Environmental Impacts of

Polyvinyl Chloride (PVC) Building Materials

A briefing paper for

the U.S. Green Building Council

prepared under the direction of the Center for Maximum Potential Building Systems

and The Healthy Building Network

by

Joe Thornton, Ph.D.

Columbia Earth Institute, Columbia University

This document was prepared for the U.S. Green Building Council under the supervision of the Center for Maximum Potential Building Systems (Austin, Texas), and the Healthy Building Network (Washington, D.C.).

This report does not represent the opinion of Columbia University, the Trustees of Columbia University, or the Columbia Earth Institute.

About the Author

Joe Thornton, Ph.D., is a research scientist in Columbia University's Earth Institute and Department of Biological Sciences. His research focuses on the health and policy implications of global chemical pollution and on the molecular evolution of animal endocrine systems. He holds Ph.D., M.A., and M.Phil. degrees in Biological Sciences from Columbia University and a B.A. from Yale University.

Dr. Thornton is the author of *Pandora's Poison: Chlorine, Health, and a new Environmental Strategy* (MIT Press, 2000), which the British scientific journal *Nature* has called "a landmark book which should be read by anyone wanting to understand the environmental and health dangers of chlorine chemistry." From the late 1980s to the mid-1990s, Thornton was research analyst and then research coordinator for Greenpeace's toxics campaigns in the United States and internationally. There, he authored seminal reports and articles on organochlorines, dioxin, breast cancer, waste incineration, risk assessment, and the precautionary principle. After moving to Columbia in 1995, Dr. Thornton was co-author of the article and American Public Health Association resolution that launched the campaign to eliminate polyvinyl chloride (PVC) products from medical devices, due to their central role in dioxin formation in medical waste incinerators. He has spoken before the U.S. Congress, the EPA Science Advisory Board, the American Association for the Advancement of Science, the American Public Health Association, the International Joint Commission, and a variety of other organizations and audiences. His work has been published in numerous scientific journals, including the *Proceedings of the National Academy of Sciences*, *Annual Review of Genomics and Human Genetics*, *Public Health Reports*, *Bioessays*, *Systematic Biology*, and *International Journal of Occupational and Environmental Health*.

CONTENTS

Summary of Findings 6

1. Introduction 13
- 1.1 Purpose 13
- 1.2 The Lifecycle of PVC 14
- 1.3 International Action on PVC 16
- 1.4 PVC and Chlorine 19

2. Production of PVC 24
- 2.1 Production of chlorine 24
  - 2.1.1 Organic by-products 25
  - 2.1.2 Mercury releases 27
  - 2.1.3 Energy consumption 28
- 2.2 Synthesis of EDC and VCM 29
  - 2.2.1 Releases of EDC and VCM 29
  - 2.2.2 Organic by-products 31
  - 2.2.3 Dioxins and related compounds 32
- 2.3 Disposal of hazardous wastes 35
- 2.4 Polymerization, formulation, and compounding 38

3. Use of PVC products 40
- 3.1 Organic by-products 40
- 3.2 Indoor air quality 40
  - 3.2.1 Release of phthalates 40
  - 3.2.2 Ashtma and other conditions 42
  - 3.2.3 Vinyl and toxic mold growth 43
  - 3.2.4 Release of lead and other stabilizers 44
- 3.3 Accidental fires 44

4. Disposal of PVC Products 47
- 4.1 Recycling 47
- 4.2 Land Disposal 48
- 4.3 Incineration 49
  - 4.3.1 The role of PVC in incinerators 49
  - 4.3.2 Dioxin formation and PVC – experimental evidence 52
  - 4.3.3 Dioxin formation and PVC – historical evidence 58

5. Background on persistent organic pollutants and PVC 62
- 5.1 Global distribution 62
- 5.2 Endocrine disruption 64
- 5.3 Dioxin and related compounds 65
  - 5.3.1 Global accumulation and exposure 65
  - 5.3.2 Health impacts 66
  - 5.3.3 Trends in dioxin contamination 68
- 5.4 Phthalates 70
  - 5.4.1 Usage 70
  - 5.4.2 Environmental fate and exposure 71
  - 5.4.3 Health impacts 71
- 5.5. Trends in PVC Markets 74

6. Conclusions 77

7. Notes 78

8. References cited 89

SUMMARY OF FINDINGS

In the last 40 years, polyvinyl chloride plastic (PVC) has become a major building material. Global vinyl production now totals over 30 million tons per year, the majority of which is directed to building applications, furnishings, and electronics.

The manufacture, use, and disposal of PVC poses substantial and unique environmental and human health hazards. Across the world, governments, companies, and scientific organizations have recognized the hazards of PVC. In virtually all European nations, certain uses of PVC have been eliminated for environmental reasons, and several countries have ambitious programs to reduce PVC use overall. Scores of communities have PVC avoidance policies, and dozens of green buildings have been built with little or no PVC. Firms in a variety of industries have announced measures to reduce PVC consumption and are using or producing alternative materials in a variety of product sectors, including building materials.

The major hazards of the PVC lifecycle discussed in this report are summarized below.

1. PVC and chlorine. Vinyl is the largest use of chlorine gas in the world, consuming about 40 percent of total chlorine production, or about 16 million tons of chlorine per year. PVC is the largest production-volume organochlorine, a large class of chemicals that have come under considerable scientific and regulatory scrutiny in the last decade because of their global distribution and the unusually severe hazards they tend to pose. Vinyl is the only major building material that is an organochlorine; alternative materials, including most other plastics, do not contain chlorine and do not pose the hazards discussed in this report.

2. Formation of by-products. At numerous points in the vinyl lifecycle, very large quantities of hazardous organochlorine by-products are formed accidentally and released into the environment.

   · Formation of hazardous organochlorine by-products begins with the production of chlorine gas. Extremely large quantities of chlorine-rich hazardous wastes are generated in the synthesis of ethylene dichloride and vinyl chloride monomer (EDC and VCM, the feedstocks for PVC). Still more by-products are created and released to the environment during the incineration of hazardous wastes from EDC and VCM production, the incineration of vinyl products in the waste stream, the recycling of vinyl-containing metal products by combustion, and the accidental burning of PVC in fires in buildings, warehouses, or landfills.

- These chemical mixtures include such pollutants as the chlorinated dioxins (polychlorinated dibenzo-p-dioxins), chlorinated furans (polychlorinated dibenzofurans), PCBs (polychlorinated biphenyls), hexachlorobenzene (HCB), and octachlorostyrene (OCS). In addition, a very large portion of these mixtures consists of chemicals that have not yet been identified or tested.

3. Hazards of by-products. The by-products of the vinyl lifecycle are of great concern, because many of their components are highly persistent, bioaccumulative, and toxic.

   - Persistence means that a substance resists natural degradation, builds up over time in the environment, and can be distributed globally on currents of wind and water. Many of the by-products of the PVC lifecycle are now ubiquitous global pollutants, which can be found not only in industrialized regions but in the planet's most remote ecosystems. Absolutely every person on earth is now exposed to these substances.

   - Bioaccumulation means that a substance is fat-soluble and therefore builds up in the tissues of living things. Most bioaccumulative substances, including many formed during the PVC lifecycle, magnify as they move up the food chain, reaching concentrations in species high on the food chain that are millions of times greater than their levels in the ambient environment. These substances also cross the placenta easily and concentrate in the breast milk of human and other mammals.

   - The by-products of the vinyl lifecycle have been shown to cause a range of health hazards, often at extremely low doses, including cancer, disruption of the endocrine system, reproductive impairment, impaired child development and birth defects, neurotoxicity (damage to the brain or its function), and suppression of the immune system.

4. Vinyl and dioxin. Among the most important by-products of the PVC lifecycle are dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxins) and a large group of structurally and toxicologically related compounds, collectively called *dioxins*. Dioxins are never manufactured intentionally but are formed accidentally whenever chlorine gas is used or chlorine-based organic chemicals are burned or processed under reactive conditions.

   - Formation of considerable quantities of dioxins has been documented during numerous stages of the vinyl lifecycle, including production of chlorine, synthesis of feedstocks, burning of vinyl products in accidental fire, and incineration of vinyl products and the hazardous wastes from PVC production.

- Vinyl is the predominant chlorine donor and therefore a major cause of dioxin formation in most of the leading dioxin sources that have been identified. When its entire lifecycle is considered, vinyl appears to be associated with more dioxin formation than any other single product.
- Dioxins are true global pollutants, now found in the tissues of whales in the deep oceans, polar bears in the high Arctic, and every human being on earth. Human infants receive particularly high doses (orders of magnitude greater than those of the average adult), because dioxins cross the placenta easily and concentrate in breast milk.
- Dioxin is the most potent synthetic carcinogen ever tested in laboratory animals and appears to be equally carcinogenic in people.
- Human development, reproduction, and the immune and endocrine systems are exquisitely sensitive to dioxin, which causes health and functional impairment at infinitessimally low doses (in the parts per trillion or even parts per quadrillion range). Toxicological studies have not been able to establish any dose level of dioxin at which biological impacts do not occur.
- The dioxin "body burden" of the general human population of the United States is already in the range at which adverse health impacts occur in laboratory animals. The dioxin exposure of the average American already poses a calculated cancer risk of one in 1,000 to one in 100 – thousands of times greater than the usual standard for an "acceptable risk."
- Dioxin at current levels therefore represents an unacceptable public health hazard; efforts to reduce dioxin formation at the source should be a high environmental and public health priority.

5. Phthalate plasticizers. In its pure form, PVC is rigid and brittle. To make flexible vinyl products, such as floor tiles and wall coverings, plasticizers must be added to PVC in large quantities – up to 60 percent of the final product by weight. The dominant class of plasticizers used in vinyl are a class of compounds called phthalates, which pose considerable health and environmental hazards. Vinyl is the only major building product in which phthalates are used extensively, and it accounts for about 90 percent of total phthalate consumption. Over 5 million tons of phthalates are used in vinyl every year.
   - Phthalates are moderately persistent, bioaccumulative substances that have become true global pollutants; they can be found in the water of the deep oceans, air in remote regions, and the tissues and fluids of every person in the world. Body burdens of some phthalates have recently been found to be surprisingly high in the bodies of the general U.S. population. Infants and toddlers are subject to exposures several times higher than those of the average adult.

- Millions of pounds per year of phthalates are released into the environment during the formulation and molding of vinyl products. Phthalates are also released when vinyl is disposed of in landfills or incinerators or when PVC products burn accidentally. An astounding 83 million tons of phthalates are currently contained in the stock of PVC products currently in use in buildings and other applications.
- Phthalates are not chemically bonded to the plastic but are merely mixed with the polymer during formulation. They therefore leach out of the plastic over time into air, water, or other substances with which vinyl comes in contact.
- Phthalates used in PVC cause a range of health effects. Diethylhexyl phthalate (DEHP, the highest-volume vinyl plasticizer and the best studied member of the class) causes cancer in laboratory animals and is considered a human carcinogen by the National Toxicology Program. Several vinyl-related phthalates, including DEHP are endocrine disrupters, mimicking or blocking the estrogen receptor and interfering with several other hormone systems. Phthalates have been found in the laboratory to damage the reproductive system of males and females, causing infertility, reduced sperm count, suppressed ovulation, and abnormal development and function of the testes and male reproductive tract.
- An expert committee of the National Toxicology Program recently reviewed the hazards of DEHP and expressed "concern that exposure [of infants and toddlers in the general U.S. population] may adversely affect male reproductive tract development" and "concern that ambient oral DEHP exposures to pregnant or lactating women may adversely affect the development of their offspring."

6. Lead and other stabilizers. Because PVC catalyzes its own decomposition, metal stabilizers are added to vinyl for construction and other extended-life applications.
   - Common PVC additives that are particularly hazardous are lead, cadmium, and organotins, with global consumption of each by vinyl estimated in the thousands of tons per year.
   - Metals do not degrade at all in the environment. All three of the major PVC stabilizers have become global pollutants.
   - Lead is an exquisitely potent developmental toxicant, damaging brain development and reducing the cognitive ability and IQ of children in infinitessimal doses. Organotins are potent endocrine disrupters that have been linked to reproductive damage in wildlife, and cadmium is a potent neurotoxin.

- Metal stabilizers are released from vinyl products when they are formulated, used, and disposed. Releases of lead stabilizers from interior vinyl building products have been documented. Metals cannot be destroyed by incineration but are released entirely into the environment, via air emissions or ash residues. Trash incinerators are the dominant source of lead and cadmium pollution, and PVC contributes a significant portion of the feed of these metals – an estimated 45,000 tons of lead each year -- to incinerators.
- Accidental fires are also major potential sources of lead, cadmium, and organotins; over 3.2 million tons of lead in the current stock of PVC in use.

7. Indoor air quality. Flexible vinyl products appear to contribute to the health hazards of poor indoor air by releasing phthalates and facilitating the growth of hazardous molds.
   - The phthalates in PVC are released into the building environment. Phthalate levels in indoor air in buildings with PVC are typically many times higher than in outdoor air. Phthalate accumulation in suspended and sedimented indoor dusts are particularly high, with concentrations in dust as high as 1,000 parts per million.
   - There is evidence that PVC-related phthalates exposure maybe linked to asthma. In laboratory animals, metabolites of phthalates used in vinyl cause asthma-like symptoms through a well-described inflammatory mechanism. Three separate epidemiological studies have found that exposure to PVC in building interiors causes significantly elevated risks of asthma and other pulmonary conditions, including bronchial obstruction, wheezing, pneumonia, prolonged cough, and irritation of the nasal passages and eyes.
   - Metal stabilizers, particularly lead, cadmium, and organotins, can also be released from vinyl products. Significant quantities of lead have been found to be released from vinyl window blinds into air and PVC pipes into water. Toxicological effects of these substances include neurological, development, and reproductive damage.
   - Because vinyl wall coverings forms a barrier impermeable to moisture, they encourage the growth of molds on wall surfaces beneath the vinyl, particularly in buildings where air condition or heating produce significant temperature and humidity differentials between rooms and wall cavities. Some molds that grow beneath vinyl produce toxic products that are released into indoor air; numerous liability suits are active on the link between vinyl-produced molds and respiratory and neurological symptoms among exposed persons. Vinyl has been cited as the interior building material most likely to facilitate the growth of these molds.

8. Occupational and local environmental exposures.

   · In the production of PVC, hundreds of thousands of tons per year of the feedstocks ethylene dichlroide (EDC) and vinyl chloride monomer (VCM) are released into the workplace and into local environments.

   · Both EDC and VCM are known human carcinogens. They are toxic to the nervous system and cause a variety of other impacts on human health.

   · Increased risks of liver cancer and brain cancer have been documented among workers exposed to VCM. There is suggestive evidence that workers involved in the manufacture of PVC products have elevated risks of testicular cancer.

   · Severe contamination of communities and waterways in the vicinity of VCM production facilities has been documented. In Louisiana, significantly elevated levels of dioxins have been found in the blood of people living near a VCM facility, several communities have been evacuated due to VCM contamination of groundwater, and extremely high levels of highly persistent, bioaccumulative by-products attributable to VCM production have been found in local waterways. In Europe, VCM production facilities have caused severe regional-scale contamination with dioxins and other by-products.

9. Energy consumption. Chlorine production is one of the world's most energy-intensive industrial processes, consuming about 1 percent of the world's total electricity output. Chlroine production for PVC consumes an estimated 47 billion kilowatt hours per year – about equivalent to the annual total output of eight medium-sized nuclear power plants.

10. Mercury pollution. The mercury-based process for producing chlorine accounts for about a third of world chlorine production. In this process, very large quantities of mercury are released into the environment. Mercury is now a global pollutant that causes severe reproductive, developmental, and neurological impacts at low doses. Chlorine production for PVC results in the release of over 200,000 pounds of mercury to air, water, and land each year.

11. Recycling. Very little PVC is recycled, and this situation is unlikely to change in the foreseeable future. Because each PVC product contains a unique mix of additives, postconsumer recycling of mixed PVC products is difficult and cannot yield vinyl products with equivalent qualities as the original. Even in Europe, where PVC recycling is more advanced than in the United States, less than 3 percent of postconsumer PVC is recycled, and most of this is "downcycled" into other products,

which means there is no net reduction in the production of virgin PVC. By 2020, only 9 percent of all PVC waste is expected to be recycled, with a maximum potential of no more than 18 percent.

When the entire lifecycle of PVC is considered, it is apparent that PVC is one of the most environmentally hazardous materials in production. Vinyl production, use and disposal is responsible for the genreration of very large quantities of persistent, bioaccumulative and toxic pollutants and releasing them into the global environment. Available data suggest that PVC has contributed a significant portion of the world's burden of persistent organic pollutants and endocrine disrupting chemicals, including dioxins and phthalates, that have accumulated universally in the environment and the bodies of the human population.

PVC is the antithesis of a green building material. Efforts to speed adoption of safer, viable substitute building materials can have significant, tangible benefits for human health and the environment.

1. INTRODUCTION

1.1 PURPOSE OF THIS DOCUMENT

The purpose of this document is to show that the lifecycle of polyvinyl chloride plastic (PVC, commonly known as vinyl) represents a significant hazard to human health and the environment. It has been prepared in support of a proposal to incorporate into the U.S. Green Building Council's (USGBC) LEED standard for commercial interiors a credit for eliminating the use of virgin PVC and other chlorinated plastics. In considering this proposal, USGBC requested separate presentations of the arguments for and against the view that PVC is not a green building material. This paper represents the position that vinyl building materials cause significant and unique environmental and public health threats. To that end, we examine the formation, release, exposure and health implications of hazardous substances during the manufacture, use, and disposal of vinyl products, as well as energy consumption associated with PVC production. A concluding section gives background on scientific and economic issues relevant to vinyl and persistent organic pollutants, including a detailed discussion of the environmental hazards of specific substances associated with the vinyl lifecycle.

In the United States, an estimated 75 percent of all vinyl consumption is in building and construction applications.[1] In the European Union, 60 percent of vinyl is used in building and construction applications, with an additional 25 percent in furniture, appliances, and electronics.[2] Where possible, hazards specific to building and construction uses are highlighted in this report, and issues specifically relevant to commercial interiors are addressed. Many of the hazards of the vinyl lifecycle are general to all PVC uses, however, and will be discussed in that context. Although the USGBC's proposed action would be implemented primarily in the United States, this report takes an international perspective on vinyl markets and environmental impacts, because many of the hazards associated with PVC are global in scale. Further, some PVC products are imported, so decisions about building materials in the United States can have considerable impacts on environmental quality in the nations where vinyl products are made. Finally, USGBC's decision could affect the choice of building materials by actors in other nations concerned with environmentally sound building practices.

1.2 THE LIFECYCLE OF PVC

PVC's lifecycle consists of three major stages – manufacture, use, and disposal (figure 1). Environmental hazards of vinyl production include the formation and release of toxic substances and the consumption of energy and resources in any and all of these steps.

1. Manufacture. PVC manufacture comprises six steps:

- The first step in producing vinyl is the production of two basic materials: ethylene gas (purified from petroleum or natural gas) and chlorine gas (synthesized from sea salt by high-energy electrolysis).
- The next step involves the production of the feedstock ethylene dichloride (EDC, also known as 1,2-dichloroethane) from chlorine and ethylene using two separate types of reaction. In chlorination, ethylene and chlorine are combined to produce EDC; hydrogen chloride formed as a by-product in this reaction is then combined with more ethylene to produce additional EDC, a process called oxychlorination.
- In the third step, EDC is converted into vinyl chloride monomer (VCM, the chemical name of which is chloroethylene), by a reaction called pyrolysis.
- In the polymerization stage, VCM molecules are linked together to yield polyvinyl chloride, typically a white powder.
- In the fifth step of PVC manufacture, called formulation or compounding, pure PVC is mixed with other chemicals – stabilizers, plasticizers, colorants, etc. -- to yield a usable plastic with desired properties. In its pure form, PVC is not particularly useful: it is rigid and brittle, and it gradually catalyzes its own decomposition when exposed to ultraviolet light. For PVC to be made into useful products, then, additives must be mixed with the polymer to make it flexible, moldable and long-lasting.[3] PVC additives include a range of toxic compounds, but the most environmentally important of these are the phthalate plasticizers and metal-based stabilizers (lead, cadmium, organotins, zinc, and other compounds).

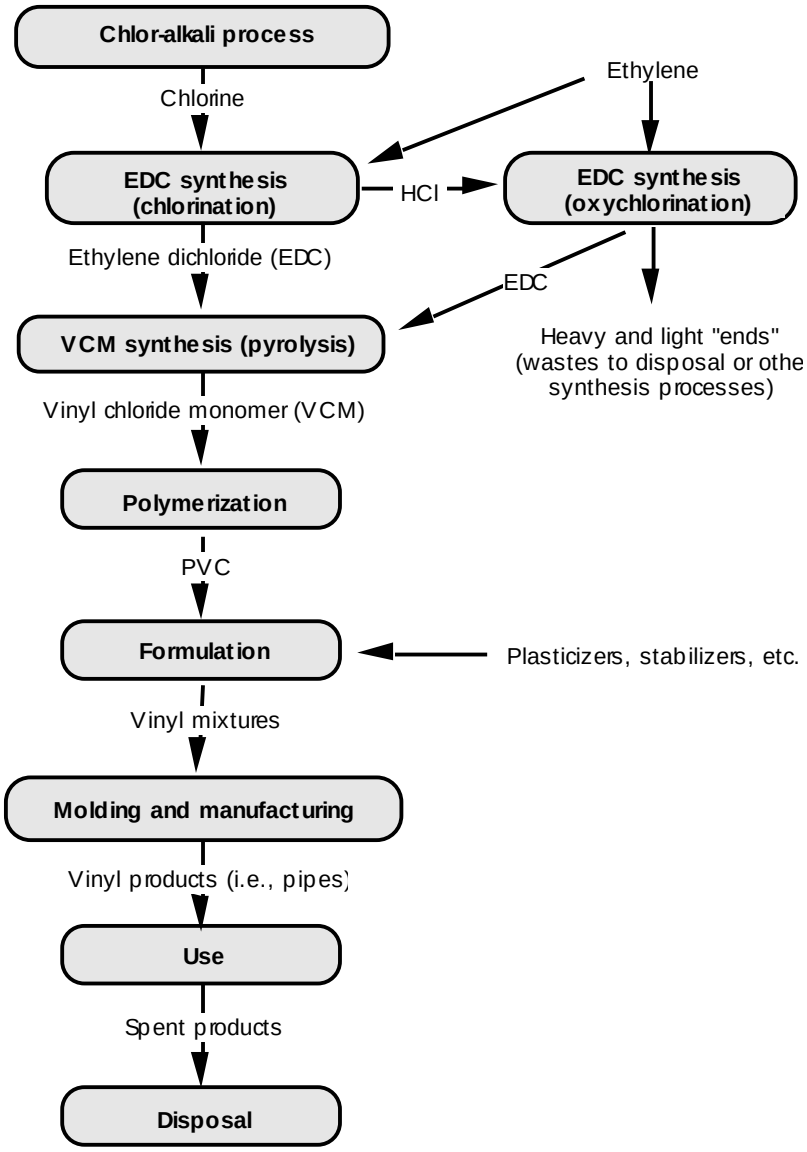


Figure 1. The lifecycle of PVC. Stages are shown in boxes with bold type; regular type and arrows show material flows.

- Finally, the formulated plastic is molded to produce the final product -- a bottle, window frame, or pipe, for instance.

` 2. Use. The second major stage in the PVC lifecycle is use of vinyl products. The duration of the product's useful life may be very short, as in PVC packaging, or rather long, as in PVC window frames. Environmental hazards during this stage include the release of toxic substances into the indoor or outdoor environment from the vinyl product or during accidental combustion. A very small portion of vinyl is recycled, a process that can lead to the dispersal of hazardous additives into the environment or a greater range of consumer products.

3. Finally, after its useful life, the vinyl product is disposed of, typically in incinerators or landfills. Environmental impacts at this stage include the long-term persistence of vinyl products in land disposal facilities, the leaching of hazardous substances out of the product, and the formation and release of unintended combustion by-products when vinyl is incinerated or processed in a secondary smelter for recycling metal products.

1.3 INTERNATIONAL ACTION ON PVC

At the time of this writing, the nations of the world are negotiating the first legally binding instrument to address global contamination by persistent organic pollutants (POPs). The draft agreement,[4] which would require each nation to eliminate the production of POPs, represents a fundamental shift from the present use of control and disposal techniques to manage chemicals. Although the treaty would take initial action on just 12 pollutants (see table 1), it would include provisions for additional substances to be addressed in the future. Several of these pollutants are produced in significant quantities during the vinyl lifecycle (table 1).

The USGBC's proposed standard comes in the context of considerable international concern and activity to restrict PVC consumption for environmental reasons. In 1995, for example, the American Public Health Association adopted a consensus resolution that hospitals should "reduce or eliminate their use of PVC plastics" wherever feasible, due to the global health and environmental impacts of the PVC lifecycle, dioxin generation in particular.[5] In a study of all major packaging materials for the Council of State Governments in the U.S., the independent Tellus Institute found that PVC is the most

environmentally damaging of all plastics.[6] A lifecycle analysis by the Danish EPA found that the common plastics polyethylene, polypropylene, polystyrene, polyethylene terephthalate (PET), and ethylene-propylene diene synthetic rubber are all clearly preferable to PVC in terms of resource and energy consumption, accident risk, and occupational and environmental hazards, including chemical exposure.[7]

Table 1. Persistent Organic Pollutants Addressed by UNEP's Internationl POPs Agreement

Aldrin
DDT
Dieldrin
Endrin
Chlordane
Heptachlor
* Hexachlorobenzene
Mirex
Toxaphene
* Polychlorinated Biphenyls (PCBs)
* Polychlorinated dibenzo-p-dioxins (PCDDs)
* Polychlorinated dibenzo-furans (PCDFs)

* Produced at one or more points during the lifecycle of PVC (see text.). Source: UNEP 1997.

Virtually all nations in the European Union have restrictions on some uses of PVC due to concerns about dioxin, release of phthalate softeners, or the difficulty of recycling and waste disposal. Among the most far-reaching policies are those of Sweden, the parliament of which voted in 1995 that both soft and rigid PVC should be gradually eliminated. In 1996 the government called for a voluntary phase-out by industry of all production of PVC. In 1999, the government adopted a bill that includes mandatory provisions to eliminate all use of PVC with hazardous additives, including phthalates and lead, and to substitute all uses of PVC with other materials wherever feasible. From 1994 to 1999, this program had already reduced total PVC use in Sweden by 39 percent.[8]

In Denmark, there is a national strategy in place to address the environmental hazards of PVC, including a tax on PVC of 0.30 USD/kg, an even higher tax on phthalates, a prohibition on the incineration of PVC, and an order to substitute alternative materials for all PVC uses which are not recycled. The German Environmental Protection Agency (UBA) has called for an end to the use of phthalates and a gradual phase-out all uses of flexible PVC.[9] UBA has also called for a ban on the use of

PVC in applications susceptible to fire due to concerns about dioxin generation.[10] In the Netherlands, government policy is to reduce the use of PVC in products that are not recycled and to eliminate the use of phthalate plasticizers and lead stabilizers in PVC. The European Union has begun an official process to review the environmental hazards of PVC and establish appropriate policy measures to safeguard the environment.[11]

Not all action on PVC is restricted to Europe. In India, the Ministry of the Environment and Forests has established rules that ban the incineration of PVC and other chlorinated plastics in medical waste incinerators. The government of Singapore, responding to the difficulty of safely disposing of PVC, has informed the Secretariat of the Basel Convention on the transboundary movement of hazardous waste that it considers PVC waste and PVC coated cables to be hazardous waste that are therefore banned from import or export.[12]

Numerous local and regional governments have specific policies to avoid PVC in construction.[13] In Germany, 274 communities – including Berlin and Bonn -- and six states have written policies to phase-out or restrict PVC. In the Netherlands, the four largest cities -- Amsterdam, Rotterdam, Utrecht, and Den Haag -- have specifications to avoid PVC whenever possible in construction. In Spain, 52 cities have declared themselves "PVC-free," with specific strategies to substitute safer alternatives for PVC construction materials. Basel, Switzerland, has guidelines for the use of environmentally friendly materials, which list PVC as environmentally harmful and a candidate for substitution whenever possible. In Austria, seven of nine states and a large number of municipalities have restrictions on PVC; the city of Linz has achieved an 85 percent PVC phase-out in public buildings, and the Vienna subway and Vienna Ost hospital have been built without PVC. The city of Berkeley, California, is considering a measure to eliminate PVC from new construction, due to concern about dioxin, endocrine disruption, and cancer during the vinyl lifecycle.[14]

Numerous major construction products have reduced or eliminated PVC entirely.[15] In an effort to utilize green building practices, the Sydney 2000 Olympics established a commitment "to minimizing and ideally avoiding the use of chlorine-based products such as PCBs, PVC, and chlorine-bleached paper." PVC use was eliminated or radically reduced in the construction of the stadium, Olympic village, hotel, and many other structures. Seville's guidelines for the 2004 Olympics specify that "we must avoid the use of PVC in construction, infrastructure, accessories and any other complements in Olympics facilities."

The Danish society of Engineers headquarters has been built without PVC, and the Nike Corporation's new European headquarters in Hilversum, the Netherlands, has been built without PVC. A number of buildings in the United Kingdom, including the new Tate Gallery of Modern Art, have been designed and built to minimize or eliminate PVC use.

A large number of private firms have also taken steps to restrict or substitute for PVC products due to environmental concern.[16] The Swedish construction firms HM and Svenska Bostder have announced that they are phasing out their use of PVC. The electronics manufacturers AEG, Sony-Europe, Vorwerk, Electrolux, Sharp, Matsushita, and Ricoh of Japan all have PVC phase-out policies. German Telekom, Nippon, and Sumitomo Electic Industry all have policies to avoid PVC in cable manufacture. The furniture and décor manufacturers and retailers EWE Kuechen (Austria), Eco AB, Innarps AB, and IKEA have policies to avoid PVC products. In transportation, virtually all of the world's major car manufacturers, including General Motors, Ford, Daimler-Benz, Volkswagen AG, BMW, Opel, Honda, Nissan and Toyota, have policies to reduce or eliminate the use of PVC in automobiles.

Particularly urgent and widespread action has focused on PVC toys, due to concern about child exposure to phthalate plasticizers in flexible PVC. In the late 1990s, government ministries in Denmark and the Netherlands found that substantial quantities of phthalates are released from PVC into saliva from vinyl teethers and chew toys. These countries, along with ministries in Belgium, Austria, Germany, Italy, Norway, Sweden, Finland, France, Greece and Spain, then sought bans on the use of soft PVC in toys. In 1997, a number of European toy retailers and manufacturers suspended sales of PVC teething rings or announced plans to eliminate all vinyl from their toy lines. In late 1998, when a wave of publicity on the issue hit the U.S. press, Toys-R-Us, Mattel and several other U.S. toy makers and retailers also announced they would stop selling some kinds of vinyl toys.[17] Subsequently, the U.S. Consumer Product Safety Commission called on the toy industry to voluntarily stop making vinyl toys that were made to be chewed and contained phthalates.[18] In 1999, the European Commission finalized an emergency ban on six phthalate plasticizers found in soft PVC toys.

1.4 PVC AND CHLORINE CHEMISTRY

PVC, EDC, VC, and the by-products formed during the vinyl lifecycle are members of a large class of problematic chemicals called organochlorines – organic (carbon-based) substances that also

contain one or more atoms of chlorine. The debate over PVC takes place in the context of broader concern about the class of organochlorines, which are regarded by many as the most environmentally problematic large class of synthetic substances.[19] PVC is the only major plastic used in buildings that contains chlorine; chlorine-free plastics include polyethylene, polypropylene, ethylene vinyl acetate, acrylonitrile-butadiene-styrene copolymer, and numerous other plastics. Polyurethane, polycarbonate, and epoxy resins are chlorine-free plastics that are currently manufactured via the organochlorine intermediates phosgene, propylene chlorohydrin, and epichlorohydrin, but technologies are being developed to produce these plastics through chlorine-free routes.[20]

The proper course of action for addressing organochlorines is a controversial topic. Some organizations and analysts, including the American Public Health Association and the International Joint Commission (a binational advisory body of the U.S. and Canadian governments charged with protection of the Great Lakes ecosystem) have called for a gradual phase-out of all uses of chlorine and organochlorines. Others, including the chemical industry and the Society of Toxicologists, have called for continuing the current system of chemical-by-chemical regulation.

The USGBC's decision on a standard for PVC in building materials does not require the proper course of action on all organochlorines to be resolved. It is useful, however, to see the question of vinyl in the context of concern about the class of chemicals of which PVC is perhaps the most important member. Vinyl and its feedstocks have the largest production volume, by far, of all organochlorines; further, a vast variety of other organochlorines are produced in considerable quantities during the lifecycle of vinyl. Many of these by-products have not been specifically identified or evaluated for their environmental behavior and toxicity, and an understanding of the general characteristics of the class of organochlorines is relevant to predicting their hazards.

Concern about organochlorines began with the recognition that they tend to dominate all major lists of "priority pollutants." For example, of the 12 POPs addressed by UNEP, all are organochlorines (table 1). Of the 11 chemicals on the IJC's Critical Track of hazardous pollutants in the Great Lakes, eight are organochlorines, as are 168 of the 362 substances on the IJC's Secondary Track.[21] Organochlorines are prominent on EPA's list of common groundwater pollutants and of contaminants at hazardous waste sites, and they constitute the majority of the list of known endocrine disrupting chemicals.[22]

Further analysis has revealed *why* organochlorines tend to be so problematic. Organochlorines tend to have several characteristics, all of which derive from fundamental chemical properties of the chlorine atom. It is the properties that make chlorine and organochlorines useful in industrial applicatiions, in fact, that are responsible for their environmental hazards.

- Reactivity of chlorine. Chlorine gas is extremely reactive, combining quickly and randomly with whatever organic matter it contacts; this property makes it an effective bleach, disinfectant, and chemical feedstock, but it also inevitably results in the generation of a diverse mixture of by-products, typically containing hundreds or thousands of organochlorines, including dioxins, whenever chlorine is used.[23]

- Persistence. The chlorine atom is extremely electronegative, which means it exerts a very strong pull on electrons that it shares with carbon atoms in an organochlorine. The addition of chlorine to organic molecules therefore changes the chemical stability of the resulting substance, often stabilizing it but sometimes destabilizing it, depending on the structure of the parent compound. In most cases, the resulting organochlorine is far less reactive than the original substance; organochlorines that do break down usually degrarde into other organochlorines, which may be more persistent and toxic than the original substance. Stability makes organochlorines useful as plastics, solvents, and refrigerants in which long-life and fire-resistance are virtues. The same property, however, makes these organochlorines persistent in the environment and therefore likely to accumulate and become globally distributed.

- In some cases, organochlorines become more unstable and reactive than the parent compound, which makes them useful as chemical intermediates. Unfortunately, this property also makes them more easily converted in the body to toxic and reactive metabolites (other organochlorines), which can then proceed to damage DNA or other essential molecules in cells.[24] Chlorine's impact on the stability of organic molecules thus has two two opposite effects, depending on the structure of the parent compound, and both are problematic from an environmental health perspective.

- Bioaccumulation. One factor that determines the solubility of a substance in water or fat is its molecular size: the larger a molecule, the more it disrupts interactions among water molecules, and the greater the tendency for the substances to be excluded from water rather than dissolve in it. The chlorine atom is very large – several times larger than an atom of carbon, hydrogen or oxygen – so

chlorination significantly increases the size of organic molecules and as a result almost invariably increases their solubility in fats and oils. The increase in fat solubility applies to the chlorination of virtually any organic substance, and it increases with each chlorine atom added.[25] Thus, for example, tetrachloroethylene, hexachlorobenzene and octachlorodibenzofuran are about 100, one thousand, and one billion times more oil-soluble, respectively, than their chlorine-free analogs (figure 2). Oil-solubility makes organochlorines effects solvents and dielectric fluids, but it is directly responsible for their tendency to bioaccumulate.

· Toxicity. Finally, organochlorines tend overwhelmingly to be toxic. The American Public Health Association has concluded that "virtually all organochlorines that have been studied exhibit at least one of a range of serious toxic effects, such as endocrine dysfunction, developmental impairment, birth defects, reproductive dysfunction and infertility, immunosuppression and cancer, often at extremely low doses, and many chlorinated organic compounds ... are recognized as significant workplace hazards."[26] According to a comprehensive independent review of thousands of individual organochlorines, chlorination of organic chemicals "is almost associated with an increase in the toxic potential. Only rarely does chlorination produce no increase or even a decrease in effects. This observation applies for all kinds of toxic effect (acute, subchronic and chronic toxicity, reproductive toxicity, mutagenicity, and carcinogenicity."[27] The tendency to be toxic makes organochlorines useful as pesticides and antibiotics, but the same quality makes them hazardous to humans and wildlife once they enter the environment.

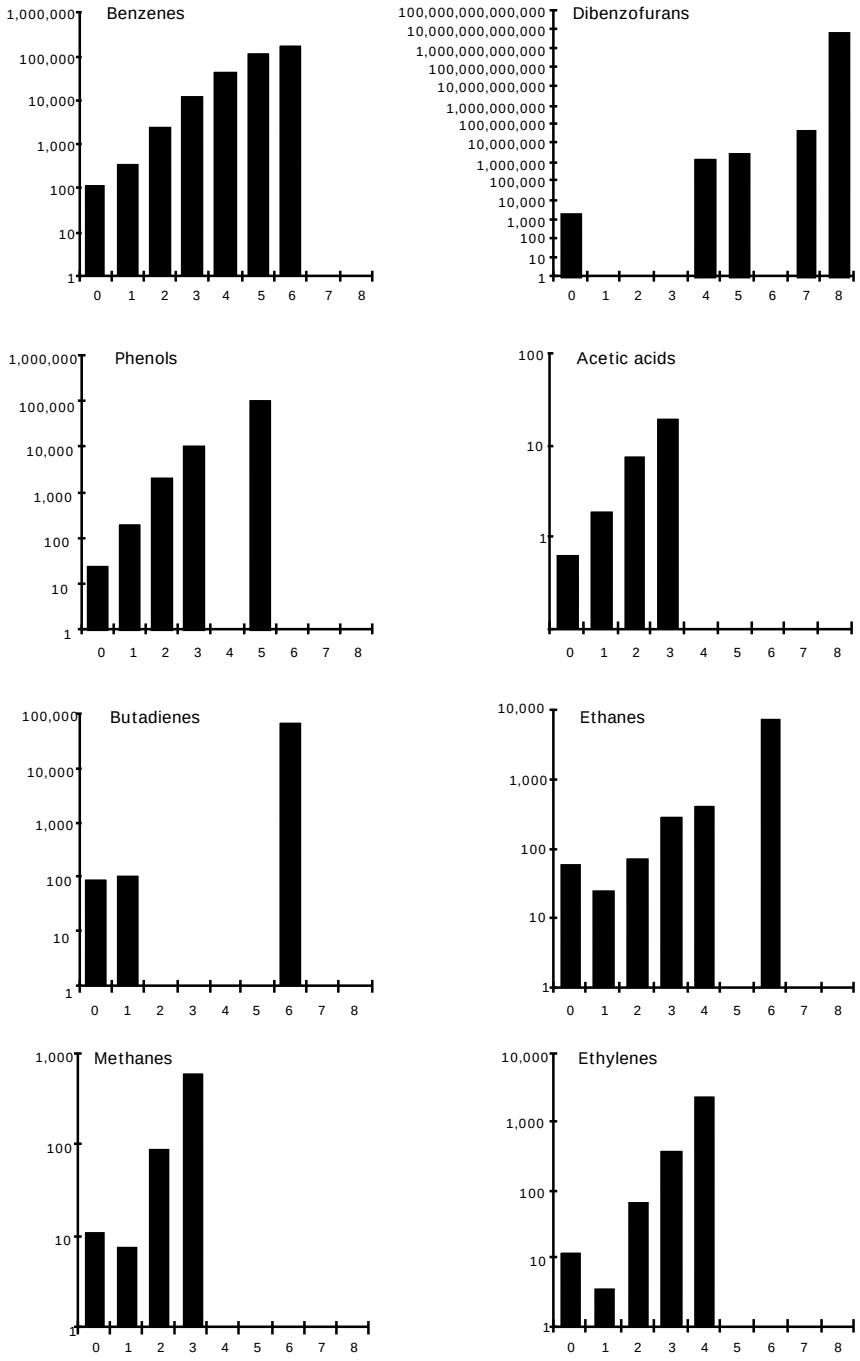


Figure 2. Effect of chlorine on oil-solubility and bioaccumulation of organic chemcials. In all groups, chlorination increases the tendency of a chemical to dissolve in fats and oils, and each chlorine atom has a greater effect. Blank cells indicate no data available. Data source: HSDB 1997.

2. PVC PRODUCTION

2.1 PRODUCTION OF CHLORINE

- The PVC lifecycle begins with the production of chlorine gas in the "chlor-alkali" process. Electricity is passed through a solution of brine to produce sodium hydroxide (alkali, or caustic soda) and chlorine gas in a fixed ratio of 1.1 to 1. Because chlorine gas and sodium hydroxide react with each other on contact, the key the process is to immediately separate the chlorine from the alkali in a specially designed electrolytic chamber, called a cell. There are three types of chlor-alkali cell in use, which differ in the ways that chlorine and alkali are separated from each other.

- The mercury process, the oldest and most energy-intensive of the three processes for chlorine production, involves two cells connected to each other. In the first cell, salt is split into chlorine gas and sodium at the cell's positive terminal (called the anode); the sodium forms an amalgam with a layer of liquid mercury, which then flows into another cell, where it reacts with water to form sodium hydroxide and hydrogen gas. The mercury process is banned in Japan, but 35.5 percent of chlorine worldwide continues to be produced using this method, with 14 percent of chlorine in North America and 65 percent of that in Western Europe produced by this technology as of 1994.[28]

- In the asbestos diaphragm process, brine enters the cell and is split at the anode, yielding chlorine gas and sodium ions. The ions then flow through a semi-permeable asbestos barrier to the other pole, where they react with water to form sodium hydroxide and hydrogen gas; the chlorine, which cannot pass through the membrane, remains near the anode. The diaphragm method was developed after the mercury process and, as of 1994, accounted for 77 percent of all chlorine production in the U.S. and 25 percent of that in Europe.[29]

- The remaining 7 percent of chlor-alkali production in both regions is based on the membrane process, the most recent of the three methods. The membrane technique is similar to the diaphragm process, except that a synthetic membrane rather than asbestos is used to separate the compartments in which chlorine and alkali are formed. The membrane process uses slightly less energy and yields products of higher purity than the other kinds of cell, but retrofitting a chlorine plant with membranes is very expensive. Because most chlor-alkali plants in the U.S. and Europe were built decades ago, few

plants in these countries use the membrane process, but most new facilities, particularly those in Asia and Latin America, are now constructed with it.[30]

The world chlor-alkali industry produced about 39 million metric tons of chlorine in 1997, 27 percent of which was in the United States. In the U.S., there are 42 chlor-alkali facilities, but over 70 percent of production capacity is located at just 12 large plants in the Gulf Coast regions of Louisana and Texas.[31] Because vinyl accounts for more than 40 percent of all chlorine consumption, it is reasonable to say that the same proportion of the environmental impacts of chlorine production – releases of toxic substances and energy demand – is associated with the vinyl lifecycle.

Table 2. Mercury releases from the world chlor-alkali industry, 1994

	Metric tons of mercury per year (1)	PVC attributable portion (2)
Consumption	229.8	91.9
Air emissions	26.3	10.5
Discharges to water	2.8	1.1
Contaminants in products	5.5	2.2
Disposed on land	157.8	63.1
Unaccounted for	36.0	14.4

Source: From Ayres 1997. These data are based on mass balances prepared by the chlorine industry for facilities in Europe and may not accurately represent global averages. (1) Assuming 39 million metric tons per year of chlorine production worldwide, of which 35.5 percent was produced by the mercury process. (2) Assuming that PVC accounts for 40 percent of chlorine consumption worldwide.

2.1.1 Organic by-products

From the moment that chlorine gas is formed in the chlor-alkali cell, it will react with any organic matter that is present to form organochlorines. For this reason, manufacturers carefully purify raw materials and equipment surfaces to remove as much organic material as possible. Nevertheless, carbon-containing substances remain present as trace impurities, in plastic materials, or from the graphite electrodes used in some types of chlor-alkali cells.[32] Chlorine combines with these organic contaminants to form persistent organochlorine by-products, such as hexachlorobenzene (HCB) and hexachloroethane (HCE), which are found in the chlorine product itself.[33] Chlorine gas has also been found to be

contaminated with PCBs, octachlorostyrene (OCS), and tetrachlorobenzene. The concentrations are rather low, but the quantities are significant: based on the levels found, the chlorine gas that is produced every year carries between 1.6 and 8.2 tons of these highly toxic and persistent by-products into the world's economy.[34] Based on an attributable fraction of 40 percent, the chlorine used for vinyl contains between 1400 and 7200 pounds of these substances annually.

Much greaterr quantities of organochlorine contaminants are deposited in the wastes from chlorine production. Swedish researchers have identified very high concentrations of dioxins and furans -- up to 650 parts per billion -- in the sludges from spent graphite electrodes used in chlor-alkali cells,[35] and very high levels of polychlorinated dibenzofurans have been found in the blood of Swedish chlor-alkali workers.[36] Severe contamination of fish and sediments with octachlorostyrene – an extremely persistent, bioaccumulative POP -- has been documented near eight North American chlorine producers, and large-scale OCS contamination of sediments in Lake Ontario has been traced to disposal of spent chlor-alkali electrodes.[37] Octachlorostyrene is now a global contaminant, with considerable levels found in the Canadian arctic, and chlor-alkali manufacture is considered an important source.[38]

Because of this problem, all chlor-alkali plants in North America and many in Europe replaced graphite electrodes with titanium substitutes during the 1970s and 1980s -- a move that industry and government assumed eliminated the formation of organochlorine by-products. But recent data indicate that even the most modern chlor-alkali plants produce dioxin-like compounds. With graphite eliminated, traces of organic chemicals are still present, primarily from plastic pipes and valves that release small quantities of their materials into the cell. A 1993 study by Swedish scientists found dioxins and furans in the sludge and plastic piping from a modern chlor-alkali plant with titanium electrodes at levels around 5 parts per trillion (TEQ).[39] Subsequent Swedish research found significant quantities of chlorinated dibenzofurans in the sludge from a chlor-alkali plant with titanium electrodes, apparently due to chlorination of organic compounds in the rubber linings of the cell.[40] In 1997, the UK Environment Agency has confirmed that a chlor-alkali plant owned by ICI Chemicals and Polymers, which replaced its graphite electrodes around 1980, continues to release dioxins in its wastewater.[41]

2.1.2 Mercury releases

Most of the mercury used in mercury chlor-alkali cells is recycled, but significant quantities are routinely released into the environment via air, water, products, and waste sludges. In the 20th century as a whole, chlor-alkali production has been the largest single source of mercury releases to the environment.[42] As recently as the 1980s, the chlorine industry was second only to fossil fuel combustion as a mercury source in Europe.[43]

Many mercury-cell plants have been retired in the last two decades, and controls on existing plants have improved, but chlor-alkali facilities remain a major source of mercury pollution. The chlorine industry is the largest mercury consumer in the United States; it is presumably even more important in Europe, where the mercury cell process is more common.[44] Based on estimates by Euro-Chlor, the trade association of the European chlorine industry, the world chlor-alkali industry *consumed* about 230 tons of mercury in 1994; this is the quantity not recycled but lost from production processes each year. Exactly where the mercury goes remains controversial, but if we use Euro-Chlor's data, about 30 tons were released directly into the air and water, 5 tons remained as a contaminant in the product, more than 150 tons were disposed on land, and 36 tons could not be accounted for (table 2).[45] The actual worldwide totals are likely to be even higher, because the well-regulated facilities of Europe are not likely to be representative of those in other regions of the world. Based on an attributable fraction of 40 percent, the PVC lifecycle is associated with the consumption of 92 metric tons of mercury (202,400 pounds) per year, of which the majority was released to air, water, or landfills.

Mercury is an extremely toxic, bioaccumulative global pollutant. Mercury compounds cause irreversible health damage to wildlife and humans, developing children in particular, including birth defects, severe neurological symptoms, kidney damage, and impaired neurological development.[46] The most tragic and infamous example of mercury pollution took place in Minimata, Japan, where the Chisso Chemical company routinely dumped mercury-contaminated waste into the local bay from the 1930s to the 1960s. Fish in Minimata Bay bioaccumulated mercury to levels 40 to 60 times higher than those in nearby ecosystems, and the local community -- in whose diet fish played a key role -- sufferred very high mercury exposures. In the early 1950s, symptoms of chronic mercury poisoning, including neurological toxicity, paralysis, coma, and death began to appear in adults, and a horrifying outbreak of severe birth defects and mental retardation occurred in children. Ultimately, mercury poisoning killed hundreds and

injured over 20,000 people in the area.[47] Chlor-alkali production is not traditionally assumed to have been the source of the Chisso's mercury releases, because the company had been using mercury as a catalyst in fertilizer production since the 1930s. As one history of the event points out, however, Chisso began using the mercury process to make chlorine for PVC plastic in 1952. In 1953, symptoms of mercury poisoning began to appear in the local population, and over the next four years the number of victims correlated with Chisso's growing production volume of vinyl chloride.[48] These facts suggest that mercury releases from the chlor-alkali process are likely to have played at least some role in the Minimata epidemic.

Mercury cells are now banned in Japan and are gradually being phased out in the U.S. and Europe, but releases of mercury remain a problem. In the 1980s, for instance, a major British chlor-alkali facility was found to be discharging up to 100 kilograms per day of mercury into local waterways; more than a decade later, mercury levels in the sediment remained extremely high.[49] In Italy, elevated levels of mercury in air, soil and plant tissues have been found in the vicinity of a mercury-based chlor-alkali plant owned by the Solvay Company.[50] In India, a 1990 study of waterways around a chlorine facility documented severe mercury contamination of fish and sediments.[51]

2.1.3 Energy consumption

Making chlorine requires enormous amounts of energy. Chlor-alkali electrolysis is one of the most energy-intensive industrial processes in the world. The production of one ton of chlorine requires about 3,000 kilowatt-hours of electricity, and the global chlor-alkali industry consumes about 117 billion kilowatt hours of electricity each year.[52] This quantity is about one percent of the world's total demand for electricity, [53] costs about five billion dollars per year, [54] and is equivalent to the annual power production of about 20 medium-sized nuclear power plants.[55] As a major energy consumer, chlorine chemistry contributes considerably to all the environmental problems -- global warming, acid rain, air pollution, generation of radioactive and other wastes from the mining, processing, and consumption of nuclear fuels, and so on -- that are associated with energy production.

Based on an attributable fraction of 40 percent of chlorine demand, the chlorine consumed in the producton of vinyl is associated with electricity consumption of approximately 47 billion kilowatt hours per year. On a per mass basis, production of chlorine to make one ton of PVC consumes about 1,800 kilowatt hours of electricity, based on the fact that pure PVC is 59 percent chlorine by weight;.

Additional energy is consumed in the chemical synthesis of EDC, VCM, and PVC and in the production of additives in the vinyl product. An estimate of the total energy consumption embodied in PVC products is beyond the scope of this document.

2.2 SYNTHESIS OF EDC AND VCM

2.2.1. Releases of EDC and VCM

During the production of feedstocks for PVC, very large quantities of EDC and VCM are released directly into the environment. Because at least 95 percent of the world's annual production of 24 million tons of VCM per year goes into PVC, these releases are almost entirely attributable to vinyl products.

Globally, the PVC industry releases at least 100,000 tons each of EDC and VCM into the air each year, plus over 200 tons of EDC and 20 tons of VCM into surface water.[56] The actual total may be quite a bit higher, because this estimate is extrapolated from emissions at a single facility in Norway: manufacturers in many other nations will have less advanced pollution control equipment and less careful plant operation than this relatively modern and well-regulated facility.

VCM and EDC are not particularly persistent, but they are both highly toxic. Thus, these releases pose the greatest hazards for communities and ecosystems near EDC/VCM manufacturing facilities. The facility in Norway, for instance, releases 40 to 100 tons of EDC each year directly into the local atmosphere.[57] In the U.S., some 12.5 million persons are exposed to EDC emissions from chemical manufacturing facilities, according to the National Institute for Occupational Safety and Health. Workers in plants that manufacture PVC or its feedstocks receive the most severe exposures to these compounds in workplace air; 81,000 U.S. workers are regularly exposed to vinyl chloride, and 77,000 are exposed to EDC.[58]

VCM is a known human carcinogen, and EDC is a probable human carcinogen according to the International Agency for Research on Cancer and theU.S. National Toxicology Program. Studies of workers exposed to VCM have shown an unambiguous increase in liver cancer. Four out of five studies have also reported an increased risk of brain cancer, a result that is statistically significant in a combined analysis.[59] Both EDC and VCM cause a variety of other toxic effects, including liver damage, neurological toxicity, immune suppression, and testicular damage.[60]

Since the carcinogenicity of VCM was established, government regulations have required considerable reductions in worker VCM exposure. In the United States, workplace VCM levels are now required to be maintained within the 1-5 parts per million range, although much higher levels continue to occur in facilities in other nations.[61] For example, a recent survey found that workers in Asia and Eastern Europe are typically exposed to VCM at levels up to 1,000 times these levels. In China, the air in dormitories for workers and their families contains VCM levels that exceed the permissible workplace level in the United States.[62]

Exposure to even the much lower levels in the U.S. industry remains a matter of concern. The mechanisms by which VCM causes cancer has been elucidated, and it is clear that VCM forms covalent bonds with DNA, leading to mutations that abolish natural controls over cell differentiation and proliferation.[63] Currently accepted biological theory indicates that mutations in a single cell can result in the development of a malignant tumor, and a single molecule of a mutagenic substance can initiate carcinogenesis; mutagenic carcinogens like VCM therefore are not likely to have a threshold below which they can cause cancer . That is, any exposure poses some risk of cancer, and the magnitude of the risk rises with the exposure.[64]

There are no studies of the health effects of EDC/VCM exposure on nearby communities. It is clear, however, that severe contamination and environmental and social disruption have occurred in several places. For example, Reveilletown, Louisiana, was once a small African-American community adjacent to a VCM/EDC facility owned by Georgia-Gulf. In the 1980s, after a plume of vinyl chloride in groundwater began to seep under homes in the area, a number of residents began to complain of health problems and brought suit against the company. In 1988, Georgia-Gulf agreed to an out-of-court settlement that provided for the permanent evacuation of the community but sealed the court records and imposed a gag order on the plaintiffs. One-hundred six residents were relocated, and Reveilletown has been demolished, the neighborhood all but wiped off the map.

The next year, as concern over air and groundwater pollution began to grow around Dow Chemical's EDC/VCM facility just five miles away in the small town of Morrisonville, near Plaquemine, Louisiana, Dow began to buy out and relocate citizens there in a "pro-active" program to avoid exposure, liability, and bad press. Morrisonville, too, is now all but abandoned.[65] On the other side of the state, in Lake Charles, Louisiana, PPG and Vista Chemical manufacture EDC and VCM, which now contaminate

water and sediments in the Calcasieu Estuary, along with several by-products of their synthesis.[66] Here, residents continue to occupy their homes, drink local water, and eat fish from the area's polluted bayous. Because many EDC/VCM manufacturing facilities are located in communities with poor and/or minority populations, this stage of the PVC lifecycle has considerable environmental justice impacts.

2.2.2 Formation of organochlorine by-products

EDC/VCM synthesis generates huge quantities of persistent, bioaccumulative by-products. There are two ways that EDC is made: ethylene can be chlorinated with chlorine gas, or it can be oxychlorinated with hydrogen chloride formed as a waste in other synthesis processes. (Almost all EDC producers use both methods in a linked cycle, because chlorination of ethylene generates hydrogen chloride as a by-product, which can then be used in oxychlorination). Both processes yield a complex mixture of reaction products, which are then distilled to yield three batches of materials: the distilled EDC product, the light ends (those substances more volatile than EDC) and the heavy ends, which are less volatile than EDC. The quantity of wastes are quite large -- about two kilograms each of heavy and light ends for each ton of EDC produced. Based on these figures, world EDC synthesis by the oxychlorination process produces at least 30,000 tons per year each of light and heavy ends.[67]

In general, the "heavies" are discarded and the light ends reprocessed in other chemical reactions. The EDC goes on to be pyrolyzed -- heated in the absence of oxygen -- to yield vinyl chloride monomer; by-products of this process include chlorobutadiene, chlorobenzene, chlorinated ethanes, ethylenes, methanes, and large amounts of complex but uncharacterized waste tars.[68] According to one industry source, the total production of chemical wastes produced in the various processes involved in EDC/VCM synthesis is 3 to 10 percent of the VCM yield, or a staggering 570,000 to 1.9 million tons of by-products each year.[69]

The heavy ends contain most of the persistent and toxic by-products. No academic or government studies have sought to identify all the compounds present in these wastes, but there are some data from industry and environmental groups. In 1990, Dow Chemical analyzed its EDC heavy ends and found they were about 65 percent chlorine, including very large quantities of several highly persistent, bioaccumulative and toxic substances: 302 parts per million PCBs, 0.3 percent hexachloroethane (HCE), 1.2 percent hexachlorobutadiene (HCBD), and 30.6 percent unidentified compounds.[70] If Dow's analysis

is representative of heavy ends in general, then EDC oxychlorination results in the worldwide production of a stunning 20,000 pounds of PCBs each year, even though these compounds were banned from intentional production some 20 years ago.[71]

The vinyl industry has claimed that these by-products are "contained" within the production equipment and are never released to the environment. But considerable releases and contamination have clearly occurred. In 1993, chemists from Greenpeace's laboratory at the University of Exeter analyzed material from a number of European EDC/VCM manufacturers. Soil and gravel samples taken near a Swedish oxychlorination reactor contained a wide variety of persistent organochlorines in the high parts per million range, and hexachlorobenzene (HCB) and HCBD were present at the remarkable levels of 1.9 and 0.6 percent by weight.[72] The next year, Greenpeace obtained samples of heavy ends from several U.S. EDC/VCM manufacturers and had them analyzed by the Exeter laboratory. In one sample from Borden Chemical, 174 organochlorines were identified, including a wide variety of highly chlorinated complex aliphatic and aromatic substances.[73].

2.2.3 Formation of dioxins

With PCBs and hexachlorobenzene in the wastes from PVC production, it is no surprise that the structurally related and extremely hazardous dioxins and furans are found in significant quantities, as well. Dioxins have been detected in the wastes from VCM synthesis and in the incineration of wastes from this process,[74] but they are formed in particularly large quantities in the production of EDC by oxychlorination. As the British chemical company ICI made clear in a submission to the government, the formation of dioxin in this process is inevitable and unpreventable:

> It has been known since the publication of a paper in 1989 that these oxychlorination reactions generate polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The reactions include all of the ingredients and conditions necessary to form PCDD/PCDFs, i.e., air or oxygen, a hydrocarbon (ethylene, etc.), chlorine or hydrogen chloride, a copper catalyst, an ideal temperature, and an adequate residence time. It is difficult to see how any of these conditions could be modified so as to prevent PCDD/PCDF formation without seriously impairing the reaction for which the process is designed.[75]

The 1989 paper to which ICI was referring was the work of a group of chemists at the University of Amsterdam, who simulated the oxychlorination process in the laboratory and found dioxin formation at a rate that would this method of producing EDC one of the world's largest sources of dioxin, if not the

largest.[76] This research generated considerable public and scientific concern, so the vinyl industry began its own sampling program. In 1993, the Norwegian PVC manufacturer Norsk-Hydro confirmed that its EDC/VCM synthesis plant produced dioxins but claimed the quantities were hundreds of times lower than the Dutch study had predicted.[77] How much dioxin is actually formed remains uncertain, because both studies have advantages and disadvantages. On one hand, the Dutch analysis may be a more accurate indicator of total dioxin generation, because the researchers were able to capture and analyze *all* the material outputs from the oxychlorination process; the Norwegian report, like any study of a full-scale facility, inevitably missed some of the by-products, which are directed into too many different wastes, products, catalysts, recirculating materials, and equipment surfaces to be completely assessed. On the other hand, the Dutch study was a laboratory simulation and the industry analysis took place at a real production facility, and there may be something about the simulation that caused more dioxin to form than during the real-world synthesis of EDC.

Whatever the exact quantities, there can be no doubt that dioxin generation occurs in amounts that are far from negligible. In 1994, government scientists found dioxins at high concentrations (up to 414 parts ber billion TEQ) in sludges from a fully modernized EDC/VCM plant in Germany, refuting the claim that only outdated EDC/VCM technologies produce dioxin.[78] The same year, ICI Chemicals and Polymers found that its vinyl chloride plant in Runcorn, UK, was producing large quantities of dioxin -- not as much as the Dutch studies predicted but more than Norsk-Hydro had estimated. Most of the dioxins at ICI were deposited in heavy-end wastes, and smaller quantities were released directly into the air and water.[79]

In the U.S., wastes from Vulcan Chemical's EDC plant in Louisiana have been found to contain dioxins and furans at the extraordinary concentration of 6.4 parts per million (TEQ), which makes them among the most dioxin-contaminated wastes ever discovered, on a par with wastes from the manufacture of Agent Orange.[80] In 2000, Norwegian scientists reported finding "extremely high" concentrations of dioxins and furans – 26.6 parts per million (60.7 ppb TEQ) – in the sludges from a VCM/EDC manufacturing plant in that country. Considerable quantities of dioxins and furans had migrated from an on-site disposal facility for these sludges into groundwater, a nearby brook, and the Gulf of Finland in the Baltic Sea. Dioxin and furan levels in several fish species in the area of the plant were 2 to 9 times higher

in fish caught from a relatively uncontaminated local comparison area. Considerable quantities of PCBs were also found in sediments near the plant and were also attributed to the production of EDC/VCM.

These extremely dangerous wastes go to one of two places. In some facilities, they are used in the manufacture of chlorinated solvents, in which case the contaminants end up in the wastes or products from those processes. In others, the wastes are disposed of, usually by incineration (see below). Not all of the by-products of EDC/VCM synthesis end up in the hazardous wastes; some escape directly into the environment. Dioxins have been detected in wastewater discharges and air emissions from a number of EDC/VCM plants,[81] and local and regional contamination of water, sediments, and shellfish has been linked to EDC/VCM manufacturers in both Europe and the United States.[82] For example, severe dioxin contamination of sediments in Italy's Venice Lagoon has been linked to an EDC/VCM manufacturing facility.[83] In the Netherlands, levels of dioxins in sediment samples in the River Rhine jump dramatically just downstream from an EDC/VCM manufacturing plant[84]; the levels are so high, in fact, that the majority of dioxins in Rhine sediments downstream from the plant, all the way to the river's mouth, and in the entire North and Wadden Seas, appear to be attributable to the facility.[85]

These dioxin releases contribute not only to environmental contamination but to human exposure, as well. In Lake Charles, Louisiana, the U.S. Centers for Disease Control has reported that residents in the Mossville area, located across the road from a large vinyl chloride manufacturing facility, have blood levels of dioxins and furans (TEQ) that average about three times greater than those of a comparison population, an increase that was statistically significant at the 95 percent confidence level. Eggs from chickens raised in the area were found to contain dioxins and furans at levels about double those in store-bought eggs, but the sample size was too small for statistically significance to be evaluated. The study did not evaluate specifically whether the increased dioxin levels were due to releases of dioxin from the EDC/VCM synthesis, from on-site incinerators for wastes from this process, or from some other process at facilities in the area.[86]

Other by-products are also present near chemical plants that make PVC feedstocks. In Lake Charles, Louisiana, the National Oceanic and Atmospheric Administration (NOAA) has found very high levels of persistent organochlorines in the water, sediment and fish of bayous near EDC/VCM facilities owned by PPG and Vista Chemical. According to NOAA, the geographical pattern of contamination indicates that PPG is the primary cause of very high levels of organochlorines in the water and sediment.

In one portion of the estuary near PPG's facility, concentrations of HCB, HCBD, and hexachloroethane exceeded 1,000 parts per million in sediment; in some samples, these three by-products represented from 0.1 percent to a mind-boggling 4.8 *percent* of the sediment's total mass.[87]

2.3 DISPOSAL OF EDC/VCM WASTE*S*

The organochlorine-rich heavy ends produced by EDC/VCM manufacture are regulated as a hazardous waste in the United States. The vast majority are disposed of by incineration, usually in on-site furnaces at the production facility. In theory, a properly designed and operated incinerator is intended to convert organochlorines by oxidation into carbon dioxide, water, and hydrogen chloride. Real-world combustion systems, however, never take this reaction to completion for all the compounds fed to them. The majority is completely oxidized, but some fraction escapes unburned, and a larger portion is converted into new organic compounds, called products of incomplete combustion (PICs). According to EPA's technical review document on hazardous waste incineration, "The complete combustion of all hydrocarbons to produce only water and carbon dioxide is theoretical and could occur only under ideal conditions.... Real world combustion systems ... virtually always produce PICs, some of which have been determined to be highly toxic."[88]

By-products form in incinerators for the same reasons they do in chemical manufacturing: multiple reaction pathways, local optima that lead to stable by-products, and deviations from optimal conditions. In incineration, the problems are particularly acute, because wastes are complex mixtures of diverse materials that can never be uniformly blended. Further, combustion is by nature a random process of bond breakage and formation; at high temperaures, most of the molecules will be completely oxidized, but some will follow alternative reaction pathways and emerge as PICs. Transient variations and upsets are a particular problem with incinerators. Good management can reduce but can never eliminate the production of PICs, as EPA's analysis made clear:

> [Deviations from optimum] usually are a consequence of a rapid perturbation in the incinerator resulting from a rapid transient in feed rate or composition, failure to adequately atomize a liquid fuel, excursions in operating temperature, instances where the combustible mixture fraction is outside the range of good operating practice, or inadequate mixing between the combustibles and the oxidant.... The amount and composition of PICs will depend in a complex and unpredictable way on the nature of the perturbation.[89]

The type of incinerator and how well it is operated will affect the *magnitude* of the PICs released, but the production of chlorinated PICs, including the most hazardous ones like the dioxins and furans, is a universal and inevitable outcome whenever chlorinated wastes are burned. As The British Department of the Environment noted, "Comprehensive tests have established that all waste incinerators, independent of type of incinerator or waste composition, are likely to produce all of the possible 75 PCDD and 135 PCDF isomers and congeners, as well as about 400 other organic compounds."[90] By-products form as products of diverse and unpredictable reactions not only in the furance but also in the cooler zones, where control over combustion conditions is virtually irrelevant.[91] Dioxins can even form in the pollution control devices or the smokestack itself, where chlorine gas, organochlorine precursors, or hydrochloric acid come in contact with organic compounds in fly ash. This process, called "de novo dioxin formation" is greatly accelerated if iron or copper catalysts are present, as they are in EDC/VCM wastes and municipal trash.[92]

This means that incinerators not only destroy organochlorines, as they are supposed to, but they also manufacture them. EPA estimates that PICs formed in the incineration process number in the thousands.[93] Of these, a small fraction has been characterized, and the rest remain unidentified. Laboratory tests show that burning methane -- the simplest possible hydrocarbon -- in the presence of a chlorine source produces more than 100 organochlorine PICs; these by-products, ranging from chlorinated methanes to dioxins, are produced by a set of reactions thought to be common to all incineration processes where chlorine is present.[94] It is much more challenging to analyze PICs in the stack gas of real-world incinerators, but over 50 organochlorines or groups of organochlorines have been identified in the emissions of hazardous waste incinerators, ranging from the structurally simple carcinogen carbon tetrachloride to highly persistent and bioaccumulative compounds like chlorinated hexanes, ethers, phenols, naphthalenes, thiophenes, dioxins, furans, and PCBs..[95]

As in other aspects of the vinyl lifecycle, the identified compounds are just the beginning. At hazardous waste incinerators, the most comprehensive research burns have identified about 60 percent of the total mass of unburned hydrocarbons in incinerator stack gases, and most field tests have had far less success in identifying the PICs emitted.[96] There is good reason to be concerned about these mystery compounds, because at least some appear to be in the same toxicological family as the ultra-toxic dioxins. German researchers measured the dioxin-like toxicity of trash incinerator fly ash using a biological test;

they found that the toxicity was up to five times greater than could be accounted for by the amount of dioxins, furans, and PCBs in the ash.[97] The remaining dioxin-like effect was presumably caused by the scores of other compounds -- such as chlorinated naphtalenes, diphenyl ethers, thiophenes, and many more -- that can cause similar health effects but were not specifically measured.

The total quantity of PICs and unburned wastes emitted from incinerators is not known precisely, but it appears to be very large. In the United States, hazardous waste incinerators must pass a trial burn that requires them to demonstrate a destruction and removal efficiency (DRE) of 99.99 percent of the organic compounds fed to them, which means that no more than 0.01 percent of several test chemicals fed into the furnace may be measured in stack emissions. But high DREs do not mean that the environment is protected, for several reasons.

- EDC heavy ends are burned in such huge amounts that even if all incinerators achieved 99.99 percent DRE, they would still emit more than 6600 pounds of unburned hazardous wastes into the air each year in the U.S. alone.[98]

- Considerably greater amounts of organochlorines are released as PICs, because “destruction” means only that the chemical tested was transformed into some substance other than the original compound, and PICs are not counted against the 99.99 percent DRE figure. EPA's Science Advisory Board has estimated that the total quantity of PICs that hazardous waste incinerators emit to the air may be up to one percent of the organic matter fed to them, an estimate that suggests that U.S. incinerators may emit more than a hundred thousand tons of organochlorines into the air each year.[99]

- Still more unburned wastes and PICs are transferred to the land or water where the ash, sludge and effluent from incinerators are disposed of; these quantities are not included in a DRE, which reflects not only destruction of waste chemicals but also their removal by pollution control devices. An incinerator with a filter that captures 95 percent of the dioxin in the stack gas deposits 20 times more dioxin in its ash than it emits to the air, without any effect on the calculated DRE.

- DREs are calculated based on an incinerator’s performance burning test chemicals that are fed in very high concentrations, but two EPA studies have found that substances in low concentrations

burn much less efficiently. Chemicals that are present in wastes in the parts per billion or parts per million range -- such as the dioxins, PCBs, and many other by-products in EDC/VCM wastes -- are subject to destruction efficiencies as low as 99 percent, implying that very significant amounts of these very hazardous substances will escape from incinerators undestroyed.[100]

- DREs measured in trial burns are unlikely to reflect emission rates during routine operation, because trial burns involved the combustion of simplified mixtures of pure chemicals under carefully controlled, closely scrutinized conditions. In daily use, incinerators generally perform less efficiently, due to the complexity of real-world wastes and the frequency of upsets, operator error, and equipment malfunction.[101] Further, the standard trial burn protocol allows the measurement of emissions to stop when the feed of waste chemicals to the incinerator stops, but it is known that emissions can continue for days, resulting in total emissions of unburned wastes that are orders of magnitude greater -- and DREs far lower -- than those measured during the trial burn.[102]

For all these reasons, an incinerator burning EDC/VCM manufacturing wastes may be certified as achieving 99.99 percent DRE when in fact it is emitting huge quantities of unburned and partially burned wastes into the environment.

2.4 POLYMERIZATION, COMPOUNDING, AND MOLDING

Polymerization of VCM to make pure PVC is a more diffusely structured industry than EDC/VCM synthesis, with smaller quantities of product made at a greater number of facilities. No estimates of the total quantity of VCM released into the workplace and the local environment in these stages are available; traditionally, worker exposure in this sector has been assumed to be higher than in any other process.[103] As discussed above, numerous studies of the health of workers in PVC polymerization facilities have been conducted, and they have established a causal connection to angiosarcoma of the liver and have also revealed statistically significant excesses of brain cancer and neurological effects among VCM-exposed workers.

Release of phthalates into the environment and occupational exposure to these substances is another issue in the later manufacturing stages of the vinyl lifecycle. In 1997, the chemical and plastics industries in the United States reported releasing 213,621 pounds of DEHP directly into the air, plus

71,004 pounds into the land.[104] Occupational exposures can be significant: in one plastics molding facility, DEHP levels have been measured at 11,500 nanograms per cubic meter, thousands of times higher than the levels typically found in outdoor air. According to the National Toxicology Program, "workers may be exposed to relatively high concentrations during the compounding of DEHP with PVC resins. The major route of exposure is inhalation. The American Chemistry Council has estimate that phthalate production workers are "generally" exposed to less than 143 micrograms per kilogram of body weight per day of DEHP, and that PVC production workers are exposed to 286 micrograms per kilograms per day, plus approximately equal amounts of BBP.[105]

There are few direct studies of possible health impacts associated with phthalate exposure in the PVC manufacturing industry. Two studies by a research group in Sweden indicate an increased risk of testicular cancer among workers in PVC manufacturing industries. In the first, a variety of occupational exposures were investigated for a possible link to testicular cancer: work in plastics production was associated with a statistically significant 2.9-fold increase in the risk of testicular cancer.[106] The second study followed-up on this finding with the goal of specifically testing and clarifying the relationship between plastics work and testicular cancer. In this report, a case-control study of 163 men with testicular cancer and twice as many men without the condition, occupational work with PVC plastic was associated with 5.6-fold increase in the risk of seminoma -- the form of testicular cancer that occurs later in life and may thus plausibly caused by occupational exposures. The increased risk was statistically significant and was highest among those men with the greatest cumulative exposures. No significant increases were seen among men who worked with other types of plastics. Because exposure to endocrine disrupting compounds – estrogenic substances in particular – can lead to testicular cancer, the authors hypothesized that exposure to phthalates used as plasticizers in PVC may be the specific cause of the increased risks.[107] These results contrast with those of a Danish study that found no relationship between work in cable manufacture – a large consumer of PVC – and testicular cancer. [108]

3. USE OF PVC PRODUCTS

PVC is not bioavailable, so the polymer itself is not toxic during its use. But vinyl products are not pure PVC: they contain both accidental contaminants and chemical modifiers that are added to the plastic on purpose, and some of these may pose health hazards. Moreover, PVC products often encounter reactive conditions -- accidental fires in particular -- which can transform the plastic into very hazardous by-products.

3.1 BY-PRODUCT FORMATION

Some portion of the diverse organochlorines by-products created in the synthesis of EDC/VCM end up in the PVC itself. In May 1994, the Swedish Environmental Protection Agency found that pure PVC plastic from two Swedish producers contained dioxins, furans and PCBs in the low parts per trillion.[109] In 1995, the UK government found dioxins and furans in the same range in PVC food packaging items, including cling film and bottles for oils and beverages.[110] Subsequently, the U.S. Vinyl Institute and the European plastics industries conducted their own studies, both of which identified trace quantities of some dioxin congeners in some samples of PVC plastic.[111] The levels were quite low, but any quantity of dioxin in consumer products is a matter of concern.

3.2 INDOOR AIR QUALITY: RELEASE OF PLASTICIZERS, STABILIZERS, AND OTHER TOXICANTS

3.2.1 Release of phthalate plasticizers

Chemical additives are particularly problematic, because they are present in PVC in very large amounts. PVC additives include a range of toxic compounds, but the most important of these are the phthalate plasticizers and metallic stabilizers. Phthalates can make up a large portion -- up to 60 percent by weight -- of the final vinyl product.[112] Flexible PVC – including flooring and wall coverings – accounts for just over half of all vinyl demand, while the remainder is rigid, unplasticized materials like siding and pipes.[113] Stabilizers, including lead, cadmium, organotins, and other compounds, are used to extend the life of PVC products exposed to light, and they are typically present in lower but still significant concentrations. About 5.4 million tons of phthalates and 156 thousand tons of lead are used in

the worldwide production of PVC every year.[114] Vinyl accounts for more than 90 percent of the total consumption of phthalates, so the health and environmental impacts of phtahatles are overwhelmingly attributable to PVC.[115]

The additives are not chemically bonded to the PVC polymer but are merely mixed into the plastic during its formulation. Over time, they leach out of vinyl products, entering the air, water or other liquids with which the product comes in contact. Most phthalates have low vapor pressures, so they are not expected to volatilize to a considerable degree; despite this prediction, they are responsible for the strong and familiar chemical smell of a new vinyl shower curtain.

Phthalate accumulation also takes place closer to home. When PVC containers and films are used to hold food products, plasticizers migrate out of the plastic and accumulate in foods, especially fatty ones like cheese and meats.[116] The common practice of storing blood and drug formulations in PVC bags causes phthalates to leach into the contents of the bag, which can result in substantial short-term phthalate exposures for the recipient.[117] Newborn infants that receive a single blood transfusion have been found to have extremely high levels of phthalates in their systems.[118] When exposure is repeated, blood levels of phthalates can be 100 to 1000 times greater than "background" and can reach the level at which liver damage and birth defects can occur in animals.[119] Phthalates are also known to be released in significant quantities into saliva when vinyl toys and teethers are sucked by small children.[120]

Of particular relevance to the health and environmental impacts of building materials is the release of phthalates into indoor air from flexible PVC. Although most phthalate plasticizers have low vapor pressure and are not expected to volatilize to a significant degree, it is clear that they are released from vinyl products into the indoor atmosphere. For example, DEHP levels in indoor air typically average 20 to 103 nanograms per cubic meter in indoor air, compared to 0.3 to 4.0 ng/m^3 in outdoor air.[121] As one review concluded, "Phthalates are typically present in indoor air at much higher concentrations than outdoor air due to their high concentrations in consumer products and building materials."[122] According to figures cited by the National Toxicology Program, inhalation accounts for about 15 percent of the average adult's daily intake of DEHP.[123]

The relatively low vapor pressure of most phthalates may contribute to the tendency to find them in much higher concentrations on dust particles than in the vapor phase. One United States study of indoor dust and air samples taken from homes and offices found substantial levels of all phthalates tested.

Levels were highest of DEHP and BBP, which were present in dust at the remarkably high mean levels of 315 and 117 parts per million, respectively.[124] Another recent study of indoor air in Norwegian residences had similar findings, reporting an average of 960 parts per million (including 640 ppm of DEHP and 110 ppm of BBP) on sedimented dust particles and 1180 parts per million (over 0.1 percent) on suspended dust particles.[125]

3.2.2. Asthma and other conditions

The high levels of phthalates in indoor air suggest the possibility that these compounds may contribute to the risk of asthma, which has been steadily increasing, particularly among children in the last 30 years. In 1997, an analysis of phthalate levels in indoor air pointed out that MEHP, the primary metabolite of DEHP, induces bronchial hyperreactivity in rats, presumably by its ability to bind to and activate the receptor for prostaglandin D2, a locally-acting hormone that triggers inflammation. This report concluded, "We propose that the increase in asthma is due to contributory factors of environmental chemicals in general, and specifically DEHP through its primary hydrolysis product MEHP, which affects the bronchial contracting receptors and thereby generates a hyperreactive condition in the lungs. This will increase the risk of a pathological development in addition to aggravation of the effects of other environmental agents."[126]

Two epidemiological studies have tested this hypothesis and found evidence that exposure to PVC in building interiors increases the risk of asthma and related conditions. The first, a case-control study of 251 Norwegian children with bronchial obstruction and an equal number of healthy children for comparison, found that the presence of PVC flooring in the home was associated with a statistically significant 1.89-fold increase in the risk of bronchial obstruction. Further analysis revealed a dose-response relationship between the amount of PVC and other plasticizer-containing materials in the home and the risk of this condition, a finding that increases confidence that the association was not a spurious one.[127]

A larger follow-up study of Finnish children found that children in homes with PVC flooring or wall covering were significantly more likely to suffer from asthma, pneumonia, persistent wheezing, prolonged cough, and phlegm in the airway. The researchers concluded, "Emissions from plastic materials indoors may have adverse effects on the lower respiratory tracts of small children…. Our findings provide additional evidence that indoor plastic materials may emit chemicals that have adverse

effects on the lower respiratory tracts of small children… and warrant further attention to the types of plastic materials used in interior decoration."[128]

A third study focused on the presence of certain breakdown products of DEHP in indoor air. This report, by Swedish researchers, examined the prevalence of symptoms of eye and nasal irritation, as well as biochemical indicators of inflammation and secretion in these tissues, in relation to the presence of 2-ethyl-1-hexanol (EH) in indoor air. The study examined the staff of four nursing homes – three with PVC flooring, and one without. EH is the primary breakdown product of DEHP in damp conditions, which sometimes occur when floors or walls that are covered with an impermeable layer of vinyl become wet. Workers in the two buildings where damp surfaces were covered with PVC were exposed to higher levels of EH and had a significantly increased prevalence of both reported symptoms of nasal and ocular irritation, as well as of the biochemical indicators. Other indoor air factors could not explain the finding, as levels of formaldehyde, molds, bacteria, ozone, and NO_2 were low in all four buildings. The authors concluded, "Emissions related to the degradation of DEHP due to dampness in the floors … may affect the mucous membranes in the eyes and nose, decrease tear film stability and increase the occurrence of ocular and nasal symptoms. The low occurrence of both symptons and signs in the building with special materials and design illustrates that it is possible to construct a new building with a minimum of adverse effects on nasal and ocular membranes."

3.2.3. Toxic mold growth

Vinyl's tendency to trap dampness can create another indoor air problem – the growth of toxic molds. Some molds that produce toxic and/or allergenic products, particularly among sensitive individuals, normally do not normally grow indoors but can grow on persistently damp surfaces that contain nutrients (including sheetrock and gypsum), if they are suitably warm and protected from drying out. Repair of the mildewed material has cost millions of dollars, and liability claims are on the rise for property damage and personal injury caused by mold growing inside buildings –including respiratory problems, skin rashes, headaches, lung disease, memory loss, and brain damage -- according to a recent report for Lawyer's Weekly, a legal industry publication.[129] Vinyl wallcoverings, because they do not breathe, are said to be the major cause of mold and mildew in interiors, according to several building industry sources.[130] The vinyl industry confirms that vinyl wall coverings have created this situation in many buildings, because PVC acts as "a vapor barrier that traps moisture inside the wall cavity, where it

condenses against the relatively cool inside surface of the wall. Prolonged exposure to these conditions will result in deterioration of the gypsum board."[131] The industry suggests that use of permeable membranes on the outside wall part of the cavity and prevention of moisture infiltration can help reduce the risk of mildew growth.[132] Because dampness and condensation can occur inside vinyl-sealed walls due to temperature and humidity differentials produced by HVAC systems, however, at least one authoritative building industry source recommends avoiding vinyl wallcoverings altogether to prevent mold and mildew growth.[133]

3.2.4. Releases of lead and other stabilizers

Metal stabilizers are also released from PVC products. Significant releases of lead have been documented from PVC window blinds,[134] and lead is also known to leach into water carried in PVC pipes that contain lead stabilizers.[135] These findings led the U.S. Consumer Product Safety Commission to warn consumers in 1996 to avoid miniblinds with lead additives.

But lead continues to be used in building-related materials, as do other hazardous additives. Lead stabilizers are commonly used in vinyl cables, window profiles, and pipes, although their use is greater in Europe than in the United States.[136] For example, lead remains the most common stabilizer in PVC products in the European Union – with consumption of over 51,000 tons per year. Lead is well-known for its clear link to impaired cognitive development in children, even at very low doses. Cadmium, another potent neurotoxin, is also used in window frames, although its use is declining. Finally, considerable quantities of organic tin compounds (about 15,000 tons per year in the EU) are used as PVC stabilizers – particular in rigid films, roofing materals, and clear rigid construction sheeting.[137] Organotins are estrogenic compounds that have been linked to widespread anomalies of reproductive development in wildlife.[138]

3.3 ACCIDENTAL COMBUSTION

Another major hazard associated with the use of PVC products is the possibility of fire. PVC is ubiquitous in modern buildings and vehicles. When vinyl burns, the primary combustion products are carbon dioxide, water, and hydrochloric acid. In several major fires, hydrochloric acid has caused severe burns to skin, eyes, and lungs and is an important cause of toxicity to firefighters and persons exposed to fire fumes and smoke. It can also cause severe damage to computers and other equipment.[139]

The hazards of PVC in fires have prompted action or positions by a number of expert organizations. The International Association of Firefighters has stated, "Due to the intrinsic hazards, we support efforts to identify and use alternative building materials that do not pose as much risk as PVC to firefighters, building occupants or communities."[140] In the United Kingdom, the Fire Brigades Union (FBU) has stated, "The FBU is now particularly concerned about the safety of PVC based building materials that are used in the construction and fitting out of buildings when involved in fire."[141] And the United States Navy has adopted specifications to substitute polyolefins for PVC-jacketed cables due to concern about hazards in the event of fire.[142]

Accidental fires represent particularly poor combustion conditions, so substantial amounts of dioxin and other organochlorines form as products of incomplete combustion in a vinyl fire.[143] Indeed, the combustion conditions in an accidental fire, where gases do not mix thoroughly, and materials cool rapidly as they escape from the flame -- are considered optimal for the rapid production of dioxins.[144] As a result, all accidental fires in modern buildings are likely to generate dioxins and other persistent, bioaccumulative organochlorines. For example, after a fire in a German kindergarten that contained substantial quantities of PVC, scientists measured dioxin levels in indoor soot at concentrations of 45,000 parts per trillion (TEQ) -- almost 300 times greater than the German government's health safety standard. This situation required the building's interior to be completely stripped -- of all floors, ceilings, wall coverings, furnishings, and so on -- sandblasted, and remediated by hazardous waste experts before children were allowed to enter again.[145] Dioxins have also been identified in the residues from burning automobiles, subway cars, and railway coaches.[146]

Even a very small amount of dioxin from each of the 621,000 structural fires and 421,000 vehicle fires in the U.S. every year could make a substantial contribution to dioxin contamination of the environment.[147] The German EPA and the German Environment Ministers have called for the use of substitutes for PVC in all areas susceptible to fire, but PVC use in construction continues to grow on a global basis.[148] As a result, a stockpile of PVC, waiting to burn, is accumulating in staggering quantities. Worldwide, over 400 million tons of PVC are "in stock" -- that is, in use in various applications, mostly construction-related, and susceptible to fire at some point.[149]

While many small fires taken together may constitute an important source of organochlorines, a single fire at a PVC factory, warehouse or disposal site can create very large quantities of pollutants. A

home contains at most a few hundred kilograms of PVC[150], but a warehouse or landfill may have hundreds of tons on-site. After a fire at a plastics warehouse in Binghamton, New York, for example, dioxin levels in soil on the site were found to be over 100 times greater than other samples from the same community.[151] According to the European Commission, fires are estimated to account for 6.6 percent of all dioxin emissions from identified sources (table 3).

PVC fires not only create dioxins and other organochlorines, they also release additives held in the plastic. The world stock of PVC in use contains a staggering 3.2 million tons of lead and 83 million tons of phthalates.[152] Lead cannot be destroyed by combustion, so accidental fires represent a major potential source of lead exposure, a hazard which looms larger as more and more PVC accumulates worldwide in building applications.

4. DISPOSAL OF PVC PRODUCTS

The final stage of PVC's lifecycle creates the most severe environmental hazards. About 30 to 50 percent of the vinyl produced annually -- some 8 to 12 million tons per year worldwide -- ends up in the trash stream.[153] Although building materials have a relatively long lifetime, significant quantities of vinyl are disposed of as cutaways in preconsumer waste and, ultimately, in demolition wastes when a product's useful lifetime ends. Construction products are often thought of as a "long-life" sector of PVC use, but vinyl products in commercial interiors, which are often renovated well before their components are physically spent, have relatively short lifetimes.

4.1 RECYCLING

One thing is true everywhere: very little postconsumer PVC is recycled. A substantial portion of preconsumer PVC – scraps and cuttings from manufacturing stages – is recycled, but the quantities of preconsumer waste represent a small fraction of the PVC waste stream. Recycling postconsumer PVC is extremely difficult because vinyl products are mixtures of PVC and additives, and each specific formulation is uniquely suited to its application. In virtually all postconsumer recycling, many formulations are mixed together, which destroys the special properties of each one. This means that recycled postconsumer PVC is always of lower quality than the original material, so it can only be used only in products without strict material requirements, such as fence posts and speed bumps.[154]

The American Association of Postconsumer Plastics Recyclers announced in 1998 that its attempts to recycle PVC had failed and that it would henceforth view vinyl products as unrecyclable contaminants in the municipal waste stream.[155]

Since recycled PVC is almost never used to make a new version of the original product, it is not recycling at all but is more accurately called "down-cycling."[156] An example of true recycling is the reprocessing of paper: the old fibers are used to make new paper products, and a new tree does not need to be cut down. In contrast, a new vinyl wall covering or floor tile must be made of new plastic. In this way, down-cycling does not reduce the amount of PVC produced each year or the total quantity of PVC building up on the planet. The illusion of recycling actually increases the global PVC burden by finding

new uses for old PVC while creating a positive image for a product that can be neither safely disposed of nor truly recycled. As the European Commission put it, while true recycling has obvious environmental benefits, "the environmental advantages of the down-cycling of mixed plastics for the production of products which substitute concrete, wood, or other non-plastic applications are less certain."[157]

In the whole of the European Union, less than 3 percent of postconsumer PVC waste is recycled, the majority of which is actually downcycling of cable and packaging wastes. According to a 2000 report by the European Commission, "high-quality mechanical recycling for post-consumer [vinyl] wastes is still in a preliminary stage and exists only for a few product groups and with low quantities" that represent only about 0.3 percent of the total mass of postconsumer vinyl wastes.[158] Sweden, a nation with an ambitious and effecting recycling program, has a total PVC recycling rate of just 2 percent in 1999, virtually all of it preconsumer waste.[159] The EC projects that only 9 percent of all PVC waste is likely to be recycled by 2020, with a maximum potential of no more than 18 percent.[160] Such low recycling rates, even with time to develop an ambitious program, indicate that PVC is not and cannot be a green building material.

There are also concerns about the environmental hazards of PVC recycling. Mechanical recycling of PVC can release additives, including phthalates and stabilizers, which are then dispersed into the recycled products, into the environment or, if they are captured, disposed of on land or in incinerators. The European Commission has recognized significant concerns about the presence of lead and cadmium stabilizers in PVC products that are recycled and their subsequent dispersal into a greater range of consumer products.[161]

4.2 LAND DISPOSAL

A significant portion of the thrown-away PVC goes to landfills, and almost all the rest is burned; the exact proportions going to land disposal and incineration vary from one country to another. In landfills, there are two concerns about PVC disposal. One is the persistence of PVC, which will typically last for centuries in a landfill, presenting a significant burden in terms of the demand for landfill space. Second is the release of additives in the plastic into groundwater. Because phthalates are not chemically bonded to the polymer, they can leach out of disposed products into landfill leachate, where they will eventually contaminated groundwater.[162]

Finally, fires can occur during or after the disposal process, releasing hazardous substances into the air, including dioxins. In Hamilton, Ontario, for example, after some 200 tons of PVC burned at a plastics recycling facility, samples of soot, ash and tree leaves from the fire area contained greatly elevated quantities of dioxins.[163] Of particular concern are landfill fires, which occur with some regulatory at landfills and waste storage sites where large quantities of PVC are present. Data on dioxin releases from landfill fires are limited, but EPA has estimated that they may be a major source of dioxin emissions in the United States.[164]

4.3 INCINERATION

4.3.1 The role of PVC in incinerators

In every inventory of dioxin sources in the world, trash incinerators and other combustion sources account for the majority of identified dioxin releases into the environment (table 3), and PVC is the predominant source of dioxin-generating chlorine in these facilities. In municipal waste incinerators, PVC contributes at least 80 percent of the organically-bound chlorine and 50 to 67 percent of the total chlorine (organochlorines plus inorganic chloride) in the waste stream, although it makes up only about 0.5 percent of the trash stream by weight.[165] In the U.S., an estimated 200,000 to 300,000 tons of PVC is incinerated in trash burners every year.[166] Large quantities of PVC also go to medical waste incinerators, where it accounts for 5 to 18 percent of the waste stream,[167] over 90 percent of the organic chlorine, and over 80 percent of the total chlorine content of medical waste.[168]

There is no doubt that burning vinyl is a source of dioxin, because numerous laboratory combustion tests involving pure PVC (or pure PVC in the presence of metal catalysts) produce considerable amounts of dioxin.[169] No one has attempted to identify the full range of by-products that form when PVC burns, but 45 organochlorines -- including persistent and toxic chlorinated benzenes, styrenes, phenols, naphthalenes, PCBs, and PCDFs -- have been found in the combustion products when the closely related plastic polyvinylidene chloride (PVDC, commonly known as Saran Wrap) is incinerated.[170]

Table 3. Inventoried dioxin sources in North America, Europe, and the world

	percent of all releases from inventoried sources				
Source type	U.S. [1]	EU[2]	U.S.[3]	Great Lakes[4]	World[5]
* Municpal waste incinerators	40.1	25.1	51.4	20.1	37.6
Ferrous metals production	NA	21.1	0.8	10.6	11.7
* Copper smelting	19.7	1.3	2.6	4.1	2.6
* Medical waste incinerators	17.4	14.2	10.3	48.7	2.8
Forest, brush, straw fires	7.6	NA	7.7	0	11.7
* Accidental fires	NA	6.6	3.7	NA	NA
Wood and coal combustion	6.0	17.1	5.9	4.4	NA
Hazardous waste incineration	5.7	0.6	2.6	8.0	22.7
Dioxin-contaminated chemicals	NA	6.6	4.7	NA	NA
* Uncontrolled trash incineration	NA	3.0	NA	NA	NA
Automobile fuels	1.4	1.9	0.4	1.4	0.4
Cement kilns (no hazardous waste)	0.6	0.4	NA	2.0	10.7

* Dioxin sources in which PVC is a major chlorine donor. NA = quantitative estimate not available. There are numerous additional dioxin sources for which none of the inventories made a quantitative estimate, due to inadequate data. Sources are listed from largest to smallest, by the percent contribution in EPA's inventory, except for sources with NA in that column, which were ordered according to their contribution in the EU inventory. Sources with less than 1 percent contribution in all inventories are not shown. References: 1. U.S. EPA 1998. Percent of all identified releases to air of PCDD/F (TEQ), based on median estimates for year 1995. Hazardous waste incineration estimate includes releases from cement kilns that burn hazardous wastes, as well as boilers and industrial furnaces. 2. A Hanberg. Compilation of EU Dioxin Exposure and Health Data. Oxfordshire: AEA Technology plc and European Commission DG Environment, 1999. Percent of all identified releases to air in the European Union. 3. Thomas and Spiro 1995. Percent of identified emissions of total PCDD/F to the air in the U.S. as of 1989 Municipal waste incinerators includes apartment incinerators. Accidental fires include structural fires, PCB fires, and PCP fires. 4. Commoner *et al.* 1995. Percent of identified emissions of PCDD/F (TEQ) to the air that reach the Great Lakes. 5. Brzuzy and Hites 1996a. Percent of identified PCDD/F releases (TEQ) to the air. Estimate for hazardous waste incineration includes cement kilns burning hazardous waste; estimate for cement kilns does not. Estimate for forest, brush, straw fires includes all biomass combustion, including wood.

As in many other processes, the identified compounds are just the beginning. In municipal incinerators, the most thorough analysis to date identified several hundred PICs, including 38 organochlorines -- chlorinated benzenes, PCBs, methanes, ethylenes, and others -- but 58 percent of the

total mass of PICs remained unidentified.[171] As noted above, a considerable portion of these mystery compounds are likely to be hazardous, and at least some are known to cause dioxin-like toxicity.

Incinerators also releases additives contained in PVC products into the environment. Over 45,000 tons of lead stabilizers in PVC enter the world's municipal trash each year.[172] Because lead cannot be destroyed by incineration, all the lead that enters an incinerator ultimately enters the environment, via stack emissions, ash, scrubber effluent, or wastewater sludges. Incinerators are now the largest source of lead emissions to the environment, and PVC is responsible for about 20 percent of the lead in the waste stream, according to Swedish figures.[173] PVC also accounts for about 10 percent of the cadmium in waste incinerators.[174] Vinyl thus appears to be a major cause not only of dioxin but of heavy metal pollution, as well.

Not all burning of vinyl takes place in high-tech incinerators. In developing countries and rural areas of industrial nations, open burning of waste is a common way to get rid of trash and debris. A recent study by U.S. EPA and the New York Department of Environmental Conservation indicates that "backyard burning" of trash in barrels can result in massive emissions of toxic chemicals, including chlorinated methanes, benzenes, phenols, and dioxins and furans. Emissions of dioxins and furans per pound of waste burned were 12,000 to 75,000 times higher than emissions from an optimally operated modern trash incinerator; EPA estimated that open burning at just two to three households would produce as much dioxin as an incinerator large enough to serve about 40,000 people.[175] Further, when more PVC was burned, average releases of all chlorinated PICs, including the dioxins, rose substantially, although the experiment did not include enough replications for the statistical significance of the increase to be evaluated. Although it is unlikely that construction or demolition waste from commercial buildings will be disposed of by uncontrolled burning, materials used in residential construction in rural areas and developing countries may be. These findings suggest that the rapidly expanding use of vinyl in developing countries, where expensive means of waste management are not available, has the potential to cause a huge increase in worldwide emissions of dioxins.

Some spent metal products that contain vinyl, including materials used in buildings such as cables and electronics equipment, are recycled or re-processed in smelters, and these facilities are also major dioxin sources. Secondary copper smelters, for example, recover copper from PVC-coated wire and cable and PVC-containing telephone cases; very high dioxin emissions have been measured at these facilities,

which are considered major dioxin sources in most inventories.[176] Most importantly, removing some of the vinyl sheathing before cables are fed to the smelters reduces dioxin emissions considerably.[177] Secondary steel smelters have also been found to emit very large quantities of dioxin, primarily because they recover metal from scrap automobiles that contain PVC.[178] Secondary lead smelters release dioxin and other organochlorines, too, due to the feed of lead automobile batteries with internal PVC separators. In the U.S., however, PVC has been recently phased out of this application, so EPA no longer considers lead smelters an important dioxin source.[179]

As table 3 shows, PVC is the primary or an important source of chlorine – and therefore of dioxin formation – in many of the major world's dioxin sources that have been identified to date. In fact, sources in which PVC is the dominant chlorine donor account for 77 and 50 percent of all inventoried dioxin emissions in the U.S. and Europe, respectively.

4.3.2 Dioxin Formation and PVC – Evidence from Combustion Experiments

Following an aggressive effort by the chemical and plastics industry, an apparent controversy has developed over whether burning PVC in incinerators results in increased dioxin emissions. The data, however, give very strong support to the view that dioxin forms when PVC and other organochlorines burn, and further that burning more PVC (or other organochlorines) results in the formation of more dioxin. This is not to say that the organochlorine content of the waste is the *only* factor involved in dioxin formation; facility design, operating conditions, and the presence of catalysts also play major roles. Chlorine is a requirement for dioxin synthesis, and preventing the introduction of organochlorines into incinerators is the best means to prevent dioxin formation. Further, because burning PVC is known to produce dioxin, a logical implication is that burning more PVC will produce more dioxin, and burning less PVC will reduce dioxin generation.

Dioxin cannot be formed without a chlorine source, so emissions from incinerators must be due to burning organochlorines, burning salt, or some combination of the two. To suggest that organochlorines are not important dioxin precursors requires the combustion of inorganic chloride salts to be the dominant source of dioxin. But several lines of evidence indicate that organochlorines (particularly PVC), not salt, are primarily responsible for dioxin emissions from combustors.

The first line of evidence comes from numerous well-conducted studies in the laboratory. Results from the laboratory are particularly convincing, because -- unlike trial burns at full-scale incinerators -- they allow combustion conditions, input materials, and emissions to be carefully controlled and accurately monitored. Studies of this type indicate that burning PVC is clearly an important dioxin source – far more so than the burning of salt:

- The German EPA has found that burning PVC or other organochlorines produces dioxin -- with concentrations in ash residues ranging from 3.2 to 662 parts per trillion (TEQ) -- but combustion of several types of organochlorine-free but chloride-containing paper, wood, cotton, or wool does not produce dioxin above the detection limit of 0.1 parts per trillion[180] (table 4).

- Two separate studies by Danish researchers have also found that burning pure PVC produces dioxin. Under some conditions, the quantities formed are quite considerable.[181][182]

- A 2000 report by Japanese researchers found that adding four percent PVC to a mixture of chlorine-free materials in a lab-scale incinerator had an "intense effect in dioxin emissions" – a more than 10-fold increase – but adding an equal quantity of salt caused at most a two-fold increase.

- When PVC is added to a mixture of chloride-containing coal and bark, dioxin concentrations in the residues increase by a factor of 10 to 100; the more PVC added, the higher the dioxin concentration.[183]

- Adding PVC during combustion of natural chloride-containing wood products increases dioxin levels in the ash by 15 to 2400 times; when large quantities of inorganic chloride chemical hardeners are added, dioxin levels rise somewhat, but are still 3 to 350 times lower than when PVC is included in the mix.[184]

- Combustion of a mixture of coal and salt produces trace quantities of dioxins and furans in the off-gas, but when elemental chlorine is added to the mix, total dioxin formation increases 130-fold.[185]

Table 4. Dioxins in ash from burning organochlorines and chloride-containing materials

Material	Total PCDD/F (ppt)	TEQ
Materials not known to contain organochlorines		
Writing paper	ND	ND
Wood	ND	ND
Cotton	ND	ND
Wool	ND	ND
Polyethylene	2.9	<0.1
Acrylonitrile-butadiene rubber	2	<0.1
Fir wood	21.4	0.65
Organochlorine plastics		
PVC plastic (pure)	244 - 2,067	3.2 - 42.2
PVC flooring material	352 - 1,847	8.2 - 14.5
PVC window frame material	7.5 - 969	8.8 - 18.1
PVC cables (copper)	669 - 2,670	11.4 - 52.6
PVC cables (no copper)	416 - 843	7.4 - 16.6
PVC gloves, hose, pipes, tape	158 - 954	2.5 - 16.5
PVDC plastic	3,304	14.1
Chloropolyethylene plastic	840	10
Polychlorobutadiene (neoprene) plastic	323 - 1,096	0.7 - 4.7
Materials containing other organochlorines		
Bleached coffee filters	6.3 - 7.7	0.15 - 0.23
Chloroparaffins	1,049	5.3
Dichloromethane	26,302	478
1,1,1-Trichloroethane	21,746	340
Tetrachloroethane	9,072	132
Trichloroethylene	120,915	149.5
Perchloroethylene	212	0.4
Epichlorohydrin	1,532	36
Chlorobenzene	16,135	0.5
p-Chloronitrobenzene	190,096	21.5
o-Chloronitrobenzene	32,293	216
p-Chlorotoluene	1,033	ND
2,4-D	178,016	361
Linuron pesticide	3,110	32

Source: Theisen 1991.

- Burning chloride-containing vegetable matter does not produce detectable PCDD/Fs, but including PVC or chlorine gas along with the plant material does.[186]

- The same pattern exists for other organochlorines. Finnish researchers have found that burning perchloroethylene in a laboratory combustion reactor produces orders of magnitude more dioxins, chlorophenols, and chlorobenzenes than burning sodium chloride does.[187] An American study found that formation of dioxin precursors rises as the proportion of organochlorines in the waste increases,[188] while three others have found that adding salt to a combustion reaction has no detectable effect on dioxin formation.[189]

In full-scale or pilot-scale incinerators (units smaller than commercial burners but similar in design), the evidence also supports a relationship between burning organochlorines and creating dioxin, but there are some contradictory studies, presumably due to the complexity of analyzing input and output streams and adjusting for fluctuating conditions:

- The Danish EPA, for example, has found that doubling the PVC content of an incinerator's waste input increases dioxin emissions by 32 percent, while doubling the chloride content increases dioxin emissions by a much smaller margin.[190]

- A team of Japanese researchers reported this year on two separate sets of experiments that showed burning a mixture of PVC and polyethylene – in which PVC is the only chlorine source – produces substantial quantities of dioxin.[191]

- Two groups of Finnish researchers have found that dioxin levels in stack gas or fly ash are very low when a mixture of coal and chlorine-free plastics is burned, but they rise substantially when PVC is added to the mix.[192]

- A 1996 study for the Dutch Environment Ministry reported that when both PVC and chloride-containing compostable matter are removed from municipal waste, emissions of chlorophenols, which are indicator compounds for dioxin formation, were extremely low. When 20 percent of the original amount of compostables was added back into the mix, emissions did not increase, but when 30 percent of the original amount of PVC was added along with the compostables, chlorophenol emissions approximately doubled.[193]

- A series of studies at a pilot-scale incinerator at the University of Florida has documented a clear relationship between the feed of PVC and the emission of chlorophenols. The authors summed up their findings: "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."[194]

- German scientists have found that removing PVC sheathings from copper cables before they are recycled in copper smelters causes dioxin emissions to drop precipitously.[195]

- Four studies have found that the addition of PVC-containing "refuse-derived fuel" to incinerators burning salt-containing organic matter like wood chips or peat results in significant increases in dioxin formation.[196]

- Adding PVC to a mixture of chlorine-free matter in a pilot-scale incinerator increased dioxin emissions substantially; when more PVC is added, more dioxin is formed.[197]

In some of these studies, a relationship was seen in the air emissions but not in the fly ash, or vice versa, reinforcing the difficulty of establishing statistically significant relationships in the complex context of burning real wastes in large incinerators.

There are a few studies with more ambiguous results, but they do not refute the link between burning organochlorines and generating dioxin. For example, a recent Swedish investigation found that dioxin formation is directly related to chlorine content, but only when chlorine levels in the fuel exceed 0.5 percent, as they do in most modern waste streams. Changes in chlorine content below this level had no statistically significant effect on dioxin emissions.[198] These results could indicate that there is a threshold below which chlorine has no impact on dioxin levels, but it is equally possible that the failure to find a correlation at low chlorine levels is an artifact of the limits of chemical and statistical analysis: as levels of both chlorine and dioxin decrease, measurement error and statistical fluctuations become more and more important, swamping a fading signal under a growing chorus of noise.

Unlike most of the studies discussed above, this report found that it did not matter whether the chlorine came in organic or inorganic form -- both gave rise to dioxin in approximately equal amounts. One of the Japanese reports also found that large quantities of salt could result in substantial dioxin emissions.[199] This finding, which contradicts the weight of evidence from the studies discussed above, should be investigated further, but from a policy perspective it does not really matter if it is right or wrong. If the many other studies are correct that chloride salts result in minimal dioxin emissions, then dioxin output depends on the organochlorine content of the waste: lowering the input of organochlorines is necessary to reduce the formation of dioxin. If, on the other hand, the Swedish study is correct, then dioxin generation depends on the waste's total level of chlorine (organic plus inorganic); lowering the quantity of organochlorines in the waste will reduce the total chlorine level and will again cause dioxin formation to fall. Either way, if we want to prevent dioxin formation in incinerators, we need to stop burning organochlorines.

Two widely cited studies, one by the New York Department of Environmental Conservation[200] and the other by the European plastics industry,[201] have come to the opposite conclusion, finding no relationship of dioxin emissions at individual trash incinerators with PVC content of the waste burned. Neither of these investigations controlled or adjusted for other factors that affect dioxin formation, including facility type, operating conditions, or other characteristic of the waste feed. This oversight will radically weaken any study's power to detect a potential relationship between PVC and dioxin formation. Indeed, an EPA reanalysis of the data from the New York study found that when combustion conditions were adjusted for, emissions of dioxins and furans increased as PVC content of the waste rose.[202]

Another study has been widely publicized as having refuted a relationship between burning organochlorines and dioxin emissions. This study,[203] promoted by the industry as a report by the American Society of Mechanical Engineers (ASME) was in fact written by a private consulting firm and was conceived, funded, and sponsored by the U.S. Vinyl Institute.[204] The so-called ASME report was not an empirical test of the relationship between burning organochlorines and making dioxin but a statistical analysis of a large quantity of non-comparable, uncontrolled, pre-existing data from many different facilities. None of these data were gathered for the purpose of examining a link between organochlorines and dioxin. This study has been soundly criticized for using inappropriate surrogate measures instead of direct measurements of organochlorine feed and dioxin emissions, and for failing to control or adjust for

other factors that affect dioxin formation. There is therefore no way to tell whether the study's failure to find a relationship between dioxin and organochlorine content of wastes was due to inadequate design or a real lack of relationship.[205]

4.3.3. PVC and dioxin – historical evidence

The theory that burning organochlorines like PVC is an insignificant dioxin source and salt is responsible for incinerator emissions of dioxin leads to several specific predictions, none of which turn out to be true. First, if salt is a more important dioxin source than burning organochlorines, forest fires should result in large dioxin releases, because plant matter is rich in salt. According to research by chemists at Indiana University, however, dioxin levels in the sediment of a U.S. lake, the watershed of which suffered a major forest fire in 1937, show no change whatsoever around or after the time of the fire.[206] More recently, Spanish scientists analyzed samples of salt-containing vegetation and soil that burned in four different 1998 forest fires; the burned materials showed no increase in dioxin levels compared to background, leading the researchers to conclude, "Natural fires seem not to be an important source of dioxin-like compounds."[207]

The second prediction implied by industry's theory is that historical levels of dioxin should track trends in the burning of salt, not the production and incineration of organochlorines. But they do precisely the opposite. Several studies have analyzed the dioxin and furan content of mummified and frozen remains of people several hundred to several thousand years old, including individuals from cultures that cooked over indoor fires and were exposed to considerable amounts of combustion emissions. All these studies have found that dioxin levels (measured as TCDD-equivalents) in ancient tissues were no more than one to two percent of the amount found in modern humans, and even this could represent contaminants deposited in the samples in modern times, especially during handling and analysis.[208]

Dioxin levels in sediment cores from lakes and seas in North America and Europe also indicate that organochlorines and not the burning of salt are responsible for the bulk of dioxin emissions (figure 3). Every study conducted to date shows that dioxin levels were extremely low before the twentieth century (when chlorine and organochlorine production began), despite the fact that natural and industrial

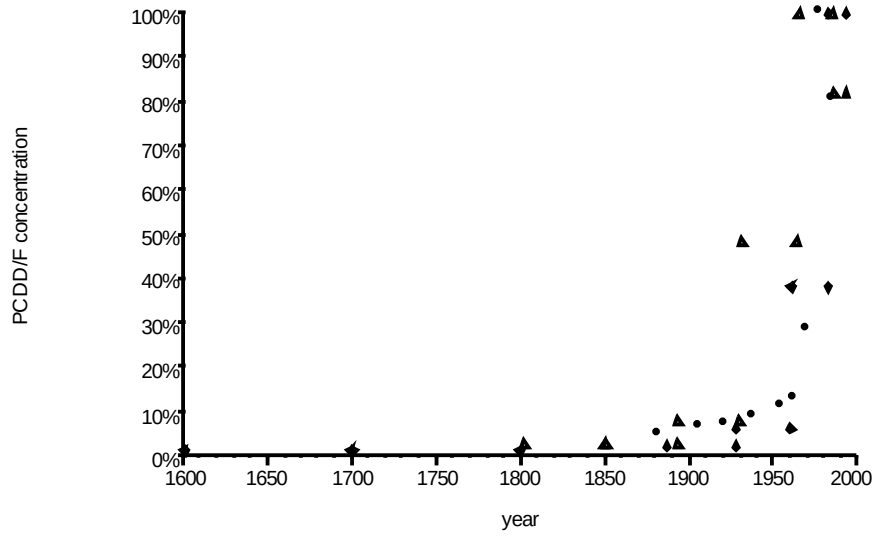


Figure 3. Dioxin deposition in European sediments. The vertical axis shows concentrations of total dioxins and furans in sediment cores from the Baltic (circles) and two German lakes -- the Wildsee (triangles) and the Herrenweiser See (squares), expressed as a percentage of the highest levels measured in each location. In all locations, levels were extremely low prior to the advent of chlorine chemistry, and they rise rapidly thereafter. Sources: Juttner et al 1997, Kjeller and Rappe 1995.

---

combustion processes were abundant in this period. Sediments in Swedish lakes show no measurable dioxin before 1945,[209] and those in the Great Lakes show none before 1920.[210] In the Baltic, dioxins and furans were present in a sediment sample dated to 1882, but the levels were 20 times lower than the peak concentrations in 1978.[211] A study of two lakes in Germany's Black Forest found that sediments from the seventeenth and eighteenth centuries contained very small quantities of dioxins and furans -- 77 and 34 times lower than the maximum concentrations from this century. Expressed as TCDD- equivalents, the ratios were even higher: 310 and 90 times greater in modern than pre-chlorine sediments.[212] In New York's Green Lake, very small quantities of dioxins and furans are present in layers from the late 1800s, but at concentrations 1500 times lower than those found in the 1960s.[213]

Only with the advent of chlorine chemistry and the incineration of its products and by-products did dioxin levels begin to rise. In all samples, dioxin concentrations began to increase slowly in the early decades of this century, and they shot up rapidly from the 1940s to the 1970s, rising 25-fold or more between 1935 and 1970.[214] This pattern is consistent with the rise of chlorine chemistry, peaking in the

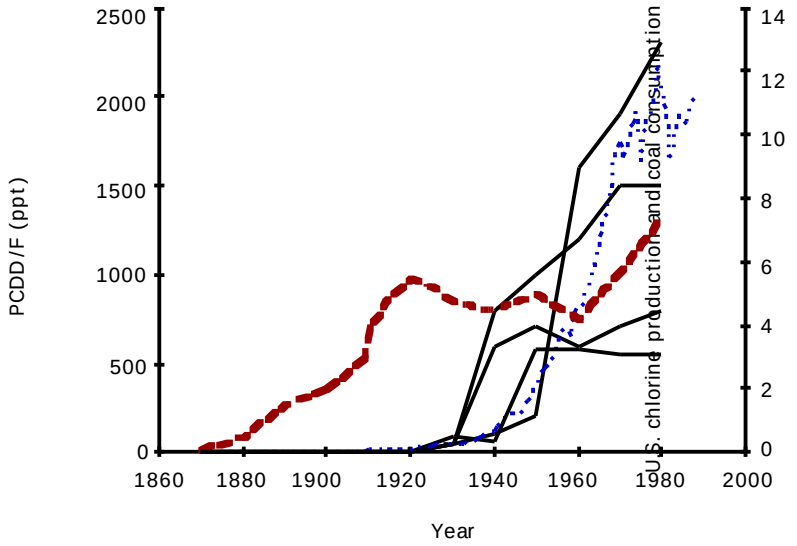
1960s or 1970s and declining somewhat thereafter, as restrictions on dioxin-contaminated pesticides and chlorinated gasoline additives went into effect.

These trends do not in any way track the history of combustion of salt, either industrial or natural. One specific study of dioxin trends in Great Lakes sediments found that dioxin levels do not follow trends in combustion of coal, which was practiced on a massive scale long before dioxin concentrations began to rise, but they do correspond quite closely to the rise of the chlorine chemical industry (Figure 4). These results suggest that industrial combustion processes -- including coal-fired power plants, steel mills, rail engines, furnaces for heating, and other industries powered by coal, which contains chloride salts -- have never been major sources of dioxin, either. The authors of the Great Lakes studies summarized their results so succinctly that they are worth quoting at length:

> 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 quantitation 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.[215]

If organochlorines have nothing to do with dioxin emissions, then why were dioxin levels in the environment non-existent or miniscule before the chemical industry began to produce them? In particular, why were dioxin levels so low during the nineteenth century, when combustion of chloride-containing materials like coal and wood was at its peak? These data make abundantly clear that burning salt is a very minor source of dioxin in the environment, virtually all of which is due to the production, use, and disposal of chlorine gas and organochlorines.

It is therefore possible that some dioxin can be formed by the combustion of chloride-containing salts, but the available evidence indicates clearly that industrially-produced materials containing organochlorines, PVC in particular, are the predominant causes of dioxin generation in incinerators. Even more importantly, they are the most readily preventable cause of dioxin formation: salts are naturally ubiquitous, but we can choose to stop producing, using, and burning organochlorines. As the Danish Technical Institute 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."[216]

Figure 4. Dioxin deposition to Great Lakes sediments. Solid lines show levels of total dioxins and furans in four sediment cores from Lake Huron; the thin dotted line shows U.S. chlorine production capacity (in millions of short tons per year); the thick dashed line shows U.S. coal combustion (in 100 millions of short tons per year). Dioxin levels were low or zero when coal combustion was at its peak, increasing only with the growth of chlorine chemistry. Sources: dioxin and coal redrawn from Czuczwa and Hites 1984; chlorine from Chlorine Institute 1991, Leder et al 1994.

In summary, PVC is the major chlorine source in the majority of the combustion facilities that dominate inventories of dioxin sources. The production and use of PVC also contributes to dioxin pollution. It therefore appears that PVC is responsible for more dioxin generation than any other single product. As more and more vinyl installed in buildings over the preceding decades enters the waste stream for disposal, the potential for dioxin generation grows accordingly. Any program to eliminate dioxin generation at the source -- a public health imperative, as discussed in section 5.3 below – should include provisions to reduce the use of PVC in applications susceptible to accidental fire or disposal by combustion.

5. BACKGROUND ON PERSISTENT ORGANIC POLLUTANTS (POPS)

USGBC's attention to the use of PVC and other hazardous substances in building materials comes in the context of extensive scientific and political activity on toxic pollutants, their global distribution, and their effects on highly exposed populations and the general public. The case for the proposed LEED standard is strengthened by an understanding of that context, and the proposed action by the USGBC would in turn strengthen international efforts to reduce persistent toxic pollution at the source. As noted above, the nations of the world are now negotiating the first global agreement to eliminate sources of persistent organic pollutants (POPs).

5.1. GLOBAL DISTRIBUTION OF POPS

The POPs treaty is, in large part, a response to scientific research that has established in the last decade that a variety of synthetic chemical pollutants are now globally distributed in the environment and food web, have damaged wildlife populations, and may have caused large-scale human health damage.[217] Global contamination has occurred because many synthetic chemicals are persistent in the environment, resisting natural degradation processes for months, years, or decades. As a result, even substances that are discharged at a relatively slow rate build up to higher levels over time and are distributed long distances on currents of wind and water. Many synthetic organic substances, because they are derived from petrochemicals, are oil soluble and therefore bioaccumulate; they build up in the fatty tissues of living things and multiply in concentration as they move up the food chain. Some bioaccumulative substances reach concentrations in species high on the food web, including humans, that are tens of millions of times greater than their levels in the ambient environment.[218]

Releases of persistent and/or bioaccumulative substances since the expansion of synthetic chemical manufacturing after World War II has resulted in the global accumulation of a large number of POPs in areas remote from any known sources of these substances, including the high Arctic,[219] the isolated rainforests of South America and Africa,[220] and remote regions of the deep oceans.[221] In the Arctic, where long residence times, cold temperatures, and long food chains combine to enhance the

persistence and bioaccumulation of organic chemicals, body burdens of humans and wildlife are as much as an order of magnitude greater than in temperate latitudes of industrialized nations.[222]

Although research and policy have focused primarily on a handful of substances – PCBs, dioxin, and about a dozen organochlorine pesticides -- global contamination cannot be reduced to a few "bad actors." In the Great Lakes, 362 synthetic chemicals have been "unequivocally identified" in the water, sediments and food chain; the list includes the most infamous POPs, but it also contains a full spectrum of less familiar substances, from solvents and chemical intermediates to a host of complex industrial specialty chemicals, by-products, and breakdown products.[223] By-products of chlorinated chemical manufacture and disposal are present in measurable quantities in the Canadian Arctic[224] and over the remote Atlantic Ocean[225], and a variety of chlorinated benzenes are ubiquitous components of rain and snow.[226] Chlorinated solvents, refrigerants, and their environmental degradation products have become truly ubiquitous contaminants of the atmosphere and vegetation.[227]

With the environment and food web ubiquitously contaminated, it should come as no surprise that the bodies of human beings are too. Exposures come through inhalation, drinking water, and food. For highly bioaccumulative substances, the vast majority of the average individual's exposure – in excess of 90 percent – comes through the food supply, primarily from animal products.[228] At least 700 xenobiotic organic chemicals are present in the adipose tissues of the general population of the United States[229]; close to 200 organochlorine pesticides, solvents, plastic feedstocks, specialty chemicals, by-products, and metabolites have been specifically identified in the in the blood, fat, milk, semen, urine, and/or breath of the general U.S. and Canadian population – people with no special workplace or local exposures to these substances. Fat-soluble chemicals that have accumulated in a woman's body easily cross the placenta and are concentrated to breast milk.

The now-ubiquitous global presence of myriad synthetic chemicals, in large-scale production for just over half a century, supports a simple inference: substances that persist or bioaccumulate cannot be integrated into natural cycles. Discharged in even very small amounts, these chemicals build up gradually in the environment and in living things. Given enough time, even very small "acceptable" discharges ultimately reach unacceptable levels. The ecosystem's assimilative capacity for persistent or bioaccumulative substances is therefore zero, and the only "acceptable" discharge is also zero. Any amount greater than zero must be expected to lead to some degree of long-term, large-scale

contamination. For this reason, strategies designed to eliminate the materials and processes that produce persistent and/or bioaccumulative substances are far superior to those that attempt to control, manage, or dispose of persistent chemicals after they have been produced.

5.2. ENDOCRINE DISRUPTION

What are the impacts of universal exposure to POPs on the health of people and wildlife? Important discoveries have emerged in the last decade from toxicology, epidemiology, and ecoepidemiology on the hazards of low-dose exposure. Traditionally, toxicological studies have focused on frank manifestations of toxicity at relatively high doses, such as death, organ damage, paralysis and tremors, cancer, and structural birth defects. Recently, however, it has been discovered that many synthetic chemicals can, at very low doses, result in subtle but significant deficits in an organism's functional capacities, such as fertility, cognition/intelligence, and immunity.

Many of these effects occur as the consequence of a newly recognized set of toxicological mechanisms – disruption of the body's endocrine system.[230] The endocrine system comprises the hormones, the glands that produce them, and the response of diverse tissues to these substances. Hormones are the body's natural signaling molecules, which circulate in the blood in very low concentrations (typically in the parts per trillion range) and trigger cascades of gene expression that control essential aspects of development, reproduction, behavior, immunity, and the maintenance of homeostasis.

In the last decade, a flurry of research has identified dozens of industrial and agricultural chemicals that disrupt the endocrine system. Some mimic or block the activity of the body's endogenous hormones by interacting directly with the hormone receptor molecules that mediate the response of cells to hormones, such as the steroids estrogen, testosterone, progesterone, and the stress hormones cortisol. Others change the rate at which the body produces or excretes its own hormones, causing unnaturally low or high levels of steroid, thyroid and retinoid hormones. Still others disrupt local signaling mechanisms that are critical to development, brain function, and the immune response, including growth factors, neurotransmitters (molecules that mediate communication among brain cells), and cytokines (intercellular signaling molecules that regulate immune function).

The U.S. National Academy of Sciences has reviewed the evidence on endocrine disruption and concluded that adverse reproductive, developmental, neurological, and immunological effects have occurred in human populations, wildlife, and laboratory animals as a consequence of exposure to hormonally active compounds found in the environment.[231] According to the Academy, effects observed include structural and functional abnormalities of the reproductive tract, reduced fertility, behavioral changes, reduced cognitive ability, and immune suppression. Many studies of wildlife have shown associations between health impacts and exposure to endocrine disrupting substances, including in large ecosystems like the Great Lakes and Baltic Sea with pollutant concentrations that are increased above universal levels by less than an order of magnitude. There is evidence that the health of the general public may have been compromised by universal exposure to these substances, but the Academy did not reach consensus on this point. The panel noted that the degree of support for this hypothesis depends on perspectives that are informed by values, including the standard of proof that should be satisfied before conclusions about public health are drawn, what kinds of effects are worthy of concern, and how scientific findings about effects on one species or stage of life should be extrapolated to others.

5.3. DIOXIN AND RELATED COMPOUNDS

5.3.1 Occurrence and exposure

The most intense scientific activity has focused on 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD, known colloquially as *dioxin)*, and a large group of structurally and toxicologically related group of compounds (including other chlorinated dibenzodioxins, dibenzofurans, PCBs, chloronaphthalenes, and many others), together referred to as *dioxins* or *dioxin-like compounds*.[1] Recent research on dioxins is particularly relevant to the PVC debate because of the evidence that the PVC lifecycle is a major source of dioxins.

[1] Because these substances all cause toxicity through a common mechanism, mixtures of dioxin-like compounds are usually expressed in terms of the toxicity of TCDD, the most toxic dioxin, as TCDD-equivalents (TEQ). To calculate the TEQ value for a mixture, the quantity of each substance is multiplied by its toxicological potency relative to TCDD, and the TEQ is the sum of these weighted quantities. A mixture with a calculated TEQ of 1 microgram is expected to have toxicity equal to 1 microgram of TCDD.

Dioxins are extremely persistent substances that break down slowly if at all in the environment. Dioxins are also powerfully bioaccumulative and are now globally distributed in the ambient environment and food web. They can be detected in the tissues and fluids of the entire U.S. population. They are cleared from the body extremely slowly: U.S. EPA estimates an average half-life for TCDD in humans of over 7 years; the body burden of the average adult therefore increases throughout life as the substance gradually accumulates in fatty tissues. Dioxins are passed transgenerationally with great efficiency; a typical nursing infant in the United States receives a daily dioxin dose 92 times greater than that of the average adult.[232]

5.3.2 Health impacts of dioxin

In 2000, U.S. EPA released its Dioxin Reassessment, a comprehensive scientific summary and analysis of research in dioxin toxicology and epidemiology, and has come to the following conclusions:

- Epidemiological and laboratory studies have established that dioxin is a human carcinogen, echoing the International Agency for Research on Cancer's determination that dioxin is a known human carcinogen.[233] Dioxin is the most potent synthetic carcinogen ever tested, causing increases in specific cancers and cancers of all sites at extremely low doses. In utero exposure to very small quantities of dioxin are associated with increased cancer of hormone-responsive organs (i.e., mammary gland) when the exposed animal reaches adulthood.

- The general public's exposures to dioxin pose a calculated cancer risk in the range of one per 100 to one per 1,000 – at least 1,000 times greater than the usual "acceptable" risk.[2] People who eat greater than average quantities of meat or fish are subject to even higher cancer risks.

- Dioxin's non-cancer effects may be of even greater concern than its carcinogenicity. Dioxin is a potent endocrine-disrupting substances, interacting with intracellular receptors and disrupting homeostasis of steroid hormones, thyroid hormone, retinoic acid, gonadotropins, and growth factors at extremely low doses. Exposure to even a single tiny dose before birth can lead to profound effects on development of the brain and reproductive system, with effects including impaired cognitive

[2] Following EPA's usual approach, this risk estimate is based on the upper bound of the 95 percent confidence interval for the carcinogenic potency of dioxin. The potency is derived from both human and animal studies. The actual risk to the public, on average, may be lower than calculated using this conservative estimate.

ability/IQ, reduced sperm density, smaller and/or malformed reproductive organs and structures, and impaired sexual behavior.

- Dioxin is a powerful immune suppressant, interfering with immune function and increasing susceptibility to infectious disease at extremely low doses

- The current body burden of the general human population is already in the range – within no more than one or two orders of magnitude -- at which dioxin has been found to cause a variety of effects in laboratory animals and human populations, including reduced sperm count, endometriosis, hormonal changes, cognitive defects, immune suppression, and impaired development of the male and female reproductive systems.[234]

- There is no evidence that there is any threshold dose of dioxin below which no adverse health impacts occur. For all responses that have been studied – including expression of target genes, growth of pre-malignant liver tumors, and changes in circulating levels of thyroid hormones -- the best estimate of dose-response relationships at very low levels of dioxin is that the severity of the impact is roughly proportional to the magnitude of dioxin exposure.[235]

Supporting the view that there is no practical threshold for dioxin toxicity, several studies have discovered that almost infinitesimally low doses have significant biological effects. For example, when rats are given a single dose of TCDD as low as 64 billionths of the animal's body weight on day 15 of pregnancy, the sexual development, behavior, and function of their male offspring are compromised.[236] Dioxin's immunotoxicity has been documented at even lower levels. Doses of TCDD as low as 2.5 parts per quadrillion -- equivalent to a mere 10 molecules per cell -- *completely* abolish the ability of cultured immune cells to respond to signals to proliferate and mount an immune defense.[237] In whole animals, dioxin produces immunotoxicity at concentrations in the spleen about five times lower than this -- on the order of just two molecules per cell.[238] If there is a threshold for dioxin, it is so low as to be absolutely irrelevant for the purposes of environmental policy and health protection.

In addition to these findings, evidence from wildlife suggests a significant current environmental health hazard from dioxin contamination. A large number of ecoepidemiological studies have established unambiguously that bioaccumulated dioxin-like compounds have caused large-scale epidemics of reduced

fertility, endocrine disruption, developmental impairment, and immune suppression in mammals, fish, and birds in the Great Lakes, the Baltic Sea, and the Wadden Sea.[239]

Together, these findings indicate that we can not assume that the general public has any margin of safety for dioxin exposure. Indeed, it is possible, though not proven, that dioxin-like compounds already contribute to society-wide rates of cancer, infertility, immune suppression, endometriosis, and impaired cognitive development. From a public health perspective, universal dioxin exposure is already too high by a considerable margin. It is essential that further releases of dioxins into the environment be eliminated wherever technically feasible.

5.3.3 Trends in dioxin contamination

Trends in dioxin levels in the environment support the conclusion that measures to reduce dioxin generation through material substitution can effectively reduce contamination and human exposure. Numerous studies of soils and sediment in Europe and North America all show that dioxin levels were very low or non-existent before the Twentieth Century (figure 3). They began to rise slowly around the turn of the century and then increased rapidly from 1940 to 1970, the period during which the chlorine industry expanded most rapidly.[240] Then, during the 1970s, many governments restricted the use of leaded gasoline (which contains chlorinated additives and was thus a major dioxin source) and major applications of some dioxin-contaminated pesticides, including 2,4,5-T and pentachlorophenol; in the same period, the U.S. Clean Air Act and similar legislation in other nations required a wide range of industrial facilities (such as incinerators, steel mills, and chemical plants) to install particulate-reducing pollution control devices, which are likely to have reduced dioxin emissions to the air, as well. Following those actions, dioxin releases to the air – as measured by dioxin accumulation in plant foliage[241] -- declined by up to 80 percent from the late 1970s to the early 1990s. As one might expect, dioxin levels in the milk of cows, which eat foliage, subsequently declined, falling by about 25 percent from 1990 to 1994.[242] Because annual sediment layers primarily reflect the deposition of dioxin from the air into surface waters, dioxin concentrations in most samples of marine and freshwater sediment also declined.[243]

But declining deposition rates do not necessarily mean a lower total burden of pollutants in the environment. Sediment layers provide a reasonably reliable record of the quantity of a substance that settled to the bottom of a body of water in any year, which roughly indicates the amount that entered the water in that year. The annual flux of persistent compounds, however, is not directly related to the total

environmental burden; if the rate at which one puts marbles into a jar declines from 100 per year to 50, the total number of marbles in the jar will continue to increase. For a record of the total amount of dioxin that has accumulated in the environment over time, soils are better than sediments, because pollutants from the recent and the distant past stay near the top of the soil, rather than being buried in annual layers. British scientists have found that dioxin levels in soil, unlike those in sediments and foliage, continued to increase without interruption right through the 1980s and into the early 1990s. They concluded, "PCDD/Fs are persistent in soils, such that declines in atmospheric deposition may not result in a decline in the U.K. PCDD/F burden for some time. It may be that even with the anticipated declines in the primary emissions of PCDD/Fs over the next decade, the rate of deposition may still exceed the rate of loss from soils."[244]

Human and wildlife tissues reflect the same pattern, with a delay because of the persistence of these compounds in our bodies. Dioxin levels in several species of wildlife in the Great Lakes declined during the late 1970s, 1980s, and early 1990s.[245] By 1993, however, levels of dioxins in the eggs of Great Lakes trout had stopped falling, reaching, in the words of one group of researchers, "a steady state or a very slow decline."[246] No human tissue analyses are available from the early decades of this century, but dioxin levels in people from the United States appear to have increased steadily during the 1960s; following the regulatory actions of the 1970s, there then appears to have been a moderate decrease in dioxin levels during the 1980s.[247] (There is some difficulty in interpreting these data: according to EPA, "It is not known whether these declines were due to improvements in the analytical methods or actual reduction in body burden levels."[248]) In the 1990s, dioxin levels in the milk of European women declined by 20 to 40 pecent.[249]

There are no reliable projections of future trends in dioxin levels. It appears that the successful actions of the past have had their effect, however. According to Swedish scientists, the declines are history, not a continuing trend. We should not expect levels to fall further, unless we take further action to restrict dioxin sources: "During the last twenty years an overall decrease in the levels [of dioxin in human tissues] is recorded. The major part of this decrease dates back to the late 1970s and the early 1980s. The situation of today seems to be quite constant and resembles what has been found for PCB. Analyses of human breast milk show a similar trend."[250]

All this information suggests a pattern with clear implications for policy. Action to reduce production and use of dioxin-generating substances has reduced emissions to the environment of these compounds. On local and regional scales, contamination of the environment and the tissues of living organisms has fallen in response, with the speed of the decline varying among different kinds of sampled material. But these pollutants are so persistent that, so long as releases continue somewhere, the global environmental burden of these compounds declines slowly if at all. If we allow releases to continue at a reduced rate, concentrations will stop declining when a new steady state is reached. If we want to reduce human and wildlife exposure, we must reduce the use of dioxin-generating materials as rapidly as possible. Because infinitessimal doses of dioxin are enough to cause health damage, the only level of dioxin exposure that should be considered acceptable from a public health perspective is zero. If we want to prevent the accumulation of dioxins and other persistent toxic chemicals in the global environment, we need to stop environmental releases altogether.

5.4. PHTHALATES

5.4.1 Use

Considerable concern has recently focused on phthalates, a class of compounds used as plasticizers in flexible PVC. Phthalates are organic chemicals used to make vinyl plastic flexible, and they can make up a large portion -- up to 60 percent by weight -- of the final product.[251] Flexible PVC – including flooring and wall coverings – accounts for just over half of all vinyl demand, while the remainder is rigid, unplasticized materials like siding and pipes.[252] PVC accounts for the vast majority of all phthalate consumption, and phthalates are the dominant class of plasticizers used in soft vinyl products.[253] An estimated 50 percent of all phthalates produced are used in building and interior materials.[254] About 5.4 million tons of phthalates are used in vinyl products worldwide each year.[255] Vinyl is the only major plastic that requires phthalates to be flexible.

Four specific phthalates are used extensively in PVC and are relevant to this discussion: diethylhexyl phthalate (DEHP, U.S. production ~ 2 million tons per year), diisononyl phhalate (DINP, 178,000 tons per year), butylbenzyl phthalate (BBP, production not reported), and diisodecyl phthalate (DIDP, 135,000 tons per year). In addition, di-n-octyl phthalate is formed as a by-product of the production of other phthalates that are used in PVC and is released to the environment during the

manufacture and use of flexible PVC.[256] The other commercially important phthalates dibutyl phthalate (DBP) and dioctyl phthalate (DOP) are not used appreciably in PVC.

5.4.2 Fate, occurrence, and exposure

Phthalates are moderately persistent in the environment; they can be degraded biologically or chemically in the presence of air, but because of their fat-solubility they quickly adsorb to sediments or enter the food chain, where they are bioaccumulative.[257] Phthalates have therefore become ubiquitous environmental contaminants, present in water, air, fish, and human tissues on a global basis.[258] Because most phthalates are far more soluble in fat than air or water, levels in outdoor air and water are typically low, although considerably higher levels of some phthalates occur in indoor air;[259] levels of DEHP in animal and human tissues can be quite high, reaching concentrations higher than such infamous pollutants as PCBs and DDT.[260]

For the general population, the greatest exposures come through the food supply, with the highest levels in fatty foods like dairy, fish, meat and oils, although indoor air makes a substantial contribution, as well.[261] Because of their higher rate of food consumption per kilogram of body weight, children ages 6 months to 4 years receive the highest exposures to phthalates, with a daily dose of DEHP (19 micrograms per kilogram of body weight) that is more than three times that of the average adult.[262] A recent U.S. Centers for Disease Control study analyzed urine samples from the general U.S. population and found surprisingly high levels of metabolites of BBP, DEHP, DINP, and DnOP (137, 21.5, 7.3 and 2.3 parts per billion, respectively), reflecting "considerable exposure" to these compounds, as well as other phthalates.[263] Because phthalates are fat-soluble, they cross the placenta easily and concentrate in breast milk.[264]

5.4.3. Health impacts

It is clear that many phthalates, including those used in PVC, are endocrine disrupters, interfering with the normal function of several classes of hormones and their receptors. In several different assays, BBP, DBP, and DINP activate the estrogen receptor, trigger expression of estrogen-responsive target genes, and, like estrogen, induce the rapid proliferation of cancer cells; DEHP reduces binding of estrogen to its receptor but does not activate gene expression, suggesting the possibility that the substance may be anti-estrogenic in effect.[265] Monoethylhexyl phthalate (MEHP, the principle metabolite of DEHP in the

body) activates another class of hormone receptors called PPARs (peroxisome proliferator-activated receptors), which are involved in liver cancer, diabetes, and the differentiation of fat cells.[266] MEHP also interferes with the interaction of follicle-stimulating hormone – a hormone secreted by the pituitary that controls steroid hormone production and other functions of the testes and ovaries – with its receptor.[267] It also activates prostaglandin D2 receptors, which are involved in inflammation and asthma,[268] and it bind to the retinoic acid receptors (RARs), which are involved in embryonic development, vision, spermatogenesis, and many other functions.[269] There is suggestive evidence that DEHP mimics the activity of thyroid hormones; the health implications of this finding have not yet been investigated, but thyroid hormones are critical to proper development of the brain, behavior, and control of metabolism.[270] Finally, several phthalates and related compounds bind to and activate the pregnane-X-receptor (PXR), a recently discovered hormone receptor that is involved in the metabolism of both exogenous compounds and endogenous steroid hormones. Based on knowledge of the function of the PXR, activation by phthalates could, in principle, result in reduced levels of circulating steroid hormones and consequent endocrine disruption.[271]

Presumably through these or other endocrine mechanisms, phthalates are well-recognized developmental and reproductive toxicants. DEHP is the best studied member of the class, and in studies of a variety of species of laboratory animals, relatively high doses of DEHP produce structural birth defects, intrauterine death, and developmental delay. DEHP also reduces fertility, ovarian weight, and circulating estrogen levels, and it suppresses ovulation in female rodents. In males, DEHP causes testicular lesions, reduced androgen levels, and atrophy of the testes. Exposure in utero or during childhood is particularly problematic, because developmental effects occur at doses up to 100 times lower than those that produce reproductive toxicity in the adult.[272] Exposure of a pregnant mother rat to DEHP or DBP disrupts the development of her male offspring's reproductive system; effects include reduced synthesis of testosterone by the fetal testis, loss of sperm-producing cells, and abnormal development of the testes, epididymes, and prostate,.[273] Extremely low levels of MEHP (10^{-7} molar, or less than 30 parts per billion) – approximately the same level as found in the urine of the general U.S. population[274] -- cause significant damage to cultured sperm-producing cells of developing rat testes.[275]

Considerably less attention has been paid to other phthalates, but some data are available. BBP is also a male reproductive toxicant, causing testicular lesions, reduced sperm counts, and increased

infertility at relatively high doses in adult males, but virtually no data are available on impacts on the development of the reproductive system.[276] Monobutyl phthalate, a metabolite of BBP, causes cryptorchidism (failure of the testes to descend) when exposure occurs in utero.[277] Much more data needs to be gathered before the full suite of effects caused by each phthalate, together with the dose required to produce these effects at different stages of development, will be understood.

In 2000, an expert committee convened by the National Toxicology Program reported on its review of the evidence on the reproductive and developmental toxicity of phthalates. The panel compared the doses of DEHP that produce developmental toxicity in animals to the levels to which infants and toddlers in the general U.S. population are routinely exposed, and concluded by stating "concern that exposure may adversely affect male reproductive tract development."[278] The panel also examined exposure that occurs across the placenta and via breast milk and concluded, "The panel has concern that ambient oral DEHP exsposures to pregnant or lactating women may adversely affect the development of their offspring." For DINP, the panel expressed "minimal concern" – as opposed to"negligible concern" or "no concern" – that exposure of pregnant women in the general population may cause adverse health impacts on their children, and "low concern" that children exposed to DINP through mouthing of vinyl products might experience development and reproductive health effects."[279]

There is very little data from human studies concerning the impacts of phthalates on development and reproduction. One study suggests that phthalate exposure of the general population may be related to endocrine disruption and altered reproductive development in girls. In Puerto Rico, the incidence of premature breast development (early thelarche, defined as breast development during the period of 2 to 8 years of age) is quite high (about 1 percent of the population) and has been rising rapidly in recent decades. This phenomenon cannot be explained by changes in nutrition or exposure to hormones used in agriculture,[280] and a similar trend has been documented in the U.S.[281] Exposure to estrogen-mimicking compounds is a plausible explanation, because estrogens trigger breast development in girls. A case-control study of girls from the general Puerto Rican population found that the levels of phthalates in the blood of girls with premature breast development were 5.9 times higher levels than in girls without premature development. Levels of DEHP, which accounted for over 80 percent of the total phthalates measured in the girls' blood, were 6.4 times higher among girls with premature thelarche. This study does not prove that phthalates caused the precocious sexual development, but, as the authors concluded, it

suggests that the estrogenic or other endocrine-disrupting effects of phthalates may have contributed to the epidemic of early thelarche.[282]

Some phthalates are also recognized carcinogens. According to the National Toxicology Program, DEHP is "reasonably anticipated to be a human carcinogen" based on consistent findings of liver cancer in laboratory animals, although the relevance of the mechanism of carcinogenicity in rodents to that in humans is controversial, and people are likely to be less quantitatively sensitive to this effect of DEHP than rodents.[283] There is also some epidemiological evidence that exposure to phthalates increases the risk of testicular cancer in plastics industry workers (see section 2.4 below).

5.5. TRENDS IN PVC MARKETS

Polyvinyl chloride plastic was first marketed in 1936[284] but did not begin to play a major role in building construction until the 1950s. Production grew rapidly from the 1960s through the 1980s, and has now reached more than 30 million tons per year. This mass estimate includes the non-PVC components of vinyl products, such as plasticizers and stabilizers; production of pure polyvinyl chloride is estimated at approximately 24 million metric tons per year worldwide.[285] Vinyl is the largest use of chlorine in the world, accounting for over 40 percent of all chlorine use in the United States, with a similar or slightly greater proportion globally.[286]

Few materials have infiltrated modern life as ubiquitously as PVC, and construction represents the largest sector of vinyl applications. In the last fifty years, vinyl -- the only major plastic that contains chlorine -- has taken the place of wood, metal, ceramics, textiles, and metals in a range of building products, including pipes, window frames, exterior siding, floor tiles, wall coverings, and wire and cable sheathings. PVC is also used in furniture, upholstery, appliance casings, toys, shower curtains, and other household items, as well as in automobile and other vehicle components, office supplies, packaging, and medical devices.

Today, PVC is not only the largest but also the fastest growing use of chlorine in the world. In fact, it is the only major chlorine application still increasing in the world's wealthy nations, and it is growing particularly rapidly in developing countries.[287] The reasons for the industry's aggressive expansion of PVC markets lie in the economics of the production of chlorine and its coproduct, sodium hydroxide (caustic soda or alkali). Alkali is a profitable and environmentally unproblematic substance

that is used as a source of sodium and hydroxide ions in a wide variety of industries. The vast majority of the world's alkali is produced by chlor-alkali electrolysis, in which chlorine and sodium hydroxide are produced together in a fixed ratio. Chlorine is a hazardous gas, so it cannot be stored, and the chemical industry can only produce as much alkali as there are markets for chlorine. In recent years, as numerous uses of chlorine (e.g., pulp and paper, solvents, and refrigerants) have been restricted for environmental reasons, a "chlor-alkali imbalance" has developed, requiring the industry to create new markets for chlorine in order to continue to take full advantage of potential sales in alkali markets.[288] According to an analyst for the chlor-alkali manufacturer Elf-Atochem, "There is a logical progression toward permanent imbalance between caustic supply and demand. Domestic chlorine consumption and chlorinated exports will set operating rates for U.S. chlor-alkali capacity, with the EDC/VCM/PVC chain leading the way."[289]

The industry's strategy to rectify the chlor-alkali imbalance is to aggressively expand markets for PVC and the feedstocks from which it is made, which are already the major global sinks for chlorine. For the last several decades, PVC production and consumption has grown at a remarkable pace, but recently, PVC markets in industrialized nations have neared saturation, because vinyl has already replaced so many traditional materials; growth in vinyl in these countries is now no greater than annual increases in gross national product.[290] This rate of growth is not nearly enough to offset the loss of chlorine demand in sectors that have been restricted -- pulp and paper, refrigerants, and solvents -- so the industry has focused on expanding exports of PVC and its feedstocks to developing nations.[291] U.S. net exports of EDC, VCM and PVC now contain about two million tons of chlorine per year -- over 15 percent of total chlorine production -- and were expected to grow by a stunning 14 percent in 1998 alone.[292] The major recipients are Latin America and Asia, where PVC consumption is expected to grow at annual rates of 7 percent or more per year, leading to a doubling of demand each decade.[293] Why these countries? As an executive of a major Japanese PVC company explained, vinyl is a uniquely marketable product for export because poor countries need to reach only minimal levels of economic and technological development before they can be encouraged to buy plastic, and these nations usually have few environmental regulations:

> 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.[294]

The rapid increase in vinyl consumption in developing countries means that, despite slow growth in PVC consumption in the wealthy nations, global demand for PVC will rise from 22 million tons per year in 1996 to 28 million tons per year 2000 -- annual growth of over 6 percent.[295] According to one industry analyst, "The most important structural changes [in the chlorine industry] will be concentration of growth in emerging markets and restructuring in industrialized markets: potential loss of 10-30 percent of current customers in industrialized markets; continued shutdown of inland plants linked to declining uses; three quarters of global demand growth in developing countries; increase in VCM and PVC trade and potential tripling in volume of global EDC trade. It appears unlikely at this point that lost markets will offset growth for PVC and other uses.[296]

6.0 CONCLUSION

The PVC lifecycle presents one opportunity after another for the formation and environmental discharge of organochlorines and other hazardous substances. This apparently innocuous plastic is, when its lifecycle is considered, one of the most hazardous materials on earth, creating large quantities of persistent, toxic organochlorines and releasing them into the indoor and outdoor environments. PVC has contributed a signficiant portion of the world's burden of POPs and endocrine disrupting chemicals, including dioxins and phthalates, that are now in the environment and the bodies of the human population. .It is beyond doubt that vinyl has caused considerable occupational disease and contamination of local enviornments as well.

PVC is the antithesis of a green building material. Efforts to speed adoption of safer, viable substitute building materials can have significant, tangible benefits for human health and the environment.

NOTES

[1] Geiser 2000.
[2] European Commission 2000.
[3] Tukker et al. 1995.
[4] United Nations Environment Programme 2000.
[5] APHA 1996.
[6] Tellus Institute 1992.
[7] Christaensen et al. 1990. PVC was not judged demonstrably inferior under certain critera (such as energy use and accident potential during manufacture) to polyurethane, acrylonitrile-butadiene-styrene, and aluminum.
[8] KemI 2000, Greenpeace 2000.
[9] European Commission 2000.
[10] UBA 1992.
[11] European Commission 2000.
[12] Greenpeace 2000.
[13] Greenpeace 2000.
[14] Burress, C. Berkeley Group Wants Toxic Ban: City asked to stop dumping of carcinogens. San Francisco Chronicle, October 3, 2000.
[15] Greenpeace 2000.
[16] Greenpeace 2000.
[17] Warren 1998, Jackson 1998, and New York Times 1998,
[18] Mayer 1998.
[19] Thornton 2000, Collins 2000, International Joint Commission 1992, APHA 1994.
[20] For a discussion of chlorine-free synthesis technologies, see Thornton 2000. and references therein.
[21] Great Lakes Water Quality Board 1987.
[22] Burmaster and Harris, Miller et al. 1991.
[23] See discussion and references in Thornton 2000.
[24] Henschler 1994.
[25] Solomon et al. 1993.
[26] APHA 1994.
[27] Henschler 1994.
[28] Leder et al. 1994. In Western Europe, the Oslo-Paris Commission on the Northeast Atlantic has recommended that mercury cells be phased out, so the proportion of mercury-related chlorine is expected to decline from these 1994 figures.
[29] Leder et al. 1994.
[30] Leder et al. 1994.
[31] Leder 1994.; see also Thornton 2000.
[32] Schmittinger et al. 1986.
[33] HSDB 1997.

[34] Concentrations of these compounds detected in chlorine gas range from 40. to 210. parts per billion (Hutzinger and Fiedler 1988). My calculation of annual loadings assumes world production of 39. million metric tons of chlorine each year. (Leder 1994)

[35] Rappe et al. 1991. report dioxins and furans in three samples of chlor-alkali electrode sludge, with total concentrations of PCDD/F of 641, 667. and 263. ppb, with TEQ values of 28, 28, and 13. ppb.

[36] Svennson et al. 1993.

[37] Kaminski and Hites 1984.

38. Barrie et al. 1997.

[39] Andersson et al. 1993.

[40] This research is summarized in Versar 1996. and EPA 1998.

[41] Environment Agency 1997.; the quantity of dioxins discharged has been estimated at 1.5. grams per year (TEQ).

[42] Lindqvist et al. 1991.

[43] Lindqvist et al. 1991, Pacyna and Munch 1991.

[44] Ayres 1997.

[45] The data from Euro-Chlor, presented in Ayres 1997, are the most comprehensive available. They have the advantage of being based on a mass balance method, so that all mercury consumed is accounted for in way or another. My calculation of total mercury releases from the chlor-alkali industry uses this range and assumes 39. million tonnes global chlorine production, 35.5. percent through the mercury process (Leder et al. 1994). The actual total may be higher, since many plants are not likely to be as well operated as those in Europe. Euro-Chlor's estimates of releases to water and air (0.2. and 1.9. grams of mercury per ton of chlorine, respectively) are somewhat lower than estimates made by other parties. One review estimates mercury releases at 3. grams per ton of chlorine for a new chlor-alkali plant, and 10. grams per ton of chlorine for a well-operated existing facility (Schmittinger et al. 1986). Real-world plants in Germany have been found to release 19. grams per ton (SRI 1993).

[46] ATSDR 1998.

[47] Harada 1995, Davies 1991.

[48] Hill and Holman 1989.

[49] Airey and Jones 1970, Johnston et al. 1993.

[50] Maserti and Ferrara 1991.

[51] Panda et al. 1990.

[52] Energy requirements vary somewhat among the chlor-alkali cell types: the mercury cell requires 3310.-3520. kilowatt hours (kwh) per tons of chlorine, the diaphragm 2830. kwh/ton, and the membrane process 2520. kwh/ton; based on the proportion of each cell type in the world industry, the average energy requirement for the industry overall is slightly under 3000. kwh/ton (SRI 1993).

[53] SRI 1993.

[54] Assuming an average global cost of 4.2. cents per kilowatt-hour for chlor-alkali customers (SRI 1993).

[55] In the U.S., 109. nuclear plants generated 673. billion kwh of electricity, for an average of about 6. billion kwh per plant per year (Energy Information Administration 1996).

[56] This calculation extrapolates from Norwegian government estimates (SFT 1993), which were derived from industry data on releases. SFT estimated that 7. grams of EDC are released to water during the production of EDC; in the production of one ton of VCM from EDC, SFT estimated releases of EDC to water, EDC to air, VCM to water, and VCM to air at 1. gram, 5000. grams, 1. gram, and 1000. grams, respectively. In the polymerization of one ton PVC, SFT estimated that 5100. grams of VCM are released into the air.

Extrapolations assume 1990. production rates of 29, 137. kilotons EDC per year, 18, 495. kilotons of VCM per year, and 18, 135. kilotons of PVC per year (SRI 1993). More recent estimates of substantial increases in VCM production (Kielhorn et al. 2000. would increase these estimates by about 50. percent.

[57] SFT 1993.

[58] ATSDR 1993, ATSDR 1995.

[59] Kielhorn et al. 2000.

[60] ATSDR 1993, ATSDR 1995.

[61] Kielhorn et al. 2000.

[62] Kielhorn et al. 2000.

[63] Kielhorn et al. 2000.

[64] Pitot and Dragan 1991.

[65] Bowermaster 1993.

[66] Curry et al. 1996.

[67] My calculations assume that about 15. million metric tons per year of EDC produced by oxychlorination (half of world production (SRI 1993), assuming integrated oxychlorination and direct chlorination process in 1.:1. molar ratios). Heavy and light ends are assumed to be produced at the rate of 2. kilograms each per ton, based on the fact that production of 168, 796. tons of EDC in Sweden per year results in the generation of 335. and 333. tons per year of heavy and light ends, respectively (TNO 1996). This figure is slightly lower than that of Rossberg et al. (1986), who estimate 2.3. and 2.9. kg heavy and light ends per ton of VCM produced, respectively. Use of more recent figures for global PVC production rates (Kielhorn et al. 2000) would increase by about 50. pecent.

[68] Rossberg et al. 1986.

[69] The lower estimate is from Papp 1996. The upper estimate is from Rossberg et al. 1986, assuming synthesis of EDC in integrated chlorination/oxychlorination facility plus pyrolysis to VCM, and includes releases to air, water, heavy ends, and light ends, except nitrogen gas vented to the atmosphere and aqueous streams

[70] Dow 1990.

[71] This calculation assumes global production of 32. million kilograms of EDC heavy ends, as discussed in the note above. Use of more recent figures for global PVC production rates (Kielhorn et al. 2000) would increase by about 50. pecent

[72] Johnston et al. 1993.

[73] Costner et al. 1995.

[74] In research at a chemical plant in Russia, Khizbullia et al. (1998) found substantial quantities of dioxins and furans in the wastewater and wastewater sludge from the pyrolytic production of VCM from EDC; very large amounts were present in the waste incinerator emissions and residues from the pyrolytic production of VCM from EDC.

[75] ICI 1994.

[76] Evers et al. 1989. That report estimated that 419. grams of dioxin TEQ are formed per 100, 000. tons of EDC produced. World production of EDC by oxychlorination is about 15. million tons per year (see note above), so this rate would result in the formation of over 60, 000. grams of dioxin (TEQ) per year. Although presumably not all the dioxins created would be released directly to the environment, this quantity is more than 50. times greater than the annual dioxin emissions to air from all trash incinerators in the U.S., the largest known source of dioxin emissions in that country. It is also double the 25, 000. grams of dioxin (TEQ) per year that EPA estimates are carried into the environment by contaminated

pentachlorophenol (EPA 1998), the largest identified source of dioxin to any environmental medium.

[77] See SFT 1993, which uses the Norsk-Hydro data to estimate dioxin releases to the environment -- not quantities generated -- at 0.1. grams per 100, 000. tons of EDC/VCM. The same document did estimate, however, that dioxins generated and directed into EDC tars or recirculating process streams were about 23. to 38. times higher than the amounts released into the environment -- a quantity still less than the generation rates found in the laboratory work of Evers et al. 1989.

[78] Lower Saxony 1994.

[79] Environment Agency 1997. Total dioxin generation associated with EDC/VCM synthesis was estimated at 27. grams (TEQ) per 200, 000. tons of VCM, for a dioxin generation rate of 13.5. grams (TEQ) per 100, 000. tons -- substantially more than the Norwegian estimate but less than the Dutch figure. If production at the same plant of perc and trichloroethylene from the heavy ends of EDC oxychlorination are included, the estimate of dioxin formation increases to 500. g TEQ per year from this plant alone. Based on this estimate, all oxychlorination processes would be among the world's largest sources of dioxin.

[80] Costner et al. 1995.

[81] DTI 1995, Environment Agency 1997, SFT 1993.

[82] Contamination in the UK is described by Environment Agency 1997, in Germany by Lower Saxony 199, 4. and in the United States by Curry et al. 1996.

[83] Ramacci et al. 1998.

[84] Evers et al. 1988.

[85] Evers et al. 1993, Evers et al. 1996.

[86] U.S. Centers for Disease Control 1999.

[87] Curry et al. 1996. In addition to EDC and VCM, PPG also makes solvents and other organochlorines at the same facility, and these processes are likely to contribute to the contamination.

[88] EPA 1990.

[89] EPA 1989.

[90] UKDOE 1989. The German chemist Otto Hutzinger, one of the world's leading experts on dioxin formation concurred: "The major obstacle to construction of new MSWI [municipal solid waste incinerators] is that incineration produces several hundred stable and toxic compounds including polychlorinated dibenzodioxins. These compounds are always present at parts per million concentrations in all MSWI units, both in the fly ash formed during combustion and in the stack emissions." (Hutzinger 1986)

[91] Dellinger et al. 1988, EPA 1994b.

[92] Gullett 1990.

[93] EPA 1989.

[94] Eklund et al. 1988. Similarly, combustion under well-controlled laboratory conditions of trichloroethylene, another relatively simply organochlorine, produces a variety of persistent organochlorine PICs, including hexachloropentadiene, highly chlorinated benzenes and indenes, PCBs, and the dioxin-like chlorofulvalenes (Blankenship et al. 1994).

[95] Trenholm 1986, Trenholm 1987, Dellinger 1988, Chang 1988, EPA 1987a, EPA 1987b, Wienecke et al. 1995.

[96] EPA 1990.

[97] Markus et al. (1997) used a calibrated bioassay to quantify the activity of the cytochrome p4501A1. enzyme, which is induced by dioxin and serves as a "sensitive and selective" marker of dioxin exposure. The total dioxin-like toxicity of the fly ash exceeded that predicted by the quantity of dioxins, furans and PCBs in the sample by a factor of two to five.

[98] Assuming incineration of 30, 000. tons of EDC heavy ends per year, as discussed above.
[99] EPA 1985.
[100] Kramlich et al. 1989, Trenholm et al. 1984.
[101] See, for instance, the 1986. analysis by U.S. EPA engineers (Staley et al. 1986), which concluded, "There are several problems with the permitting process [based on trial burns]. First, the trial burn data only indicate how well the incinerator was operating during the time that the data were being taken, typically only a period of a few days. No information is obtained on how the incinerator might respond if fuel, or especially waste, conditions change. Waste streams vary widely in composition and one incinerator may burn many different toxic substances over its useful life, resulting in unavoidable and frequent changes in waste feed conditions. It is difficult to generalize the results of a trial burn to predict how the composition of the incinerator exhaust will change under these varying conditions."
[102] Licis and Mason 1989.
[103] National Toxicology Program 2000.
[104] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a
[105] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a, 2000b
[106] Hardell et al. 1998.
[107] Hardell et al. 1997, Ohlson and Hardell 2000.
[108] Hansen 2000.
[109] In this study, two samples of pure PVC contained 0.86. to 8.69. ppt TEQ (SEPA 1994).
[110] The UK Ministry for Agriculture, Fisheries, and Food found that PVC food packaging contained dioxins at levels ranging from 2.6. to 6.9. ppt TEQ (MAFF 1995).
[111] Wagenaar et al. 1996, Carroll et al. 1996.
[112] DTI 1995.
[113] These figures are for Western Europe (European Commission 2000).
[114] My calculations are extrapolated from the figures for Sweden, where the lead input into PVC equals 0.653. percent of total PVC production, and the phthalate input equals 22.6. percent (TNO 1996), assuming 24. million tons of PVC production worldwide.
[115] TNO 1996.
[116] DTI 1995.
[117] Pearson and Trissel 1993, Goldspiel 1994.
[118] Plonait et al. 1993.
[119] Dine et al. 2000, National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a
[120] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a, 2000c
[121] Rudell 2000.
[122] Rudell 2000.
[123] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a
[124] Rudell et al. in press
[125] Oie et al. 1997.
[126] Oie et al. 1997.
[127] Jaakola et al. 1999.
[128] Jaakola et al. 2000.
[129] Hsieh 2000.
[130] Lstiburek and Carmody 199, 4. Downs 2000.

[131] Vinyl Institute 2000.

[132] Vinyl Institute 2000.

[133] Lstiburek and Carmody 2000.

[134] The Consumer Product Safety Commission issued a particularly trenchant warning on the release of lead from PVC blinds, as reported in Chicago Tribune 1996.

[135] DTI 1995. Lead is much more commonly used as a stabilizer in pipes in Europe than in the United States.

[136] European Commission 2000.

[137] European Commission 2000.

[138] National Research Council 2000.

[139] Markowitz et al. 1989, Markowitz 1989, Wallace 1990.

[140] Quoted in Greenpeace 2000.

[141] Quoted in Greenpeace 2000.

[142] Greenpeace 2000.

[143] Wirts et al. 1998, Christman 1989, Theisen et al. 1989.

[144] TNO 1996.

[145] Fiedler et al. 1993. The soot samples contained dioxins and furans at 25, 251. nanograms (TEQ)/kg, which Fiedler et al. note is equivalent about 15, 000. nanograms (TEQ)/square meter. In contrast, the Hessen Ministry of the Interior's recommended maximum for wipe samples from surfaces in homes and offices is 10. nanograms (TEQ)/square meter.

[146] In a report for U.S. EPA, Versar (1996) cites analytical results that found dioxin and furan production from the combustion of vehicles that contained PVC at the following rates: .044. mg TEQ/automobile, 2.6. mg TEQ/subway car, and 10.3. mg TEQ/rail car.

[147] The U.S. Vinyl Institute, (Carroll 1995) based on one study of soot residues within a limited radius of a fire at a plastics facility, has calculated that accidental fires are probably a relatively small contributor to the total dioxin burden. But over 90. percent of the dioxins produced in a structural fire are in the gaseous phase and escape into the atmosphere (Versar 1996, EPA 1998), so calculations from soot alone will underestimate total dioxin emissions from accidental fires by at least an order of magnitude . EPA (1998) has concluded that the data are currently inadequate to make a firm quantitative estimate of the contribution of accidental structural fires to national dioxin emissions.

[148] UBA 1992, German Environment Ministers 1992.

[149][149] I have extrapolated from figures for Sweden (TNO 1996), which indicate that the stock of PVC in use (2. million tons) equals 22.47. years of current PVC production. I have assumed a similar stock-to-production ratio worldwide, and annual PVC production of 19.1. million tons per year (DTI 1995). Use of more recent figures for global PVC production would increase this estimate substantially (Kielhorn et al. 2000)

[150] Carroll 1995. notes that an average house contains 14. to 367. kg PVC, depending on the size and date of construction/remodeling.

[151] Schecter and Kessler 1996. Deutsch and Goldfarb (1988) also reported elevated dioxin levels in a university building after an interior fire in a lecture hall that contained PVC components.

[152] I have extrapolated from figures for Sweden (TNO 1996), which indicate that the stock of PVC in use (2. million tons) equals 22.47. years of current PVC production, which contains 15, 000. tons of lead 288. thousand tons of phthalates. I have assumed a similar stock-to-production ratio worldwide, and annual PVC production of 19.1. million tons per year (DTI 1995). Use of more recent figures for global PVC production rates (Kielhorn et al. 2000) would increase by about 50. pecent

[153] TNO 1996.

[154] European Commission 2000.

[155] Association of Post-Consumer Plastics Recyclers 1998.

[156] DTI 1995.

[157] European Commission 2000.

[158] European Commission 2000.

[159] KemI 2000.

[160] European Commission 2000.

[161] European Commission 2000.

[162] European Commission 2000.

[163] Hamilton-Wentworth 1997, Socha et al. 1997. The latter reference notes that dioxin levels in tree leaves downwind from the fire were 7. to 100. times above normal. Apparently, pollutants on the leaves was apparently washed from the leaves into the general environment by rain, because levels on leaves declined significantly after the first post-fire rainstorm.

[164] Based on preliminary data, EPA (1998a) estimates that landfill fires may emit on the order of 1000. grams of dioxins and furans (TEQ) to the air each year in the U.S., second only to trash incinerators among U.S. dioxin sources to the air.

[165] Danish EPA 1993, Ecocycle 199, 4. DTI 1995, TNO 1994. In the European Union, PVC contributes 39. to 66. percent of the total chloine content in waste streams being incinerated, with an average of 50. percent (European Commission 2000).

[166] Assuming U.S. municipal waste incinerator capacity of 48. million tons per year (Versar 1996), 80.% capacity utilization, and PVC content of 0.5. to 0.8. percent.

[167] According to two studies, 9.4. percent (Marrack et al. 1988) and 15. percent (Hasselriis and Constantine 1993) of infectious red-bag waste in the U.S. is PVC, and as much as 18. percent of non-infectious hospital wastes are PVC (Hasselriis and Constantine 1993) In Denmark, PVC accounts for about 5. percent of all medical waste. (DTI 1995).

[168] See discussion in Green 1993.

[169] Christmann et al. 1989a, Theisen et al. 1989, Theisen 1991.

[170] Yasuhara and Morita 1988. See also Blankenship et al. 1994.

[171] Jay and Stieglitz 1995.

[172] I have extrapolated from the relevant figures for Sweden, where 249. tons of PVC enter the waste stream each year (TNO 1996), assuming 19.1. million tons of PVC production worldwide, each year (DTI 1995).

[173] TNO 1996.

[174] European Commission 2000.

[175] Lemieux 1997. PCDD/F emissions (total) from an avid recycler with high PVC content in their waste averaged 269.6. micrograms per kilogram of waste burned; from a non-recycler with much lower quantities of PVC, and PCDD/Fs averaged 44.30. ug/kg of waste. There were only two runs for each type of trash, so conclusions about the role of PVC in dioxin emissions are tentative. EPA contrasted these high levels of dioxin emissions to those from a municipal waste combustor, which EPA estimated at 0.0035. ug/kg of waste. This figure may be lower than many incinerators in the real-world, but the point that uncontrolled burning of waste produces relatively high quantities of dioxin is almost certaintly correct. My estimate of the number of households required to produce the same amount of PCDD/Fs assumes, as EPA's report does, an incinerator burning 182, 000. kilograms of waste per day, as compared to an average of 4.9.

kilograms per day in non-recycling households.

[176] Christmann 1989, EPA 1994a, Versar 1996.

[177] Christmann et al. 1989.

[178] Schaum et al. 1993, EPA 1994b, Aittola et al. 1993.

[179] Versar 1996.

[180] Theisen 1991.

[181] Christman

[182] Vikelsoe and Johansen 2000.

[183] Kopponen et al. 1992.

[184] Kolenda et al. 199, 4. Wilken 1994.

[185] Mahle and Whiting 1980.

[186] Liberti 1983.

[187] Halonen et al. 1995.

[188] Altwicker et al. 1993. In this study, increasing the feed of organically-bound chlorine results in a substantially higher ratio of chlorophenols to chlorobenzenes in the combustion products; chlorophenols are considered precursors for dioxin formation.

[189] Bruce et al. 1991. found that addition of potassium chloride, sodium chloride, or calcium chloride to a combustion reaction had no effect on the quantities of dioxins and furans formed and deposited in the fly ash. Addink et al. 1998. added sodium chloride to fly ash and found that it did not participate in the *de novo* formation of dioxins and furans. Lenoir et al. 1991. burned sodium chloride with polyethylene in a fluidized bed combustor and found no effect on the amount of dioxins and furans emitted.

[190] Danish Environmental Protection Agency 1993.

[191] Tamade et al. 2000, Yoneda et al. 2000.

[192] Mattila et al. 1992, Ruuskanen et al. 199, 4. Frankenhauser et al. 1993.

[193] This study by Kanters et al. (1996) focused on emissions of chlorophenols as a surrogate for dioxin, due to the difficulty and expense of dioxin sampling and analysis.

[194] Wagner and Green 1993.; this study also measured emissions of chlorophenols as a dioxin surrogate.

[195] Christmann et al. 1989b.

[196] Vesterinen and Flyktmann 1996, Halonen et al. 1993b, Hutoari et al. 1996, Manninen et al. 1996. In all of these studies, dioxin levels in fly gas or flue gas increased with increasing feed of refuse-derived fuel to the burner, which was significantly higher in chlorine content than the organic matter used in comparison runs.

[197] Hatanaka 2000.

[198] Wikstrom et al. 1996.

[199] The report of Hatanaka et al. 2000. also found that NaCl and PVC resulted in similar increases in dioxin formation, although the unusually high concentration of NaCl added was thought to have resulted in less optimal combustion conditions, possibly increasing dioxin emissions indirectly.

[200] Visalli 1987.

[201] Mark 1994.

[202] EPA 1988.

[203] Rigo et al. 1995.

[204] Goodman 1994.

[205] Costner 1997, Thornton 2000.

[206] Brzuzy and Hites 1996b.

[207] Martinez et al. 2000.

[208] Reviewed in Schecter 1991. Original research reports include Ligon et al. 1989, Schecter et al. 1988, and Tong et al. 1990.

[209] Reviewed in Alcock and Jones 1996.

[210] Czuczwa and Hites 1986, Czuczwa et al. 198, 4. Czuczwa and Hites 1985.

[211] Kjeller and Rappe 1995.

[212] Juutner et al. 1997.

[213] Reviewed in Alcock and Jones 1996. Echoing these findings, EPA scientists, in a study of 11. lakes in remote parts of the U.S., found that PCDD/F concentrations in pre-1930. sediments were at most one-tenth the levels in more recent layers (Cleverly et al. 1996).

[214] Brzuzy and Hites 1996b.

[215] Czuczwa and Hites 1984. Additional data are reported in Czuczwa and Hites 1986. and Czuczwa and Hites 1985.

[216] DTI 1995.

[217] Thornton 2000, Colborn et al. 1996.

[218.] Tatsukawa and Tanabe 1990, Allan et al. 1991.

[219.] Gregor and Gummer 1989, Patton et al. 1991, Barrie et al. 1997.

[220] Simonich and Hites 1995.

[221.] Ono et al. 1987.

[222] Dewailly et al. 1993, Norstrom et al. 1990.

[223] Great Lakes Water Quality Board 1987.

[224] Barrie et al. 1997.

[225.] Fuhrer and Ballschmitter 1998.

[226.] Brun et al. 1991.

[227.] DeLorey et al. 1998, Makhijani and Gurney 1995, Plumacher and Schroder 1993.

[228.] EPA 2000.

[229.] Onstot et al. 1987.

[230] National Research Council 2000, Guillette 1999.

[231] National Research Council 2000.

[232] EPA 2000.

[233] McGregor et al. 1998.

[234] See discussion in EPA 2000, as well as DeVito et al. 1995. and Tryphonas et al. 1995.

[235] Tritscher et al. 199, 4. Kohn et al. 1996, Portier et al. 1996.

[236] Peterson et al. 1992.

[237] Neubert and colleagues (1991) documented this effect in primate lymphocytes at TCDD concentrations as low as $10.^{-14.}$ moles per liter.

[238] Kerkvliet (1994) reports that TCDD concentrations in the spleen as low as 2.*10.-15. moles per liter caused immunotoxicity in laboratory rats.

[239] See for example Olsson et al. 199, 4. Reijnders 1986, deSwart et al. 1996, Giesy et al. 1994a, 1994b

240. Alcock and Jones (1996) provide an excellent review of dioxin trends. Specific papers include Czuczwa and Hites 198, 4. Czuczwa and Hites 1985, Czuczwa and Hites 1986, Czucwa, Veety and Hites 198, 4. Juttner et al. 1997, Kjeller et al. 1995, Kjeller et al. 1991a, Kjeller et al. 1996.

241. Kjeller et al. 1996.

242. Reviewed in Alcock and Jones 1996.

243. Sediment cores from two Black Forest lakes, for example, show a contradictory pattern. One shows that dioxin levels in the layer dated 1985.-1992. were lower than in that from the period 1964.-1985. The other, however, shows that dioxin levels in 1982.-1992. were higher than in 1960.-1982. (Juttner et al. 1997).
244. Alcock and Jones 1996. Kjeller et al. 1991a also provide a useful discussion.
245. Allan et al. 1991. Alcock and Jones (1996) also review studies that suggest a decline in PCDD/Fs in Baltic wildlife during the same period.
246. Huestis et al. 1997.
247. Stanley et al. 1990.
248. EPA 1994b.
249. Reviewed in Alcock and Jones 1996.
250. Johansson 1993.
[251] DTI 1995.
[252] These figures are for Western Europe (European Commission 2000).
[253] DTI 1995.
[254] Oie 1997.
[255] My calculations are extrapolated from the figures for Sweden, where the the phthalate input into PVC equals 22.6. percent (TNO 1996), assuming 24. million tons of vinyl produced per year worldwide.
[256] National Toxicology Program 2000.
[257] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a, 2000b, 2000c.
[258] Giam et al. 1978, Blount et al. 2000.
[259] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[260] Giam et al. 1978, National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[261] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[262] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[263] Blount et al. 2000.
[264] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[265] Harris et al. 1997, Jobling et al. 1995.
[266] Maloney and Wasman 1999.
[267] Heindel et al. 1989.
[268] Ole et al. 1997.
[269] Pagnetto et al. 2000.
[270] Badr 1992.
[271] Masayuma et al. 2000.
[272] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[273] Gray et al. 1999, Lambright et al. 2000.
[274] Blount et al. 2000.
[275] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[276] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000b.
[277] Imajima et al. 1997.
[278] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
[279] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000c.
[280] Colon et al. 2000.

[281] Hermann-Giddens et al. 1997.
[282] Colon et al. 2000.
[283] National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a, National Toxicology Program 2000.
[284] Taylor 1957, Aftalion 1957.
[285] Kielhorn et al. 2000. report global vinyl chloride production capacity of 27. million tons per year. Assuming 95. percent utilization of VCM in PVC and 95. percent operating rates, global PVC production is likely to be about 24. million tons per year.
[286] Leder et al. 1994.
[287] Growth is expected in a few much smaller applications, such as phosgene for polycarbonate and propylene chlorohydrin for propylene oxide, but the increases in these chlorine uses are less than one-tenth the growth expected in PVC (Mears 1995).
[288] Leder et al. 1994.
[289] Tullos 1995.
[290] Endo 1990.
[291] Leder et al. 1994.
[292] Mears 1995.
[293] Waltermire 1996.
[294] Endo 1994.
[295] Svalander 1996.
[296] Tittle 1995.