ToxCat SPECIAL

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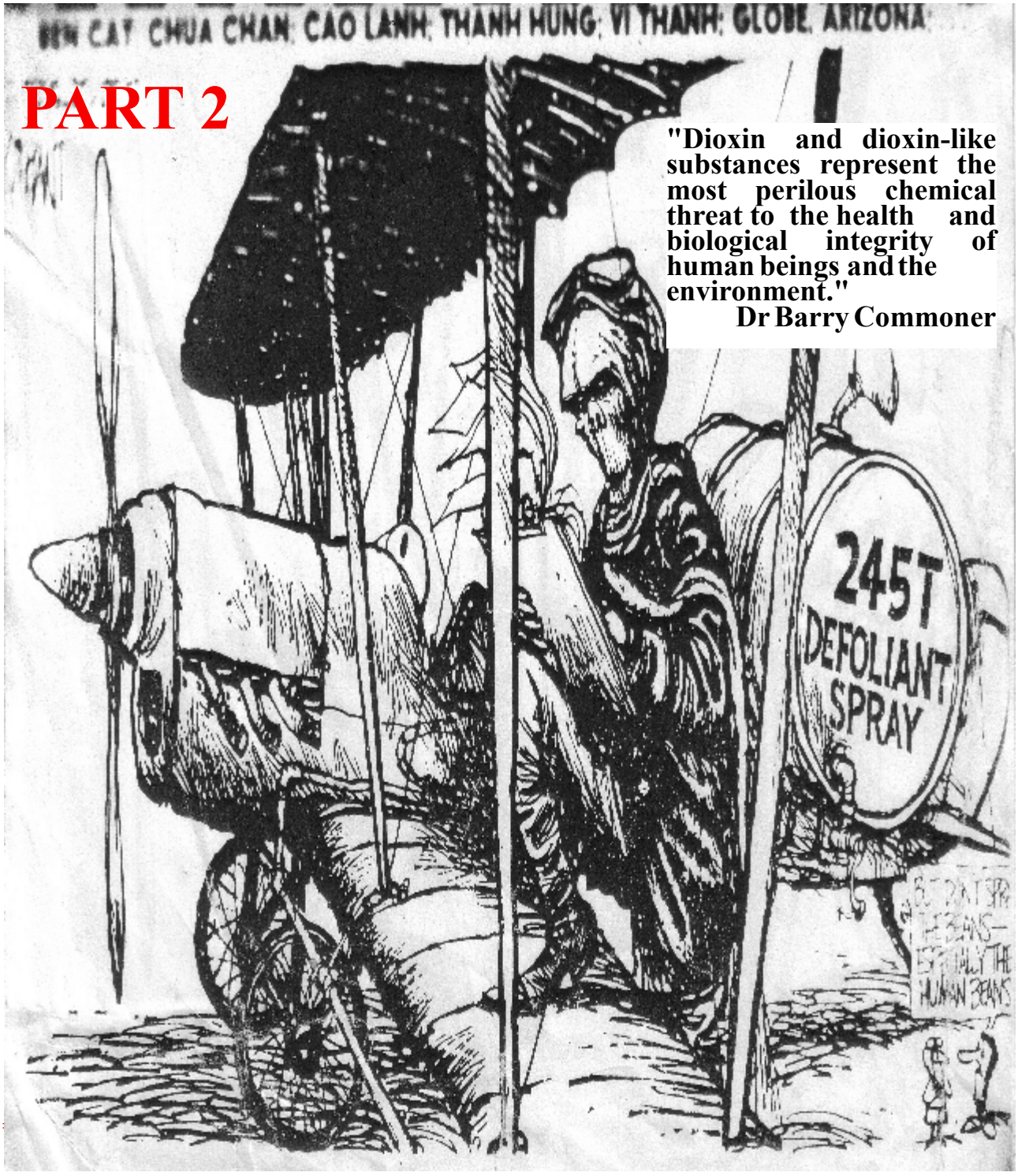
A Beginners Guide to: *DIOXIN*

PART 2



"Dioxin and dioxin-like substances represent the most perilous chemical threat to the health and biological integrity of human beings and the environment."

Dr Barry Commoner

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Communities Against Toxics (CATs) is a network of long suffering citizens and communities in Great Britain and Ireland living with incinerators, waste treatment plants, toxic waste landfills, chemical installations and other unsafe, polluting industrial facilities.
Founded in 1990, CATs operates as a non-profit making, non-party political organisation dedicated to increasing public and political awareness on environmental issues and whenever possible strengthening democracy at a local level.
To help communities protect the environment from industrial pollution and political apathy, CATs endeavours to provide information and expertise at reasonable cost and whenever possible free of charge to members of the poorer sections of society and groups in country's with transitional economies.
CATs survives on membership subscriptions and donations from sympathetic Foundations and receives no financial support from government sources or industry. CATs members newsletter ***ToxCat*** is published every two months.
Other publications available to members and subscribers include:

ToxCat 'Beginners Guide' to *Incinerator Emissions & their known impact on human health.*
ToxCat 'Beginners Guide' to *Epidemiological Studies Around Incinerators*
ToxCat 'Beginners Guide' to *Endocrine Disrupters*
ToxCat 'Do You Want a Boy or a Girl?

In the pipeline:
ToxCat 'Living with Incinerators' - Community Case Studies

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Front cover cartoon taken from Billee Shoecraft's *'Sue the Bastards'* artist unknown

Mylece

By
Carol van Strum

In the rainforests of coastal Oregon, berry vines and alder trees spring up almost overnight on untended clearing. Dense jungle quickly swallows abandoned homesteads and orchards, where only daffodils and the occasional apple tree remain amid the ferns and saplings, blooming tributes to years of human toil. Vast thickets of brush carpet the scarred earth of clearcuts and old logging roads.

By the 1970s the dioxin-tainted herbicides 2,4,5-T and 2,4-D had become indispensable tools for replacing such "unwanted vegetation" with plantations of Douglas fir seedlings.

The U.S. Environmental Protection Agency's (EPA), 1979 ban of herbicide 2,4,5-T sent shock waves through the lumber and chemical industries, which predicted the loss of 20,000 timber jobs and blamed marijuana growers for the ban. In heavily sprayed Lincoln County which had comprised most of the Alsea Study area, a county commissioner vehemently denounced the 2,4,5-T ban on local radio programs that the ban was prompted by marijuana growers to protect their illegal crops. Echoing earlier Dow Chemical Company statements, the commissioner proclaimed that any health problems attributed to herbicides were actually caused by smoking marijuana.

Driving along the coast with her two small children, Melyce Connelly heard the commissioner's radio broadcast. His words rankled the forty-mile drive to a home and sanctuary that no longer promised safety.

A single mother at age 22, Melyce clung doggedly to the log house she and her ex-husband had built themselves, determined to wrest a living from her few cleared acres along the river. With help from neighbours, she ploughed land, drove truckloads of manure and coaxed a small paradise out of forest soil. Her garlic field paid the mortgage, and beds of herbs, sweet basil; lemon thyme, rosemary, dill, sage parsley, shallots, sold fresh to coastal restaurants supplemented her winter income from teaching exercise classes.

For herself she grew flowers, and from March to November the log house basked in a sea of holly hocks, roses, lilies cosmos, daisies, narcissus columbine dahlias, and daffodils. Her business card was a photo of herself, laughing under a cascade of flowers on her porch, with a giant hibiscus blossom in her hair.

Shortly before the commissioner's radio broadcast, Melyce learned for the first time that the EPA had found dioxin in a neighbours water supply directly upstream from her home. The neighbour had lost two babies through miscarriages and other child with birth defects. As Melyce said, "You can't help wonder if there's connection."

After the 2,4,5-T ban, the Forest Service announced it would substitute 2,4-D in its spray plans for that year, which included the headwaters of Ryan Creek, the watershed for Melyce's farm. She and other neighbours met with district ranger who had them mark their water on his map and promised those areas would not be sprayed.

Three days later however, Melyce woke to the sound of a helicopter spraying Ryan Creek. Within the next few days, all her young chicks and ducklings died and her six-month-old son developed persistent, bloody diarrhoea. In the surrounding valley over the next month, every pregnant woman in her first trimester miscarried, and several children were hospitalised with near-fatal cases of spinal meningitis. Melyce carefully preserved the chicks and ducklings that had died, putting them in her freezer in hope that she could get them analysed some day.

Alarmed by these events the Lincoln County Health Department initiated a study of health problems following the spraying in the valley. The EPA had taken over the county's effort wader the auspices of its Alsea Study. Publicity about the study had prompted the commissioner's remarks about marijuana growers.

Still fuming Melyce took from her freezer some of the frozen bodies of her chicks and ducklings, and drove over 50 miles to the county offices in Newport. Carrying her infant son and the bag of frozen poultry, she marched unannounced into the commissioner's office and thumped the bag on his desk. .

"Open it," she commanded. As the startled commissioner peeled tin foil from the small, frozen bodies, Melyce placed her son on his desk as well and took off his diaper.

"Now, sir," she said, "you tell me those ducklings died from smoking too much marijuana. You tell me those chicks died from smoking too much marijuana." Fighting back tears, her voice shaking, she thrust a bloody, soiled diaper at him. "You tell me this child has bloody shits day after day from smoking too much marijuana. Tell me to my face, Mr. Commissioner!"

The next day, the commissioner went on the air again with a public apology. Information had been brought to his attention, he said, that convinced him of grave health risks from herbicide exposure. For the rest of his time in office, Commissioner Andy Zedwick led a tireless campaign against the aerial spraying of herbicides in Lincoln County, joining the county medical society in sponsoring ballot measures to restrict such uses.

When the EPA took over the county's health study of her valley, Melyce accompanied researchers on their sample collection efforts, and gave them the bodies of her chicks and ducklings for dioxin and herbicide analyses. Promised results of the study within 90 days, Melyce hounded the agency for four years, only to be told finally that many of the samples, including her birds, had never

been analysed, and that results of others were inexplicably "mixed up" with Dow Chemical samples from Midland, Michigan.

In 1984, EPA researchers returned to the valley to resample a single site, the water supply of Melyce's neighbour, where dioxin had been found in 1979. In the five years since 2,4,5-T was banned, dioxin levels had increased four-fold in sedimentsupstream from Melyce's home. Despite the increase, to thehighest dioxin levels in stream sediments ever reported in the Pacific Northwest; the EPA made no effort to collect further samples in the valley, and announced that the levels foundpresented no "immediate" health risk.

On July 4, 1989, ten years after Ryan Creek was sprayed with 2,4-D, Melyce Connelly died at age 32 of brain, lung, and breast cancer. Friends and neighbours gathered in Melyce's gardens for the last time to spread her ashes among the flowers and trees she loved. Shortly thereafter, the new owners of the property bull-dozed the gardens and garlic fields, and the house she had built burned to ground a few weeks later in an accidental fire. Berry vines and alder saplings now thrive in the clearing where her house and gardens once stood, the old pathways emerging ghost-like every spring in rows of bobbing daffodils.

Not until 1993, thirteen years after requiring manufacturers to test 2,4-D products for dioxin, did EPA admit that 2,4-D-which had been sprayed over Ryan Creek after the 2,4,5-T ban, was also contaminated with the most toxic form of dioxin, 2,3,7,8-TCDD. Yet according to Dr. Anthony Colluci, a former EPA official, the EPA had known of TCDD in 2,4-D by the early 1970s.

The use of 2,4-D in forestry and on residential lawns, roadsides, golf courses, and school grounds continues to this day, (1996) with EPA approval.

Melyce, by Carol Van Strum was taken from;
***Dioxin:* The Orange Resource Book *Synthesis/Regeneration* 1995**

A Secret Risk Assessment and a Leaked Memo

The citizens of all industrialised countries are assured by politicians the regulatory bodies in place will make sure industry adhere to the 'strict' regulations in place to protect public health. However, there are several examples where the regulators actually concealed data that could cause problems for industry should it released into the public domain.

The United State Environmental Protection Agency position throughout the 1980's was that incineration was safe, despite the fact that every incinerator is known to produce dioxin. To prove the safety of these facilities EPA 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 cal-

In the case of dioxin, over the years EPA's "standard" risk assessment had assumed that airborne dioxin only entered humans through their lungs. Dioxin that falls to the ground and then incorporated into the food chain and consequently eaten by human and animals had always been ignored in EPA's risk assessment.

However, the EPA's team of scientists conducting the official "reassessment" of dioxin's toxicity published a report in the summer of 1992 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.[1]

It was well known that dioxin accumulate in the food chain, and that meat, milk and fish are the major sources of dioxin exposure for humans. [1]

When Greenpeace researcher Joe Thornton did his own risk assessment on the Waste Technologies Incinerator situated on the banks of the Ohio River in East Liverpool, Ohio, using the technique recommended in the EPA's draft report, including dioxins in beef and milk, he found that WTI posed risks 10,000 times higher that EPA had calculated. To counter the findings of Thornton, EPA did its own food-chain risk assessment, which was not released to the public, but which came to light in court.[2]

The EPA's secret risk assessment concludes that dioxin from WTI is

1,000 times more dangerous than the 'official' published EPA risk assessment said it was.

Leaving aside the serious ethical issue of EPA refusing to publish important health and safety information about the WTI incinerator, an internal memo from Richard Guimond, acting chief of EPA's Office of solid Waste Emergency Response, dated January 22, 1993 was leaked to Greenpeace. It stated "There are very serious implications associated with adopting risk assessment procedures based on indirect exposure routes for air emission sources."[3]

Translation: if food-chain exposures are now to be counted in incinerator risk assessments, may incinerators will be found to be unacceptably dangerous.

Source: *Rachel's* Hazardous Waste News #325 February 17, 1993

[1] U.S. Environment Protection Agency, *Estimating Exposure t Dioxin-like compounds* [EPA/600/6-88/005B] Workshop Review Draft. (Washington, D.: U.S. Environmental Protection Agency, August, 1992).

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

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

Fraudulent Manipulation of Studies - A Few Facts

It has been proven that some sections of the chemical industry and government officials have shamefully and deliberately used 'selected' data and fraudulent, industry-sponsored studies to down grade the impact of dioxins on human health.

Yet even today, despite the amount of times politicians and high ranking officials, academics and government scientists have been caught 'fiddling the books' and manipulating data, some people still believe they are beyond deceit, pillars of virtue, God-like figures whose word should never be questioned. Anyone who dares question what they say should be treated with utter contempt.

Yet one of the most eminent scientists of the past 100 years, Sir Richard Doll, was receiving large amounts of money from Monsanto for something like 15 years. If his honesty and honour was truly beyond question why was this not made public before his death?

Everyone accepts that politicians and lawyers lie. But there have always been unscrupulous people in all walks of life, thieves, liars, even murderers are to be found among the best educated as well as on the streets of the poorest neighbourhoods. Not all villains walk around with hoods over their heads, the biggest ones often wear smart, pin-stripped

We once discussed the idea of setting up a web site with a manikin type figure that opened its mouth and when money was inserted said things like "no one ever died from dioxin" and "dioxin is breathed in and out."

Unfortunately it seems there is an ever-increasing number of academics and scientists only too willing to become 'coin-operated.' They will cloud data to obtain money, whether it is in the form of a brown envelope or funding for a University project.

The fraudulent manipulation of testing data has occurred on numerous occasions resulting in some of the guilty companies being prosecuted. Professor Samuel Epstein wrote:

"The overwhelming bulk of all benefit and risk data, on which regulatory decisions are based, comes from the industries themselves being regulated. These data are either generated and interpreted by in-house scientific staff or by commercial laboratories and universities under contract to industry.[1]

Prof Epstein cited a number of examples of industry explaining away carcinogenesis and manipulating data to suit their needs.

Dow claimed in 1971 that the herbicide 24-D was tested on rats and found to be non-teratogenic, although tabular data indicated the production of a wide range of congenital defects. But since the affected progeny were shown to be capable of surviving in early infancy, Dow decided that the birth defects were of no particular consequence and should be ignored. To bolster this position, Dow redefined the standard term teratology, as congenital defects inconsistent with survival or optimal function. Under this definition Thalidomide-type defects and most congenital heart defects would be excluded.

Industrial Biotest Lab, Northbrook, Illinois faced with a federal investigation in April 1977 for fraud and submission of questionable test data, destroyed files dealing with toxicological and carcinogenicity testing of thousands of federally approved drugs, pesticides, food additives and industry chemicals. The President of the company AJ. Frisque, has admitted that he ordered the shredding of laboratory documents, but claimed this was because of a "misunderstanding."

Allied Chemicals suppressed data for about 10 years on the carcinogenicity and the toxic effects on reproductive and central nervous system of the organic pesticide kepone. As a consequence workers exposed to very high levels while working in grossly deficient working

conditions developed crippling neurological and other diseases.

Dr. Epstein feature resulted in a barrage of letters from industry and apologist. James D. Wilson of Monsanto Industrial Chemicals Co, St. Louis, Missouri wrote:

"Among the people employed [at Monsanto] are scientists who stand with the best in academia or government labs. They will not compromise their personal ethics not endanger their scientific standing to participate in deception. They set the standards the rest of us live by. Ultimately our survival is tied to our creditability - in the trust customers and the public place in our word.

Nevertheless, sometimes misguided people will shade results, or falsify it, to give results they believe their bosses or customers want. And sometimes scientists make honest errors of act or judgement. Monsanto guards against this by appropriate review procedures, good laboratory practices codes and the like and by employing god scientists, proud of their scientific credentials. They know that their work will be judged by their peers in the scientific community, and they act accordingly."

Fraudulent studies are not a thing of the past, on February 18 (2008) it was reported in *Chemical & Engineering News* that a chemist had submitted fraudulent research documents on metals including lead, nickel, copper, manganese, arsenic, palladium, cobalt, thallium, and selenium. He also published research on measuring carbosulfan, deltamethrin, bediocarb, pyrethroids, and quinalphos.

In all it is believed he plagiarized and/or falsified more than 70 research papers that were published in a wide variety of Western scientific journals between 2004 and 2007.[2]

Some journal editors say it is one of the most spectacular and outrageous cases of scientific fraud they have ever seen.

Like any case of scientific fraud, it raises the question "what is inciting people to do this even though it is deeply wrong?"

"Partly we have to blame our own selves," says Purnendu K. (Sandy) Dasgupta, a chemistry professor at the University of Texas, Arlington, and U.S. editor of *Analytica Chimica Acta.*

Citing the enormous pressure on scientists everywhere to publish and win grants. Dasgupta says editors and reviewers are overwhelmed and reliant on the honour system at the heart of scientific publishing. "Plagiarism can be guarded against," he says, "but out-and-out fraud is hard to guard against."

One well known case of a honourable, 'upper class' stealing from the poor was the scandal in Britain of Westminster council leader Dame Shirley Porter selling public housing for votes (at a loss of £27 million to the council - which of course means the ratepayer.)

In many countries including France, Germany and the UK bribes were treated as legitimate business expenses which could be claimed for tax deduction purposes.

UK multinationals routinely pay commissions to gain contracts from other governments -- We know at least one UK government minister has assisted them in this process. Jonathan Aitken, a former Minister for Defence Procurement, was jailed in June 1999 because he lied in court about his visits to France and Switzerland in 1993 to attend a secret meeting to negotiate contracts for an arms deal.[3]

* Monsanto admitted bribing a representative of the Indian government in relation to GM crops.

* In Grenoble a former mayor and government minister, together with a senior executive of the private water company Lyonnaise des Eaux (now Suez-Lyonnaise), received prison sentences in 1996 for receiving and giving bribes to award the city's water contract to a Lyonnaise subsidiary. In Angoulème, a former mayor and one-time minister was jailed for two years for taking bribes companies bidding in public tenders, including Générale des Eaux (now Vivendi).20 Executives of Générale des Eaux were also convicted of bribing the mayor of St-Denis (Ile de Réunion) to obtain the town's water concession.

The involvement of these companies in the spread of incineration throughout Europe leads one to question the unhealthy eagerness of the EU Commissioners and many UK/EU politicians to assist them to build these incinerators and the fact that the European Investment Bank is supplying the necessary finance in many cases.

Then of course we have governments and their departments keeping data that might damage the interests of industry secret:

* The British government kept secret the fact that BSE could 'jump' species for something like 10 years, while an unsuspecting nation consumed meat of dubious quality. BSE has resulted in the deaths of 165 people (as of June 2007) with many more expected due to the long incubation period of the prion.

* The UK's Environment Agency (EA) kept secret data showing a municipal solid waste incinerator in Winchester was emitting huge amounts of dioxin for 4 years.

It is not only by deliberately manipulating and falsifying data that scientists can protect industrial interests. They can also ignore certain chemicals and omit important studies/findings.

* Britain's EA and Food Standard Agency (FSA) failed to included PCBs in their study on the impact of incinerator ash contaminated with heavy metals and dioxin spread on food producing allotments, footpaths, playing fields and flower beds in and around Newcastle upon Tyne, England for approx 6 years. The EA claimed the PCBs would have been destroyed in the incinerator. However, temperature as high as 1300 degrees are necessary for the destruction of PCBs and most mu-

nicipal waste incinerators burn at 800 to 950 degrees.

* They also failed to include children under 10 years of age, the most susceptible section to chemical impact, in their health impact studies. They then declared there was "no adverse health effects," despite dioxin levels of 4224 ng/kg being found in ash that had been in the open environment for approximately four to six years.

* When conducting studies on the body burden of dioxin the French scientists of Afssa failed to included dioxin-like PCBs in their calculations. They also failed to consider the intake during the first two years of a child's life, the period intake is highest resulting in a seriously flawed study keeping the truth from the French nation.

Are the scientists who deliberately omit children and dioxin-like PCBs from health studies, or fail to mention epidemiological studies containing data that would be damaging to industries interest, any less contemptible than those who deliberately manipulate and falsify figures. I think not!

References:
[1] 'Polluted Data' (The *Ecologist* Vol. 9 Nos 8/9 Nov/Dec 1979).
[2] *Chemical & Engineering News:* Sci- ence & Technology February 18, 2008 Volume 86, Number 07 pp. 37-38
[3] Corner House Briefing 19 Exporting Corruption, Privatisation, Multinationals and Bribery, by Dr. Susan Haw- ley. June 2000

Monsanto's Fraudulent Studies

The following is the memo sent by Cate Jenkins Ph.d with reference to some studies conducted by Monsanto.
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY WASHINGTON D.C. 20460
OFFICE OF SOLID WASTE AND EMERGENCY RESPONSE....
MEMORANDUM
DATE: November 15, 1990.
SUBJECT: Criminal Investigation of Monsanto Corporation - Cover-up of Dioxin Contamination in Products - Falsification of Dioxin Health Studies.

FROM: Cate Jenkins, Ph.D., Chemist Regulatory Development Branch (OS 332) Characterization and Assessment Division,
TO: John West, Special Agent in Charge Office of Criminal investigations Center U.S. Environmental Protection Agency Building 53, Box 25227 (303) 236-5100 Kevin Guarino, Special Agent Office of Criminal Investigations National Enforcement Investigations Center, EPA
As per our meeting yesterday, I am summarizing information available to me supporting allegations of a long pattern of fraud by Monsanto Corporation. The fraud concerns 2,3,7,8-tetrachlorodibenzodioxin (dioxin) contamination of Monsanto's dioxin-exposed workers. You indicated that you would contact me regarding the specific documents which would be useful to your investigation.

SIGNIFICANCE OF MONSANTO'S DIOXIN FRAUD

You stated that pursuing a criminal prosecution against Monsanto would require a prior determination of the significance of the fraud. In order for proceedings to be initiated by EPA, the fraud would need to have affected the regulatory process at EPA and Monsanto to have knowingly submitted the falsified data and health studies to EPA in order to affect the regulatory process. Monsanto has in fact submitted false information to EPA which directly resulted in weakened regulations under RCRA and FIFRA since these regulations do not take into account tetrachlorinated dioxin contamination in trig, tetra, and pentachlorophenols, as well as 2,4-dichlorophenol and its phenoxy acetate (2,3-D, a currently used herbicide). In addition, Monsanto's failure to report dioxin contamination of the disinfectant in Lysol has prevented any ban or other alleviation of human exposures to dioxins in this product.

The Monsanto human health studies have been submitted to EPA by Monsanto as part of public comments on proposed dioxin rules and Agency-wide dioxin health studies are continually relied upon by all offices of EPA to conclude that dioxins have not caused cancer or other health effects (other than chloracne) in humans. Thus, dioxin has been given a lesser carcinogenic potential ranking, which continues to be the basis of less stringent regulations and lesser degrees of environmental controls. The Monsanto studies in question also have been a key basis for denying compensation to Vietnam Veterans exposed to Agent Orange and their children suffering birth defects from such parental exposures.[1]

Monsanto would not be able to support a claim that independent researchers were responsible for the falsifications, because Monsanto personnel compiled all data utilised by these researchers. In addition the National Institute of Environmental Health Sciences partially funded one of the Monsanto studies in question providing a basis for charges of the fraudulent use of governmental funds.[2]

DIOXIN CONTAMINATION OF MONSANTO PRODUCTS

Monsanto covered-up the dioxin contamination of a wide range of its products. Monsanto either failed to

report contamination, substituted false information purporting to show no contamination or submitted samples to the government for analysis which had been specially prepared so that dioxin contamination did not exist.

The earliest known effort by Monsanto to cover-up dioxin contamination of its products involved the herbicide used in Vietnam Agent Orange (2,4,5- trichlorophenoxy acetate, 2,4,5-T). Available internal Monsanto correspondence in the 1960s shows a knowledge of this contamination and the fact that the dioxin contaminant was responsible for kidney and liver damage, as well as the skin condition chloracne."

Early internal Monsanto documents reveal that samples of 2,4,5-T and other chlorinated herbicides and chlorophenols submitted to the U.S. Department of Agriculture in the 1970s were "doctored." In other words, highly contaminated samples were not submitted to the government, and Monsanto samples of penta tetra-, tetra-tri-, dichlorophenol, and associated herbicides never contained tetrachlorinated dioxins. These analyses were subsequently adopted by EPA in a 1980 publication and were used without any data from other sources as the basis for 1984 regulations under RCRA. As a result, these regulations do not control the chlorophenol phenoxy acetate products as acutely hazardous due to their contamination of tetrachlorinated dioxins.

Monsanto also submitted assertions to EPA that process chemistry would preclude the formation of tetrachlorophenol or its phenoxy acetate.

Evidence from the Kemner v Monsanto proceedings revealed that this process chemistry claimed by Monsanto was not always used. In fact, off-specification dichlorophenol known to be contaminated with tetrachlorinated dioxin, was being used as a feedstock to make pentachlorophenol and other chlorinated products. The result of this alternate synthesis route is the introduction of dioxins as a contaminants.

EPA also relied on these "process chemistry" arguments by Monsanto as a basis for not regulating most chlorophenols and 2,4-D for their tetrachlorinated dioxin content

Another Monsanto document introduced as evidence the above proceedings shows cross-contamination of range of Monsanto products with tetrachlorinated dioxin by the following mechanism: The same production equipment is used without cleaning for all chlorinated phenolic products

In 1984, when promulgating the dioxin regulations under RCRA, EPA was only made aware of the cross contamination problem in the event that 2,4-D was made on equipment previously used to make 2,4,5-T. Thus, EPA again was subverted from promulgating adequate regulations for products other than 2,4-D that were cross-contaminated with dioxins.

Members of the Canadian Parliament recently directed investigations by the Royal Canadian Mounted Police and government scientist into the dioxin contamination of disinfectants such as Lysol containing Monsanto's Santophen (ortho-dichloro-para-phenol), and directed laboratory, analyses of existing stocks. This disinfectant uses the ortho-dichlorophenol, discussed above, as a feedstock would introduce any dioxins present into the disinfectant a 1984 letter to the Canadian government, Monsanto asserted that their disinfectant contained no dioxin. This was later refuted by testimony by Monsanto's chemist.

FRAUDULENT DIOXIN HEALTH STUDIES

As you indicated today, demonstrating criminal fraud in the epidemiological studies performed by Monsanto on its dioxin-exposed workers would necessitate bringing in appropriate groups in EPA capable of performing scientific study audits[3] You indicated, however, that NEIC did no, believe this would be a barrier to the investigation. The following are a few key instances where obvious fraud utilised in the conduct of these studies:

Dr. Raymond Suskind at the University of Cincinnati hired by Monsanto to study the workers at Monsanto 's Nitro, West Virginia plant. Dr. Suskind stated in published studies in question that chloracne, a skin condition was the prime indicator of high human dioxin exposures, and no other health effects would be observed in the absence of this condition. Unpublished studies by Suskind, however indicate the fallacy of this statement. No workers except those having chloracne were ever examined by Suskind or included in his study. In other words, if no workers without chloracne were ever examined for other health effects. There's no basis for asserting that chloracne was "the hallmark of min intoxication."[4] These conclusions have been repeatedly utilised by EPA, the Veterans Administration, etc., to deny any causation by dioxin of health effects of exposed citizens, if these persons did not chloracne.

The results of Dr. Suskind's studies also were diluted by the fact that the exposed group contained not only individual having chloracne (a genuine, but not the only effect of dioxin exposure), but also all workers having any type of condition such as chemical rash. The workers could have had no or negligible dioxin exposures, but they were included in the study as part of the heavily exposed group. tact was revealed only by the careful reading of the published Suskind study[5] Further, Dr. Suskind utilised statitics on the skin conditions of workers compiled by a Monsanto clerical worker, without any independent verification.[6]

Dr. Suskind also covered-up the documented neurological damage from dioxin exposures. At Workers Compensation hearings, Suskind denied that the workers experienced any neurological health effects. In the Kemner, et.al v Monsanto proceedings, however, it was revealed that Suskind had in his possession at the time examinations of the workers by Monsanto's physician,

Dr. Nestman. documenting neurological health effects. In his later published study. Dr. Suskind denied the continuing documented neurological health effects suffered by the workers, falsely stating that symptoms "had cleared."

All of the Monsanto dioxin studies also suffer another fatal flaw. The purported "dioxin unexposed" control group as selected from other workers at the same Monsanto plant. An earlier court settlement revealed not only that these supposedly unexposed workers were exposed dioxins. but also to other carcinogens. One of these carcinogens, para-amino biphenyl, was known by Monsanto to be a human carcinogen and it was also known that workers were heavily exposed.

Another Monsanto study involved independent medical examinations of surviving employees by Monsanto physicians. Several hundred former Monsanto employees were too ill to travel to participate in the study. Monsanto refused to use the attending physicians reports of the illness as part of their study, saying that it would introduce inconsistencies. Thus, any critically ill dioxin-exposed workers with cancers such as Non-Hodgkins lymphoma (associated with dioxin exposures), were conveniently excluded from the Monsanto study.

There are numerous other flaws in the Monsanto health studies. Each of these misrepresentations and falsifications always served to negate any conclusions of adverse health effects from dioxins. A careful audit of these studies by EPA's epidemiological scientists should be obtained as part of y our investigation.

The false conclusions contained in the Monsanto studies have recently been refuted by the findings of a recent study by the National Institute of Occupation Safety and Health (NIOSH). This NIOSH study, recently circulated by Dr. Marilyn Fingerhut for review, found a statistically significant increase in cancers at all sites in the Monsanto workers, when dioxin exposed workers at Monsanto and other industrial locations were examined as an aggregate group.[7]

Please do not hesitate to contact me regarding documents to support your investigation, which include testimony and evidentiary documents from the on-going *Kemner v Monsanto* litigation, earlier litigation in West Virginia brought by the Monsanto workers, ongoing investigations by the Canadian government internal Monsanto documents, as well as documentation of the submission of the fraudulent data and studies by Monsanto to support the rulemaking process under RCRA and other EPA authorities.

CC: Admiral E. Zumwalt
Senator Thomas Daschel.
Congressman Ted Weiss. American Legion.
National Vietnam Veteran's Coalition.
Oklahoma Agent Orange Foundation.
Independent International Agent Orange Network.
Vietnam Veterans of New Zealand.
Greenpeace, U.S.A.
Earth First.
Natural Resources Defense Counci.l
Environmental Defense Fund.
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Mike Petruska Chief, Regulatory Development Branch.
Carrol G. Wills, Acting Director, NEIC, EPA/Denver.

References

1) The American Medical Association, concerned about the veracity of one of the Monsanto studies published in its journal, stated that a reassessment would be undertaken if the outcome of appeal of the Kenmer v Monsanto litigation did not reverse the verdict impugning the credibility of the Monsanto studies.
2) You indicated that NEIC would be reticent to receive documents of this nature suspected to be under a court protective order, but assured me that you would pursue legal routes to obtain them independently.
3) You should be cautioned regarding any consultation with Dr. Renate Kimbrough at EPA regarding the review of the Monsanto studies. Dr. Kimbrough was contacted by Monsanto during the Kennner v. Monsanto litigation and provided expert testimony, while an employee of the Centers for Disease Control, on behalf of Monsanto. Dr. Kimbrough has provided expert testimony on behalf of other defendant corporations responsible for dioxin pollution even co-authoring papers with these defendants.
4) Suskind examined only one worker without chloracne (Mr. Kiley), and dismissed this individual's health complaints as being those of a complainer.
5) Later studies by the Centers for Disease Control have demonstrated that any manifestation of chloracne in humans is not correlated with the blood dioxin levels. [n other words, individuals with lower blood dioxin have been observed to develop chloracne, those with higher blood levels did not.
6) The deposition of Ms. Jan Young of Monsanto, previously under a protective order, is in the process of release pursuant to a motion by Greenpeace, USA.
7) This NIOSH study does have an inherent design weakness that would diminish the capability of detecting excess cancers. This is because Monsanto and the other dioxin-producing companies were allowed to independently select the group of dioxin-exposed workers to be studied by NIOSH.

In the late sixties nine babies between the ages of six and fourteen days were rushed to St. Louis, Children's Hospital. They were sweating excessively, their heart rate was increased and they had breathing difficulties. Tests revealed enlarged livers and two babies died soon after being admitted to the hospital.
An investigation to determine the cause of the illnesses revealed the expectant mothers had been lying between sheets laundered with a product containing pentachlorophenol. Despite rinsing the chemical was still present in the sheets and had penetrated the mothers' bodies, crossed the placenta barrier and infected the developing babies.

TOO MUCH SECRECY

We should be able to look to industrial scientists and regulatory officials for 'safe' solutions. However, corporations are only interested in developing technologies that reap profits, and will manipulate and falsify data to suit their interests.

The industry can divert technology research into channels that are directly contradictory to the known facts of human needs/well-being. The chemical and incineration industries for example, conduct a never-ending search for obscure and futile data attempting to cloud and lessen the impact of the ever-increasing mountain of data showing the link between chemicals, incineration, its by-products, and ill health.

'When an American federal report criticised the cigarette industry for not facing up to the health hazards or even admitting they exists, the industry's PR machine protested that the report was a 'shockingly intemperate defamation of an industry which led the way in medical re- search to seek answers in the ciga- rette controversy.'[1]

Secrecy, as exhibited by the U.S. EPA, its 'risk assessment,' and the Alsea study, has happened far too many times, with the blessing of many high ranking political figures. Industry's interests have always far out weighed society's and its well-being, and continues to do so today.

The truth is scientific objectivity depends on a process whereby the results of research are subject to scrutiny and peer reviewing by others working in the filed to enable amendments etc. Without a free flow of information this is impossible.

Once science is done in secret it is on the way to becoming non-science, for errors, which are bound to be made, and manipulated and falsified data will not be picked up.

'...[T]he closer one gets to a complete understanding of a situation the more rationally one can plan, but secrecy hinders the development and dissemination of understanding. Of course in the short term secrecy can be and is defended as a rational means to protect the narrow interests of a business enterprise against its rivals.'[1]

Professor Barry Commoner described one example where a secret U.S. Government committee was set up to estimate the dangers of atomic fallout poisoning. Their estimate (later declassified), was an underestimate by an order of magnitude. A major reason for the error was that the committee assumed that strontium 90 would enter plants only through the roots. Had a botanist been on the committee he could have informed them that many plants absorb nutrients through the leaves. Unfortunately no botanist was present, and as the committee was secrete nobody could tell them of this rather elementary (to a botanist) fact.[2]

[1] Harry Rothman, *Murderous Providence, a Study in Industrial Societies,* Rupert Hart-Davis, 1972. ISBN 0 246 10515 1

[2] *Chemical and Engineering News* 10 January 1969

Zambon *et al.*, 2007. **Sarcoma risk and dioxin emissions from incinerators and industrial plants: a population- based case-control study (Italy)**. *Environ. Health* 6:19

Abstract

Background. It is not clear whether environmental exposure to dioxin affects the general population. The aim of this research is to evaluate sarcoma risk in relation to the environmental pollution caused by dioxin emitted by waste incinerators and industrial sources of airborne dioxin. The study population lives in a part of the Province of Venice (Italy), where a population-based cancer registry (Veneto Tumour Registry – RTV) has been active since 1987. Methods Two hundred and five cases of visceral and extravisceral sarcoma, confirmed by microscopic examination, diagnosed from 01.01.1990 to 31.12.1996, were extracted from the RTV database. Diagnoses were revised using the actual pathology reports and clinical records. For each sarcoma case, three controls of the same age and sex were randomly selected from the population files of the Local Health Units (LHUs). The residential history of each subject, whether case or control, was reconstructed, address by address, from 1960 to the date of diagnosis. All waste incinerators and industrial sources of airborne dioxin in the Province of Venice were taken into account, as was one very large municipal waste incinerator outside the area but close to its boundaries. The Industrial Source Complex Model in Long Term mode, version 3 (ISCLT3), was used to assess the level of atmospheric dispersion. A specific value for exposure was calculated for each point (geo-referenced address) and for each calendar year; the exposure value for each subject is expressed as the average of specific time-weighted values. The analysis takes into account 172 cases and 405 controls, aged more than 14 years.

Results. The risk of developing a sarcoma is 3.3 times higher (95% Confidence Interval – 95% CI: 1.24 – 8.76) among subjects, both sexes, with the longest exposure period and the highest exposure level ; a significant excess of risk was also observed in women (Odds Ratio OR = 2.41, 95% CI: 1.04 – 5.59) and for cancers of the connective and other soft tissue (International Classification of Diseases, ninth Revision – ICD-IX 171), both sexes (OR = 3.27, 95% CI: 1.35 – 7.93).

Conclusion. Our study supports the association between modelled dioxin exposure and sarcoma risk.

Seveso

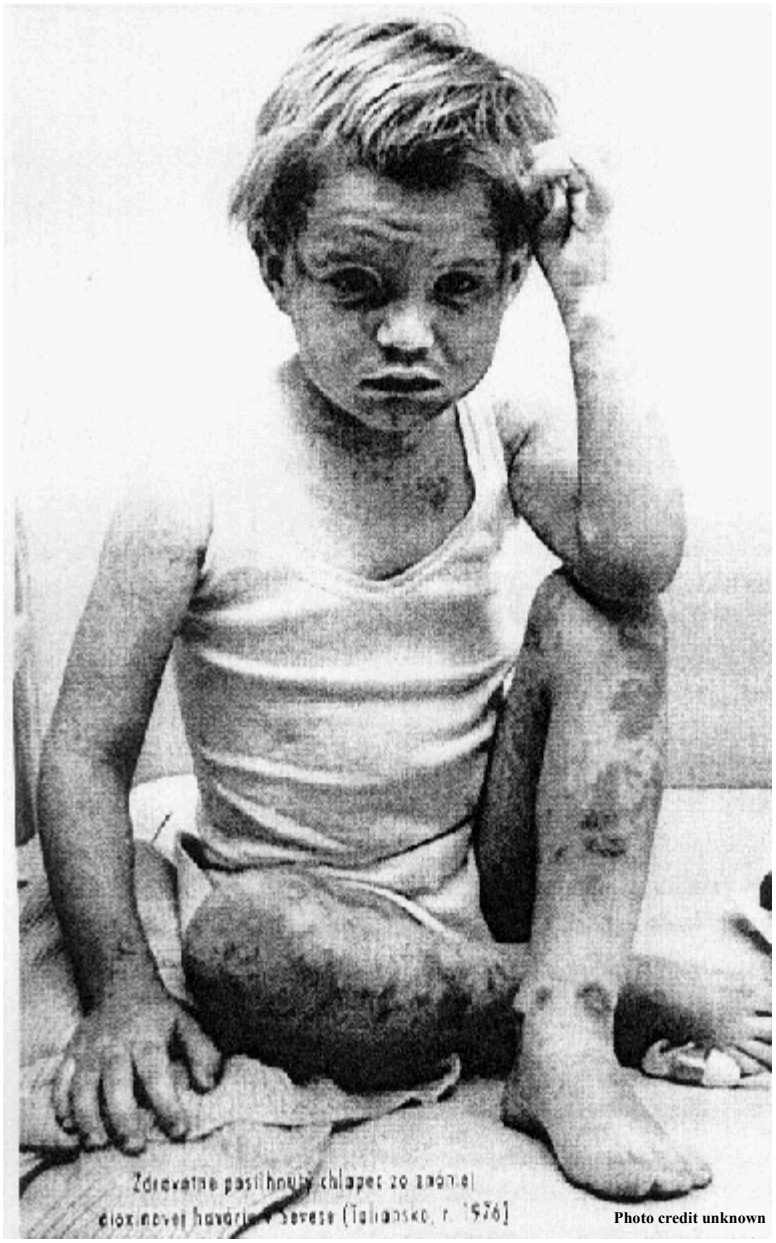


Photo credit unknown

It was on July 10th 1976 that the lives of the people of Seveso, Italy, were touched forever by dioxin. A massive explosion at the Hoffman-La Roche chemical plant caused a visible chemical cloud (officially estimated to be between 200 to 300 grams of dioxin) to rise about 50 meters and carried southeast by the wind. The toxic cloud enshrouded the municipalities of Meda, (population 19,000) Seveso, (17,000) Desio (33,000) Cesano Maerno (34,000) Barlassina (6,000) and Boviso Masciago (11,000).

In all the explosion contaminated a region with a population of around 121,000, 12 miles from Milan. Within a few hours children in the area exhibited the first sign of health problems with acute diarrhoea, vomiting, and burn-like skin lesions, appeared.

One of these was Stefania Senno who was just three years old and playing on a balcony in her family home when the cloud covered her. A few days later her face became disfigured. Stefania is now 33 and despite four operations her face still shows the ravages of dioxin.

On the Monday men climbed onto the top of the reactor to collect samples to ascertain exactly what had been released into the atmosphere. On removing the manhole cover they find a large solid grey mass. Unable to chip anything off the mass they took swabs of the chemicals thrown onto the plant by the explosion.

These samples were taken to the Dubendorf laboratories in Switzerland for analysis, but it was known the results would not be available until the Thursday.[1]

The authorities began an investigation five days after the accident, when animals such as rabbits began to die en masse.

The results of the first of the Dubendorf laboratories analysis ar- dioxin concentration at 3 parts per per thousand, a thousand times the quantity expected in crude TCP.

The same day reports were coming in of pets, chickens and rabbits dying in the area of ICMESA. Children were being taken to the local doctor with blisters on their faces. Von Zwehl was extremely reluctant to say is was dioxin had been released, insisting it was basically TCP in the cloud.

But birds were falling out of the trees, dogs and cats staggered like drunks before falling over and dying. By Friday dead birds were scattered around the streets. Cats, dogs, chickens and rabbits were dying, mothers were rushing their children to hospital. The workers had gone on strike demanding to know the details of the accident and the contents of the chemical cloud.

The Mayor, Francesco Rossi was extremely worried. The local doctor had recommended evacuating the population nearest the plant and as Mayor he was responsible for the safety and health of his community. Yet if he called for the population to be evacuated unnecessarily it would reflect badly on local industry and could possibly damage the political standing of his

He looked to the Deputy Prefect of Milan for advice. “Go ahead with the evacuation if you think it is necessary” was his advice.

Von Zwehl still kept the word ‘dioxin’ close to his chest and his refusal to reveal the results analysis of the Dubendorf resulted in him being threatened with arrest.

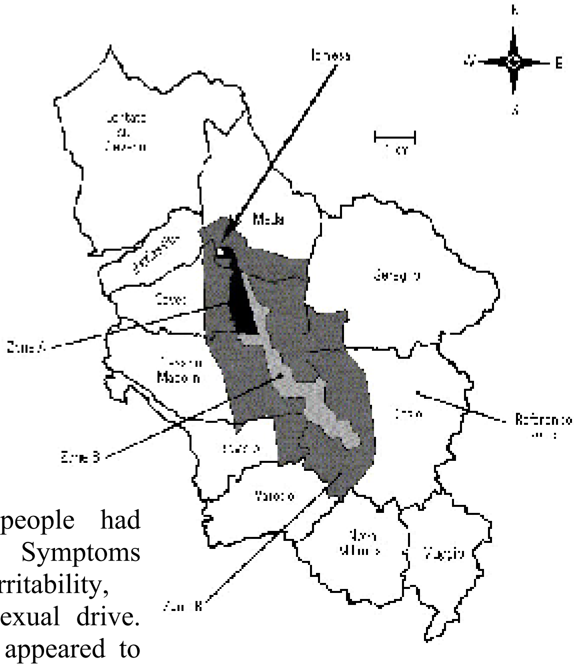
It wasn’t until 23rd July that it was announced to the public that dioxin *was* involved. Although Dr Ernesto Bergamaschini, aSeveso general practitioner who worked as factory doctor to ICMESA told a scientific meeting a year later that’ he knew about the dioxin on Thursday July 15,’ the day he had talks with Dr. Giuseppe Reggiani director of Clinical Research for Hoffmann-La Roche.

Within three weeks, some 736 people living closest to the plant were evacuated.

About 37,000 people are believed to have been exposed to the chemicals and approximately 4% of local farm animals died. Those that didn’t, roughly 80,000 animals, were killed to prevent contamination from filtering up the food chain.

Because of the publicity on the teratogenicity of dioxin, abortions were made available to the exposed women.

Studies of the situation at the ICMESA plant revealed that dioxin was probably escaping periodically from the plant over a two-year period prior to the explosion. Two and a half months after the explosion, children and young people began to develop chloracne.



A year later 130 people had confirmed chloracne. Symptoms included nervousness, irritability, loss of appetite and sexual drive. Spontaneous abortions appeared to double; the true level of birth defects could not be determined because of the abortions.

In 1977 it was discovered that 280 children in an area north of the contaminated area were suffering from chloracne.

Studies covering 1976-1986, a short period in which to find cancer occurrences, showed an increase in heart diseases and connective and soft tissue cancer. Both men and women showed an increase in rare blood and liver cancers. [2]

“Any doctor who is honest in this area will admit that the increase in miscarriages is very considerable” said Dr. Nino Rossi. “They are happening between the third and fifth month of pregnancy...” “...[I]f you go into any of the hospitals around here, Desio, Giussano, Seregno, Mariano - and ask where the women who had miscarriages came from

The general manager of the plant Herwig von Zwehl wrote an official letter to the Health Department.

BY HAND Monday 12 July 1976

For the attention of the Health Officer

Subject; incident on July 10 1976

We can confirm our discussion and the information we gave you when you visited us today:

An incident occurred in our works on Saturday10 July at about 12.40 p.m.

The plant was closed at the time fro the normal Saturday rest day.

Only maintenance and modification staff were on the premises and they were not involved in the department in question.

The reason for the incident is still being investigated.

The timing of the accident leads us to believe that an unexplained exothermic chemical reaction occurred in a reactor which had been left to cool. It had been loaded with the following substances?

Tetrachlorobenzene, ethylene glycol and caustic soda which had reacted together to form crude trichlorophenol.

When work stopped (6.00a.m. Saturday) the reactor containing the crude product has been left closed, as is customary, without agitation or heating.

We are unaware what happened from that time until 12.40 p.m. To when the safety disc ruptured and allowed a cloud of vapour to escape which, after affecting the inside of the factory, was carried by the wind towards the south-east and quickly dispersed over the area. Since we are not in a position to evaluate the substances present in the vapour or to predict their exact effects, but knowing the final product is used in manufacturing herbicides, we have advised householders in the vicin- ity not to eat garden produce.

For the moment we have suspended work in this plant, concentrating our research on explaining the causes of the accident, to avoid similar cases in the future.

Thanking you for your courteous collaboration, and with best wishes.

ICMESA

eight out of ten will be from Seveso or Meda."

In fact the pregnancy loss rate in Seveso rose from 10% to 20" in the forth quarter of 1976.[1]

Birth defects also rose in Seveso and Cesano Maderno (per 1000 birth) from 0 in 1976 to 12.7 in 1977 and 16.7 in the first half of 1978;

In Meda and Desio they rose from 1.2 to 23.0 in 1977 and 21.8 in first half of 1978;

Other seven towns; rose from 1.8 (1976) to 9.8 (1977) to 14.00 first half of 1978.[1]

Birth Defects

According to the official statitics the total number of birth defects in the 11 towns was 53 in 1978, compared with 38 the previous year and four in 1976. Dr. Alberto Colomb didn't agree with these figures claiming they were only the cases reported by doctors and hospitals. They ignored any that came to light when mothers had their post natal check-up or when they had their babies innoculated against polio.

The earlier figures also excluded defects detected when the children attend nursery school.

Dr. Colombi claimed that another 100 cases had been officially ignored despite the details being in the offical files. [1]

Dr. Colombi had already challeged official figures for 1978 and been proved right with 53 defects per thousand compared with the official figure of 19 per thousand.

Fifteen years after theaccident cancer deaths from all forms of cancer had increased with a 3 fold increase in rectal cancer and a significant increase in blood cancer in men. There was also an increase in blood cancer and a 6 fold increase in Hodgkin's disease and myeloma in women.[2][3]

"For years industry and government agencies have said there was 'no problem' although many pregnant women in the area had spontaneous abortions. We have now seen a rise in cancers among the community of Seveso and there is a real problem." said Dr. Massimo Donati, a MD living in Seveso.[4]

Later studies of children born to parents exposed during the accident found that between 1977 immediately after the accident) and 1984, substantially more females than males were born (48 to26) consistent with other evidence that dioxin modifies hormonal balance.[5]

The Seveso accident is likely the most systematically studied dioxin contamination incident in history. In the words of Dr. Paolo Mocarelli of the Hospital of Desio: "a chance experiment on human beings. Probably the strongest effect is on reproduction."

Dr, Mocarelli was put in charge of a laboratory set up two weeks after the accident to test people for health

Dioxin Exposure, from Infancy through Puberty, Produces Endocrine Disruption and Affects Human Semen Quality. Paolo Mocarelli, Pier Mario Gerthoux, Donald G. Patterson Jr., Silvano Milani, Giuseppe Limonta, Maria Bertona, Stefano Signorini, Pierluigi Tramacere, Laura Colombo, Carla Crespi, Paolo Brambilla, Cecilia Sarto, Vittorio Carreri, Eric J. Sampson, Wayman E. Turner, and Larry L. Needham

Abstract:

Background:

Environmental toxicants are allegedly involved in decreasing semen quality in recent decades; however, defini- tive proof is not yet available. In 1976 an accident exposed residents in Seveso, Italy, to 2,3,7,8-tetrachlorodiben- zo-p-dioxin (TCDD) .

Objective: The purpose of this study was to investigate reproductive hormones and sperm quality in exposed males.

Methods: We studied 135 males exposed to TCDD at three age groups, infancy/prepuberty (1–9 years) , puberty (10–17 years) , and adulthood (18–26 years) , and 184 healthy male comparisons using 1976 serum TCDD levels and semen quality and reproductive hormones from samples collected 22 years later.

Results: Relative to comparisons, 71 men (mean age at exposure, 6.2 years ; median serum TCDD, 210 ppt) at 22–31 years of age showed reductions in sperm concentration (53.6 vs.72.5 million/mL ; $p = 0.025$) ; percent progressive motility (33.2% vs. 40.8% ; $p < 0.001$) ; total motile sperm count (44.2 vs. 77.5 x 106 ; $p = 0.018$) ; estradiol (76.2 vs. 95.9 pmol/L ; $p = 0.001$) ; and an increase in follicle-stimulating hormone (FSH ; 3.58 vs. 2.98 IU/L ; $p = 0.055$) . Forty-four men (mean age at exposure, 13.2 years ; median serum TCDD, 164 ppt) at 32–39 years of age showed increased total sperm count (272 vs. 191.9 x 106 ; $p = 0.042$) , total motile sperm count (105 vs. 64.9 x 106 ; $p = 0.036$), FSH (4.1 vs. 3.2 UI/L ; $p = 0.038$) , and reduced estradiol (74.4 vs. 92.9 pmol/L ; $p < 0.001$) . No effects were observed in 20 men, 40–47 years of age, who were exposed to TCDD (median, 123 ppt) as adults (mean age at exposure, 21.5 years) .

Conclusions: Exposure to TCDD in infancy reduces sperm concentration and motility, and an opposite effect is seen with exposure during puberty. Exposure in either period leads to permanent reduction of estradiol and increased FSH. These effects are permanent and occur at TCDD concentrations < 68 ppt, which is within one order of magnitude of those in the industrialized world in the 1970s and 1980s and may be responsible at least in part for the reported decrease in sperm quality, especially in younger men.

Environ Health Perspect 116:70–77 (2008) doi:10.1289/ehp.10399 available via http://dx.doi.org/ [Online 29/10/2007]

problems. The first day on the job, heinitiated a series of tests that today have surpassed 1 million. He saved a blood sample from each person in case it became possible to measure TCDD (dioxin) someday. This became possible in 1987 and the 30,000 (approx) samples Mocarelli put in the refrigerator following the accident have paid dividends as he works with the U.S. Centers for Disease Control and Prevention to unravel the accident and its implications more than two decades later.

It was also noted that excessive numbers of people had died of heart attacks, perhaps brought on by ischemic heart disease.

Ischemic heart disease has been noted in other studies in dioxin and refers to a narrowing of the arteries with a consequent reduction of blood flow to the heart which can result in a heart attack.

A report published in the *American Journal of Epidemiology* in 1993[5] covering the decade from 1976 to 1986, a short period in which to find cancer occurrences as the latency period for cancers varies from 7 to 40 or 50 years revealed several elevated disease rates among the exposed group.

Dr. Linda Birnbaum, director of environmental toxicology for U.S. EPA, told the Associated Press that the new study "is one more nail in the coffin" for dioxin.[6]

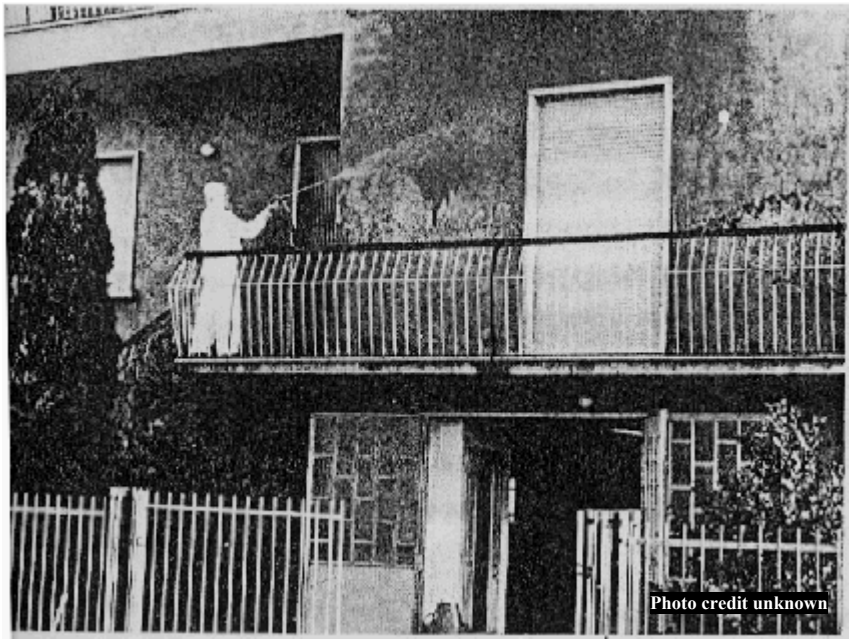


Photo credit unknown

Birnbaum, who was coordinating EPA's multi-year "scientific reassessment" of dioxin said, "This, together with other studies, clearly supports that dioxin has the potential to cause cancer in people, just as it does in every animal it's been tested in. The weight of the evidence is becoming overwhelming," she told Associated Press reporter Paul Raeburn.

Any 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 the three areas, labeled zones A, B, R.

The small A zone was most heavily contaminated, but its 724 residents were evacuated. ("Heavy" contamination means that each square yard of land contained 13 to 494 micrograms of dioxin; a microgram is a millionth of a gram and there are 28 grams in an ounce.)

The B zone was less heavily contaminated but its 4824 residents were not evacuated; zone B contained 43 micrograms of dioxin per square yard of soil, or less. The R zone was even less contaminated (average contamination being 4.3

Hautarzt. 1976 Jul;27(7):328-33. **[Chloracne, porphyria cutanea tarda, and other poisonings due to the herbicides]**[Article in German] Jirásek L, Kalenský J, Kubec K, Pazderová J, Lukás E.
In 80 industrial workers producing herbicides (2,4,5-trichlorphenoxyaceticacidsodium and sodiumpentachlorphenolate) in Czechoslovakia the following signs of intoxication caused by 2,3,6,7-tetrachlordibenzodioxin were found: Dermatological: Chloracne and Porphyria cutanea tarda. Internal: Disorders of the metabolism of porphyrins, fats, carbohydrates, plasmaproteins. Neurological: Mainly lesions of the peripheral neurone. Psychiatric: Neurasthenic syndrome and organic lesions.
Differences from the usual course of chloracne were observed. Porphyria cutanea tarda acquisita was most obvious, one patient suffered and died from severe atherosclerosis, hypertension and diabetes. Many patients developed polyneuropathy, as verified both by EMG and autopsy. Two patients died from bronchogenic carcinoma. PMID: 134006 [PubMed - indexed for MEDLINE]The development and prognosis of chronic intoxication by tetrachlordibenzo-p-dioxin in men. *[Arch Environ Health.* 1981][Acne chlorina and porphyria cutanea tarda during the manufacturing of herbicides] [*Cesk Dermatol.* 1973][Acne chlorina, porphyria cutanea tarda and other manifestations of general poisoning during the manufacture of herbicides. II] [*Cesk Dermatol.* 1974]Leads from the MMWR. Porphyria cutanea tarda and sarcoma in a worker exposed to 2,3,7,8-tetrachlorodibenzodioxin-Missouri. [*JAMA.* 1984]Angiosarcoma, porphyria cutanea tarda, and probable chloracne in a worker exposed to waste oil contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin. [*Br J Ind Med.* 1993] [Acne chlorina and porphyria cutanea tarda during the manufacturing of herbicides] *Cesk Dermatol.* 1973 Oct; 48(5):306-17.

micrograms per square yard), so its 31,647 residents were probably exposed to low levels. Another 181,579 people living beyond zone R serve as a control group living in "non-contaminated" areas.

Zone A is the closest to the accident site. R extends several miles distance. Zone B is between the two.

The assumption is that people's exposure varied with distance from site of the accident.

In zone A, women had elevated cancers of the gall bladder and biliary tract (the system that delivers bile from the liver to the small intestine). They also had elevated occurrences of circulatory diseases and of chronic rheumatic heart disease. Men in zone A had elevated occurrences of cerebrovascular disease (such as stroke). In zone B men had elevated melanomas (serious skin cancers) and cancer of the lining of the chest cavity (pleura); women in zone B has elevated incidence of soft tissue sarcomas. In zone R, men had elevated incidences of cancers of the lining of the chest cavity (pleura) and they had increased incidence of all blood diseases and 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 average for the entire decade. In the case of cancers, which would only begin to occur after a latency period, the time of interest was the second five years of the decade. The researchers reported results for each half of the decade.

During the second half there were no elevated cancer levels found in zone A. In zone B men showed elevated incidence of cancers of the lung, cancer the lining of the chest (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.

Whereas this particular study did not prove that dioxin exposure caused the cancers or other serious ailments from which these people suffered in abnormally high numbers (mainly disease of the heart, blood and other arteries). It did confirm 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."*[7]

Which is exactly what pyromaniacs claim when they say *"there is no evidence that the population was harmed apart from developing chloracne, which is a nasty skin complaint."*

In addition to the studies at Seveso, a 1988 U.S government study had shown that Vietnam Veterans exposed to Agent Orange (contaminated with dioxin) suffered from elevated incidence of cancers, liver damage, cardiovascular deterioration, and degeneration of the endocrine system.

The study found that 4.59% of the Ranch Hands have some kind of cancer, compared to 2.33% of an 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 risk is increased by a factor of 2.6), whereas the risk for "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.

This Seveso study was not the first to indicate that dioxin causes cancer in humans. [8][9][10][11]

Swedish researchers in the late 1970s began reporting that exposure to phenoxy herbicides (2,4-D and 2,4,5-T) caused a 3-fold to 6-fold increase inthe risk of soft tissue sarcomas and lymphomas. Phenoxy herbicides are contaminated with dioxin during manufacture.

Germany, BASF workers

German workers who manufactured chlorophenols and phenoxy herbicides had their adipose tissues sampled in 1988, 32 years after known special factory exposure and analysed for TCDD.[1] All 6 workers studied had chloracne from high exposure as well as genetic sensitivity to dioxins. The average concentration was 49 ppt TCDD on a lipid basis, about 10 times higher than the then mean level of TCDD in humans from industrial countries, and the range was 11-141 ppt. .

These six patients were involved in direct contact with dioxins during a dioxin cleanup in 1953. They all developed persistent chloracne. Other medical signs and symptoms were noted after exposure including fatigue, headache, memory impairment, severe pain in the abdomen and extremities, liver pathology, elevated serum lipids, conjunctivitis, insomnia and gastrointestinal system pathology. These symptoms were documented in the patients' medical records. The medical problems listed above can be caused by dioxins although other causes are also possible.

In light of the history of chloracne following exposure plus the other reported or observed medical problems, it seemed to us reasonable to conclude that there were some probable causal linkages between the ingestion of the 2,3,7,8-TCDD, which was documented at the work site, and at least some of the subsequent illnesses, including the severe characteristic skin lesion, chloracne.

[1]. Schecter A, Ryan JJ. Polychlorinated dibenzo-p-dioxin and dibenzofuran levels in human adipose tissues from workers 32 years after occupational exposure to 2,3,7,8-TCDD. *Chemosphere* 1988;17(5):915-20.

In both animal and human studies (notably epidemiological analyses of cancer rates following the accidentin Seveso,) TCDD exposure has increased cancer incidence and mortality at all cancer sites rather than at a few specific sites. In 1997, the International Agency for Research on Cancer upgraded TCDD to a Group 1 human carcinogen on the basis of mechanistic data. Considering subsequent dose–response assessments for TCDD and cancer, Kyle Steenland, a professor of environmental and occupational health at Emory University, and colleagues argued in the September 2004 issue of EHP that "TCDD exposure levels close to those in the general population may be carcinogenic and argue for caution in setting the upper ranges of long-term permissible exposure to dioxins."

Although TCDD is carcinogenic, it is not directly genotoxic. A report in the 8 January 2008 Proceedings of the National Academy of Sciences now demonstrates one of the ways that TCDD may promote cancer's growth and spread.

A new study describes a novel mechanism of TCDD action that focuses on the mitochondria: "We found that TCDD induces tumor cell proliferation and invasion by directly acting on mitochondrial transcription machinery and inducing mitochon-

'Capitalism means progress, and progress can lead sometimes to some inconvenience.'
Dr. Adolph Jann, President of Hoffmann-La Roche.

drial respiratory stress," says principal investigator Narayan G. Avadhani, a biochemistry professor at the University ofPennsylvania. Such mitochondrial dysfunction inhibits apoptosis in malignant cells and increases the invasive potential of cancer. Mitochondrial dysfunction is also associated with conditions such as heart disease, diabetes, obesity, blindness, deafness, kidney disease, and neurodegenerative disorders, as well as with aging.

"[The respiratory stress-signaling] cascade culminates in the activation of a large number of nuclear genes that affect various cellular processes including cell metabolism, proliferation, and apoptosis," says lead author Gopa Biswas, a researcher in Avadhani's lab. "We have now established that TCDD alters cellular morphology and physiology through a similar mechanism."

It is generally accepted that adverse effects of TCDD result from its activation of the Ah receptor, with effects occurring at very low exposures. In the presence of TCDD, the Ah receptor has been shown to either induce or suppress the transcription of numerous genes that have been linked with cancer development via changes in tumor suppressor proteins, oncogenes, growth factors, and cell cycle proteins, among other factors.

Mitochondrial dysfunction may entail a more fundamental mechanism. It appears that TCDD-induced mitochondrial stress signaling in cancer cells is propagated in part through the Ah receptor but also acts through mechanisms that are independent of the Ah receptor, such as by inducing protein kinase C and extracellular signal–regulated kinases.

"Our findings show that at subtoxic levels of ten to fifty nanomolar, TCDD is sufficient to cause mitochondrial dysfunction and induce the signaling cascade," says Avadhani. "These results raise concerns over the adverse health implications of dioxins and PCBs even at very low levels."

Recognition that the carcinogenic effects of environmental toxicants may originate in disruption of mitochondrial biology could prove important for the future development of cancer prevention and treatment procedures related to TCDD and other dioxin exposures. "The new findings

In 1963 an explosion occurred in a 2-4-5-T factory owned by Philips Duphar in Amsterdam, Holland. Fifty workers developed Chloracne and suffered internal damage and serious psychological disturbances as a result, when workers tried to decontaminate the plant six months later. All but one of the workers wore deep-sea diving suits and industrial facemasks, nine men contracted Chloracne, and three of them died within the next two years. The worker who was not as well protected was still being treated in 1976 for severe effects and was unable to work.

In 1973 the plant was still so contaminated with Dioxin that it had to be dismantled, embedded in concrete, and buried at sea. Between 1965 and 1969 a 2-4-5-T production plant near Prague, Czechoslovakia, developed leaks in its processing area. Workers developed Chloracne and exhibited weight loss, libido diminution and insomnia.

Maximum symptoms were observed about one to two years after the initial exposure but lasted over eight years in some of the exposed workers. Several workers died of severe liver damage, and workers' families also became sick. Contaminated equipment was buried in a mine shaft.

Other studies of workers exposed to 2-4-D and 2-4-5-T were conducted by Festisov (1966), Long (1969), Poland (1971), Sundell (1972) and Piper (1973). These studies showed exposed workers exhibiting symptoms including fatigue, headaches, loss of appetite, stomach and kidney pain, upper respiratory distress, decreased hearing, smell and neurological responses, high serum albumin values, skin and eye irritations and concentrated TCDD (Dioxin) levels in body fat and liver tissue... Further tests showed TCDD, the contaminant in 2-4-5-T, to be an extremely toxic agent with a slow effect rate and diverse symptomatology including edema, necrotic changes of the liver, gastric hyperplasia and ulceration, hemmoroglus of gastrointestinal tract and other organs, atrophy of the kidneys, thymus and other lymphoid organs and tissues. Later, symptoms appear to lead to decreased immune responses.

suggest that the risk of cancer may be reduced by avoiding or lowering exposure to environmental mitochondrial toxicants as well as [possibly] by optimizing mitochondrial energy metabolism by nutritional and medicinal means," says Egil Fosslien, a pathology professor emeritus at the University of Illinois at Chicago. [12]

'Had the effects of dioxin poisoning [in Seveso] been more dramatic, had people dropped in the streets as the animals did, had the poison been visible or radioactive or detectable in some simple way, the story would have been different. But dioxin is the most insidious of substances, working in a un-news-worthy manner producing damage that may not become obvious for years, and may only be clearly demonstrable by a well-founded epidemiological study. Extract from *The Super Poison.*

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[12] http://www.ehponline.org/docs/2008/116-3/forum.html#canc
M. Nathaniel Mead. Cancer and TCDD: The Mitochondrial Connection*Environmental Health Perspectives* Volume 116, Number 3, March 2008

[Note: This assessment addresses only dioxin releases in stack gases and does not include the far greater releases in residues, especially fly ash, despite its invocation of the Stockholm Convention.] *Chemosphere* Volume 73, Issue 10 , November 2008, Pages 1632-1639 .doi:10.1016/j.chemosphere.2008.07.068

Practices in dioxin emission reduction by special regulatory enforcement and utilizing advanced control technologies for incinerators in Korea .**Ki-Heon Kim [a] , Bong-Jin Chung [b] , Sang-Hyeob Lee [c] and Yong-Chil Seo [c]** , [a]National Institute of Environmental Research, Inchon, Republic of Korea [b]Department of Environmental Engineering, Suwon University, Suwon, Republic of Korea [c]Department of Environmental Engineering, YIEST, Yonsei University, 234 Maeji-Ri, Wonju, Kangwon, Republic of Korea.

Abstract

This study strives to estimate the emission of dioxin and furthermore attempts to find the best technological control methods available for waste incinerators by investigating the emission status thereof. In order to incorporate the Stockholm Convention, a particular stringent law was promulgated in Korea and in recent years incinerators were forced to utilize better technological control. After the enforcement of special dioxin emission regulation in 2003, the average concentration of dioxin emitted from municipal and industrial waste incinerators decreased from 15.25 and 12.86 ng TEQ Nm^{-3} to 5.53 and 4.96 ng TEQ Nm^{-3} in 2001 and 2004, respectively. Based on test results at commercial plants, several best arranged sets of air pollution control devices (APCDs) were suggested in order to provide guidelines to help operators. These sets included combinations of spray dry absorbers, bag type filters, wet scrubbers, selective catalytic reductions and electrostatic precipitators. Different suggestions and real installations of APCD arrangement were investigated during the years around the regulation in effective. The results were presented depending on the capacity of the incinerators and different waste streams to observe the efforts to reduce dioxin emission by operators of incineration plants. The annual amount of dioxin emission from the incinerators is expected to be 212.5 g-TEQ in 2011 and 234.3 g-TEQ in 2015, respectively, compared to 891.6 g-TEQ recorded in 2001. The enforcement of new regulation and the installation of better APCDs showed the significant effect on such reduction. This reduction in dioxin emission from incinerators confirmed the nation's commitment to the regulatory requirement set by the Stockholm Convention.

Coalite Chemicals

In April 1968 an explosion at the Coalite and Chemicals works in Bolsover, England, resulted in falling masonry killing a chemist and showering workers with dioxin.

Seventy nine cases of chloracne were recorded and Dr. Jenny Martin, Consultant Chemical Pathologist at Chesterfield Royal Hospital, was commissioned by Coalite to research the effect on the workers.

When the study was completed Coalite told Dr. Martin that it did not wish to have the information published and informed her of the nature of the control group used for the study.

Realising the study had been devalued by Coalite including management staff in the control population, instead of restricting it to the chemical workers, Dr.Martin arranged a second study without Coalite's involvement.

She published the results of blood chemistry from eight workers suffering from chloracne in The Lancet in February 1979.

Shortly after this her home was broken into and only the medical records of the Coalite workers were stolen. Nothing else was taken.[1]

"The police were very sharp with me and gave me a four hour grilling." Dr. Martin told me: "You would have thought I was the criminal and not the victim. Mind you, a number of people were not happy with me putting the plant in the news again."

Dr. Martin said a number of people exposed to dioxin at Coalite died from heart problems which biochemical tests linked to chemicals.[1]

The debris from the explosion was dumped at a secret site, but the 'cover-up' didn't stop there. The plant was again in the national news in 1991 when it was responsible for what was then, the worst case of dioxin contamination recorded in the UK.

Coalite had been sending its waste to the Cleanaway incinerator in Ellesmere Port for years before the company decided it would be better to construct their own facility and this, desipte claims to the contrary by management, had been burning well below the necessary temperature to destroy the waste. This resulted in large amounts of dioxin being spewed over the surrounding area causing the milk of 27 farms to exceed the British governments 'acceptable' level of dioxin contamination.[2]

A third farm that produced calves for veal was found to have dioxin levels of 3.4ng/kg of whole milk.

Faced with what would be a serious blow to the financial interests of the dairy industry, and the government politically if the extent of the contamination and the events leading up to it had been made public. The British Government's experts simply 'diluted' the toxicity of dioxin by raising the acceptable level of contamination from 1 picogram per kilogramof body weight a day (pg/kg/bw/a day) to that of the World Health Organistions of 10/pg/kg/bw/a day, thereby, with the simple action of a pen, magically detoxifying the milk of 25 farms and making it 'fit for human consumption.'

This 'fact-free' detoxification not only avoided what would have been a national disaster for the industry. It kept under wraps the serious dishonesty and incompetence of British politicians who had ignored recommendations as long ago as 1982 for an inquiry into potential dioxin pollution from the Coalite plant.

The two farms still over the safe limit had levels of dioxins equating to 1.21 and 0.85 ng/kg of whole milk, compared with a guideline 'action level' of 0.7ng/kg.

Managing director of Coalite Chemicals, Peter Stefanini, said he would not comment on the call for a public enquiry as this was a matter for the government.

"Acceptable" daily doses of dioxin (pg/kg/day) at the time of the Coalite scandal.	
USEPA	0.006
State of California	0.007
CDC	0.03
US Food & Drug Admin	0.06
Nat Res Conc of Canada	0.07
Germany	1-10
Netherlands	4
Canada & Ontario	10
World Health Org	10
UK	1

"We are forever hearing of breast milk being a source of dioxin. Let's get it straight! Breast milk is not a source of dioxin. It's the industry that put it there that's the source!
Pat Costner, Greenpeace

He said: 'We conducted our own investigations into the incinerator at the time these issues first cropped up because everyone recognised that incinerators are potential sources of dioxins.

'Our own conclusions are that the incinerator has been working within the standards laid down. HMIPis also aware of how our incinerator operates . . . I would be surprised if our incinerator was producing sufficient levels of dioxin to be responsible for the levels found in the milk.'

Stefanini points out that the dioxin levels represent toxic equivalent calculations of 17 forms of dioxin relative to 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD). He argued that emission samples from milk, the incinerator stack and sediment around the plant have all been found to contain varying proportions of the different dioxins, making it difficult to identify a precise source.

The Coalite executive said that while it is right that the Ministry of Agriculture, Farms and Fisheries (MAFF) should set safe levels for dioxins and act when these are exceeded, a number of assumptions are made in setting the levels and they actually represent extremely low concentrations.

A number of disturbing features came to light during investigations into this affair. The contamination of the milk had not been discovered because of people's concerns about the plant and dioxin, but simply because MAFF were doing random milk testing around the country.

Because the government failed to monitor the plant despite the concerns aired in 1982, no-one knows how long the incinerator had been operating below the optimum temperature and pumping dioxin over the countryside and people of the area.

Researching into the incident for the book *Waste not Want not!* (Earthscan) and as anti-incineration activists we looked deeper into the affair than most people. We visited the local Environmental Health Department where their spokewoman was adamant that the plant didn't have an incinerator, despite the fact it was visible from the factory gates.

On our second visit to the planning department we were met by a number of police officers who question us and checked over our vehicle explaining they "thought it strange an Irishman and a Scouser looking at the plans of a chemical plant."

When we were released we went back to the planning office and explained why were in the area. We asked for a certain dated file but were handed a different file to the one we had requested. "No, we want this particular file" we said. "No, this is the one you want" was the quite reply. In this file we saw there was no incinerator shown on one page of plans of the plant, but it appeared on the next page. No planning application had been lodged or passed in-between dates, and the first mention of the incinerator was a letter to the company from the planning authority stating 'we have no objections to the expansion of the incinerator which has operated with no problems for three years.'

As the Coalite in-house incinerator was closed by the regulators, there was no alternative on-site method to dispose of the heavily dioxin contaminated chemical waste. This resulted in it being stored in holding tanks on site for months.

Given their experiences and first-hand knowledge of the persistence and toxicity of dioxin, Joe Holland,

one of the workers at Coalite we had interviewed about the contamination interviewed about the contamination, phoned me saying “the workers were scared to walk past the tanks.”

This highly toxic waste was eventually transported by road tanker, along with the contaminated milk from the two farms still above the new ‘acceptable’ level, to the Cleanaway hazardous waste incinerator at Ellesmere Port, Cheshire.

Local citizens who campaigned against thebuilding of this plant expressed great concern as they were aware the plant already had a history of fires, explosions, dump stack operations and as many as seven colourful chemical releases in one month.

Hearing of where the waste was going and the incidents at the plant Joe Holland phoned me expressing his concern saying the waste: “should not be disposed of in such a densely populated area.”

The concerns of Joe Holland and the Ellesmere Port community were not shared by Henry Pullen, a director of Cleanaway and one time chemist of Purle and Monsanto.

He told the local media: “this waste is no different than any other,” but then Henry Pullen did not live in Ellesmere Port and had never worked at Coalite.

Coalite were in the news again a few years later when news that dioxin levels in milk from farms around the plant increased markedly in October 1996 was released by MAFF in August 1997.[3]

The contamination was close to levels which led to a ban on sales of produce despite the incinerator being closed in 1991. In February 1996, Coalite was prosecuted for failing to use the "best practicable means" to prevent pollution from the plant and fined £150,000. [4]

Monitoring of milk around the site showed a rapid decline in dioxin levels after the incinerator closed. On most farms, levels appeared to be approaching the norm for industrialised areas - until samples from two farms showed a return almost to the 1991 peak levels (see table below).

The levels in milk from Farm B far exceeded the maximum tolerable concentration of 16.6ng per kilogram of milk fat, expressed as the toxic equivalent (TEQ) of the most toxic dioxin 2,3,7,8-TCDD. However, Farm B produces suckling cattle and does not provide milk for human consumption.

MAFF carried out further sampling in and concluded that as milk from Farm F did not exceed the tolerable concentration there was “no risk to human health”. This level is set to ensure that extreme consumers of milk do not exceed the Department of Health's tolerable daily intake for dioxins - which has been criticised as 100 or even 1,000 times too high to protect health.[5]

The Environment Agency says that no likely sources of atmospheric dioxin pollution remaiedn at the Coalite works. Provisional results of air samples taken in the weeks before last October’s milk samples did not show elevated dioxin levels.

“The indications are that there was no aerial release,” a spokesman said.

However, after a prolonged dry period heavy rain fell a few days before the milk was sampled. The spokesman suggested that cattle uprooted the grass and ate large amounts of soil, although dioxins in soil are generally believed to be poorly absorbed by cattle.

If soil is confirmed as the source of the contamination, the findings will call into question the wisdom of allowing highly contaminated soils to be grazed by livestock. Occasional ingestion of such soil by livestock could be a significant route of dioxins passing into the human food chain.

Dioxin levels in milk (ngTEQ/kg milk fat)

	July 1991	Aug 1992	Sept 1993	July 1994	Aug 1995	Oct 1996
Farm A	21	7.4	41	5.8	-	-
Farm B	85	48	25	27	26	62
Farm F	6.0	2.S	2.0	2 4	2.3	6 0
Farm H	5 2	3.2	2.5	21	2 7	

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[3] Food surveillance sheet 124: Dioxins and PCBs in cows’ milk from the Bolsover area.
[4] ENDS Report 253, pp 48-49
[5] ENDS Report 255, pp 3-5
[6] Food surveillance sheet 123: Dioxins and PCBs in cows’ milk from farms close to industrial sites: 1996 survey results. From MAFF, 0171 238 6235.

GENERAL INFORMATION Part 2

Other examples of farm produce being contaminated by dioxin from municipal solid waste incinerators (MSW) are:

* In Rijnmond, Holland in 1989, the milk from 16 farms was so contaminated by dioxin from a nearby MSW that the fat was skimmed off and sent to a nearby toxic waste incinerator for disposal.

* In 1998 a number of MSW's in France were closed because of heavy dioxin contamination of farm produce.

Under Wraps

Secrecy is never far away when dioxin emissions threatens an incinerators operations. The result of samples taken in 1989 and 1991 from around a MSW in the city of Winchester, England, were kept under wraps by the Regulator's Her Majesties Inspectorate of Pollution (HMIP) until 1994. When they were finally made public it was revealed dioxin levels similar to those around the Coalite plant.

HMIP was obviously more concerned with safeguarding the company's interests than protecting the health of the people of Winchester.

Reassessment

In 1990, under pressure from the powerful chlorine industry who, despite the ever-increasing literature on the multiple effects on health of dioxin, considered regulations too restricting and costly. The U.S.EPA undertook a reassessment program of dioxin using all available data i.e., studies of rats, mice, guinea pigs, rabbits, cattle, marmosets, monkeys and humans.

Employing 100 scientists from outside their organisation, as well as their own in-house staff, they concluded after three years of research that:

* the largest source of dioxin was municipal and clinical waste incinerators:

* dioxin was more toxic than had been originally believed:

* dioxin was capable of damaging health in ways not widely anticipated i.e. immune system suppression, endocrine system/hormone disruption:

* that these non-cancer effects occurred at levels 100 times below the level that caused cancer.[1]

The EPA emphasised that dioxin damages the immune system directly and indirectly, and concluded that even low doses attack the immune system by directly reducing the number of B cells that develops in the bone marrow, then circulate throughout the blood and lymph, fighting off invaders.

Despite these findings and acknowledging that municipal waste incineration as being responsible for up to 85% of the UK's dioxin contamination. The Department of the Environment, (under the leadership of John Gummer) were determined to act on recommendations made in 1993 by 28 representatives of the packaging industry, collectively known as The Producers Responsibility Group (PRG) (later to become VALPACK a front for the packaging industry) to undertake a building program of 'close to home recycling plants' i.e. waste to energy incinerators, throughout the UK.

The government's 'guess-estimate' that the most modern incinerator plants would contribute 6% to 18% of this country's future dioxin contamination,[29] was calculated with 10 energy from waste plants operating.

This was before the announcement that an additional 100 MSW incinerators that will, in their opinion, be necessary if the UK is to comply with EU Legislation over the next 15 years or so.

What is even more disturbing about this scheme is the fact that in April 1999 Environment Minister Michael Meacher, a key player in the production of the *Waste Strategy 2000* told a House of Lords inquiry into 'Waste Incineration' in 1999 that: "Incinerator plants are the source of serious toxic pollutants;

Oh, S., Ro, K., Chung, K., 2003. **Induction of Cytochrome P4501A and Endocrine Disrupting Effects of School Incinerator Residues.** *Environmental Monitoring and Assessment* 83: 35-45

Abstract

The emission of the dioxin-like compounds from on-site waste incinerators of seven schools in Kyonggi Province of Korea was evaluated by determination of the cytochrome 4501A(CYP1A) catalytic activity and antiestrogenic activity using cell culture microbioassay. The residue samples were extracted in a Soxhlet apparatus using toluene for 20 hr. The concentrated crude extracts were fractionated with a basic alumina column. Dioxin-like compounds were then extracted.

Induction of CYP1A activity in a rat(H4IIE) hepatoma cell line was used as indicator of biologicaleffect of incinerator residues and measured as 7-ethoxyresorufin-O-deethylase(EROD) activities. The EROD activities of fraction I extracts (one of the two extracts) in the H4IIE cells were from 0.044±0.002 to 4.424±0.351 ng-TEQ g-1 (TCDD Toxicity equivalent), showing relatively high inducing capacity. Antisetrogenicity of the extracts was measured as decrease in E2-induced cell proliferation. Most of the extracts showed antiestrogenic activity in MCF7-BUS cell. The TEQ levels of the incinerator residues and the antiestrogenic activities were in good correlation, strongly suggesting that the potent toxic emissions were indeed produced from the on-site school waste semi-incinerators and could cause the antiestrogenicity.

dioxins, furans, acid gases, particulates, heavy metals, and they all need to be treated very seriously.

"...There must be absolute prioritisation given to human health requirements ...and protection of the environment.

"I repeat that the emissions from incinerator processes are extremely toxic. Some of the emissions are carcinogenic. We know scientifically that there is no safe threshold below which one can allow such emissions..."

Speaking on industry's claim that 'dioxins are natural' during a presentation to the 102nd Meeting of the Great Lakes Water Quality Board, Chicago, Illinois, July 15th 1993 Dr. Linda Birnbaum (Director of Environmental Toxicology Division U.S.EPA) said:

"People have done analyses of Egyptian mummies from more than 2,000 years ago and frozen Eskimos from northern Canada. The levels are below detection limit. Dioxin is a product of modern industrialisation."

When asked if there was a 'threshold', a level below which no effects occur for dioxin, Dr. Birnbaum replied: "There is no threshold for immunotoxic responses to dioxin." In other words, no level of dioxin below which the immune system is not affected.

Human studies

Many industrialists and pyromaniacs still maintained humans are not as sensitive as animals to dioxin. They point out the differences in the sensitivity of a guinea pig compared to a rat. However, research shows this vastly different sensitivity is not strictly true as far as humans are concerned as Dr. Birnbaum stated:

"...[W]ith respect to dioxin, people react similarly to animal responses. ...[T]here is a large amount of data showing for example, that changes in biochemical properties such as enzyme induction in some hormonal states and in growth factors, occur at similar body burdens in animals as they do in people.

"In the on-going occupational study conducted by National Institute of Occupational Safety and Health (NIOSH) looking at workers who were exposed to dioxin. These adult males are showing decreases in the levels of their circulating testosterone at body burdens very similar to the body burdens in adult rats. In immunotoxicity testing, human lymphocytes and cultured cells respond to the same concentration of dioxin in the media as mouse and monkey celis. In terms of developmental toxicity you find similar responses at similar concentrations of TCDD.

"For example, if you take out the embryonic palate of a rat and the embryonic palate of a human, put them in culture and expose them to the same concentration in the media, you get a similar response.

"Similarly, the body burden associated with chloracne is essentially the same as the body burden causing chloracne in monkeys, in hairless mice or in rabbit ears. Animals with a lot of hair --like regular mice and regular rats -- do not develop chloracne. But hairless mice do and the body burden there is essentially the same. Cancer appears to occur at similar body burdens in animals as in humans."

Although animals studies have consistently demonstrated dioxins toxicity, the evidence for toxicity to humans was essentially circumstantial until methods were developed to measure dioxin in human tissue.

Dr. Arnold Schecter, a world renowned expert on dioxin told delegates at one conference he had analysed tissue from the bodies of Eskimos who froze to death over 100 years ago and "found as close to zero as you can get... The point is dioxin are new, they are not something that has always been around."

Speaking on thedifference between animals and humans Dr Schecter said; "Humans are not all that different from other mammals Human cells have cytoplasm, nucleii, mitochondra and so on - just as do other mammals.

"Since it became possible to measure dioxins in humans a number of studies have linked dioxin exposure and toxicity."

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Diabetes

There has been a notable increase in the incidence of diabetes in veterans exposed to dioxin. One study conducted over 20 years on Air Force Veterans exposed to Agent Orange showed that those exposed to dioxin have an increased incidence of diabetes and heart disease.

The body burden that seems to produce an increase in diabetes range from 99 to 140 ng kg. The average American has a body burden of around 13 ng/kg, only a factor of 8 below the lowest level thought to create diabetes.[3] This might only seem a very tiny amount, and as an absolute quantity it is. But compared to the amount that causes major problems in animals and humans, 13 ng/kg qualifies asa major public health problem. It should be noted that in laboratory animals chloracne occurs at body burdens as low as 23ng/kg, and in humans has occurred as body burdens as low as 96 ng/kg.

The EPA published a study that cites examplesof humans getting chloracne with body burdens only 3 times as high as the U.S. average body burden and estimate that 5% of Americans, some 12.5 million people have body burdens twice the average..[4]

Workers Study

A study of 1,189 workers at a pesticide manufacturing plant in Hamburg, Germany, who were exposed between 1952 and 1984 found an increase in deaths compared to a control group consisting of 2,528 non-dioxin exposed workers in the same region. Exposure was related to higher death rates.

They found an increase in all deaths, including cancer deaths and ischemic heart diseases among the dioxin exposed workers, compared to same-aged individuals in the control group. The disease related deaths

increased with the dose of dioxin to which the workers were exposed.

The authors concluded that the results of the study "support the hypothesis of a dose related effect of PCDD/F [dioxin and furans] on a cancer and ischemic heart disease mortality."[5]

Infections

Another study of the health of 158 workers exposed to dioxin during the BASF explosion in 1953 compared them to 161 unexposed workers.

Researchers found the exposed group suffered more frequent infections and parasitic diseases during the 36 years after the explosion. Especially noticeable were increases in respiratory infections, thyroid diseases, disorders of the peripheral nervous system and appendicitis.

Mental disorders were also increased. Altogether the highly exposed group had 18% more recorded episodes of illness than the control group.[6]

Public Perception

The editorial in the September 1993 issue of Epidemiology points out some of the public policy implications of the conclusion that dioxin causes cancer in humans.[7] The author of the editorial, Swedish dioxin researcher Olav Axelson, says that the "biological effects of TCDD [dioxin]" are "a first order public health concern."

"There seems to be an urgent and costly need to change or improve industrial and other processes so as not to produce dioxins (and the toxicologically similar chlorinated dibenzo-furans). For example, there is a need to restrict the use of chlorine in paper bleaching. Incineration of waste material at too low temperature should be avoided as well as the 'combustion' of organochlorine compounds in general," Axelson said.

Dioxin was declared a Class 1 carcinogen, or "known human carcinogen," by the International Agency for Research on Cancer (IARC), zation in February, 1997.

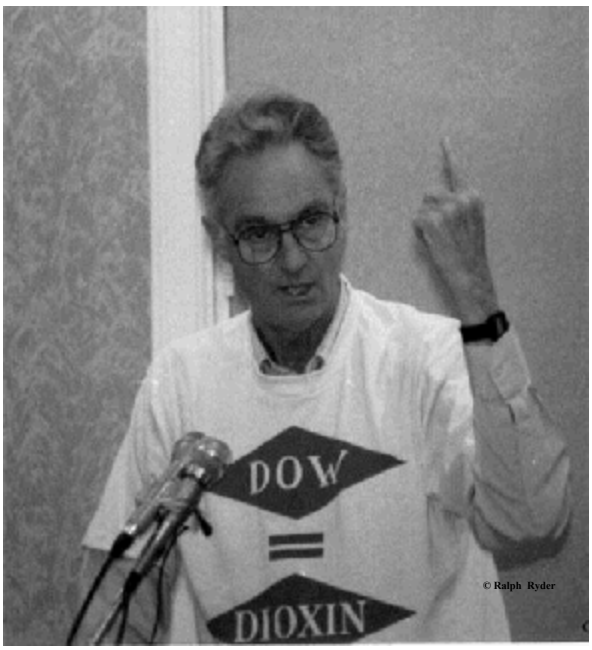
Despite this, and a mountain of peer-reviewed studies showing a wide range of disturbing health impacts far worst than chloracne. Dame Barbara Clayton, Past President of the National Society for Clean Air and Environmental Protection (NSCAEP), when giving evidence to a House of Lords Select Committee inquiry (1999) declared

"...If you look at the massive exposure of people to dioxins as a result of two major accidents there is no evi-dence that the population was harmed apart from developing severe chloracne which is a nasty skin complaint, but that was with massive exposure."[8]

This is a well-know industry-scripted line regurgitated 'parrot fashion' at every opportunity by pyromaniacs. However, given the evidence available at that time it is certainly not a statement one would expect from such a eminent member of Britain's scientific community - and whom one would assume had studied all the available information to present to such an important the inquiry.

My bewilderment was further increased when Dame Clayton told the committee:

As well as the deliberate manipulation and falsifying of figures by industry, another method of distortion is omission. Citizens are mislead not only by what is said, but even more by what is left unsaid



Peter Montaque Ph.D editor of *Rachel's* Democracy and Health News addresses delegates at the '3rd Citizens' Dioxin Conference in Baton Rouge 1996.

"the public look on dioxins as the very severe chemical..." - "...there is no reason to have that view but it is very much the public perception...." An absolutely unbelievable statement when you consider the mountain of peer reviewed papers available at that time. After studying dioxin intensely for a decade the U.S.EPA had clearly described dioxin 'as a serious public health threat' (September 1994) and two years`earlier had stated 'dioxin is much more toxic than previously known.'

The agency said, 'Indeed, these [dioxin] compounds are extremely potent in producing a variety of effects in experimental animals based on traditional toxicology studies at levels hundreds or thousands of times lower than most chemicals of environmental interest.'

And: "There is adequate evidence from studies in human populations as well as in laboratory animals and from ancillary experimental data, to support the inference that humans are likely to respond with a plethora [an abundance] of effects from exposure to dioxin and related compounds."

I also think we should also consider the statement by epidemiologist Richard Clapp a researcher into the impact of dioxin on solders in Vietnam who described it as "the Darth Vadar of chemicals." While Dr. Linda Birnbaum (U.SEPA) has described it as "the badiest of the bad."

Of course the question scientists, academic and Health Authority officials should be asking is: 'Why did Professor Clayton fail to mention any of the peer-reviewed studies to the committee?' After all these were published several years earlier and it is not unreasonable to expect an expert to have kept up with the evidence before asking to be allowed to speak to such an important body.

Other studies the eminent Professor omitted to mention include: [9][10][11][12][13] all showing far more serious health impacts than a 'nasty skin disease' from dioxin exposure, and all published well in advance of the inquiry.

Perhaps another question to be asked is "which two major accidents the Professor was referring to? Yusho? Yu-Cheng? Seveso?

I have mentioned the effects in Seveso earlier, and briefly mentioned the epidemic poisoning at Yusho and Yu-Cheng where severe developmental effects were observed in infants and children born to mothers exposed to dioxin-like polychlorinated dibenzofurans /biphenyls (PCDFs/PCBs) including: Intrauterine growth retardation,

Dr. Linda Birnbaum

Low birth weight,
Hyperpigmentation,
Natal teeth,
Increased incidences of skin and respiratory infections,
Neurodevelopmental delay,
Alterations in sexual development.
All conditions far worst than a 'nasty skin complaint.'

Breastmilk

Scientists have known for years that pollutants/chemicals were accumulating in women's breast milk. We now know that the body burden of a new born child is increased by the simple, loving act of breastfeeding.

"When we looked at the children of the women chemical workers 25 years after their exposure had stopped, we found elevated levels in the children, which we think comes from nursing". said Dr. Arnold Schecter.Dr. Schecter found that there had been a transfer of dioxin from mother to infant, and more of a transfer in stillborns. He noted up to 50% of the dioxin in one mothers body being transferred to the twins she nursed for two years.

"Nursing is highly desirable in general and yet we are unhappy with the high levels of dioxins, furans, and PCBs found in nursing mothers' breast milk" he said.[14]

The U.S.EPA found that breast milk levels of dioxins toxic equivalent (TEQ) were about 20 ppt lipid, or about 1 ppt of milk. Since infants are fed solely on breast milk, that is 200 picograms TEQ per feeding, or 800 picograms TEQ per day. The (revised) U.S.EPA maximum allowable dose of .01 pg/kg/day for adults would lead to a lifetime dose of 20,000 picograms. Thus an infant ingesting typical breast milk would accumulate a lifetime dose of dioxin TEQ in about 25 DAYS.[15]

Even though the British government has set a much higher tolerable daily intake (TDI) figure than the U.S., a Committee on Toxicity of Chemicals in Food Consumer Products and the Environment (COT) report in 1997 indicated that young breast fed babies may be exposed to as much as 17 times the UK's TDI of dioxin-like chemicals in their body.

The average levels of PCBs and dioxins for a two month old breast

Nishijo *et al.,* 2007. **Effects of maternal exposure to 2,3,7,8-tetrachlorodibenzo-*p*- dioxin on fetal brain growth and motor and behavioral development in offspring rats.** *Toxicology Letters.* Article in Press.

Abstract

The effects of maternal exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) during pregnancy on fetal brain growth and neurobehavioral development in early developmental stages were investigated using rat offspring. TCDD in corn-oil (0.1mg/kg) was orally administrated to the dams from the 9th to 19th gestational day. When TCDD effects on the fetal brain weight were analyzed on the 19th gestational day, weight ratio of the brain to the whole body, and that of the forebrain without the cerebral cortex to the whole brain were larger in the exposed group than those of the control group, suggesting premature fetal brain development. TCDD effects on motor functions were investigated using newborns in an inclined plane task. Motor development assessed by righting response on an inclination was delayed in the exposed offspring in the 8th–12th postnatal day, especially in male. Also, TCDD effects on active avoidance behavior in a shuttle box were investigated using the offspring after weaning. Latency in the active avoidance learning was longer, and locomotor activity was reduced in the exposed male offspring in the 41st–44th postnatal day. The results demonstrated that maternal TCDD exposure delayed fetal brain growth and neurodevelopment of the offspring in early stage, especially in male rats.

fed baby is estimated to be 170 TEQ pg/kg/bw/aday), when the recommended TDI intake in the UK is 10.[16]

World Health Organisation

On June 4th 1998 after a 4 day debate, 40 specialist from 15 countries within the World Health Organisation (WHO) declared they had lowered what they had maintained for a decade was a TDI of dioxin from 10 pg/kg/bw/a day - to 1 to 4 pg/kg/bw/a day.[17]

They issued the statement: "The experts recognised that subtle effects may already occur in the general population in developed countries at levels of 2 to 6 pg/kg/bw/a day."

This being the case, one must ask why they are talking in terms of the 'tolerable' level being 1 to 4 pg/kg/bw/a day and simply not 1 to <2 pg/kg/bw/a day?

But even this figure is worrying. Whereas the Belgium limit for dioxin in chickens is 5ppt, the U.S.EPA calculate that five ounces of chicken meat contaminated with 3 ppt of dioxin would contain a total dioxin load of 420 picograms, or about 600 times what the U.S.EPA might consider an adults's acceptable daily intake of 0.7 picograms per day.

Even the 'nasty skin disease' pyromaniacs speak of as being the only result of high dioxin exposure has some terrible results

Put another way: if an adult ate 43 5-ounce servings of chicken containing 3 ppt of dioxin, they would exceed the U.S.EPA's recommended LIFETIME dose of dioxin from those 43 meals alone. Many of us eat far more than 43 servings of chicken every year.[18]

Unfortunately it seems that the lies, omissions and detoxification of dioxin is not confined to the industrial boardroom, the corridors of Westminster or the House of Lords. Even the experts of WHO are tacitly accepting the permanent chemical pollution of air, water and food.

To reach the revised figure of 1 to 4pg/kg/bw/a day, they took the lowest observed level that caused problems in laboratory animals and reduced it by a factor of ten. Normal practice in such circumstances would be to apply a safety factor of 100, but, if they had applied this, they would have been declaring much of

Kim *et al.,* 2007. **Enrichment of PCDDs/PCDFs in the cooling system of municipal solid waste incineration plants.** *Waste Management* 27: 1593-1602 **Sam-Cwan Kim [a], Kil-Chul Lee [a], Ki-Heon Kim [a], Myung-Hee Kwon [a] and Geum-Ju Song [a]** National Institute of Environmental Research (NIER), Environmental Research Complex, Kyungseo-Dong, Seo-Ku, Inchon 404-170, Republic of Korea . Accepted 13 July 2006. Available online 29 September 2006.

Abstract

This study measured the levels of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDDs/PCDFs), destroyed or formed in combustors and re-synthesized in cooling systems. For the proper control of PCDDs/PCDFs in municipal solid waste (MSW) incinerators, three grate-type MSW incinerators were selected, two of which had boilers, and one of which had a water spray tower (WST) as a cooling system. At the combustor outlets, dusts were in the range of 1640–4270 mg/S m^3 and PCDDs/PCDFs were in the range of 0.103–2.619 ng- TEQ/S m^3, showing the different values according to the grate structure of combustor and the flow direction of flue gas. After the flue gases passed through the cooling system, PCDDs/PCDFs at the waste heat boiler (WHB) outlets were enriched to levels that were 10.8–13.6 times higher than those at the furnace outlets, but PCDDs/PCDFs at the WST outlet was reduced to 5% of the level found at the furnace outlet. The emission patterns, such as the ratio of PCDFs to PCDDs, the ratio of gaseous-phase to particulate-phase PCDDs/PCDFs, and the compositional percentiles of each 2,3,7,8-substituted congener varied according to the types of air pollution control devices (APCDs). Reducing re-synthesis in the cooling system rather than enhancing the removal efficiencies of the APCDs seems to be more effective for lowering the levels of PCDDs/PCDFs in MSW incineration plants.

the food of the industrialised countries dangerously contaminated.

This could well have proved politically dangerous and might well have cause panic among consumers when the wider public realised the extent of their subtle poisoning by the chemical industry and its associates, which of course, as a major emitter of dioxin, includes the incinerator industry.

Regulatory Bodies Will Protect You

In the UK citizens are assussed the Environment Agency will protect them from polluting incinerators. The reality is however its officials have been working hand in hand with the operators to spread dioxin around. The following is taken from an article I had published in *The Ecologist* Vol 31 No 8 October 2001.

...on 3 July 2001, BBC's *Newsnight* featured a report on the use of incinerator fly ash and bottom ash on the allotments at Byker, Newcastle, and in breeze block type buildings and road aggregate at the Edmonton incinerator in London. This mixture contained a number of toxins including: arsenic, cadmium, mercury, lead, zinc, nickel, copper and Polychlorinated Dibenzodioxin, more commonly known as dioxin.

Dioxin is perhaps best known as a contaminate of the herbicide Agent Orange, used in the Vietnam War to kill foliage. It is a recognised carcinogen causing cancer in every species every tested. The United States Environmental Protection Agency believes itis responsible for 100cancer deaths every day in the US. It causes Vitamin K deficiency in babies, disrupts the immune system, mimics hormone function, and interrupts the thyroid, which in turn causes developmental and neurological problems in children.

It has been calculated that up to 8,000 cancer cases will result in Belgium due to the dioxin food contamination that took place there in 1999.[1][2] And now, in the UK we are building roads and houses with it and spreading it on our vegetable patches.

After watching the programme, concerned citizens swamped building block companies with calls for information.[3] But few people were aware that the operators of the Edmonton plant had been mixing fly and bottom ash for approximately 30 years. They continued this practice despite being informed in 1977, along with the rest of the incinerator industry and the regulatory bodies of the UK, that incinerator fly ash is heavily contaminated with heavy metals and dioxin.[4]

At last, communities around the country are waking up to the failings of the Environment Agency (EA) to protect public health from

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the dubious practices of the waste industry, despite claims that 'incinerators... are the most regulated industrial process in the UK'.[5] In particular, two appalling failures of the EA to protect public health are the Byker and Edmonton incinerator ash scandals, both discovered and made public by concerned citizens.

At Byker the EA, the Health and Safety Executive, and Newcastle and North Tyneside Health Authority, were all blissfully unaware of the use of highly contaminated ash from the Byker incinerator on allotments, farms, school playing fields, bridle paths etc, for seven years.

While the EA may not have known what was going on at Byker, the evidence is that at Edmonton they knew full well of the extremely high levels of dioxin in its mixed ash, yet still did not stop the company from 'recycling' it.

The EA claims it is not its job to monitor the ash produced by incinerators, but rather 'the plant operator has a duty of care under the environment protection Act 1990 to ensure that the waste is transferred to a suitable disposal contractor. [This) contractor in turn has a duty of care under the Environment Protection Act 1990 to ensure it disposes of waste at a suitably licensed landfill'.[6]

Amazingly, while gathering evidence to prosecute the operators of the Byker plant and Newcastle City Council for spreading contaminated ash around Newcastle, the EA was simultaneously sitting on a working Ash Group working hand in hand and encouraging the use of similar mixed ash as road aggregate and in breeze block type building bricks at Edmonton.

David York, managing director of Ballast Phoenix, the company that handles the 150,000 tonnes of ash generated at Edmonton each year, admitted on *Newsnight* that tens of thousands of tonnes of the finer (fly) ash containing 'higher levels of dioxin' [than bottom ash] had been used in masonry blocks that went into houses. However, he dismissed the possibility of this presenting a health hazard when a house owner drilled into a block saying it will be 'a short, one-off exposure.'

You think that's bad? When asked about the toxicity of dioxin concentrations in the mixed Edmonton ash, environment minister Michael Meacher replied: 'The Environment Agency has no information on the toxicity of dioxin concentration in ash mixed before that date [August 2000].'[7]

But evidence shows that the EA had plenty of data on the levels of dioxin in Edmonton ash well before August 2000.

During the court action by North London Waste Ltd against activists of Greenpeace, a fax dated 24 July 1998 from Henry Cheung to Peter Montgomery the Environment Agency inspector responsible for regulating the Edmonton plant since 1996, was produced as evidence. This showed a lab analysis of the dioxin and furan levels in Edmonton's electrostatic precipitator (ESP) fly ash measured at 10,800ng/kg I-TEQ (nanograms per kilogram international toxic equivalent). The handwritten note reports a 14 to 1 ratio of bottom to fly ash produced and has a set of calculations showing the final levels of dioxins in the mixed ash as being 771ng/kg T-TEQ. These levels are much higher than the 'background levels' spoken of by minister Michael Meacher when he said `the Agency was informed by the operator that test results showed the dioxin levels of mixed ash to be close to background levels' (ie those found in normal urban soil)."

Furthermore, tests conducted in 1996 on ESP fly ash from UK plants were in the region of 6,600 and 31,100ng/kg TEQ (Cams *et al* 1996). Commissioned for a study by the EA itself in 1997 and 1999, AEA Technology wrote: 'For this study we use the range 6,600-31,100ng/kg TEQ to cover the variability found in UK plants'.

In other words, apart from any documentation on the public register such as the fax, the EA itself had indeed commissioned and published measurements from UK incinerators showing the extreme- ly high levels of dioxins in ESP fly ash long before August 2000. This data has been available from 1996, but, if we look at the public register at Edmonton we find it contains a 1993 ESP fly ash dioxin analysis, showing 3,600ng I-TEQ/kg levels."'

So Edmonton was mixing fly ash with bottom ash knowing full well it contained as much as 3,600ng/kg to 10,800ng/kg of dioxins. Tests conducted by *Newsnight* on a sample block made from 30 per cent Edmonton ash showed 343ng/kg. Therefore the level of dioxin contamination in this fine mixed ash would be in excess of 1100ng/kg, significantly higher than the 200ng/kg (peaking at 900ng/kg)left as result of Agent Orange in Vietnam, where they are still reporting birth defects and elevated dioxin levels in human tissue 30 years on.

Yet with all this knowledge, the EA not only didn't stop the practice, but moreamazingly granted Ballast Phoenix, the company using the mixed ash, a waste licensing exemption. And all this from the supposed regulatory body!

In addition, workers who handled the ash at Edmonton for Ballast Phoenix were not given any warning as to the toxicity of its contents or provided with protective clothing. Nor have any ever been tested for dioxin body levels.

Although the operators of EfW incinerators are given the overall responsibility of monitoring themselves and presenting the data to the EA, sometimes an independent company is entrusted with the taskof conducting or checking some of the data. Conveniently, a number of these independent companies also appear to be subsidiaries of the very companies they are supposed to be

checking. For example the Teesside site is managed by SITA. Its ash is tested by EUS Laboratories Ltd and AES Ltd. The air emissions are tested by AES Ltd, which is owned by Suez Lyonnaise des Faux - which owns SITA. [10]

It is highly probable that there were more companies than just Ballast Phoenix using mixed Edmonton ash. We know a seminar was held by Aggregate Industries (owners of Bardon Aggregates, who promoted the event) and hosted by Ballast Phoenix at the Edmonton incinerator in May 2000. There an official from the Department of the Environment, Transport & Regions (DETR) was proudly showing people around the ash storage facility proclaiming that as well as Edmonton, ash from the incinerators at South East London Combined Heat & Power plant (SELCHP), Tyseley (Birmingham), Dudley (Birmingham), Stoke and Cleveland (Teesside) had all been used in 'recycling'. So why was York so reluctant to tell *Newsnight* where the ash had been used, when it was obviously done with the approval of the DETR? Could it be that, given the data on the level of dioxin concentrations in the ash from the ALA Technology study, along with the company's own ash-testing data, York could be aware that perhaps the ash contains much higher levels of dioxin than he cares to admit - perhaps higher than his friends at EA would find acceptable:'

Whatever the reason, we know that incinerator ash has been used in Waltham Abbey by-pass with the approval of Essex County Council; car parks at Ford's Dagenham plant; Netherend Lane, Cradley Health in Birmingham; and in roads in Stoke, Dudley and Essex.

Incineration advocate Malcolm Chilton has claimed: 'Processed ash entering the construction market has dioxin concentrations of between 20-5O ng/kg, which falls within the range of 'naturally occurring soils.' Yet the reality is the dioxin level in soil is not naturally occurring at all. It is there as a result of emissions from incinerators. Even the UK government acknowledges that up to 85 per cent of the country's present dioxin contamination comes from incinerators.[11]

The operators of Edmonton no longer officially recycle fly ash. They claim they stopped doing this in August 2000 - conveniently just before the EA was to give evidence to a House of Commons Committee who had been informed of the mixed ash 'recycling' methods employed by SITA and London Waste Ltd, by a representative of the Public Interest Consultants.

However, there were plans to recycle nearly 60,000 tonnes of ash as 'assorted grades of aggregate every year' at SITA's Teesside plant, 'with support from Ballast Phoenix'.[12]

This is worrying because when asked if they had tested the bottom ash for dioxin [before its use as building material] Jon Garvey, former regional director of SITA based at their plant in Cleveland, replied: 'We haven't tested for dioxins, because they are assumed not to be there...'"[13]

Wherever these ashes have been used, be they roads, paths, playing fields, landfills, building blocks or anywhere else, can justifiably be considered a reservoir of dioxin that could be released at any time. This could take five, 10, 25 or 50 years - no human containment method lasts forever. It could even be released next year when a house holder begins a bit of DIY or unsuspecting workmen dig up the road to lay or repair cables or pipes and release clouds of dioxin/metal-containing dust when cutting through the roads surface.

Furthermore, it is clear that there are people working in the incineration industry who, in order to reduce companies' costs, have no qualms about spreading a compound estimated to be 167,000 times more toxic than cyanide on areas where children play and in people's homes.

What is bewildering is that the government requires the industry to spend millions of pounds on fitting anti-pollution devices to capture the most deadly toxins known to man - and then allows them to spread these around the open environment in roads and houses - and has two departments, the EA and DEFRA, actively encouraging them to do so - while the government itself subsidies the practice with hundreds of millions of pounds' worth of taxpayers' money.

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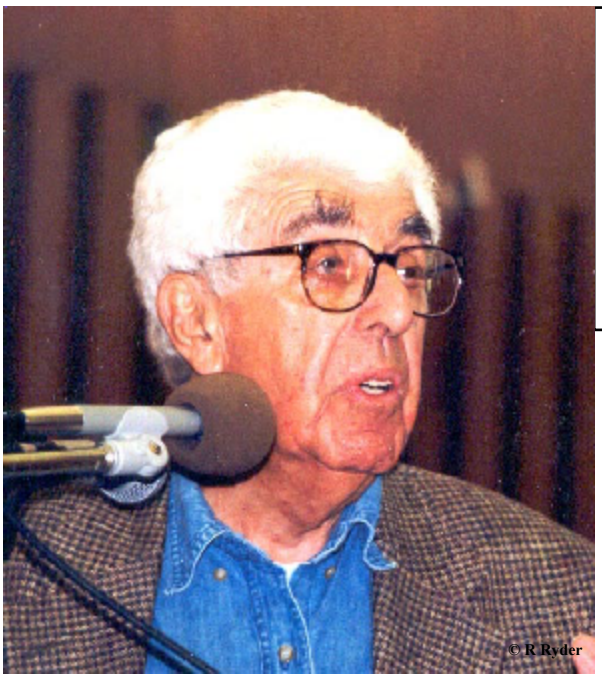
Source; No Smoke Without A Liar. *The Ecologist* Vol 31 No 8 October 2001

...it is clear that there are people working in the incineration industry who, in order to reduce companies' costs, have no qualms about spreading a compound estimated to be 167,000 times more toxic than cyanide on areas where children play and in people's homes.



Keynote Address at the Second Citizens Conference on Dioxin,
St. Louis, Missouri, July 30, 1994

The Political History of Dioxin

by Barry Commoner, Center for the Biology of Natural Systems

"It is fitting that this conference, which marks a momentous turning point in the notorious history of dioxin, should take place in St. Louis. It was not far from here that the threat of dioxin to the general public first became apparent—when a local dealer mixed dioxin-contaminated chemical waste into used oil and sprayed it in horse arenas, killing animals and sickening children. It was here that the enormous power of dioxin to disrupt our lives was demonstrated—when, for the first time, chemical contamination caused an entire town, Times Beach, to close down.

It was the local chemical company—Monsanto—that first began the ma nufacture ofpolychlorbiphenyl in Anniston, Alabama—a type of process, we now know, that inevitably produces dioxin-like substances as well. And the first unwitting discovery that such materials create dangerous industrial hazards to chemical workers was made in the early 1930s when most of the workers in the Monsanto plant became sick.

We also meet at a crucial time in the history of dioxin. I am convinced that 1994 will be seen as the year in which—despite every effort of the chemical industry and its journalistic allies to confuse and misinform us—the true dimensions of the ominous threat of dioxin to human health became known. The profound significance of its diverse attack on living things has now become clear: Dioxin and dioxin-like substances represent the most perilous chemical threat to the health and biological integrity of human beings and the environment.

The history of dioxin is a sordid story—of devastating sickness inflicted unawares, on chemical workers; of callous disregard for the impact of toxic wastes on the public; of denial after denial by the chemical industry; of the industry's repeated efforts to hide the facts about dioxin and, when these become known, to distort them. Our task here is to learn from this history—not only from the data generated by the rapidly growing list of scientific studies, and the crucial facts unearthed by grassroots activists—but also from the attempts of the chemical industry and its allies to distort them. We need to learn what must be done, now, not merely to diminish—but to end—the menace of dioxin and its many toxic cousins to life.

A good place to start is right here, in Missouri, with the events that led to the evacuation of Times Beach. On May 26, 1971, 2,000 gallons of what was supposed to be waste oil were sprayed on the soil in a nearby horse arena.

Three days later the arena was littered with dead birds; four days later three horses and the ringmaster were sick. By June, 29 horses, 11 cats and four dogs had died; in August the six-year-old daughter of one of the owners was admitted to St. Louis Children's Hospital with a severe kidney disorder.

Several other children and grown-ups reported less serious ailments. It was not until August 1974, after a foot of soil was removed and replaced. that the area could shelter healthy horses, pets, and birds. This was the beginning of a decade of study, controversy, and concern that climaxed when Times Beach was evacuated.

It took three years of work by state and US health laboratories to pin down the cause of all this sickness and death. Dioxin, at a level of 30-53 parts per million, was identified in samples of the arena soil. By then it was clear that the "waste oil" included chemical residue from a plant in Verona, Mo., that had been synthesizing trichlorophenol— an intermediate of 2,4,5-T—the herbicide "Agent Orange" that the US had sprayed in huge amounts in the war against Vietnam.

Note: The term 'dioxin' is used to connote the group of 210 similar substances—polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans. Certain types of polychlorbiphenyls (PCBs) have similar biological effects and are included among "dioxin-like" substances.

Why should a chemical plant designed to produce trichlorophenol also produce dioxin? The explanation lies in the special nature of manufacturing chemicals, which is very different from manufacturing anything else. When a car, let us say, is made, pieces of metal, glass, rubber and many other materials are assembled, but the matter of which is not changed. The waste is just some left-over wiring, paint fumes, or perhaps a cracked windshield, produced in amounts much less than the car itself and reducable by good housekeeping.

But the purpose of chemical manufacturing is to change matter, to rearrange atoms and make new molecules. In such a chemical reaction huge numbers of molecules jostle around, their constituent atoms assembling and disassembling in many different molecular arrangements. The chemist learns to favor the production of a particular molecule by controlling temperature, pressure, and other conditions and, more precisely, by introducing a catalyst. But the process is never perfect; some unwanted molecules that happen to be very stable and resist further transformation will persist as waste.

Dioxins are just such very stable compounds. In the production of trichlorophenol—or for that matter in most reactions involving organic (carbon-containing) chemicals and chlorine— dioxin is likely to be formed, and once formed, to persist as an unwanted waste. By its very nature, such waste—much of it toxic—is built into chemical manufacturing. Toxic waste is not simply a matter of poor housekeeping or bad management; it is an inescapable part of chlorine-based chemical production. Moreover, some of the industry's actual products, for example solvents, are themselves toxic, and many produce toxic substances—including dioxin—when an effort is made to dispose of them, especially by incineration.

Since the early 1970s a geat deal has been written about why dioxin should be so dangerous in such small amounts. But the fact that dioxin-like compounds—complex, highly chlorinated organic chemicals—are very toxic has been known, or should have been known, much earlier. Again, there is a Missouri connection, for the discovery was made in the 1930s in Monsanto's Anniston, Alabama, plant. Within a year after the plant opened, most of the workers had developed chloracne and a wide range of other symptoms.

In 1936 two Atlanta physicians published a case history in the Archives of Dermatology and Syphilology about one of the Monsanto workers described as:

"O.D., a Negro aged 26 [who] began work in the distillation of chlorinated diphenyl in April 1930." They reported that the patient had a severe case of chloracne and observed that the patient, even in December 1933, "complained of lassitude, loss of appetite and loss of libido." Some sense of the authors' ability to appreciate the significance of these symptoms, later shown to be characteristic of dioxin poisoning, can be gained from their additional comment;" "His complaint of lassitude was not borne out by anything more than the usual temperament of the Negro toward work..."

We have heard the same sorry tale very often since then: "The only human disease attributable to dioxin is chloracne." But step by distressing step, the full range of the devastating effect of dioxin on people has confirmed the reality of O.D.'s symptoms, and much more.

The carcinogenic effect of dioxin played a key role in the evacuation of Times Beach and in the general assessment of its risk. In 1978, the first comprehensive animal tests showed that rats and mice raised on a dioxin-containing diet developed an excess incidence of cancer. In 1985 the EPA issued its first formal cancer risk assessment of dioxin. It concluded, from the animal tests and consideration of the possible mechanisms of chemical induction of cancer, that a dosage of 0.006 picograms per kilograms of body weight per day—which in an adult person amounts to a daily intake of 14 trillionths of an ounce—would represent a lifetime cancer risk of one in a million. This singled out dioxin as the most potent synthetic carcinogenic chemical. EPA estimated that people would be exposed to the one-per-million risk if they lived near soil contaminated at the level of one part per billion. When soil in Times Beach was found to considerably exceed this level, the EPA decided to evacuate the town.

Apart from the terrible disruption of the lives of the people of Times Beach, what does this decision tell us? Why should EPA and other government agencies try to establish such a cut-off level—a dividing point between remedial action and doing nothing? I suppose that one reason is simply bureaucratic timidity—a way of avoiding a decision based on personal judgment; it is safer, for the bureaucrat if not for the rest of us, to rely instead on some number, arrived at by "objective science" rather than by responsible human beings.

But there is much more to the notion of a "safe" level of exposure than protecting human health or environmental quality. For Syntex (USA) Inc.—the company responsible for the dioxin clean-up costs in Missouri—it is a matter of money. In 1986 Syntex staff members published a graph showing the relation between different clean-up standards and the expected costs of achieving them in the Missouri dioxin-contaminated sites. It showed, for example, that if the soil-contamination standard of one part per billion were relaxed to 10 parts per billion, Syntex would need to spend 65% less on the clean-up.

The Syntex people proposed that the 1985 EPA risk assessment should be sharply reduced. This would not only save Syntex money, it would also reduce the need to clean up many superfund sites; it would improve the environmental acceptability of the incinerators; it would weaken the claims of the veterans who were exposed to Agent Orange in Vietnam, and it would affect the outcome of numerous court cases. EPA did not refute the Syntex proposal, adhering to the Reaganesque line that environmental hazards must be balanced against thecost of remedying them.

It is no wonder, then that polluters declared open season on dioxin risk estimates. Their techniques varied. Some of the most imaginative efforts were made by companies that built incinerators— major sources of environmental dioxin.

They usually accepted the EPA's estimate of dioxin's high carcinogenic potency, but tried to get around it by showing that the dioxin would be so diluted once it left the incinerator smokestack that the people exposed would fall within the one-in-a-million cancer risk standard of "acceptability."

The prize for the most imaginative example of dioxin detoxification by dilution goes to the author of the environmental impact statement for the proposed—and still not built- trash-burning incinerator at the Brooklyn Navy Yard in New York. Here is his prize-winning idea: Dioxin emitted into the air by the incinerator would fall to the ground and there become mixed into the upper 10 cm of the soil. This would greatly reduce the dioxin, so that when it finally came into contact with the people of Brooklyn, it would result in the risk—magically—of just under one in a million. Unfortunately, most of Brooklyn is not covered with soil, but with asphalt and houses.

Perhaps embarrassed by such ludicrous efforts to evade the consequences of its 1985 cancer risk assessment, EPA decided to make life easier for the industry's inventive risk assessors by revising the risk assessment itself.

Was dioxin really so potent that absorbing only 14 trillionths of an ounce would carry the one-in-a-million lifetime cancer risk? With the director of EPA's Office of Research and Development as chairman, a Workgroup of EPA staff reviewed the 1985 document and re-examined its data and reasoning. They had little to say about the data and concentrated their attention on the fact that there were several diffcrent theories about how chemicals like dioxin might cause cancer. Most of the alternative theories predicted a dioxin cancer potency much lower than the 1985 risk assessment and were incompatible with the theory that guided it. If these alternative theories were right, then the assessment's theory had to be wrong.

We need to learn what must be done, now, not merely to diminish—but to end—the menace of dioxin and its many toxic cousins to life.

What to do? In spectacular intellectual feat (its originality seriously compromised by the fact that it had been suggested by a recent manifesto from the Reagan/Bush Office of Management and Budget), the Workgroup decided that the "scientifically sound" thing to do was to average the potency values indicated by the different theories. Because the high potency value of the 1985 assessment's theory was outweighed by the more numerous low-potency theories, the average turned out to be 16 times less stringent than the 1985 risk assessment.

When the Workgroup's draft was sent out for review in 1987, I was among those invited to respond. (Such strange things sometimes happen when a bureaucracy tries to navigate the risky passage between science and politics.) Tom Webster and I prepared a detailed point-by-point criticism of the Workgroup report.

But what really counted was a much simpler point—which I had the opportunity to make in a speech to the entire Washington EPA staff in January 1988 (another strange event): If the low-potency theories are right, then the original high-potency theory is wrong, and vice versa—a situation that can hardly be corrected by averaging their mutually contradictory results.

This and other criticism of the Workgroup's 1987 attempt to revise the 1985 risk assessment had an effect: A revised draft was issued that scrapped the first one. Now the Workgroup decided that the low-potency models were inadequate and accepted a version of the original high-potency model as the basis for its analysis. Then, without any factual evidence to support it, the Workgroup nevertheless decided that the original 1985 risk assessment "may be an overestimate," although the "scientific data do not permit an estimate of the extent of the overestimate." So, having decided that the original potency estimate was too high, and not knowing by how much (which logically could be only 1% of its original value—a difference totally lost in the range of uncertainty of the estimate)—the Workgroup concluded that the true value is—once again—exactly 16 times lower than the 1985 estimate. That the same decision for a 16-fold reduction of dioxin's cancer potency was based on two sets of mutually contradictory reasons suggested that the result was unencumbered by factual scientific analysis.

Stated a little less politely, I would credit the Workgroup with a new, highly innovative approach to the evaluation of dioxin's toxicity: fact-free detoxification. All this became clear in public hearings on the draft Workgroup report, with the result that it died a quiet death

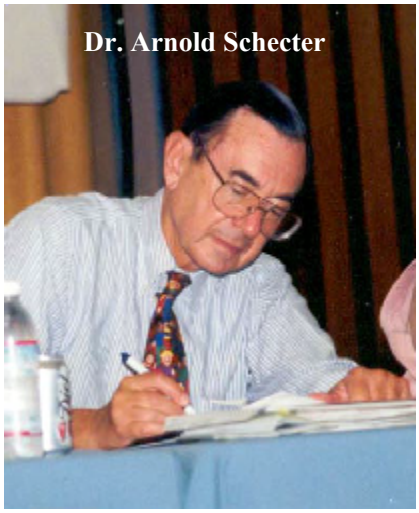
somewhere in the EPA bureaucracy. The 1985 cancer risk assessment survived.

Thus far, the attempts to downgrade the EPA's 1985 risk assessment had avoided a direct challenge to the data on which it was based—chiefly, the results of a rat feeding test carried out by a researcher at the Dow Chemical Company. In 1986 it was confirmed that paper mills using chlorine bleach produced dioxin in their waste water at levels that would exceed a standard based on the 1985 risk assessment. This was the result of chlorine reacting with chemical constituents of wood. Seeking to avoid proposed regulations that would restrict the use of chlorine, the paper industry decided to challenge the results of the Dow rat test. They borrowed the original slides from Dow and assembled a panel of "independent" toxicologists who examined each slide and decided for themselves whether cancer was present or not. Since they were not animous in their decisions, the cancer frequency was decided by majority vote. This reduced the original potency figure by half—hardly a significant change. And once more, under this new assault—detoxification by recount—the 1985 risk assessment survived. Nevertheless, the paper companies asked EPA to "rethink" it.

This brings us to October 1990 and a place called the Banbury Center in Long Island. There, under the sponsorship of EPA and the Chlorine Institute—an industry group—toxicologists and biochemists were convened to consider the "Biological Basis for Risk Assessment of Dioxins and Related Compounds." The purpose of the conference was to review new data about how dioxin caused cancer in order to provide a "scientific" basis for a new risk assessment. The "new data" were studies that actually went back to the 1970s. They showed that dioxin's effects were exerted through a receptor—a particular protein in animal cells called Ah—that tightly bound dioxin and facilitated its action, through the cell's genetic system, on protein synthesis.

At Banbury, the relationship between the science and politics of dioxin, until then a kind of cautious flirtation, came into full flower. On the scientific side the conference made a lot of sense, for it came on the heels of a rapid expansion of what was known about the biological effects of dioxin and dioxin-like compounds such as PCBs. The participants agreed that most, if not all, of these newly recognized effects (actually many of them were seen earlier in industrial exposures such as "O.D."s) were mediated through dioxin's primary interaction with the Ah receptor. A few participants proposed an additional, highly controversial conclusion: that the effect of a chemical operating through a receptor must have a threshold, a dose below which there would be no effect. They also claimed that the existence of a threshold would justify downgrading the dioxin potency, but many other participants disagreed.

The latter were surprised to learn, from news stories based on a press release prepared by a conference participant hired by the Chlorine Institute (but not originally identified in that way), that there was a consensus—that the dioxin risk should be downgraded.


Dr. Arnold Schecter

The EPA participants in the Banbury Conference hurried back to Washington with news that prompted the Administrator, William K. Reilly, to predict that a new reassessment would in fact reduce the dioxin risk. This set the stage for the latest chapter in dioxin's sordid history: a new, Banbury-inspired, re-evaluation of the 1985 risk assessment. This has now been completed and is scheduled for release in September. But we already know what it will say, thanks to a leak of the report's conclusion a few weeks ago. The new attempt to downgrade the dioxin hazard, like all the earlier ones, has failed. But in failing, it has not simply confirmed the important but narrow result of the 1985 risk assessment that dioxin is an enormously potent carcinogen. It has also greatly expanded the range and biological impact of dioxin's effects, at levels of exposure already experienced by the entire US population.

If, as a skeptic like myself might conclude, the Banbury Conference was set up to urge EPA to find new "scientific" reasons for downgrading dioxin's cancer potency, the planners made two serious tactical mistakes. First, by concentrating attention on the receptor theory, the Conference validated the growing, but until then largely unconnected evidence, about the non-cancer effects of dioxin, such as hormonal and developmental disruptions, at concentrations even lower than those that induce cancer. The second mistake arose out of the threshold concept itself, for it raised the question of whether the dioxin level carried in people's bodies was already at or above the threshold. If so, added exposure would then be expected to have an effect on the incidence of cancer and other disease

regardless of whether or not the threshold theory was correct.

The threshold proponents proposed that a dietary intake ofone to three picograms per kilogram per day would be "safe." Unfortunately, EPA studies of the body burden of dioxin showed that the US average has already reached that level. Earlier, Tom Webster and I had shown that this level indicated a cancer risk of 330 per million. Apparently Americans are sufficiently exposed to some very general source of dioxin to put us all well above the "acceptable" cancer risk of one in a million, and within range of its numerous other harmful effects. That source, according to the forthcoming EPA report, is chiefly food.

Dr. Arnold Schecter, who has so courageously pioneered in this area, has recently completed a study of the dioxin content of US food, which he has kindly allowed me to share with you. His basic conclusion is that a typical daily diet delivers between 0.3 to 3.0 picograms per kilogram of body weight (a level that represents a lifetime cancer risk of 50-500 per million). The new EPA risk assessment reports that if dioxin-like types of PCB are included in the assessment, the average US intake is 3-6 picograms per kilogram per day, representing a cancer risk of 500 to 1,000 per million. As the new EPA report states—rather delicately—"the weight of the evidence suggests concern for the impact of these chemicals on humans at or near current background levels."

Stated more simply, the situation is this: The general spread of dioxin and dioxin-like chemicals in the US environment has already exposed the entire population to levels of these extremely toxic substances that are expected to cause a number of serious health effects. These include an average risk of cancer of 100 or more per million in the entire US population—100 times greater than the risk standard that has triggered EPA remedial action, for example at Times Beach the EPA document also acknowledges that the newly appreciated hazards of dioxin go far beyond the risk of cancer. At or near the observed levels of dioxin and dioxin-like compounds in the US population, the expected non-cancer effects include:

*disruption of endocrine hormone systems, especially those related to sexual development;

*disruption of critical stages of embryonic development, for example of the nervous system;

*damage to the developing immune system, leading to increased susceptibility to infectious diseases.

These are *intergenerational defects,* they are imprinted for life on the developing fetus by the effect of dioxin on the mother and sometimes the father. In its recent Seventh Biennial Report on the environmental impact of persistent toxic substances such as dioxin on the Great Lakes, the International Joint Commission has bluntly confronted the catastrophic implication of this threat, stating:

Surely, there can be no more compelling self-interest to force us to come to grips with this problem than the spectre of damaging the integrity of our species and its entire environment.

Why should such biologically powerful agents arise from the normal activities of the chemical industry? Why should ordinary commercial products like PCB, or a routine by-product of numerous chemical industry processes like dioxin act in the body as though they were hormones?

Dioxin and dioxin-like chemicals have become widely known as "environmental hormones" because they enter into the complex network of natural hormones that govern sexual development and other embryonic processes—and disrupt them. They are man-made chemicals that, present in only miniscule amounts, can powerfully alter the natural biochemical processes that determine how animals develop, grow, and behave. However, dioxin is not in fact a hormone, a term that is properly restricted to chemical substances that are produced inside the cells of living things, and not in the reactors of the chemical industry. There is a crucial molecular difference between dioxin and hormones. Dioxin is distinctively characterized by its chlorine atoms, which, when linked to particular carbon atoms in its molecular structure, give rise to dioxin's powerful toxic properties. In contrast, no natural hormone is chlorinated.

What should we call a man-made substance that is not a hormone but acts like one—inducing powerful, often destructive changes in biochemical processes? We already have a generic name for such substances, chemicals that are designed to powerfully modify cellular chemistry, but in useful ways: pharmaceutical drugs. It makes more sense, I believe, to call dioxin an "environmental drug" than an "environmental hormone," for it helps to explain why dioxins and dioxin-like substances are so menacing to human health and environmental quality.

Unlike ordinary pharmaceutical drugs, dioxins were not subjected to years of testing in the laboratory, and in patients, in order to make sure that they do more good than harm. Unlike

Dioxin and its chemical cousins have been administered, wholesale, to everyone...whether they want it or not; and certainly not under the watchful care of a physician. Thus, like the pharmaceutical companies, the entire chemical industry is also in the drug business—but in a wildly unregulated and extremely dangerous way.

ordinary drugs, they are not prescribed by a physician for the use of an individual patient in order to counter a previously diagnosed ailment. Instead, dioxin and dioxin-like substances were massively released into the environment long before their enormous biological powers were studied, let alone understood.

Dioxin and its chemical cousins have been administered, wholesale, to everyone—whether old, young, or not yet born; whether well or sick; whether they want it or not; and certainly not under the watchful care of a physician.

Thus, like the pharmaceutical companies, the entire chemical industry is also in the drug business, but in a wildly unregulated and extremely dangerous way.

How can we bring this rogue sector of the chemical industry under control and protect ourselves from its powerful threats? The world owes the International Joint Commission (IJC), its staff and scientific consultants (and indeed, Greenpeace and the other grassroots organizations that have participated in this work) a huge debt of gratitude for their efforts to understand this issue and develop constructive ways of dealing with it. In its most recent (Seventh) Biennial Report, the IJC spells out its key conclusions:

"Persistent toxic substances are too dangerous to the biosphere and to humans to permit their release in any quantity, and ."All persistent toxic substances are dangerous to the environment, deleterious to the human condition, and can no longer be tolerated in the ecosystem, whether or not unassailable scientific proof of acute or chronic damage is universally accepted.

"The production and release of these substances into the environment must, therefore, be considered contrary to the Agreement legally, unsupportable ecologically and dangerous to the health generally. Above all, they are ethically and morally unacceptable. The limits on allowable quantities of these substances entering the environment must be effectively zero, and the primary means to achieve zero should be the prevention of their production, use, and release rather than their subsequent removal."

Clearly, this means that changes must be made in the chemical industry to alter or eliminate the processes that give rise to dioxins and dioxin-like substances. These dangerous chemicals can be formed in many of the industry's organo-chlorine reactions, or whenever the products of these reactions, such as PVC, are burned. What needs to be done about that has also been made clear in an earlier (the Sixth) IJC Report:

We know that when chlorine is used as a feedstock in a manufacturing process, one cannot necessarily predict or control which chlorinated organics will result, and in what quantity. Accordingly, the Commission concludes that the use of chlorine and its compounds should be avoided in the manufacturing process.

This proposal and the campaigns developed by Greenpeace and other environmental organizations, have already launched the issue of "banning chlorine" into the domain of public debate. We have already heard the replies from the industry and its friends. One argument, advanced by the chemist G.W. Gribble, is that "[C]hlorine is as natural to our world as carbon, oxygen, and hydrogen." Of course that is true, but the point is that chlorinated organic compounds are not so natural. They are rare in living things; only about 600 such substances have been identified, compared with tens of thousands of different organic substances made by living things that are not chlorinated.

Moreover, not a single chlorinated organic compound has been identified as natural in mammals.

In Gribble's compilation of 611 chlorinated (and other halogenated organic) compounds produced by living things, there are numerous examples from fungi, higher plants, algae, sponges, jellyfish, worms, and other marine animals.

There is exactly one entry under mammals—a chlorinated compound found in the urine of a group of cattle. Recently I called the author of the paper cited by Gribble, Dr. K-C Luk. He told me that he had no way of knowing whether the chlorinated compound was a natural metabolic product or was acquired by the cattle from the environment. Given the huge amount of unnatural chlorinated compounds that beset modern agriculture, I would bet on the environment.

In fact, these data are very illuminating. It looks as though in the early evolution of living things, a few organochlorine compounds were included in their biochemical systems. But when the first mammals—or possibly vertebrates—emerged, chlorine was abruptly excluded from this new form of life. As a result, chlorinated organic compounds like dioxin are incompatible with the distinctively complex hormonal systems and developmental processes that are characteristic of vertebrates, especially mammals. The chemical industry has violated this biological taboo, and we are all paying dearly for this transgression—for, in the words of the IJC, it has created "the spectre of damaging the integrity of our own species [and probably of other vertebrates as well] and its own environment."

The industry's chief defense against shutting down the use of chlorine in chemical manufacturing is that it is essential to the manufacturing of most of its products (true), which are in turn essential to most other industries and agriculture (not so true). It is true that

synthetic organic chemicals—plastics, pesticides, detergents, and solvents—have deeply penetrated the modern world. This was done not so much by creating new industries as it was by taking over existing forms of production. After all, we did have food before synthetic pesticides, and there was furniture, flooring, and paint long before plastics. In fact, as pointed out by one of the leaders in the development of the petrochemical industry, Lord Beeching, it grew through a virulent form of industrial imperialism:

Instead of producing known products to satisfy existing industrial needs, it [the petrochemical industry] is, increasingly, producing new forms of matter which not only replace the materials used by existing industries, but which cause extension and modification of those industries ... To an increasing degree it forces existing industries to adapt themselves to use its products.

I believe that this is where the industry is most vulnerable. As the source of persistent dangerously toxic substances, the chemical industry must change its methods of production—and where necessary its products—beginning with the elimination of chlorine. Of course, the industry will use its enormous wealth and political power to resist such a far-reaching change. But some of its equally powerful corporate customers—paper mills, electronics manufacturers, and the food industry—may be less rigid. Yes, they have been invaded by the chemical industry's products that they use. But with those products have come the built-in toxic accompaniments and the economic liability for their damage.

We now know, for example, that the US population is exposed to dioxin not so much from the chemical industry's direct emissions, but chiefly from food that has been contaminated with dioxin entering the food-chain, especially beef and dairy products. These industries, already suffering from reduced consumption to avoid fat and cholesterol, are now likely to be hit once more, this time by the dioxin problem. Sooner or later, to protect their own economic interests—properly encouraged by grassroots activists—they will use their own corporate power to help persuade the chemical industry to change its ways.

Already the paper industry has begun to make plans for ending chlorine bleaching processes. There are even whispers from the chemical industry itself that they have got the message; very quietly, I have heard, their chemists are looking for ways to take chlorine out of their processes.

These are some of the reasons why we are at a turning point not only in the history of dioxin, but of the chemical industry itself. What has brought us to this point, I am convinced, is the environmental movement—at its powerful grassroots: the numerous community campaigns against trash-burning incinerators; the valiant battles against hazardous waste incinerators at East Liverpool and Jacksonville; the struggles at Times Beach and Love Canal; the campaign for justice for the veterans exposed to Agent Orange. Let this conference, here in the place where it all began, be the start of new campaigns and new victories—for the sake of the environment and the people who live in it. END

While it is indisputable that the chemical industry has brought society many benefits, it is leaving a terrible legacy for future generation to contend with. Its processes and irresponsible 'dilute and disperse' methods of waste management continue to poison the food chain, and each and everyone of us daily resulting in a lot more than a "nasty skin complaint."

ten Tusscher *et al.*, 2007. **Perinatal dioxin exposure, cytochrome P-450 activity, liver functions and thyroid hormones at follow-up after 7–12 years.** *Chemosphere.* Article in Press.

Abstract

Objectives: Prenatal and lactational exposure to Dutch ‘‘background’’ dioxin levels may cause health effects spanning many years. In addition, perinatal studies have shown a relationship between dioxin exposure and thyroid disturbance. To assess the later health effects of prenatal and lactational dioxin exposure on liver function we measured plasma ALAT and ASAT levels amongst our longitudinal cohort, as was done perinatally and at 2½ years. The children underwent a caffeine loading test to determine CYP1A2 activity. To assess the later effects on thyroid function we measured plasma TSH and FT4.

Study design: A longitudinal cohort of 37 healthy children (age 7–12, mean 8.2 years), with documented prenatal and lactational dioxin exposure, ingested 3 mg caffeine/kg BW 6 h prior to blood withdrawal. Paraxanthine/caffeine molar ratio, ALAT, ASAT, TSH and FT4 were determined in venous blood.

Results: Linear regression of ASAT and ALAT revealed no relation with prenatal and lactational dioxin exposure. No correlation was found between the paraxanthine/caffeine molar ratio and prenatal and lactational dioxin exposure. Linear regression of TSH and FT4 revealed no relation with prenatal and lactational dioxin exposure.

Conclusion: This follow-up has shown a normalisation of previously abnormal ALAT and ASAT levels, indicating a transient effect. CYP1A2 activity, measured by means of a caffeine-loading test, revealed no correlation with the prenatal and lactational exposures. A normalisation of previously abnormal thyroid hormone homeostasis was seen, also possibly indicating a transient effect. This study provides new data on long-term follow-up after perinatal dioxin exposure to

Meijer, L., Weiss, J., Van Velzen, M., Brouwer, A., Bergman, A., Sauer, P., 2008. **Serum Concentrations of Neutral and Phenolic Organohalogens in Pregnant Women and Some of Their Infants in The Netherlands.** *Environmental Science & Technology.* Article in Press.

Abstract

As part of a large European Union (EU)-funded comparative toxicology and human epidemiology study, EU-Compare, a selection of organohalogen compounds (OHCs) was analyzed in maternal serum, collected at the 35th week of pregnancy, and in cord serum of a number of their infants to determine maternal concentrations and to investigate the extent of transplacental transfer of these compounds. Eight neutral OHCs were analyzed: one polychlorinated biphenyl (PCB: CB-153), 4,4 ’ DDE, five polybrominated diphenyl ethers (PBDEs: BDE-47,BDE-99, BDE- 100,BDE-153,and BDE-154), and hexabromocyclododecane (HBCDD). Five phenolic OHCs were analyzed: three hydroxylated PCBs (4OH-CB-107, 4OH-CB- 146, and 4OH-CB-187), one hydroxylated PBDE (6OH-BDE-47), and pentachlorophenol (PCP). All OHCs, except 6OH-BDE-47, were present in maternal and cord serum. The historically identified OHCs showed the highest concentration: 4,4'-DDE (median value 89 ng/g lipid in maternal serum and 68 ng/g lipid in cord serum) and PCP (median value 970 pg/g serum in maternal serum and 1500 pg/g serum in cord serum). HBCDD and the PBDEs were present at much lower concentrations. We conclude that OHCs are present in the serum of pregnant women, and all compounds tested are transferred over the placenta. Because transfer is occurring at a critical stage of infant development, investigation of the health impact is urgent.

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Transplacental transfer was observed for all the neutral and phenolic OHCs analyzed in this cohort. The ratios ranged between 0.5 and 1.0. These ratios are in accordance to transplacental transfer ratios observed in similar cohorts in other industrialized countries (Table 4). The number of cord serum samples analyzed and the percentage of BFR concentrations above the LOD and LOQ in cord serum was low, especially for HBCDD. Therefore, the calculated transfer ratios for the BFRs should be considered with some caution.

In this paper serum concentrations of eight neutral and five phenolic OHCs in pregnant women and some of their infants was presented. Except for the phenolic 6OH-BDE-47, all other neutral and phenolic OHCs could be detected in maternal and cord serum. No difference in serum BFR concentration between 20th and 35th week of pregnancy was observed. All the neutral and phenolic OHCs present in the serum of pregnant women were transferred over the placenta to the infants, including the BFRs. Our study indicates that the human fetus is exposed to a large number of different environmental contaminants, including the historically identified OHCs as well as the more recently used BFRs. Given the negative effects of exposure to these compounds in animals, and in line with earlier found negative effects of comparable compounds like PCBs, more health studies are needed to investigate the possible influence of these compounds of the human fetus.

Shellart, N., Reits, D., 2008. **Influences of perinatal dioxin load to visual motion and oddball stimuli examined with an EEG and MEG analysis.** *Clinical Neurophysiology.* Article in Press. doi:10.1016/j.clinph.2008.03.002

Abstract
Objective: With MEG and EEG the effect of perinatal dioxin load of 38 healthy 7- to 12-year-old children was studied to assess possible disturbances of visual development.
Methods: Latencies and amplitudes of the motion (N2 with subcomponents) and oddball responses (N200 and P3b) were analysed after age correction.
Results: With increasing load, latencies increased and the amplitudes of the oddball components tended to be reduced. The latency increase between the high- and low-loaded children was about 13 ms ($P < 0.004$) and the oddball response showed an amplitude decrease of 12% ($P = 0.009$).
Conclusions: It may be concluded that, during the end-80s/early-90s, exposure to background levels in industrialized regions seems to have resulted in small underdevelopment or damage to visual motion processing and visual cognition.
Significance: Since dioxin pollution by incinerators still exists in many regions in developing countries and also still, although at a smaller scale, in the industrialized world, perinatal loads of similar magnitude and possibly more as measured in this study may occur and as a consequence might affect the developing brain.
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The amplitude and latency effects of the high-loaded children are smaller than those of the patients and children with cognitive complaints. The high-loaded group has a prenatal exposure 1.8 times that of the low-loaded group. Since the effects are underestimated, the actual effect upon the high-loaded group can be nearly twice as much. This strengthens our indications that a high perinatal dioxin exposure is assumed to have resulted in some neural underdevelopment and/or damage. Since dioxins and PCDFs have short half-lives (Leung *et al.,* 2006), these disorders probably occur in the first years of life.
To our knowledge no other perinatal dioxin study has been performed using EEG or MEG, but a PCB study resulted in similar effects on the event-related potentials (Chen and Hsu, 1994).
Despite various molecular studies about dioxin poisoning, the mechanism of how dioxins affect latency and amplitude of neural responses is still unknown or speculative. Consequently, at present, we confine with the conclusion that there are indications of injurious interference in cerebral function with respect to visual cognitive and motion processing after perinatal exposure to background levels of dioxins at that time.

Aristizábal *et al.,* 2008. Baseline levels of dioxin and furan emissions from waste thermal treatment in Colombia. *Chemosphere.* Article in Press. doi:10.1016/j.*chemosphere.*2007.03.078

Abstract
Background data of polychlorinated dibenzodioxin and dibenzofuran (PCDD/Fs) emissions from the incineration sector in Colombia are presented. Monitoring was carried out during a two-year period, 2003–2005. Twelve plants were sampled for stack gas emissions of dioxins and total solid particulate (TSP). Additionally, PCDD/Fs in several fly ash samples were analyzed. Most incinerators burned industrial refuse materials and medical residues. A wide range of PCDD/Fs emission levels were found. In particular, levels ranging from 6.9 to 343.8 ng I- TEQ/Nm3 were determined in plants without any air pollution control system (APCS). In contrast, 0.5–39.2 ng I-TEQ/Nm3 levels were found in plants with APCS while 8.5–67.5 ng I-TEQ/g were measured in fly ash samples. TSP values ranged from 14 to 448 mg/Nm3. This study also evaluated the impact of implementing different control systems in an incinerator. Finally, for comparison purposes several samples were analyzed by both high resolution gas chromatography coupled to high resolution mass spectrometry (HRGC- HRMS) and high resolution gas chromatography coupled to ion-trap low resolution mass spectrometry–mass spectrometry (HRGC-IT LRMS/MS). Overall, I-TEQ values deviated about 20–30% between both techniques.
[from body of text] Conclusions
Background PCDD/F emissions and TSP obtained from this study reveal high contribution of emissions from medical and industrial waste incinerators. The concentrations from plants without any APCS are significantly higher than levels monitored in plants equipped with APCS. However, only two plants with APCS achieved the limit values set in the Regulation (Resolución 0886, 2004).
…
In addition, the management of fly ash from incinerators should be a major concern prior to final disposal since they also contain high dioxin concentrations. Adequate and safe disposition of fly ash is necessary due to the potential high risks to human health and the environment.

Journal of Hazardous Materials Volume 160, Issue 1 , 15 December 2008, Pages 37-44
doi:10.1016/j.jhazmat.2008.02.077 Polychlorinated dibenzo-*p*-dioxins/dibenzofuran mass distribution in both start-up and normal condition in the whole municipal solid waste incinerator

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Abstract
Although many researches focused on the polychlorinated dibenzo-*p*- dioxins/dibenzofuran (PCDD/F) emissions from stack, in the bottom ash and in the surrounding environment, researches focused on PCDD/F mass distributions in the whole incineration plant have seldom been addressed. This study determined PCDD/F emissions in the whole plant. A high-resolution gas chromatograph/high- resolution mass spectrometer was utilized for analyzing 17 PCDD/F species. Experimental results displayed that PCDD/Fs were formed during fly ash from super heater (SH), economizer (EC), semi-dryer absorber (SDA) and fabric filter (FF) was transferred to fly ash pit. Mass distribution ratios of PCDD/Fs in g I-TEQ (Toxicity Equivalency Quantity) per week from stack, SH, EC, SDA, FF, generation and bottom residue (BR) in start-up operations were 14.6%, 0.1%, 8.3%, 1.0%, 41.7%, 33.4% and 0.9%, respectively. Above results indicated that main PCDD/F source in the MSWI was from fly ash. However, the fly ash is easily controlled and PCDD/F emitted from stack flue gases will be difficult to be handled. Therefore, we should pay more attention on PCDD/F emission from flue gases especially from start-up procedure. Besides, fly ash should be controlled by sodium hypophosphite before being landfilled. MSWI did require further detoxification treatments for the solid residues and flue gases.

This new study suggests that dioxin emissions from incinerators are matched in their toxicity by another, related class of chemicals: chlorinated and brominated polycyclic aromatic hydrocarbons.

Occurrence and Profiles of Chlorinated and Brominated Polycyclic Aromatic Hydrocarbons in Waste Incinerators. Yuichi Horii,† Gon Ok,‡ Takeshi Ohura,§ and Kurunthachalam Kannan*†
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Abstract:
Chlorinated polycyclic aromatic hydrocarbons (ClPAHs) have been reported to occur in urban air. Nevertheless, sources of ClPAHs in urban air have not been studied, due to the lack of appropriate analytical methods and standards. In this study, we measured concentrations of 20 ClPAHs and 11 brominated PAHs (BrPAHs) in fly ash and bottom ash from 11 municipal/hazardous/industrial waste incinerators, using analytical standards synthesized in our laboratory. Concentrations of total ClPAHs and BrPAHs in ash samples ranged from <0.06 to 6990 ng/g and from <0.14 to 1235 ng/g, respectively. The concentrations of ClPAHs were approximately 100-fold higher than the concentrations of BrPAHs. 6-ClBaP and 1-ClPyr were the dominant compounds in fly ash samples. The profiles of halogenated PAHs were similar to the profiles reported previously for urban air. 1-BrPyr was the predominant BrPAH in fly ash. Concentrations of 6-ClBaP, 9,10-Cl2Phe, 9-ClAnt, and 6-BrBaP in fly ash were significantly correlated with the corresponding parent PAH concentrations. Significant correlation between ÓClPAH and ÓPAH concentrations suggests that direct chlorination of parent PAHs is the mechanism of formation of ClPAHs during incineration of wastes; nevertheless, a comparable correlation was not found for BrPAHs. There was no significant correlation between the capacity and temperature of an incinerator and the concentrations of ÓCl-/BrPAHs in ash samples, although lower concentrations of all halogenated PAHs were found in stoker-type incinerators than in fixed grate-type incinerators. Toxicity equivalency quotients (TEQs) for ClPAHs in ash samples were calculated with ClPAH potencies. Average TEQ concentrations of ClPAHs in fly ash and bottom ash were15800 pg-TEQ/g and 67 pg-TEQ/g, respectively. Our results suggest that the extent of dioxin-like toxicity contributed by ClPAHs in ash generated during waste incineration is similar to that reported previously for dioxins. Waste incineration is an important source of Cl-/BrPAHs in the urban atmosphere.

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