The Vinyl Institute
A Division of The Society of The Plastics Industry, Inc.

Robert H. Burnett
Executive Director

August 12, 1994

TO: VI Executive Board

RE: Greenpeace Attack

Enclosed for your reading pleasure is the latest assault by our friends at Greenpeace - "Achieving Zero Dioxin". By any measure, it is the slickest and most professional looking document they have produced, but as usual, riddled with inaccuracies and outright false statements.

As a top priority, we have asked Edward Howard and Company along with several member company representatives to take this apart and highlight the PVC-specific areas that require refutation. The format and distribution of our rebuttal will be similar to that used for the "Truth About Vinyl" piece which is our response to the Greenpeace handout at NPE entitled "No Future for PVC".

We anticipate having this for final review and distribution no later than Monday, August 22.

Bob

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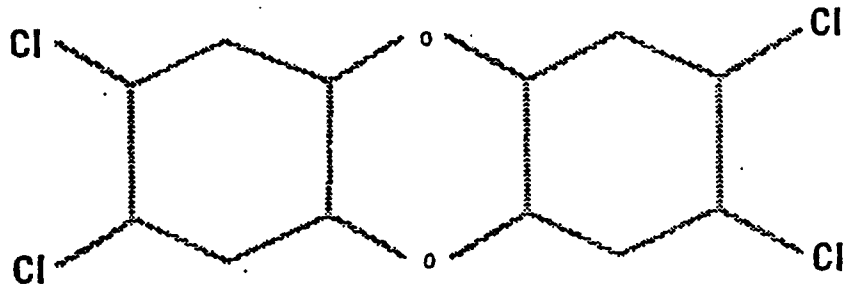
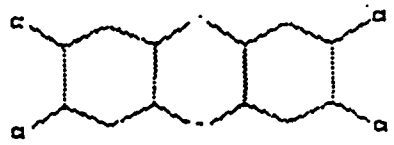
ACHIEVING ZERO DIOXIN



An Emergency Strategy for Dioxin Elimination



GREENPEACE

ACHIEVING ZERO DIOXIN

An Emergency Strategy for Dioxin Elimination

by Joe Thornton

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Thanks to Rick Hind, Lisa Finaldi, Jack Weinberg, Scott Brown, Lynn Thorp, and Tom Webster for their review and assistance in the preparation of this document, and to the 4 million Greenpeace supporters who made this report possible.

Printed on totally chlorine-free paper.

July 1994

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SUMMARY OF FINDINGS

EPA's scientific reassessment of dioxin indicates that dioxin pollution poses a large-scale, long-term threat to public health and the environment. Although the reassessment has not yet been released in final form, EPA documents associated with the project -- published scientific papers, released draft chapters, public presentations, and research updates-- make the following points clear:

- Dioxin concentrations in the food supply and the bodies of the general human population are now in the range at which effects on reproduction, development, and the immune system are known to occur in laboratory animals. Health damage has been documented in humans at dioxin body burdens just slightly higher than those found in the general population. And a substantial body of evidence indicates that wildlife populations are experiencing reproductive and developmental effects at background concentrations of dioxins and related compounds.
- The developing infant and fetus are at the greatest risk from dioxin exposure. At very low doses, dioxin can lead to permanent hormonal changes, birth defects, reduced sperm count, infertility, endometriosis, and impaired ability to learn. Dioxin is also a very potent immune suppressor, leading to increased susceptibility to infectious disease.
- The weight of evidence indicates that dioxin causes cancer in humans. Current "background" dioxin contamination results in cancer risks to the general population ranging from 1 in 1,000 to 1 in 10,000. These risk estimates suggest that dioxin is responsible for 350 to 3500 cancers in the U.S. annually.
- There does not appear to be a threshold or "safe dose" below which effects do not occur. EPA's research has been unable to identify a level of exposure, no matter how low, at which dioxin does not bind to its receptor, interact with DNA, and induce enzymes and growth factors that are involved in both cancer and non-cancer effects.

This body of evidence suggests that dioxin exposure may be causing subtle effects now among large sectors of the general public, but the lack of an uncontaminated control group makes it impossible to determine with certainty whether this is the case. The data clearly shows, however, that existing environmental concentrations of dioxin place the human population *at risk*, and any increase in these concentrations will increase that risk and/or the severity and number of persons affected. Because the potential health damage is severe -- and because the entire public is exposed and potentially affected -- this situation calls for immediate action to reduce human exposure to dioxin and prevent further dioxin releases.

DIOXIN IS FORMED THROUGHOUT CHLORINE CHEMISTRY

Dioxin is never manufactured on purpose. It is formed as an unintentional by-product in scores of processes in which chlorine and chlorine-derived chemicals are produced, used, and disposed. In fact, the evidence suggests that virtually or absolutely all chlorine-related products and processes are associated with dioxin formation at some point in their lifecycle. The many industrial processes in which the formation of dioxins and related compounds has been identified are shown in table 1.1.

The formation of dioxin begins with the production of chlorine gas itself in the "chlor-alkali" process. It was formerly believed that only older facilities using graphite electrodes produced dioxin, but new Swedish data indicate that dioxins are formed in even the most modern chlor-alkali plants with titanium electrodes. Because the production of chlorine itself results in dioxin formation, all "downstream" processes in which chlorine is used are linked to dioxin, as well.

Dioxin appears to be formed whenever chlorine is used in industrial processes in the presence of organic (carbon-containing) substances. Thus, dioxin and related compounds have been identified from pulp mills that use chlorine or chlorine dioxide bleach; from chemical plants that use chlorine to produce other chemicals; from metallurgical plants that use chlorine to produce refined metals; and even from the use of chlorine to disinfect water and wastewater.

Dioxin is formed as a by-product when chlorine is used to produce organochlorine chemicals. It has long been recognized that dioxins occur in the production of aromatic (benzene-based) organochlorines, such as chlorobenzene, chlorophenols, PCBs, and many pesticides. But dioxin is also formed in the manufacture of the full range of organochlorines, from the simple to the complex, from cyclic to aliphatic compounds, including solvents, feedstocks for PVC plastic, and specialty chemicals and intermediates. In addition, dioxins have been identified in some inorganic chlorine compounds, such as ferric and copper chlorides and hypochlorite bleach.

Dioxins can also be formed when chlorinated organic chemicals are used – particularly in environments that are reactive, heated, or alkaline. Thus, dioxins are formed when chlorinated solvents are used as degreasing agents along with sodium hydroxide, when chlorinated chemicals are burned in vehicle fuels as scavengers or additives, when organochlorine catalysts are used in oil refining, and when organochlorine pesticides are heated in the pressure-treatment of lumber. In addition, dioxins can also be formed when organochlorines are used as intermediates in the production of other chemicals – even those in which chlorine does not appear in the final product, such as the pesticide parathion or the chemical nitrophenol. Dioxins are even formed when some other organochlorines, such as chlorophenols, are released into the environment and are then subject to biological, physical, or chemical transformation.

Finally, dioxins are formed whenever any organochlorine chemical – product or waste – is burned. Combustion is the largest sector of dioxin sources, and dioxins have been identified from all types of incinerators that burn organochlorines, including incinerators for hazardous waste, garbage, medical waste, sewage sludge, and so on. Dioxins are also formed when organochlorines are introduced into combustion-based recycling processes – such as when PVC-coated copper cables or automobiles are recycled in secondary smelters. Very large quantities of dioxin are formed when chlorinated chemicals – particularly PVC plastic – burn in accidental fires at homes, offices, and industrial facilities.

RANKING DIOXIN SOURCES

In its reassessment documents, EPA makes a preliminary attempt to catalogue dioxin sources, identifying two of the largest known sources of dioxin:

- Incinerators that burn chlorinated wastes, including garbage, hazardous waste, and hospital waste, along with other facility types in the combustion sectors;
- Pulp mills that use chlorine and chorine-dioxide bleaches.

But EPA's list neglects dozens of other dioxin sources that have been identified in the scientific literature and government reports and underestimates actual emissions from incinerators. As a result, the agency's list accounts for only 10 to 50 percent of the 25,000 grams of dioxin that EPA estimates are deposited into the environment each year. Major sources neglected by EPA include the following:

* All references to EPA's dioxin source estimates are based on EPA reassessment documents prior to July 1994. Future EPA documents may revise these estimates.

- Chemical industry. Dioxin is produced in the manufacture of chlorine and a full range of chlorinated organic chemicals, including PVC, pesticides, solvents, and other chemicals used in rubber, dyes, plastics, and other products.
- PVC plastic. Throughout its lifecycle, PVC appears to be responsible for more dioxin formation than any other product. When PVC feedstocks are manufactured, large quantities of dioxins are formed and are released in air emissions, water releases, solid and liquid wastes. Recent Swedish studies have found that the PVC plastic itself is contaminated with dioxin. PVC is also the largest source of chlorine – and thus the major dioxin precursor – in most of the largest combustion sources of dioxin, including hospital waste incinerators, trash incinerators, and copper and steel smelters. Finally, the accidental burning of PVC in home and building fires appears to be an extremely large dioxin source. Preliminary estimates suggest that PVC may account for up to one-third of all dioxin formation.
- Garbage incinerators. EPA's estimate of combined dioxin emissions from the nation's 120 garbage incinerators is 15 times *lower* than measured dioxin emissions from a *single* incinerator in Columbus, Ohio. Garbage burners appear to be the largest point sources of dioxin, with total nationwide emissions hundereds of times higher than EPA estimates.
- Cement kilns that burn hazardous waste. High dioxin emissions have been documented from these facilities, in which more hazardous waste is burned than in officially designated "incinerators."
- Hazardous waste incinerators. EPA underestimates actual emissions from these facilities by basing its estimate on a trial burn at a single facility. Data from real incinerators – which operate under non-optimal or upset conditions – indicate that EPA has underestimated actual dioxin releases by more than 20 times.
- Ash and dusts from all types of waste incinerators, which may carry up to 100 times more dioxin into the environment than air emissions.
- Metallurgical industries in which chlorine or organochlorines are present, such as the steel, nickel, and magnesium industries.
- Environmental transformation – other organochlorines released into the environment can be transformed into dioxins by natural processes.

EMERGENCY ACTION : A NATIONAL STRATEGY FOR DIOXIN ELIMINATION

The emerging science makes clear that current dioxin contamination poses a serious threat to public health and the environment, with future generations at the greatest risk if dioxin pollution continues. Because these compounds are so persistent in the environment and the human body, dioxins that have already accumulated will not disappear quickly. In response to its findings, EPA should initiate a comprehensive national dioxin elimination program, with the goal of reducing human exposure and preventing the formation and discharge of dioxin into the environment from all sources. The program should be based on these principles:

- Zero *means zero*. Dioxin releases must be eliminated, not simply reduced. Because dioxin is so persistent, the ecosystem has no capacity to safely "assimilate" dioxin inputs. Even dilute discharges build up in the environment over time, eventually reaching unacceptable concentrations. Thus, zero is the only acceptable discharge of dioxin, and the current crisis demonstrates the failure of the "acceptable discharge" approach to persistent toxic substances. Given the current health threat, it would be wholly inappropriate for EPA to permit the continued release of *any* additional dioxin into the environment.
- Pollution prevention, not control. Once dioxin is produced, it is too late to prevent its discharge into the environment; pollution control devices, filters, treatment systems, and disposal methods such as burning and burying simply shift captured chemicals from one environmental medium to another or delay their release until a later date. Dioxin discharges can be eliminated only through primary prevention: industrial processes and feedstocks that result in dioxin formation must be changed so that no dioxin is formed.

Because dioxin occurs throughout the field of chlorine chemistry, this goal appears to require the planned phase-out of chlorine and chlorinated organic feedstocks from industrial processes.

- Address all dioxin sources. EPA must address all known and suspected sources of dioxin in order to bring future releases of dioxin to zero. All identified dioxin sources must be subject to action, and EPA should initiate research to identify all unknown or suspected sources.
- Set priorities. Because dioxin is associated with the many uses of chlorine in our industrial economy, eliminating dioxin will require substantial technical and economic conversion. The largest dioxin-producing sectors for which the alternatives are available and feasible now should be subject to immediate action. Sectors that require longer implementation phases should be placed on timelines for dioxin elimination.

STEP 1: GENERAL POLICY CHANGES

1. National policy. EPA should establish a national Zero Dioxin program with the goal of eliminating the formation and release of dioxin within 10 years, through the elimination of those products, feedstocks, and processes that lead to dioxin formation.

2. Moratorium on new dioxin permits. EPA should grant no new permits for facilities to release dioxin into any environmental medium.

3. Sunset existing dioxin permits. EPA should modify all existing dioxin release permits to include timetables for the reduction and eventual elimination of all dioxin releases.

4. Comprehensive approach to all sources. For the many dioxin sources that are not currently permitted or regulated, EPA should begin a process to identify and prioritize all dioxin sources and require their elimination within 10 years. All facilities that manufacture, use, or dispose of chlorine or chlorinated organic chemicals should be required to test for dioxin formation in their products, processes and wastes. Any identifiable release of dioxin and related compounds should be reported under federal Right-to-Know programs.

STEP 2: IMMEDIATE PRIORITIES: PHASE-OUT MAJOR DIOXIN SOURCES

EPA should take immediate action in the largest dioxin-producing sectors. In these sectors, regulatory action can have the most significant impact on total dioxin pollution.

1. Incineration. Incinerators that burn chlorinated wastes are the largest known producers of dioxin. EPA should take the following actions:

- Place an immediate moratorium on permits for new combustion facilities that burn chlorinated wastes and on permits for expanded capacity at existing units.
- Modify immediately all permits for combustion devices to include sunset provisions to reduce and finally eliminate the input of chlorinated wastes and products. This policy should include incinerators, boilers, kilns and furnaces that burn hazardous wastes; incinerators for trash, hospital waste, sewage sludge, tires, and soils; and metal smelters. Minimize the burning of chloride-containing wastes (i.e., food and yard wastes) through trash separation programs.
- In turn, focus waste reduction programs on eliminating the generation of chlorinated incinerable wastes, particularly solvents, PVC, and wastes from the production and use of chlorinated substances in the chemical industry.
- Immediately ban the addition of chlorinated compounds to fuels, including gasoline, diesel, and others.

2. Pulp and Paper. The use of chlorine, chlorine dioxide, and other chlorine-based bleaches in the paper industry is the largest source of dioxin discharges directly to waterways and one of the largest dioxin-producing sectors overall. In other parts of the world, totally chlorine-free pulp bleaching is rapidly becoming the industry standard. EPA should establish rapid timelines for the phase-out of all chlorine-based bleaches in the pulp and paper industry.

3. PVC plastic. Throughout its lifecycle, PVC results in more dioxin formation than any other single product. EPA should immediately establish a PVC phase-out program, with progressive reductions toward zero in the production and use of PVC. Particularly rapid phase-outs should be applied to short-life uses of PVC (packaging, toys, etc.), PVC in areas susceptible to fire (construction, appliances, and automobiles), and PVC in products that are recycled in combustion-based processes (i.e., cables and cars). EPA should also modify all discharge permits for chemical plants that produce PVC feedstocks to include sunset provisions on dioxin discharges to all environmental media.

STEP 3: SECONDARY ACTIONS: PHASE-OUT OTHER CHLORINE USES

While immediate action occurs in the priority sectors discussed above, the following dioxin-producing sectors should be placed on timetables for phase-out.

1. Chlorinated solvents. In production, incineration, and some uses, chlorinated solvents cause dioxin formation. EPA should establish a rapid timetables for phasing-out the production and use of these chemicals.

2. Chlorine-related pesticides. Dioxins are formed in the production, some uses, and disposal of chlorine-containing pesticides and in the production of non-chlorinated pesticides made with chlorinated intermediates. EPA should establish a phase-out timetable for all chlorine-related pesticides.

3. Other chlorinated organic chemicals. Dioxins are formed in the production, some uses, and disposal of other chlorinated organic chemicals, such as intermediates, catalysts, and specialty chemicals. EPA should establish timelines for phasing-out the production and use of all other chlorinated organic chemicals. Any uses that serve a compelling social need (i.e.,pharmaceuticals) for which alternatives may not yet be available should receive temporary exemptions.

4. Metallurgy and inorganic chemicals involving chlorine. Dioxins are formed when chlorine and chlorinated compounds are used in metallurgical processes and in the production of some inorganic chlorinated chemicals. EPA should establish phase-out timetables for the use of chlorine in high-temperature metallurgy, and for the manufacture of inorganic chlorinated chemicals associated with dioxin formation.

5. Wastewater treatment. Dioxins and related compounds are formed in the chlorination of drinking water and sewage and in the incineration of sludge from wastewater treatment plants. EPA should establish timelines for the implementation of chlorine-free water treatment methods while insuring that adequate disinfection continues. In most cases, chlorine-free methods for water disinfection can be implemented more quickly than the use of chlorine as a residual can be eliminated.

6. Remediation. A substantial quantity of dioxin and PCB-contaminated materials are now present in landfills, sediments, and extant industrial wastes. EPA should intensify the development and application of non-combustion methods for degradation of these materials.

TABLE 1.1.
SUMMARY OF PROCESS THAT FORM DIOXIN AND RELATED CHEMICALS

Production of chlorine gas
- Chlorine electrolysis with graphite electrodes
- Chlorine electrolysis with titanium electrodes

Chemical industry – use of chlorine gas
- Chlorinated aromatic chemicals – manufacture (chlorobenzenes, chlorophenols, PCBs, others)
  - Pesticides
  - Dyes
  - Specialty chemicals
- Chlorinated solvents – manufacture (trichloroethylene, tetrachloroethylene, carbon tetrachloride)
- PVC plastic – manufacture of feedstocks (ethylene dichloride, vinyl chloride)
  - Production wastes
  - Effluent
  - Sludge from effluent treatment
  - Air emissions
  - PVC plastic products
- Other aliphatic organochlorines – manufacture (epichlorhydrin, hexachlorobutadiene)
- Some inorganic chlorides – manufacture (ferric and copper chlorides, sodium hypochlorite)

Uses of chlorine gas – other industries
- * Pulp and paper – chlorine bleaching
  - Mill effluent
  - Mill sludge
  - Pulp and paper products
  - Emissions from sludge incinerators
- Water and wastewater disinfection
- Refined metals – manufacure with chlorine (Ni, Mg)

Uses of organochlorines
- Manufacture of chlorine-free chemicals with chlorinated intermediates (nitrophenols, parathion, others)
- Degreasing/extraction with organochlorine solvents in alkaline or reactive environments
- Oil refining with organochlorine catalysts
- Use of pesticides with heat (wood treatment, etc.)
- Iron/steel sintering with organochlorine cutting oils, solvents, or plastics
- * Burning gasoline or diesel fuel with organochlorine additives
- Use of chlorine-based bleaches and detergents in washing machines and dishwashers

Incineration, Recycling, and Fires (primary dioxin precursor in parentheses)
- * Medical waste incinerators (PVC)
  - Air emissions
- * Municipal waste incinerators (PVC)
  - Air emissions
  - Ash residues
- * Hazardous waste incinerators (solvents, chemical manufacturing wastes)
  - Air emissions
  - Ash residues
- Cement kilns burning hazardous waste (solvents, chemical manufacturing wastes)
  - Air emissions
  - Cement kiln dust
- Accidental fires in homes and offices (PVC)
- Fires at industrial facilities (PVC, PCBs, other chlorinated chemicals)
- Aluminum recycling/smelting (PVC)
- Steel and automobile recycling/smelting (PVC)
- * Copper cable recycling/smelting (PVC)
- Aluminum recycling/smelting (PVC)
- * Wood burning (pentachlorophenol wood preservatives, PVC)

Environmental transformation
- Transformation of chlorophenols to dioxins in the environment

* Addressed by EPA in documents related to its dioxin reassessment. (Cleverly 1993, Schaum 1993)

List includes sectors in which formation of dioxin or related compounds (PCBs, chlorinated dibenzofurans, and/or hexachlorobenzene) has been confirmed in chemical analyses, as well as sectors in which dioxin formation is "known or suspected" according to EPA (EPA 1980, EPA 1985, PCTN 1985) or NATO (Hutzinger 1988).

POLITICAL AND ECONOMIC IMPLICATIONS

Because dioxin can be formed at some point in the lifecycle of all or virtually all chlorine-related processes, the only feasible method for preventing dioxin releases is to target the critical step – the production of chlorine and related compounds. Controlling dioxin formation at later steps in the chlorine "use-tree" would require specific regulations on scores of processes and thousands of chemicals. Because dioxin is an inevitable by-product of the field of industrial chlorine chemistry, addressing dioxin pollution requires a comprehensive approach to the class of chlorine-based products and processes.

Numerous scientific and political institutions have called for a phase-out of chlorine chemistry in order to eliminate the formation and release of persistent toxic substances. For instance, the International Joint Commission on the Great Lakes has called on the U.S. and Canadian governments to bring to zero all discharges of persistent toxic substances to all environmental media; particularly priority was placed on the dioxin family. In 1992 and 1994, the IJC recognized that dioxin formation occurs throughout the field of chlorine chemistry and that the mix of by-products formed in the lifecycle of chlorine and related compounds cannot be prevented or controlled. Thus, the IJC has called on the governments to phase-out all uses of chlorine and chlorinated organic chemicals as industrial feedstocks.

In 1993, the American Public Health Association made a similar recommendation, finding that "the only feasible and prudent approach to eliminating the release and discharge of chlorinated organic chemicals and consequent exposure is to avoid the use of chlorine and its compounds in manufacturing processes." APHA resolved that chlorinated organic compounds should be treated as a class for phase-out, with exceptions only for uses that can be demonstrated to pose no significant hazard or for which no alternatives can be found.

In 1992 and 1993, the 15-nation Paris Convention on the Northeast Atlantic and the 21-nation Barcelona Convention on the Mediterranean Sea agreed that the discharge of persistent, bioaccumulative toxic substances – particularly organohalogens such as dioxins – must be brought to zero on rapid timelines. And in early 1994, the White House itself began to move towards the comprehensive approach to the field of chlorine chemistry in its proposal for the Clean Water Act, proposing to develop "a national strategy to reduce, substitute, or prohibit the use of chlorine and chlorinated compounds" in several important use sectors.

For all major dioxin sources, chlorine-free alternatives are available and feasible now. There are few or no *technical* barriers to the elimination of dioxin releases associated with paper bleaching, PVC, solvents, sewage and drinking water disinfection, pesticides and many chemical manufacturing processes. In many cases, these alternatives result in direct economic benefits as investments in process changes are paid off with reduced costs for chemical procurement, pollution control and disposal, liability, increased resource efficiency, and more reliance on skilled labor then chemicals.

But phasing out all dioxin sources will require substantial conversion. In some industries – particularly the manufacture of chlorine-based chemicals – the transformation may be difficult. Workers and communities should not bear the economic burden of changes to protect public health and the environment. The Zero Dioxin Strategy should be guided by a transition planning process involving labor, communities, and all other stakeholders. Revenue from a tax on on chlorine and related chemicals should be distributed to ensure that investment in alternative products and processes takes place in the same locations where chlorine-based processes were formerly used; it should also be used to protect, compensate, and provide future opportunities for workers and communities that are affected by conversion.

2: NEW SCIENCE ON DIOXIN HAZARDS

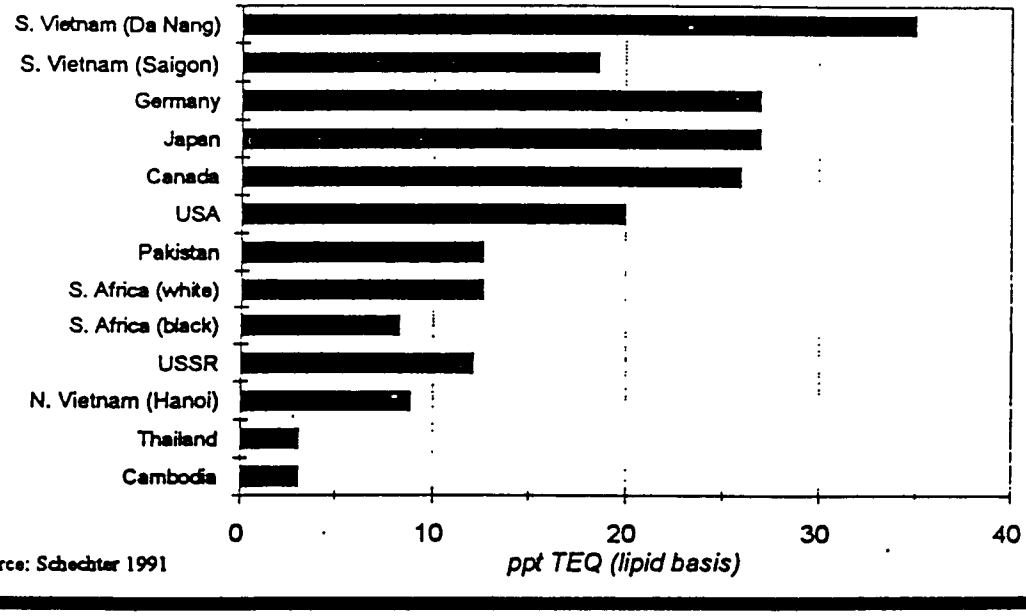
Exposures to dioxin

According to EPA, an average person in the U.S. is exposed to 3 to 6 picograms* of dioxin (TEQ) ** per kilogram of body weight per day, including dioxin-like PCBs. [EPA 1994] An average adult has accumulated a body burden of 40 to 60 parts per trillion in his or her fat. About 90 percent of these exposures occur due to contamination of the food supply, particularly through fish and animal products. The remainder occur through inhalation of contaminated air and ingestion of contaminated water.

In most industrialized nations of the world, dioxin body burdens and exposures are in the same range as in the U.S.; levels are somewhat lower in developing nations. [Schecter 1991] Concentrations of dioxins in breast milk from numerous countries are shown in figure 2.1.

Infants receive even higher exposures. Because mothers' milk is highly contaminated, an average infant that nurses for one year receives 60 pg/kg/day of dioxin, not including dioxin-like PCBs – 10 to 20 times greater than an average adult dose. An average infant receives 4 to 12 percent of his or her entire lifetime exposure within the first year of life – a critical period of development and sensitivity to toxic chemicals.

Figure 2.1.
Dioxin in Human Milk from Selected Countries



Source: Schechter 1991

* A picogram is one one-trillionth of a gram.

** TEQ, or toxic equivalents, expresses the total quantity of dioxin-like compounds relative to the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Unless noted, all values given in this document are expressed as TEQ.

Once emitted into the environment, dioxins persist for decades or centuries. Moreover, dioxins are also highly bioaccumulative, so they multiply in concentration as they move up the food chain. Thus, even dilute discharges of dioxin accumulate in the environment over time.

Dioxins can also be transported long distances on air and water currents; wildlife migration and food shipments result in further dispersion. As a result, dioxins contaminate the environment, wildlife, and humans in areas thousands of miles from any known sources, including the Arctic and the deep oceans. Persons living near or working in facilities that are dioxin sources and those who consume higher-than-normal quantities of dioxin-contaminated foods are subject to even greater exposures.

Trends in Dioxin Contamination

When persistent toxic substances are released into the environment, they are degraded slowly – if at all. Over time, these substances accumulate in air water, soil and sediment, and bioaccumulation results in even higher concentrations in the foodweb and the bodies of the general population. Trends in contamination by these substances tend to follow this general pattern: new or increased discharges of a chemical result in slowly increasing environmental concentrations; the increase levels off when a "steady-state" is reached – the point at which the rate of "removal"– either through degradation or accumulation in sediments, soils, and vegetation – matches the rate of release. Any given rate of release will correspond to a specific steady state. Thus, if discharges are reduced, levels will slowly decline and then plateau at a new steady state. For instance, PCB concentrations in water and the food chain increased steadily from the 1940s to the 1960s, after which they leveled off. After the production of PCBs was banned in the 1970s, levels decreased during the 1980s. The decline has now stopped, however, plateauing at a level still considered unsafe. [IJC 1991]

Data on trends in dioxin contamination suggest a similar pattern. Sediment cores show that levels rose consistently from 1940 to 1970 and then reached a relatively steady state. [Czuczwa 1987] Regulatory changes in the 1970s – increased controls on some incinerators, restrictions on leaded gasoline (which contains chlorinated additives) and bans on several highly dioxin-contaminated pesticides – resulted in reduced dioxin emissions, after which concentrations declined slowly in Great Lakes sediments and in some but not all species of wildlife over the perioed 1977-1987. [Allan 1991]

Data on trends in dioxin contamination of human tissues are very sparse but seem to fit this pattern. One study [Stanley 1990] found that levels may have decreased slightly in the 1980s following consistent increases during the preceding decades; "however, it is not known whether these declines were due to improvements in the analytical methods or actual reduction in body burden levels," according to EPA. [EPA 1994] If these declines are real, they probably reflect the pattern described above. According to Swedish scientists:

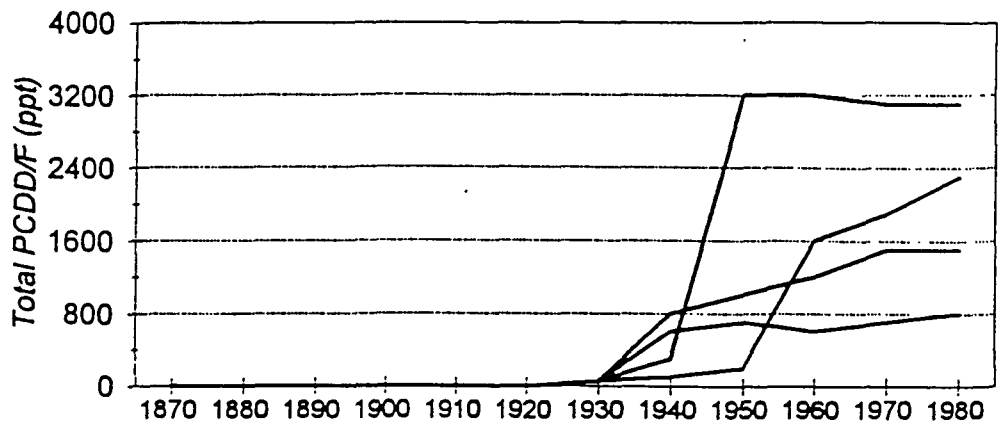
> *"Within the Swedish Dioxin Survey, levels and trends of PCDD/PCDF have been determined in various media. During the last twenty years an overall decrease in the levels is recorded. The major part of this decrease dates back to the late 1970s and the early 1980s. The situation of today seems to be quite constant and resembles what has been found for PCB. Analyses of human breast milk show a similar trend." [Johansson 1993]*

This pattern suggests that reducing industrial releases of persistent toxic substances will result in decreased contamination of the environment and human tissues. If releases continue at a reduced rate,

concentrations will decline only until a new steady state is reached. Further, the "removal" of these substances at steady-state into sediments, soil, and vegetation is actually a form of accumulation, and at least some of these sequestered stores will eventually be released again into the ambient environment, where they will be available for uptake by wildlife and humans. Thus exposure to highly persistent and toxic substances – and the build-up of these compounds in human tissues – can be prevented only if releases of these compounds are brough to zero.

Figure 2.2.

Trends in dioxin contamination in Great Lakes sediments.



Source: Czuczwa and Hites 1986.

Roots of the Reassessment

In April 1991, under pressure from the chlorine and paper industries, EPA announced a multi-year reassessment of the toxicity of dioxin. The project was ostensibly intended to incorporate into EPA regulations new science that had been developed since the agency last formulated dioxin policy in 1985. (Another reassessment, also begun under pressure from dioxin-producing industries, proposed in 1987 to weaken dioxin standards by a factor of 16, but was rejected by EPA's Science Advisory Board. [EPA 1988])

EPA announced the dioxin reassessment at the height of a scientific and public relations campaign in which dioxin-producing industries and their scientists argued that new data on the mechanisms of dioxin's toxicity implied that dioxin is less of a hazard to human health than previously thought. In particular, an understanding had emerged that dioxin's effects begin when a molecule of the chemical binds to a "receptor" protein within the cell, then enters the cell nucleus and interacts with DNA to trigger a cascade of biochemical reactions, including the synthesis and metabolism of hormones, their receptors, enzymes, growth factors, and other substances. Industry had argued that dioxin's interaction with the receptor meant that dioxin was less

toxic than previously thought and that there would be a threshold, or safe dose of dioxin, below which no adverse effects would occur. [Bailey 1992]

Contrary to the industry's intent, however, EPA's reassessment has concluded that dioxin is more toxic than previously thought and poses a significant hazard to the health of the general population. Although the final draft for public review is slated for release in summer 1994, drafts of the reassessment, research updates, published scientific papers and presentations, all by EPA and the scientists working on the reassessment clearly indicate the direction of EPA's final report.

Finding #1: "Background" dioxin pollution is a threat to the general population.

EPA's reassessment indicates that dioxin pollution threatens the health of the general public. Several lines of evidence support this conclusion.

First, EPA scientists have concluded that the general population's current body burdens and exposures are already in the range at which health effects occur in laboratory animals, including rodents and primates. Persons subject to higher-than-average exposures – because they live near local dioxin sources or regularly consume highly-contaminated foodstuffs – are at even greater risk. According to a draft of EPA's reassessment:

> *Subtle changes in enzyme activity indicating liver changes, in levels of circulating reproductive hormones in males, in reduced glucose tolerance potentially indicative of risk of diabetes, and in cellular changes related to immune function suggest the potential for adverse impacts on human metabolism, reproductive biology, and immune competence at or within one order of magnitude of average background body burden levels....*
>
> *Individuals at the high end of the general population range may be experiencing some of these effects. Some more highly exposed members of the population may be at risk for frankly adverse effects including developmental toxicity, reduced reproductive capacity based on decreased sperm counts and potential for increased fetal death, higher probability of experiencing endometriosis, reduced ability to withstand an immunological challenge and others. [EPA 1994]*

EPA's Assistant Administrator for Research and Development echoed this conclusion:

> *"Certain non-cancer effects, including changes in endocrine function associated with reproductive function in animals and humans, behavioral effects in offspring of exposed animals, and changes in immune function in animals have been demonstrated. Some data suggest that these effects may be occurring in people at body burden levels that can result from exposures at or near current background.... Risks from ubiquitous background levels of dioxin in the general population need to be carefully considered." [Bretthauer 1992]*

This conclusion was reinforced in a recent scientific publication by EPA's chief toxicologist on the dioxin reassessment:

> *"Results in enzyme induction from both rats and mice would suggest that at current environmental levels, (1-10 pg/kg/day) people may be experiencing small but significant increases in these markers of response. Highly exposed populations may be at special risk. Since animal studies suggest that changes in hepatic enzyme induction occur at body burdens similar to those at which immunotoxicity in mice*

and permanent effects on the reproductive system occur in rats, it is reasonable to hypothesize that subtle effects on these parameters may be occurring in the human population. [Birnbaum 1993]

And EPA's dioxin research team restated this conclusion in a presentation to an international scientific conference in late 1993:

"[EPA's] documents reflect input from the expert panel that concluded that current body burdens of dioxin and related compounds (TEQ) are at or near the point where responses would be expected to occur., As the panel recommended, additional emphasis was placed on non-cancer endpoints, since effects on the immune system and developmental reproductive effects appear to be occurring at extremely low levels". [Birnbaum 1993a]

Second, there is substantial evidence to indicate that populations of wildlife species high on the food chain are suffering health damage due to reproductive and developmental impairment due to "background" exposures to dioxin and related compounds. In the Great Lakes, for instance, exposure to dioxin-like compounds has been linked to large-scale hormonal, reproductive, and developmental impairment among numerous species of predator birds, fish and wildlife; these impacts are primarily transgenerational, affecting the offspring of the exposed organism. [Giesy 1994, IJC 1991]

Finally, according to EPA, effects on humans – including hormonal and metabolic changes – have been documented at dioxin body burdens and exposures that are only slightly higher than those to which the general population is subject. [EPA 1994] For instance, while the average American's dioxin body burden is estimated at 9 ng/kg, workers with dioxin body burdens of just 13 ng/kg have been found to have significantly decreased levels of serum testosterone. Further, laboratory experiments on primates have also documented health effects at dioxin exposures and body burdens at or near those of the general human population. For instance, at body burdens just 3 times greater than that of the average U.S. resident, rhesus monkeys suffer significantly increased incidence of endometriosis, while marmosets experience altered immune system responses. [EPA 1994] These data indicate that if there is any "margin of safety" left between the current exposure of the average American and the level at which health damage begins, this margin is a small one.

In summary, the evidence suggests that dioxin exposure *may* be causing effects now among large sectors of the general public, but the lack of an uncontaminated control group makes it impossible to determine with certainty whether this is the case. The evidence clearly shows, however, that existing environmental concentrations of dioxin place the human population *at risk,* and any increase in these concentrations will increase that risk and/or the severity and number of persons affected.

Finding #2: Cancer hazard to humans

In 1985, EPA found that dioxin was the most potent synthetic carcinogen yet tested. [EPA 1985] Since that time, industry representatives have attempted to argue that dioxin may be carcinogenic in laboratory animals but not in humans. EPA's reassessment reviewed the laboratory and epidemiological data, however, and concluded that dioxin poses a severe cancer hazard to both humans and animals.

According to EPA's draft reassessment:

With regard to carcinogenicity, a weight-of-the evidence evaluation suggests that dioxin and related compounds are likely to present a cancer hazard to humans.... While the epidemiological data alone are not yet deemed sufficient to characterize the cancer hazard of this class of compounds as being "known," the unequivocal evidence in animal studies, inferences drawn from mechanistic data, and the suggestive evidence of recent epidemiology studies support the characterization of dioxin and related compounds as likely cancer hazards. [EPA 1994]

According to a summary by EPA's dioxin research team:

"Dioxin does cause cancer in humans." [EPA 1992]

In fact, EPA estimates that "background" dioxin exposures to dioxin-like compounds result in very high cancer risks throughout the general population.

*Modeling estimates suggest that current background exposures may result in upper bound population cancer risk estimates **in the range of one in ten thousand (10-4) to 1 in a thousand (10-3) attributable to exposure to dioxin and related compounds.** [EPA 1994] (emphasis added)*

Thus, dioxin exposure currently poses cancer hazards that are 100 to 1000 times greater than the standard "acceptable" risk of one cancer per million. According to EPA's risk estimates, dioxin would cause from 350 to 3500 cancers each year in the U.S. – up to 3 percent of all cancers.

EPA's finding that dioxin can be considered a human carcinogen is reinforced by the recent conclusion of the U.S. National Institutes of Medicine, a division of the National Academy of Sciences. After reviewing several hundred epidemiological reports on dioxin and cancer, the panel concluded that there is "sufficient evidence" of an association between exposure to dioxin and dioxin-contaminated herbicides and soft-tissue sarcoma, non-Hodgkins' lymphoma and Hodgkin's disease in humans. The panel also found "limited/suggestive evidence" for an association between dioxin exposure and respiratory cancer, prostate cancer, and multiple myeloma. Limited/suggestive evidence refers to strong evidence that nevertheless cannot complete exclude a possible role for chance or confounding factors. [Fallon 1993]

Finding #3: Severe effects on reproduction and development

Recent research indicates that the effects of dioxin on the reproductive, developmental, and immune system effects are even more severe than cancer. EPA's reassessment has confirmed that dioxin interferes with a wide range of biochemical substances, including sex and thyroid hormones, insulin, growth factors, and enzymes. These changes can result in an array of effects on reproduction, development and immune system function, as shown in table 2.2.

Many of the non-cancer effects found in dioxin-exposed rodents have already been documented in primates and human populations exposed to dioxin or dioxin-like compounds, including alterations in male sex hormones, altered thyroid function, immune suppression, impaired neurological function on development, reduced penis size, behavioral changes, immune system effects, and birth defects.

TABLE 2.2

Health Effects Associated with Dioxin and Related Chemicals

- Metabolic changes: weight loss, wasting syndrome, fetal death, altered glucose and fat metabolism
- Hormonal changes (including alterations in sex, thyroid, and other hormones)
- Birth defects
- Impaired neurological development and subsequent cognitive deficits
- Male reproductive toxicity:
  - reduced sperm count
  - testicular atrophy
  - abnormal testis structure
  - reduced size of genital organs
  - feminization of hormonal and behavioral responses
- Female reproductive toxicity
  - hormonal changes
  - decreased fertility
  - inability to maintain pregnancy
  - ovarian dysfunction
  - endometriosis
- Immune suppression and increased susceptibility to infectious diseases
- Effects on the liver, thymus, spleen, bone marrow, and skin
- Diabetes

Source: adapted from EPA 1994.

According to EPA, "In mammals, postnatal functional alterations involving learning behavior and the developing reproductive system appear to be the most sensitive to perinatal dioxin exposure." [EPA 1994] During key periods of development, the fetus or infant is exposed to large quantities of dioxin as the mother's body burden is partially liberated and transferred to the infant across the placenta and via breast milk. Doses low enough to cause no visible changes in an adult can produce serious and long-lasting effects on infant or fetal development. These impacts may be invisible at birth but appear later as functional deficits during childhood, puberty or adulthood, leading to reduced fertility, altered behavior, diminished intellectual capacity, and impaired immune defenses. According to EPA's draft reassessment:

> *Recent laboratory studies have suggested that altered development may be among the most sensitive TCDD endpoints.... Developmental toxicity endpoints are observed at lower TCDD exposure levels than are endpoints of male and female reproductive toxicity.... Of particular interest to the risk assessment process is the fact that a wide variety of developmental events, crossing three vertebrate classes and several species within each class, can be perturbed, suggesting that dioxin has the potential to disrupt a large number of critical developmental events at specific developmental stages. Not only can these changes lead to increases in embryo/fetal*

mortality, but they can disrupt organ system structure and irreversibly impair organ function, even after only transient perinatal TCDD exposure. [EPA 1994]

Finding #4: No safe dose

EPA's reassessment indicates that any dose of dioxin, no matter how small, can cause biochemical changes that may lead to a wide range of effects on the health and function of an organism. There does not appear to be any threshold or safe dose below which dioxin does not cause these biochemical changes. According to a recent reassessment update by EPA's research team:

No evidence for low dose nonlinearity was observed for sensitive biochemical responses. [Birnbaum 1993a]

EPA based this conclusion on research conducted by the National Institute for Environmental Health Sciences (NIEHS), which investigated the relationships between dioxin dose and certain effects, including receptor binding, enzyme and growth factor induction, and certain other key parameters. For none of the effects tested was a threshold evident. [Portier 1993] According to NIEHS:

"[Our findings] are consistent with the knowledge that for some receptor-mediated responses, there is a proportional relationship between receptor occupancy and biological response, even at low ligand concentrations. The results presented here illustrate that a threshold for the biological effects of TCDD exposure cannot be assumed simply on the basis that dioxin response is receptor-mediated.... If TCDD-mediated effects on cytochrome p-450 induction or EGF receptor binding are reliable surrogates for toxicity or toxicity is induced by similar mechanisms, the risks from exposure to TCDD are as high or possibly higher than were estimated by the EPA using a linear model. If this is the case, there should be considerable concern for the high levels of TCDD already present in human tissues." [Portier 1993]

It is possible that certain effects that follow these biochemical disruptions will be related to dioxin exposure in a non-linear or threshold fashion. But according to EPA, "This point is somewhat moot, however, given that background exposures to dioxin are ubiquitous and associations between dioxin exposure and certain types of cancer have been noted at body burdens within 1-2 orders of magnitude of average background body burdens, obviating the need for large-scale low dose extrapolations." [EPA 1994] Even if there is a safe dioxin dose, we are already at, near, or above that threshold. Additional exposures cannot be considered "safe."

These findings have extraordinary implications for public health and regulatory policy. Since no dioxin exposure can be considered safe – and any additional exposure may result in incremental increases in the severity of toxic effects – there is no longer any such thing as an "acceptable" discharge that does not pose a hazard to public health. This fact implies that the regulatory policies and models used in the U.S. and Europe is no longer appropriate and have failed to protect public health. These regulatory systems are based on permitting discharges within specified limits, so long as they are not predicted to result in exposures that exceed an individual's "acceptable daily intake." If any additional dioxin exposure poses a public health hazard, the basis for granting permits is no longer valid, and the regulatory framework must shift to the prevention of releases.

EPA has admitted as much, although it has not even begun to establish a prevention-based policy. In particular, EPA admits that the use of a reference dose (RfD) – and its corollary, the "acceptable daily intake" –

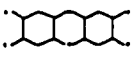
is not appropriate, since these standards are based on the assumption that there can be a safe level of exposure:

> *In the case of dioxin and related compounds, calculation of an RfD based on human and animal data and including standard uncertainty factors to account for species differences and sensitive subpopulations* ***would result in reference intake levels on the order of 10-100 times below the current estimates of daily intake in the general population.*** *For most compounds where RfDs are applied, background exposures are generally low, are not persistent and are not taken into account. Dioxin and related compounds present an excellent example of a case where background levels in the general population are likely to have significance for the evaluation of the relative impact of incremental exposures associated with a specific source.* ***Since RfDs refer to the total chronic dose level, the use of the RfD in evaluating incremental exposure in the face of a background intake exceeding the RfD would be inappropriate....*** *[EPA 1994] (emphasis added)*

In light of the existing public health threat from dioxin, regulatory policy must shift from approving "acceptable" exposures and discharges to preventing dioxin formation and release. According to EPA,

> *The weight of the evidence suggests concern for the impacts of these chemicals on humans at or near current background levels. Additional, incremental exposures occurring as a result of proximity to a point source of release or specific human activity patterns such as consumption of high levels of more highly contaminated foods, should be evaluated relative to background levels and the impact of the incremental exposure on both transient and steady-state body burdens.*
>
> *This situation is somewhat akin to the scientific approach taken for evaluating lead in children. This approach has been useful in providing public health-based advice to decisionmakers faced with difficult regulatory choices. [EPA 1994]*

EPA thus identifies the need for a wholesale shift in the regulatory approach to dioxin. EPA should immediately suspend its practice of approving additional discharges and exposures through permits, controls, acceptable discharges, and "reference doses." Instead, the agency should develop a coordinated national program to reduce human exposure to dioxin and prevent further release into the environment of dioxin and related compounds.



3: FORMATION OF DIOXINS

In its Reassessment, EPA makes a preliminary attempt to catalogue dioxin sources, and it correctly identifies incinerators that burn chlorinated waste and chlorine-bleaching pulp mills as primary sources. But EPA's list omits dozens of other dioxin sources* that have been identified in the scientific literature and government reports -- including those prepared by researchers within EPA. As a result, EPA's list of dioxin sources accounts for only 10 to 50 percent of the 25,000 g/year of dioxin that the agency estimates are emitted into the environment each year

Dioxin is never manufactured on purpose. Instead, it is formed as an unintentional by-product in scores of processes in which chlorine and chlorine-derived chemicals are produced, used, and disposed. In fact, the data suggest that all chlorine-based products may be associated with dioxin production at some point in their lifecycle. Dioxin and related compounds are produced in the following processes, implicating the entire field of chlorine chemistry:

- The chlor-alkali process in which elemental chlorine gas is produced from salt;
- All uses of chlorine gas (and other chlorinated oxidizing and bleaching agents) in industrial processes, such as the pulp, metallurgical, and water treatment sectors;
- Use of chlorine in the chemical industry to produce chlorinated organic (and some inorganic) compounds with a wide range of structures;
- Some uses of chlorinated organic chemicals, particularly those in reactive, alkaline, or heated environments;
- Incineration, combustion-based recycling, or accidental burning of any chlorine-containing product or of any waste from any of the above processes.

DIOXIN AND CHLORINE CHEMISTRY

Elemental chlorine gas is a highly reactive substance; it combines almost immediately with organic (carbon-containing) materials to produce organochlorines. The formation of organochlorines takes place quickly and randomly; this process is impossible to prevent and difficult to control. Thus, complex mixtures of hundreds or thousands of organochlorines are formed whenever chlorine comes into contact with organic material. These complex organochlorine mixtures frequently -- and possibly always -- contain dioxins and related compounds.

Virtually all industrial chlorine chemistry is suspected of generating dioxin-like compounds at some point during manufacture, use, and disposal. A number of lines of evidence support this judgment. First, dioxins are produced when chlorine is manufactured from salt in the chlor-alkali process, as discussed in chapter 5. Dioxin is thus implicated in all "downstream" processes -- the entire field of chlorine chemistry.

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* Throughout this document, processes are considered sources of dioxins or dioxin-like compounds if they involve the formation of any of the following: chlorinated dibenzo-p-dioxins, chlorinated dibenzofurans, hexachlorobenzene, or octachlorostyrene. Hexachlorobenzene itself contains dioxins nd furans at concentrations over 200 ppm. [Esposito 1980]. Given the structural similarity of HCB to dioxin and the occurrence of dioxin in HCB, the presence of HCB in any product or process points to the presence of dioxin. According to one study: "Hexachlorobenzene, octachlorostyrene, and other highly chlorinated compounds are formed under similar conditions as PCDD/F, and their presence in the emissions of a technical process should therefore be a good indicator for reaction mechanisms which may also create PCDD/PCDF. [Oehme 1989]

Second, dioxins are formed as trace by-products in the synthesis of the full range of chlorinated organic chemicals. In its preliminary dioxin reassessment documents, EPA notes,

> *"Dioxin-like compounds can also be formed during the manufacture of certain compounds such as chlorinated phenols, benzenes, and others. The releases associated with chemical manufacturing could not be quantified due to lack of test data. Potentially such releases could occur via the product itself or as emissions to the air, land or water." [Schaum 1993]*

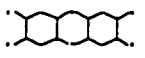
But the problem is much broader than this language suggests. Dioxins are known to be formed in the production not just of a few aromatic (benzene-based) chemicals, as EPA implies, but in a wide range of organochlorines, including those that do not resemble dioxin in their chemical structures – from simple to complex molecules, from aliphatics to aromatics, including solvents, pesticides, feedstocks for plastics, chemical intermediates, dyes, and others. Tables 3.2, 3.3, 3.4 and 3.5 present industrial and agricultural chemicals that are known or suspected of dioxin formation during their manufactur. Many but not all are benzene-based; aliphatic organochlorines associated with formation of dioxin and related compounds include 1,2-dichloroethane, carbon tetrachloride, trichloroethylene, tetrachloroethylene, hexachlorobutadiene, and epichlorohydrin. [Heindl 1987, Hutzinger 1988, Rossberg 1986].

Although a large number of chlorinated chemicals have not yet been tested for dioxin formation, the wide variety of products known to be associated with dioxin formation supports the hypothesis that dioxins are formed *throughout a full range of uses* in the chemical industry. On the other hand, the data contradict the notion that dioxin is associated only with a handful of products. The formation of dioxin in these chemical manufacturing processes cannot be prevented. Although producers attempt to exercise control over production conditions – such as temperature, pressure, and material purity – transient variations always occur, resulting in the production of complex by-product mixtures, including dioxin. In 1987, EPA recognized this fact:

> *The manufacture of halogenated organic chemicals results in the formation of small amounts of undesired side reaction by-products. These contaminants may be contained in the product chemical, separated into a processing step residue, or lost to the air or wastewater as a pollutant.... PCDDs and PCDFs are formed from a wide variety of chemicals, involving complex reaction pathways.... The large number of isomers is one of the reasons for the complexity and difficulty in the analysis and determination of dioxins and furans [as impurities in other chemicals.] (Lee 1987)*

Dioxins can also be formed when organochlorines are used, particularly in reactive environments – i.e., at elevated temperature or in chemical reactions. Dioxin production has thus been identified from the burning of vehicle fuels with chlorinated additives, the use of organochlorine catalysts in the steel industry, treatment of wood with pentachlorophenol, synthesis of chemicals and pharmaceuticals using organochlorines as intermediates, extractants, or reaction media, degreasing and cleaning of equipment with chlorinated solvents in the presence of strong alkalis or at elevated temperature.

Finally, dioxins are formed when any organochlorine is burned – in an incinerator, recycling facility, or in an accidental fire. According to EPA, "The formation of dioxin is generally understood to occur as a result of burning organic material with chlorine-containing material." (EPA 1986) Thus dioxins have been identified in the emissions and residues of the all types of incinerators burning wastes containing any organochlorine-



contaminated wastes, including hazardous wastes, garbage, medical waste, and sewage sludge. Large quantities of dioxins are also formed when organochlorine substances – such as PVC – burn in accidental fires or are introduced into combustion-based industrial processes such as primary and secondary smelters for copper, steel, aluminum, and iron.

Incineration is the dominant disposal method for spent organochlorine products (i.e., solvents, PVC, etc.) and for the chlorinated wastes that are produced whenever any organochlorine is manufactured. EPA has estimated that 37 percent of all wastes fed to incinerators are halogenated and that approximately 12.8 million metric tons of halogenated incinerable hazardous wastes are generated each year in the U.S. Of this quantity, 1.04 million metric tons are chlorinated solvents, 1.25 million metric tons are liquid chemical wastes, and 9.78 million metric tons are halogenated solids – primarily from the chemical manufacturing industry. [Oppelt 1987, Oppelt 1993]

In theory, incinerators are designed to convert organochlorines into hydrochloride acid, carbon dioxide, and water; real-world incinerators, however, never take this reaction to completion. The problem is not limited to poorly operated or aging incinerators; dioxins are formed in even the most sophisticated "state-of-the-art" incinerators According to EPA:

> *The complete combustion of all hydrocarbons to produce only water and carbon-dioxide is theoretical and could occur only under ideal conditions.... Real-world combustion systems virtually always produce PICs [products of incomplete combustion] some of which have been determined to be highly toxic. [EPA 1990]*

Dioxins are formed as products of the diverse and unpredictable reactions that take place in the furnace and the cooler zones of an incinerator. Localized and short-term variations from ideal combustion occur constantly in incinerators; when large-scale upsets (explosions, flame-outs, puffs, etc.) take place, even greater quantities of dioxin and other PICs are formed and released.

OTHER SOURCES: THE MYTH OF "NATURAL" DIOXIN

Industry advocates have argued that large amounts of dioxin are produced by "natural" sources, particularly forest fires, the domestic burning of wood, and the combustion of coal. This argument, put forward by Dow scientists as the "Trace Chemistry of Fire" theory, is intended to refute the idea that public policy should focus on the elimination of industrial sources of dioxin, particularly those associated with chlorine chemistry.

One aspect of the "Trace Chemistry of Fire" theory is correct, while the rest has been soundly disproven. It is possible for trace amounts of dioxin to be produced from salt, if enough energy is present to convert stable ionic chlorides into chlorine or organochlorines. Thus, dioxin can be formed in incinerators when salt is burned; according to one study, the quantity of dioxin formed per unit of chlorine is about half that when organochlorines are burned. [Danish EPA 1993] Other studies indicate that dioxin formation from salt combustion may be even lower: a German EPA study found high dioxin concentrations in the ash residue from the combustion of chlorinated plastics, chlorinated solvents, and pesticides, but concentrations were orders of magnitude lower or non-detectable when chlorine-free materials such as wood, paper, and chlorine-free plastics were burned, as shown in table 3.1. [Pohle 1991]

Notwithstanding the fact that burning organochlorines produces *more* dioxin, it is true that burning salt at high temperatures can produce dioxin. Thus, the introduction of salt to incinerators should be minimized. Since food and yard wastes – the major sources of salt in incinerators – are best composted and can be safely land disposed if necessay, there is no barrier to reducing the burning of salt-containing materials. The Netherlands, for instance, has a program to separate this "green waste" for compost from other types of municipal waste.

TABLE 3.1.
DIOXINS IN ASH FROM THE BURNING OF
ORGANOCHLORINES AND CHLORIDE-CONTAINING MATERIALS

Material (# of samples)	Total PCDD/F(ppt)	Total 2378-PCDD/F ppt	Total PCDD/F (TEQ- ppt)
Writing paper	ND	ND	ND
Wood, Cotton, Wool	ND	ND	ND
Polyethylene	2.9	ND	<.01
Chlorine-free polychloprene alternative [1]	2	ND	<0.1
Fir wood (w/residues?)	21.4	1.51	0.65
Coffee filters (2)	6.3-7.7	0.61-0.76	0.15-0.23
PVC plastic (10)	244-2067	7.5-122.1	3.2-42.2
PVDC plastic	3,304	20.5	14.1
Chloro-polyethylene plastic	840	27.6	10
Chloroparaffin	1,049	14	5.3
Polychloroprene plastic	323-1096	2.2-14.0	0.7-4.7
PVC flooring production samples	352-1847	25.7-38.4	8.2-14.5
PVC window-frame production samples	7.5-969	22.3-52.1	8.8-18.1
PVC cables (w/copper)	669-2970	26.2-129.4	11.4-52.6
PVC cables (w/o copper)	416-843	20.7-46.6	7.4-16.6
Other PVC products [2]	158-954	7.2-492	2.5-16.5
Dichloromethane	26,302	975	478
1,1,1-trichloroethane	21,746	831	340
Tetrachloroethane	9,072	37.9	132
Trichloroethylene	120,915	18.5	149.5
Perchloroethylene	212	1	0.4
Chlorobenzene	16,135	0.7	0.5
p-Chloronitrobenzene	190,096	29.7	21.5
o-Chloronitrobenzene	32,293	1,331.9	216
p-Chlorotoluene	1,033	ND	ND
Epichlorohydrin	1,532	117	36
2,4-D	178,016	168	361
Lunuron (pesticide)	3,110	253	32

1. Acrylonitrile-butadiene rubber]
2. PVC gloves, hose, pipes, tape, etc.

Source: Pohle 1991

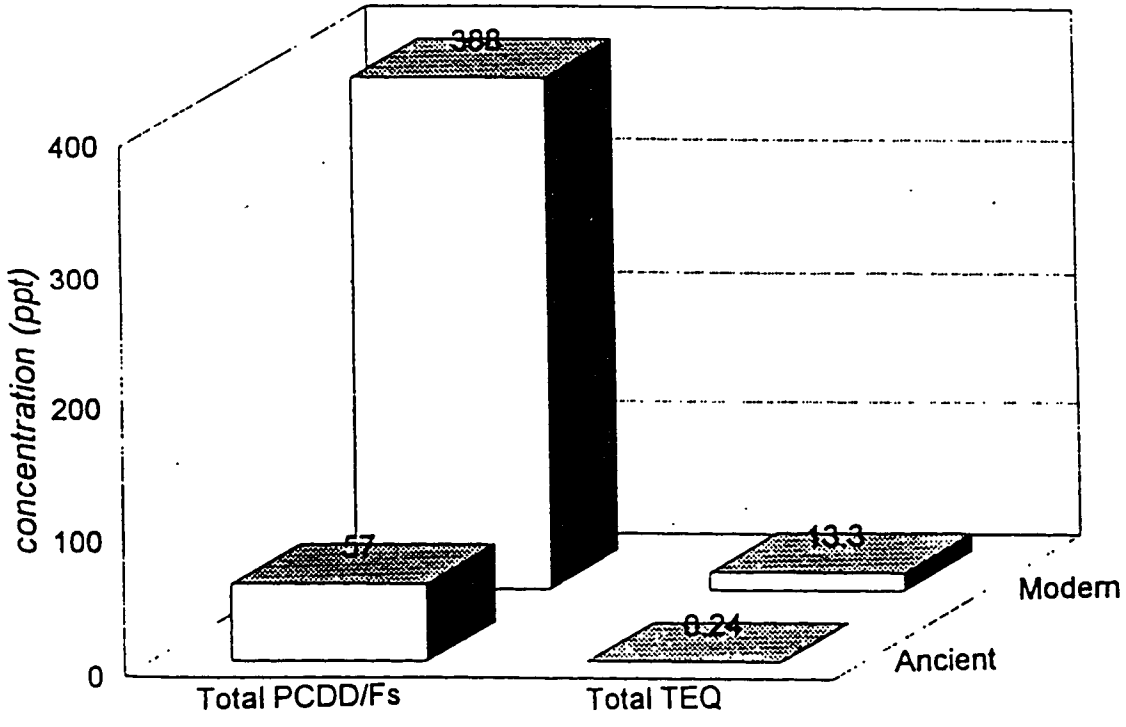
But the most important part of the Trace Chemistry of Fire theory – that much or most dioxin can be explained by "natural" combustion unrelated to chlorine chemistry – has been proven false. Several lines of evidence indicate that virtually all dioxin found in the environment is produced by industrial sources associated with the production, use, and combustion of chlorine and organochlorine chemicals – not salt combustion.

Second, historical trends in dioxin levels in human tissues and sediment cores clearly show that dioxin pollution was not a problem before industrialization and the development of chlorine chemistry in particular. Studies of sediment and soil patterns in the Great Lakes show that dioxin pollution was negligible or non-existent before the rise of chlorine chemistry in the Twentieth Century, and that levels skyrocketed only when chlorine chemistry rapidly expanded after World War II, as shown in figure 2.2. [Czuczwa 1986]

Further, two studies have found that dioxins and furans are almost completely absent from the tissues of pre-industrial humans who habitually burned wood indoors and were thus highly exposed to wood smoke. Dioxin levels (TEQ) were less than 2 percent of the levels found in current human tissues, as shown in figure 3.1.. [Schechter1991] Based on these studies, EPA has concluded:

> *"The theory that much of today's body burden could be due to natural sources (such as forest fires) has been largely discounted by testing of ancient tissues which show levels much lower than those found today." [Schaum 1993]*

Figure 3.1
Dioxin concentrations in ancient and modern humans.



Source: Schechter 1991.

Third, the data do not show that the burning of wood per se produces significant amounts of dioxin. Organochlorine pesticides, solvents and other chemicals have been distributed throughout the global environment, and they have been deposited on and absorbed into trees, their wood and foliage, and other forest materials. Thus, a forest fire or a domestic wood fire burns industrial chemicals in addition to natural wood, and dioxin emissions are most likely a reflection of these chlorine inputs. According to EPA,

> *"Only one test has directly measured CDD/CDFs in the smoke from forest fires, and the authors cautioned that all or a portion of these emissions could represent resuspended material from aerial deposits rather than originally formed material." (Schaum 1993)*

Further, a recent industry study found that the burning of natural wood results in very low dioxin emissions, while the burning of waste wood chips and wood in the presence of household waste results in dioxin emissions that are higher by a factor of 100 to 1000. [Schatowitz 1993] Danish studies indicate that the high dioxin emissions documented in emissions from wood stoves were caused by the burning of wood impregnated with the wood preservative pentachlorophenol. [Vikelsoe 1993] For this reason, the German EPA has prohibited the burning of PVC-coated wood in home or industrial stoves and furnaces.

EPA's estimates that forest fires emit 300 to 3000 grams/year of dioxin per year, industrial wood burners 70-1600 g/year, and domestic wood burners 40-400 g/year. If these figures were accurate, these "natural" sources would account for only 1.6 to 20 percent of EPA's estimate of total U.S. dioxin releases. But EPA admits that these numbers are almost certainly significant overestimate. EPA derived its figures for both sources by combining total particulate generation rates with the levels of dioxin found in chimney soot, admitting that "dioxin levels in soot are likely to be much higher than what is actually emitted on particulates due to accumulation in chimneys." [Schaum 1993]

For all the reasons discussed above, then, it appears that the contribution of salt combustion to total dioxin emissions is less than one percent, while the vast majority is related to chlorine chemistry. The burning of chlorides in combustion facilities should be minimized through the removal of food and yard waste, but the focus of any dioxin reduction program must be on the products and processes associated with industrial chlorine chemistry.

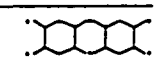


Table 3.2
Pesticides with Dioxin Formation Known or Suspected in Manufacture

Dioxin contamination known

Brenox //methyl-5-(2,4-dichlorophenoxy-2-nitrobenzonate
Chloranil // 2,3,5,6-tetrachloro-2,5-cyclohexadiene-1,4-dione
Chlorophenols, "various"
2,4-D// (2,4-dichlorophenoxy)acetic acid
2,4-DB salts//2,4-dichlorophenoxybutyric acid
2,4-DP//2-(2,4-dichorophenoxy)propionic acid
Dicamba, dimethylamine salt // 3,6-dichloro-2-methoxybenzoic acid,
Dicamba //3,6-dichloro-2-methoxyenzoic acid
Dicapthon//Phosphorothioic acid, o-(2-chloro-4-nitrophenyl)-o,o-dimethyl ester
Dichlofenthion // Phosphorothioic acid, o-2,4-dichlorophenyl o,o-dialkyl ester
Disul sodium (sesone) //2,4-dichlorophenoxy ethyl sulfate, sodium salt
DMPA
Erbon
Erbon // 2,2-dichloropropanoic acid, 2-(2,4,5-trichlorophenoxy) ethyl ester
Hexachlorophene // 2,2'-methylene-bis(3,4,6-trichloro-phenol)
Isobac 20 // 2,2'-methylene bis (3,4,6-trichlorophenol), monosodium salt
Nitrofen// 2,4-dichlorophenyl-p-nitrophenyl ether
Pentachlorophenol (PCP) and salts
Ronnel //,phosphorothioic acid, o,o-dimethyl-O-(2,4,5-trichlorophenyl) ester
Sesone
Silvex/2-(2,4,5-trichlorophenoxy)propionicacid
2,4,5-T // (2,4,5-trichlorophenoxy) acetic acid
2,4,5-trichlorophenol
2,3,4,6-tetrachlorophenol
Tetradifon

Dioxin contamination "possible"

Akton // O-(2-chloro-1-(2,5-dichlorophenyl)-vinyl)- O,O-diethyl phosphorothioate
Alkyl dimethyl 3,4-dichlorobenzyl ammonium chloride
Carbonphenothion // Phosphorodithioic acid, s-((4-Chlorophenyl)thio)methyl) o,o-daithyl ester
Carbophenothion
Chloranil
Chlorbromuron
5-chloro-2-(2,4-dichlorophenoxy)phenol
1-(4-chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone
Chloroneb // 1,4-dichloro-2,5-dimethoxybenzene
Chlorothalonil
Chloroxuron // 3-(4-(4-chlorophenoxy) phenyl-1,1-dimethylurea
Chlorthiophos
Cycloate // 4-tert-butyl-2-chlorophenyl methyl methylphosphoramidate
DCPA//2,3,5,6-tetrachloro-1,4-benzene- dicarboxylic acid, dimethyl ester
DDT
Decachlorobis (2,4-cyclopentadiene-1-yl)
Dichlobenil // 2,6-dichlorobenzonitrile
Dichlofenthion//O-(2,4-dichlorophenyl)-O,O-diethyl phosphorothioate
Dichlone
Dichlone // 2,3-dichloro-1,4-naphthalenedione
Dicofop-methyl
2,4-dichlorophenyl p-nitrophenyl ether (TOK)
2,4-dichloro-6-(o-chloroanilino)-s-triazine
2,6-dichloro-4-nitroaniline
Diflubenzuron // N-[(4-chlorophenyl) amino)carbonyl)-2,6-difluorobenzamide]
Dimethyl tetrachloroterephthalate
Dinitrobutylphenol, ammonium salt
4,6-dinitro-o-cresol
Diuron// 3-(3,4-dichlorophenyl)-1,1-dimethylurea
Endosulfan
Erbon
Fenvalerate // cyano(3-phenoxyphenyl)methyl-4- chloro -alpha-(1-methylethyl)benzeneacetate
Fluvalinate// N-2-chloro-4-trifluoromethyl)-phenyl-DL- valine(+)-cyano-(3-phenoxyphenyl) methyl ester
Fungaflor // 1-(2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl)-1H-imidazole
Gardona // 2-chloro-1-(2,4,5-trichlorophenyl) vinyldimethyl phosphate
Hexachlorobenzene
Imazalil
Ioxynil
Kelthane // 1,1-bis(chlorophenyl)-2,2,2-trichloroethanol
Lindane // g-hexachlorocyclohexane
MCPA // (4-chloro-o-tolloxyl) acetic acid
MCPB // 4-(2-methyl-4-chlorophenoxy) butyric acid
Mecoprop // 2-(4-chloro-2-methylphenoxy) propionic acid
2,2'-methylene-bis(3,4,6-trichloro-phenol)
2,2'-methylene-bis(4-chlorophenol)
Monuron trichloroacetate // 3-(p-chlorophenyl)- 1,1- dimethylurea trichloroacetate
Neburon // 1-butyl-3-(3,4-dichlorophenyl)-1-methylurea
Oxadiazon // 2-tert-butyl-4-(2,4-dichloro-5--isopro poxy-phenyl)delta-1,3,4-oxadiazolin-5-one)
o-Benzyl-p-chlorophenol
o-Dichlorobenzene
O-(4-bromo-2,5-dichlorophenyl)-O,O-dimethyl phosphorothioate
Parathion // phosphorothioic acid, o,o-diethyl-o-(4-nitrophenyl) ester
Pentachlorobenzonitrile
Pentachloronitrobenzene
Pentachlorophenyl laurate
Pipecolinopropyl-3,4-dichlorobenzoate
Piperalin // 3-(2-methylpiperidino) propyl-3,4-dichlorobenzoate
Planavin // 4-methylsulfonyl)-2,6- dinitro-N,N-dipropylaniline
Profenofos // O-(4-bromo-2-chlorophenyl)- O-ethyl S-propyl phosphorothioate)
Propanil // 3,4-dichloropropionanilide
p-Chlorophenyl-2,4,5-trichlorophenylsulfide
p-Dichlorobenzene
Rabon // 2-chloro-1-(2,4,-dichlorophenyl) vinyl diethyl phosphate
Ronilan // 3-(3,5-dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidine-dione
Sesone
Sodium pentachlorophenate
1,2,4,5-tetrachloro-3-nitrobenzene
Tetrachloroisophthalnitrile
Tetrachlorophenate
Tetrachlorophenols
2,2'-thiobis(4-chloro-6-methylphenol)
2,3,6-trichlorobenzoic acid
Trichlorobenzoyl chloride
2,3,6-trichlorophenylacetic acid or sodium salt

Sources: Esposito 1980, PTCN 1985.

Note: original list included compounds likely to produce any halogenated dibenzodioxins, including brominated, fluorinated, and iodinated compounds. These compounds have been omitted from this list. However, non-halogenated products made from chlorinated intermediates have been maintained.

Table 3.3.
Commercial Chemicals with Dioxin Formation Known or Suspected During Manufacture

HIGH PROBABILITY OF DIOXIN FORMATION

4-bromo-2,5-dichlorophenol
2-chloro-4-fluorophenol
2,4-dibromophenol
2,3-dichlorophenol
2,4-dichlorophenol
2,5-dichlorophenol
2,6-dichlorophenol
3,4-dichlorophenol
2,4,5-trichlorophenol
2-chloro-1,4-diethoxy-5-nitrobenzene
5-chloro-2,4-dimethoxy-aniline
chlorohydroquinone
o-chlorophenol
2-chloro-4-phenylphenol
4-chlororesorcinol
3,5-dichlorosalicylic acid

POSSIBILITY OF DIOXIN FORMATION

o-chlorofluorobenzene
3-chloro-4-fluoro-nitrobenzene
3-chloro-4-fluorophenol
4-chloro-2-nitrophenol
chloropentafluorobenzene
3,4-dichloroaniline
o-dichlorobenzene
3,4-dichlorobenzaldehyde
3,4-dichlorobenzotrichloride
3,4-dichlorobenzotrifluoride
1,2-dichloro-4-nitrobenzene
3,4-dichlorophenylisocyanate
1,2-dihydroxybenzene-3,5-disulfonic acid, disodium salt
2,5-dihydroxybenzenesulfonic acid
2,5-dihydroxybenzenesulfonic acid, potassium salt
2,4-dinitrophenol
2,4-dinitrophenoxyethanol
3,5-dinitrosalicylic acid
fumaric acid
hexachlorobenzene
maleic acid
maleic anhydride
o-nitroanisole
2-nitro-p-cresol
o-nitrophenol
pentachloroaniline
o-phenetidine
phenol (from chlorobenzene)
1-phenol-2-sulfonic acid, formaldehyde condensate
phenyl ether
phthalic anhydride
picric acid
sodium picrate
1,2,4,5-tetrachlorobenzene
tetrachlorophthalic anhydride
1,2,4-trichlorobenzene
2,4,6-trinitroresorcinol

Source: Esposito 1980.
Original list included compounds likely to produce any halogenated dibenzodioxins, including brominated, fluorinated, and iodinated compounds. These compounds are omitted from this list. Non-halogenated products made from chlorinated intermediates included.

Table 3.4.
20 Pesticides Known or Suspected to be Contaminated with Hexachlorobenzene

* DCPA (Dacthal)
* Chlorothalonil (Daconil 2787, Bravo, Termil)
* Pentachloronitrobenzene (PCNB,Terrachalor)
5,6,7,8-Tetrachloroquinoxaline (Chlorquinox)
Pentachlorophenol (Chlorphen)
Pentachlorophenol zinc salt
Pentachlorophenol sodium salt
Pentachlorophenol potassium salt
Dehydroabietylamine pentachlorophenate
1,2,4,5-Tetrachloro-3-nitrobenzene (Folosan)
2,2,2-Trichloro-N-(pentachlorophenyl)acetimidoylchloride
2,3,5,6-tetrachloro-N-methoxy-N-methyl-terephthalamate
2,4,5 Trichlorophenoxyacetic acid (2,4,5-T)
2,3,6-Trichlorobenzoic acid (2,3,6-TBA)
Endrin
Allyl chloride/dichloropropene
1,2,3-Trichloropropane
Dehydroabietylamine pentachlorophenate
* Picloram

* Manufacturers have acknowledged the presence of hexachlorobenzene as an impurity in these products.
Source: PCTN 1985.

Table 3.5.
Chemicals Known to Generate Hexachlorobenzene in Manufacture

Carbon tetrachloride
Chlorinated benzenes
Chlorine
Hexachlorocyclopentadiene
Pentachloronitrobenzene
Pentachlorophenol
Tetrachloroethylene
Trichloroethylene

Source: USEPA 1985b.

4: PRIORITY SECTORS: MAJOR DIOXIN SOURCES

EPA's investigation, along with other information, points to three major processes as the largest dioxin sources. These sectors – incinerators, chlorine-bleaching of pulp and paper, and PVC plastic – account for the majority of known dioxin emissions.

1. INCINERATION OF CHLORINE-CONTAINING WASTES

The largest group of identified dioxin sources are waste incinerators. EPA estimates that total dioxin emissions from such plants total 565 to 5343 g/year. Because EPA omitted certain major dioxin sources in this category (cement kilns burning hazardous waste, contaminated ash residue from waste incinerators and other combustors, etc.) and underestimates emissions from others, actual emissions are even higher. EPA identifies the following incinerator types as major dioxin sources [Shaum 1993, Cleverly 1993]:

* Hospital waste incinerators, which burn primarily packaging, containers and disposable implements such as gloves and tubing. Chlorine is present primarily from PVC. EPA estimates annual dioxin emissions at 500-5100 g/year.

* Garbage incinerators. EPA estimates air emissions at 60-200 g/year, but 1992 tests on a *single* garbage incinerator in Columbus, Ohio, revealed average dioxin emissions of 984 g/year (TEQ). [OEPA 1994] In other words, a single incinerator was found to emit up to 16 times more than EPA's total estimate for all of the nation's 120 incinerators. Of these, 40 are like the Columbus facility in their use of electrostatic precipitators – pollution control devices that operate at temperatures in which dioxin formation is maximized. [Connett 1994] If the Columbus incinerator trial burn is representative, emissions from these 40 incinerators alone would total close to 40,000 g/year of dioxin, and sa range of 4,000-4,0,000 g/year can be assumed until more data are available.

In additionto air emissions, much greater quantities are deposited in the ash residues from these facilities, which are typically disposed in landfills, where leaching occurs over time. Chlorine is present primarily from PVC, with additional amounts entering in bleached paper, other organochlorine chemicals (i.e., solvents, paint strippers, paints, and pesticides) and chloride from vegetable materials.

* Hazardous waste incinerators. EPA estimates air emissions from these facilities at only 2.5-8.4 g/year. This figure, however, significantly underestimate actual emissions, for a number of reasons. First, it is derived from a single trial burn at one incinerator; it is widely recognized that trial burns underestimate actual emissions, because neither operating conditions nor the materials burned resemble those of daily operation. Further, EPA does not include dioxin in ash and liquid residues from hazardous waste incinerators; releases in these forms may be 100 times greater than those in air emissions – or even greater – if pollution control devices are at least 99 percent effective, as incinerator operators claim.

Trial burns in 1993 at the Waste Technologies Industries "state-of-the-art" hazardous waste incinerator revealed mean dioxin emissions of 1.2 grams per year (TEQ), or 13.6 ug/ton of waste burned. [ENSR 1993] An estimated 2 million tons of hazardous waste are burned each year in incinerators, according to EPA. If all U.S. incinerators achieved the dioxin emissions found in an idealized trial burn at this "state-of-the-art," annual air emissions would total some 27.2 grams per year. Real-world operation and upset conditions may increase dioxin emissions by a factor of up to 10; emissions from this sector would thus total up to 272 grams per year.

If pollution control devices are 90 to 99 percent effective, ash from these incinerators would carry an additional 270 to 27,000 grams of dioxin (TEQ) into the environment per year.

* Sewage sludge incinerators. EPA estimates emissions from these facilities at 1-26 g/year, based on tests at three facilities. Dioxin emissions are the result of organochlorines in the sludge, primarily due to the chlorination of sewage, with smaller amounts entering in industrial discharges into sewer systems.

* Drum and barrel reclamation facilities that recycle used chemical containers. EPA estimates annual dioxin emissions at about 2 g/year. Chlorine enters these facilities as chemical residues in drums and barrels.

* Vehicle fuels. Chlorinated compounds, such as 1,2-dichloroethane, are added to leaded gasoline and some other fuels as scavengers and for other purposes. The combustion of these chemicals in engines results in dioxin formation. EPA estimates that vehicle emissions result in the release of 8 to 870 grams of dioxin per year (TEQ). Fuels that do not contain chlorinated additives do not produce dioxin emissions. [Marklund 1990] Germany has prohibited the use of chlorinated additives in vehicle fuels.

Table 4.1
Dioxin Formation from Incinerators

Facility type	*Dioxin Releases (g/yr - TEQ)* EPA estimate	revised estimate
Hospital waste incinerators	500-5100	
Municipal waste incinerators		
air emissions	60-200	4,000-40,000
ash	–	600-20,000
Hazardous waste incinerators		
air emissions	2.5 - 8.4	27-270
ash	–	270-27,000
Cement kilns burning hazardous waste		
air emissions	–	800
dust	–	118
Other industrial furnaces burning hazardous waste	–	ND
Sewage sludge incinerators	1-26	
Tire burning (incinerators, kilns, etc.)	–	ND
Vehicle fuels	8-870	
Drum and barrel reclaimers	1-3	
Carbon regeneration facilities	1	

Sources: Schaum 1993, Cleverly 1993. Revisions explained in this text. All estimates are preliminary and are subject to significant uncertainty.

In addition to these combustion sources, there are several types of incinerators that are known dioxin sources from which EPA did not estimate dioxin emissions, including:

* Cement kilns that burn hazardous waste. Although cement kilns burn more hazardous waste than officially designated commercial incinerators and are known to be important dioxin sources, EPA made no estimate of dioxin emissions from these facilities. Data indicate, however, that these are important dioxin

sources. A 1993 testing program for EPA examined dioxin emissions at 15 U.S. cement kilns that burn hazardous waste; mean dioxin emissions per kiln were estimated at 20 grams of dioxin (TEQ) per year. [Schreiber 1993] Based on this figure, total dioxin emissions from the 34 waste-burning cement kilns and the 6 waste-burning aggregate kilns in the U.S. would total 800 g/year. These estimates do not include emissions from the approximately 200 other boilers/industrial furnaces that burn approximately 500,000 tons of hazardous waste per year.

Cement and aggregate kilns release even greater amounts of dioxin in their solid residues, including kiln dust and the cement products themselves. In a 1994 Report to Congress, EPA found dioxins and furans in dust from five of six waste-burning cement plants sampled, with average concentrations of 42.2 ppt TEQ. [EPA 1993] Waste-burning cement kilns generate about 2.78 million metric tons of dust per year. [EPA 1993] Based on these figures, dust from these kilns carries 118 grams of dioxin (TEQ) into the environment each year. EPA found that these dusts enter the environment during storage, transport, and disposal, and concluded that significant uptake into the foodchain and human exposures are possible. [EPA 1993]

Dust from aggregate kilns adds an additional but unknown quantity of dioxin. Dioxins are also deposited into cement and aggregate products, but no data are available to estimate annual quantities.

* Tire incinerators. Only limited data are available on emissions from tire burners. One test at a "state-of-the-art" dedicated tire incinerator in Modesto, CA, found significant dioxin emissions from that facility. Burning 5800 pounds per hour of tires, the incinerator emitted dioxins and furans at a rate of .0236 grams per year (TEQ). [CARB] Tires are burned in cement plants, pulp mill boilers, tire incinerators, and utility boilers. [Blumenthal 1992] Because no data are available on the total number of tires burned each year, total national annual emissions from such facilities cannot be estimated. Small quantities of chlorinated chemicals are added to tires as additives, stabilizers, plasticizers, etc.

2. CHLORINE BLEACHING AT PULP MILLS

EPA identifies the use of chlorine and chlorine-based bleaches in the pulp and paper industry as the largest source of dioxin discharges directly into waterways. Large quantities of dioxin are also released into the air, into paper products, and into the land when contaminated sludge is disposed. Total dioxin emissions from pulp mills are estimated at 238-1166 grams/year (TEQ). [Schaum 1993, Cleverly 1993] EPA makes no estimate of dioxin releases from the incineration of pulp mill sludge, a process known to generate dioxins.

TABLE 4.2

DIOXIN RELEASES FROM PULP MILLS (g/yr - TEQ)

Medium	Low	High
Water	56	360
Land (sludge)	84	300
Products	97	500
Kraft liquor boilers	1	6
Sludge incineration	ND	ND
TOTAL	238	1166

Sources: U.S. EPA (Schaum 1993, Cleverly 1993) ND = no data.

Many pulp mills have switched from chlorine gas to chlorine dioxide bleaches, thus reducing dioxin emissions by as much as 80 percent. EPA's estimates of dioxin releases reflect this change, with the lower end of the range representing estimates from a "modernized" paper industry using chlorine dioxide bleach. According to EPA, the upper estimate was based on a 1988 survey, and the lower estimate represents conditions believed to be more representative of current release rates, though not yet independently confirmed.* Even under this scenario, dioxin releases to all media still total over 235 g/year, making even "modernized" chlorine bleaching pulp mills one of the largest dioxin-producing sectors known.

Significant quantities of dioxin have also been identified in consumer products made from the pulp mill sludges and wastes. Dioxin concentrations ranging from 9.5 to 200 ppt have been detected in tall oil acid, tall oil resins, and soaps and detergents made from tall oils from chlorine-bleaching pulp mills. [Rappe 1990]. Paper products made from chlorine dioxide-bleached pulp also contain dioxin; concentrations range from 0.2 to 1.3 ppt. [Gruber 1993]

3. *PVC PLASTIC*

PVC plastic is the largest single use of chlorine, consuming almost 4 million tons of chlorine in the U.S. – about 30 percent of all the chlorine produced. PVC is used in pipes, flooring, wall coverings, packaging, furniture, automobiles, office products, appliances, cables, and certain medical products.

Since PVC is the largest single use of chlorine, it is no surprise that it results in more dioxin production during its lifecycle than any other product. Dioxin is produced and released in the manufacture of PVC feedstocks, the burning of wastes from production of PVC and its feedstocks, the incineration of PVC products in trash and hospital incinerators, the secondary smelting of metal products containing PVC (such as automobiles and PVC-coated copper cables), and the accidental burning of PVC in fires.

TABLE 4.3
Dioxin sources associated with PVC: preliminary estimates

Process	*Dioxin released (TEQ g/yr)*	*% caused by PVC*	*Dioxin released due to PVC (TEQ g/yr)*
Wastes from EDC/VC synthesis	500-1000	100	500-1000
PVC products	10-100	100	10-100
Municipal Waste Incinerators	4,000 *	50	2,000
Hospital Waste Incinerators	500-5100	75	375-3825
Copper reclamation plants	230-310	75	166-230
Steel reclamation plants	10-110	50	5-55
Building fires	500-5,000	75	375-3750
TOTAL			3,430 - 10,960

* Low end of revised national estimate, based on trial burn at Columbus, Ohio, incinerator. (OEPA 1994)
Due to limited database, all estimates are preliminary and subject to significant imprecision. Basis for estimates explained in text.

PVC Manufacture

A series of European studies have clearly established that very large quantities of dioxin are formed in the production of vinyl chloride monomer – the building-block for PVC. Dioxin formation is particularly significant in the "oxychlorination" stage, when the feedstock ethylene dichloride is combined with hydrochloric acid and oxygen – in the presence of a copper catalyst – to produce VCM. Dioxins have been detected in the wastes (or "tars") from this process, in air emissions, water discharges, and in sediments downstream near discharge pipes from these facilities. Dioxins are only one group in a complex cocktail of by-products formed in VCM manufacture, including hexachlorobenzene, hexachlorobutadiene, hexachloroethane, carbon tetrachloride, and other "heavy" and "light" organochlorines.

In May 1994, the Swedish Environmental Protection Agency found that PVC plastic itself contains measurable quantities of dioxins and furans. [SEPA 1994] Pure PVC suspension from two Swedish PVC producers was found to contain a full range of congeners of dioxins, furans, and PCBs, shown in table 4.4. Total concentrations, including PCBs, ranged from 0.86 to 8.69 ppt (TEQ). Based on these figures and annual U S. production of 5.7 million tons, PVC products carry an estimated 4 to 40 grams of dioxin (TEQ) into the U.S. environment each year.

TABLE 4.4
DIOXIN IN PVC PLASTIC PRODUCTS

Dioxin congener	Sample 1 - PVC plastic (ppt)	Sample 2 -- PVC Plastic (ppt)
2378-TCDD	<0.1	0.1
12378-PeCDD	<0.2	0.6
123478-HxCDD	<0.4	0.3
123678-HxCDD	0.4	1.5
123789-HxCDD	<0.4	1.1
1234678-HpCDD	26.	5.4
2378-TeCDF	0.6	2.7
12348(78)-PeCDF	0.2	8.2
23478-PeCDF	0.2	6.2
234678-HxCDF	<0.2	8.2
123478(9)-HxCDF	0.1	4.9
123678-HxCDF	<0.1	4.
123789-HxCDF	<0.3	1.6
1234789HpCDF	<0.4	2.4
1234678-HpCDF	1.	13.
OCDF	<0.9	7.4
OCDD	55.	6.
3344-TeCB	7.80	31.00
PeCB (118)	450.00	399.00
33445-PeCB	1.20	18.00
PeCB (105)	58.00	68.00
334455-HxCB	<.70	4.70
Total PCDD/F/B	<604.2	594.3
Total (TEQ)	0.86	8.69

Source: Swedish EPA 1994. Two samples of pure PVC suspension from two Swedish PVC plants were analysed.

Numerous earlier studies have shown that dioxins are produced in significant quantities in the production of EDC as a feedstock for PVC plastic:

- A 1989 study by the University of Amsterdam estimated, based on a laboratory simulation of the oxychlorination process for the production of EDC that 419 grams of dioxin (TEQ) are formed for each 100,000 tons of EDC produced. [Evers 1989]
- In early 1994, the German EPA found extraordinarily high dioxin concentrations in wastes and wastewater treatment sludge from ICI's VCM/PVC plant in Wilhelmshaven, Germany, as shown in table 4.5. This plant is considered a modern factory. [Lower Saxony 1994]
- VCM plants appear to be the most important source of dioxins in the North Sea and the River Rhine, according to a 1993 study of the dioxin fingerprint in sediments conducted by the Dutch Government and the University of Amsterdam . [Evers 1993]
- In 1990, dioxins were found in the wastewater discharges from Solvay's VCM plant in Rheinberg, Germany; total discharges were estimated at up to 8 grams TEQ/year. [Adelt 1990]
- In 1992, Norsk Hydro analyzed wastes from its VCM plant in Stenungsgund, Sweden; dioxins were found in four waste streams, and total dioxin discharges into wastes were estimated at 321 grams TEQ/year. [Hydro-Plast 1992] A 1993 study by the University of Umeaa confirmed the occurrence of dioxins in wastes from this facility. [Andersson 1993]
- A 1988 study by the University of Amsterdam found that the fingerprint of dioxin congeners in sediments from the River Rhine suggested that HTA* VCM plant was a major source of dioxin into that river. [Evers 1988]
- A 1988 study by the Stichting Reinwater, a Dutch environmental organization, found that concentrations of dioxins and furans in the Rhine were significantly higher downstream from Solvay's VCM plant in Rheinberg, Germany, than upstream. [Verhoog 1988]
- A 1992 study by several Swedish universities found high levels of hexachlorobenzene, pentachlorobenzene and several congeners of dioxins and furans in sediments near Norsk Hydro's plant in Stenungsund. [Cato 1992]
- In 1993, Greenpeace commissioned the University of Amsterdam and SAL laboratory in the UK to analyze samples of effluent from a Solvay VCM plant and soil samples from the vicinity of a Norsk-Hydro oxychlorination reactor in Sweden. Both contained significant quantities of dioxins and furans, as shown in Table 4.6. [Johnston 1994]

*Hals Troisdorf Ag

Table 4.5
Dioxin from ICI's VCM/PVC plant in Wilhelmshaven, Germany

	Dioxin concentration ng/kg TEQ
Metal sludge	408,270
Metal sludge cake	413,790
Wastewater treatment sludge	7,199
Sediment near discharge pipe	1.7 - 3.9
Mussels near discharge	0.71 - 4.44

Source: Lower Saxony Ministry of Environmental Affairs, 1994.

Thus, this body of evidence shows that dioxins can be produced at a rate of up to 400 g TEQ per 100,000 tons of EDC. Using a very conservative estimate of 5 to 10 grams dioxin (TEQ) per 100,000 tonnes of EDC [Wang-Anderson 1992], and assuming U.S. production of 10.4 million tonnes of EDC per year, dioxin formation from this sector is estimated at 500-1000 grams (TEQ) per year.

Table 4.6
Dioxins in Effluent and Soil from two EDC/VCM plants in Europe

	Effluent		*Waste (dry wt.)*	
Congener	pg/L	pg/L (TEQ)	ng/g	ng/g (TEQ)
2378-TCDD	nd	-	nd	-
12378-PeCDD	nd	-	nd	-
123478-HxCDD	nd	-	nd	-
1234678-HpCDD	21	0.2	nd	-
OCDD	125	0.1	1.7	.001
Other isomers			5.02	
2378-TCDF	12	1.2	nd	-
12378-PeCDF	7.3	0.4	nd	-
23478-PeCDF	7.3	3.7	nd	-
123478-HxCDF	12	1.2	3.4	0.34
123678-HxCDF	18	1.8	.65	0.07
234678-HxCDF	nd	-	nd	-
123789-HxCDF	nd	-	1.7	0.17
1234678-HpCDF	228	2.3	17	0.17
1234789-HpCDF	24	0.2	nd	-
OCDF	4621	4.6	6700	6.7
Other isomers			165.8	
Total	5068.3	15.7	6885.2	7.5

Source: Johnston et al 1994.. TEQ based on NATO-ITEQ system.

The Norwegian government has also estimated., based largely on industry-supplied data, annual dioxin emissions from its VC/EDC plants into various media: 25 to 250 mg to air, 6-100 mg into wastewater, 420 mg into sludge, 7.3 grams into EDC tars, and 10 grams in internal process streams. [SFT 1994] Assuming that U.S. production of VC/EDC is 15 times larger than that in Norway [SRI 1993], annual dioxin releases from the U.S. industry can be estimated as follows:

Air:	0.38 to 3.8 g/year (TEQ)
Water	0.09 to 1.52
Sludge	6.384
Tars	110.
Internal streams	152
Total	268 - 272 g/year (TEQ)

EPA did not acknowledge the body of evidence indicating that PVC manufacture is a major dioxin source, citing a "lack of data." According to the agency,

> *Recently, some claims have been made that significant dioxin emissions may occur during the production of vinyl chloride monomer and associated products. However, insufficient emission data are currently available to evaluate these reports. [Schaum 1993]*

Incineration of PVC products

PVC is the largest waste source associated with dioxin emissions from garbage and hospital waste incinerators. In numerous studies of combustion processes, PVC has been identified as a precursor for dioxin formation. [Christman 1989, Thiesen 1989] According to one scientific report,

> *"Samples from real fire accidents as well as from laboratory combustion tests ... demonstrate that in case PVC-containing materials take part in combustion processes, PCDD/Fs can be found in the decomposition products in considerable concentrations. Therefore, these results confirm the classification of PVC-containing materials as PCDD/F precursors. [Thiesen 1989]*

The German EPA has estimated that rigid PVC – such as bottles and pots – is responsible for approximately 50 percent of all chlorine fed to municipal waste incinerators, though it constitutes only 0.5 percent of the total mass of wastes. [Brahms 1989]

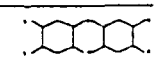
The Dutch Environment Ministry has estimated that PVC constitutes 40 percent of all chlorine – in either organic or inorganic form – fed to garbage incinerators, and the majority of organic chlorine. Analytical studies by the Ministry demonstrated that reducing the fraction of PVC burned results in a corresponding reduction in the emissions of dioxin and chlorophenols. [Boerekamps-Kanters 1993]

Several reports have found a direct relation between the amount of PVC in the waste fed into an incinerator and the amounts of dioxin emitted. [Ozvacic 1990; Danish EPA 1993]. In the Netherlands, where an effective garbage separation system removes most of the organic chlorides in compostable materials such as food and wood wastes, PVC remains the only major source of chlorine and the major dioxin precurso; reducing PVC feed results in significant decreases in dioxin emissions. [Boerekamps Kanter 1993] Based on these findings, the Dutch Environment Ministry concluded:

> *"These new experiments by the University of Leiden demonstrate clearly a relation between the content of PVC in household waste and dioxin formation in waste incinerators. On the basis of these experiments there is no reason to reconsider present policies regarding PVC applications: the main feature of this policy is that PVC applications for which no feasible system of recycling and reuse can be established the use of more environmentaily sound alternative materials is to be preferred." [Netherlands Environment Ministry 1994]*

In 1993 the Danish EPA found that "during incineration chlorinated dioxins and dibenzofurans are formed, and although emissions are reduced significantly by flue gas cleaning and better operation conditions, these persistent substances can still be spread and accumulated through food chains. PVC is the most significant individual source of chlorine in municipal waste." [Danish EPA 1993]

PVC is also the largest single source of chlorine in hospital waste incinerators. Chlorinated plastics (PVC) account for 9.4 percent of the total weight of "red bag" infectious waste, according to one study. [Marrack 1988] PVC is used in hospitals for packaging, containers, bags, tubing, and other uses.



Recycling of PVC-containing products

EPA has identified secondary copper smelters as one of the largest known dioxin sources, releasing 230-310 g/year TEQ. [Schaum 1993] Dioxin is produced at these facilities when PVC-coated copper wire and cables are heated at very high temperatures for recycling. PVC is the primary source of chlorine feed to these units. According to one Germany study,

> *Considerable amounts of dioxins and furans have been found in the flue gas as well as in soil from the near vicinity of copper reclamation plants. In these facilities, scrap copper containing varying quantities of PVC-coated cables is precleaned by combustion or pyrolysis and then recycled in a copper smelter. In the ambient air near a copper smelter, we could find in emission measurements surprisingly high concentrations of dioxins and furans. [Christman 1989]*

Recycling of steel with PVC is another major dioxin source. The two major sources of chlorine inputs are vinyl residues – particularly from automobiles – and chlorinated cutting oils. According to the authors:

> *This pilot study clearly shows that PCDDs and PCDFs are formed during scrap metal melting processes. Combustion of PVC has been reported as one source of PCDDs and PCDFs in different combustion and pyrolytic processes. In this study, PVC is also found to give the highest emissions. [Tysklind 1989]*

A 1993 study by Finnish researchers also found very high emissions of dioxin from a metal reclamation plant and concluded that PVC was the most important dioxin precursor. According to the authors:

> *The measured emissions may indicate that a large part of the chlorinated organic compounds are formed due to the chlorine in PVC plastic (PVC plastic in cables and car components). [Aittola 1993]*

According to one Swedish study of such facilities, secondary steel smelters discharge dioxins in quantities ranging from 200 to 2200 ng per ton of steel (TEQ). An estimated 50.1 million tons per year of steel are recycled in U.S. secondary smelters. [U.S. DOC 1993] Dioxin emissions from this sector can thus be estimated at 10-110 grams per year (TEQ).

In its preliminary dioxin reassessment documents, EPA noted that "The secondary smelters which recover metal from waste products such as scrap automobiles have the potential for dioxin formation due to the plastic and associated chlorine in the feed material. Other countries such as Germany have identified this industry as potentially important." [Schaum 1993] EPA did not estimate dioxin emissions from any metal-PVC recyclers, however, except copper smelters.

PVC in Fires

Almost half of all PVC is used in building, construction, and furniture, including pipes, flooring, wallpaper, siding, wastebaskets, molded furniture, and other products. PVC probably constitutes the single most abundant synthetic material in the home.

Just as burning PVC in an incinerator or smelter results in dioxin formation, so does accidental burning of PVC in home and burning fires. Because combustion conditions in these fires cannot be optimized, dioxin

formation may be even greater than in incinerators. Numerous studies have associated the presence of PVC in fires with major dioxin emissions.

- German scientists have identified dioxin concentrations of up to 10,000 ng/m2 on surfaces from accidental fires with large quantities of PVC and concentration of up to 200 ng/m2 after "normal" fires in homes and offices. Soot samples from a fire at a German kindergarten – where substantial quantities of PVC construction material burned – have been found to contain dioxin concentrations as high as 45 ppb TEQ – equivalent to 15,000 ng/m2. [Fiedler 1993]
- On July 1, 1993 a fire destroyed a plastics plant near Montreal, consuming an estimated 15,000 kg of PVC in the factory. Tests by the Quebec Environment Ministry showed that ash from the fire was contaminated dioxins and furans at concentrations of 18 ppb (TEQ). Soil near the plant contained dioxins and furans at 0.55 ppb. Dioxin production in the fire was estimated at 40 to 85 grams, although the basis for this estimate is unknown. [Quebec Environment Ministry 1993] According to that figure, this single fire would be one of the largest known dioxin sources in Canada.
- In October of 1992 Microplast, a PVC recycling company in Lengrich, Germany, caught fire. The German Environmental Protection Agency (UBA) found that residues in the warehouse contained dioxin concentrations of 13.7 ppb TEQ. Cabbages growing 600 meters away showed an 88-fold increase in dioxin concentration above background levels. Cabbages growing 2.4 km downwind still exceeded the maximum acceptable limit for food of 1 ng/kg and were banned for sale. [UBA 1992]
- On November 27, 1992, a fire completely destroyed Euromat, a PVC factory in Diest, Belgium, which produced PVC granules for cars, cable, shoes, and the medical industry. Approximately 100 tons of PVC burned, and dioxins were found in fire residues in concentrations up to 87 ppt.

PVC is now ubiquitous in construction projects. Any fire in a modern building is an important dioxin source, and any modern building is a potential dioxin source. Because PVC fires have been linked to significant dioxin production, the German Environment Ministry has called for the use of substitutes for PVC in all areas susceptible to fires.

> *With fires in which PVC products are involved, the residues contain higher amounts of dioxin than is the case with fires without PVC materials. The gases from PVC fires also include hydrogen chloride. PVC materials should not be used where the clean-up measures which may be necessary lead to significant disruption of the public infrastructure....*
>
> *PVC products in the building industry should be substituted for in those areas of use in which considerable dangers to the environment and health occur, and extensive clean-up measures become necessary, as a result of the possible formation of dioxin and hydrogen chloride in fires.[German Env. Ministers 1992]*

The German EPA and Ministry of Health have made a similar order:

> *"The use of plastics containing chlorine and bromine should be completely excluded, as far as is possible. UBA and BGA propose a ban on the use of plastics containing chlorine and bromine in apparatus susceptible to fire, in the manufacture of chip-board, as well as the labelling of plastics containing chlorine and if necessary a ban on the use of PVC in packaging." [UBA 1992]*

An estimated 500,000 fires occur each year in Germany. [Fielder1993] Based on the production of 40-85 g of dioxin (TEQ) at a single fire in a PVC factory in Canada, and the findings of comparably high concentrations of dioxin in residues from fires in "normal" buildings, total U.S. dioxin emissions from accidental fires may range from 500 to 5000 grams per year.

5: SECONDARY PRIORITIES: OTHER DIOXIN SOURCES

A number of other sectors and product groups have been identified as important sources of dioxin during their manufacture, use, and/or disposal. These sectors include solvents, pesticides, other chlorine-based chemicals, water and wastewater treatment, and metallurgical processes.

1. CHLORINE PRODUCTION

Dioxin contamination begins at the very first stage of chlorine chemistry – the production of chlorine gas through the electrolysis of salt water. Carbon-containing compounds can be present as impurities, in plastic materials, or from the graphite electrodes in some electrolytic cells. [Schmittinger 1986] Once produced, chlorine immediately reacts with these organic compounds to form organochlorine by-products. Chlorine gas has been found to be contaminated with dioxin-like compounds in concentrations ranging from 40 to 210 ppb, including the PCBs decachlorobiphenol and nanochlorobiphenyl, octachlorostyrene, and tetrachlorobenzene. [Hutzinger 1988] Based on these estimates, the 13 million tons of chlorine produced in the U.S. each year would thus contain from 472,000 to 2,480,000 grams of these dioxin-like compounds each year (not TEQ).

Very high concentrations of dioxin (up to 650 ppb total) have been found in the sludges from spent graphite electrodes used in this process. [Rappe 1991] The dioxin-like compounds octachlorostyrene, HCB, and hexachloroethane are also found in these sludges. Most chlorine plants have replaced graphite electrodes in recent years with titanium substitutes.

For years it was assumed that the removal of graphite electrodes eliminated dioxin formation from chlorine production . But data recently released by chlorine manufacturers in Sweden indicate that even the most modern chlor-alkali plants produce substantial quantities of dioxin. It appears that trace quantities of organic materials – particularly from plastic pipes and valves – are reacting with chlorine to produce dioxins. According to studies by the University of Umeaa, dioxins have been detected in sludge and plastic piping from chlor-alklai plants in concentrations of 4.65 and 5.13 ppt (TEQ), respectively. [Umeaa 1994] These data are presented in table 5.1

Because production of chlorine itself results in dioxin formation, all "downstream" products that rely on chlorine are thus associated with dioxin formation.

2. PESTICIDES

For several decades, dioxin has been identified as a contaminant of some pesticides, such as 2,4,5-T, one of the major components of Agent Orange. New information suggests, however, that the problem is not limited to a few chemicals but actually extends throughout the entire range of chlorine-based synthetic pesticides.

The chlorine industry has argued that 96 percent of all synthetic organic pesticides contain chlorine or are manufactured using chlorinated intermediates. [CRA 1993] As a consequence, dioxins appear to occur as by-products in the manufacture of virtually all synthetic pesticides. Dioxin-like compounds are known or

Table 5.1
Dioxins in Waste from A Modern Chlor-Alkali Plant

Congener	Sludge (ng/L)	Polymer waste (ng/L)
2378-TCDD	.021	.0025
Total TCDD	.19	.02
2378-TCDF	.47	.69
Total TCDF	2.17	2.91
12378-PeCDD	.037	.016
Total PeCDD	.37	.06
12378-PeCDF	1.94	2.74
23478-PeCDF	.70	.79
Total PeCDF	7.48	7.66
123478-HxCDD	.16	.039
123678-HxCDD	.09	.008
123789-HxCDD	.11	.011
Total HxCDD	1.62	.13
123478-HxCDF	20.5	30.6
123678-HxCDF	3.79	7.99
123789-HxCDF	7.26	.77
234678-HxCDF	.84	.42
Total HxCDF	36.0	25.3
1234678-HpCDD	.72	.13
Total HpCDD	1.48	.22
1234678-HpCDF	58.2	38.5
1234789-HpCDF	19.4	7.09
Total HpCDF	101	57.9
OCDD	5.45	.69
OCDF	63.4	87.1
Total TEQ	4.65	5.13

Source: University of Umeaa 1993. Samples are sludge and plastic pipe from a Swedish chlor-alkali plant using titanium electrodesTEQ values using NATO method.

suspected contaminants of approximately 100 pesticides, ranging from chlorophenols and chlorophenoxy acetic acids to dichloropropene, lindane, atrazine, and simazine. (See tables 3.2 and 3.4.)

For instance, dioxins and furans have been detected at 160 ppt (TEQ) in 2,4-D, one of the most widely used herbicides in the U.S. [Schecter 1993] U.S. production of 2,4-D is estimated at 65 million pounds per year [EPA 1991]; dioxin contamination of this pesticide alone would account for the release 4.7 g of dioxin per year, plus additional amounts from production emissions and wastes. Dioxin concentrations in pesticides can

be very high: for instance, wastes from the production of lindane may contain dioxin concentrations as high as 50 ppm (TEQ) [Scholz 1987].

Dioxins are also associated with pesticides that do not contain chlorine but are made with chlorinated intermediates. For instance, dioxins are known or suspected contaminants in parathion and ammonium dinitrobutylphenol. [Esposito 1980, PCTN 985]

Dioxins are also produced when the chlorine-containing wastes from pesticide manufacture are incinerated. Because these products are usually produced in multi-step, complex processes, waste generation at pesticide plants is typically quite high; further, virtually all such wastes contain chlorine, since chlorine is involved in the production of almost all pesticides. Most pesticide production wastes are incinerated, leading to additional dioxin formation and release in the combustion process.

In some cases, dioxins can be produced when chlorine-containing pesticides are used. For instance, dioxins have been identified as by-products when pentachlorophenate wood preservatives are heated to the temperatures used in pressure-treatment of lumber. [Rappe 1978]

Chlorinated pesticides and the wastes from their production also produce dioxins when incinerated. EPA has estimated that 13,200 tons of incinerable pesticides are generated each year, plus presumably much larger quantities of chlorinated production wastes. [Oppelt 1987]

3. CHLORINATED SOLVENTS

Solvents account for close to 10 percent of all chlorine production, and they are a correspondingly significant source of dioxins. Dioxins occur in the manufacture, use, and disposal of chlorinated solvents.

Dioxin-like compounds are formed when chlorinated solvents are manufactured. Dioxins and furans have been identified in tetrachloroethylene, and 1,2-dichloroethane in concentrations up to 50 ppt (sum PCDD/Fs). [Heindl 1987] In addition, hexachlorobenzene has been identified in carbon tetrachloride, trichloroethylene, and tetrachloroethane. [Rossberg 1986] According to a German research team that found dioxin in a number of solvents and other aliphatic organochlorines, "These results suggest that the synthesis of short-chain chlorinated hydrocarbons can lead to PCDD/PCDF formation." [Heindl 1987]

Table 5.2
Solvents and other Aliphatic Organochlorines
Contaminated with Dioxin and Related Compounds

Compound	Contaminants detected	References
Carbon tetrachloride	HCB	Rossberg 1986
1,2-Dichloroethane	OCDF	Heindl 1987
Epichlorchydrin	HpCDD, HxCDD, OCDD, HpCDF,OCDF	Heindl 1987
Hexachlorobutadiene	OCDF	Heindl 1987
Tetrachloroethylene	OCDD, HCB	Heindl 1987, Rossberg 1986
Trichloroethylene	HCB	Rossberg 1986
Trichloroethylene *	TCDF, PeCDF, HxCDD,	Heindl 1987
	HxCDF, HpCDD, HpCDF, OCDD, OCDF	Heindl 1987

* Used in the presence of NaOH

U.S. production of chlorinated solvents totals approximately 3 million tons per year. If dioxin contamination averages only 1 ppt, these products would carry an estimated 2 grams per year of dioxin into the environment. However, because most solvents are quite volatile – and dioxin is not-- the presence of dioxin in these products suggests that significantly greater amounts may be present in the heavier wastes from chlorinated solvent production. Although large quantities of hexachlorobenzene have been identified in wastes from these processes, no data are available on dioxin levels in these residues.

The use of chlorinated solvents can also result in dioxin formation. Dioxins can be formed when chlorinated solvents are used for degreasing in the presence of alkalis – a common circumstance in many industrial applications. For instance, combining trichloroethylene with sodium hydroxide results in the formation of dioxins, furans, hexachlorobenzene, and octachlorostyrene. "It should be pointed out that trichloroethylene in alkaline medium is used occasionally in industrial processes: for the degreasing of metals, a combination of trichloroethylene, alkaline cleaners and emulsifiers is used at elevated temperatures. [Heindl 1987] The German Environmental Agency has confirmed this conclusion, finding that dioxins are produced in the following processes: "synthesis/extraction with chlorinated solvents, metal finishing, and dry cleaning." [Drechler 1992]

Dioxin concentrations of 140 ppt have been identified in the distillation sludge from the use of perchloroethylene in dry cleaning. [Lexen 1992] The source of these dioxins, however, has not been determined: they may be formed in the dry cleaning or distillation process, occur as contaminants in the "virgin" perchloroethylene, or are accumulated in clothes and then washed out in the cleaning process.

Chlorinated solvents are also a primary source of chlorine – and thus of dioxin emissions – in hazardous waste incinerators. Although some portion of spent chlorinated solvents are recycled, the vast majority are burned in incinerators or cement kilns. In 1983, about 1.1 million metric tons of chlorinated solvents were available for incineration, according to EPA estimates. [Oppelt 1987]

4. WATER AND WASTEWATER TREATMENT

The chlorination of sewage and drinking water results in the formation and release of dioxins in a number of ways. First, the process of chlorination itself appears to produce dioxin-like compounds. Swedish researchers have found that chlorinated dibenzofurans are formed when chlorine-free dibenzofurans naturally present in water are treated with chlorine. The authors wrote,

> *"The pattern identified in this report .... could be called the chlorine pattern. For the tetraPCDFs it consists of the same isomers as the pulp leaching pattern. However, the chlorine pattern also contains higher chlorinated PCDFs, but no PCDDs. [Rappe 1989]*

Other researchers have identified chlorinated phenoxyphenols, also called "pre-dioxins" as a by-product of the chlorination of water. [Ondeira 1989]

The incineration of chlorinated residues from wastewater and drinking water treatment plants results in further dioxin emissions. EPA has identified sewage sludge incinerators as a source of dioxin, with total emissions estimated at 1-26 grams (TEQ) per year. Further, carbon filters used to remove contaminants from drinking water accumulate chlorinated disinfection by-products; when these filters are "recharged" at high

temperature, dioxins are formed and released. German scientists have found that the regeneration of carbon filters, "heavily loaded with organic chlorine," results in dioxin formation; little or no dioxin is released when no chlorine is added. [Hutzinger 1988] EPA estimates dioxin releases from this sector at less than 1 g/year.

5. CHLORINATED AROMATIC CHEMICALS.

As discussed above, dioxin has been identified as a by-product in the production, use and disposal of the full spectrum of organochlorine compounds.

Dioxins are contaminants in a wide-range of chlorinated benzenes, chlorinated phenols, and other chlorinated chemicals with ring structures. According to NATO scientists:

> *All processes involving the manufacture of chlorophenol derivatives also are suspected of producing PCDD and PCDF. Most likely all technical chlorophenols and chlorophenol derivatives contain small amounts of PCDD and PCDF. These values show that all aromatic chlorinations are suspect of causing PCDD/ formation. Moreover, radical-side-chain chlorinations such as the chlorination of toluene are also relevant, because a small degree of nuclear chlorination can also occur. The following products are manufactured by chlorination or radical chlorination: chloro-nitrobenzenes, chloroaniline, 2,3,6-trichlorophenoxyacetic acid. [Hutzinger 1988]*

These compounds are used in a wide variety of processes in the chemical industry. Many are used in the production of pesticides, and some chlorophenols are commercial pesticides themselves. Chlorinated aromatics are used in the manufacture of specialty chemicals and additives for plastics, nylon, synthetic rubber, dyes, and pharmaceuticals. Extremely high concentrations of dioxins have been identified in several types of synthetic dyes – including phthalocyanine, dioxazine, and other dyes – as well as candles and other products made with these dyes. [Hutzinger 1988] The Swedish Environmental Protection Agency has identified dioxins in wastes from the pharmaceutical industry, in concentrations of approximately 2.5 ppt. [Lexen 1992]

6. OTHER CHLORINATED ORGANICS

Other chlorinated organic chemicals are associated with dioxin formation. The production and use of many of these compounds in the chemical industry have been identified as dioxin sources. In addition, whenever these products are used, wastes are produced containing a diverse cocktail of chlorinated wastes. The majority of these wastes are burned, leading to additional dioxin emissions.

For instance, epichlorohydrin – an intermediate used in the production of epoxy resins – is itself contaminated with dioxin. Dioxins and related compounds have also been found in concetnrations as high as 425 ppt (total) in hexachlorobutadiene, a short-chain aliphatic organochlorine used in the production of rubber and plastics. [Hutzinger 1988]

In addition, chlorinated intermediates are by definition used in highly reactive environments, and the formation of dioxin is a likely outcome. According to NATO scientists,

> *"All industrial chemical processes by which chlorine or hydrogen chloride is cleaved from intermediate products must be critically regarded with respect to PCDD/PCDF formation. Such chemicals and substance which contain no chlorine in the end product (in particular those resulting from eliminations, dehydrochlorination, and alkaline hydrolyses) should be especially considered." [Hutzinger 1988]*

Air emissions and wastewater effluents from oil refineries that use organochlorine compounds to regenerate spent catalysts. Concentrations of total dioxins and furans in in liquid effluent were found to be as high as 100 ppq, while concentrations in air emissions were over 200 ng/M3 – greater than that found in many incinerator stack gases. [Thompson 1990]

Chlorinated wastes from the production and use of these other organochlorines also results in dioxin formation and release. Production of any organochlorine results in the generation of waste liquids, solids, and sludges, and incineration is the dominant disposal method for these wastes. EPA has estimated that a 9.78 million metric tons of halogenated solvents, 0.72 million metric tons of halogenated sludges, and 1.25 million metric tons of halogenated liquids (excluding solvents) were available for incineration in 1983. [Oppelt 1987]

7. INORGANIC CHLORINE CHEMICALS: CHLORIDES AND BLEACHES

According to NATO,

> *"The processes to manufacture metal chlorides, chlorine, NaOCl (sodium hypochlorite) and hydrochloric acid are suspected of causing PCDD/F formation." [Hutzinger 1988]*

Of particular concern are inorganic chlorine compounds containing iron or copper, since these metals are catalysts that accelerate dioxin formation. Tests at the University of Bayreuth have found detectable quantities of dioxin in chlorides of iron, aluminum, and copper. [Hutzinger 1988] Swedish researchers have found that iron chloride used for water treatment contains significant quantities of dioxin and furans. [Rappe 1991]

Sodium hypochlorite (common household bleach) has been found to contain hexachlorobenzene and tetrachlorobenzene – markers of possible dioxin contamination – in the low parts per trillion range. [Hutzinger 1988] In addition, the use of chlorine-based bleaches and detergents in home clothes washing machines and dishwashers has been found to increase the concentration of dioxins in wastewater from these appliances. According to Swedish researchers, net production of dioxin is 8 pg TEQ per dishwashing cycle and 6 pg TEQ per clothes washing cycle. [Rappe 1992]

Table 5.3.
Dioxins and Related Compounds in Inorganic Chlorides

(ppb)

Compound	HpCDD	HpCDF	OCDD	OCDF	PCB-9	PCB-10	OCS	HCB	TCB
Iron chloride (FeCl3)	-	12	-	48	-	830	280	4000	-
Aluminum chloride (AlCl3)	-	-	0.1	34	200	-	77	1100	-
Copper chloride (CuCl)	-	0.08	0.2	0.08	-		.3	1	0.2
Copper chloride (CuCl2)	0.03	0.1	0.6	0.5	0.09	0.08	0.04	4	-
Titanium tetrachloride	-	-	-	-	-	0.02	-	-	-
Silicon tetrachloride	-	-	-	-	-	0.02	-	-	-
Sodium hypochlorite (NaOCl)	-	-	-	-	-	-	-	.001	.004

PCB-9 = nanochlorobiphenyl, PCB-10 = decachlorobiphenyl, OCS = octachlorostyrene; TCB = tetrachlorobenzene.
Source: Hutzinger 1988

8. METALLURGICAL PROCESSES

Elemental chlorine gas used in the production of certain metals combines with any organic material present to form organochlorine by-products, including dioxins. High concentrations of dioxins and related compounds have been identified n the emissions from several types of metal processing plants.

Dioxins may be formed when chlorine is used in the production of refined nickel and magnesium. Annual emissions from one magnesium plant in Norway have been estimated at 500 g/year (TEQ) to air and water, while emissions from a nickel plant have been estimated at 1 g/year (TEQ) to water alone. Emissions of hexachlorobenzene from the magnesium factory were estimated at over 350,000 grams per year. In addition high concentrations of PCDFs have accumulated in fish tissues near the nickel production facility. [Oehme 1989]

Recently, iron and steel plants have been found to be major sources of dioxin. German analyses show that such plants may emit dioxin in their stack gas in concentrations of 3 - 10 ng/M3 (TEQ). [Lahl 1993] The Swedish and Dutch governments have also identified iron sinter plants as very large sources of these compounds. [Rappe 1992] Dioxins are produced in this process because of the introduction of chlorinated chemicals such as cutting oils and solvents. According to one analysis:

> *"Sintering plants serve for recycling of dusts, scrap and abrasion from other processes of the metallurgical plant to recover the iron for further use in the blast furnace. But this reasonable waste management method is accompanied by the problem of introducing traces of chlorine and organic compounds responsible for the generation of PCDD/F within these plants. [Lahl 1993]*

According to one analysis, air emissions of dioxins from these plants acount for 300-1000 g/year (TEQ) in Germany alone. [Lahl 1993] U.S. production of steel is approximately twice as great as that in Germany; dioxin emissions from this sector in the U.S. may thus range from 600 to 2000 grams per year, making this one of the largest of all known dioxin sources. [U.S. DOC 1993]

9. Environmental transformation of other organochlorines

Several studies have found that chlorophenols may be transformed under environmental conditions into dioxins and related compounds. These transformations may occur due to enzymatic or photolytic reactions. [Oberg 1990, Oberg 1992, Plimmer 1973]

Several studies have found that the concentration of dioxins in sewage sludge and compost actually increases over time with no apparent input of dioxins. It appears that chlorophenols – and possibly other organochlorines – are being transformed into dioxin during the composting process. [Oberg 1992, Shafer 1992] Typically, the total TEQ in such a mixture may increase by 2- to 3-fold during composting. [Oberg 1992]

If chlorophenols can be transformed into dioxin, it is plausible that some other organochlorines – particularly other aromatics such as chlorobenzenes, PCBs, phenoxy herbicides, and others – could be changed into dioxins as well. The production of dioxin due to the environmental transformation of organochlorines released into the environment from industrial and agricultural sources may be a significant unquantified dioxin source. No data are available from which to estimate dioxin production due to this phenomenon.

6: RECOMMENDATIONS: THE ZERO DIOXIN PROGRAM

NEED FOR EMERGENCY ACTION

EPA's reassessment indicates that dioxin contamination poses a serious threat to public health. Because dioxin is so persistent in the environment and in the human population, contamination that has already occurred will not disappear quickly. One analysis found that if all exposures to PCBs could be stopped immediately, it would take six generations before body burdens would return to non-detectable levels. [Swain 1988]

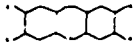
The first principle of environmental and public health practice is prevention. It is not ethical or necessary to delay action until it has been conclusively proven that dioxin contamination has caused large-scale damage to health and the environment. By then, it will be too late. The evidence indicates that current dioxin contamination poses current or future threats to the general population, and it is possible that the damage may have begun already.

This information provides more than adequate scientific support for a shift in regulatory policy, based on the principles of public health protection. Environmental policy on dioxin must shift to preventing further releases and exposures. EPA should initiate a comprehensive dioxin elimination program, including immediate action to eliminate the largest dioxin sources and medium-term measures to address additional dioxin-producing sectors. Based on the conclusion that the environment and the human population are already excessively loaded with these compounds, EPA must act now to bring all additional emissions of dioxins to zero.

Policy changes on dioxin can be modeled, in part, on experience with another pollutant – lead. In the 1970s and 1980s, health scientists found that lead -- a persistent metal emitted by a multiplicity of sources, including vehicles, chemical plants, and paints – – had accumulated in the environment and in the bodies of the human population to levels that threatened the health of a large fraction of the nation's children. Subsequently, EPA policy – once a source-by-source approach that sought to control and permit lead releases -- was transformed into an overall strategy to prevent lead emissions from every identifiable source, with the goal of reducing lead exposure and body burden for every American. Although EPA has not strictly followed this policy, the result has been significant achievements, including the phase-out of leaded gasoline and paints – the largest single sources of lead emissions– and consequent reductions in environmental contamination and blood lead levels in young children.

With the lead model in mind, EPA should immediately establish a national program that applies a prevention-based approach to all known sources of dioxin, while identifying and targeting additional, yet-to-be-identified sources. The goal of the program should be to eliminate all discharges of dioxin and achieve substantial reductions in environmental concentrations, human exposures, and human body burdens of dioxin and related compounds.

The large number of dioxin-producing products and processes makes the implementation of a dioxin elimination program more difficult than a lead prevention policy. Installing dioxin-free alternatives in the full range of industrial sectors will require substantial technical and economic conversion. On the other hand, the health hazard posed by dioxin is compelling, and measures to protect public health and the environment should



not be delayed. In the largest dioxin-producing sectors for which the alternatives are available and feasible, action should be taken now; those sectors that require longer implementation phases should be placed on timelines for dioxin elimination. Thus, a dioxin elimination program should be divided into tiers for immediate and medium-term action.

PRINCIPLES OF THE ZERO DIOXIN PROGRAM

1. ZERO MEANS ZERO. Sources of dioxin must be eliminated, not simply reduced. The ecosystem may have the capacity to "assimilate" limited quantities of chemicals for which there are substantial natural biogeochemical cycles (such as carbon or nitrogen compounds), but it has no ability to assimilate persistent synthetic toxic substances - such as dioxins. Even dilute releases of these chemicals build up in the environment over time, eventually reaching levels that threaten public health and the environment. Thus, the ecosystem's assimilable capacity for dioxin and related compounds is zero. The current crisis, in which dioxin levels have reached the range that threatens public health, demonstrates the failure of the "acceptable discharge" approach to dioxin.

Given the current health threat, it would be wholly inappropriate for EPA to continue to permit the release of dioxin into the environment – even in reduced amounts. To protect public health, inputs of dioxin and related compounds into the environment must be eliminated.

2. POLLUTION PREVENTION, NOT CONTROL. Once dioxin is produced, it is too late to prevent its discharge into the environment; pollution control devices, filters, treatment systems, and disposal methods such as burning and burying simply shift captured chemicals from one environmental medium to another or delay their release until a later date. Pollution control is an inappropriate strategy for a class of persistent chemicals that resist treatment and disposal methods, for which the ecosystem has no assimilable capacity, and which have already accumulated to excessive levels in the environment.

The goal of eliminating dioxin discharges can only be achieved if the Zero Dioxin Program focuses on prevention. The industrial processes and feedstocks that result in dioxin formation must be changed so that no dioxin is formed. Products that involve the formation of dioxin at some point during their lifecycle – manufacture, use, or disposal – must be altered if possible or otherwise banned. Processes that result in dioxin formation, such as incineration or the pulp bleaching, must eliminate the feedstocks that lead to dioxin production or be shut down altogether.

3. ADDRESS ALL DIOXIN SOURCES. The program must address all known and suspected sources of dioxin in order to bring future releases of dioxin to zero. In its reassessment documents, EPA has focused on only a few of the many sources of dioxin; these account for only 10 to 50 percent of all dioxin releases. The Zero Dioxin Program must take a comprehensive approach to all dioxin sources, including those that are suspected.

4. SET PRIORITIES FOR DIOXIN ELIMINATION. Some products and processes are known to account for large quantities of dioxin during their lifetime. The Zero Discharge program should be implemented so that the largest sources – and those for which alternatives are most readily available – are phased-out on the shortest timelines.

STEP 1: ESTABLISH A NATIONAL ZERO DIOXIN PROGRAM

1. National goal. EPA should establish a national Zero Dioxin program with the stated goal that all releases of dioxin be brought to zero within 10 years through the elimination of those products, feedstocks, and processes that lead to dioxin formation.

2. Moratorium on new dioxin permits. EPA should grant no new permits to release dioxin into any environmental medium.

3. Sunset existing dioxin permits. EPA should modify all existing dioxin release permits to include timetables for the reduction and eventual elimination of all dioxin releases.

4. Comprehensive approach to all sources. For the many dioxin sources that are not currently permitted or regulated, EPA should begin a process to identify and prioritize all dioxin sources and require their elimination within 10 years. All facilities that manufacture, use, or dispose of chlorine or chlorinated organic chemicals should be required to test for dioxin formation in their products, processes and wastes. Any identifiable release of dioxin and related compounds should reported under federal Right-to-Know programs.

STEP 2: IMMEDIATE PRIORITIES: MAJOR DIOXIN SOURCES

In the largest dioxin-producing sectors, EPA should take immediate action to reduce and eliminate dioxin releases.

1. Incineration. Incinerators that burn chlorinated wastes are the largest known producers of dioxin. EPA should take the following actions:

- Place an immediate moratorium on permits for new combustion facilities that burn chlorinated wastes and on permits for expanded capacity at existing units.
- Modify immediately all combustion device permits to include sunset provisions to reduce and finally eliminate the input of chlorinated wastes and products. This policy should apply to all combustion devices, including but not limited to incinerators, boilers, kilns and furnaces that burn hazardous wastes; incinerators for trash, hospital waste, sewage sludge, and tires; and metal smelters and sintering plants.
- In turn, focus waste reduction programs on eliminating the generation of chlorinated incinerable wastes, particularly solvents, PVC, and wastes from the production and use of chlorinated substances in the chemical industry.
- Immediately ban the addition of chlorinated compounds to fuels, including gasoline, diesel, and jet fuel.
- Minimize the burning of chloride-containing wastes (i.e., food and yard wastes) in trash incinerators through garbage separation programs.

2. Pulp and Paper. The use of chlorine, chlorine dioxide, and other chlorine-based bleaches in the paper industry is the second largest dioxin-producing sector and the largest source of dioxin discharges directly to waterways. Oxygen-based and other alternative bleaching methods are available and in use in 55 pulp mills throughout the world to produce a range of high-quality, bright paper suitable for the most demanding uses.[Albert 1994] EPA should establish rapid timelines for the phase-out of all chlorine-based bleaches in the pulp and paper industry.

3. PVC. In manufacture, use, accidental combustion, recycling and disposal, PVC results in more dioxin formation than any other single product. Wood, metals, ceramics, glass, wood, paper, and chlorine-free plastics provide the necessary technical properties to replace PVC in virtually all uses; over 80 communities, along with numerous hospitals and manufacturers of automobiles, consumer products, flooring, furniture, electronics equipment, and office products in several European nations have virtually eliminated PVC in a full range of applications. EPA should take the following actions

- Establish a PVC phase-out program, with progressive reductions toward zero in the production and use of PVC.
- Immediately ban short-life PVC uses, such as packaging, toys, and non-essential medical supplies.
- Initiate a phase-out of all uses of PVC in areas susceptible to fire and in products subject to combustion-based recycling (i.e., construction, furniture, automobiles, and cable sheathing).
- Initiate a phase-out of PVC in all other uses. Exceptions can be made if there are minor PVC uses that serve compelling social needs for which alternatives are not yet available (i.e., certain medical uses).
- Modify all discharge permits for chemical plants that produce PVC feedstocks to include sunset provisions on dioxin discharges to all environmental media, including deposition of dioxin into wastes and residues for disposal or treatment.

4. Chlorinated aromatic chemicals. This class of chemicals, with their close structural relationship to dioxin and related chemicals, result in significant quantities of dioxin formation in manufacture, use and disposal. EPA should immediately ban all open uses of chlorinated aromatic compounds (i.e., pesticides) and establish a phase-out program for all other uses.

STEP 3: SECONDARY ACTIONS: BEGIN CHLORINE PHASE-OUT

As discussed in this report, all uses of chlorine and chlorinated organic chemicals are susepcted of dioxin formation at one or more points in their lifecycle – from the production of chlorine itself to the disposal and recycling of chlorinated products and wastes. Thus, a dioxin phase-out program is tantamount to a phase-out of chlorine chemistry, and all uses of chlorine must be critically regarded for dioxin formation.

Phasing-out all uses of chlorine and chlorinated organic chemicals, of course, involves significant economic and technological change that will require phased implementation. While immediate action takes place in the priority sectors discussed above, the following dioxin-producing sectors should be placed on timetables for phase-out, with schedules based on the magnitude of releases, the availability of alternatives, and the ease of their implementation.

1. Chlorinated solvents. During production, incineration, and some uses, chlorinated solvents result in dioxin formation. Viable water-based, mechanical, and chlorine-free chemical alternatives are available for cleaning, degreasing, and coating in a full range of industries. EPA should establish a rapid timetable for the phase-out of the production and use of all chlorinated solvents.

2. Chlorine-related pesticides. Dioxins are formed in the production, some uses, and disposal of chlorine-containing pesticides and in the production of non-chlorinated pesticides made with chlorinated intermediates. The National Academy of Sciences has found that farmers can adopt organic farming methods, reduce or eliminate the use of synthetic pesticides, and enhance their profits and crop yields. [NAS 1989] EPA should establish a phase-out timetable for all chlorine-related pesticides.

3. Other chlorinated organic chemicals. Dioxins are formed in the production, some uses, and disposal of all other chlorinated organic chemicals, such as intermediates, catalysts, and specialty chemicals. Alternatives are available now for most such uses (i.e., chlorine-free alternatives to chlorohydrin and phosgene intermediates have been developed by major chemical companies) [Robert 1994] and can be developed for others. EPA should establish timelines for phasing-out the production and use of all other chlorinated organic chemicals. Uses that serve a compelling social need (i.e., pharmaceuticals) for which no alternatives are available should be exempted from the program.

4. Metallurgy and inorganic chemicals involving chlorine. Dioxins are formed when chlorine and chlorinated compounds are used in metallurgical processes and in the production of some inorganic chlorinated chemicals (certain metal chlorides, hypochlorites, etc.). EPA should establish phase-out timetables for the use of chlorine in high-temperature metallurgy and for all inorganic chlorinated chemicals in which dioxin formation has been identified.

5. Wastewater treatment. Dioxins and related compounds are formed in the chlorination of drinking water and sewage and in the incineration of sludge from water treatment plants. Effective alternatives – including ultraviolet light, ozone, hydrogen peroxide, slow sand filtration, and membrane filtration – are in use in water systems across the world. EPA should establish timelines for the implementation of chlorine-free water treatment methods while insuring that adequate disinfection continues. In most cases, chlorine-free methods for water disinfection can be implemented more quickly than the use of chlorine as a residual can be eliminated.

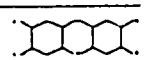
6. Remediation. A substantial quantity of dioxin and PCB-contaminated materials are now present in landfills, sediments, and stored industrial wastes. EPA should intensify the development and application of non-combustion methods for the degradation of these materials.

Political and economic implications

Faced with severe health hazards of environmental pollution, society has repeatedly taken large-scale action to prevent further harm. In addition to the lead example, the U.S. government has banned or restricted numerous individual toxic substances as well as the class of polychlorinated biphenyls. More recently, most of the world's governments have agreed to phase-out the production and use of the class of chlorofluorocarbons and other ozone-depleting substances.

An even more ambitious approach is necessary and justified to address the severe hazards posed by the accumulation of dioxin in the environment. Calls for a phase-out chlorine chemistry to eliminate the production of persistent toxic pollution, however, have ample precedent:

- The International Joint Commission on the Great Lakes has recognized the fact that dioxin formation occurs throughout the field of chlorine chemistry and that the mix of by-products formed in the lifecycle of chlorine and chlorinated organic chemicals cannot be prevented or controlled. Thus the IJC has called on the U.S. and Canada to begin a phase-out of all uses of chlorine and chlorinated organic chemicals as industrial feedstocks. [IJC 1992, IJC 1994]
- The American Public Health Association has made a similar recommendation. In 1993, the APHA found that "the only feasible and prudent approach to eliminating the release and discharge of chlorinated organic chemicals and consequent exposure is to avoid the use of chlorine and its compounds in manufacturing processes." On this basis, the organization resolved that chlorine and chlorinated organic



compounds be treated as a class for phase-out, with exceptions to be made only for uses that can be demonstrated to pose no significant hazard or for which no alternatives are available. [APHA 1994]

- The 15-nation Paris Convention on the Northeast Atlantic and the 21-nation Barcelona Convention on the Mediterranean Sea have agreed that the discharge of persistent, bioaccumulative toxic substances – particularly organohalogens such as dioxins – must be brought to zero on rapid timelines.
- The White House itself has also begun to move towards the comprehensive regulation of the field of chlorine chemistry. In its proposal for the Clean Water Act, President Clinton proposed to develop "a national strategy to reduce, substitute, or prohibit the use of chlorine and chlorinated compounds." Specifically, this recommendation consisted of an 18-month study and a 12-month period of policy formulation and review, with the focus on major chlorine uses, including pulp bleaching, PVC plastic, solvents, and water treatment.

Without question, phasing-out dioxin sources will require substantial technological and economic transformation, as numerous products and processes are converted to chlorine-free alternatives. Although this conversion will require substantial investment in some sectors, most of the alternative products and processes provide economic benefits in terms of increased employment, improved efficiency, decreased expenses for chemical procurement, waste disposal, liability and remediation, and the elimination of the social costs associated with damage to health and the environment. The medium- and long-term economic benefits to industry and society have been clearly demonstrated for chlorine-free pulp bleaching, elimination of chlorinated solvent use in manufacturing and dry cleaning industries, pesticide-free agriculture, and numerous chlorine-free methods of chemical manufacturing. [Thornton 1994]

The transition is significant enough that there will be substantial costs and benefits and the possibility of economic dislocation in sectors that manufacture chlorine and chlorine-based chemicals. If it is guided by careful planning, the economic benefits of this conversion process can be maximized, the costs minimized, and both distributed equitably. Thus, the long-term aspects of the Zero Dioxin Program should be subject to a transition planning process, with the participation of all stakeholders – including labor, communities, environmentalists, chemical users and chemical producers. The goal of this process should be to insure that the safest and most beneficial alternatives are used, to minimize economic dislocation by directing investment in dioxin-free alternatives to those communities where dioxin-producing processes have been phased out, and to protect workers and communities who do experience economic dislocation by providing income protection, health coverage, and the opportunity for substantial advanced education, retraining, and/or placement. The International Joint Commission, labor unions [OCAW 1993], the APHA, and environmental organizations have all called for such transition planning and/or worker protection as essential components of any chlorine phase-out program.

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