

# Dioxin From Cradle to Grave

**By Joe Thornton**  
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### **Thanks to the Following Contributors:**

Editor: Joel Tickner

Editorial Assistance: Thomas Belazzi, Pat Costner, Charlie Cray, Exeter Lab, Kathy O'Keefe, Beverley Thorpe, Wytze van der Naald, Bill Walsh

Production Assistance: Jay Townsend

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## Summary of Findings

The purpose of this report is to show that the lifecycle of polyvinyl chloride plastic, also known as PVC or vinyl, is a major source of dioxin pollution. When PVC is manufactured, disposed of by incineration, burned accidentally, or when products containing PVC (usually as a coating or covering on a metal product) are recycled in combustion-based processes, large quantities of dioxin are formed. In fact, PVC is a major chlorine donor -- and thus a significant cause of dioxin generation -- in a large percentage of identified dioxin sources. There are strong grounds for holding PVC responsible for a substantial and growing proportion of global dioxin production and release. Fortunately, cleaner substitutes exist for almost all uses of PVC.

### **Dioxin is a Global Health Risk**

Dioxin and dioxin-like compounds are a family of toxic substances produced accidentally in a host of industrial processes involving chlorine or chlorine-based chemicals. These compounds are extremely long-lived in the environment, and, because they are fat soluble, they concentrate in the tissues of humans and other species high on the food chain. Since the advent of the modern chemical industry, dioxins have gradually accumulated on a truly global basis -- from the local farm to the deep oceans, from the Gulf of Mexico to the North Pole. Every person is now exposed to significant amounts of dioxins, primarily through the food supply. Even larger quantities of dioxin are passed from one generation to the next via mothers' milk and across the placenta.

Evidence gathered in the last decade, much of it presented in the U.S. Environmental Protection Agency's (EPA) "Dioxin Reassessment," indicates that global dioxin contamination poses a long-term threat to the health of the general human population. Of particular concern is the ability of tiny doses of dioxin to interfere with the body's hormones which could lead to a wide variety of effects on reproduction, child development, and the immune system, as well as cancer. Wildlife populations -- fish, birds, and marine mammals -- have already been severely affected by dioxin pollution. In the human population, dioxin exposure may already have played a role in the incidence of cancer, endometriosis, cognitive deficits in developing children, and other conditions and diseases.

We do not know the extent to which dioxin exposure has already affected human health on a global scale, but we know with certainty that universal exposure to these chemicals represents a risk to public health. Any increase in exposure will increase that risk. Because dioxin is so persistent in the environment, continued discharges will add to the global dioxin burden, increasing the threat to public health. We must bring dioxin creation and releases to zero as quickly as possible, with priority given to the largest dioxin sources.

## Dioxin Sources

There are two ways to characterize dioxin sources: we can focus on the facilities that release dioxin into the environment, or we can identify the materials that cause industrial and natural processes to produce dioxin in the first place. EPA and others have used the first method to identify the major dioxin sources in the U.S. and other nations. All of these inventories note that incinerators and other combustion sources are the largest sources of dioxin: garbage incinerators, medical waste incinerators, incinerators and cement kilns that burn industrial hazardous waste, metals smelters, and other combustion processes account for over 90 percent of dioxins and furans entering the environment from known sources. Pulp and paper mills that use chlorine-based bleaches and manufacturers of chlorinated organic chemicals are the two most important dioxin sources not related to combustion.

With this approach, EPA has offered a preliminary characterization of dioxin discharge points, but the resulting list of sources is long, diverse, and confusing. The chemical industry has used this list of dioxin sources to argue that a dioxin curtailment strategy would require a byzantine set of regulations addressing virtually every industrial activity and combustion process. Many dioxin sources, however, can be tied together by the chlorine-containing feedstocks they share. The superior approach, thus, emphasizes the materials that cause dioxin formation in diverse processes. It turns out that four products - - PVC plastic, chlorinated solvents, chlorinated pesticides, and chlorine bleached paper -- are the major causes of dioxin releases from almost all of the sources identified by the various inventories.

EPA's current method of identifying dioxin sources does not point to clear strategies to prevent dioxin pollution. Focusing on discharge points leads to an emphasis on better management and control technologies for each dioxin discharger. These technologies are typically very expensive, cannot eliminate all dioxins, and thus perpetuate dioxin contamination of air, water, or land. In contrast, the materials-based approach focuses on those substances that, when introduced into industrial or other processes, cause dioxin formation. By avoiding the production of these materials, we can prevent virtually all dioxin generation.

In general, the cause of dioxin formation is the material that supplies chlorine for incorporation into dioxin. Dioxin formation requires only three things: a source of chlorine, a source of organic matter, and a reactive environment in which these materials can combine. The latter two are ubiquitous; synthetic chlorine production is the uniquely preventable factor in dioxin generation. In virtually all dioxin sources, the chlorine donor is a material -- a plastic, solvent, bleach, or pesticide, for instance -- that has been produced by the chemical industry. \*

Most studies in laboratory, pilot scale, and full-scale incinerators confirm this view: reducing the input of PVC and other chlorinated organic chemicals to incinerators reduces dioxin formation. While a few studies have found no such relationship, the

weight of the evidence from these studies confirms that reducing chlorine input in incineration will decrease dioxin output.

## **PVC and Dioxin**

Dioxins are produced throughout the lifecycle of PVC plastic. PVC manufacture begins when chlorine gas is produced by the energy-intensive electrolysis of brine, a process in which dioxin is formed. Next, chlorine is combined with ethylene to produce ethylene dichloride (EDC). In this process, large quantities of dioxin are formed. Some portion of these dioxins are released in air emissions and wastewater discharges. Samples taken downstream from EDC manufacturers in the U.S. and Europe indicate significant contamination of sediment and the foodchain in the vicinity of these plants. The largest quantities of dioxin are directed into the wastes or tars that result from EDC synthesis, according to European studies. Greenpeace analyses at U.S. chemical facilities indicates that the wastes from this process are among the most dioxin-contaminated wastes known. These chlorine-rich wastes are typically incinerated, producing and releasing dioxins into the environment.

EDC is then converted into vinyl chloride monomer (VCM), which is polymerized, formulated, and formed into a final PVC-containing product. After PVC products are sold, they produce dioxins if they encounter reactive conditions, as they do when a building or vehicle burns in an accidental fire. After their useful lifetime, PVC products that are incinerated or serve as coatings or coverings on metals recycled in smelters create additional dioxins. Incinerators and smelters in which PVC is burned are among the most important dioxin sources yet identified. More than 1 billion pounds of PVC may be burned in U.S. trash and medical waste incinerators and in accidental structural fires each year. The unknown quantities of PVC burned in industrial and warehouse fires, automobile fires, metals smelters, and wood combustion add to this total.

The lifecycle of PVC plastic may thus be the cause of more dioxin formation than that of any other single material. Sources in which PVC is a major chlorine donor account for a significant proportion of the identified dioxin emissions in the EPA's inventory. PVC is a significant, and sometimes predominant, chlorine donor in most of the major dioxin sources, including municipal waste incinerators, medical waste incinerators, smelters for copper and accidental fires and open waste burning. Wastes from PVC production are important chlorine donors in hazardous waste incinerators, and PVC is an important chlorine donor in wood combustion.

Nearly 40% of all the chlorine produced by the chemical industry is used in PVC, so it is not surprising that PVC is a significant chlorine source in so many dioxin producing processes. PVC is also an unusually fast-growing dioxin source. While production of many other chlorinated compounds is declining and others hold steady, U.S. and worldwide production of PVC and its feedstocks is rapidly growing, both for use in the U.S. and for export, primarily to developing nations. Finally, a certain percentage of longer lived PVC products (e.g., construction materials) will be reaching the end of their

useful lives in the coming years. If burned, these PVC stocks may lead to a new influx of dioxin pollution.

## **Recommendations**

The health risk posed by dioxin calls for immediate action. As a major cause of dioxin pollution and numerous other environmental impacts, PVC must be a priority in any dioxin prevention program. Greenpeace recommends the following elements of a materials-based dioxin prevention strategy for PVC:

### **1. Prevent PVC-Related Dioxin Pollution**

EPA should announce a PVC sunset program, the intent of which is to progressively reduce the production and use of PVC in the U.S. to zero. Priority should be given to those use sectors that cause the most dioxin formation during their lifecycle (e.g., those most likely to be incinerated or involved in fires) or are most easily replaced with safer, chlorine-free substitutes. Specific action should include:

- A moratorium on permits for new production facilities/expansions for EDC, VCM, and PVC and modification of permits at existing plants to require that dioxin releases to all media, including wastes destined for disposal, be brought to zero within 5 years.
- A moratorium on permits for new incinerators and other waste combustion facilities, and modification of existing permits to require that dioxin emissions to all media to be brought to zero within 5 years by eliminating the input of chlorinated wastes and product.
- A phase-out of medical waste and municipal solid waste incineration.
- Rapid phase-outs of:
  - all short-life PVC uses (packaging, toys, IV-bags, etc.);
  - PVC products in areas susceptible to fire (construction materials, PVC coated cables, appliances, and vehicles); and
  - recycling of metals containing PVC residues in combustion-based processes (i.e., electrical cables, automobiles).

### **2. Acknowledge and Investigate the Role of PVC in Dioxin Formation**

First, EPA must acknowledge the important role of PVC in dioxin formation. Since at least 1990, EPA has had information that the incineration and manufacture of PVC produce dioxins. Since 1993, extensive information on these subjects has been submitted to the agency. Nevertheless, EPA has failed to acknowledge or substantively investigate the critical role of PVC as a chlorine donor in major dioxin sources.

EPA has made no attempt to collect or analyze samples of waste, wastewater, or air emissions from the nation's 14 EDC/VCM facilities or the incinerators that burn wastes

from these plants. Instead, the EPA is allowing the industry trade organization, the Vinyl Institute, to voluntarily "self-characterize" the industry's dioxin emissions. The industry will collect samples from its own plants, analyze their dioxin content, interpret the data, and submit it to EPA. Although there is a "peer review" committee to examine the methodology and results of the vinyl industry's self-characterization, the industry will ultimately choose where, when, and how samples will be taken and analyzed, and which data are suitable for submission. By the time the agency's reassessment is finalized, the industry is expected to have only submitted data to EPA on dioxin contamination of wastewater and resins -- the two media with the lowest and most difficult to detect concentrations of dioxins --but no data on dioxins in wastes, tars, sludges; or incinerator emissions, ashes, or sludges, which, directly or indirectly, are responsible for the vast majority of dioxin emissions.

### **3. Focus Dioxin Policy on Materials**

EPA should reorient its dioxin research and policy towards pollution prevention. This requires a fundamental shift away from the agency's current reliance on pollution control techniques for managing dioxin sources. The agency should establish a goal of zero dioxin discharges to all environmental media and focus on reducing the production and use of materials that cause dioxin generation during their lifecycle. Research on dioxin sources should also be reoriented to emphasize the identification of dioxin-producing materials.

### **4. Address Environmental Justice Concerns Associated with the PVC Lifecycle**

Pollution associated with the lifecycle of PVC has a disproportionate effect on low-income and minority communities. Incinerators where municipal, medical, and hazardous wastes rich in PVC and its by-products are burned tend to be located more predominantly in African-American, Latino, and Native American communities. A similar pattern holds true for the manufacture of PVC, as well. There are 14 U.S. facilities, mostly located in the Gulf Coast of Louisiana and Texas, that produce ethylene dichloride and/or vinyl chloride monomer feedstocks for PVC. The mean percentage of "non-white" residents in these communities is 57 percent higher than the national average, a preliminary analysis of census data indicates.

Pollution caused by PVC production and disposal is thus an environmental justice issue. EPA should apply its environmental justice policy to investigate and initiate action to prevent dioxin formation during the lifecycle of PVC plastic. President Clinton's Executive Order 12898 on Environmental Justice requires that the agency improve "research relating to the health and environment of minority populations" and reduce pollution in these communities.

### **5. Ensure a Just Transition**

Any plan to protect health and the environment from dioxin sources -- including a PVC sunset program -- must prevent or compensate for the economic and social dislocation

that results. A tax on the production of EDC/VCM would help to drive the transition away from PVC and finance the costs associated with it. The revenues from such a tax could be used for transitional measures to ensure that a PVC phase-out is just, equitable, and orderly. In particular, a transition fund could be used to assist workers and communities affected by the transition, provide for educational opportunities, income protection, and health insurance for those affected by a PVC phase-out, and finance research and development into safer PVC alternatives.

## Notes

\* Although very small quantities of dioxin can be produced when natural chloride salts are burned in some high-temperature processes, the vast majority of dioxin is associated with burning or processing the products and wastes of industrial chlorine chemistry. Thus, eliminating the feed of PVC and other organically-bound chlorine compounds to incinerators will substantially reduce dioxin formation.

## Introduction

The purpose of this report is to show that the lifecycle of polyvinyl chloride plastic, also known as PVC or vinyl, is a major source of dioxin pollution. When PVC is manufactured, disposed of by incineration, burned accidentally, or when products containing PVC (usually as a coating or covering on a metal product) are recycled in combustion-based processes, large quantities of dioxin are formed. In fact, PVC is a major chlorine donor -- and thus a significant cause of dioxin generation -- in a large percentage of identified dioxin sources. These are strong grounds for holding PVC responsible for a substantial and growing proportion of global dioxin production and release. Fortunately, cleaner substitutes exist for almost all uses of PVC.

**TABLE 1: The Many Uses of PVC**

Food Wrap	Coatings	Automotive seats	Credit Cards
Flooring	Upholstery	Medical instruments	Bottles
Siding	Garden Hose	Doors	Apparel
Pipe	Shower Curtains	Window Frames	Toys
Wall covering	Wire/Cable insulation	Molding	Appliance housing

Source: Society of the Plastics Industry (SPI), 1995

This report represents the culmination of several years of research on the part of Greenpeace scientists and policy analysts. Greenpeace will continue to conduct literature reviews and primary research to demonstrate and better characterize the links between PVC and other chlorinated compounds and dioxin pollution. It is our hope that this research will convince government agencies and non-governmental organizations throughout the world of the need to address the materials that lead to dioxin pollution, the only method that will lead to true pollution prevention for dioxin. The report is divided into the following sections: (1) the health threats posed by dioxin; (2) strategies for dioxin prevention; (3) the PVC-dioxin pollution links, with analysis of the role of PVC in dioxin formation from different sources; (4) strategies for a PVC phase-out; and (5) recommendations.

# Chapter 1 – Dioxin Poses a Global Health Threat

## History: From Rare Contaminant to Global Health Threat

In the 1950s, dioxin\* was first discovered as the cause of severe health problems among workers who had been exposed to the by-products of explosions in chemical plants that manufactured certain chlorine-based pesticides. In these accidents, dioxin was formed and released into the workplace environment, causing systemic health problems among workers.

In the 1960s and 1970s, dioxin was identified as a contaminant in the pesticides themselves -- the components of Agent Orange -- and health problems began to emerge among soldiers and civilians exposed to Agent Orange in the Vietnam War. Subsequently, toxicological and epidemiological studies showed that dioxin was an extraordinarily potent carcinogen and caused damage to a variety of organs and systems in laboratory animals.

In the 1980s, the scope of the problem suddenly exploded. Dioxin, it was discovered, is formed not just in the manufacture of a few pesticides, but in a wide range of industrial processes involving chlorine or chlorinated materials. Trash incinerators and pulp and paper mills that used chlorine as a bleaching agent were found to release particularly large quantities of dioxin. The scope of environmental contamination by dioxin also turned out to be much greater than previously thought: dioxin was discovered in air, water, and wildlife on a truly global basis -- from the Great Lakes to the deep oceans to the North Pole. Significant dioxin concentrations were found in the bodies of the general human population and "market-basket" studies of the human food supply. By the end of the 1980s, it was clear that every person in the world is now exposed to dioxin.

Only in the 1990s, however, has the health risk posed by universal dioxin exposures become clear. In 1994, the U.S. Environmental Protection Agency (EPA) released its long-awaited "Dioxin Reassessment" [EPA, 1994a, 1994b], a project originally begun when the chemical and paper industries pressured the agency to revise downward its estimate of dioxin's toxicity and thus weaken regulations on dioxin sources. Contrary to the industry's intent, however, EPA's reassessment concluded that dioxin may pose a long-term, large-scale hazard to the health of the general population. New toxicological and epidemiological studies have indicated that in addition to its potential to cause cancer in humans and in animals, extraordinarily small quantities of dioxin can disrupt the body's hormone system, leading to severe effects on reproduction, off-spring development, and the function of the nervous and immune systems.

## Global Dioxin Pollution

Two aspects of the environmental behavior of dioxin-like compounds make them particularly troublesome. First, they are extraordinarily persistent, resisting physical, chemical, and biological degradation for decades and longer [Paustenbach et. al., 1992, Webster and Commoner, 1994]. As a result, even dilute discharges accumulate in the



environment over time, reaching particularly high levels in aquatic sediments and in the food chain. Because they are so long-lived and can be transported long distances through the atmosphere, dioxins are now distributed on a truly global basis [Schechter, 1991, Brzuzy and Hites, 1996]. Inuit natives of Arctic Canada, for instance, have some of the highest body burdens of dioxins, furans, and polychlorinated biphenyls (PBCs) recorded, due to a diet dependent on fish and marine mammals from a local food chain contaminated by dioxin from distant industrial sources [Dewailly, 1994].

Second, dioxins are highly oil-soluble but insoluble in water; they thus bioaccumulate in fatty tissues and are magnified in concentration as they move up the food chain. In species high on the food chain, dioxin body burdens are typically millions of times greater than the levels found in the ambient air, soil, and sediments [Environment Canada, 1992]. Dioxins are also extraordinarily persistent in human tissues: estimated half-lives in humans are typically 5 to 10 years [EPA, 1994a].

At the apex of the food chain, the human population is particularly contaminated. A spectrum of dioxin-like compounds has been identified in the fat, blood, and mother's milk of the general population [Schechter, 1991]. Virtually all human exposures to these compounds occur through the food supply, particularly through consumption of fish, meat, eggs, and dairy products [Furst and Wilmers, 1991]. Significant quantities are passed from mother to child, during the most sensitive stages of development, across the placenta and via mother's milk [Schechter, 1991]. The daily PCDD/PCDF dose of an average nursing infant in the U.S. is 10 to 20 times greater than the average adult exposure [Birnbaum, 1994]. A nursing infant thus receives about 10% of the entire lifetime exposure to these compounds during the first year of life [EPA, 1994a].

### **Health Effects of Dioxin**

Biochemical studies have shown that dioxins act as powerful "environmental hormones." Like the body's natural hormones, dioxins can cross cell membranes and alter the activity of genes that regulate the body's processes of development and self-maintenance. A molecule of any dioxin-like compound can bind to a specific "receptor" protein within the cell; this complex then enters the cell nucleus and interacts with DNA to change the expression pattern of certain genes. The resulting changes trigger a cascade of biochemical reactions, including the synthesis and metabolism of hormones, their receptors, enzymes, growth factors, and other substances. Unlike a natural hormone, however, dioxin resists metabolic degradation and has an extraordinarily high affinity for its receptor [Birnbaum, 1994]. Tiny doses of these "false signals" can thus have powerful effects on processes regulated by hormonal mechanisms, including cell proliferation and differentiation, as well as organismal reproduction, development, metabolism, and immune function (see Table 2). Dioxins can thus be thought about as "toxic transmitters," interfering with, confusing, and disrupting natural body systems, such as hormone, immune, and neurological systems.

## **TABLE 2: Toxicological Effects of Dioxin-like Compounds**

### Modulation of hormones, receptors, and growth factors

- Steroid hormones and receptors (androgens, estrogens and glucocorticoids)
- Thyroid hormones
- Insulin Melatonin
- Vitamin A
- EGF and receptor
- TGF-a and TGF-b
- TNF-a, IL1b, c-Ras, c-ErbA

### Carcinogenesis

#### Immune system effects

- Suppression of cell-mediated and humoral immunity
- Increased susceptibility to infectious challenge
- Auto-immune response

#### Developmental impacts

- Birth defects
- Fetal death
- Impaired neurological development and subsequent cognitive deficits
- Altered sexual development

#### Male reproductive toxicity

- Reduced sperm count
- Testicular atrophy
- Abnormal testis structure
- Reduced size of genital organs
- Feminized hormonal responses
- Feminized behavioral responses

#### Female reproductive toxicity

- Decreases fertility
- Inability to maintain pregnancy
- Ovarian dysfunction
- Endometriosis

#### Other effects

- Organ toxicity (liver spleen, thymus, skin)
- Diabetes
- Weight loss
- Wasting syndrome
- Altered glucose and fat metabolism

Sources: Adapted from U.S. EPA, 1994a and Birnbaum, 1994

In laboratory animals, exposure to dioxins, particularly 2,3,7,8-TCDD, has been associated with a remarkable variety of toxicological effects (see Table 2). Some of these effects have occurred at extraordinarily low doses. For instance, exposure of monkeys to just 5 parts per trillion of 2,3,7,8-TCDD concentrations in the diet caused impaired neurological development and endometriosis [Rier, et. al., 1993, Bowman, et. al., 1989]. Pregnant rats receiving a single small dose of 2,3,7,8-TCDD on day 15 of pregnancy, had male offspring which appeared normal at birth, but at puberty were demasculinized, with altered reproductive anatomy, reduced sperm count, feminized hormonal responses, and feminized sexual behavior [Mably, et. al., 1991]. Very low doses of dioxin have also produced immune system changes in rats and monkeys [Hong, et. al., 1989, Neubert, et. al., 1992, Yang, et. al., 1994, Lucier, 1991, Enan, et. al., 1992]. The recent finding that the genome of the HIV-1 virus contains regulatory sequences that bind the dioxin-receptor complex and activate transcription of viral genes is cause for concern that dioxin-like chemicals may also play a role in the expression of infectious disease [Yao, et. al., 1995].

Dioxins clearly cause cancer. All 18 studies on the carcinogenicity of 2,3,7,8-TCDD have been positive, demonstrating that dioxin is a multisite carcinogen in both sexes in the rat, mouse, and hamster by all routes of exposure investigated [Huff, 1994]. EPA has estimated that current background exposures pose cancer risks as high as one-in-one-thousand, a level that exceeds "acceptable" risk standards by up to a thousand times and, if accurate, could correspond to as many as 3,500 U.S. cancer deaths per year due to dioxin exposure [EPA, 1994a]. According to one review, "Epidemiological data from occupationally exposed workers now show accumulating and convincing evidence that exposures to TCDD are associated with several cancers in humans: respiratory, lung, thyroid gland, connective and soft tissue sarcoma, hematopoietic system, liver, and all cancers [Huff, 1994]." The most toxic dioxin, 2,3,7,8-TCDD was recently classified as a known human carcinogen by the International Agency for Research on Cancer (IARC), and is classified as a probable human carcinogen by the Environmental Protection Agency, and the National Institute for Occupational Safety and Health. The new IARC classification provides an important international recognition of the potential for dioxin to impact human health.

While the noncancer effects of dioxin in humans have received less attention, there is evidence that PCDD/F exposure reduces male sex hormone levels and libido [EPA, 1994a, Egeland, et. al., 1994, Wolfe, et. al., 1994, Webster and Commoner, 1994] and increases the risk of diabetes and related metabolic conditions [Wolfe, et. al., 1992, Sweeney, 1992]. In human infants, several studies indicate dioxin-mediated effects on physical, cognitive, and sexual development [Hsu, et. al., 1994, Chen, et. al., 1992]. Infants born to mothers who had consumed two to three meals per month of Great Lakes fish were underresponsive and hyporeflexic at birth and subsequently exhibited dose-dependent deficits in visual recognition memory and activity levels; these deficits were still present at follow-up at age 4. PCBs were measured in umbilical cord serum as a marker of total pollutant exposure, and the severity of developmental deficits correlated with PCB levels [Jacobson, et. al., 1990, 1992]. Finally, there is evidence of alterations in thyroid hormone levels, increased incidence of intracranial hemorrhage, and immune

suppression associated with lactational exposure to PCDDs, PCDFs, and PCBs in Europe and Arctic Canada [Weisgas-Kuperus, et. al., 1996, Pluim, et. al., 1993, Koppe, et. al., 1991, Dewailly, et. al., 1993].

### **A Global Risk to Public Health**

Every member of the general population, from the moment of conception until death, is now exposed to dioxin-like compounds due to ubiquitous contamination of the food supply. Several lines of evidence suggest that these "background" exposures may pose significant public health risks.

- There does not appear to be a safe or "threshold" dose below which no health effects occur [EPA, 1994a, Tritscher, et. al., 1994]. In fact, for those effects that have been investigated, the available data indicate a dose-response relationship that is linear or even supra-linear [Portier, et. al., 1990]. This indicates that low doses of dioxin cannot be assumed safe.
- The "background" exposures and body burdens of PCDD/F in the general human population are approaching levels that clearly produce biological impacts in laboratory animals. Two analyses have concluded that the current human body burdens are equal to or within one or two orders of magnitude of the levels that produce metabolic, reproductive, developmental, and immunological effects in laboratory animals [Devito, et. al., 1995, Webster and Commoner 1994]. This information suggests that at least part of the general human population is now at risk of adverse health impacts due to dioxin exposures.
- It is clear that levels of dioxin-like compounds in the environment have already caused large-scale effects on wildlife populations, particularly fish-eating birds and marine mammals. The most severe effects appear to be endocrine-mediated impairment of development, reproduction, neurological, and immune function [Reinjders and Brasseur, 1992, Fox, 1992, Ross, 1995, SAB, 1989,1991,1993]. If dioxin levels are high enough to cause these effects in wildlife, then humans--who are high on the food chain but have slower generation times--are likely to be at risk as well.

EPA's chief toxicologist on the dioxin reassessment reinforced the conclusion that the health of the general population is at risk in a recent scientific publication:

"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, 1994]."

It is possible that universal exposure to dioxin-like compounds has already had large-scale impacts on human health. For instance, incidence rates for many types of cancer have been increasing internationally for several decades [Davis, et. al., 1991, Hoel, et. al., 1992]. Further, several authors have reported a global trend since the 1950s of falling sperm counts and increasing abnormalities of the human male reproductive tract. [Carlsen, et. al., 1992, Lancet, 1995, Sharpe and Skakkebek, 1993, Auger, et. al., 1995]. Dioxins and other persistent organic pollutants that disrupt the endocrine system – none of which were produced in significant quantity before World War II -- may play a role in these trends which have occurred during the period of increasing exposure to these compounds. The observations from laboratory, epidemiological, and wildlife studies do not establish dioxin pollution as the cause of trends in human reproductive health or cancer rates, but they suggest a sound basis for concern and preventive action.

### **A Zero Discharge Policy for Dioxin**

Although we do not know the extent to which dioxin exposure has already affected human health on a global scale, we can conclude with certainty that universal exposure to these compounds represents a risk to public health. Any increase in exposure will increase that risk and/or the severity of toxic effects. Only by significantly reducing dioxin exposure can public health be protected.

The finding that public health is now at risk has extraordinary implications for environmental policy. Even if there is a safe dioxin dose, we are already at, near, or above that threshold. 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 such thing as an "acceptable" discharge that poses insignificant risk to public health. Given this current health hazard, the only acceptable level of dioxin release into the environment is zero.

Merely reducing dioxin releases is not enough. The data on trends in dioxin and PCB levels in the environment indicate that exposures will continue at an unsafe level so long as dioxin releases continue --and longer. Persistent pollutants eventually reach a steady-state level in the environment, determined by the magnitude of discharges. New or increased industrial inputs of dioxins cause environmental concentrations to increase slowly until they equilibrate at a point when the rate of "removal"-- either through degradation or accumulation in sediments, soils, and vegetation -- matches the rate of release. If inputs are reduced, levels will slowly decline and then plateau at a new steady state. Thus, concentrations of PCBs in water and the food chain increased steadily from the 1940s to the 1960s, after which they leveled off; after PCBs were 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 dioxin levels rose consistently from 1940 to 1970 as the manufacture and incineration of chlorine-based chemicals expanded [Czuczwa and Hites, 1986]. In the early 1970s, regulatory changes --including increased controls on certain types of incinerators and restrictions on leaded gasoline (which contains chlorinated additives), and several highly dioxin-contaminated pesticides -- reduced dioxin emissions; after a lag, environmental concentrations declined slowly from the late 1970s to the late 1980s [Environment Canada, 1992, Alcock and Jones, 1996]. Dioxin levels in human tissues (as well as breast milk) may also have declined, although the data are less clear [Schechter, 1996].

This pattern suggests that reducing industrial releases of persistent toxic substances, such as dioxins, will result in decreased contamination of the environment and human tissues. If releases continue at a reduced rate, however, exposure and contamination will also continue. Further, although these substances may be "removed" into sediments, soil, and vegetation, 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 it is essential that releases of dioxins be brought to zero as quickly as possible. The more quickly dioxin releases are brought to zero, the more quickly the health risk will abate.

## Notes

\* The term "dioxin" is used throughout this report to refer to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). The term "dioxins" generally refers to the class of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) with between 1 and 8 chlorine atoms. Where research addresses only a subset of the class, they are referred to specifically. The group of dioxin-like compounds includes all substances similar in chemical structure and biological effects to 2,3,7,8-TCDD; among these are certain polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), as well as a number of coplanar polychlorinated biphenyls (PCBs). The toxicity of a mixture of dioxin-like compounds can be expressed in toxic equivalency (TEQ) units, a single term that represents the sum of the concentrations of each dioxin-like substance, adjusted by its toxicity relative to that of 2,3,7,8-TCDD, the most toxic dioxin. A spectrum of dioxin-like compounds are invariably produced that form 2,3,7,8-TCDD.

## Chapter 2 - Dioxin Sources: Focusing on Prevention

### Overview of Dioxin Sources

Several inventories of dioxin sources in the U.S., Europe, and globally have been prepared. All these inventories are preliminary, and further investigation is likely to lead to quantitative revisions. Not all dioxin sources have been identified, and these inventories omit known dioxin sources for which data are inadequate to make quantitative estimates of dioxin releases. (A listing of identified dioxin sources is provided in Table 3).

Nevertheless, these inventories represent the most complete database on dioxin sources currently available, and the information they contain provides a starting point from which to begin discussion of a dioxin prevention strategy.

According to all available inventories, combustion-related processes account for the majority of identified dioxin emissions.\* In the U.S., this sector accounts for over 90% of the dioxins and furans entering the environment from known sources (see Table 4). According to EPA, medical waste incinerators, municipal waste incinerators, hazardous-waste-burning incinerators and cement kilns, industrial and residential wood-burning facilities, and sewage sludge incinerators are the largest identified dioxin sources. Also important in this category are combustion-based metallurgical processes (including copper, lead, and steel smelters/recyclers), combustion of automobile and truck fuels, and the burning of wood products. Minor sources within this category include coal-fired utilities, drum and barrel reclamation, tire incineration, carbon reactivation furnaces, and forest fires. Accidental fires in homes, offices and industrial fires may be an important dioxin source, but inadequate information was available for EPA to develop an estimate.

Three independent studies have confirmed EPA's picture of dioxin generation, particularly the central role of incinerators (see Table 4). One, a comprehensive report by the Center for the Biology of Natural Systems, found that six source sectors accounted for 91.5 percent of all identified dioxin deposition into the Great Lakes: medical waste incinerators, municipal solid waste incinerators, iron ore sintering plants, hazardous waste incinerators and cement kilns, and secondary copper smelters [Cohen and Commoner, 1995]. A Princeton University study found that trash incinerators, hospital waste incinerators, hazardous waste incinerators, open garbage burning, and copper and steel recyclers were responsible for 83 percent of dioxin air emissions in the U.S. [Thomas and Spiro, 1995]. A worldwide inventory of dioxin sources placed municipal waste incineration, hazardous waste incineration, and ferrous metal smelting at the top of its list [Brzuzy and Hites, 1996a] (see Table 5).

### **TABLE 3: Summary of Processes that Form Dioxin and Related Chemicals**

#### Production of chlorine gas

Chlorine electrolysis with graphite electrodes Chlorine  
electrolysis with titanium electrodes

#### Use of chlorine gas - chemical industry

Synthesis of chlorinated aromatic chemicals  
Synthesis of chlorinated solvents (TCE, perc, carbon tetrachloride)  
Synthesis of feedstocks for PVC plastic  
Synthesis of other aliphatic organochlorines  
Manufacture of some inorganic chlorides

#### Use of chlorine gas -- other industries

Pulp and paper -- chlorine bleaching  
Water and wastewater disinfection  
Production of refined metals -- manufacture with chlorine (Ni, Mg)

#### Use of organochlorines

Chemical industry: use of organochlorine intermediates in chemical synthesis  
Solvents: Use in reactive environments  
Oil refining with organochlorine catalysts  
Use of pesticides in presence of heat (wood treatment, etc.)  
Iron/steel sintering with organochlorine cutting oils, solvents, or plastics  
Burning gasoline or diesel fuel with organochlorine additives  
Chlorine-based bleaches and detergents: use in washing machines/dishwashers

#### Combustion of Organochlorines

Medical waste incinerators  
Municipal waste incinerators  
Hazardous waste incinerators  
Cement kilns burning hazardous waste  
Accidental fires in homes, offices, and industrial facilities  
Aluminum recycling/smelting  
Steel and automobile recycling/smelting  
Copper cable recycling/smelting  
Aluminum recycling/smelting  
Wood burning (w/organochlorine residues, pesticides, etc.)

#### Environmental transformation

Transformation of chlorophenols to dioxins in the environment

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. For full reference information, see EPA, 1994b, Thornton, 1994, and Thornton, 1995.



**TABLE 4: Dioxin Sources in the U.S.**

% of total identified emissions

<b>Sources:</b>	<b>U.S. EPA(1)</b>	<b>Cohen(2)</b>	<b>Thomas (3)</b>
Medical waste incinerators	45	48.7	10
Trash and apartment incinerators	42	20.1	65
Haz. waste incinerators/Cement kilns	4*	8	2.5
Pulp mills	3	ND	0.1
Wood burning	3	1.9	5.8
Copper recycling	2	4.2	2.5
Forest fires/Agricultural burning	0.7	ND	7.5**
Vehicle fuels	0.7	1.4	0.7
Sewage sludge incineration	0.2	0.6	1
Dioxin-contaminated chemicals	<1	ND	4.5
Lead recycling	<0.1	ND	ND
Steel smelters	ND	10.6	0.8
Open waste burning	ND	ND	2.5
Accidental fires	ND	ND	0.7**

(1) From U.S. EPA, 1994. Percent of releases of PCDD/F (as TEQ) to air, water, and land from all identified sources, using median estimates. Sources listed by rank in EPA inventory.

(2) Percent of identified airborne emissions of PCDD/F (as TEQ) to the Great Lakes (Commoner and Cohen, 1995).

(3) Percent of identified air emissions sources of PCDD/F in the U.S. (Thomas and Spiro 1995).

\* Based on EPA's estimate that dioxin emissions from cement kilns burning hazardous waste are one order of magnitude greater than that from kilns burning traditional fuels (EPA, 1994b).

\*\* Estimate characterized by major uncertainty, according to Thomas and Spiro, 1995.

**TABLE 5: World Dioxin Sources to the Atmosphere**

<b>Source Sector</b>	<b>emissions (kg TEQ/yr)</b>	<b>range</b>
Municipal waste incineration	1130	680 - 1580
Cement kilns burning hazardous wastes	680	400 - 960
Ferrous metals production	350	210 - 490
Cement kilns (no haz. wastes)	320	190 - 450
Medical waste incineration	84	49 - 119
Secondary copper smelting	78	47 - 109
Leaded fuel combustion	11	6 - 16
Unleaded fuel combustion	1	0.6 - 1.4
Total	3000	2400 - 3600

Source: Brzuzy and Hites, 1996a. Range represents 90% confidence limits

There are also a number of important dioxin sources not related to combustion. For instance, pulp and paper mills rank third among all dioxin source sectors in EPA's inventory. Chemical manufacturing is the other important non-combustion dioxin source. EPA has identified over 100 pesticides contaminated with dioxin, along with a wide range of chlorinated solvents, feedstocks for plastics, and chemical intermediates [EPA, 1994b]. This list includes representatives of the full range of chlorinated compounds,

from simple aliphatic to complex aromatics. EPA does not provide a quantitative estimate of total dioxin releases associated with the use of chlorine in the chemical industry. Given the huge quantity of organochlorine products manufactured worldwide – approximately 11,000, containing some 25-30 million tons of chlorine each year -- dioxin contamination in the parts per billion or even parts per trillion range would make the production of organochlorines an important dioxin source. Finally, the use of chlorine in metallurgical processes [Oehme, 1989], water disinfection [Rappe, 1991], and the manufacture of some inorganic chemicals [Heindl and Hutzinger, 1987], are also known to produce dioxins, but in lesser quantities than those sectors discussed above.

### **Focusing on the Materials that Create Dioxin**

PCDD/Fs are never manufactured on purpose but are accidental by-products of a host of processes that involve chlorine-based chemicals. A strategy for eliminating dioxin releases must thus begin by identifying and organizing dioxin sources. The appropriate method to characterize and identify dioxin sources, however, has proven to be quite contentious. There are two methods by which dioxin sources can be identified and grouped. One approach emphasizes the types of facilities that release dioxin into the environment. The other focuses on the materials that are introduced into industrial processes and turn them into dioxin sources.

The facility-type approach has been taken by the chemical industry and most regulatory agencies [TNO, 1994, SEPA, 1996, EPA, 1994a]. Inventories produced by this method list the many industrial processes in which dioxins are known to be produced, and organize them by industrial sector (waste disposal, metallurgy, chemical manufacture, etc.). This approach gives the impression that dioxin is formed by so many unconnected sources that a curtailment strategy would require a byzantine set of regulations addressing virtually every industrial activity and combustion process known. In the U.S., some industry representatives have used this approach to confuse and mystify discussions of dioxin sources. In its "Dioxin Reassessment Briefing Packet," the Chlorine Chemistry Council of the Chemical Manufacturers Association (CCC/CMA) has written:

"Among the natural sources of dioxin are forest fires, volcanoes, and compost piles. Man-made sources of dioxin include municipal, hospital and hazardous waste incinerators, motor vehicles, residential wood burning and a variety of chemical manufacturing processes. With so many sources, it is not surprising that scientists have detected dioxins virtually everywhere they have looked [CCC, 1994]."

The superior approach emphasizes the materials that cause dioxin formation in these diverse processes. Many dioxin sources can be tied together by the common chlorine-containing feedstocks they share. For example, the use of chlorinated solvents in industry can cause dioxin formation during the manufacture of these chemicals, some of their uses, and their disposal by incineration. The production of chlorine-bleached paper may result in dioxin discharges via pulp mill effluent and air emissions, black liquor boilers, sludge incinerators and land disposal facilities, wastewater treatment plants that receive

sludge or effluent from these plants, and --once the paper products have been used -- from trash incinerators and recycling facilities that process waste paper [EPA, 1994a]. Thus, a single material can create, throughout its lifecycle, a great diversity of dioxin sources. By focusing on the materials that give rise to dioxin, the second method reveals opportunities to replace dioxin-generating products with materials that do not result in dioxin formation. For example, the switch to non-chlorine bleaching methods in the pulp and paper industry could essentially eliminate the industry's dioxin pollution.

A materials-based approach leads to effective dioxin prevention measures. The resulting solutions are relatively straightforward, clearly implemented programs such as chemical phase-outs, product reformulation, and materials substitution. The focus on discharge points, in contrast, leads to an emphasis on pollution control and management technologies for each type of dioxin-releasing facility. Because dioxin sources comprise dozens of industrial sectors and thousands of individual facilities, ongoing technological management of these sources can be expensive, technically difficult, and administratively demanding. Improving operating conditions of an industrial process can reduce but not prevent dioxin generation (see EPA, 1989, for example). Since some of the dioxins will escape destruction in pollution control devices, releases of dioxins to air, water, or land will occur. Some dioxin sources -- such as accidental fires -- cannot be controlled at all. Pollution management is thus characterized by partial effectiveness, diminishing returns, and continual intervention and expense. Prevention, in contrast, requires retooling of a process to avoid the root dioxin-generating material, but once this investment is made, associated dioxin releases can be essentially prevented.

### **Synthetic Chlorine Production: The Uniquely Preventable Factor in Dioxin Formation**

The vast majority of dioxin generation can be prevented if the chlorine-containing materials that cause dioxin synthesis can be kept out of industrial and other reactive processes. In fact, a large proportion of dioxin emissions from a multiplicity of sources can be attributed to a handful of individual chlorine-related products and processes. PVC plastic, chlorine-based pulp bleaches, pesticides, and chlorinated solvents are likely the most important causes of dioxin from the majority of the significant sources identified in the available inventories (see Table 6).

**TABLE 6: Summary of Dioxin Sources and Their Chlorine Donors**

<b>Dioxin-generating process</b>	<b>Primary chlorine donor</b>
Municipal waste incineration	PVC, bleached paper, household hazardous waste
Medical waste incineration	PVC
Pulp mills	Chlorine-based bleaches
Hazardous waste incineration	Spent solvents, chemical industry wastes, pesticides
Wood burning (industrial and residential)	PVC, pentachlorophenol, chemical hardeners
Secondary copper smelting	PVC-coated cables, PVC in telephones and electronic equipment, chlorinated solvents/cutting oils
Chemical manufacturing	Chlorine or organochlorines as a reactant
Home and building fires	PVC, pentachlorophenol, PCBs, chlorinated solvents

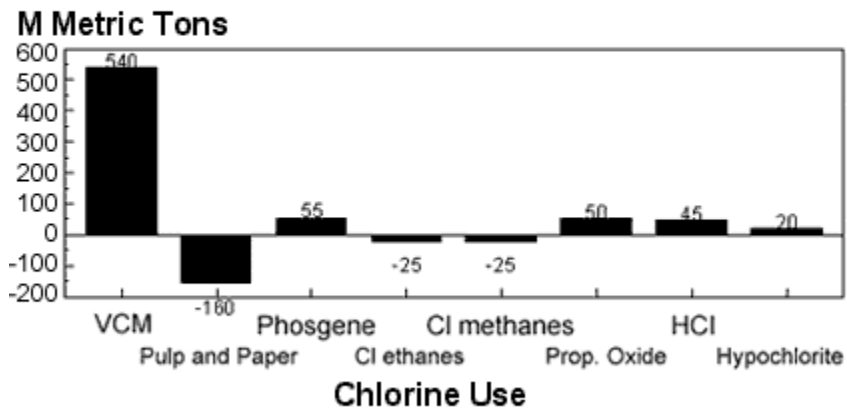
Vehicle fires  
 Ferrous metal smelting  
 Vehicle fuel combustion  
 Forest fires  
 Sewage sludge incineration  
 Secondary lead smelting

PVC, chlorinated cutting oils  
 PVC, chlorinated cutting oils, chlorinated solvents  
 Chlorinated additives  
 Pesticides, deposition of airborne organochlorines  
 Chlorination by-products  
 PVC

Sources: EPA, 1994b, Versar, 1996, Thomas and Spiro, 1995, Dempsey and Oppelt, 1993, Wilken, 1994, Heindl and Hutzinger, 1987, Marklund, 1990, Tysklind, 1989, Aittola, et. al., 1990, Green, 1993, Brzuzy and Hites, 1996a, 1996b.

Compared to other chlorinated materials, PVC production and demand are on the rise, and thus its contribution to the global dioxin burden is likely to increase substantially (see Figure 1). The appropriate priority targets for a prevention strategy should thus be this group of materials that are the main causes of dioxin formation. By shifting focus from the point of discharge to an upstream emphasis on the production and use of dioxin-generating materials, we can prevent these materials from entering reactive environments before they can turn into dioxin.

**FIGURE 1**  
**World Chlorine Demand**  
 Projected Annual Average Growth 1995-2005



Source: Meers, 1995

The ultimate preventable cause of dioxin generation is the input material that supplies chlorine for incorporation into dioxin. Although a number of potential pathways for dioxin formation have been proposed, all require the following: a source of chlorine, a source of organic matter, and a thermally or chemically reactive environment in which these materials can combine [EPA, 1994b]. It is the presence of chlorine donors that turns industrial processes with reactive conditions into dioxin sources, and these chlorinated materials are the appropriate focus for preventive efforts. In most cases, the chlorine donor is a product or waste of industrial chlorine chemistry. Organic matter is ubiquitous, and reactive environments (such as fires and industrial processes) are common; synthetic chlorine production is the uniquely preventable factor in dioxin generation. It is because chlorine chemistry and its products have become so widespread that dioxin formation now appears to be ubiquitous.

The field of industrial chlorine chemistry provides a chain of opportunities for dioxin synthesis in which the three necessary factors are present. Dioxin generation begins with the production of chlorine gas in a chlor-alkali plant -- the process in which large quantities of electricity are used to transform a salt solution (sodium chloride) into elemental chlorine gas, sodium hydroxide, and hydrogen. Opportunities for dioxin synthesis continue with the use of chlorine in industrial or municipal processes, where the chlorine gas itself creates the reactive environment. Further dioxin generation takes place when organochlorines are used in reactive environments, recycled by combustion, or disposed of by incineration. Once released into the natural or human environment, chlorinated organic chemicals are subject to additional transformation processes that can produce dioxins, including photolysis, forest fires, and building fires. Thus, dioxin appears to be formed at some point in the lifecycle of almost all chlor-alkali products and processes (see Table 3).

Some authors, particularly those representing the chemical industry, have contended that dioxin is not predominantly associated with chlorine chemistry. In 1980, Dow Chemical advanced the position that dioxin occurs naturally in the environment and has thus been with us "since the advent of fire." With this "Trace Chemistries of Fire" theory [Bumb, 1980], Dow argued that dioxins are formed in any combustion process, including forest fires, volcanoes, and household stoves due to the presence of natural chloride salts, like table and sea salts. Dow and its allies continue to claim that the importance of "natural dioxin" renders ineffective policies that focus on industrial sources of dioxin. Responding to concern raised by EPA's 1994 dioxin reassessment, for instance, the Chlorine Chemistry Council emphasized the importance of volcanoes and forest fires as dioxin sources. Similarly, the Vinyl Institute has relied extensively on a study it commissioned that purported to find that dioxin is formed in almost any combustion device irrespective of differences in quantities of chlorinated chemical products present [Rigo, 1995] (see Case Study 1). The Vinyl Institute has recently even questioned whether the extensively researched health effects of dioxin are real [Vinyl Institute, 1996a].

### **The "Trace Chemistries" theory has been proven false.**

According to one recent review, "There is no experimental evidence to support the abundant, natural production of PCDD/F [Brzuzy and Hites, 1996b]." A number of lines of evidence indicate that dioxin contamination is due to the manufacture and dispersal of chlorinated organic chemicals, not due to the natural presence of chloride salts.

- If any dioxin is produced naturally, the quantities are negligible. EPA shows that more than 99 percent of all dioxin in the US comes from industrial sources [EPA, 1994b] and a global inventory indicates that a maximum of 3% of dioxin deposition is caused by the burning of "biomass," including the combustion of wood contaminated with chlorinated pesticides and other chlorinated materials, and atmospheric deposition products [Brzuzy and Hites, 1996b].

- The data in Dow's original paper indicate that natural sources are insignificant. Soil samples from Dow's own chlorine chemical manufacturing and incineration facility in Midland, Michigan contained dioxin in concentrations thousands of times higher even than in other urban areas, which are themselves contaminated by industrial sources. And while trace amounts of dioxin were found in combustion residues from automobiles, home fireplaces, and cigarettes, the dioxin levels found in particulate matter from Dow's own incinerators were orders of magnitude higher [Bumb, 1980].
- The dioxin levels found in the preserved tissues of ancient human beings indicate that dioxin was not a significant pollutant before the advent of chlorine chemistry. These studies have found that dioxin levels in the tissues of ancient humans --including those who cooked over indoor fires --are no more than one to two percent of the amount found in modern humans [Schechter, 1991, Ligon, 1989]. According to EPA, "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 [EPA 1994b]."
- Studies of sediments in the Great Lakes [Czuczwa and Hites, 1986] and elsewhere [reviewed in Alcock and Jones, 1996] show that dioxin was virtually non-existent until the 20th Century; levels began to climb towards their current concentrations only after World War II. One study was conducted on a lake, the watershed of which suffered a forest fire in 1937, but dioxin levels showed no response to this event [Brzuzy and Hites, 1996b]. The general dioxin trend, particularly in the Great Lakes, corresponds to the development and expansion of the chlorine chemical industry but bears no relationship to the increasing combustion of coal.

According to one study:

"There is an abrupt increase in PCDD and PCDF concentrations around 1940.... Starting at this time, the production of chlorinated organic compounds such as chlorobenzenes and chlorophenols increased substantially. These compounds are used in a variety of products, including building supplies, herbicides and packaging. Much of these materials eventually become incorporated in solid wastes. The trend for the production of chloro organic compounds is very similar to the sedimentary PCDD and PCDF profiles. The agreement between these two trends is convincing despite the uncertainties introduced by sediment mixing and the errors inherent in the dating and quantification techniques....It is clear that the high levels of dioxins and furans found in presently accumulating sediments are not due to the advent of fire [Czuczwa and Hites, 1984]."

## **Dioxin From Incinerators: The Role of Chlorine**

PVC is of particular concern because it is an important chlorine donor in many types of incinerators. Following its "Trace Chemistries of Fire" theory, the chemical industry has recently argued that incinerators release dioxin without relation to the amount of chlorinated organic materials that are fed to them [Rigo, et. al., 1995]. Of course, chlorine feed is not the only factor involved in dioxin formation; facility design, operating conditions, and the presence of catalysts also play significant roles. Without the products and wastes of the chlorine industry, only natural chloride-containing salts could serve as chlorine donors. But a host of studies indicates that reducing the feed of chlorinated organic compounds to incinerators is the best way to reduce dioxin formation.

First, there is no experimental support for the claim that combustion of chloride salts can produce significant quantities of dioxin. As EPA points out, no studies have adequately insured that purportedly chlorine-free materials do not contain residues of organochlorine contaminants now ubiquitous in air, water, and biological samples [EPA, 1994b]. On the other hand, it is clear that when chlorinated organic substances are added to a mixture that contains salt, dioxin formation increases by orders of magnitude. Removing these chlorinated substances from incinerator waste streams should thus significantly reduce dioxin generation.

A number of laboratory studies have clearly demonstrated the central role of chlorinated organic substances in dioxin formation in incinerators. The German EPA, for instance, found that combustion of chlorinated containing plastics and other chemicals produced higher dioxin concentrations in ash residues than combustion of chloride-containing but chlorine-free paper, wood, cotton, or wool [Thiessen, 1991]. Dioxin concentrations were found to increase considerably as more PVC was added to a mixture of chloride-containing coal and bark [Kopponen, 1992]. Wilken (1994) also found the addition of PVC during the combustion of wood products to increase dioxin levels in the ash [Wilken, 1994]. In a laboratory experiment, combustion of coal in the presence of salt produced trace quantities of dioxins in offgas, but when elemental chlorine was added to the mix, total dioxin formation increased considerably [Mahle and Whiting, 1980]. Finally, combustion of vegetable matter does not produce PCDD/Fs, but combustion of vegetable matter in the presence of chlorine gas or PVC plastic results in formation of chlorophenols and PCDD/Fs [Liberti, 1983].

In full-scale incinerators, the Danish EPA has found that doubling the PVC content of an incinerator's wastefeed increases dioxin emissions by 32 percent, while doubling the chloride content increases dioxin emissions by a much smaller margin [Danish EPA, 1993]. A 1993 study for the Dutch Environment Ministry reported that reducing the PVC feed results in a corresponding reduction in dioxin emissions, particularly when chloride concentrations are kept low [Kanters and Louw, 1993]. According to the Danish Technical Institute (1995), citing a 1994 report by Wikstrom, et. al., PCDD/F and PCBs emissions are directly correlated with the level of chlorine in the fuel when total chlorine levels are greater than one percent. Extensive studies at an incinerator at the University of

Florida have documented a clear relationship between emissions of dioxin-indicator compounds and the feed of PVC [Wagner and Green, 1993]. According to the authors:

"These experimental, phenomenological and theoretical studies of toxic emissions from incineration all support the physically intuitive hypothesis that reduction of chlorinated plastics in the input waste stream results in reduction of aromatic chlorinated organic emissions.... We are convinced that, when all other factors are held constant, there is a direct correlation between input PVC and output PCDD/PCDF and that it is purposeful to reduce chlorinated plastics inputs to incinerators [Wagner and Green, 1993]."

A recent Swedish study noted that dioxin formation is directly related to chlorine content only when chlorine levels in the fuel exceed 0.5 percent [Wikstrom, et. al., 1996]. It is possible that a relationship exists below this level but cannot be statistically detected due to the inherent limitations in measuring dioxins at low concentrations [Costner, 1997].

Several studies have not found a relationship between incinerator dioxin emissions and PVC content of the waste feed. For instance, two trial burns at municipal waste incinerators found no statistically significant association between PVC feed and dioxin emissions [Visalli, 1987, Mark, 1994]. Neither of these investigations, however, controlled or adjusted for variations in other factors that are also known to affect dioxin emissions, such as operating conditions and wastefeed composition. A potential relationship between PVC and dioxin may thus have been masked by fluctuations in other factors. Indeed, an EPA reanalysis of the data from one of these studies [Visalli, 1987] indicates that when combustion conditions were held constant, emissions of dioxins and furans tended to increase as PVC content of the waste rose [EPA, 1988].

In summary, although some dioxin can be formed from the combustion of chloride containing salts, anthropogenic materials containing organochlorines are clearly the predominant and most readily preventable dioxin precursors in combustion sources. Indeed, as the Danish Technical Institute (1995) has written, "It is most likely that the reduction of the chlorine content of the waste can contribute to the reduction of the dioxin formation, even though the actual mechanism is not fully understood."

### **Case Study 1: The Rigo, et al. Study: Science for Sale**

The Vinyl Institute (VI) and the Chlorine Chemistry Council (CCC), industry trade organizations, and others, including national and international government bodies have asserted that there is no relationship between chlorine feed and dioxin generation in incinerators, based on a recent study sponsored by the PVC industry itself [Rigo, et. al., 1995]. This report by Rigo, Chandler, and Lanier, was not experimental; the authors did not generate new data but instead compiled existing trial burn data from a large number of incinerators and concluded that there is no statistically significant relationship between chlorine input and dioxin concentrations in stack gases.



Rigo, et. al.'s failure to find a relationship between dioxin in stack gases and chlorine is more likely the result of bad study design than the fact that no relationship exists. Rigo, et. al. did not control or adjust for changes in combustor design, waste type, operating parameters, or any other factors, so it is not surprising that a relationship with chlorine was not detected. In general, when an outcome is determined by many factors, a significant relationship with one factor will only be evident if the others are held constant or adjusted for statistically. Otherwise, the "noise" caused by the other factors will drown out the "signal" -- the relationship between the factor of interest and the outcome. Chlorine input is clearly not the only factor that determines the magnitude of dioxin emissions from incinerators: combustion conditions, other components in the waste, and other variables also affect the amount of dioxin that will be released.

The Rigo, et. al. study is undermined by the use of inappropriate data, as well. Most of the trial burns which provided the data for the authors' analysis were not designed to examine the relationship between chlorine input and dioxin output, so the resulting data are not suitable for a statistical analysis. The Rigo, et. al. study is also marred by a reliance on the concentrations of dioxins and chlorine in stack gas rather than total mass of chlorine input and dioxin output; if the total flow of stack gas increases, concentrations can decrease even if dioxin generation or chlorine feed increases; the variables the authors examined are thus inappropriate for evaluating a relationship between chlorine input and dioxin output. Also, the authors addressed only the relationship between certain measures of chlorine input and dioxin concentrations in combustor gases and failed to examine the more important measure of chlorine input and total dioxin output (gases, fly ash, scrubber water). There is every reason to expect that dioxin concentrations in stack emissions could decrease at the same time that total dioxin input increases. (For an extensive discussion of the Rigo, et. al. report, see Costner, 1997).

Despite these flaws, a reanalysis of the statistical evaluations made by Rigo, et. al. [Costner 1997] indicates that, contrary to the report's text, the majority of incinerators examined did show a positive relationship between chlorine input and stack gas emissions. Indeed, on a facility by facility basis, a positive relationship between hydrogen chloride (a surrogate for chlorine feed) and dioxin was found at 15 of 22 municipal waste incinerators examined. Confidence levels for a positive correlation were >95 percent at five facilities, >90 percent at two facilities and >80 percent at eight facilities. Of the seven facilities exhibiting a negative correlation, results from one facility had a confidence level of >90 percent, while the remaining six had confidence levels of <80 percent. These results suggest that stack emissions of dioxin are related to chlorine content in the waste after all. Rigo, et. al.'s statistical evaluations for medical waste incinerators also showed a predominance of positive correlations. In an analysis of the Rigo, et. al. raw data for cement kilns, a predominance of positive correlations was also demonstrated. Given the flaws in study design, data quality, and statistical re-evaluation, no defensible conclusions regarding the relationship of chlorine input with dioxin output can be drawn from the Rigo, et. al. study.

There are other reasons to be skeptical of the Rigo report. A Vinyl Institute memo [Goodman, 1994] indicates the financial and political interests that informed the writing of Rigo's report:

"The Vinyl Institute has created an Incineration Task Force in anticipation of adverse EPA actions regarding dioxins and furans.... Dick Magee brought forward a proposal from the American Society of Mechanical Engineers to hire Rigo & Rigo Associates, Inc., of Cleveland, OH. The purpose of ASME as the contractor is to provide unassailable objectivity to the study....

"The Incineration Task Group interviewed Dr. H. Gregory (Greg) Rigo, principal of Rigo & Rigo Associates, Inc. by phone and found him to be extremely knowledgeable about incineration and to have several proprietary databases VI had not discovered. He is also user friendly, i.e., willing to set his priorities to our needs) and appears to be sympathetic to Plastics, Vinyl, PVC and Cl2....

"The ASME proposal calls for \$130,000 for the Rigo & Rigo/ASME study. Since there are many unanswered questions regarding dioxins and since VI may want to use Greg Rigo as an expert witness or advocate to talk about the report, I am proposing an additional \$20,000 as a contingency fund, for a total of \$150,000 to be fully funded by VI [Goodman 1994, emphasis added]."

## Notes

\* Most of these inventories deal primarily with emissions to the atmosphere; releases to land and water are not included. Further, most account for only a small percentage of the total estimated dioxin flux into the environment (10%-50%). Thus, there may be dioxin sources that have not yet been quantified, or the contribution of addressed sources may be underestimated.

# Chapter 3 – PVC's Role in Dioxin Formation

## The PVC Lifecycle

The lifecycle of PVC represents a large – and possibly the largest – single material source of dioxins. The PVC lifecycle has three stages. In all of them, dioxins are known to be produced.

- **Manufacture.** The first step in PVC's lifecycle is the production of elemental chlorine gas by the energy-intensive electrolysis of salt. Dioxin is produced in this process. This chlorine is then reacted with ethylene to produce ethylene dichloride (EDC), a process in which very large quantities of dioxin are produced. EDC is then converted in another chemical reaction to vinyl chloride monomer (VCM), which is then polymerized to form pure PVC plastic. Pure PVC is then mixed with various additives and used to produce PVC-containing products. Dioxins may be formed in some of these processes, as well. The wastes from the manufacture of PVC and its feedstocks are incinerated in most cases, producing yet more dioxins.
- **Use.** PVC is used in a wide range of consumer, construction, and medical products. In some uses, PVC may encounter reactive conditions, as when a building with PVC cables burns in an accidental fire. When PVC burns, dioxins are produced.
- **Disposal.** After their use, PVC-containing products may be dumped in landfills, burned in incinerators or furnaces, or, in some cases, recycled. When land disposed, PVC products have an essentially infinite lifetime. When burned in incinerators or landfill fires, PVC produces dioxins. When PVC serves as a coating or covering on metals that are recycled in smelters, dioxins are formed.

Indeed, PVC is an important, and sometimes the predominant, chlorine donor in sources responsible for the vast majority of all identified dioxin releases in the EPA's Dioxin Reassessment and in Cohen and Commoner's analysis of dioxin emissions in the Great Lakes [EPA, 1994b, Cohen and Commoner, 1995]. PVC provides a substantial proportion of the organically-bound chlorine in incinerators for trash and medical waste. PVC is also a significant chlorine source in some secondary metals smelters. In accidental fires, wood combustion, and open burning, PVC also contributes substantially to dioxin emissions. Compared to other chlorinated materials, PVC production is on the rise, and thus its contribution to the global dioxin burden is likely to increase substantially.

## PVC in Incinerators

Incinerators are responsible for the majority of identified dioxin releases, both in the U.S. and globally. What are the materials that result in dioxin formation in these combustion sources? As the Swedish EPA points out, "In combustion processes in which both chlorine and a carbon source are present, PCDD/Fs can be formed [SEPA, 1996]." Similarly, U.S. EPA's dioxin reassessment notes, "Dioxin-like compounds can be generated and released to the environment from various combustion processes when chlorine donor compounds are present [EPA, 1994b]." One recent survey of emissions

data from a wide range of combustion facility types indicates a "clear dependence" of dioxin and furan emissions on chlorine content of the waste feed [Thomas and Spiro, 1995]. The material that donates the chlorine is thus the appropriate focus for prevention efforts, be it PVC, chlorinated solvents, etc.

Although many materials can serve as chlorine-donors in combustion-based processes, one -- PVC plastic -- plays an important role in most of the major dioxin sources. This is not surprising, because PVC is the most abundant product of chlorine chemistry, consuming over 30 percent of all the chlorine produced in the U.S. each year. In Denmark alone, for instance, some 13,500 tons of PVC is incinerated each year [DTI, 1995]. The quantity that ends up in U.S. incinerators is uncertain, but total PVC production is immense: over 5 million tons per year in the U.S., and 20 million tons per year worldwide [SRI, 1993]. It has been unambiguously shown in the laboratory that burning PVC results in the generation of dioxin [Thiessen, 1991, Christmann, 1991]. Given the large quantities of PVC produced and sold in the U.S. and the fact that the burning of PVC results in the generation of dioxins, it is clear that incineration of PVC will likely result in large dioxin emissions.

### **Municipal Waste Incinerators**

In all inventories, municipal waste incinerators are the number one or two source of dioxin emissions to the environment. In the U.S., there are 211 trash incinerators with combined capacity of 48 million tons of waste each year. EPA's median estimate for dioxin emissions from these facilities is 4800 grams per year (TEQ). Municipal waste incinerators also produce an estimated 7 million metric tons of ash annually, which is disposed in landfills. The more effective the incinerator's emissions control device, the more severely contaminated its ash. Ash from trash incinerators carries about 1800 grams per year of dioxin (TEQ) into the land, based on an average concentration of 258 ppt TEQ [Versar, 1996].

PVC is common in packaging and consumer products. The major chlorinated plastic, PVC contributes a large percentage of the organically-bound chlorine found in municipal waste. PVC accounts for an estimated 0.5 to 0.8 percent of municipal waste. Despite this low percent by weight, PVC can contribute at least 80 percent of organically-bound chlorine and as much as 67 percent of the total chlorine (organochlorines plus inorganic chloride) in the waste stream [Danish EPA, 1993]. Other studies have made similar findings, with PVC comprising 50 percent or more of total chlorine in the waste stream [Kanters and Louw, 1993, Ecocycle, 1994, DTI, 1995, Brahms, 1989]. An estimated 200,000 to 300,000 tons of PVC is incinerated each year in U.S. trash incinerators, based on national incinerator capacity of 48 million tons per year [Versar, 1996], 80 percent capacity utilization, and PVC content ranging from 0.5 to 0.8 percent.

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 one of the only major source of chlorine and, subsequently, one of the only major dioxin precursors. Dutch incinerator studies indicate that reducing PVC feed causes significant

decreases in dioxin emissions [Kanters and Louw, 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 for PVC applications for which no feasible system of recycling and reuse can be established, the use of more environmentally sound alternative materials is to be preferred [Netherlands Environment Ministry, 1994]."

More recent Dutch research [Kanters, et. al., 1996] confirms these findings. In a laboratory experiment, chlorophenol emissions (an indicator for dioxins) were lowest when the majority of chlorine-containing substances were removed from the wastes. When 20% of the original quantity of compostables was included, emission doubled from this baseline; when 30% of the original quantity of PVC was included, emissions doubled again [Kanters, et. al., 1996].

### **Medical Waste Incinerators**

In U.S. dioxin inventories, medical waste incinerators (MWIs) are the largest or second largest dioxin source. There are about 2400 medical waste incinerators in the U.S., which burn about 848,000 tons of waste per year [Versar, 1996]. EPA's original inventory estimated annual dioxin emissions in the range of 1600 to 16,000 grams of dioxin (TEQ) into the air each year, with a median estimate of 5100 grams, making MWIs the largest dioxin source in the inventory [EPA, 1994b]. Although other analyses have confirmed that MWIs are major dioxin sources, the American Hospital Association has argued that the actual total is much lower [AHA, 1995]. EPA is expected to lower its quantitative estimate, but there is little doubt that medical waste incinerators will remain a significant dioxin source.

The majority of the available chlorine in waste from hospitals and other health care institutions comes from PVC plastic, which enters these facilities as packaging and in many disposable medical products [Green and Wagner, 1993, Coppinger, 1996]. PVC is the most commonly used polymer in the medical device arena. An estimated 700 million pounds per year of PVC are used in medical devices in the United States, with an annual growth rate of 6.4%. [Chemical Week, 1995]. PVC is used in packaging, gloves, infusion bags, tubing, trays, and numerous other medical applications. Since an estimated 80 percent of U.S. medical waste is incinerated [AWMA, 1994], as much as 280,000 tons of PVC may be burned in medical incinerators each year.

While municipal trash contains about 0.5 percent PVC, an estimated 9.4 to 15 percent of all infectious waste is PVC [Marrack, 1988, Hasselriis, 1993], and as much as 18 percent of non-infectious hospital wastes are PVC [Hasselriis, 1993]. According to one report for the city of New York, PVC gloves and IV-bags alone account for over 90 percent of the

organic chlorine and over 80 percent of the total chlorine content of medical waste [Green, 1993]. All PVC products taken together could likely contribute as much as 90 percent or more of the organically-bound chlorine and 80 percent of the total chlorine fed to medical waste incinerators. The large quantities of PVC in the medical waste stream is one reason why medical waste incinerators are such a significant dioxin source. The authors concluded that the substitution of PVC plastic is an effective way of reducing the environmental impacts of such incinerators [Green, 1993].

### **Hazardous Waste Incinerators and Cement Kilns Burning Hazardous Waste**

Commercial incinerators and kilns accept complex mixtures of chemical wastes from a wide variety of sources, while many chemical companies operate on-site incinerators, furnaces, and boilers for disposal of their own wastes. EPA estimates that hazardous waste incinerators and kilns release 120 to 1200 grams of dioxin (TEQ) per year, with a median estimate of 409 grams per year [EPA, 1994b]. These estimates are based upon carefully controlled trial burns, so actual emissions during routine operation or upsets may be significantly higher [Costner and Thornton, 1991]. As of 1994, there were 298 permitted incinerators, boilers and furnaces burning 2.3 million tons of hazardous waste per year; 34 cement kilns burned an additional one million tons of hazardous waste each year (including medical waste containing PVC). In addition to dioxin in air emissions, incinerators produce highly contaminated ash which is disposed on land. If pollution control devices are 99 percent effective, ash from these facilities will carry about 100 times more dioxin into the environment than air emissions will. Waste-burning cement kilns produce large quantities of contaminated dust -- an estimated 3 million tons per year with dioxin concentrations averaging 42.2 ppt TEQ [EPA, 1993]. Based on these estimates, kiln dusts would carry 118 grams of dioxin (TEQ) into each year. EPA has found that dusts are released to the environment during storage, transport, and disposal, which often takes place by dumping into quarries or piles [EPA, 1993].

Because hazardous waste destined for incinerators is predominantly organic chemical waste, the organically-bound chlorine content is much higher than in municipal waste, often reaching 10 percent of total waste feed. The major chlorine donors in incinerated hazardous wastes belong to two categories: wastes from the manufacture of organochlorine chemicals and spent chlorinated solvents [Oppelt, 1986, Dempsey, 1993]. Wastes from the manufacture of PVC feedstocks are likely a significant contributor to the first category: EDC/VCM manufacture is the largest use of chlorine in the chemical industry -- accounting for more than half of all chlorine used within the organic chemicals sector [SRI, 1993]; millions of tons of chlorine-rich wastes are generated by these processes that are incinerated in on-site or commercial facilities. Given the large amount of PVC production wastes incinerated each year, it would appear that the PVC lifecycle is an important indirect chlorine donor and dioxin precursor in hazardous waste incinerators, as well.

## Metals Recycling

In many cases, products containing PVC (usually as a coating or covering on a metal product) are recycled in combustion-based processes. The result is the formation of significant amounts of dioxin. Indeed, PVC is an important chlorine donor in the secondary processing facilities that are considered major dioxin sources. The recycling of PVC itself has not been thoroughly analyzed for the creation of dioxins, and is thus not discussed here.

Secondary copper smelters are major dioxin sources, releasing 74-740 grams of dioxin per year (TEQ), according to EPA. These facilities produce dioxins in large part because they process for recycling large quantities of PVC-coated copper cables, as well as PVC telephone cases and other PVC-laden products at high temperatures [Christmann, 1989a]. A Swedish study described the creation of PCDD/Fs from the recycling of PVC coated wire in a copper smelter [Marklund, et. al., 1986]. In its investigation of a secondary copper smelter, EPA fed a secondary copper smelter a mixture of electronic telephone scrap and other plastic scrap (likely PVC coated), as well as metal scrap, residues, and slag [EPA, 1994a]. The estimated TEQ emission factor for this facility is 779ng/kg of scrap metal smelted [Versar, 1996]. The presence of large amounts of PVC along with copper -- a catalyst that increases the rate of dioxin formation -- makes these major dioxin sources. According to one German 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 imission measurements surprisingly high concentrations of dioxins and furans [Christmann, et. al., 1989a, b].

Removal of a part of the cable sheathings (typically PVC) before reclamation in a copper smelter resulted in reductions in PCDD/F values of 17%, 48% and 35% (in three different smelters) [Christmann, 1989b]. Other researchers [see Fiedler and Hutzinger, 1990] investigating this same plant found that there is little information on PCDD/F formation in copper recycling. However, after reduction of plastic inputs in this plant (presumed to be PVC, though not specified as such), the researchers noted that stack gas concentrations of PCDD/Fs fell from 200 to 70 ng TEQ/Nm<sup>3</sup>.

Secondary steel smelters are important dioxin sources in some inventories [Cohen and Commoner, 1995, Thomas and Spiro, 1995], although EPA did not make a quantitative estimate. EPA did note, however, "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]."

These facilities may produce dioxin in part because of the presence of PVC residues in steel scrap, particularly from automobiles, in which PVC may be used for upholstery, interior moldings, underseal, and other parts (EPA, 1994a identifies PVC as one of the chlorinated materials entering these processes). Chlorinated cutting oils provide significant amounts of chlorine for dioxin formation. An analysis [Aittola, 1993] of a metal reclamation facility in Finland with seven different processes for copper, aluminum, and various steels, found that a large part of the emissions of chlorinated aromatic compounds in these processes are formed due to PVC plastic and cables and car components. EPA estimates that between 500 and 850 pounds of auto "fluff" (plastic and other non-metal materials removed from cars destined for reclamation) is generated per car. For 1990 models about 20 pounds is PVC [Carroll, et. al.]. While much of the fluff can be removed by magnetic separation and landfilled or stockpiled, separation techniques are likely not 100 percent effective; at least some PVC is likely to reach the smelting operation. According to one experimental study of a Swedish steel recycler:

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].

Secondary lead smelters also release dioxin. According to a recent report for EPA, "Historically, the source of CDD/CDFs at secondary lead smelters is the PVC separator used in lead acid batteries. In 1990, about 1 percent of scrap batteries processed at lead smelters contained PVC separators....Less than 0.1 percent of scrap batteries contained PVC separators in 1994 and this trend is continued because no US manufacture of lead-acid automotive batteries currently uses PVC in production [Versar, 1996]." This change can be viewed as a model for dioxin prevention: dioxin releases were virtually eliminated by phasing-out PVC from the products that ultimately end up in combustion facilities.

Some inventories suggest that primary steel smelters may be major dioxin sources, as well [Lahl, 1993, Lahl, 1994, Brzuzy and Hites, 1996a]. In these facilities, dioxin may be produced due to a few chlorine donors: the use of chlorinated solvents and cutting oils in the production process, paints, and the reintroduction of dusts and slag containing chlorinated materials from recycling of scrap. PVC present as part of the scrap charges to steel mills may contribute to the emissions of chlororganics reported by Oberg and Allhammar, including dioxins [see Johnston, 1993 on Oberg and Allhammar, 1989]. Recycled scrap steel can play an important role in some types of primary steel production. One government study of electrical arc steel manufacture found the potential for the creation of toxic organic micropollutants because of feedstocks that contain organic materials such as oils, emissions, greases, and plastics; chlorinated compounds in these materials may give rise to PCDD/F formation [Air Board, 1995].



## **Other Combustion Sources**

Dioxin formation in the burning of natural wood is negligible. In contrast, industrial and residential wood burning can become an important dioxin source when wood with chemical additives or residues is burned. EPA identifies the industrial and municipal burning of wood as a large dioxin source [EPA, 1994a]. Since much of the wood incinerated is construction debris, and markets for PVC siding, window frames, wallpaper, and flooring are increasing rapidly, this source is likely to increase in the future. The major chlorine donors in this sector appear to be scrap wood with PVC residues, chip board with chloride-containing hardeners, and treated wood preserved with pentachlorophenol [Wilken, 1994, Vikelsee, 1993, Kolenda, et. al., 1994]. The German Federal Environmental Authority has urged that no chlorine-containing fuels be burned in any furnace, from private fireplaces to industrial plants [Wilken, 1994].

## **Accidental Fires**

PVC is now ubiquitous in modern buildings in flooring, siding, pipes, furniture, wallpaper, and other materials. Construction applications account for more than 60 percent of PVC use [SRI, 1993]. An average house contains 14 to 367 kilograms of PVC, depending on size and date of construction/remodeling [Carroll, 1995]. Industrial and institutional buildings contain even greater quantities. In Denmark alone, it is estimated that 1.2 million tons of PVC are used in existing buildings, which could burn in the case of accidental fires [DTI, 1995]. Combustion conditions associated with an accidental fire -- low oxygen and rapidly cooling temperatures away from the fire -- result in poor combustion conditions and high rates of dioxin formation [SEPA, 1996]. The Danish Technical Institute (1995) summarized:

"Rigid PVC does not burn by itself and will not start a fire. If other materials support a fire, PVC will burn under the formation of carbon oxides and hydrochloric acid fumes.... Burning PVC also yields large amounts of soot-containing smoke. The soot will contain several different substances. The presence of chlorine in the material gives the potential for contribution to the formation of polychlorinated dioxins and dibenzofurans during the fire. If formed, it seems that these substances have the highest tendency to be present in the soot. The amount will depend on the fire conditions such as oxygen available, temperature, catalyst available such as copper, and the amount of chlorinated material, eg PVC, involved in the fire....Laboratory tests with different types of PVC product report formation of PCDD/F in the level of microgram per kilo."

Samples of soot taken from fires in PVC-containing buildings have been found to contain dioxins in concentrations as high as 10,000 nanograms (ng) (TEQ)/per square meter (m<sup>2</sup>) on surfaces and 45 parts per billion (ppb) TEQ in ash and soot [Fiedler, 1993, UBA, 1992]. Fires in homes, schools and office buildings can produce dioxin concentrations in the 200 ng (TEQ)/sm<sup>2</sup> range [DTI, 1995]. Dioxins found in soot, however, represent only a small part of the problem: more than 90 of the dioxins produced in a structural fire are

found in the gaseous phase and escape into the atmosphere [Versar, 1996]. PVC fires may thus make a substantial but yet unquantified contribution to dioxin contamination of the environment.

According to one industry spokesman, as much as three-quarters of all PVC manufactured goes into the building and construction market for uses such as piping, siding, window frames, wallpaper, cabling, flooring, and other uses [Reisch, 1994]. As a result, any fire in a modern building is likely to be a source of dioxin. The contents of many buildings, including furniture, appliances, computer housings, toys, and other consumer products present in a typical home, are also potential sources of dioxin. Despite the PVC's potential contribution to the U.S. dioxin burden from accidental fires, EPA, in its draft dioxin reassessment, has failed to adequately account for the contribution of accidental fires to the nation's dioxin burden and in particular the contribution of PVC as a chlorine donor.

In the U.S. alone, there are 621,000 structural fires per year [Versar, 1996]. As noted in a recent report for the revision of U.S. EPA's dioxin reassessment, "PVC building materials and furnishings, textiles and paints containing chloroparaffins, and other chlorinated organic compound-containing materials appear to be the primary sources of the chlorine [Versar, 1996]." Based on the number of fires in Versar, 1996 and the Vinyl Institute's estimate of the amount of PVC in a typical house, Greenpeace estimates that 9,000 to 240,000 tons of PVC may burn in house fires each year.

Large, and potentially unknown, quantities of PVC may burn when accidental fires occur at plastics manufacturing, storage, or recycling sites. For instance, after a single fire at a plastics warehouse in Binghamton, New York, dioxin levels in soil on the site were found to be over 100 times greater than other samples from the same community. Dioxin levels in the soot were as high as 23 ng/m<sup>2</sup> TEQ, approximately the same as the state's maximum level for reentry into dioxin-contaminated buildings [Schechter, 1996]. A 1992 fire at Microplast, a PVC recycling company in Lengrich, Germany, resulted in dioxin contamination of residues in the facility and an 88-fold increase in dioxin concentrations in cabbages grown on farms downwind from the facility [UBA, 1992]. A recent study [Meharg, 1996] of three fires involving large quantities (approximately 1000 tonnes) of plastics (PVC and polyethylene) estimated that 50-100 tonnes of aromatic compounds (both chlorinated and non-chlorinated) were produced from each of the three fires investigated.

Substantial dioxin releases from the accidental burning of stockpiled waste containing PVC have also been documented. For example, several fires involving automobile fluff stockpiled at automobile reclamation facilities have occurred. In one fire, between 39,000 and 48,000 pounds of the fluff were burned. Laboratory studies by EPA indicate that burning one kilogram of fluff generates air emissions of approximately 0.0072g of PCDD/PCDFs. The Agency estimates that approximately 2 billion pounds of automobile fluff are generated annually, most of it ultimately disposed of in landfills. If all of this fluff were burned in open fires, more than 31 kg of these PCDD/PCDFs would be released into the air per year [Costner, et. al., 1995 citing Ryan and Lutes, 1993]. This

quantity of fluff is so large that it would contribute significantly to the national dioxin burden, even if burned in controlled combustors.

Landfill fires represent a largely unquantified source of dioxins. PVC may comprise between 50 and 75 percent of the total chlorine in landfills [Johnston, 1993]. Other chlorine sources may include organic household wastes, pesticides, and solvents. A large percentage of the PVC used in construction applications will reach the end of its useful life in the coming years, and overall use of PVC is increasing. With a significant portion of this PVC destined for landfills, landfill fires may become an important source of dioxin emissions.

Accidental burning of PVC in automobiles (e.g., dash boards, underseal, upholstery) could represent another important, yet unquantified, dioxin source. According to a report for the U.S. EPA, "Accidental fires in vehicles are uncontrolled combustion processes that typically result in relative high emissions of incomplete combustion products, including CDD/CDFs, because of poor combustion conditions. PVC vehicle components and other chlorinated organic compound-containing materials appear to be the primary sources of the chlorine. In 1993, approximately 421,000 [vehicle] fires were reported in the US [Versar, 1996]." Dioxins and furans have been identified in residues from the experimental burning of automobiles (0.044 - 0.052 mg TEQ/car), a subway car (2.6 mg TEQ), and a railway coach (10.3 mg TEQ) [Versar, 1996]. Given the number of automobile fires per year and the amount of dioxin generation per vehicle burnt, vehicle fires could thus represent an important, and still unknown source of dioxin emissions.

Due to the large amounts of dioxin that can be produced in accidental fires, the German Environment Ministry has called for the use of substitutes for PVC in all areas susceptible to fires:

"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 Environmental 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 labeling of plastics containing chlorine and if necessary a ban on the use of PVC in packaging [UBA, 1992]."

## **Open Burning of Wastes**

In dioxin inventories for industrialized nations, open burning is not considered an important dioxin source. In some nations, especially developing nations, the greater prevalence of this practice and the poor combustion conditions encountered may make open burning of chlorine-containing wastes an important dioxin source. Open burning of trash or construction waste that contains PVC may result in significant but unquantified dioxin emissions.

## **Manufacture of PVC Feedstocks**

In addition to the significant dioxin formation associated with PVC during disposal and accidental combustion, dioxin is also generated during the manufacture of the product and its feedstocks. Since at least 1983, the chemical industry has known that dioxin is formed during the manufacture of the PVC feedstocks vinyl chloride (VCM) and 1,2-dichloroethane (EDC) [Beekwilder, 1989, cited in Andersen and Knapp, 1993]. Despite the industry's prior knowledge, it was not until 1989 that a series of sampling programs in Europe brought the problem to the attention of the scientific community, governments, and the public at large. While only a portion of the dioxin generated during PVC production is released into the environment (some of the dioxins are recycled in internal processes or destroyed during incineration), PVC production represents a potentially large dioxin source.

## **Dioxin in Wastes**

Dioxin formation is particularly significant in the oxychlorination process, in which ethylene is combined with hydrochloric acid and oxygen in the presence of a copper catalyst to produce EDC. Dioxins produced in this process follow one of three paths: the majority remains with the EDC product, and smaller amounts are distributed to the wastewater and offgases from the process [DTI, 1995]. Dioxins contained in the latter two fractions enter pollution control devices and are then released to the environment via air emissions, wastewater discharges, ash residues, and treatment sludges. The EDC product fraction is then purified, and much of the dioxin is partitioned into the "heavy ends," wastes that are non or semi-volatile. These wastes are primarily disposed of by incineration -- a process that produces and releases dioxins and furans from the incineration of the many other chlorinated organic compounds in the wastes [Costner, et. al., 1995]. Some of these wastes, however, may be shipped offsite for treatment or disposal.

The quantities of dioxin formed in wastes appear to be very large. \* Laboratory simulations at the University of Amsterdam demonstrated dioxin formation during oxychlorination at a rate equivalent to 419 grams of dioxin (TEQ) per 100,000 tons of EDC produced (4.2ng/g EDC) [Evers, 1989]. A 1994 analysis at a fully modernized EDC/VCM plant in Germany found dioxins in process sludges at concentrations as high as 414 ppb [Lower Saxony, 1994], refuting the claim that only outdated EDC/VCM technologies produce dioxin. An analysis made by ICI Chemicals and Polymers at its

vinyl chloride monomer production plant in Runcorn, UK, found that more than 27g TEQ of dioxins are produced in solid and liquid wastes each year (per 200,000 tons) [ICI, 1994, reproduced in Environment Agency, 1997]. If dioxins created during the integrated production of perchlorethylene and trichlorethylene (produced using by-products of the VCM production) are included, this number increases to more than 500g TEQ per year, much of which is currently deposited in off-site salt caverns.\*\* Admitting the inevitability of producing dioxins in VCM production, ICI notes that, "It is difficult to see how any of these [process] conditions could be modified so as to prevent PCDD/PCDF formation without seriously impairing the reaction for which the process is designed [ICI, 1994]."

In the U.S., dioxins, furans and PCBs have been identified in oxychlorination wastes from the Vulcan Chemical plant in Louisiana at concentrations as high as 6000 ppb TEQ. [Costner, et. al., 1995]. These levels suggest that oxychlorination wastes are among the most dioxin-contaminated wastes ever identified, with concentrations in the same range as wastes from the production of Agent Orange, an notorious herbicide used for defoliation during the Vietnam War.

Production wastes from EDC synthesis also contain PCBs. In 1990, Dow Chemical found that "heavy end" wastes from EDC distillation at its Louisiana facility contained PCBs at concentrations up to 302 parts per million -- well over the legal limit [Dow, 1990]. In fact, EPA had proposed to regulate PCB formation from EDC synthesis as early as 1988, but withdrew its proposal under pressure from the Vinyl Institute and numerous EDC-producing corporations [Costner, 1995].

The fact that dioxins are formed and partitioned into wastes does not mean that they will be released into the environment. Some dioxins are retained in cycling streams in the processes; some may be destroyed in incineration. But at least some portion of the dioxins created will be released to the environment through incomplete combustion or accidental releases; indeed in its sampling of wastes at the Vulcan Chemical plant and other EDC/VCM facilities, Greenpeace investigators found wastes in barrels and other readily accessible storage containers [Costner, et. al., 1995]. In addition, the chlorine-rich wastes created in the EDC/VCM production process contain many other organochlorines, which, when incinerated, can create and release dioxins into the environment as products of incomplete combustion. The quantity of dioxin generated as products of incomplete combustion may, in fact, be far greater than that in the original waste that escapes combustion [Costner, et. al., 1995].

In addition to incinerator air emissions, dioxin-contaminated fly ash and scrubber water from on and off-site incineration facilities must be treated and disposed of. Fly ash and scrubber water may account for up to 88 percent of dioxins created by the incinerator process [Huang and Beuknes, 1995]. Ashes and waste water treatment sludges are typically land-disposed, while effluents are ultimately discharged to waterways. When landfilled, the dioxins in these wastes can threaten groundwater.

Wastes from EDC manufacture is of special concern in developing countries, where incineration technologies are primitive and inadequate landfilling could occur. In the end, the wastes associated with the production of PVC should be considered dioxin-contaminated wastes that pose special threats to air, water, and land.

### **Releases to Air and Water**

Dioxins, furans, PCBs, hexachlorobenzene, and other dioxin-like compounds have been identified in the air emissions and effluent discharges (and wastewater treatment sludges) from several EDC/VCM plants [ICI, 1994, SFT, 1993, Johnston, 1994]. These wastewater dioxin concentrations are of particular interest in relationship to dioxin in sediments. For example, in one study, up to 80 percent of the dioxin in sediment samples from the Rhine River (Netherlands) were attributed to an upstream EDC/VCM facility [Evers, et. al., 1988, Evers, 1989]. In an update to their study [Evers and Olie, 1996], these same researchers linked decreased dioxin levels in sediments to the halting of production of the pesticide 2,4,5-T, an example of a materials policy applied to dioxin; however, they found that "the congener patterns of PCDD and PCDF in Rhine sediments revealed that the production of vinyl chloride monomer is still an important source of especially OCDF and other higher chlorinated congeners."

Another research effort identified dioxin concentrations ranging from 433 to 922 ppt TEQ in sediments from a harbor on which an EDC/VCM facility is sited [Evers, et. al., 1989b, Wenning, et. al., 1992]. Studies in the Netherlands, Sweden, and Germany have all found elevated levels of dioxins in sediments, water, and biota downstream from EDC/VCM discharge points [Evers, 1988, Evers, 1993, Cato, 1992, Costner, et. al., 1995, Lower Saxony, 1994, Verhoog, 1988, as cited in Andersen and Knapp, 1994].\*\*\* In certain areas of the North Sea and Baltic, EDC/VCM synthesis appears to be a primary source of environmental dioxin contamination. [Evers, 1989, Evers, 1988]. High concentrations of dioxins have been identified in shellfish and sediments in the Venice lagoon in the vicinity of Enichem's VCM/EDC/PVC production facility [Fabbri, 1996]. Greenpeace found total dioxin concentrations of more than 2,900 parts per trillion (15.4ppt TEQ) in sediments taken slightly downstream from the discharge point of the Geon Corporation in LaPorte, Texas [Costner, et. al., 1995]. This concentration is approximately four times higher than the average concentration reported for North American sediments in EPAs draft dioxin reassessment.

A National Oceanic and Atmospheric Administration (NOAA) report found high levels of hexachlorobenzene, PCB's, hexachlorobutadiene – all contaminants that tend to be formed as byproducts along with dioxin -- in the sediments and water of estuarial bayous surrounding the PPG and Condea Vista PVC facilities in the Lake Charles, Louisiana area. Fish in these waters are also highly contaminated [NOAA, 1996]. These two firms, along with Conoco, Inc. appeared to make major contributions to the area's contamination burden.

## **Dioxins in PVC Products**

Dioxins have also been found in the PVC product itself. In May 1994, the Swedish Environmental Protection Agency found that PVC plastic 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. Total concentrations, including PCBs, ranged from 0.86 to 8.69 ppt (TEQ). In 1995, the UK Ministry of Agriculture, Fisheries, and Food (MAFF) noted the presence of low levels of PCDDs and PCDFs in PVC articles manufactured for use either as food packaging or food processing equipment [Wagenaar, et al, 1996]. In 1992, BF Goodrich (now Geon) submitted information to U.S. EPA indicating that dioxins had been found in concentrations up to 170 parts per trillion (total PCDD/F) in resin and pipes made from "post-chlorinated PVC" (CPVC), an engineering thermoplastic that Goodrich manufactured and sold at the time and has since discontinued [Mattia, 1992, MRI, 1991]. Dioxins were also found in the workplace air in the extrusion of this product [Mattia, 1992]. Since then, the plastics industry in Europe and the U.S. Vinyl Institute have undertaken studies to quantify dioxin levels in PVC resins and products [Wagenaar, et. al., 1996, Carroll, et. al., 1996]. Both studies identified trace levels of dioxin contamination (mainly OCDFs) in some samples. The industry suggests that these levels are insignificant and that the results of the Swedish and British government studies are due to contamination. But the evidence clearly warrants a comprehensive, independent investigation to characterize the presence of dioxins in PVC resins and products.

## **Estimates of Total Dioxin Formation**

Given the paucity of plant-specific data, it is not possible to estimate with confidence the total quantities of dioxin produced by production processes for PVC feedstocks. As a preliminary estimate, we can extrapolate from the analysis of ICI's Runcorn Plant, which found dioxin production of 27 g TEQ per 200,000 metric tonnes of EDC produced [Environment Agency, 1997]. Based on annual U.S. production of 11 million metric tonnes of EDC [VI, 1996], U.S. EDC/VCM plants would create an estimated 1485 g TEQ dioxin per year, assuming similar rates of dioxin generation. At least some portion of this dioxin would be released to air, water, and land.

The vinyl industry has argued that European data can not be applied to U.S. plants. The industry has not supplied data on dioxin contamination of wastes from its facilities in this country, however, to fill the gap. In the meantime, there is no reason to suppose that dioxin formation will be substantially different from similar facilities in Europe. Indeed, the U.S. Vinyl Institute has confirmed the presence of low concentrations of dioxin in waste water samples from four U.S. plants that manufacture EDC/VCM [Vinyl Institute, 1996]. As previously noted, samples from U.S. EDC/VCM plants obtained by Greenpeace and analyzed at an independent laboratory contained extremely high levels of dioxin [Costner, et. al., 1995]. More recent samples of "light end" wastes from Borden's facility in Louisiana contained 310 ppt TEQ, while samples of on-site remediation wastes at the PPG facility in Lake Charles, Louisiana -- from an old area for the dumping of tars -- contained dioxins and furans at the extremely high level of 76,239 ppt [Santillo, 1996].

As a result, there can be no doubt that substantial quantities of dioxins can be produced in PVC production in Europe and in the U.S.

### **Dioxins in Chlorine Production**

Even at the very root of the PVC lifecycle, dioxins can be produced. More than 30 percent of the chlorine produced in the world is directed into PVC [SRI, 1993], so that PVC again is associated with more dioxins produced in the chlor-alkali process than any other product. In this process, chlorine gas is produced by passing a powerful electric current through a solution of salt water. Carbon-containing materials are present in these reactions as trace contaminants, as plastics in plant equipment, or as components of electrodes. During and after the chlor-alkali reaction, chlorine combines with organic material to produce dioxins and other contaminants. Very high concentrations of dioxins (up to 650 ppb total), along with hexachlorobenzene, octachlorostyrene, and hexachloroethane, have been also found in the sludges from spent graphite electrodes used in this process [Rappe, 1991]. These results were found in plants using graphite electrodes.

Although all chlorine plants in North America and many in Europe have replaced graphite electrodes in recent years with titanium substitutes, questions arise as to dioxin formation even at the most modern chlor-alkali plants. Researchers [Strandell, et. al., 1994 as reported in Versar, 1996] have reported that metal electrode sludge from a facility in Sweden contained high levels of PCDFs that might have been formed by the chlorination of polycyclic aromatic hydrocarbons present in the rubber linings of the electrolytic cell. This research raises questions as to the extent to which modern chlorine production processes create dioxins.

### **Unanswered Questions**

An accurate quantification of the total contribution of the PVC lifecycle to the global dioxin burden is not possible at this time due a lack of important information. Data gaps include site-specific emissions to all media and waste streams from EDC/VCM/PVC plants; dioxin releases associated with accidents, near-misses, pressure releases and other production upsets at EDC/VCM/PVC production facilities (which may occur with regularity); amounts of PVC on metals destined for recycling; amounts of PVC involved in accidental fires (structural and transportation); and amounts of PVC otherwise burned in landfills and other combustion processes.

Despite these uncertainties, there is no doubt that PVC is the cause of major dioxin releases from medical waste and municipal waste incineration, and appears to be an important chlorine donor in accidental fires and a number of metals recycling processes. Dioxin generation in many other aspects of the PVC lifecycle appears to be significant, but remains to be quantified. For example, assessment of the total dioxin generation from PVC manufacture complicated by the use of different production technologies and the co-production of chlorinated solvents [Stringer, et. al., 1996]. Important questions remain unanswered concerning the full extent of dioxin generation caused by the PVC lifecycle.



Gathering the required objective information is a task that should be pursued aggressively by independent parties, not delegated to the industry itself.

### **Case Study 2: The PVC Industry's Voluntary Dioxin Study: Science or Public Relations**

A 1994 "Crisis Management Plan for the Dioxin Reassessment" prepared for the Vinyl Institute (VI) by a public relations firm precisely forecasts the actions the industry should take -- and has taken -- since the release of EPA's dioxin reassessment. The document instructs the industry how to portray scientific and technical information "to avoid deselection of PVC by major customers and to prevent punitive regulation of PVC by EPA, Congress, or the state legislatures [Burnett, 1994]."

According to the document, the Vinyl Institute initiated the strategy just before EPA released its dioxin reassessment, because "EPA will likely conclude that the incineration of chlorinated compounds is the single largest known contributor of dioxin.... We believe that PVC will be specifically mentioned, and potentially slated for further regulation." The strategy advises industry representatives on how to respond in the media, and it suggests that the industry enter into joint scientific activities with EPA in order to avoid future regulation.

According to the strategy:

"The short-term objective of the plan is to mitigate the effects of potential negative press coverage by positioning the vinyl industry as a proactive and cooperative entity, working in tandem with EPA to characterize and minimize sources of dioxin.... The vinyl industry must actively and aggressively communicate with the media its commitment to working with EPA to characterize and minimize any dioxin in the PVC lifecycle while at the same time asserting that based on reliable data available to date, the industry believes its contribution is minimal. Cooperative positioning is the key element; solely asserting that PVC is a minor contributor to the problem will only serve to misposition the industry as uncaring and unresponsive in the minds of target audiences.... The most effective message VI can deliver to respond to Greenpeace and leverage its own position in the dioxin debate is the following: The vinyl industry supports EPA's objective of identifying and reducing dioxin emissions and is working with EPA to determine the best means of achieving that goal [Burnett, 1994]."

Soon after the strategy was written, EPA and the Vinyl Institute announced a plan by which the industry would cooperate with EPA's Dioxin Reassessment by "self-characterizing" dioxin emissions from PVC production. This industry program is now the only source of new data on dioxin/PVC links that enters the dioxin reassessment process. The industry collects samples from its own plants, analyzes their dioxin content, interprets the data, and submits it to EPA. While the process and results are overseen by a

"peer review" panel, the industry ultimately can choose where, when, and how samples will be taken and analyzed, and which data are suitable for submission. Because information about the samples -- including the facility from which they were taken -- is confidential, there is no way for the review committee, EPA or the public to know whether sampling times or locations accurately represent typical or worst-case dioxin releases or have been specifically selected to provide favorable data. Nor is it possible to otherwise evaluate, confirm, or act on the information.

The VI self-characterization will not look at releases of dioxins throughout the entire PVC lifecycle but will consider only dioxin releases during PVC production. The program only tests certain potential dioxin discharge routes without evaluating the total generation of dioxins at these facilities [Vinyl Institute 1996]. The sampling program will not examine many materials that are potentially important sources of dioxin transfer and release, including recirculating materials in the manufacturing process, products transferred to other facilities in the EDC/VCM/PVC manufacturing chain, and wastes transferred to regulated hazardous waste treatment, storage and disposal facilities. Of particular concern is the failure to evaluate highly-contaminated production wastes; the industry omits these wastes from its program because they do not leave the "system boundary" of the facility, although their incineration, land disposal or accidental release will likely result in their escape across this imaginary boundary. Finally, the program will not address the significant dioxin emissions associated with the disposal of PVC products.

Thus far, the industry has chosen to submit information only on those aspects of the PVC production that are known to contain small amounts of dioxin (wastewater and resins) [Carroll, 1996]. By the time EPA finishes its reassessment, no data are expected to have been submitted on those aspects of production that could cause major dioxin emissions. Data on wastewater treatment sludges and on incinerator stack gases are not expected until mid-1997.\*\*\*\* No data from Phase 2 of the industry's characterization program, including vinyl chloride monomer, spent catalyst, and chlorinated solvent co-products will be available for some time to come. Meanwhile, the industry continues to grow rapidly, building new production facilities and expanding sales, exports, and production capacity.

The VI's testing program to date has demonstrated numerous limitations. First, the peer review committee was not brought into the program until it was already in progress, with most of the Phase 1 sampling complete. The committee thus had no meaningful opportunity to evaluate the design of the sampling program or to audit its performance. Still, the committee has noted several limitations in the study including the voluntary participation of VI member companies, possible undocumented differences in field sampling protocols by participating companies, the relatively low number of wastewater treatment systems sampled, lack of documentation on fugitive releases of EDC prior to purification, and the fact that the study has been designed to characterize normal process operations and not upsets or accidents, which occur with regularity [Vinyl Institute, 1996]. Additionally, there is no attempt in the sampling program to conduct single-plant, multi-media studies (total dioxin emissions from a single plant).

It is reasonable for any industry with potential impacts on public health and the environment to be subject to independent evaluation, not "self-characterization." The vinyl industry in particular has demonstrated a need for oversight. As early as 1949, the industry knew that worker exposure to vinyl chloride monomer, a known human carcinogen, could cause liver damage in humans. Even with information from additional studies in the 1950s and 1960s, the industry failed to take action to protect worker and public health from VCM exposure. Only when studies revealed an association with rare liver cancer deaths in the early 1970s did the industry admit the hazards of vinyl chloride. The U.S. Second Circuit Court noted, "Indeed, the record shows what can only be described as a course of continued procrastination on the part of the industry to protect the lives of its employees." When the Occupational Safety and Health Administration (OSHA) proposed a lower occupational health standard for VCM exposure, the industry took OSHA to court, arguing that the new limit would devastate the industry. But when the court upheld OSHA's exposure limit, the industry was able to implement new technologies and reduce exposure levels within 1 year while expanding its operations [Ashford and Caldart, 1991].

## Notes

\* Researchers have noted that dioxins contained in EDC wastes are generally dominated by the octa-chlorinated dibenzofurans (OCDFs). These octa-chlorinated dioxins and furans receive relatively low weighting in TEQ schemes [Johnston and Troendle, 1993]. Thus, the mass balance of total dioxins in EDC wastes (and emissions from these plants) is much larger.

\*\* The integrated process appears to be used by some facilities in the United States. This integrated process could lead to the generation of wastes with even higher dioxin levels.

\*\*\* In some of these studies other production facilities may have existed in the area or production of other chlorinated chemicals may have occurred at the same facilities.

\*\*\*\* While these results may be submitted to EPA before the dioxin reassessment is released, they are not likely to be incorporated into the final document.

## Chapter 4 – Phasing Out PVC

### A PVC Sunset to Prevent Dioxin

PVC contributes to dioxin generation at numerous points throughout its lifecycle, and appears to cause more dioxin formation than any other single material. Reducing the production and use of PVC is a simple and effective avenue to prevent PVC-related dioxin pollution. By replacing PVC with alternative, chlorine-free materials, dioxin formation associated with PVC can be eliminated entirely. Given the importance of the PVC lifecycle in the nation's dioxin burden, a PVC phase-out must be a top priority in any dioxin prevention strategy.

If we do not address PVC and other dioxin-generating materials, the only alternative is to attempt better management of dioxin sources. But these approaches are inherently inadequate to protect health and the environment from dioxin. They are also very costly. For instance, EPA has recently proposed new regulations for dioxin emissions for medical waste incinerators. These rules would require MWIs to install new pollution control devices, which would, according to EPA, more than double the per-ton cost of medical waste disposal at existing incinerators, and increase the annual national bill for medical waste incineration to \$754 million from the current \$340 million [EPA, 1995]. Even with the new devices, medical waste incinerators will remain an important source of dioxins to the air, and contamination of ash will increase substantially. In contrast, preventing PVC from entering the medical waste stream would almost eliminate dioxin releases from MWIs to all environmental media.

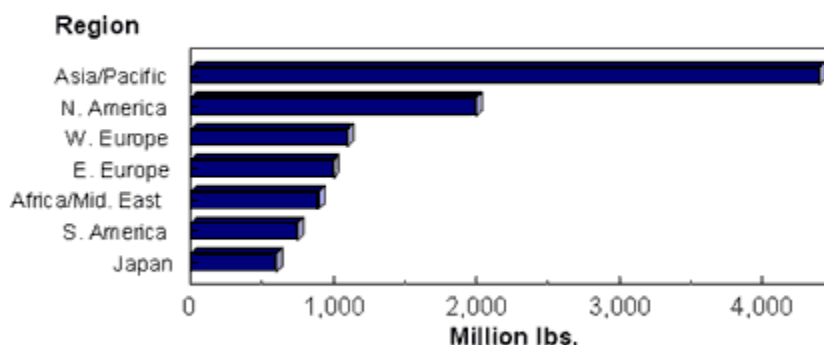
This picture is typical. No technological upgrade can ever eliminate the generation of dioxin. In the most sophisticated incinerator, for example, products of incomplete combustion will always be formed [EPA, 1989]. So long as chlorinated materials are burned in incinerators, by-products of combustion will always include dioxin. Since the global ecosystem has zero assimilative capacity for persistent pollutants, mere reductions of dioxins through pollution management is clearly inadequate.

In developing countries, pollution control and management strategies are particularly inappropriate. A significant amount of the PVC and PVC feedstocks produced in the U.S. are exported to developing nations, where the use of PVC plastic is growing at the rate of 7% to 9% per year [Waltermire, 1996] (see Figure 2). State-of-the-art incineration technology and pollution control devices require very large capital investments to install; further, they require fiscally and technically demanding operation and maintenance regimes. It is not practical to expect that all incinerators in all nations of the world will be outfitted and operated at such expense. To assume that all the PVC in the world can be directed to such optimally operated state-of-the-art incinerators is, thus, far from realistic. Further, pollution control devices cannot address dioxin releases from certain points of the PVC lifecycle, such as accidental fires or open burning.

FIGURE 2

## World Regional PVC Growth

Expected PVC Demand Increase, 1994-1998



Source: Waltermire, 1996

Note: Average annual growth rate highest in developing countries

(e.g., Eastern Europe 11%, Africa/Mid. East 9%, Asia/Pacific 9%, vs. Europe 2%)

It is far more effective to avoid the production and dispersal of PVC and other chlorinated materials which inevitably lead to dioxin generation in the first place. This strategy is appropriate for both industrialized and developing nations alike. Restricting the production and use of PVC and encouraging the use of alternative materials will prevent PVC from entering incinerators, smelters, and accidental and deliberate fires. It will also prevent dioxin generation that would have occurred during the manufacture of these products and their feedstocks.

Because some uses of PVC are more easily replaced by alternatives than others, a PVC phase-out must begin by setting priorities. PVC used in disposable products that are likely to be incinerated (e.g., IV bags, packaging), or products for which there are readily available drop-in replacements (e.g., pipes) should be phased-out on relatively short timelines. Meanwhile, research and development should continue on the few uses for which alternatives are not yet on the market, such as some medical device applications.

### Alternatives to PVC

Alternatives are available now for the vast majority of all uses of PVC, and the most appropriate alternative is unique to each PVC application. PVC use patterns are presented in Table 7. Construction applications, such as pipes, fittings, sidings, and window profiles, account for over 50 percent of PVC consumption. Other PVC uses include furniture, wall and floor coverings, automobiles, electronic equipment, wire and cable coatings, packaging, and medical devices. In industrialized nations, packaging is a relatively minor PVC use, but it plays a more important role in nations with developing or transitional economies where consumerism is on the increase [Endo, 1991].

**TABLE 7: World PVC Consumption by Type (1990) - in thousands of metric tons**

	U.S. & Canada	Latin America	Japan	Asia (rest)	Western Europe	Oceania	Eastern Europe	Middle East	Africa
<u>Flexible PVC</u>									
Film and sheet	169	84	382	571	424	16			
Floorings	191	30	97	66	43	7			
Synthetic leather	80	53	83	30	175	7			
Wire and cable cover	222	42	246	202	436	17			
Other	527	58	167	273	517	13			
<u>Rigid PVC</u>									
Blow molding (bottles, etc.)	110	72	524	17					
Film and sheet	77	31	258	195	601				
Pipe and fittings	1585	212	692	612	1450	100			
Siding/window profiles	481	12	487						
Other	509	32	156	155	266	13			
Unidentified	725	130	1848	258	244				
Total	3951	626	2081	2830	5254	190	1848	258	244
Percent of World Consumption	22.9%	3.6%	12.0%	16.4%	30.4%	1.1%	10.7%	1.5%	1.4%

Source: SRI, 1993. Blank cells indicate no data.

Note: Overall Building and Construction accounts for the greatest percentage of PVC use (60 percent or more). Consumer uses, packaging, electrical and electronic, transportation, furniture, and others account for much smaller, yet often significant, percentages of PVC use.

The most appropriate substitute depends upon the qualities required for each PVC application. A detailed discussion of every PVC use and its alternative is beyond the scope of this document. Nevertheless, existing information indicates that safer, effective substitutes are available now for all major PVC uses. These include "traditional materials" --wood, metal, paper, glass, ceramics, etc. -- and sometimes chlorine-free plastics, in particular polyethylene and polypropylene. Preliminary information on alternatives for a number of PVC applications is presented in Table 8.

A study by the consulting firm Prognos AG for the state of Hessen, Germany, found that 95 percent of all PVC uses could be easily substituted using available alternatives. Prognos found further that while some alternatives were slightly more expensive, a large percentage of PVC uses could be substituted cost-effectively (no or little net increase in costs) and with a potential increase in employment of four percent [Plimke, 1994]. The Enquete Commission of the German Bundestag also concluded that alternatives were available for the vast majority of PVC uses:

"Except for special products with particular characteristic requirements, such as blood packs or electrically conducting flooring for clinically clean rooms, it is safe to assume that there are materials able to replace PVC in all its uses, these in many cases being marketed by the same manufacturers as those producing PVC [Enquete-Kommission, 1994]."

**TABLE 8: Alternatives to Selected PVC Uses**

<b>PVC use</b>	<b>Alternative Material</b>
Window profiles	Wood, aluminum
Pipes	Concrete, steel, galvanized iron, copper, clay, chlorine-free plastics, including high-density polyethylene (PE), polypropylene (PP), acrylonitrile-butadiene-styrene (ABS), and polyisobutylene.
Flooring	Linoleum, wood, stone, rubber, PE and PP.
Cable coatings	PE, ethylene-vinylacetate copolymer (EVA); polyamide, silicone, and other thermoplastic elastomers.
Packaging	No packaging at all, glass, paper and cardboard, PP, PE, and polyethylene terephthalate (PET).
Wall coverings	Paint, tiles, paper-based wallpaper.
Roof-sheeting	Synthetic rubber, polyolefin sheeting, traditional materials made from tar, wood, and other materials.***
Gutters	Galvanized iron.
Shutters and blinds	Wood, aluminum, and chlorine-free plastics.
Furniture	Wood, metal, textiles, leather, and chlorine-free plastics such as butadiene-polyamide copolymer.
Office supplies	Metal, wood, PP, PE.
Automobiles	Metal, textiles, chlorine-free plastics, including polyolefins and ABS.
Medical uses	Glass, latex, chlorine-free plastics including PP, PE, PET, EVA, polybutylene terephthalate, block copolymers, and silicones.

Sources: Belazzi 1993, Enquete Kommission 1994, CRA 1993, Wagener 1993.

### **Progress Towards a PVC Phase-Out**

A large number of communities, manufacturers, and hospitals have begun or completed successful efforts to eliminate PVC from their product lines or facilities. For instance, over 200 communities in Europe -- including major cities in Austria, the Netherlands, Germany, Sweden, Luxembourg, Denmark, and Norway -- have policies to restrict or avoid the use of PVC in public construction projects; many have successfully built major new buildings without PVC. In transportation, the Euro-Tunnel, and subway systems in London, Vienna, Bilbao, Dusseldorf, and Berlin are PVC-free. The British, German, and U.S. Navies do not employ PVC for ship-board uses. The Olympic stadium and other parts of the Sydney 2000 Olympic village are being built with a commitment to avoid or minimize the use of PVC.

In industry, there are numerous examples of manufacturers that have successfully replaced PVC in their product lines. Volkswagen has stopped using PVC in its vehicles, and Mercedes, BMW and Opel have adopted similar policies. Sony-Europe, AEG, Ikea furniture, Herlitz, and Tarkett, as well as numerous retailers throughout the world, have adopted PVC phase-out policies for their lines of appliances, furniture, office equipment, flooring, and product packaging.

Major governments and advisory bodies have also begun to call for a phase-out of PVC. The Swedish Parliament has called for a phase-out of specific PVC uses [Porter, 1996]. The Danish government has instituted a goal of phasing out important PVC additives due

to their numerous negative environmental properties. The government plans to promote PVC-free construction in all public building bids, and to institute policies against PVC incineration. The Spanish Senate has asked the government to replace the use of PVC in packaging with non-polluting substitutes, and to study the possibilities of a total PVC phase-out in Spain [Greenpeace, 1996]. The International Joint Commission on the Great Lakes has called for a sunset policy to eliminate the use of chlorine and chlorinated organic compounds in industry [IJC, 1991, 1993]. In 1993, the American Public Health Association (APHA) made a similar recommendation. In 1996, the American Public Health Association noted that PVC was the major source of dioxin emissions from medical waste incinerators, and resolved that effective chlorine-free products should be substituted for PVC in medical uses [APHA, 1996].

Despite this progress in some industrialized nations, the PVC industry is rapidly expanding its markets in developing countries, particularly in Asia and Latin America [Waltermire, 1996]. As markets for vinyl plastic become saturated in wealthy countries, the industry is relying for its growth on exports of PVC, EDC, and VCM from the U.S. to developing nations -- as well as on the construction of new production facilities in these countries [Endo, 1991, Christiansen, 1995] (see Case Study 3). As a result, dioxin and other pollution problems associated with the PVC lifecycle are transferred to developing nations. It is particularly unlikely that pollution control strategies -- the construction and maintenance of "state-of-the-art" incinerators and adequate landfill capabilities, for instance -- will be effective in these nations, especially when they are of questionable effectiveness in the developed countries. Thus, addressing the production of PVC becomes particularly important.

### **Socioeconomic Implications**

A PVC phase-out must include measures to address the socioeconomic impacts of the transition away from PVC. Although effective alternatives are available now for all major PVC uses, many workers and communities involved in the production of PVC and its feedstocks would be affected by a shift to alternative materials.

Phasing out PVC will affect different sectors of the PVC industry in different ways. At later stages of the PVC lifecycle, economic dislocation would likely be less significant than at earlier stages. Compared to the chemical companies at the base of the PVC lifecycle, PVC formulators and manufacturers of PVC-containing products are smaller and more numerous. These companies will have the most promising opportunities to switch to alternative materials. A flooring manufacturer, for example, may be able phase-out PVC and phase-in alternative materials such as linoleum and polyolefins; workers at existing plants would continue to make these reformulated flooring products. The most significant impacts of a PVC phase-down would likely be at the earliest stages of the PVC chain, where economic activity is most concentrated, highly capitalized, and technology-intensive. Facilities that produce chlorine, EDC, VCM, or PVC polymer are highly reliant on sales of PVC. For such facilities, significant economic disruption would occur as PVC is phased out. Phasing out PVC, however, would likely result in some growth in the manufacture of alternative materials, such as chlorine-free plastics.



### **Case Study 3: The Global PVC Industry**

The PVC industry is concentrated in industrialized nations. In 1990, 76 percent of the world's PVC was produced in Europe, the U.S., Canada, Japan, and Australia, and 77 percent of the world's PVC was consumed in these countries, as well. Production of the feedstocks for PVC -- ethylene dichloride and vinyl chloride monomer -- showed an even more extreme pattern: these nations produced 85 and 79 percent of the world's total production of these chemicals, respectively [SRI, 1993].

In recent years, however, the Northern chemical industry has targeted developing countries for its growth, likely because the Northern market for PVC is approaching saturation and these countries represent growing consumer societies. Through the 1990s, consumption of PVC in Japan, Europe, and North America is expected to grow by only 2, 2.8 and 3.4 percent per year. In Latin America and Asia, however, PVC consumption will grow at annual rates of 6 to 7 percent per year, doubling by the end of the decade [Roberts, 1994]. According to the industry, PVC demand correlated closely with economic development. People in industrialized countries thus presently consume 6 to 18 times more PVC per capita than developing countries [Waltermire, 1996]. According to one PVC industry representative, continued demand growth for PVC depends to a great extent on markets in developing nations:

"Demand for PVC in the high-population developing countries will grow rapidly after their GNP per capita reaches \$500 per year. On the other hand, in the world's major industrialized countries where per capita GNP is over \$10,000/year, the use of PVC has come close to its maturity and the growth rate of PVC may not be as much as the GNP growth rate. The concern over the disposal of waste material is one of the reasons for advanced society to refrain from excessive use of plastics [Endo, 1991]."

To create and meet growing demand for PVC, the chemical industry is aggressively expanding both its product exports to developing countries and its construction of new PVC factories in these nations. In 1992, twenty-one new PVC production facilities were slated for construction in Asia, Latin America, and the Middle East, compared to one each in Japan and Western Europe, three in Eastern Europe and none in North America [ECN, 1992]. While PVC production rates in Japan, Europe, and North America are expected to grow at only about 2 to 3 percent per year through the 1990s, PVC production growth rates are predicted at 6.5 percent per year in Southeast Asia, 8.1 percent in the Middle East, 14.1 percent in India and Pakistan, and 5.7 percent per year in Latin America [Endo, 1990].

Exports of PVC and its feedstocks EDC and VCM to nations with developing economies are also significant. Trade statistics [SRI, 1993] show that the U.S. and Canada account for 67 percent of the world's EDC exports, and Asia is the world's largest importer. The U.S. is also the world's biggest exporter of VCM and PVC, with Latin America and Asia accounting for the bulk of the imports. Exports of PVC and precursors from the U.S. and

Europe to developing countries are increasing rapidly, and these exports are considered a critical part of the growth strategy for the Northern chlorine industry [Christianssen, 1995].

## **Recommendations**

### **A PVC Sunset to Prevent Dioxin**

The health risk posed by dioxin calls for immediate action to reduce and ultimately eliminate the production and use of PVC. PVC is the single largest use of elemental chlorine and its production is expanding. It is also known that dioxin is generated as a byproduct during PVC production, use, or treatment for disposal. There are strong grounds for holding PVC responsible for a substantial and growing proportion of global dioxin production and release. Other chlorinated materials (pesticides, solvents, pulp and paper bleaches, for example) are also responsible for significant dioxin generation, and these too must be addressed as part of a larger strategy to eliminate dioxin. Greenpeace recommends the following steps towards a materials-based dioxin prevention strategy for PVC:

#### **1. Prevent PVC-related Dioxin Pollution**

Because of PVC's important role in the formation and release of dioxin, and its other numerous adverse environmental and health impacts, EPA should announce a program to sunset the production and use of PVC. The stated intent of this program should be to progressively reduce the production and use of PVC in the U.S. to zero. Priority should be given to those use sectors that cause the most dioxin formation during their lifecycle or are most easily replaced with safer, chlorine-free substitutes. Longer timelines for phase-out are appropriate for the small percentage of uses for which alternatives are not yet on the market. Specific actions should include:

- A moratorium on permits for new production facilities/expansions for EDC, VCM, and PVC and modification of permits at existing plants to require that dioxin releases to all media, including wastes destined for disposal, be brought to zero within 5 years.
- A moratorium on permits for new incinerators and other waste combustion facilities, and modification of existing permits to require that dioxin emissions to all media to be brought to zero within 5 years by eliminating the input of chlorinated wastes and product.
- A phase-out of medical waste and municipal solid waste incineration.
- Rapid phase-outs of:
  - all short-life PVC uses (packaging, toys, IV-bags etc.);
  - PVC products in areas susceptible to fire (PVC coated cables, construction materials, appliances, and vehicles); and

- recycling of metals containing PVC residues in combustion-based processes (i.e., electrical cables, automobiles).

## **2. Acknowledge and Investigate the Role of PVC in Dioxin Formation**

First, U.S. EPA must acknowledge the important role of PVC in dioxin formation. Since at least 1990, EPA has had information that the incineration and manufacture of PVC produce dioxins. Since 1993, extensive information on these subjects has been submitted to the agency. Nevertheless, EPA has failed to acknowledge or substantively investigate the critical role of PVC in major dioxin sources.

EPA has made no attempt to collect or analyze samples of waste, wastewater, or air emissions from the nation's 14 EDC/VCM facilities or the incinerators that burn wastes from these plants. Instead of using its authority under the Toxic Substances Control Act (TSCA) to require dioxin sampling by the industry, the EPA is allowing the Vinyl Institute (VI), the industry's trade organization, to sample and "self-characterize" its dioxin output. Despite the late arrival of a "peer review" committee, the industry chooses where, when, and how samples will be taken and analyzed, and which data are suitable for submission (see Case Study 1). It is unclear whether these results can be considered either trustworthy or useful.

While the PVC industry is in the process of "self-characterizing" its pollution, it continues to grow rapidly, building new production facilities and expanding production capacity. At least nine of the nations EDC, VCM, or PVC producers have begun new construction or announced plans for major expansions. At least 21 new plants are in construction around the world, primarily in Asia, Latin America, and the Middle East (see Case Study 3). Thus, while the industry studies itself, its total dioxin releases not only go unaddressed, but can actually increase. EPA should immediately establish its own independent investigation of dioxin production associated with the manufacture, use, and disposal of PVC and its feedstocks.

## **3. Focus Dioxin Policy on Materials**

EPA should reorient its dioxin research and policy towards pollution prevention. This requires a fundamental shift away from the agency's current reliance on pollution control techniques for managing dioxin sources. The agency should establish a goal of zero dioxin discharges to all environmental media and focus on reducing the production and use of materials that cause dioxin generation during their lifecycle. This shift requires that research on dioxin sources also be reoriented to emphasize the identification of those dioxin-producing materials.

#### 4. Address Environmental Justice Concerns Associated with the PVC Lifecycle

Pollution associated with the lifecycle of PVC has a disproportionate effect on low-income communities and peoples of color. The incinerators where PVC-related municipal, medical, and hazardous wastes are burned tend to be located in African-American, Latino, and Native American communities. These facilities -- particularly municipal, medical, and hazardous waste incinerators -- are major dioxin sources.

A similar pattern holds true for the manufacture of PVC, as well. There are 14 U.S. facilities that produce ethylene dichloride and/or vinyl chloride feedstocks for PVC. The mean percentage of "non-white" residents in these communities is 57 percent higher than the national average, a preliminary analysis of census data indicates (see Table 9). The higher "non-white" fraction in communities with EDC/VCM facilities is statistically significant at a level of confidence greater than 90 percent.

Pollution caused by PVC production and disposal is thus an important environmental justice issue. EPA should apply its environmental justice policy to investigate and initiate action to prevent dioxin formation during the lifecycle of PVC plastic. President Clinton's Executive Order 12898 on Environmental Justice requires that the agency improve "research relating to the health and environment of minority populations" and reduce pollution in these communities.

**TABLE 9: Demographics of Communities with EDC/VCM Production Facilities**

Company	Location	Zip	Products	% White	% "Non-white"
Borden	Geismar, LA	70734	EDC/VC	57	43
Dow	Freeport, TX	77541	EDC/VC	79	21
Dow	Plaquemine, LA	70764	EDC/VC	59	41
Formosa	Baton Rouge, LA	70821	EDC/VC	ND	ND
Formosa	Pt. Comfort, TX	77978	EDC/VC	96	4
Georgia-Gulf	Plaquemine, LA	70764	EDC/VC	59	41
Geon	LaPorte, TX	77571	EDC/VC	88	12
Occidental	Convent, LA	70723	EDC	33	67
Occidental	Deer Park, TX	77536	EDC/VC	97	3
Oxymar	Ingleside, TX	78362	EDC/VC	89	11
PPG	Lake Charles, LA	70601	EDC/VC	58	42
Vista	Lake Charles, LA	70669	EDC/VC	85	15
Vulcan	Geismar, LA	70734	EDC	57	43
Westlake	Calvert City, KY	42029	EDC/VC	100	0

Average of EDC/VC communities 73.6 26.4

Average of all U.S. Communities 83.2 16.8

% difference in EDC/VC communities vs. U.S. average -12% +57%

Source: 1980 U.S. Census data.

## 5. Ensure a Just Transition

Any plan to phase-out PVC must prevent or compensate for economic or social dislocation that result from these measures to protect health and the environment from dioxin sources. A tax on the production of EDC/VCM could help to drive the transition away from PVC and finance the costs associated with it.\* The tax could be applied to all producers and exporters of these products, at a level that makes alternatives economically attractive. The resulting revenues should be used to develop transitional measures to ensure that a PVC phase-out is just, equitable, and orderly. A transition fund should be established to assist workers and communities affected by the transition, provide educational opportunities, income protection, and health insurance for those affected by a PVC phase-out, and finance research and development into safer PVC alternatives. An orderly and effective PVC phase-out process would begin by prioritizing measures to achieve maximum PVC reduction with minimum social impact. As discussed above, such measures would include immediate restrictions on those applications of PVC for which alternatives are most readily available and those which lead most directly to the greatest generation of dioxins.

**TABLE 10: U.S. Producers of Ethylene Dichloride and Vinyl Chloride**

Production capacity in million lbs/yr (1995)

<b>Company</b>	<b>Location</b>	<b>EDC</b>	<b>VCM</b>
Borden Chemical	Geismar, LA	745	950
Dow Chemical	Freeport, TX	2,820	900
Dow Chemical	Plaquemine, LA	2,100	2,150
Formosa	Baton Rouge, LA	1,195	1,280
Formosa Pt	Comfort, TX	1,205	800
Geon	LaPorte, TX	1,705	1,400
Georgia Gulf	Plaquemine, LA	2,075	1,280
Occidental	Convent, LA	800	ND
Oxymar	Ingleside, TX	2,310	1,400
PPG	Lake Charles, LA	2,700	625
Vista Chemical	Lake Charles, LA	1,250	900
Vulcan Chemical	Geismar, LA	300	ND
Westlake	Calvert City, KY	1,300	1,000
<b>TOTAL</b>		<b>22,092</b>	<b>13,785</b>

Source: SRI 1992 and Chemical Week 1995

**TABLE 11: U.S. PVC Producers**

PVC capacity in million lbs/yr (1992)

<b>Company</b>	<b>Location</b>	<b>Capacity</b>
Borden	Geismar, LA	375
Borden	Iliopolis, IL	300
Certainteed	Lake Charles, LA	200
Formosa	Delaware City, DE	280
Formosa Pt	Comfort, TX	700
Georgia Gulf	Delaware City, DE	140
Georgia Gulf	Plaquemine, LA	700
GEON	Avon Lake, OH	380
GEON	Deer Park, TX	285
GEON	Henry, IL	185
GEON	Louisville, KY	275
GEON	Pedricktown, NJ	340
GEON	Plaquemine, LA	185
Goodyear	Niagara Falls, NY	115
Keysor Century	Saugus, CA	60
Occidental	Addis, LA	300
Occidental	Burlington, NJ	270
Occidental	Pasadena, TX	750
Occidental	Pottstown, PA	200
Shintech	Freeport, TX	1,000
Union Carbide	Texas City, TX	125
Vista	Aberdeen, MS	450
Vista	Oklahoma, OK	350
Vygen	Ashtabula, OH	120
Westlake	Calvert City, KY	290
Westlake	Pensacola, FL	200
TOTAL		8,595

Source: SRI 1992; Chemical Week 1995b

**Notes**

\* A thorough description of a just transition program is contained in GLU, 1995.

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