of publishing them in the scientific literature (C&EN, Nov. 20, 1978, page 7). It concluded that the dioxins in the river did not get there from the discharges of its plant, but rather from "normal combustion processes that occur everywhere."

"We now think dioxins have been with us since the advent of fire," Bumb said at the time. "The only thing that's different is our new ability to detect them in the environment."

Not surprisingly, the report was received with skepticism by most of the press and by some government officials. The conclusions were so clearly favorable

to the company that, as Bumb puts it, "Our motives were immediately suspect."

However, Dow's report does not exactly say that there is no connection between the Dow plant and the dioxins found in the river. "Dow has never said that the dioxins in the Tittabawassee didn't come from Dow," says Bumb. "We say they probably are coming from this plant. But there is a difference. We say that the dioxins that are in the Tittabawassee, we believe, are due to combustion and not due to discharges from the pesticide or herbicide plants."

Dow also doesn't say, according to Bumb, that because dioxins are being produced throughout the environment and are ubiquitous, their contamination in pesticides is not important.

"If you think it's our position to say it's fine that pesticides contain dioxins, we're producing them everywhere, so let's not worry about them, that's not what we're saying," he insists.

"The concentration of dioxins in the environment from combustion and the concentration from the use of pesticides are separate issues, and we have never tried to make them the same issue."

Taken as a separate issue, Dow insists, its study of dioxins from combustion is a fascinating and significant piece of environmental chemistry. One of the first things that it did after setting up the task force was to take dust samples from its own analytical laboratories to calibrate the "baseline" level for its analytical techniques. Many studies of 2,3,7,8-

2,3,7,8-TCDD's toxicity has been widely studied on a variety of animals

2, 3, 7, 8-Tetrachlorodibenzo-*p*-dioxin is, without question, very toxic stuff. But a consensus has not yet developed among scientists as to just how toxic it is, especially to humans.

Toxicologically, 2,3,7,8-TCDD is a puzzling compound. It has been one of the most extensively studied of all chemicals, but so far its mechanism of action has not been established. Acute toxic doses vary as much as 1000 times from one animal species to another. Indeed, it doesn't seem to affect even the same organs in all animal species. Many of its oncogenic and teratogenic effects are both contested and confusing. And what its toxicological effect is on man, based on animal studies and limited human exposure, is currently the subject of fiery debate.

Much of this debate is being focused by the Environmental Protection Agency's pending review of the continued registration and use of pesticides that contain trace amounts of 2,3,7,8-TCDD as an impurity. EPA is attempting to pull together all available data on human and animal toxicity to evaluate the possible hazards involved in use of those pesticides. Its report, based on a year of intensive scientific review and public comment, is due in April.

Meantime, some of what's known about 2,3,7,8-TCDD toxicity follows:

Acute toxicity. The most sensitive laboratory animal to 2,3,7,8-TCDD is the guinea pig, for which the LD₅₀ level, or the dose needed to cause death in half the animals receiving it, is 0.6 microgram per kg of body weight given as an oral dose. This toxicity level is comparable to that of some of the most toxic natural substances, such as mold toxins, and it makes 2,3,7,8-TCDD one of the most acutely toxic substances known.

Other lab animals are much less sensitive than the guinea pig, however. Male rats have an oral LD₅₀ of 22 micrograms per kg; female rats are only about half as sensitive with an LD₅₀ of 45 micrograms per kg. Recent data on monkeys set their oral LD₅₀ for 2,3,7,8-TCDD at less than 70 micrograms per kg. Rabbits are less sensitive, at 115 micrograms per kg, and dogs are even less sensitive.

In some animals TCDD leads to death by causing lesions in the liver, Dr. Richard J. Kociba of Dow Chemical Toxicology Research Laboratory explains. But in some other species, including the very sensitive guinea pig, no single organ appears damaged enough to be responsible for the animal's death.

Instead, the animal seems to waste away in a syndrome that looks much like starvation.

Estimates of acute toxicity to humans are undergoing revision based on evidence collected following a chemical plant release in Seveso, Italy, in July 1976. The explosive release spread 2,3,7,8-TCDD, contained as a contaminant in trichlorophenol, along with other contained substances over an area of several hundred acres near the plant (C&EN, Aug. 23, 1976, page 27). Estimates vary as to how much 2,3,7,8-TCDD was dispersed over this area but center on several kilograms.

"If you calculate it out, that dose should have killed every human being within miles," based on projections from acute toxicity in guinea pigs, says Kociba. "But, to the best of my knowledge, the only thing that was observed in humans following the Seveso incident was some chloracne [a skin condition associated with 2,3,7,8-TCDD exposure] and some transient liver toxicity that has pretty well reversed itself in the months following the episode," he says. No human deaths have been directly attributed to 2,3,7,8-TCDD exposure at Seveso, Kociba says, "When we extrapolate to man, it appears that TCDD

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TCDD in the environment indicate that it breaks down fairly rapidly via photodegradation in the air. Where it persists, it usually does so in soil, water, or animal tissues.

Dow found, however, not a background level of dioxin in the lab dust samples but levels in some samples of more than 8700 ppb for all dioxin species combined. These samples contained up to 2.6 ppb of 2,3,7,8-TCDD and the other 11 isomers that the analytical procedure used could not separate from the 2,3,7,8 isomer.

It is the 2,3,7,8 isomer that contaminates trichlorophenol and its derivatives. These dust samples, however, contained an array of dioxins including octachloro, heptachloro, and hexachloro species and tetrachloro isomers that could be distinguished from the 2,3,7,8 isomer.

"That told us that the problem was not a waterborne problem, but an airborne problem, because where we collected the samples in the laboratory had never been and could never be under water," Bumb explains.

He goes on, "So we then said, all right, if the soil or air in Midland has dioxins in it, then we have to find control areas where there are not dioxins. So we went around to other cities in Michigan, cities outside of Michigan, and the upper part of the lower peninsula of Michigan and took samples. ... We found varying amounts of the dioxin isomers, some of which included TCDD, some hexa, some hepta, and some octa species. These were dust and soil samples. We found chlorinated dioxins everywhere we looked, in

Dioxins, though ubiquitous, concentrate near Dow plant

Type of chlorinated dibenzo-p-dioxins

Parts per billion	Tetra (TCDD)				
	2,3,7,8 fraction ^a	Others	Hexa	Hepta	Octa
Soil samples					
Dow plant	16	17	280	3200	20,500
	100	18	120	650	6,300
Rural sites	N.D. ^b		N.D.	N.D.	N.D.
	N.D.		N.D.	N.D.	0.1
Metropolitan sites	N.D.		1.2	1.6	2.0
	0.03		0.31	3.3	22.0
Dust samples					
Dow research building	1.0	0.5	18	240	980
	2.3	2.3	35	1200	7,500
Midland, Mich.	0.03 ^b		0.2	2.3	19
	0.04		0.4	3.9	31
Metropolitan sites	N.D.		0.09	0.8	3.5
	N.D.		0.3	1.0	3.8
	0.04		0.34	3.2	8.2

^a This fraction contains 2,3,7,8-TCDD and 11 of the 21 other TCDD isomers. ^b TCDD isomers were separated only for samples taken at the Dow plant. Notes: Table shows data from only a few of the samples taken include sample with the highest total dioxin concentration taken from each type of site. N.D. = no detectable peak at 2.5 times background.

every sample we took, although not necessarily TCDD.

"We were now coming to the conclusion that finding them in cities and in the soil was not a function of any use of pesticides or herbicides because these products aren't used in cities. We were coming to the conclusion that they were probably due to combustion."

Why combustion? Because of the high concentrations of dioxins found in cities

and the fact that the route of entry into the environment was by air. "The only things that have large volume exhausts in cities are combustion sources," Bumb says, "so that's got to be one route."

"We then looked at what you've got to consider unusual sources," Bumb explains, "for example, the mufflers of diesel automobiles. We grilled steaks. ... We scraped in fireplaces and checked home electrostatic precipitators, and the dioxins

on an acute basis is probably much less toxic than for some of the laboratory animals," he concludes.

Oncogenic effects. Oncogenicity is the property of a substance to produce or induce benign or malignant tumors in living animals. Most of the work on the oncogenicity of 2,3,7,8-TCDD by itself (and not as a contaminant in pesticides) has been done using the Sprague Dawley strain of rats. Kociba and his associates at Dow have conducted lifetime studies using 100 rats at each of three exposure levels and 172 control animals. Animals at the highest dose level, 2200 ppt in their food, showed many toxicological effects, particularly in the liver. They had a higher incidence of tumors of the liver, lung, and mouth. However, this strain of rats has a significant incidence of spontaneous tumors and showed a decrease in the number of these tumors in the pituitary, uterus, mammary gland, pancreas, and adrenal gland.

At the intermediate dose level of 210 ppt, Kociba found some toxic effects to the liver and other organs, but no abnormal incidence of tumors. The lowest dose, 22 ppt, showed neither oncogenic nor other toxicologic effects for the lifetime of the animals.

Preliminary data from a study currently under way, sponsored by the National Cancer Institute, using the same animal strain shows a similar increase in the incidence of certain tumors at high dose levels and a low dose level that can be tolerated for the lifetime of the animal without any increase in tumors, Kociba says. However, a study in the same rat species conducted by Dr. J. P. Van Miller and associates at the University of Wisconsin has found an increased incidence of tumors in random organs at doses as low as 5 ppt.

One study finds an elevated incidence of liver cancers among Vietnamese following the wide application of Agent Orange, a defoliant contaminated with 2,3,7,8-TCDD, by the U.S. military in that country in 1961 and 1962. The increase, from 159 or 3% of all cancers over a seven-year period to 791 or 10% of all cancers, begins in 1962. Cancer ordinarily has a much longer latency period than this, and the study does not rule out other possible sources of the increased cancer incidence. Seveso residents have not yet shown an increase in cancer incidence following their 2,3,7,8-TCDD exposure.

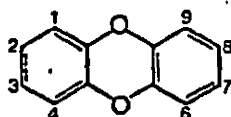
Teratogenicity. 2,3,7,8-TCDD causes a number of birth abnormalities in some

strains of mice and rats, including cleft palate, and kidney, skeletal, and intestinal tract abnormalities. These effects can be seen at levels as low as 0.01 microgram per kg in some studies with rats. The compound is also toxic to the fetus at levels as low as 0.125 microgram per kg in rats and 0.3 microgram per kg in mice. Because of the narrow range that is found between the no-effect level on the fetus and the lethal effect on the mother, some researchers classify 2,3,7,8-TCDD as a weak animal teratogen.

The evidence from Seveso concerning teratogenic effects on humans so far has all been negative, according to Kociba. Extensive data from Italian health officials and the World Health Organization gave "absolutely no indication of increase in birth defects, abortions, or other teratogenic effects that could be correlated with the Seveso exposure," he says.

However, EPA is studying eight women who live in or near Alsea, Ore., near forests that are sprayed periodically with herbicides containing 2,3,7,8-TCDD and who say there is a close correlation between the time the forests were sprayed and 10 miscarriages the women had between 1973 and 1977.

Dioxin isomers cause naming problems



The trouble with using nonscientific names for chemical compounds is that they can be imprecise, leading to confusion as to exactly what compound is being cited. A perfect example is dioxin. This term is the one most commonly used, especially in the nonscientific literature, for any or all of the chlorinated dibenzo-*p*-dioxins.

The parent compound, at least as far as names are concerned, is shown above. It can be chlorinated, singly or in multiples, at any of eight positions (numbered 1 to 4 and 5 to 9 in the diagram). Thus, theoretically there are 75 possible chlorinated species—one octachloro molecule; two isomers each of the monochloro and heptachloro forms; 10 each of the dichloro and hexachloro forms; 14 each of the trichloro and pentachloro molecules; and 22 tetrachlorodibenzo-*p*-dioxins.

Of these compounds, by far the most toxic to laboratory animals, and pre-

sumably to humans, is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. As it is commonly used, the term dioxin almost always applies to this compound, but how many of the other 74 chlorinated dioxins are included as well is often not clear.

And this compound also is called TCDD—for tetrachlorodibenzo-*p*-dioxin. This term, too, sometimes means only the 2,3,7,8-isomer, and sometimes combinations of that isomer with any or all of the other 21 tetrachloro species.

The problems with dioxins are not limited to imprecise nomenclature. The analytical chemistry of the dioxins is advancing very rapidly, but it is still not possible to separate all 75 compounds from one another. This field is advancing so rapidly, however, that, for many studies, including Dow's, the level of separation possible when the study was begun already has been surpassed by the time the study is completed. Thus, using consistent nomenclature even within one study is sometimes tricky.

In this article, dioxin means any members of the entire family. TCDD means any or all of the tetrachloro isomers. The specific isomer 2,3,7,8-tetrachlorodibenzo-*p*-dioxin is referred to as 2,3,7,8-TCDD.

at combustion sources probably will show fixed ratios of the various dioxins, as the European investigators find.

As the dioxins persist in the environment, however, oxidation and reduction reactions occur simultaneously, and dioxins formed at different sources mix. The result is a band of dioxin concentrations that doesn't match what's expected from any known source.

"And that's what we do find," says Bumb.

Dow also finds that the ability of dioxins to persist increases greatly when they are bound to particulate matter, compared to when they are free. For instance, free dioxins are destroyed, or at least reduced to levels that cannot be detected by current methods, by incineration at 800° C with the proper combustion air conditions. But dioxins bound to particulate matter can pass through incinerators operating at temperatures as high as 1150° C with no change in concentration.

It is this particulate-bound dioxin that is probably being emitted by combustion sources. Its environmental fate has not been studied so well as that of free 2,3,7,8-TCDD as it occurs in pesticides and which photodegrades fairly rapidly in the air and on leaves.

The Dow report has not convinced Michigan's Department of Natural Resources (DNR), for whom it was prepared, that the dioxins in Tittabawassee River fish did not come from discharges from Dow's manufacturing plant. Particularly worrisome are the data showing that dioxin levels around the Dow plant and in Midland are higher than the sample levels found anywhere else, says Dennis L. Swanson of DNR's Office of Toxic Materials Control.

An alternative explanation for the findings, he suggests, is that the dioxins found were made by Dow in production processes and not destroyed in the company's incinerators.

In a letter to Dow in mid-January, DNR director Howard A. Tanner outlined what he considers to be deficiencies in the Dow report and asked for further information in a number of areas. Of first importance, Swanson says, is the request for a total mass balance for dioxins and related organochlorine precursors found at the Midland plant. Swanson's other requests center on the same question: Are the dioxins found in the environment near Midland missing from the Dow plant?

To do a mass balance for all organochlorine compounds, accounting for them to the level that would be needed to see a loss of the amount that's being found in the environment near Midland, would be a tremendously expensive undertaking, says Bumb. "I'm sure we could do it," at a cost, he quickly estimates, of \$3 million to \$5 million, "but I'm not sure we would gain much more insight into what we've discovered and what is being confirmed by other scientists."

As for the fact that the highest dioxin levels are found near the Dow plant, Bumb sees two reasons for this finding. The first is that Dow has more experience

were always there. And, again, we saw an unusual distribution of TCDD, hexa, hepta, and octa forms."

At about this point, the Dow group became aware of European investigators who were drawing similar conclusions based on investigations of emissions from municipal incinerators and industrial heating facilities. K. Olie, P. I. Vermeulen, and O. Hutzinger of the University of Amsterdam, for example, found organochlorine compounds in the fly ash of municipal incinerators located in Amsterdam and two other Dutch cities. Gas chromatography-mass spectroscopy analysis (GC-MS) detected only hexa, hepta, and octachloro isomers in the Amsterdam incinerator ash, but these three types of dioxins as well as penta and tetrachloro isomers were detected in the fly ash from Arnhem and Alkaar.

The data are not qualitative, but the researchers conclude that municipal incinerators may be a source of some of the organochlorine compounds in the environment. "The amount of chlorodibenzodioxins and chlorodibenzofurans entering the atmosphere via flue gases is probably quite small," they suggest. "However, because of the extreme toxicity of some of the components and the fact that most incineration plants are located in densely populated areas, extensive monitoring of such facilities might be advisable."

Another group of European researchers who have made similar findings includes Dr. Hans Rudolf Buser and Dr. Hans-Paul Bosshardt of the Swiss Federal Research Station and Dr. Christopher Rappe

of the University of Umea, Sweden. These researchers have done quantitative studies of the polychlorinated dibenzo-*p*-dioxins and dibenzofurans they have isolated from the fly ash of a municipal incinerator and an industrial heating facility, both in Switzerland.

GC-MS analysis of these fly ashes gives an almost identical pattern for the dioxins formed at each of these combustion sources, although the municipal incinerator has higher concentrations of the more highly chlorinated dioxins. The pattern is the same as that found when a mixture of various chlorophenols is burned under similar conditions. However, the municipal incinerator burned mainly household and some industrial refuse, not chemical wastes, and the industrial heater burned used industrial oils.

Dow, too, has been investigating the ratios of one form of dioxin to another in its environmental samples. It has found that these samples do not match the ratios that would be expected if their source were the contaminants found in either trichlorophenol or pentachlorophenol. Moreover, dioxins can be reduced stepwise from the octachloro compound all the way to the free dioxin. These compounds appear to exist in equilibrium with one another, and the Dow researchers have calculated the equilibrium proportions of each compound.

Although they can establish the equilibrium ratio under experimental conditions, Dow researchers say that predicting what levels will be found in the environment is a much more difficult matter. They suggest that samples taken directly

insampling in Midland and knows how to select the place where dioxins are most likely to be. The other is that the Dow plant is a very large combustion source, comparable to a city of about 820,000 people. Thus it can be expected to be a source of dioxins.

In addition to asking for more information from Dow, DNR is setting up a protocol for its own environmental survey to see if dioxins are indeed ubiquitous in Michigan. The state has no facilities for analyzing dioxins at the parts-per-trillion level, so analysis of samples will have to be done by a contractor, at a cost of about \$800 per sample.

Michigan's Department of Public Health also has evaluated the Dow report. This department is interested in the issue because edible fish species are found in the Tittabawassee River. Thus, dioxins in the river pose a potential health hazard. The point is somewhat moot, however, because the fish also contain unacceptable levels of polychlorinated biphenyls, leading the department to issue warnings not to eat the fish.

The health department's conclusions about the Dow report are different from those of DNR. A staff memorandum forwarded to Dow by department director Maurice S. Reizen states the department's agreement with Dow that dioxins are ubiquitous and are produced by a variety of combustion sources. It recommends further research on dioxin-containing particulates. That research, it says, should include studies of their dispersion in the environment and an evaluation of their removal by waste-treatment processes.

Dow itself has asked for a panel of outside chemists to review the analytical technique used in its study. The panel is headed by Dr. Lockhart B. Rogers, an analytical chemist at the University of Georgia. Rogers says the panel will try to determine the probable validity of the data, but will not try to arrive at any environmental conclusions. He hopes the review can begin this month.

Another chemist, Dr. Michael L. Gross of the University of Nebraska, remarks: "I have difficulty with the statement that dioxins are ubiquitous." Gross has analyzed many environmental samples for

2,3,7,8-TCDD for EPA. TCDD's are not always present in the samples he has analyzed, down to levels of 5 ppt, he says. He was not looking for other dioxins.

"What I'm saying is that the conclusion may be awfully premature," he adds. He questions, among other things, where the chlorine is coming from to make chlorinated dioxins at combustion sites. The compounds probably would be thermodynamically favored molecules under fire conditions, he says, but he thinks formation of polychlorinated species might require more chlorine than is typically present in fuels.

"I think that Dow is probably right that you can form dioxins in fires, but I'm sure some fires are worse than others," he says.

Says Dr. Thomas O. Tiernan of Wright State University, Dayton: "I think that Dow's conclusions are all possible, but it will take a great deal more work to prove them." Tiernan, too, has been involved extensively in EPA's environmental monitoring program for dioxins. He sees no difficulty in finding enough chlorine in the environment to supply dioxin-forming reactions. Chlorine is widely used in water treatment, he says. In addition, he has found polychlorinated biphenyls in "everything from Arctic ice to toenails," and these compounds could serve as a source of chlorine for dioxin formation.

Dr. Alvin L. Young of the Air Force's Occupational & Environmental Health Laboratory in Texas says, "I was very skeptical when I first saw the Dow report." The European work that reaches similar conclusions has been changing his mind, however. Young has been involved for the past decade with the Air Force's extensive environmental monitoring program for 2,3,7,8-TCDD.

He says recent work by Dr. B. Jansson and Dr. G. Sundstrom of the National Swedish Environment Protection Board finds TCDD at levels ranging from 10 to 140 ppm in the combustion products of chlorinated phenols. These levels can be reduced to less than 0.01 to 0.02 ppm when the combustion occurs in the presence of copper salts. The TCDD is not 2,3,7,8-TCDD, but the much less toxic 1,3,6,8 isomer, he says.

"I would now say the Dow work is



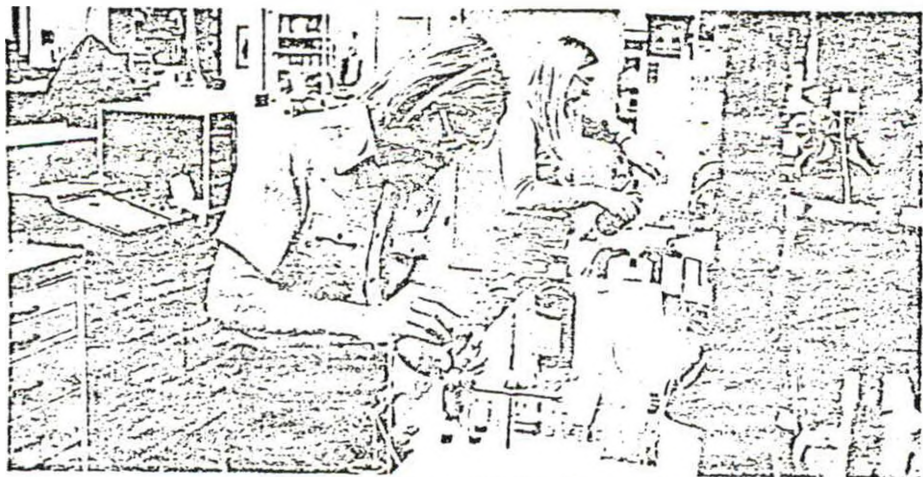
Swanson: total mass balance for dioxins

probably correct," Young says. "I'm now wondering if our dioxin monitoring program is pointing in the right direction. Perhaps we should be looking for all the dioxins in the environment instead of just 2,3,7,8-TCDD. Are the others there in concentrations high enough to be a hazard?"

According to Dr. Matthew S. Meselson of Harvard University, "Dow is making more of this interesting information than is probably justified." It may be true that combustion is putting a wide variety of dioxins into the environment, he says, but it is important from the health viewpoint to distinguish among the various dioxins. By far the most toxic is 2,3,7,8-TCDD, and "when 2,3,7,8-TCDD has been found in the environment, it has been found, to my knowledge, only near chlorinated phenols," he says. It may be that overall, combustion sources are introducing more dioxins into the environment than any other source, he says, but the "hot spots" of 2,3,7,8-TCDD contamination are always associated with chlorinated phenoxy compounds.

If the data exist anywhere to refute or substantiate Dow's claim that combustion sources are emitting measurable amounts of dioxins into the environment, those data are at EPA. The agency has sponsored many environmental monitoring programs for dioxins. EPA's programs were not set up to look at combustion sites as possible sources of dioxins, and in many cases the monitoring was done only for 2,3,7,8-TCDD and not the other dioxins that Dow finds more prevalent. However, the data, taken together, may shed some light on Dow's hypothesis.

EPA so far has not commented on the Dow findings. It is currently preparing an evaluation of the Dow report, expected to be released in the next few weeks. Among the sources of input for this evaluation are reviews of the report that have been prepared by all of EPA's regional offices that have had some experience with dioxins. Several of EPA's advisory committees with expertise in this matter also have been asked for comment. □



Dioxin analyses being performed in Dow's analytical laboratory

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