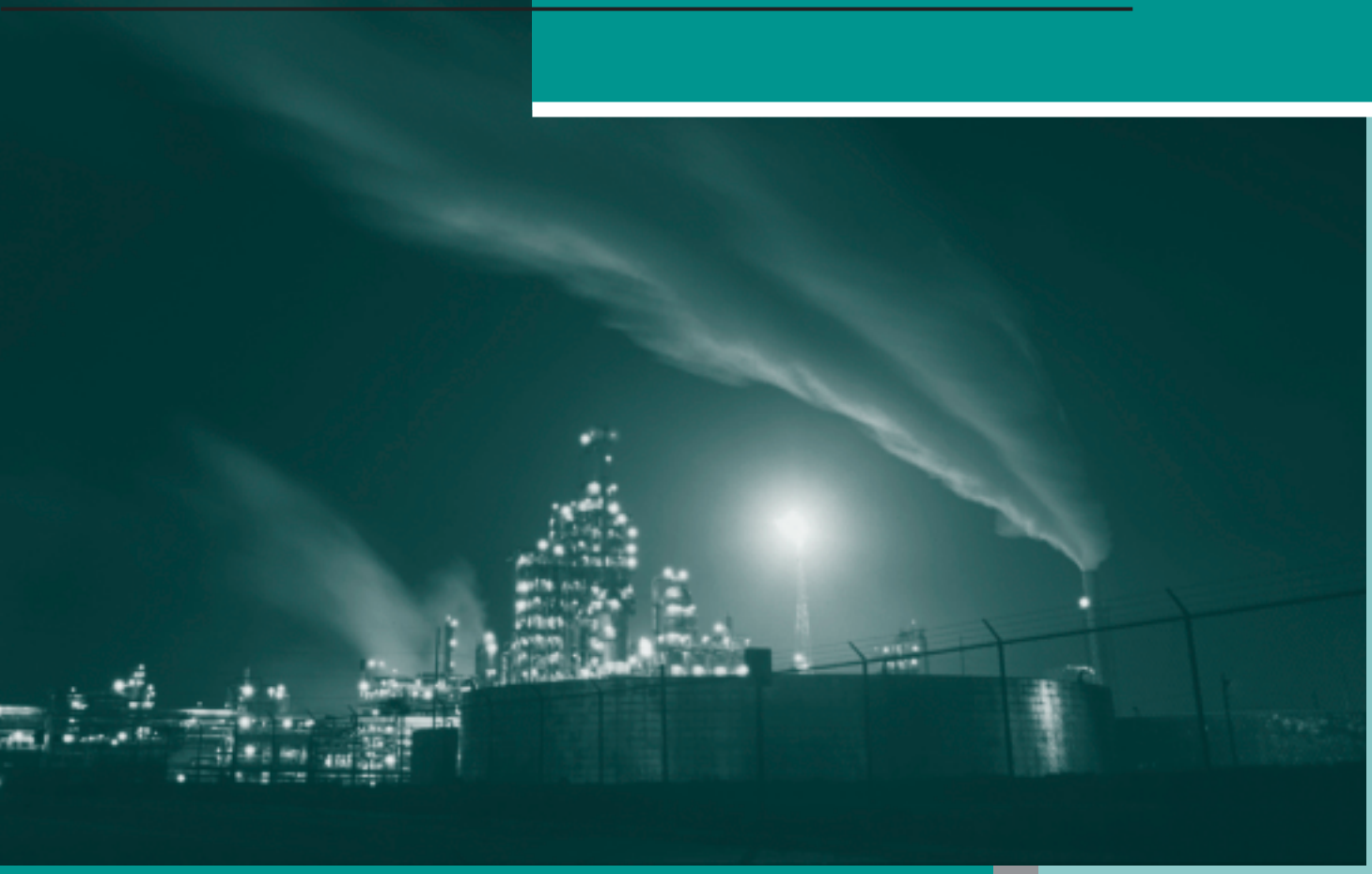
Environmental Impacts of Polyvinyl Chloride Building Materials

by Joe Thornton, Ph.D.



A Healthy Building Network Report

Environmental Impacts of Polyvinyl Chloride Building Materials

by Joe Thornton, Ph.D.

A Healthy Building Network Report

This report was prepared by the author and does not represent the opinions of The University of Oregon or any of its affiliates.

Washington, D.C.
The United States

ISBN 0-9724632-0-8

Printed in the United States of America on acid-free, recycled paper, using soy-based ink.

Photo Credits:

p. 1 © Greenpeace

p. 41 © Leuders/Greenpeace.

p. 53 © Basel Action Network. PVC coated wire waste sorted and burned by villagers in Guiyu, China. Cancer causing polycyclic aromatic hydrocarbons and dioxins will result from burning wires made from PVC and brominated flame retardants.

p. 77 © Bryce lankard/Greenpeace 1996. Cows graze outside PVC manufacturing facility in Giesmar, Louisiana. Dioxin emissions to the environment move up the food chain. Fatty foods such as meat and dairy products are the primary source of dioxin exposure to most humans.

p. 97 © Stone/Greenpeace 200. Lake Charles, Louisiana area children suffer health impacts due to pollution from PVC manufacturing facilities.

Acknowledgements

The Healthy Building acknowledges the following people and organizations for their important contributions to this report.

Dr. Joe Thornton's willingness to accept this assignment on short notice and under a short deadline is testament to his ongoing commitment to the related goals of sound science and environmental justice.

Timely and helpful peer review were provided by the following experts:

Barbra Batshalom, Founder & Executive Director, the Green Roundtable (www.greenroundtable.org)

Mark Rossi, Health Care Without Harm (www.noharm.com)

Ted Schettler, MD, MPH of the Science Environmental Health Network (www.sehn.org)

Jan Stensland, Principal, Inside Matters (www.insidematters.com)

Gail Vittori, Co-Director, Center for Maximum Potential Building Systems (www.cmpbs.org)

HBN also appreciates the thoughtful review and contributions of Paul Bogart, Charlotte Brody, RN, Health Care Without Harm (www.noharm.org); Gary Cohen, Environmental Health Fund; Charlie Cray; Lisa Finaldi, Greenpeace (www.greenpeaceusa.org); Jamie Harvie of the Institute For A Sustainable Future (www.isfusa.org); Jeannette McCulloch, Valerie Denney Communications (www.vdcom.com); Anne Platt McGinn, Worldwatch Institute (www.worldwatch.org).

Tom Lent of the Healthy Building Network over saw final editing and production by Dragon Well Digital Media Group.

Funding for this project was provided by: Alida Messinger Charitable Lead Trust; The John Merck Fund; the Mitchell Kapor Foundation; and the New York Community Trust.

The Healthy Building Network is a project of the Institute for Local Self-Reliance (www.ilsr.org) which provides excellent institutional support for our work, especially Mr. Jonathan Burnworth.

Bill Walsh
National Coordinator
Healthy Building Network
November, 2002

About the Author

Joe Thornton, Ph.D., is Assistant Professor in the Center for Ecology and Evolutionary Biology at the University of Oregon. 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 U.S. and international toxics campaigns. There, he authored seminal reports and articles on organochlorines, dioxin, breast cancer, waste incineration, risk assessment, and the precautionary principle. In 1995, Dr. Thornton joined Columbia University's Earth Institute, where he wrote *Pandora's Poison* and expanded his research into the basic science of the biological systems that can be damaged by toxic chemicals. He also co-authored the article and American Public Health Association resolution that launched the campaign to eliminate polyvinyl chloride (PVC) products from medical uses due to their central role in dioxin formation in medical waste incinerators. Dr. Thornton 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 *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

Tables

Figures

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. This paper discusses the hazards of the PVC lifecycle that have led to this large scale movement away from PVC products.

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

PVC production is the largest use of chlorine gas in the world. PVC consumes about 40 percent of total chlorine production, or approximately 16 million tons of chlorine per year worldwide. PVC is the largest production-volume organochlorine, a large class of chemicals that have come under scientific and regulatory scrutiny in the last decade because of their global distribution and the unusually severe hazards they tend to pose. PVC (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.

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

Production: Formation of hazardous organochlorine by-products begins with the production of chlorine gas. Extremely large quantities—on the order of one million tons per year—of chlorine-rich hazardous wastes are generated in the synthesis of ethylene dichloride and vinyl chloride monomer (EDC and VCM, the feedstocks for PVC).

Combustion: 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.

By-products of PVC production are highly persistent, bioaccumulative, and toxic. The chemical mixtures produced in the synthesis of EDC and VCM include such extremely hazardous and long-lived 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. Many of the by-products of the vinyl lifecycle are of great concern, because of their persistent bioaccumulative toxicity:

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.

Toxicity. The feedstocks, additives, and by-products produced and released during the lifecycle of PVC have been shown to cause a range of health hazards, in some cases 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
- Immune system suppression.

When its entire lifecycle is considered, vinyl appears to be associated with more dioxin formation than any other single product.

Dioxins. Among the most important by-products of the PVC lifecycle are dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin) and a large group of structurally and toxicologically related compounds, collectively called dioxins or dioxin-like compounds. 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.

Dioxins are formed during numerous stages of the vinyl lifecycle. Formation of dioxins has been documented in production of chlorine, synthesis of the feedstocks EDC and VCM, burning of vinyl products in accidental fires, and incineration of vinyl products and the hazardous wastes from PVC production.

Vinyl is a major dioxin source. Vinyl is the predominant chlorine donor and therefore a major and preventable 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 global pollutants. Dioxins are now found in the tissues of whales in the deep oceans, polar bears in the high Arctic, and virtually 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.

There is no known safe dose of dioxin. Dioxin causes damage to development, reproduction, and the immune and endocrine systems at infinitesimally low doses (in the low parts per trillion). Toxicological studies have not been able to establish a "threshold" dose below which dioxin does not cause biological impacts.

Dioxin is a potent carcinogen. Dioxin is the most potent synthetic carcinogen ever tested in laboratory animals and is a known human carcinogen.

Dioxin poses health risks to the general public that are already too high. 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."

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."

Phthalate plasticizers. In its pure form, PVC is rigid and brittle. To make flexible vinyl products, such as roofing materials, 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 group 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 have become global pollutants. Phthalates are moderately bioaccumulative and moderately persistent under some conditions. They can now be found in the water of the deep oceans, air in remote regions, and the tissues and fluids of the general human population. Infants and toddlers are subject to exposures several times higher than those of the average adult.

Massive quantities of phthalates are released into the environment each year. Millions of pounds of phthalates are released annually 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. More than 80 million tons of phthalates are estimated to be contained in the stock of PVC products now in use in buildings and other applications.

Phthalates leach out of vinyl products. 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 damage reproduction and development. Phthalates have been found to damage the reproductive system, causing infertility, testicular damage, reduced sperm count, suppressed ovulation, and abnormal development and function of the testes and male reproductive tract in laboratory animals. They are known carcinogens in laboratory animals.

DEHP exposure is already too high. An expert committee of the National Toxicology Program recently reviewed the hazards of diethylhexyl phthalate (DEHP, the most common vinyl plasticizer) and expressed "concern that exposure [of infants and toddlers in the general U.S. population] may adversely affect male reproductive tract develop-

ment" and "concern that ambient oral DEHP exposures to pregnant or lactating women may adversely affect the development of their offspring." The average American's dose of the plasticizer DEHP is now approximately equal to EPA's reference dose – the maximum "acceptable" exposure based on studies of health impacts in laboratory animals.

Lead and other heavy metal 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 in the environment. All three of the major PVC stabilizers resist environmental breakdown and have become global pollutants.

Metal stabilizers are highly toxic. Lead is an exquisitely potent developmental toxicant, damaging brain development and reducing the cognitive ability and IQ of children in infinitessimal doses. Cadmium is a potent neurotoxin and carcinogen, and organotins can suppress immunity and disrupt the endocrine system.

Metal stabilizers are released through out the vinyl product lifecycle. 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 a dominant source of lead and cadmium pollution, and PVC contributes a significant amount of these metals—an estimated 45,000 tons of lead each year—to incinerators.

Accidental fires in buildings and landfills are also potentially important sources of lead, cadmium, and organotins. In a fire, metals in PVC will be released to the environment; an astounding 3.2 million tons of lead are present in the current stock of PVC in use. Potential lead releases from this stored PVC must be viewed as a major potential health hazard.

Flexible PVC harms 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.

PVC products release phthalates 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.

PVC phthalate exposure may be 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 human 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.

PVC products can release heavy metals into the building environment. Metal stabilizers, particularly lead, cadmium, and organotins, can be released from vinyl products. Significant quantities of lead have been found to be released from vinyl window blinds into air and from PVC pipes into water. Toxicological effects of these substances include neurological, development, and reproductive damage.

Vinyl has been cited as the interior building material most likely to facilitate the growth of toxic molds.

Vinyl wall covering encourages toxic mold growth. Because vinyl wall coverings form a barrier impermeable to moisture, they encourage the growth of molds on wall surfaces beneath the vinyl, particularly in buildings where air conditioning or heating systems produce significant temperature and humidity differentials between rooms and wall cavities. Some molds that grow beneath vinyl produce toxic substances that are released into indoor air and are suspected causes of severe human health problems. 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.

Workers and communities are exposed to toxic substances due to PVC production. In the production of PVC, many thousands of tons per year of the feedstocks ethylene dichloride (EDC) and vinyl chloride monomer (VCM) are released into the workplace and into local environments.

PVC feedstocks cause cancer and other health impacts. Both EDC and VCM cause cancer in laboratory animals; VCM is classified as a known human carcinogen and EDC is a probable human carcinogen. Increased risks of liver cancer and brain cancer have been documented among workers exposed to VCM. They are toxic to the nervous system and cause a variety of other impacts on human health There is preliminary evidence that workers involved in the manufacture of PVC products may have elevated risks of testicular cancer.

There is no safe VCM exposure level. Although workplace exposures in U.S. chemical and plastics facilities have been significantly reduced from the levels of the 1960s, there is no threshold below which VCM does not increase the risk of cancer, so current exposures in the U.S. continue to pose cancer hazards to workers. Further, occupational exposure to VCM remains extremely high in some facilities in Eastern Europe and Asia.

VCM production facilities are major polluters. 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 contamination with dioxins and other by-products.

Chlorine production consumes enormous amounts of energy. Chlorine production is one of the world's most energy-intensive industrial processes, consuming about 1 percent of the world's total electricity output. Chlorine production for PVC consumes an estimated 47 billion

kilowatt hours per year—equivalent to the annual total output of eight medium-sized nuclear power plants.

Chlorine production causes 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. The vinyl lifecycle is associated with the continuing release of many tons of mercury into the environment each year.

Even in Europe, where PVC recycling is more advanced than in the United States, less than 3 percent of post-consumer PVC is recycled, and most of this is merely "downcycled."

PVC is extremely difficult to recycle. 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, post-consumer recycling of mixed PVC products is difficult and cannot yield vinyl products with equivalent qualities to the original. Even in Europe, where PVC recycling is more advanced than in the United States, less than 3 percent of post-consumer PVC is recycled, and most of this is merely "downcycled" into other products, which means there is no net reduction in the production of virgin PVC. By 2020, only 9 percent of all post-consumer PVC waste in Europe is expected to be recycled, with a maximum potential of no more than 18 percent.

PVC is one of the most environmentally hazardous consumer materials ever produced. The PVC lifecycle presents one opportunity after another for the formation and environmental discharge of organochlorines and other hazardous substances. When its entire lifecycle is considered, it becomes apparent that this seemingly innocuous plastic is one of the most environmentally hazardous consumer materials produced, creating large quantities of persistent, toxic organochlorines and releasing them into the indoor and outdoor environments. PVC has contributed a significant portion of the world's burden of persistent organic pollutants and endocrine-disrupting chemicals—including dioxins and phthalates—that are now present universally in the environment and the bodies of the human population. Beyond doubt, vinyl has caused considerable occupational disease and contamination of local environments as well.

In summary, the feedstocks, additives, and by-products of the PVC lifecycle are already present in global, local, and workplace environments at unacceptably high levels. Efforts to reduce the production and release of these substances should be environmental and public health priorities.

It is time to phase out PVC building materials. The hazards posed by dioxins, phthalates, metals, vinyl chloride, and ethylene dichloride are largely unique to PVC, which is the only major building material and the only major plastic that contains chlorine or requires plasticizers or stabilizers. PVC building materials therefore represent a significant and unnecessary environmental health risk, and their phase-out in favor of safer alternatives should be a high priority.

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.

The hazards posed by dioxins, phthalates, metals, vinyl chloride, and ethylene dichloride are largely unique to PVC. It is time to phase out PVC building materials.

Introduction

one

Introduction

PURPOSE OF THIS REPORT

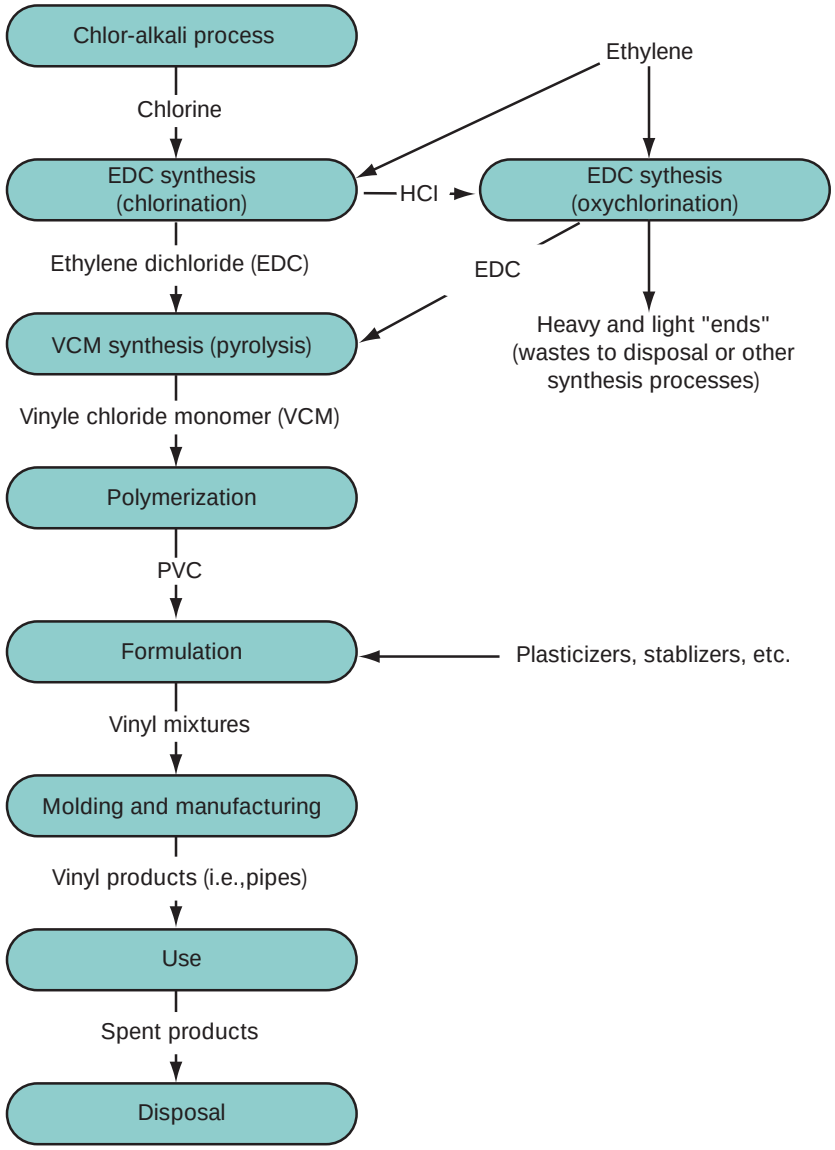
The purpose of this report is to present information on the environmental and health hazards associated with the lifecycle of polyvinyl chloride plastic (PVC; commonly known as vinyl). This report is not intended as a complete review of all aspects of the PVC lifecycle; rather, the most important evidence indicating that PVC poses important hazards to health and the environment have been surveyed from a perspective consistent with the precautionary principle. Individual chapters on the manufacture, use and disposal of vinyl products discuss the formation, release, exposure, and health implications of hazardous substances during the manufacture, use, and disposal of vinyl products are considered, as well as energy consumption associated with PVC production. The final chapter presents background information on scientific and economic issues relevant to vinyl and persistent organic pollutants, including a detailed discussion of the environmental hazards of dioxin-like compounds and phthalate plasticizers, two particularly hazardous classes of substances associated with the vinyl lifecycle.

In the United States, building and construction applications account for an estimated 75 percent of all vinyl consumption.[1] In the European Union, 60 percent of vinyl is used in building and construction applications, with an additional 25 percent in appliances, electronics, and furniture.[2] In this report, hazards specific to building and construction uses are highlighted whenever possible; however, many of the hazards of the vinyl lifecycle are general to all PVC uses and will be discussed in that context. Because many of the hazards associated with PVC are global in scale, this report takes an international perspective on vinyl markets and environmental impacts. Further, significant quantities of vinyl and vinyl products are imported, so decisions on building materials in the United States have implications for environmental quality and worker and community health wherever PVC is manufactured.

TOXIC RELEASES THROUGHOUT THE LIFECYCLE OF PVC

The PVC lifecycle is marked by 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.

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



Manufacture

PVC manufacture is comprised of five major steps—ethylene and chlorine gas production, feedstock production, polymerization, formulation or compounding, and molding.

1. *Ethylene and chlorine gas production*—ethylene gas (purified from petroleum or natural gas) and chlorine gas (synthesized from sea salt by high-energy electrolysis) are two basic materials for vinyl production.

2. *Feedstock production*—ethylene dichloride (EDC, also known as 1,2-dichloroethane) can be produced from chlorine and ethylene by chlorination or oxychlorination. 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 in a process known as oxychlorination. EDC is then converted into vinyl chloride monomer (VCM; the chemical name of which is chloroethylene), by a reaction called pyrolysis.

3. *Polymerization*—VCM molecules are linked together to yield polyvinyl chloride, typically a white powder.

4. *Formulation or compounding*—pure PVC is mixed with other chemicals—stabilizers, plasticizers, colorants, and the like—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, 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.

5. *Molding*—the formulated plastic is molded to produce the final product, such as a bottle, floor tile, or pipe.

Usage

The second major stage in the PVC lifecycle is the use of vinyl products. The duration of the product's useful life may be short (PVC packaging, with a lifetime measured in days or weeks) or moderate (PVC floor tiles or roofing materials, which have an average lifetime of 9 to 10 years).[4]

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, especially where large quantities of PVC are used, such as vinyl roofing membranes or siding.

Disposal

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 product being leached of hazardous substances, and the formation and release of unintended combustion by-products when vinyl is incinerated or processed in a secondary smelter for recycling metal products. Only a 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.

INTERNATIONAL ACTION TO ELIMINATE PVC

UNEP POPs agreement

In the fall of 2000, international negotiations were completed on the first legally binding instrument to address global contamination by persistent organic pollutants (POPs).[5] The agreement, which will require each nation to eliminate the production of POPs, represents a fundamental shift from the present control and disposal techniques used to manage chemicals. Although the treaty takes initial action on just 12 pollutants, it includes provisions for additional substances to be addressed in the future. Four of these pollutants are produced in significant quantities during the vinyl lifecycle (table 1).

International analyses

Currently there is 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.[6] In a study of all major packaging materials for the Council of State Governments in the

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.

Table 1 Persistent organic pollutants addressed by UNEP's International POPs Agreement

Pollutant
Aldrin
DDT
Dieldrin
Endrin
Chlordane
Heptachlor
Hexachlorobenzene (HCB)*
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.

Source: UNEP 1997.

United States, the independent Tellus Institute found that PVC is the most environmentally damaging of all plastics.[7] 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.[8]

Restrictions in the EU

Almost all European Union nations have restrictions on uses of PVC. These restrictions address concerns about dioxin exposure, release of phthalate softeners, or the difficulty of recycling and waste disposal. Among the most far-reaching policies are those of Sweden, whose parliament voted in 1995 to gradually eliminate soft and rigid PVC. In 1996, Sweden called for a voluntary industry phase-out of all PVC production. In 1999, it adopted a bill that includes mandatory provisions to eliminate use of PVC with hazardous additives—including phthalates and lead—and to substitute PVC use with other materials wherever feasible. From 1994 to 1999, this program had reduced total PVC use in Sweden by 39 percent.[9]

Denmark established a national strategy to address the environmental hazards of PVC, including a tax on PVC of $0.30 U.S. per kilogram, a higher tax on phthalates, a prohibition on the incineration of PVC, and an order to substitute alternative materials for all non-recyclable PVC use. The German Environmental Protection Agency (UBA) has called for an end to the use of phthalates and the gradual phase-out all uses of flexible PVC.[10] Due to the concern for dioxin generation, UBA has also called for a ban on the use of PVC in applications susceptible to fire.[11] The policy in the Netherlands 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 to establish appropriate policy measures to safeguard the environment.[12]

In Germany, 274 communities—including Berlin and Bonn—and six states have written policies to phase-out or restrict PVC.

Restrictions elsewhere in the world

Not all action on PVC is restricted to Europe. India's Ministry of the Environment and Forests has established rules that ban the incineration of PVC and other chlorinated plastics in medical waste incinerators. Singapore has informed the Secretariat of the Basel Convention on the transboundary movement of hazardous waste that it considers PVC-containing waste and PVC-coated cables to be hazardous waste that are therefore banned from import or export.[13]

Restriction on PVC use in construction

Numerous local and regional governments have specific policies to avoid PVC in construction.[14] In Germany, 274 communities—including Berlin and Bonn—and six states have written policies to phase-out or restrict PVC. The Netherlands' four largest cities—Amsterdam, Den Haag, Rotterdam, and Utrecht—have specifications to avoid PVC whenever possible in construction. Fifty-two cities in Spain 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, listing 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 reportedly achieved an 85 percent PVC phase-out in public buildings, and the Vienna subway and Vienna Ost hospital were constructed without PVC.

Nearly all of the world's major car manufacturers, including BMW, Daimler-Benz, Ford, General Motors, Honda, Nissan, Opel, Toyota, and Volkswagen AG have policies to reduce or eliminate the use of PVC in automobiles.

Numerous major construction projects have reduced or eliminated PVC entirely. 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."[15] PVC use was eliminated or radically reduced in the construction of the hotel, Olympic village, stadium, 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."[16] The headquarters for the Danish society of Engineers has been built without PVC, as has the new European headquarters for Nike in Hilversum. Many buildings in the United Kingdom, including the new Tate Gallery of Modern Art, have been designed and built to minimize or eliminate PVC use.[17]

Many private firms have also taken steps to restrict or substitute PVC products.[18] The Swedish construction firms HM and Svenska Bostder have announced that they are phasing out PVC use. In Japan, the electronics manufacturers AEG, Electrolux, Matsushita, Ricoh, Sharp, Sony-Europe, and Vorwerk have PVC phase-out policies. German Telekom, Nippon, and Sumitomo Electric Industry have policies to avoid PVC use in cable manufacture. The furniture and décor manufacturers and retailers Eco AB, EWE Kuechen (Austria), IKEA, and Innarps AB have policies to avoid PVC products. In transportation, nearly all of the world's major car manufacturers, including BMW, Daimler-Benz, Ford, General Motors, Honda, Nissan, Opel, Toyota, and Volkswagen AG have policies to reduce or eliminate the use of PVC in automobiles.

PVC restrictions in toys

Particularly urgent and widespread action has focused on PVC toys, due to concern for exposure of children 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. As a result, these countries, along with ministries in Austria, Belgium, Finland, France, Germany, Greece, Italy, Norway, Spain, and Sweden, 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, Mattel, Toys-R-Us, and several other U.S. toy makers and retailers announced that they would stop selling certain vinyl toys.[19] Subsequently, the U.S. Consumer Product Safety Commission called on the toy industry to voluntarily stop making vinyl chewing toys that contained phthalates.[20] In 1999, the European Commission finalized an emergency ban on six phthalate plasticizers found in soft PVC toys. Although toys represent a small fraction of total PVC consumption, the trend away from vinyl use in toys is significant for the entire industry. As one plastics industry spokesman commented, "In the long run, the industry will go the way toys go."[21]

PVC AND THE CASE AGAINST ORGANOCHLORINES

EDC, PVC, VCM, 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—regarded by many as the most environmentally problematic class of synthetic substances.[22] PVC is the only major plastic used in buildings that contains chlorine. Chlorine-free plastics include polyethylene (PE), polypropylene (PP), polyethylene terephthalate (PET), acrylonitrile-butadiene-styrene (ABS) copolymer, ethylene vinyl acetate (EVA), and numerous others. Polyurethane, polycarbonate, and epoxy resins are chlorine-free plastics that are currently manufactured via the organochlorine intermediates chlorohydrin, epichlorohydrin, phosgene, and propylene, but technologies are being developed to produce these plastics through chlorine-free routes.[23]

The proper course of action for addressing organochlorines is controversial. Some organizations and analysts, including the American Public Health Association and the International Joint Commission (a binational advisory body of the United States 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 advocated continuing the current system of chemical-by-chemical regulation.[24]

Of the 12 POPs addressed by United Nations Environmental Programme (UNEP), all are organochlorines, and four are produced by the PVC lifecycle.

Decisions about the use of PVC in building materials do not require the proper course of action on all organochlorines to be resolved. The question of vinyl should be seen, however, 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 are known to be hazardous; many more have not been specifically identified or evaluated for their environmental behavior and toxicity, and an understanding of the general characteristics 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 United Nations Environmental Programme (UNEP), all are organochlorines, and four are produced by the PVC lifecycle (see table 1). Of the 11 chemicals on the International Joint Commission's Critical Track of hazardous pollutants in the Great Lakes, eight are organochlorines, as are 168 of the 362 Great Lakes contaminants on the IJC's Secondary Track.[25] 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.[26]

Systematic problems with organochlorines

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

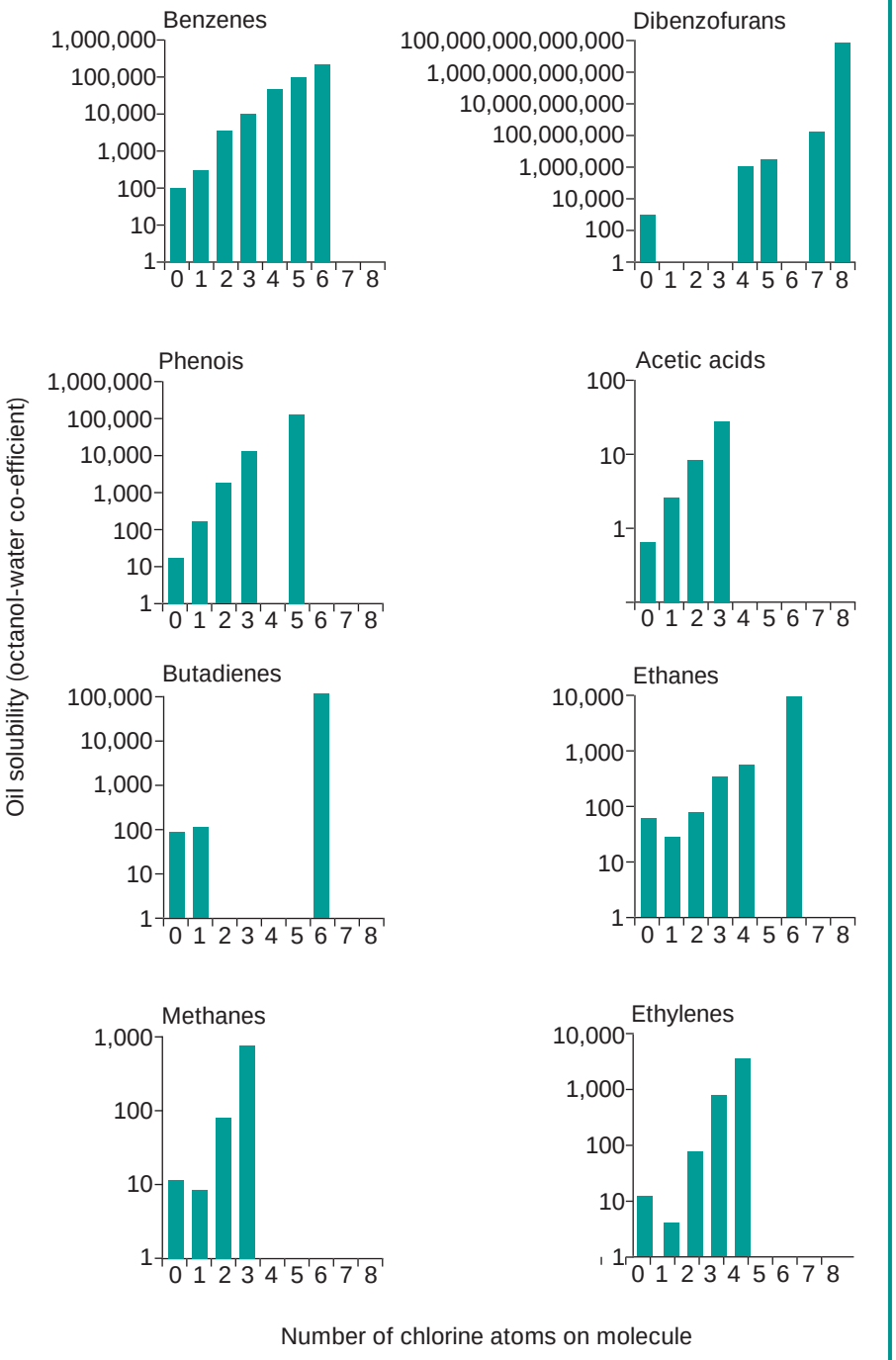
Reactivity of chlorine: Chlorine gas is extremely reactive, combining quickly and randomly with any organic matter it contacts. This property makes it an effective bleach, disinfectant, and chemical feedstock, but also results in the generation of a diverse mixture of by-products, typically containing hundreds or thousands of organochlorines—including dioxins—whenever chlorine is used.[27]

Persistence. The chlorine atom is extremely electronegative, which means it exerts a strong pull on the electrons it shares with carbon atoms in an organochlorine—significantly stronger than the hydrogen atom it usually replaces. The addition of chlorine to organic molecules therefore changes the chemical stability of the resulting substance—often stabilizing 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 degrade into other organochlorines, which may be more persistent and toxic than the original substance. Stability makes organochlorines useful as plastics, solvents, and refrigerants—applications for which long-life and fire-resistance are virtues. This 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, making them useful as chemical intermediates. But 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.[28] Chlorine's impact on the stability of organic molecules thus has one of 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 nondissolvable in water. 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 solubility in fats and oils. The increase in fat solubility applies to the chlorination of virtually any organic substance, and increases with each chlorine atom added.[29] Thus, for example, tetrachloroethylene, hexachlorobenzene and octachlorodibenzofuran, are about one hundred, one thousand, and one billion times more oil-soluble, respectively, than their chlorine-free analogs (figure 2). Oil-solubility makes organochlorines useful as solvents

Figure 2 Effect of chlorine on oil solubility and bioaccumulation on organic chemicals



Note: In all groups, chlorination increases the tendency of a chemical to dissolve fats and oils, and each chlorine atom has a greater effect. Blank cells indicate no data available.

Source: HSDB 1997.

and dielectric fluids, but it is directly responsible for the tendency to bioaccumulate.

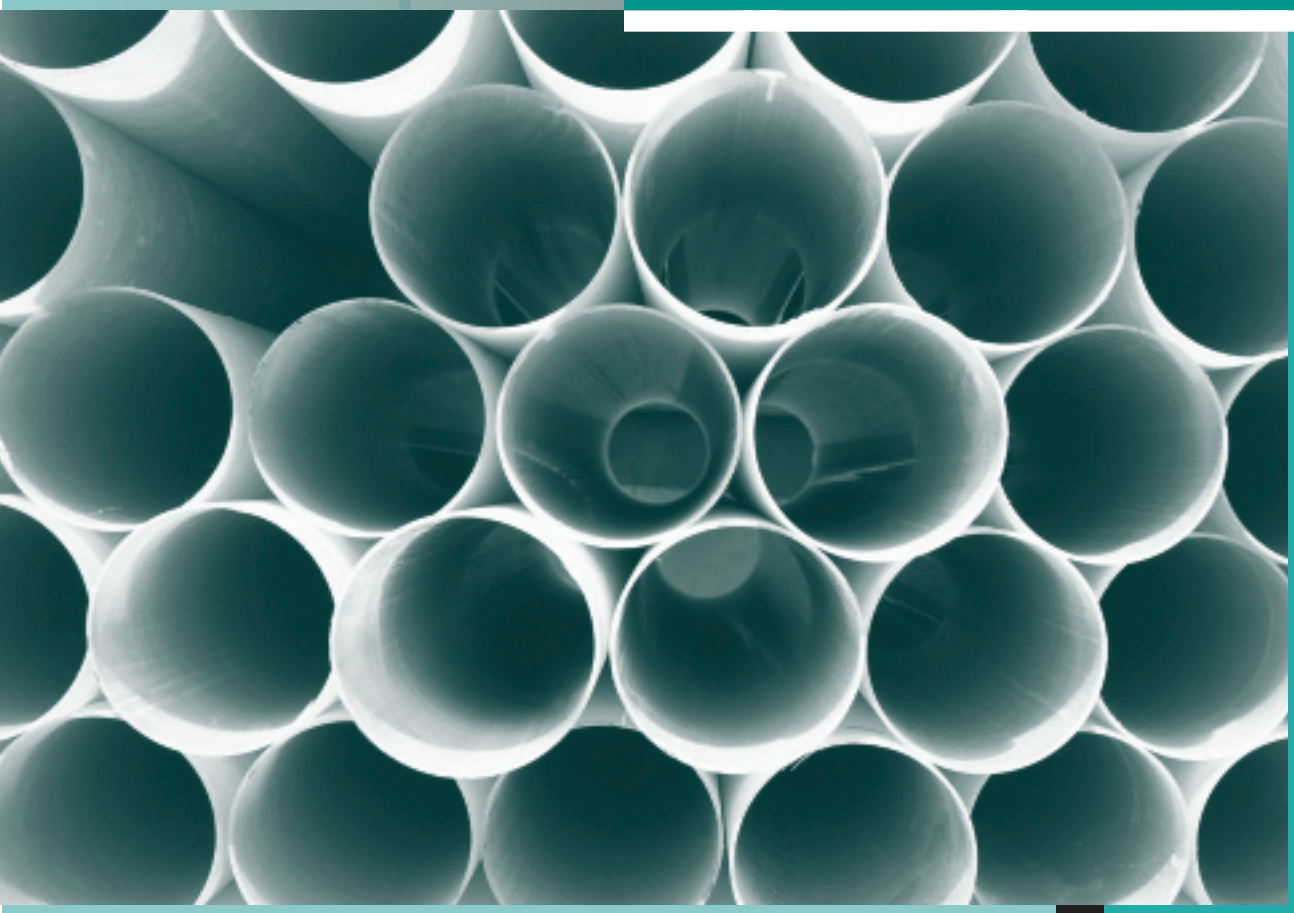
Toxicity. Organochlorines tend to be overwhelmingly toxic. The American Public Health Association has concluded that "virtually all organochlorines that have been studied exhibit at least one of a range of serious toxic effects, such as endocrine dysfunction, developmental impairment, birth defects, reproductive dysfunction and infertility, immunosuppression and cancer, often at extremely low doses, and many chlorinated organic compounds…are recognized as significant workplace hazards."[30] According to a comprehensive independent review of thousands of individual organochlorines, chlorination of organic chemicals "is almost always 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)."[31] The general toxicity of organochlorines makes them useful as pesticides and antibiotics, but also hazardous to humans and wildlife once they enter the environment.

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."

Notes

1. Geiser 2000.
2. European Commission 2000.
3. Tukker et al. 1995.
4. Schneider and Keenan 1997.
5. United Nations Environment Programme 2000.
6. APHA 1996.
7. Tellus Institute 1992.
8. Christaensen et al. 1990. PVC was not judged demonstrably inferior under certain criteria (such as energy use and accident potential during manufacture) to polyurethane, acrylonitrile-butadiene-styrene, and aluminum.
9. KemI 2000; Greenpeace International 2000.
10. European Commission 2000.
11. UBA 1992.
12. European Commission 2000.
13. Greenpeace International 2000.
14. Greenpeace International 2000.
15. Quoted in Greenpeace International 2000.
16. Quoted in Greenpeace International 2000.
17. Greenpeace International 2000.
18. Greenpeace International 2000.
19. Warren 1998; *New York Times* 1998.
20. Mayer 1998.
21. Warren 1998.
22. Thornton 2000; Collins 2001; International Joint Commission 1992; APHA 1994.
23. For a discussion of chlorine-free synthesis technologies, see discussion and references in Thornton 2000.
24. Karol 1995.
25. Great Lakes Water Quality Board 1987.
26. Burmaster and Harris 1982; Miller et al. 1991; Guillette and Crain 1999.
27. See discussion and references in Thornton 2000.
28. Henschler 1994.
29. Solomon et al. 1993.
30. APHA 1994.
31. Henschler 1994.

PVC Production



two

PVC Production

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 (also called 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 to the process is to separate immediately the chlorine from the alkali in a specially designed electrolytic chamber, called a cell. There are three types of chlor-alkali cells 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 connected cells. 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 production worldwide is by 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. 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.[1]

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. 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 United States and 25 percent in Europe.[2]

The remaining 7 percent of chlor-alkali production is based on the membrane process, the most recently developed 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 expensive. Because most chlor-alkali plants in the United States and Europe were built decades ago, few use the membrane process. But most new facilities—particularly those in Asia and Latin America—are now constructed with it.[3]

The world chlor-alkali industry produced nearly 39 million metric tons of chlorine in 1997; 27 percent of that was made in the United States. There are 42 chlor-alkali facilities in the United States, but more than 70 percent of production capacity is located at just 12 large plants in the Gulf Coast region of Louisiana and Texas.[4] Because vinyl accounts for more than 40 percent of all chlorine consumption, it is reasonable to estimate that the same proportion of the environmental impacts of chlorine production—releases of toxic substances and energy demand—is associated with the vinyl lifecycle.

Chlorine gas produced each year for vinyl production carries between 1,400 and 7,200 pounds of highly persistent and toxic organochlorine by–products — including HCB, hexachloroethane (HCE), PCBs, OCS, tetrachlorobenzene—into the world's economy.

Dioxin and other organic by-products

From the moment that chlorine gas is formed in the chlor-alkali cell, it will react with any organic matter present to form organochlorines. For this reason, manufacturers purify raw materials and equipment surfaces carefully to remove as much organic material as possible. Nevertheless, carbon-containing substances remain as trace impurities—in plastic materials or from the graphite electrodes used in some types of chlor-alkali cells.[5] Chlorine combines with these organic contaminants to form persistent organochlorine by-products, such as HCB and hexachloroethane (HCE), which are found in the chlorine product itself.[6] Chlorine gas can also be contaminated with PCBs, octachlorostyrene (OCS), and tetrachlorobenzene. The concentrations are

moderate—40 to 210 parts per billion (ppb)[7] —but the quantities are significant: based on the levels found, the chlorine gas produced each year carries between 1.6 and 8.2 tons of these highly persistent and toxic by-products into the world's economy.[8] Based on an attributable fraction of 40 percent, the chlorine used for vinyl contains between 1,400 and 7,200 pounds of these substances annually.

Severe contamination of fish and sediments with OCS—an extremely persistent, bioaccumulative POP—has been documented near eight North American chlorine producers.

Much greater quantities of organochlorine contaminants are deposited in chlorine production wastes. Swedish researchers have identified high concentrations of dioxins and furans[9] in the sludges from spent graphite electrodes used in chlor-alkali cells,[10] and high levels of polychlorinated dibenzofurans have been found in the blood of Swedish chlor-alkali workers.[11] The levels were up to 650 ppb, with TEQ values up to 28 ppb. (Because dioxin-like compounds cause toxicity through a common mechanism, mixtures of these substances can be expressed relative to 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.)

Severe contamination of fish and sediments with OCS—an extremely persistent, bioaccumulative POP—has been documented near eight North American chlorine producers. Additionally, large-scale OCS contamination of sediments in Lake Ontario has been traced to disposal of spent chlor-alkali electrodes.[12] OCS is now a global contaminant, with considerable levels found in the Canadian arctic—and chlor-alkali manufacture is considered an important source of that contamination.[13]

As a result of these problems, 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 near 5 ppt (TEQ).[14] 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.[15] In 1997, the UK Environment Agency 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.[16]

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 into the environment.[17] As recently as the 1980s, the chlorine industry was second only to fossil fuel combustion as a mercury source in Europe.[18]

Many mercury-cell plants have been retired in the past two decades, and controls on existing plants have improved, but chlor-alkali facilities remain a 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.[19] According to the Chlorine Institute (an industry organization) the chlor-alkali industry in the United States consumes 176,769 pounds of mercury per year.[20] By definition, materials consumed are not recycled but are released directly to the environment, into wastes, or in the product itself. Without providing documentation, the Vinyl Institute has argued that only 20 percent of the chlorine produced by the mercury process in the United States is used to produce PVC.[21] Based on these two figures, vinyl manufacture in the United States alone is associated with the release of more than 35,000 pounds (about 16 metric tons) of mercury into the environment each year.

The U.S. industry's figures probably significantly underestimate actual mercury releases. More detailed estimates, derived by superior mass-balance accounting methods, are available based on studies conducted

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

	Metric tons of mercury per year[1]	PVC attributable portion[2]
Total 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

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 and that an equal fraction of the chlorine produced in mercury cells is used for PVC; the Vinyl Institute has argued that the attributable fraction for mercury is 20 percent in the United States.

Note: These data are based on mass balances prepared by the chlorine industry for facilities in Europe and may not accurately represent global averages.

Source: Ayres 1997.

by Euro-Chlor, the trade association of the European chlorine industry.[22] This information indicates that 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 extrapolate from the Euro-Chlor estimates, 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). Based on these figures and 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 is released into air, water, or landfills. If we use the Vinyl Institute's U.S.-based estimate that 20 percent of the chlorine produced by the mercury process is used to produce PVC, production of chlorine for vinyl would account for the release of 46 tons per year of mercury each year. The actual worldwide totals are likely to be even higher since the well-regulated facilities of Europe are not likely to be representative of those in other regions of the world.

Mercury is an extremely toxic, bioaccumulative global pollutant. Mercury compounds cause irreversible health damage to wildlife and humans—especially to developing children, resulting in birth defects, impaired neurological development, kidney damage, and severe neurological destruction.[23] The most tragic and infamous example of mercury

pollution happened 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 population—among whom a diet of fish played a key role—suffered high mercury exposures. In the early 1950s, symptoms of chronic mercury poisoning, including neurological toxicity, paralysis, coma, and death began to appear in adults in the community, and a horrifying outbreak of severe birth defects and mental retardation occurred in children. Ultimately, mercury poisoning killed hundreds and injured more than 20,000 people in the Minimata area.[24] 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.[25] This pattern suggests that mercury releases from the chlor-alkali process are likely to have played a role in the Minimata epidemic.

The PVC lifecycle is associated with the consumption of 92 metric tons of mercury (202,400 pounds) per year, of which the majority is released into air, water, or landfills.

Today, mercury exposure remains a major environmental health problem. According to a recent report by the U.S. National Academy of Science, "Individuals with high [mercury] exposure from frequent fish consumption might have little or no margin of safety—in other words, exposures of high-end consumers are close to those with observable adverse effects. Those most at risk are children of women who consumed large amounts of fish and seafood during pregnancy. The committee concludes that the risk to that population is likely to be sufficient to result in an increase in the number of children who struggle to keep up in school and who might require remedial classes or special education."[26]

Mercury cells are now banned in Japan and are gradually being phased out in the United States and Europe as well, 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 mer-

cury into local waterways; more than a decade later, mercury levels in the sediment remained extremely high.[27] 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.[28] In India, a 1990 study of waterways around a chlorine facility documented severe mercury contamination of fish and sediments.[29]

The global chlor-alkali industry consumes about 117 billion kilowatt-hours of electricity each year, equivalent to the annual power production of about 20 medium-sized nuclear power plants.

Major energy consumption

Chlorine production 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.[30] This quantity is about 1 percent of the world's total demand for electricity,[31] costs about $5 billion per year,[32] and is equivalent to the annual power production of about 20 medium-sized nuclear power plants.[33] As a major energy consumer, chlorine chemistry contributes considerably to all the environmental problems—global warming, air pollution, acid rain, mercury emissions, 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 vinyl production 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 required for the manufacture of PVC is beyond the scope of this document.

SYNTHESIS OF EDC AND VCM

Releases of EDC and VCM carcinogens

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

Undoubtedly, extremely large quantities of EDC and VCM are released into the air and water each year. As part of the U.S. Toxics Release Inventory (TRI), manufacturers of PVC and its feedstocks self-reported emissions of 887,000 and 798,000 pounds of VCM and EDC, respectively, directly into the environment in 1998; an additional 2 million pounds of VCM and 27 million pounds of EDC were reported sent to sewage treatment plants or off-site waste facilities.[34] These figures may significantly underestimate actual releases, however, because of widely recognized problems with the TRI database. Reported emissions are not based on actual monitoring of releases, and estimation methods vary greatly among facilities. Most importantly, TRI release estimates are self-reported by the industry and are thereby subject to no independent verification.

More valid figures come from estimates made by the Norwegian government in 1993. Based on a detailed mass balance conducted at a Norwegian facility, specific estimates of per-ton releases of EDC and VCM from vinyl manufacture were made (table 3). Extrapolating these estimates using 1990 vinyl production figures,[35] we can calculate that the PVC industry releases at least 100,000 tons each of EDC and VCM into the air each year—plus more than 200 tons of EDC and 20 tons of VCM into surface water. Worldwide VCM production has increased by about 50 percent in the past decade,[36] which increases the associated estimates of total releases by a similar fraction. The actual total may be even higher since this estimate was extrapolated from emissions at a single facility in Norway: manufacturers in many other nations have less advanced pollution control equipment and less careful plant operation than this relatively modern, well-regulated facility.

Table 3 Releases of EDC and VCM in PVC production stages; Norwegian government estimates, 1993

	Grams released per ton of product			
Product	EDC to air	EDC to water	VCM to air	VCM to water
EDC synthesis (1 ton)	–	7	–	–
VCM synthesis (1 ton)	5000	1	1000	1
PVC polymerization (1 ton)	–	–	5100	–

– Indicates releases <1 g/ton.

Source: SFT 1993.

EDC and VCM are not particularly persistent—but both are highly toxic. Releases of these compounds therefore pose the greatest hazards for communities and ecosystems near EDC/VCM manufacturing facilities. The facility studied in Norway, for instance, releases 40 to 100 tons of EDC each year directly into the local atmosphere.[37] In the United States, some 12.5 million people 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 highest exposures to these compounds in workplace air—81,000 U.S. workers are regularly exposed to vinyl chloride, while 77,000 are exposed to EDC.[38]

According to the International Agency for Research on Cancer and the U.S. National Toxicology Program, VCM is a known human carcinogen, and EDC is a probable human carcinogen. Studies of workers exposed to VCM have shown an unambiguous increase in liver cancer. Four out of five studies also report an increased risk of brain cancer that is statistically significant in a combined analysis.[39] Both EDC and VCM cause a variety of other toxic effects, including immune suppression, liver damage, neurological toxicity, and testicular damage.[40]

Since the carcinogenicity of VCM was established, government regulations have required considerable reductions in worker VCM exposure. In the United States, workplace VCM levels within the 1–5 ppb range are now required—orders of magnitude lower than typical concentrations of the 1950s and 1960s—but much higher levels continue to occur in facili-

ties elsewhere.[41] 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 the typical U.S. concentrations. In China, the air in dormitories for plastics industry workers and their families contains VCM levels that exceed the permissible workplace level in the United States.[42]

Although occupational exposure to EDC and VCM has declined significantly in the United States, even the much lower levels that now characterize the domestic vinyl industry remain a matter of concern, because there appears to be no safe dose of these compounds. The mechanisms by which EDC and VCM cause cancer have been elucidated, and it is now clear that both substances are genotoxic—causing irreversible damage to DNA. Indeed, VCM has been shown to form chemical bonds with DNA, leading to mutations that abolish natural controls over cell differentiation and proliferation.[43] Currently accepted biological theory indicates that mutations in a single cell can result in the development of a malignant tumor; similarly, a single molecule of a genotoxic substance can damage DNA. Carcinogens such as VCM therefore are not likely to have a threshold below which they do not increase the risk of cancer. That is, any exposure to this carcinogen poses some risk of cancer, and the magnitude of the risk increases with the level of exposure.[44] The same is true of other health impacts mediated by DNA damage, including certain birth defects and genetic diseases. Thus, reduced levels of EDC and VCM in U.S. PVC production facilities reduce but do not eliminate the occupational health risks from these chemicals.

EDC and VCM are genotoxic—causing irreversible damage to DNA—and are not likely to have a threshold below which they do not increase the risk of cancer.

The health effects of community EDC and VCM exposure remain largely unstudied. It is clear, however, that severe environmental contamination and social disruption have occurred in several communities near EDC and VCM production facilities. As an example, Reveilletown, Louisiana, was once a small African-American community adjacent to a EDC/VCM 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 complained of health problems and brought a lawsuit 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 since been demolished.[45]

The next year, as concern about air and groundwater pollution grew around Dow Chemical's EDC/VCM facility five miles from Reveilletown 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.[46] On the other side of the state, in Lake Charles, Louisiana, PPG and Vista Chemical manufacture EDC and VCM, which, along with by-products of their synthesis, now contaminate water and sediments in the Calcasieu Estuary.[47] Residents here 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.

Formation of PCBs and other organochlorine by-products

EDC/VCM synthesis generates large quantities of persistent, bioaccumulative by-products. EDC is made in two ways: ethylene is chlorinated with chlorine gas, or ethylene is oxychlorinated with hydrogen chloride that has been formed as a waste in other synthesis processes. Most EDC producers use both methods in a linked cycle, since 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 (less volatile than EDC). The waste quantities 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.[48]

In general, the heavy ends are discarded and the light ends reprocessed in other chemical reactions. EDC goes on to be pyrolyzed—heated in the absence of oxygen—to yield VCM. By-products formed in this process include chlorinated ethanes, chlorobenzene, chlorobutadiene, ethylenes, methanes, and large amounts of complex but uncharacterized waste tars.[49] According to industry sources, the total amount of chemical wastes produced in the various processes involved in EDC/VCM synthesis is

estimated as between 3 to 10 percent of the VCM yield—a staggering 570,000 to 1.9 million tons of by-products each year.[50]

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 data from industry and environmental groups. In 1990, Dow Chemical analyzed its EDC heavy ends and found them to be about 65 percent chlorine, including large quantities of highly persistent, bioaccumulative, and toxic substances: 302 ppm PCBs, 0.3 percent HCE, 1.2 percent hexachlorobutadiene (HCBD), and 30.6 percent unidentified compounds.[51] If Dow's analysis is representative of heavy ends in general, then EDC oxychlorination results in the worldwide production of a remarkable 20,000 pounds of PCBs each year, even though these compounds were banned from intentional production some 20 years ago.[52]

EDC oxychlorination results in the worldwide production of a remarkable 20,000 pounds of PCBs each year, even though these compounds were banned from intentional production some 20 years ago.

The vinyl industry claims that these by-products are "contained" within the production equipment and thereby never released into the environment. But releases and contamination have clearly occurred. In 1993, chemists from the Greenpeace 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 ppm range, and HCB and HCBD were present at the remarkable levels of 1.9 and 0.6 percent by weight.[53] The following 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 substances with a range of chemical structures, many of them highly persistent, bioaccumulative, and toxic.[54]

Dioxin and furan formation

With PCBs and HCB 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. In research at a chemical plant in Russia, substantial quantities of dioxins and furans were identi-

fied in the wastewater and wastewater sludge from the pyrolytic production of VCM from EDC—as well as in the waste incinerator emissions.[55] But the largest quantities of dioxin are formed 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:

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.[56]

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 make this method of producing EDC one of the world's largest—if not the largest—sources of dioxin.[57] These authors estimated that 419 grams of dioxin TEQ are formed per 100,000 tons of EDC produced—a rate equivalent to more than 60,000 grams of dioxin (TEQ) per year from the world vinyl industry.[58] Although not all the dioxins created would be released directly into the environment, this quantity is more than 50 times the annual dioxin emissions from all trash incinerators in the United States—the largest known source of U.S. dioxin emissions. It is also double the 25,000 grams of dioxin (TEQ) per year that EPA estimates are carried into the environment by contaminated pentachlorophenol[59]—the largest identified source of dioxin to any environmental medium.

This research generated considerable public and scientific concern. As a result, 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.[60] How much dioxin is actually formed remains uncertain, because both studies have strengths and weaknesses. On one hand, the Dutch analysis may be a more accurate indicator of total dioxin generation because the researchers captured and analyzed 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 equipment surfaces, products, recirculating materials, and wastes to be completely assessed in a large-scale industrial context. On the other hand, the Dutch study was a laboratory simulation, and the industry analysis took place at a real production facility; there may be reasons why the simulation caused more dioxin to form than a real-world synthesis of EDC.

Dioxin releases in wastes

Whatever the exact quantities, there can be no doubt that dioxin generation occurs in far from negligible amounts. In 1994, government scientists found dioxins at high concentrations (up to 414 ppb TEQ) in sludges from a fully modernized EDC/VCM plant in Germany, refuting the claim that only outdated EDC/VCM technologies produce dioxin.[61] 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 substantially 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.[62]

In the United States, wastes from Vulcan Chemical's EDC plant in Louisiana have been found to contain dioxins and furans at the concentration of 6.4 ppm (TEQ), which makes them among the most dioxin-contaminated wastes ever discovered, on a par with wastes associated with the manufacture of Agent Orange.[63] In 2000, Norwegian scientists reported finding "extremely high" concentrations of dioxins and furans—26.6 ppm (60.7 ppb TEQ)—in the sludges from a VCM/EDC manufacturing plant. 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 region of the plant were 2 to 9 times higher than levels in fish caught from a relatively uncontaminated local comparison area. Considerable quantities of PCBs were also found in sediments near the plant and were attributed to the production of EDC/VCM.[64]

Residents in the Mossville area—located across the road from a large vinyl chloride manufacturing facility—have blood levels of dioxins and furans (TEQ) averaging nearly three times those of a comparison population.

The wastes from EDC synthesis have one of two major destinations. In some facilities, wastes are used in the manufacture of chlorinated solvents—wherein the contaminants end up in the wastes or products from those processes. In other facilities, the wastes are disposed of, usually by incineration. (A discussion of environmental releases from this practice follows). But not all 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,[65] and local and regional contamination of water, sediments, and shellfish has been linked to EDC/VCM manufacturers in Europe and the United States.[66] For example, severe dioxin contamination of sediments in Italy's Venice Lagoon has been linked to an EDC/VCM manufacturing facility.[67] In the Netherlands, levels of dioxins in sediment samples in the River Rhine jump dramatically immediately downstream from an EDC/VCM manufacturing plant.[68] 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.[69]

These dioxin releases contribute not only to environmental contamination but also to human exposure. In Lake Charles, Louisiana, the U.S. Centers for Disease Control 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) averaging nearly three times 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 nearly double those in store-bought eggs; but the sample size was too small for statistical 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 production wastes, or from some other process at facilities in the area.[70]

Other by-products are also present near chemical plants that make PVC feedstocks. In Lake Charles, Louisiana, the National Oceanic and Atmospheric Administration (NOAA) found 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 high levels of organochlorines in the water and sediment. In one portion of the estuary near PPG's facility, concentrations of HCB, HCBD, and HCE exceeded 1,000 ppm in sediment; in some samples, these three by-products represented from 0.1 percent to an extraordinary 4.8 percent of the sediment's total mass.[71]

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.[72] As previously discussed, 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 revealed statistically significant excesses of brain cancer and neurological effects among VCM-exposed workers. In the United States, worker exposure in this sector has declined in recent decades.

Occupational health & phthalates

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, chemical and plastics industries in the United States reported releasing 213,621 pounds of the plasticizer diethylhexyl phthalate (DEHP) directly into the air, plus 71,004 pounds into the land.[73] 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.[74] 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."[75]

Cancer risk from PVC manufacturing

There are few direct studies of health impacts and 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 study, a variety of occupational exposures were investigated for a possible link to testicular cancer. Work in plastics production was found to cause a 2.9-fold increase in the risk of testicular cancer.[76] The second study tested and clarified the relationship between plastics work and testicular cancer. In this report, using a case-control study of 163 men with testicular cancer and 326 without the condition, occupational work with PVC plastic was associated with a 5.6-fold increase in the risk of seminoma—a form of testicular cancer that occurs later in life and may thus plausibly be caused by occupational exposure. The increased risk was statistically significant and highest among those men with the greatest cumulative exposures. No significant increases in the testicular cancer rate were seen among men who worked with other types of plastics. Because exposure to endocrine-disrupting compounds can lead to testicular cancer, the authors hypothesized that exposure to phthalates used as plasticizers in PVC—some of which are known endocrine disrupters and testicular toxicants—may be the specific cause of the increased risks.[77] 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.[78]

Occupational work with PVC plastic was associated with a 5.6-fold increase in the risk of seminoma—a form of testicular cancer.

DISPOSAL OF EDC/VCM WASTES

The organochlorine-rich heavy ends produced by EDC/VCM manufacture are regulated as a hazardous waste in the United States. The majority of these are disposed of by incineration, usually in on-site furnaces at the production facility. In theory, a properly designed and operated incinerator converts organochlorines by oxidation into carbon dioxide, hydrogen chloride, and water. Real-world combustion systems, however, never take this reaction to completion for all the compounds fed into them. Most compounds are 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."[79]

Dioxin and other byproduct formation in incinerators

By-products form in incinerators for the same reasons as 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—wastes are complex mixtures of diverse materials that can never be uniformly blended. Further, combustion is by nature a stochastic process of bond breakage and formation; at high temperatures, 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 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.[80]

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."[81]

By-products form from diverse and unpredictable reactions not only in the furnace but also in the cooler zones, where control over combustion conditions is nearly irrelevant.[82] Dioxins can even form in pollution control devices or smokestacks, where chlorine gas, hydrochloric acid, or organochlorine precursors 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.[83]

Incinerators not only destroy organochlorines, they also manufacture them.

This means that incinerators not only destroy organochlorines, as they are supposed to, but also manufacture them. EPA estimates that PICs formed in the incineration process number in the thousands.[84] Of these, some have 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 in which chlorine is present.[85] It is much more challenging to analyze PICs in the stack gas of real-world incinerators, but more than 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, dioxins, ethers, furans, naphthalenes, PCBs, phenols, and thiophenes.[86]

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.[87] There is good reason to be concerned about these mystery compounds because at least some appear to be in the same toxicological family as dioxins. German researchers measuring the dioxin-like toxicity of trash incinerator fly ash

using a biological test found that toxicity was up to five times greater than could be accounted for by the amount of dioxins, furans, and PCBs in the ash.[88] The remaining dioxin-like effect was presumably caused by scores of other compounds—such as chlorinated naphthalenes, diphenyl ethers, thiophenes, and many others—that can cause similar health effects but were not specifically measured.

Efficient incineration and high emissions

The total quantity of PICs and unburned wastes emitted from incinerators is not known precisely, but it appears to be 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.

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.

- EDC heavy ends are burned in such immense quantities that even if all incinerators achieved 99.99 percent DRE, they would still emit more than 6,600 pounds per year of unburned hazardous wastes into the air in the United States alone.[89]

- Much greater amounts of organochlorines are released as PICs. "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 1 percent of the organic matter fed to them.[90] This estimate suggests that incineration of heavy ends from vinyl manufacture would emit some 660,000 pounds of PICs each year.

- 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 into the air, without any effect on the calculated DRE.

- DREs are calculated from an incinerator's performance when burning test chemicals that are fed in 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 ppb or ppm 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 significant amounts of these hazardous substances will escape intact from incinerators.[91]

- DREs measured in trial burns are unlikely to reflect emission rates during routine operation because trial burns involve 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.[92] Further, the standard trial burn protocol allows the measurement of emissions to stop when the feed of waste chemicals to the incinerator stops, but 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.[93]

For 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.

FAILURE OF THE VINYL INSTITUTE'S SELF-CHARACTERIZATION OF DIOXIN RELEASES

The Vinyl Institute (VI) has argued that its role in dioxin formation is minimal, primarily based on its own study, called the "dioxin self-characterization."[94] In this report, the Vinyl Institute concludes that the U.S. PVC industry releases about 13 grams of dioxin (TEQ) per year to the environment. This estimate is also the basis for the Vinyl Institute's contention that vinyl production is responsible for only a small fraction of identified dioxin releases in the United States.[95]

Pathways that contain the largest amounts of dioxin were completely ignored.

There are three reasons to be skeptical of the industry's reassurances about its dioxin emissions:

- Even if the Vinyl Institute's estimate is accurate, 13 grams of dioxin per year is still highly significant, justifying action to reduce vinyl consumption. U.S. EPA's current standard for the acceptable daily dioxin intake of an average adult (weighing 70 kg) is 0.153 billionths of a gram per year.[96] Based on its own estimates, then, the vinyl industry's annual releases of dioxin into the environment equal the acceptable annual dose for about 85 billion people. (Not all of the dioxins released by the industry will result in direct human exposures, of course. The point of this calculation is to demonstrate that, because dioxin is so exquisitely toxic, a quantity of dioxin that appears small on a mass basis is in fact extremely significant from a toxicological perspective.)

- The Vinyl Institute's figures on its dioxin releases are likely to be gross underestimates because they omit the majority of the dioxin produced during the vinyl lifecycle. The industry's self characterization analyzed several potential pathways for dioxin release, finding low to moderate quantities of dioxins and furans in samples of EDC, PVC products, air emissions, and wastewater and sludge from its treatment. But numerous pathways that contain the largest amounts of dioxin—along with many PVC-related processes that are major dioxin sources—were completely ignored. No data, for example, were gathered on dioxin contamination of chemical streams that

re-circulate in the manufacturing process, of light ends and other wastes used in other synthesis processes, and—most importantly, because these are known to be so severely contaminated—heavy ends, tars, and other hazardous wastes that are sent to disposal facilities. Nor did the program address what is apparently the largest PVC-related dioxin source—the burning of vinyl in incinerators, smelters, and accidental fires. Thus, the industry's estimates are grossly incomplete and do not effectively refute the argument that the lifecycle of PVC is a major dioxin source.

The Vinyl Institute report did not address what is apparently the largest PVC-related dioxin source—the burning of vinyl in incinerators, smelters, and accidental fires.

- The Vinyl Institute's estimates have not been independently verified. In this self-characterization, the PVC industry decided when and where to take samples, how to collect them, how to analyze dioxin content, which data to present, and how to interpret this data before submitting their results to EPA. While an independent panel reviewed the submission, the industry chose which data the panel saw. Information about the samples—including which facility they came from—was completely confidential, so neither reviewers nor the public had the opportunity to determine whether sampling times and locations accurately represented typical dioxin releases. Most importantly, no one was able to independently evaluate, confirm, or act on the information.

It would be naïve to take at face value the industry's own estimates of the magnitude of its releases of dioxin—a substance that is the subject of major public concern and regulatory activity—particularly when those estimates conflict with a large body of information gathered by independent sources, such as those cited above.

Notes

1. Leder et al. 1994
2. Leder et al. 1994.
3. Leder et al. 1994.
4. Leder et al. 1994; see also Thornton 2000.
5. Schmittinger et al. 1986.
6. HSDB 1997.
7. Hutzinger and Fiedler 1988.
8. My calculation of annual loadings assumes world production of 39 million metric tons of chlorine each year (Leder et al. 1994).
9. Polychlorinated dibenzofurans (PCDFs, or furans) are structurally related to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD, known colloquially as dioxin), the best studied and most hazardous of the dioxin-like compounds, a large group of structurally and toxicologically related group of compounds that includes not only

the furans but also some PCBs, chloronaphthalenes, and many others), together referred to as dioxins or dioxin-like compounds.
10. Rappe et al. 1991.
11. Svennson et al. 1993.
12. Kaminski and Hites 1984.
13. Barrie et al. 1997.
14. Andersson et al. 1993.
15. This research is summarized in Versar, Inc. 1996 and EPA 1998.
16. Environment Agency 1997.
17. Lindqvist et al. 1991.
18. Lindqvist et al. 1991; Pacyna and Munch 1991.
19. Ayres 1997.
20. Chlorine Institute 2000.
21. Burns 2000.
22. The data from Euro-Chlor, presented in Ayres 1997, are the most comprehensive available. The data are based on a mass balance method, so that all mercury consumed is accounted for in one way or another. My calculation of total mercury releases from the chlor-alkali industry uses this range and assumes 39 million tons 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 International 1993).
23. ATSDR 1998.
24. Harada 1995; Davies 1991.
25. Hill and Holman 1989.
26. National Academy of Sciences 2000.
27. Airey and Jones 1970; Johnston et al. 1993.
28. Maserti and Ferrara 1991.
29. Panda et al. 1990.
30. Energy requirements vary somewhat among the chlor-alkali cell types: the mercury cell requires 3310-3520 kilowatt-hours per ton of chlorine, the diaphragm 2,830 kilowatt-hours per ton, and the membrane process 2,520 kilowatt-hours per ton. Based on the proportion of each cell type in the world industry, the average energy requirement for the industry overall is slightly under 3,000 kilowatt-hours per ton (SRI International 1993).
31. SRI International 1993.
32. Assuming an average global cost of 4.2 cents per kilowatt-hour for chlor-alkali customers (SRI International 1993).
33. In the United States, 109 nuclear plants generated 673 billion kilowatt-hours of electricity, for an average of about 6 billion kilowatt-hours per plant per year (Energy Information Administration 1996).
34. National Institutes of Health 1998.
35. Production estimates are 29,137 kilotons EDC per year, 18,495 kilotons of VCM per year, and 18,135 kilotons of PVC per year (SRI International 1993).
36. Kielhorn et al. 2000.
37. SFT 1993.
38. ATSDR 1993; ATSDR 1995.
39. Kielhorn et al. 2000.
40. ATSDR 1993; ATSDR 1995.
41. Kielhorn et al. 2000.
42. Kielhorn et al. 2000.
43. Kielhorn et al. 2000.
44. Pitot and Dragan 1991.
45. Bowermaster 1993.
46. Bowermaster 1993.
47. Curry et al. 1996.
48. My calculations assume that about 15 million metric tons per year of EDC produced by oxychlorination (half of world production (SRI International 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 Centre for Technology and Policy Studies 1996). This figure is slightly lower than that of Rossberg et al. (1986), who estimate 2.3 and 2.9 kilogram heavy and light ends per ton of VCM produced, respectively. Use of more recent figures for global PVC production rates (Kielhorn et al. 2000) increases this estimate by about 50 percent.
49. Rossberg et al. 1986.
50. 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.
51. Dow Chemical 1990.
52. This calculation assumes global production of 32 kiltons of EDC heavy ends per year, as discussed in the section above. Use of more recent figures for global PVC production rates (Kielhorn et al. 2000) would increase this estimate by about 50 percent.
53. Johnston et al. 1993.
54. Costner et al. 1995.
55. Khizbullia et al. 1998.
56. ICI Chemicals and Polymers 1994.

57. Evers 1989.
58. Based on early 1990s world production of EDC by oxychlorination of about 15 million tons per year (see note above).
59. EPA 1998.
60. The conclusions from this study are summarized in SFT 1993.
61. Lower Saxony Ministry of Environmental Affairs 1994.
62. 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 grams TEQ per year from this plant alone. Based on this estimate, all oxychlorination processes would constitute one of the world's largest sources of dioxin (Environment Agency 1997).
63. Costner et al. 1995.
64. Isosaari et al. 2000.
65. DTI 1995; Environment Agency 1997; SFT 1993.
66. Contamination in the UK is described by Environment Agency 1997; in Germany by Lower Saxony Ministry of Environmental Affairs 1994; and in the United States by Curry et al. 1996.
67. Ramacci et al. 1998.
68. Evers et al. 1988.
69. Evers et al. 1993; Evers et al. 1996.
70. U.S. Centers for Disease Control 1999.
71. Curry et al 1996.
72. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
73. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
74. Rudell et al. forthcoming.
75. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
76. Hardell et al. 1998.
77. Hardell et al. 1997; Ohlson and Hardell 2000.
78. Hansen 2000.
79. EPA 1990.
80. EPA 1989.
81. UKDOE 1989.
82. Dellinger et al. 1988; EPA 1994b.
83. Gullett 1990.
84. EPA 1989.
85. 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).
86. Trenholm and Lee 1986; Trenholm and Thurnau 1987; Dellinger et al. 1988; Chang et al. 1988; EPA 1987a and 1987b; Wienecke et al. 1995.
87. EPA 1990.
88. 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.
89. Assuming incineration of 30,000 tons of EDC heavy ends per year.
90. EPA SAB 1985.
91. Kramlich et al. 1989; Trenholm et al. 1984.
92. 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."
93. Licis and Mason 1989.
94. Vinyl Institute 1998.
95. Burns 2000.
96. EPA 1985. The "acceptable" dose (0.006 pg/kg of body-weight/day) is the daily exposure that poses a calculated lifetime cancer risk of one per million.

Use of PVC Products

three

Use of PVC Products

PVC is not bioavailable, so the polymer itself is not toxic during 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—that can transform the plastic into hazardous by-products.

BY-PRODUCT FORMATION

Some portion of the diverse organochlorine 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 at concentrations ranging from 0.86 to 8.69 ppt TEQ.[1] 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.[2] 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.[3] The levels were very low, but any quantity of dioxin in consumer products is a matter of concern.

INDOOR AIR QUALITY: RELEASE OF TOXICANTS

Because chemical additives are present in PVC in large amounts, they are particularly problematic. 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.[4] Flexible PVC—including

flooring, roofing membranes, and wall coverings—accounts for more than half of all vinyl demand, while the remainder is rigid, unplasticized materials such as pipes and siding.[5] 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 each year in the worldwide production of PVC.[6] Vinyl accounts for more than 90 percent of the total consumption of phthalates, so the health and environmental impacts of phthalates are overwhelmingly attributable to PVC.[7]

Vinyl accounts for more than 90 percent of the total consumption of phthalates, so the health and environmental impacts of phthalates are overwhelmingly attributable to PVC.

The additives are not chemically bonded to the PVC polymer but are mixed into the plastic during its formulation. Over time, these additives leach out of vinyl products, entering the air, water, or other liquids with which the product comes in contact. 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.[8] The common practice of storing blood and drug formulations in PVC bags causes phthalates to leach into the container's contents, which can result in substantial short-term phthalate exposures for the recipient.[9] Newborn infants receiving a single blood transfusion have been found to have extremely high levels of phthalates in their systems.[10] When exposure is repeated, blood levels of phthalates can be 100 to 1,000 times greater than "background" and can reach levels at which liver damage and birth defects can occur in animals.[11] Phthalates are also released in significant quantities into saliva when small children suck on vinyl toys and teethers.[12]

Release of phthalate plasticizers

Of particular relevance to the health and environmental impacts of building materials is the release of phthalates into indoor air from flexible PVC. The plastics industry has argued that most phthalates have low vapor pressures; therefore they are not expected to volatilize much.[13] But this prediction is not borne out by experience: empirical data make clear that phthalates are released in considerable amounts from vinyl products into the indoor atmosphere. For example, DEHP levels in indoor air average 20 to 103 nanograms per cubic meter, compared to 0.3 to 4.0 nanograms per cubic meter in outdoor air.[14] 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."[15] According to figures cited by the National Toxicology Program, inhalation accounts for about 15 percent of the average adult's daily intake of DEHP.[16]

The relatively low vapor pressure of most phthalates may explain their tendency to be present on dust particles in higher concentrations than in the vapor phase. One U.S. 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 butyl benzyl phthalate (BBP), which were present in dust at the remarkably high mean levels of 315 and 117 ppm, respectively.[17] Another recent study of indoor air in Norwegian residences had similar findings, reporting an average of 960 ppm (including 640 ppm of DEHP and 110 ppm of BBP) on sedimented dust particles and 1,180 ppm (more than 0.1 percent) on suspended dust particles.[18] This study also found that a large portion of the phthalate-contaminated dust particles were small enough to be taken into the airway and lungs. As discussed later, phthalates impair reproduction and development, and some are suspected carcinogens.

Phthalate connections to 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—the cases of which have been steadily increasing in recent decades—particularly among children. In 1997, an analysis of phthalate levels in indoor air pointed out that MEHP—the primary metabolite of DEHP—induces bronchial hyper-reactivity 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 hyper reactive condition in the lungs. This will increase the risk of a pathological development in addition to aggravation of the effects of other environmental agents."[19]

Three 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 study of 251 Norwegian children with bronchial obstruction, with an equal number of healthy children for comparison, found that the presence of PVC flooring in the home was associated with a statistically significant 1.9-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 between exposure and risk is not a spurious one.[20]

PVC flooring in the home was associated with a statistically significant 1.9-fold increase in the risk of bronchial obstruction.

A larger follow-up study in Finland found that children in homes with PVC flooring or wall covering were significantly more likely to suffer from asthma, persistent wheezing, pneumonia, 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."[21]

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. 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. The study examined the staff of four nursing homes—three with PVC flooring, and one without. Workers in the two buildings with damp PVC surfaces were exposed to higher levels of EH and had significantly increased symptoms of nasal and ocular irritation, as well as of 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 symptoms 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."[22]

Vinyl wall coverings are said to be the major cause of mold and mildew in interiors, according to several building industry sources.

This evidence does not prove that PVC is a major cause of asthma, but it justifies concern about the role of indoor exposure to phthalate plasticizers in relation to this widespread condition and action to reduce exposures.

Toxic mold growth

Vinyl's tendency to trap dampness can create another indoor air problem—the growth of toxic molds. Some molds produce toxic and/or allergenic products, particularly among sensitive individuals. These molds do not normally grow indoors but can grow on persistently damp surfaces that contain nutrients (including gypsum and sheetrock), 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 headaches, skin rashes, memory loss, respiratory problems, lung disease, and brain damage.[23] Vinyl wall coverings, because they are impermeable to water vapor, are said to be the major cause of mold and mildew in interiors, according to several building industry sources.[24] The vinyl industry confirms that vinyl wall coverings have created this situation in many buildings; 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."[25] 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.[26] Because dampness and condensation can occur inside vinyl-sealed walls from temperature and humidity differentials produced by heating and air conditioning systems, however, at least one authoritative building industry source recommends avoiding vinyl wall coverings altogether to prevent mold and mildew growth.[27]

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,[28] leading to a warning by the U.S. Consumer Product Safety Commission. Lead is also known to leach into water carried in PVC pipes that contain lead stabilizers.[29]

But lead continues to be used in building-related materials, as are other hazardous additives. Lead stabilizers are commonly used in pipes, vinyl cables, and window profiles, although their use is greater in Europe than in the United States.[30] Lead accounts for nearly 70 percent of all vinyl stabilizers in Europe, with consumption of more than 51,000 tons of lead in PVC annually, based on 2000 estimates by the European Union.[31] Lead is an infinitely persistent substance and is exquisitely toxic to the developing brain—even in tiny amounts. In November 2000, the Danish government took action to ban the use of virtually all lead compounds, including those in PVC cables, gutters, pipes, roofing, and windows, by no later than 2003.[32]

Lead is an infinitely persistent substance and is exquisitely toxic to the developing brain—even in tiny amounts.

PVC is also associated with other toxic metals. According to the European Commission, 50 tons (110,000 pounds) of cadmium—also a highly neurotoxic and infinitely persistent metal—are used in vinyl each year in Europe, although quantities are declining. Consumption of organotin compounds in vinyl is estimated at 15,000 tons, mostly in rigid films, roofing materials, and clear rigid construction sheeting.[33] Organotins used in vinyl can suppress the immune systems, cause birth defects, damage the liver, bile duct, and pancreas, and may pose hazards to the aquatic organisms when released into the environment.[34] Further, the mono- and di-butyl tin compounds used in PVC are contaminated with tributyl tin (TBT), a potent endocrine-disrupting compound that has caused major damage to marine wildlife populations.[35]

ACCIDENTAL COMBUSTION RELEASES

The possibility of fire is another major hazard associated with the use of PVC products. Vinyl manufactures often stress the materials' fire resistant properties—due to the high fraction of chlorine in PVC—as an advantage for hospitals, schools, and other public buildings. In fact, chlorine's resistance to combustion represents a hazard, not a benefit.

Hydrochloric gas releases

PVC is now ubiquitous in modern buildings and vehicles. When vinyl burns, the primary combustion products are carbon dioxide, hydrochloric acid, and water. 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 fumes and smoke. It can also cause severe damage to computers and other equipment.[36] When large masses of PVC are present—as in vinyl siding or roofing membranes—the hazards may extend to building occupants and the surrounding community.

The hazards of PVC in fires have prompted action or positions by a number of expert organizations. The U.S. military has adopted specifications to avoid PVC-jacketed cables in aircraft, space vehicles, and enclosures in which offgassing may occur in the event of fire.[37] 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."[38] The International Association of Firefighters has stated,

> Because of its majority chlorine content, when PVC burns in fires two hazardous substances are formed which present acute and chronic hazards to fire fighters, building occupants, and the surrounding community. These are hydrogen chloride gas and dioxin. Hydrogen chloride is a corrosive, highly toxic gas that can cause skin burns and when comes into contact with the mucous lining of the respiratory tract creates hydrochloric acid, which can cause severe respiratory damage. Exposure to a single PVC fire can cause permanent respiratory disease.
>
> Dioxin is an unintentional by-product of PVC combustion, and would most likely be left behind in ash and debris from a PVC fire. While only small amounts of dioxin may be formed as the result of burning PVC, it is one of the most toxic substances known to science. Dioxin is a known human carcinogen and has been linked to reproductive disorders, immune suppression, and endometriosis, and other diseases in laboratory animals.

Due to its 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.[39]

Dioxin formation

Accidental fires provide very poor combustion conditions, so substantial amounts of dioxin and other organochlorines form as products of incomplete combustion in a vinyl fire.[40] 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.[41] As a result, all accidental fires in buildings containing PVC are likely to generate dioxins and other persistent, bioaccumulative organochlorines. For example, in Germany after a fire in a kindergarten that contained substantial quantities of PVC, scientists measured dioxin levels in indoor soot at concentrations of 45,000 ppt (TEQ)—almost 300 times greater than the German government's health standard. This situation required the building's interior to be completely stripped of PVC—all floors, ceilings, wall coverings, furnishings, and so on—sandblasted, and remediated by hazardous waste experts before children were allowed to enter again.[42] Dioxins have also been identified in the residues from burning automobiles, railway coaches, and subway cars.[43]

In Germany after a fire in a kindergarten that contained substantial quantities of PVC, scientists measured dioxin levels in indoor soot at concentrations of 45,000 ppt (TEQ)—almost 300 times greater than the German government's health standard.

Even a small amount of dioxin from each of the 621,000 structural fires and 421,000 vehicle fires in the United States each year could substantially contribute to dioxin contamination of the environment.[44] 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.[45] As a result, a stockpile of PVC, waiting to burn, is accumulating in immense quantities. Worldwide, more than 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.[46] The Vinyl Institute has argued that PVC fires are probably a relatively small contributor to the total dioxin burden, based on a study that quantified dioxin levels in soot residues within a limited radius of a fire at a plastics facility.[47] But more than 90 percent of the dioxins produced in a structural fire are in the gaseous phase and escape into the atmosphere,[48] and an additional amount is transported

beyond the local area, so this study is likely to have underestimated total dioxin emissions by at least a factor of ten. EPA 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.[49]

While many small fires taken together may constitute an important source of organochlorines, a single fire at a large commercial building, disposal site, PVC factory, or warehouse can generate large quantities of pollutants. A home contains at most a few hundred kilograms of PVC,[50] but a large building may contain much more. For example, a vinyl-lined roof on an average-sized school contains more than 10 tons of PVC,[51] and a plastics warehouse or landfill may have hundreds of tons on-site. After a fire at a plastics warehouse in Binghamton, New York, dioxin levels in soil on the site were found to be more than 100 times greater than other samples from the same community.[52] Elevated dioxin levels have also been reported in a university building after an interior fire in a lecture hall that contained PVC components.[53] According to the European Commission, fires are estimated to account for 6.6 percent of all dioxin emissions from identified sources (table 4).

Lead and phthalate releases

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.[54] Since lead cannot be destroyed by combustion, accidental fires represent an important potential source of lead exposure—a hazard that looms larger as more and more PVC accumulates worldwide in building applications.

Notes

1. SEPA 1994.
2. MAFF 1995.
3. Wagenaar et al. 1996; Carroll et al. 1996.
4. DTI 1995.
5. These figures are for Western Europe (European Commission 2000); in the U.S., where unplasticized vinyl siding is more widely used, the relevant figure may be slightly lower.
6. 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 Centre for Technology and Policy Studies 1996), assuming 24 million tons of PVC production worldwide.
7. TNO Centre for Technology and Policy Studies 1996.
8. DTI 1995.
9. Pearson and Trissel 1993; Goldspiel 1994.

10. Plonait et al. 1993.
11. Dine et al. 2000; National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
12. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a and 2000c.
13. American Chemistry Council 2000.
14. Rudell 2000.
15. Rudell 2000.
16. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
17. Rudell et al. forthcoming.
18. Oie et al. 1997.
19. Oie et al. 1997.
20. Jaakola et al. 1999.
21. Jaakola et al. 2000.
22. Wieslander et al. 1999.
23. Hsieh 2000.
24. Lstiburek and Carmody 1994; Downs 2001.
25. Vinyl Institute 2000.
26. Vinyl Institute 2000.
27. Lstiburek and Carmody 1994.
28. Chicago Tribune 1996.
29. DTI 1995.
30. European Commission 2000.
31. European Commission 2000.
32. ENDS 2000.
33. European Commission 2000.
34. European Commission 2000; Ema et al 1996; Merkord et al 2000; De Santiago and Aguilar-Santelises 1989.
35. Kemi 2000.
36. Markowitz et al. 1989; Markowitz 1989; Wallace 1990.
37. U.S. Navy 1986.
38. Cameron 1996.
39. Duffy 1998.
40. Wirts et al. 1998; Christman 1989; Theisen et al. 1989.
41. TNO Centre for Technology and Policy Studies 1996.
42. Fiedler et al. 1993.
43. Versar, Inc. 1996.
44. Versar, Inc. 1996.
45. UBA 1992; German Environment Ministers 1992.
46. I have extrapolated from figures for Sweden (TNO Centre for Technology and Policy Studies 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).
47. Carroll 1995.
48. Versar 1996; EPA 1998.
49. EPA 1998.
50. Carroll 1995.
51. Assuming a roof size of 80,000 square feet and a mass of 0.31 pounds of vinyl roofing membrane per square foot (Cummings 2001).
52. Schecter and Kessler 1996.
53. Deutsch and Goldfarb 1988.
54. I have extrapolated from figures for Sweden (TNO Centre for Technology and Policy Studies 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 this estimate by about 50 percent.

Disposal of PVC Products

four

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.[1] EPA has estimated that at least 1.5 million tons of PVC are disposed of annually in the United States.[2] Although some building materials have a relatively long lifetime, significant quantities of vinyl are disposed of as cutaways in preconsumer waste and, ultimately, in demolition or renovation 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—often renovated well before their components are physically spent—have relatively short lifetimes.

THE FAILURE OF 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 amount 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 post-consumer vinyl recycling, many formulations are mixed together, destroying the special properties of each. As a result, recycled post-consumer PVC is always of lower quality than the original material, so it can be used only in products without strict material requirements, such as fence posts and speed bumps.[3]

Since recycled PVC is almost never used to make a new version of the original product, down-cycling is a better term for the process than recycling.[4] 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. 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."[5]

In the European Union countries, less than 3 percent of postconsumer PVC waste is recycled—the majority of which is actually down-cycling 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."[6] Sweden, a nation with an ambitious and effecting recycling program, had a total PVC recycling rate of just 2 percent in 1999, nearly all of it preconsumer waste.[7] The European Commission 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.[8] Such low recycling rates, even with time to develop an ambitious program, indicate that PVC is not and cannot be a green building material.

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.[9]

There are also concerns about the potential environmental hazards of PVC recycling. Mechanical recycling of PVC can release additives, including phthalates and stabilizers, which may then be 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.[10]

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.

LAND DISPOSAL LEACHATES AND FIRES

A significant portion of discarded PVC ends up in landfills, and almost all the remainder is burned—the exact proportions vary from one country to another. In landfills, there are three concerns about PVC disposal. First, the persistence of PVC, which typically lasts for centuries in a landfill, presents a significant burden in terms of the demand for landfill space. Second, the release of additives in the plastic can contaminate groundwater. Because phthalates and metals are not chemically bonded to the polymer, they can leach out of disposed products into landfill leachate, eventually contaminating groundwater.[11]

EPA estimates that landfill fires may emit on the order of 1,000 grams of dioxins and furans (TEQ) into the air each year in the United States, potentially making them the largest U.S. dioxin sources to the air.

Third, fires can occur during or after the disposal process, releasing hazardous substances into the air, including dioxins and metals. In Hamilton, Ontario, for example, after some 200 tons of PVC burned at a plastics recycling facility, samples of ash, soot, and tree leaves from the fire area were found to contain elevated quantities of dioxins.[12] Of particular concern are landfill fires, which occur with some regularity at landfills and waste storage sites where large quantities of PVC are present. Data on dioxin releases from landfill fires are limited, but EPA estimates that landfill fires may emit on the order of 1,000 grams of dioxins and furans (TEQ) into the air each year in the United States, potentially making them the largest U.S. dioxin sources to the air.[13]

INCINERATION

Role of PVC in incineration

Dioxin releases: 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 4), 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.[14] In the United States, an estimated 200,000 to 300,000 tons of PVC is inciner-

Table 4 Inventoried dioxin sources in North America, Europe, and the world
Percent of all releases from inventoried sources

Source type	United States [1]	European Union[2]	United States [3]	Great Lakes[4]	World[5]
* Municipal 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.

1. 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. Percent of all identified releases to air in the European Union.

3. Percent of identified emissions of total PCDD/F to the air in the United States as of 1989. Municipal waste incinerators include apartment incinerators. Accidental fires include structural fires, PCB fires, and PCP fires.

4. Percent of identified emissions of PCDD/F (TEQ) to the air that reach the Great Lakes.

5. 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.

Source: U.S. EPA 1998; Hanberg et al. 1999; Thomas and Spiro 1995; Cohen et al. 1995; Brzuzy and Hites 1996a.

Note: 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.

ated in trash burners every year.[15] Large quantities of PVC also go to medical waste incinerators, where PVC accounts for 5 to 18 percent of the waste stream—more than 90 percent of the organic chlorine, and more than 80 percent of the total chlorine content of medical waste.[16] As table 4 shows, combustion sources in which PVC is a major source of chlorine—and therefore of dioxin formation—make up the bulk of the world's major dioxin sources identified to date. In fact, sources in which PVC is the dominant chlorine donor account for 77 percent of all inventoried dioxin emissions in the U.S. and 50 percent in Europe.

Unidentified PICs: Without a doubt, burning vinyl is a source of dioxin. Numerous laboratory combustion tests involving pure PVC (or pure PVC in the presence of metal catalysts) produce considerable amounts of

dioxin.[17] 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, naphthalenes, PCBs, PCDFs, phenols, and styrenes—have been found in the combustion products when the closely related plastic polyvinylidene chloride (PVDC; commonly known as Saran Wrap) is incinerated.[18]

In municipal waste incinerators, PVC contributes at least 80 percent of the organically-bound chlorine in the waste stream.

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, ethylenes, methanes, PCBs, and others—but 58 percent of the total mass of PICs remained unidentified.[19] 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.

Lead and other additives: Incinerators also release additives contained in PVC products into the environment. An estimated 45,000 tons of lead stabilizers in PVC enter the world's municipal trash each year, based on Swedish figures.[20] Because lead cannot be destroyed by incineration, all 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 into the environment; PVC is responsible for about 20 percent of the lead in the waste stream, according to Swedish figures.[21] In the European Union, vinyl contributes from 1 to 28 percent of the lead and 10 percent of the cadmium entering municipal waste incinerators.[22]

Backyard burning: Not all vinyl burning takes place in high-tech incinerators. In developing countries and rural areas of industrial nations, open burning of waste is a common method to rid 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 benzenes, methanes, phenols, as well as 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.[23] Further, when more PVC was burned, average releases of

dioxin and all other chlorinated PICs rose substantially; 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. The rapidly expanding use of vinyl in developing nations, where expensive means of waste management are not available, has the potential to cause a major increase in worldwide emissions of dioxins.

Smelter releases: Some spent metal products that contain vinyl—including materials used in buildings such as cables and electronics equipment—are recycled or reprocessed 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. High dioxin emissions have been measured at these facilities, which are considered major dioxin sources in most inventories.[24] Most importantly, removing some of the vinyl sheathing before cables are fed to the smelters reduces dioxin emissions considerably.[25] Secondary steel smelters have also been found to emit large quantities of dioxin, in part because they recover metal from scrap automobiles that contain PVC.[26] Secondary lead smelters release dioxin and other organochlorines, too, due to the feed of lead automobile batteries with internal PVC parts. In the United States, however, PVC has been recently phased out of this application, so EPA no longer considers lead smelters an important dioxin source.[27]

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, strongly support 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. Conversely, because burning PVC is known to produce dioxin, 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 the result of burning organochlorines, burning salt, or some combination of the two. To suggest that organochlorines are not important dioxin precursors requires burning organochlorines to produce little or no dioxin and the combustion of inorganic chloride salts to be the predominant source of dioxin. Several lines of evidence indicate that organochlorines—particularly PVC—are the most important and most preventable cause of dioxin emissions from combustors.

Chlorine is a requirement for dioxin synthesis, and preventing the introduction of organochlorines into incinerators is the best means to prevent dioxin formation.

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, emissions, and input materials to be carefully controlled and accurately monitored. Studies of this type indicate that burning PVC is clearly an important dioxin source.

- The German EPA has found that burning PVC or other organochlorines produces dioxin—with concentrations in ash residues ranging from 3.2 to 662 ppt (TEQ). But combustion of several types of organochlorine-free but chloride-containing cotton, paper, wood, or wool does not produce dioxin above the detection limit of 0.1 ppt[28] (table 5).

- Two separate studies by Danish researchers have found that burning pure PVC produces dioxin. Under some conditions, the quantities formed are quite large.[29]

- A 2000 report by Japanese researchers found that adding 4 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. Adding an equal quantity of salt caused at most a two-fold increase.[30]

Table 5 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.

Numerous well-conducted studies indicate that organochlorines—particularly PVC—are the most important and most preventable cause of dioxin emissions from combustors.

- When newspaper or chlorine-free plastics are burned in a laboratory-scale incinerator, dioxin generation is extremely low. When PVC is added to the mix, dioxin levels in fly ash increase by a factor of 200 to 1,200—compared to a 13- to 45-fold increase when salt is added.[31]

- 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.[32]

- Adding PVC during combustion of natural chloride-containing wood products increases dioxin levels in the ash by 15 to 2,400 times. When large quantities of chemical hardeners containing inorganic chloride are added, dioxin levels rise somewhat, but are still 3 to 350 times lower than when PVC is included in the mix.[33] A Swiss study confirmed these results, finding that dioxin levels in fly ash from the burning of waste wood that has been glued, painted, or otherwise processed are up to 2,000 times higher than in ash from the burning of natural wood.[34]

- 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.[35]

- Burning chloride-containing vegetable matter does not produce detectable PCDD/Fs, but including PVC or chlorine gas along with the plant material does.[36]

- The same pattern exists for other organochlorines. Finnish researchers have found that burning perchloroethylene in a laboratory combustion reactor produces more dioxins, chlorobenzenes, and chlorophenols than burning sodium chloride.[37] An American study found that formation of dioxin precursors rises as the proportion of organochlorines in the waste increases,[38] while three others have found that adding salt to a combustion reaction has no detectable effect on dioxin formation.[39]

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 also some contradictory studies, possibly due to the difficulty of analyzing complex input and output streams and adjusting for fluctuating operating conditions:

- The Danish EPA 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.[40]

- A team of Japanese researchers has reported 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.[41]

- Two groups of researchers from Finland 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 rise substantially when PVC is added to the mix.[42]

- 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—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.[43]

- 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."[44]

- German scientists have found that removing PVC sheathings from copper cables before they are recycled in copper smelters causes dioxin emissions to drop precipitously.[45]

- 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.[46]

- According to a 2000 study by Japanese researchers, adding PVC to a mixture of chlorine-free matter in a pilot-scale incinerator increases dioxin emissions substantially; when more PVC is added, more dioxin is formed.[47]

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.

Two widely cited studies, one by the New York Department of Environmental Conservation[48] and the other by the European plastics industry,[49] 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 characteristics of the waste feed. This oversight radically weakens any study's ability 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.[50]

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.[51] 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.

Salt combustion and dioxin formation

Some studies indicate that burning large quantities of salt can produce dioxin. For instance, paper mills often burn logs that have been soaked in saltwater, and these incinerators have much higher concentrations of dioxin in their emissions than burning unsoaked wood.[52] The Swedish report discussed above found that it did not matter whether the chlorine came in organic or inorganic form—both gave rise to dioxin in approximately equal amounts.[53] One of the Japanese studies also found that burning large quantities of salt in a lab-scale incinerator could result in substantial dioxin emissions.[54] Another study found that burning PVC caused a much greater increase in dioxin formation than salt did, although the two substances caused similar increases in dioxin when municipal incinerator fly ash—which contains a wide range of organochlorines and other compounds—was included in the mix.[55]

These results conflict with the findings of the other studies discussed above, so the importance of salt in dioxin formation in incinerators remains an open question. From a policy perspective, however, it does not really matter how much dioxin salt combustion can produce. If burning chloride results in negligible dioxin emissions, then dioxin output depends almost entirely 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, burning salt can produce dioxin in amounts comparable to the burning of PVC and other organochlorines, then dioxin generation depends on the waste's total level of chlorine

From a policy perspective, it does not really matter how much dioxin salt combustion can produce. If we want to prevent dioxin formation in incinerators, we need to stop burning organochlorines.

(organic plus inorganic). Lowering the quantity of organochlorines in the waste will then reduce the total chlorine level and reduce dioxin formation. Either way, if we want to prevent dioxin formation in incinerators, we need to stop burning organochlorines.

The VI's ASME study is deeply flawed for several reasons—and does not provide an adequate basis to dismiss the many studies that do establish a link between dioxin generation and the combustion of PVC and other organochlorines.

Whatever the quantities, salts are ubiquitous in organic matter and cannot be removed easily from production, commerce, or the waste stream. In contrast, PVC use is highly preventable. Alternatives for most uses are currently available, ranging from traditional materials to chlorine-free polymers.[56] Any program to eliminate dioxin generation at the source should include provisions to reduce the use and combustion of PVC.

Vinyl Institute's flawed ASME dioxin study

To rebut evidence linking incineration of vinyl to dioxin formation, the Vinyl Institute (VI) frequently cites a single study, purportedly by the American Society of Mechanical Engineers (ASME), a professional society representing 125,000 mechanical engineers worldwide, "[which] found little or no correlation between chlorine input and dioxin emissions from incinerators."[57] This study is deeply flawed for several reasons—and it does not provide an adequate basis to dismiss the many studies that do establish a link between dioxin generation and the combustion of PVC and other organochlorines.

ASME study is biased

Several vinyl industry documents shed light on the purpose and origins of the ASME report. Just before U.S. EPA released its draft Dioxin Reassessment in 1994, the Vinyl Institute commissioned the public relations firm Nichols-Desenhall Communications to prepare a *Crisis Management Plan for the Dioxin Reassessment*. According to the plan, "EPA will likely conclude that the incineration of chlorinated compounds is the single largest known contributor of dioxin…We believe that PVC will be specifically mentioned and potentially slated for further regulation." In order to "prevent punitive regulation of PVC by EPA, Congress, or the state legislatures," the plan advised the Vinyl Institute how to present itself in the media and "strongly urge[d] VI to aggressively defend the industry's credibility through the use of third party sources to debunk…EPA's misleading claims."[58]

The industry took the advice of its public relations firm. In 1994, the Vinyl Institute's Incineration Task Force hired the consulting firm Rigo and Rigo, Inc. to prepare an "independent" analysis, which found that the amount of dioxin released by incinerators has no relation to the amount of chlorinated organic materials fed into them.[59] The Vinyl Institute arranged to have the report published as a product of the prestigious ASME, an independent professional organization. According to Vinyl Institute documents, one of the leaders of the Vinyl Institute's Incineration Task Force, Dick Magee, was also an active ASME member; Magee brokered an arrangement in which the Vinyl Institute would hire and fund Rigo to write a report that would be released under the ASME banner. According to an internal Vinyl Institute memo from 1994, the purpose of ASME's involvement was to create the illusion of "third-party" authority, and that the Rigo report was conceived, carried out, and rewarded in a spirit of public relations, not unbiased analysis. The memo reads:

> The Vinyl Institute has created an Incineration Task Force in anticipation of adverse EPA actions regarding dioxins and furans…Dick Magee brought forward a proposal from the American Society of Mechanical Engineers to hire Rigo & Rigo Associates, Inc., of Cleveland, OH. The purpose of ASME as the contractor is to provide unassailable objectivity to the study…
>
> The Incineration Task Group interviewed Dr. H. Gregory (Greg) Rigo, principal of Rigo & Rigo Associates, Inc. by phone and found him to be extremely knowledgeable about incineration and to have several proprietary databases VI had not discovered. He is also user friendly, i.e., willing to set his priorities to our needs, and appears to be sympathetic to Plastics, Vinyl, PVC, and Cl^2…
>
> The ASME proposal calls for $130,000 for the Rigo & Rigo/ASME study. Since there are many unanswered questions regarding dioxins and since VI may want to use Greg Rigo as an expert witness or advocate to talk about the report, I am proposing an additional $20,000 as a contingency fund, for a total of $150,000 to be fully funded by VI.[60]

Methodological flaws

The methodology of the Rigo report is no less flawed than its origins, undermining the reliability of its claim that burning organochlorines is not related to dioxin formation. The study was not experimental, so it could not directly refute the existence of a chlorine-dioxin link. Instead of generating new data, the authors compiled existing trial burn measurements from a large number of incinerators, statistically analyzed the relationship between indicators of chlorine feed and dioxin releases, and concluded that there was no statistically significant relationship between the two. A statistical analysis of this type is particularly sensitive to design problems: if the putative cause and effect are not measured accurately, or if confounding factors are not taken into account, then a meaningful relationship is likely to go undetected. In fact, the study's data and methods on dioxin output and chlorine input were flawed in several ways, suggesting that it's failure to detect a causal link is more likely an artifact of bad study design than a meaningful finding that no relationship between chlorine and dioxin actually exists:

The study's data and methods on dioxin output and chlorine input were flawed in several ways, suggesting that it's failure to detect a causal link is more likely an artifact of bad study design than a meaningful finding that no relationship between chlorine and dioxin actually exists.

1. The Rigo study failed to take account of differences among facilities. Chlorine input is not the only factor that determines the magnitude of dioxin emissions from incinerators; combustion conditions, the types and quantities of substances in the waste, and other variables also affect the amount of dioxin that will be released. Because of this complexity and constant fluctuations of many factors, statistical relationships between stack emissions and indicators of waste input or combustion conditions can seldom be established, even at individual incinerators.[61] Massively compounding this problem, Rigo used data from a large number of incinerators operating under widely variable conditions, but did not control or adjust for differences in facility type, operating parameters, waste type, or other factors. A statistical summary of data from many different facilities, with no attempt to control or adjust for confounding factors, cannot be expected to detect a signal within such extensive noise. Even a strong relationship between organochlorine input and dioxin output is likely to go undetected in a study designed in this manner.

2. The Rigo study also used data from unreliable sources. The emissions data in Rigo's analysis came almost exclusively from trial burns

designed for permitting purposes, without the proper kinds of controls and measurements necessary to evaluate the relationship between chlorine input and output. Moreover, trial burn data are notoriously problematic. First, with their highly simplified designs, these trials do not accurately represent the way incinerators operate in the real world, when waste composition and operating conditions constantly fluctuate. Further, they do not measure the much larger quantity of chemicals that are released after the feed of waste to the incinerator has stopped.[62] In fact, many trial burns have conducted their evaluations of low- or no-chlorine wastes after chlorinated wastes have been burned, so the later stack samples become contaminated by continuing emissions from earlier runs. The use of results from trial burns of this sort thoroughly scrambles any relationship that might otherwise have been recognizable between chlorine input and dioxin input.

3. Rigo's study relied on the wrong kinds of measurements. To investigate a link between the amounts of organochlorines burned and the amount of dioxin produced by incinerators, Rigo should have examined the statistical relationship between the mass of organochlorines fed to an incinerator and the mass of dioxins released. Instead, the study tracked "surrogate" measures for both of these parameters, measuring the concentration of hydrogen chloride (HCl; the primary by-product of organochlorine combustion) in the stack gas as a rough indicator for the mass of organochlorines in the feed; as a surrogate for the mass of dioxin released, it examined the concentrations of dioxin in the stack gas.[63] The problem is that concentrations do not accurately represent quantities, for several reasons:

- If the total flow of stack gas increases (as it generally will if more waste, and thus more chlorine, is fed to the incinerator), the concentrations of dioxin in the gas may decrease, even if a larger amount of dioxin is being emitted.

- Stack gas measurements omit pollutants directed into fly ash, bottom ash, and scrubber water, so changes in the efficiency of pollution control devices can reduce the concentration of dioxin in stack emissions while total dioxin formation increases. Control devices can also reduce the concentration of HCl while total organochlorine input

rises. (Because pollution control devices have different capture efficiencies for dioxin and hydrochloric acid, the concentrations of these materials in the stack gas after it passes through this equipment will not reflect the ratios of the amounts that were actually produced by the incinerator).

- Hydrogen chloride is formed not only by the combustion of organochlorines but also by the burning of chloride salts. It is therefore not a reliable indicator of the amount of organochlorines fed to an incinerator.

The variables that Rigo analyzed are thus grossly inappropriate substitutes for the quantities that are truly of interest; Rigo's failure to find a relationship between the surrogates he used says nothing about whether a link actually exists between organochlorine input and dioxin generation.

All the flaws discussed above cripple the ASME study's ability to establish a link between chlorine and dioxin. A finding of "no relationship" is only as good as a study's power to detect a relationship, and in this case that power can only be described as pathetically weak. On the basis of Rigo's analysis, no reliable inferences can be drawn about whether a relationship exists between the amount of organochlorines burned and the amount of dioxin formed in an incinerator. With more than twenty well-designed studies coming to the opposite conclusion—that burning PVC and other organochlorines produces dioxins, and burning less reduces dioxins—Rigo's findings are far from persuasive. The weight of evidence from laboratory, pilot, and full-scale tests clearly establishes that vinyl is an important source of dioxin in incinerators, fires, and combustion-based recycling facilities, which together are responsible for the majority of identified dioxin releases in the world.

PVC and dioxin relationship—historical evidence

The theory that burning organochlorines like PVC is an insignificant dioxin source and that salt is responsible for incinerator emissions of dioxin implies 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

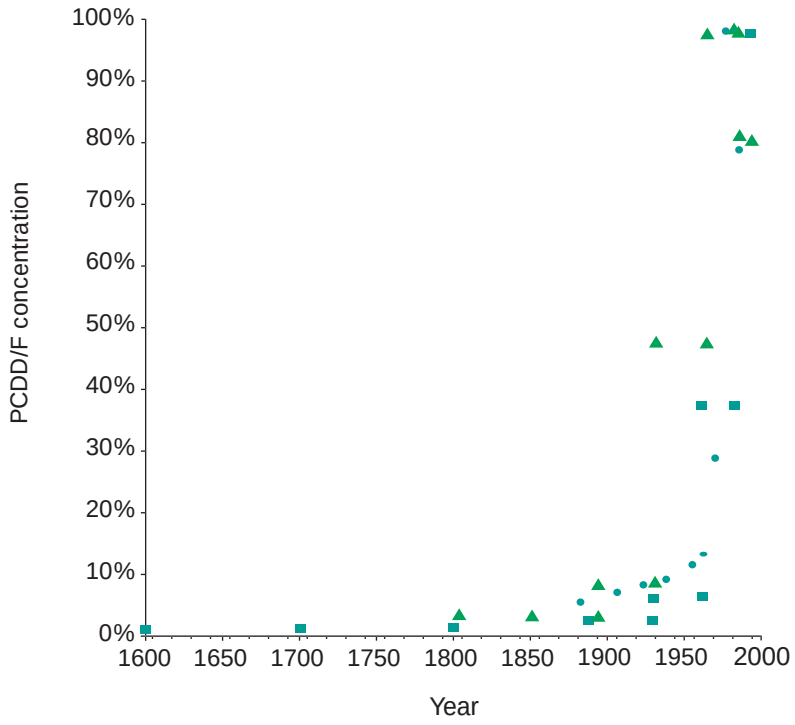
plant matter is rich in salt. According to research by chemists at Indiana University, however, dioxin levels in the sediment of a U.S. lake, the watershed of which suffered a major forest fire in 1937, show no change whatsoever around or after the time of the fire.[64] More recently, scientists in Spain 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 levels, leading the researchers to conclude, "Natural fires seem not to be an important source of dioxin-like compounds."[65]

The second prediction implied by industry's salt 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 reverse. Several studies have analyzed the dioxin and furan content of mummified and frozen remains of people who lived several hundred to several thousand years ago, including individuals from cultures that cooked over indoor fires and were exposed to considerable amounts of combustion emissions. These studies have found that dioxin levels (measured as TCDD-equivalents) in ancient tissues were no more than 1 to 2 percent of the amount found in modern humans, and even this amount could represent contaminants deposited in the samples in modern times, especially during handling and analysis.[66]

The theory that burning organochlorines like PVC is an insignificant dioxin source and that salt is responsible for incinerator emissions of dioxin implies several specific predictions, none of which turn out to be true.

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 20th century when chlorine and organochlorine production began, despite the fact that natural and industrial combustion processes were abundant in this period. Sediments in Swedish lakes show no measurable dioxin before 1945,[67] and those in the Great Lakes show none before 1920.[68] 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.[69] A study of two lakes in Germany's Black Forest found that sediments from the seventeenth and eighteenth centuries contained small quantities of dioxins and furans—77 and 34 times lower than the maximum concentrations from this century. Expressed as

Figure 3 Dioxin deposition in European sediments



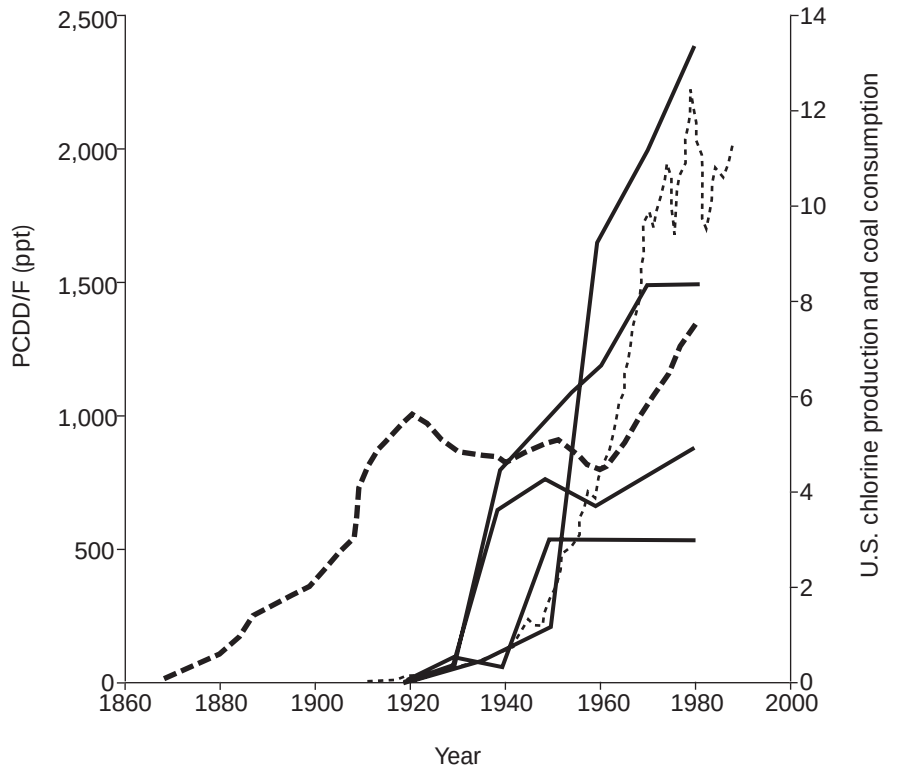
Note: The vertical axis shows concentrations of total dioxins and furans (ppt) in sediment cores from the Baltic (circles) and two German lakes—the Wildsee (triangles) and the Herrenweiser See (squares), expressed as a percentage of the highest levels measured in each location. In all locations, levels were extremely low prior to the advent of chlorine chemistry, and they rise rapidly thereafter.

Source: Juttner et al 1997; Kjeller and Rappe 1995.

TCDD-equivalents, the ratios were even higher: 310 and 90 times greater in modern than in pre-chlorine sediments.[70] In New York's Green Lake, small quantities of dioxins and furans are present in layers from the late 1800s, but at concentrations 1,500 times lower than those found in the 1960s.[71]

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, then shot up rapidly from the 1940s to the 1970s—rising 25-fold or more between 1935 and 1970, then declining somewhat thereafter.[72] This pattern is consistent with the rise of chlorine chemistry, which peaked in the 1960s

Figure 4 Dioxin deposition in European sediments



Source: Juttner et al 1997; Kjeller and Rappe 1995.

Every study conducted to date shows that dioxin levels were extremely low before the 20th century when chlorine and organochlorine production began.

or 1970s, followed by restrictions on dioxin-contaminated pesticides and chlorinated gasoline additives went into effect.

Although these trends follow the production of chlorine, they do not even approximately track the history of combustion of salt, either industrial or natural. One study of dioxin trends in Great Lakes sediments found that dioxin levels do not follow trends in combustion of coal—practiced on a massive scale long before dioxin concentrations began to rise—but they do correspond closely to the rise of the chlorine chemical industry (figure 4). These results suggest that industrial combustion processes—including coal-fired power plants, furnaces for heating, rail engines, steel mills, and other industries powered by coal (which contains

chloride salts)—have never been major sources of dioxin. 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 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.[73]

Dioxin concentrations began to increase slowly in the early decades of this century, then shot up rapidly from the 1940s to the 1970s consistent with the rise of chlorine chemistry.

If organochlorines have nothing to do with dioxin emissions, then why were dioxin levels in the environment non-existent or minuscule before the chemical industry began to produce them? In particular, why were dioxin levels so low during the 19th century when combustion of chloride-containing materials such as coal and wood was at its peak? These data make abundantly clear that the majority of dioxin in the environment is due to the production, use, and disposal of chlorine gas and organochlorines.

In conclusion, while it is likely that some dioxin can be formed by the combustion of chloride-containing salts, the available evidence indicates that industrially produced materials containing organochlorines—PVC in particular—are the dominant cause of dioxin generation in incinerators. More importantly, these materials 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."[74]

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—should include provisions to reduce the use of PVC in applications susceptible to accidental fire or disposal by combustion.

Notes

1. TNO Centre for Technology and Policy Studies 1996.
2. Franklin Associates 1997.
3. European Commission 2000.
4. DTI 1995.
5. European Commission 2000.
6. European Commission 2000.
7. KemI 2000.
8. European Commission 2000.
9. APR 1998.
10. European Commission 2000.
11. European Commission 2000.
12. Hamilton-Wentworth 1997; Socha et al. 1997. Socha et al. notes that dioxin levels in tree leaves downwind from the fire were 7 to 100 times above normal. Apparently, pollutants on the leaves were washed from the leaves into the general environment by rain, because levels on leaves declined significantly after the first post-fire rainstorm.
13. EPA 1998.
14. Danish EPA 1993; Ecocycle Commission of the Government of Sweden 1994; DTI 1995; TNO Institute of Environmental and Energy Technology 1994.
15. This assumes U.S. municipal waste incinerator capacity of 48 million tons per year (Versar 1996), 80 percent capacity utilization, and PVC content of 0.5 to 0.8 percent.
16. According to two studies, 9.4 percent (Marrack 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). See also Green 1993.
17. Christmann et al. 1989; Theisen et al. 1989; Theisen 1991.
18. Yasuhara and Morita 1988. See also Blankenship et al. 1994.
19. Jay and Stieglitz 1995.
20. I have extrapolated from the relevant figures for Sweden, where 249 tons of PVC enter the waste stream each year (TNO Centre for Technology and Policy Studies 1996), assuming 19.1 million tons of PVC production worldwide, each year (DTI 1995).
21. TNO Centre for Technology and Policy Studies 1996.
22. European Commission 2000.
23. 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 certainly 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.
24. Christmann 1989; EPA 1994a; Versar, Inc. 1996.
25. Christmann et al. 1989.
26. Lahl 1994; Schaum et al. 1993; EPA 1994b; Aittola et al. 1993.
27. Versar, Inc. 1996.
28. Theisen 1991.
29. Christman 1989; Vikelsoe and Johansen 2000.
30. Ishibashi et al. 2000.
31. Takasuga et al. 2000. When fly ash from a municipal incinerator was added to the mix, baseline concentrations of dioxin were higher, and, as discussed below, addition of PVC or salt yielded similar increases in dioxin levels.
32. Kopponen et al. 1992.

33. Kolenda et al. 1994; Wilken 1994.
34. Wunderli et al. 2000.
35. Mahle and Whiting 1980.
36. Liberti et al. 1983.
37. Halonen et al. 1995.
38. 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.
39. 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.
40. Danish Environmental Protection Agency 1993.
41. Tamade et al. 2000; Yoneda et al. 2000.
42. Mattila et al. 1992; Ruuskanen et al. 1994; Frankenhauser et al. 1993.
43. 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.
44. Wagner and Green 1993; this study also measured emissions of chlorophenols as a dioxin surrogate.
45. Christmann et al. 1989.
46. Vesterinen and Flyktmann 1996; Halonen et al. 1993; Hutoari and Vesterin 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.
47. Hatanaka et al. 2000.
48. Visalli 1987.
49. Mark 1994.
50. EPA 1988.
51. Wikstrom et al. 1996.
52. Pandompatam et al. 1997.
53. Wikstrom et al 1996.
54. 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.
55. Takasuga et al. 2000.
56. Thornton 2000.
57. Burns 2000.
58. Burnett 1994.
59. Rigo et al. 1995.
60. Goodman 1994.
61. Illustrating this point, even carbon dioxide, a widely accepted indicator of combustion conditions, is not consistently related to the emission of unburned wastes, according to Staley et al. 1986 and EPA 1990.
62. Licis and Mason 1989.
63. Rigo analyzed Hydrogen chloride in stack gas for municipal and medical waste incinerators. For hazardous waste incinerators, his analysis was based on the percent chlorine in the waste feed, a parameter that also does not reflect the mass of chlorine. If the percent of chlorine stays the same and total waste feed is increased, then the mass of chlorine feed will increase but would not have been noted using Rigo's approach. Further, increasing the waste feed typically increases the stack gas flow rate, which will tend to reduce dioxin concentrations even if the mass of dioxin emitted increases.
64. Brzuzy and Hites 1996b.
65. Martinez et al. 2000.
66. Schecter 1991; Ligon et al. 1989; Schecter et al. 1988; Tong et al. 1990.
67. Reviewed in Alcock and Jones 1996.
68. Czuczwa and Hites 1986; Czuczwa et al. 1984; Czuczwa and Hites 1985.
69. Kjeller and Rappe 1995.
70. Juutner et al. 1997.
71. 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).
72. Brzuzy and Hites 1996b.
73. Czuczwa and Hites 1984. Additional data are reported in Czuczwa and Hites 1986 and 1985.
74. DTI 1995.

Background
Persistent Organic Pollutants (POPs)

five

Background on Persistent Organic Pollutants (POPs)

In recent years, there has been extensive scientific and political activity on toxic pollutants, their global distribution, and their effects on highly exposed populations and the general public. An understanding of that context strengthens the case for action to restrict PVC in building applications, and action on PVC would in turn strengthen international efforts to reduce persistent toxic pollution at the source.

GLOBAL DISTRIBUTION OF POPs

Recently the first global agreement to eliminate sources of persistent organic pollutants (POPs) has been negotiated. The POPs treaty is, in large part, a response to scientific research in the past decade that has identified a variety of synthetic chemical pollutants that are now globally distributed in the environment and food web, have damaged wildlife populations, and may have caused large-scale human health damage.[1] Global contamination has occurred because many synthetic chemicals are persistent in the environment, resisting natural degradation processes for months, years, or even decades. As a result, even substances that are discharged at a relatively slow rate build up to higher levels over time and are distributed widely by air and water. Because many synthetic organic substances are derived from petrochemicals, they 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 tens of millions of times greater than their levels in the ambient environment in species high on the food web, including humans.[2]

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 POPs in areas remote from any known sources of these substances, including the high Arctic,[3] the isolated rainforests of South America and Africa,[4] and remote regions of the deep oceans.[5] 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.[6]

Although research and policy have focused primarily on a handful of substances—dioxins, PCBs, 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 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.[7] By-products of chlorinated chemical manufacture and disposal are present in measurable quantities in the Canadian Arctic[8] and over the remote Atlantic Ocean,[9] and a variety of chlorinated benzenes are components of rain and snow.[10] Chlorinated solvents, refrigerants, and their environmental degradation products have become truly ubiquitous contaminants of the atmosphere and vegetation.[11]

With the environment and food web ubiquitously contaminated, it should come as no surprise that people are contaminated as well. Human exposures come through inhalation, drinking water, and eating 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.[12] At least 700 xenobiotic organic chemicals are present in the adipose tissues of the general population of the United States.[13] Close to 200 organochlorine by-products, metabolites, pesticides, plastic feedstocks, solvents, and specialty chemicals have been specifically identified in the blood, breath, fat, milk, semen, or urine of the general U.S. and Canadian populations—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 in breast milk.

"Acceptable" substances that persist or bioaccumulate cannot be integrated into natural cycles. The ecosystem's assimilative capacity for persistent or bioaccumulative substances is therefore zero, and the only "acceptable" discharge is also zero.

The now-ubiquitous global presence of synthetic chemicals—in large-scale production for just over than half a century—supports a simple inference: substances that persist or bioaccumulate cannot be integrated into natural cycles. Discharged in even small amounts, these chemicals build up gradually in the environment and in living things. Given enough time, even small "acceptable" discharges 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 or bioaccumulative substances are far superior to those that attempt to control, manage, or dispose of persistent chemicals after they have been produced.

ENDOCRINE DISRUPTION

What are the impacts of universal exposure to POPs on the health of people and wildlife? Important discoveries have emerged in the past 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 cancer, organ damage, paralysis and tremors, structural birth defects, and death. Recently, however, it has been discovered that many synthetic chemicals can, at minute doses, result in subtle but significant deficits in an organism's functional capacities, such as fertility, immunity, cognition and intelligence.

Many of these effects occur as the consequence of a newly recognized set of toxicological mechanisms—disruption of the body's endocrine system.[14] 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, circulating in the blood in low concentrations (typically in the ppt range) and triggering cascades of gene expression that control essential aspects of development, behavior, immunity, reproduction, and the maintenance of homeostasis.

In the past 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 mediating 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, retinoid, and thyroid 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 developmental, immunological, neurological, and reproductive effects have occurred in human populations, wildlife, and laboratory animals as a consequence of exposure to hormonally active compounds found in the environment.[15] According to the Academy, effects observed include functional and structural 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.

The U.S. National Academy of Sciences concluded that adverse developmental, immunological, neurological, and reproductive effects have occurred in human populations, wildlife, and laboratory animals as a consequence of exposure to hormonally active compounds found in the environment.

DIOXIN AND RELATED COMPOUNDS

Occurrence and exposure

The most intense scientific activity has focused on 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD, also known colloquially as dioxin) and other structurally similar compounds that act through a similar toxicological mechanism (called, collectively, dioxin-like compounds). Research on dioxins is particularly relevant to the PVC debate because of the evidence that the PVC lifecycle is a major source of dioxins.

A typical nursing infant in the United States receives a daily dioxin dose 92 times greater than that of the average adult.

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 more than 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.[16]

Health impacts of dioxin

In 2000, U.S. EPA released its Dioxin Reassessment,[17] 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 findings of both the U.S. National Toxicology Program and the International Agency for Research on Cancer.[18] *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 exposures to small quantities of dioxin are associated with increased cancer of hormone-responsive organs (such as mammary glands) 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.[19] People who eat greater than average quantities of meat or fish are subject to even higher cancer risks. (EPA's estimates are based on numerous assumptions and may or may not accurately reflect actual risks.)

- Dioxin's non-cancer effects may be of even greater concern than its carcinogenicity. *Dioxin is a potent endocrine-disrupting substance*, interacting with an intracellular receptor and disrupting the action of gonadotropins, retinoic acid, steroid hormones, thyroid hormone, 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 ability and IQ, reduced sperm density, smaller 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 at or near the level at which dioxin has been found to cause a variety of effects in laboratory animals and human populations*, including cognitive defects, endometriosis, hormonal changes, immune suppression, reduced sperm count, and impaired development of the male and female reproductive systems.[20]

- *There is no evidence of a 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 low levels of dioxin is that the severity of the impact is roughly proportional to the magnitude of dioxin exposure.[21]

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 behavior, function, and sexual development of their male offspring are compromised.[22] 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.[23] 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.[24] If there is a threshold for dioxin, it is so low that it is irrelevant for the purposes of environmental policy and health protection.

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.

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 that bioaccumulated dioxin-like compounds have caused large-scale epidemics of developmental impairment, endocrine disruption, immune suppression, and reduced fertility in mammals, fish, and birds in the Baltic Sea, the Great Lakes, and the Wadden Sea.[25]

Together, these findings indicate that we cannot 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, endometriosis, immune suppression, impaired cognitive development, and infertility. From a public health perspective, universal dioxin exposure is already too high by a considerable margin. Further releases of dioxins into the environment should be eliminated wherever technically feasible.

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 show that dioxin lev-

els were very low before the 20th 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.[26] Then, during the 1970s, many governments restricted the use of leaded gasoline (which contains chlorinated additives and thus produces dioxin when burned) 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 chemical plants, incinerators, and steel mills) 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[27]—declined by nearly 80 percent between the late 1970s and the early 1990s. As one might expect, dioxin levels in the milk of cows, which eat foliage, subsequently declined, falling by about 25 percent between 1990 to 1994.[28] 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.[29]

If there is a threshold for dioxin, it is so low that it is irrelevant for the purposes of environmental policy and health protection.

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 settles 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 indicators 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 UK 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."[30]

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.[31] 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."[32] No human tissue analyses are available from the early decades of this century, but dioxin levels in people from the United States increased steadily during the 1960s. Following the regulatory actions of the 1970s, levels declined during the 1980s.[33] Concentrations continued to fall in most European nations during the 1990s, although the data for the United States are ambiguous.[34]

These pollutants are so persistent that, so long as releases continue somewhere, the global environmental burden of these compounds declines slowly, if at all.

There are no reliable projections of future trends in dioxin levels. Apparently, the successful actions of the past have had their effect, however. According to Swedish scientists, the declines are history, not a continuing trend. "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."[35] In Germany, dioxin levels in human milk have stopped falling and increased slightly in the late 1990s.[36]

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 should reduce the use of dioxin-generating materials rapidly. Because infinitesimal 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.

Because infinitesimal 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.

PHTHALATES

Usage

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.[37] Flexible PVC—including flooring and wall coverings—accounts for just more than half of all vinyl demand, while the remainder is rigid, unplasticized materials like pipes and siding.[38] PVC accounts for the vast majority of all phthalate consumption, and phthalates are the dominant class of plasticizers used in soft vinyl products.[39] An estimated 50 percent of all phthalates produced are used in building and interior materials.[40] About 5.4 million tons of phthalates are used in vinyl products worldwide each year.[41] 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 near 2 million tons per year), diisononyl phthalate (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.[42] The other commercially important phthalates dibutyl phthalate (DBP) and dioctyl phthalate (DOP) are not used appreciably in PVC.

Because phthalates are fat-soluble, they cross the placenta easily and concentrate in breast milk.

Fate, occurrence, and exposure

Phthalates are moderately persistent in the environment. They can be degraded biologically or chemically in the presence of air in days or weeks; in anaerobic conditions, like those often found in groundwater, little if any degradation occurs, with a hydrolysis half-life of 2000 years.[43] Because phthalates are soluble in fat, they quickly adsorb into sediments or enter the food chain, where they can bioaccumulate.[44] Although some phthalates are partially metabolized in humans, DEHP tends to bioaccumulate in aquatic invertebrates and fish. Bioconcentration factors measured in fish range from 42 to 2680, indicating that fish swimming in water contaminated with DEHP accumulate in their tissues levels of DEHP that are up to thousands of times greater than the concentration in the water.[45]

Because of this behavior and the large quantities produced, phthalates have become ubiquitous environmental contaminants, present in air, water, fish, and human tissues on a global basis.[46] Because most phthalates are 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;[47] levels of DEHP and metabolites in animal and human tissues can be quite high, reaching concentrations higher than such infamous pollutants as PCBs and DDT.[48]

For the general population, the greatest exposures to phthalates come through the food supply, with the highest levels in fatty foods like dairy, fish, meat, and oils, although indoor air contributes substantially as well.[49] 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.[50] 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 ppb, respectively), reflecting “considerable exposure” to these compounds, as well as other phthalates.[51] Because phthalates are fat-soluble, they cross the placenta easily and concentrate in breast milk.[52] The authors of the CDC study concluded, “Some individual exposures

are substantially higher than previously estimated for the general population," and these "data indicate a substantial internal human dose of DBP, DEP, and BBP, [the metabolites of which] are of particular concern because of their developmental and reproductive toxicity in animals."[53]

Health impacts

Phthalates are well-recognized developmental and reproductive toxicants. DEHP is the most 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, developmental delay, and intrauterine death. DEHP also reduces estrogen levels, fertility, and ovarian weight, 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—developmental effects occur at doses up to 100 times lower than those that produce reproductive toxicity in the adult.[54] Exposure of a pregnant mother rat to DBP or DEHP 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.[55] Extremely low levels of MEHP (100 nanomolar, or less than 30 ppb)—approximately the same level as found in the urine of the general U.S. population[56]—cause significant damage to cultured sperm-producing cells of developing rat testes.[57]

Phthalate exposure in utero or during childhood is particularly problematic—developmental effects occur at doses up to 100 times lower than those that produce reproductive toxicity in the adult.

The general human population is exposed to levels of DEHP that justify public health concern. EPA's reference dose (RfD) for DEHP is 20 micrograms per kilogram of body-weight per day.[58] An RfD is the "acceptable" exposure level, predicted by EPA based on toxicological studies, at which significant risk of health effects will not occur. But the average daily dose in the United States for children ages 6 months to 4 is 19 micrograms per kilogram of body weight, approximately the same as EPA's RfD.[59] And researchers at the Centers for Disease Control have estimated that the daily DEHP exposure of adults in the general U.S. population, based on the 95th percentile and maximal levels found in the general population's urine, is as high as 3.6 and 46 micrograms per kilogram per day—in the same range as EPA's RfD, and considerably higher for some individuals.[60]

The general public's DEHP exposure allows little or no clear margin of safety.

These results make clear that the general public's DEHP exposure allows little or no clear margin of safety. Furthermore, EPA's RfD for DEHP was established in 1986 based on a 1953 study that examined changes in liver weight in rodents exposed to DEHP, supplemented by other studies from 1982 and 1984. This RfD was calculated before any of the relevant studies of the effects of chronic low-level exposure to DEHP and metabolites on reproduction and development were published. Since these types of impacts may occur at substantially lower doses than liver damage, even the RfD may not represent a truly safe dose.[61] It is therefore prudent from a public health perspective to reduce exposures to DEHP as rapidly as possible.

Considerably less research has been conducted on the toxicity of other phthalates, but some data are available. Like DEHP, BBP is 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.[62] Monobutyl phthalate, a metabolite of BBP, causes cryptorchidism (failure of the testes to descend) when exposure occurs in utero.[63] 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 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 a "concern that exposure may adversely affect male reproductive tract development."[64] The panel also examined exposure that occurs across the placenta and via breast milk and concluded, "The panel has concern that ambient oral DEHP exposures to pregnant or lactating women may adversely affect the development of their offspring." Based on the available data, the panel expressed low, minimal, or negligible concern about other phthalates.

There are few epidemiological data concerning the impacts of phthalates on human 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 from 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,[65] and a similar trend has been documented in the United States.[66] Exposure to endocrine-disrupting compounds is one 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 more than 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.[67]

Some phthalates may be carcinogens. According to the National Toxicology Program (NTP), DEHP is "reasonably anticipated to be a human carcinogen" based on consistent findings of liver cancer in laboratory animals. There has been some debate about whether the mechanism of carcinogenicity in rodents is relevant to humans. Based on this issue, the International Agency for Research on Cancer now lists DEHP as an animal carcinogen but as unclassifiable with regard to human carcinogenicity. But the NTP has not adopted this view, and scientists at the National Institute for Environmental Health Scientists have criticized the IARC's action as lacking a sound scientific basis in current understanding of DEHP's mechanism of toxicity.[68]

TRENDS IN PVC MARKETS

Polyvinyl chloride plastic was first marketed in 1936[69] 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 estimate includes the non-PVC components of vinyl products, such as plasticizers and stabilizers; production of pure polyvinyl chloride is now estimated at approximately 24 million metric tons per year worldwide.[70] Vinyl is the largest use of chlorine in the world, accounting for more than 40 percent of all chlorine use in the United States, with a similar or slightly greater proportion globally.[71]

Few materials have infiltrated modern life as ubiquitously as PVC, and construction represents the largest sector of vinyl applications.

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

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.[72] 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 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 safely stored; the chemical industry therefore can only produce as much alkali as there are markets for chlorine. In recent years, as numerous uses of chlorine (for example, pulp and paper, refrigerants, and solvents) 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 potential sales in alkali markets.[73] 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."[74]

The industry's strategy to rectify the chlor-alkali imbalance is the aggressive expansion of markets for PVC and the feedstocks from which it is made—already the major global sinks for chlorine. For the past several decades, PVC production and consumption has grown at a remarkable pace, but recently PVC markets in industrialized nations have neared saturation. Vinyl has already replaced so many traditional materials, that growth in vinyl in these countries is now no greater than annual increases in gross national product.[75] This rate of growth is not nearly enough to offset the loss of chlorine demand in sectors that have been restricted so the industry has focused on expanding exports of PVC and its feedstocks to developing nations.[76] U.S. net exports of EDC, PVC, and VCM now contain about two million tons of chlorine per year—more than 15 percent of total chlorine production—and were expected to grow by a stunning 14 percent in 1998 alone.[77] 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.[78] 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 about the disposal of waste material is one of the reasons for advanced society to refrain from excessive use of plastics.[79]

The rapid increase in vinyl consumption in developing countries means that, despite slow growth in PVC consumption in the wealthy nations,

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.

global demand for PVC will rise from 22 million tons per year in 1996 to 28 million tons per year 2000—an annual growth rate of more than 6 percent.[80] 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.[81]

Notes

1. Thornton 2000; Colborn et al. 1996.
2. Tatsukawa and Tanabe 1990; Allan et al. 1991.
3. Gregor and Gummer 1989; Patton et al. 1991; Barrie et al. 1997.
4. Simonich and Hites 1995.
5. Ono et al. 1987.
6. Dewailly et al. 1993; Norstrom et al. 1990.
7. Great Lakes Water Quality Board 1987.
8. Barrie et al. 1997.
9. Fuhrer and Ballschmitter 1998.
10. Brun et al. 1991.
11. DeLorey et al. 1998; Makhijani and Gurney 1995; Plumacher and Schroder 1993.
12. EPA 2000a.
13. Onstot et al. 1987.
14. National Research Council 2000; Guillette and Crain 1999.
15. National Research Council 2000.
16. EPA 2000a.
17. EPA 2000a.
18. McGregor et al. 1998; National Toxicology Program 2001.
19. 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.
20. See discussion in EPA 2000a, as well as DeVito et al. 1995 and Tryphonas 1995.
21. See also Tritscher et al. 1994; Kohn et al. 1996; Portier et al. 1996.
22. Peterson et al. 1992.
23. Neubert and colleagues (1991) documented this effect in primate lymphocytes at TCDD concentrations as low as 10-14 moles per liter.
24. Kerkvliet (1994) reports that TCDD concentrations in the spleen as low as 2×10^{-15} moles per liter caused immunotoxicity in laboratory rats.
25. See for example Olsson et al. 1994. Reijnders 1986; de Swart et al. 1996; Giesy et al. 1994a and 1994b.
26. Alcock and Jones (1996) provide an excellent review of dioxin trends. Specific papers include Czuczwa and Hites 1984, 1985 and 1986, Czucwa et al. 1984, Juttner et al. 1997, Kjeller and Rappe 1995, Kjeller et al. 1991, and Kjeller et al. 1996.
27. Kjeller et al. 1996.
28. Reviewed in Alcock and Jones 1996.
29. 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).
30. Alcock and Jones 1996. Kjeller et al. 1991 also provide a useful discussion.
31. 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.

32. Huestis et al. 1997.
33. Stanley et al. 1990.
34. LaKind et al. 2001.
35. Johansson 1993.
36. Furst 2000.
37. DTI 1995.
38. These figures are for Western Europe (European Commission 2000).
39. DTI 1995.
40. Oie et al. 1997.
41. My calculations are extrapolated from the figures for Sweden, where the phthalate input into PVC equals 22.6 percent (TNO Centre for Technology and Policy Studies 1996), assuming 24 million tons of vinyl produced per year worldwide.
42. National Toxicology Program 2000.
43. HSDB 1997.
44. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a, 2000b, and 2000c.
45. HSDB 1997.
46. Giam et al. 1978;, Blount et al. 2000; HSDB 1997.
47. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
48. Giam et al. 1978; National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
49. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
50. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
51. Blount et al. 2000.
52. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
53. Blount et al. 2000.
54. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
55. Gray et al. 1999; Lambright et al. 2000.
56. Blount et al. 2000.
57. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
58. EPA 2000b.
59. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
60. Kohn et al. 2000. The median exposure level was estimated to be 0.71 micrograms per kilogram per day.
61. EPA 2000b.
62. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000b.
63. Imajima et al. 1997.
64. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction 2000a.
65. Colon et al. 2000.
66. Hermann-Giddens et al. 1997.
67. Colon et al. 2000.
68. Melnick 2001.
69. Taylor 1957; Aftalion 1991.
70. 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.
71. Leder et al. 1994.
72. 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).
73. Leder et al. 1994.
74. Tullos 1995.
75. Endo 1990.
76. Leder et al. 1994.
77. Mears 1995.
78. Waltermire 1996.
79. Endo 1990.
80. Svalander 1996.
81. Tittle 1995.

Conclusion

six

Conclusion

The PVC lifecycle presents one opportunity after another for the formation and environmental discharge of organochlorines and other hazardous substances. When its entire lifecycle is considered, it becomes apparent that this seemingly innocuous plastic is one of the most environmentally hazardous consumer materials produced, creating large quantities of persistent, toxic organochlorines and releasing them into the indoor and outdoor environments. PVC has contributed a significant portion of the world's burden of persistent organic pollutants and endocrine-disrupting chemicals—including dioxins and phthalates—that are now present universally in the environment and the bodies of the human population. Beyond doubt, vinyl has caused considerable occupational disease and contamination of local environments as well.

In summary, the feedstocks, additives, and by-products of the PVC lifecycle are already present in global, local, and workplace environments at unacceptably high levels. Efforts to reduce the production and release of these substances should be environmental and public health priorities. The hazards posed by dioxins, phthalates, metals, vinyl chloride, and ethylene dichloride are largely unique to PVC, which is the only major building material and the only major plastic that contains chlorine or requires plasticizers or stabilizers. PVC building materials therefore represent a significant and unnecessary environmental health risk, and their phase-out in favor of safer alternatives should be a high priority.

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.

References

Addink, R., F. Espourteille, and E.R. Altwicker. 1998. Role of inorganic chlorine in the formation of polychlorinated dibenzo-p-dioxins/dibenzofurans from residual carbon on incinerator fly ash. *Environmental Science and Technology* 32:3356–3359.

Aftalion, F. 1991. *A History of the International Chemical Industry*. Translated by O.T. Benfey. Philadelphia: University of Pennsylvania Press.

Airey, D., and P.D. Jones. 1982. Mercury in the River Mersey, its estuary and tributaries during 1973 and 1974. *Water Research* 16:565–577.

Aittola, J.P., J. Paasivirta, and A. Vattulainen. 1993. Measurements of organochloro compounds at a metal reclamation plant. *Chemosphere* 27:65–72.

Alcock, R.E., and K.C. Jones. 1996. Dioxins in the environment: a review of trend data. *Environmental Science and Technology* 30:3133–3143.

Allan., R, D., Peakall, V. Cairns, J. Cooley, G. Fox, A. Gilman, D. Piekarz, J. van Oostdam, D. Villeneuve, and M. Whittle. 1991. *Toxic Chemicals in the Great Lakes and Associated Effects, Volume I: Contaminant Levels and Trends*. Ottawa: Environment Canada.

Altwicker, E.R., R.K.N.V. Konduri, C. Lin, and M.S. Milligan. 1993. Formation of precursors to chlorinated dioxin/furans under heterogeneous conditions. *Combustion Science and Technology* 88:349–368.

American Chemistry Council. 2000. Response of the Phthalate Esters Panel of the American Chemistry Council to a briefing paper submitted by Joe Thornton, Ph.D., acting under the direction of the Center for Maximum Potential Building Systems and the Healthy Building Network. Arlington, VA, American Chemistry Council.

Andersson, P., K. Ljung, G. Soderstrom, and S. Marklund. 1993. *Analys av Polyklorerade Dibensofuraner och Polykloerarde Dibensodioxiner i Processprover fran Hydro Plast AB*. Umea, Sweden: Institute of Environmental Chemistry, University of Umea.

APHA (American Public Health Association). 1996. Resolution: Prevention of dioxin generation from PVC plastic use by health care facilities. Adopted at APHA Annual Meeting, New York, November.

———. 1994. Resolution 9304: Recognizing and addressing the environmental and occupational health problems posed by chlorinated organic chemicals. *American Journal of Public Health* 84:514–515.

APR (Association of Postconsumer Plastics Recyclers). 1998. APR takes a stand on PVC. Press release. Washington, DC, April 14.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Mercury (update). Washington, DC: U.S. Public Health Service.

———. 1995. Toxicological Profile for Vinyl Chloride (update). Washington, DC: U.S. Public Health Service.

———. 1993. Toxicological Profile for 1,2-Dichloroethane. Washington, DC: U.S. Public Health Service.

Ayres, R. 1997. The life-cycle of chlorine, part I: chlorine production and the chlorine-mercury connection. *Journal of Industrial Ecology* 1:81–94.

Badr, M.Z. 1992. Induction of peroxisomal enzyme activities by di-2-ethylhexylphthalate in thyroidectomized rats with parathyroid replants. *Journal of Pharmacology and Experimental Therapy* 263:1105–1110.

Barrie, L., R. Macdonald, T. Bidleman, M. Diamond, D. Gregor, R. Semkin, W. Strachan, M. Alaee, S. Backus, M. Bewers, C. Gobeil, C. Halsall, J. Hoff, A. Li, L. Lockhart, D. Mackay, D. Muir, J. Pudykiewicz, K. Reimer, J. Smith, G. Stern, W. Schroeder, R. Wagemann, R. Wania, and M. Yunker. 1997. Chapter 2: Sources, occurrence and pathways. In: J. Jensen, K. Adare, R. Shearer eds.. *Canadian Arctic Contaminants Assessment Report*. Ottawa: Indian and Northern Affairs Canada.

Blankenship, A., D.P.Y. Chang, A.D. Jones, P.B. Kelly, I.M. Kennedy, F. Matsumura, R. Pasek, and G.S. Yang. 1994. Toxic combustion by-products from the incineration of chlorinated hydrocarbons and plastics. *Chemosphere* 28:183–196.

Blount, B.C., M.J. Silva, S.P. Caudill, et al. 2000. Levels of seven urinary phthalate metabolites in a human reference population. *Environmental Health Perspectives* 108:979–982.

Bowermaster, J. 1993. A town called Morrisonville. *Audubon* 95(4):42–51.

Bruce, K.R., L.O. Beach, and B.K. Gullett. 1991. The role of gas-phase Cl2 in the formation of PCDD/PCDF during waste combustion. *Waste Management* 11:97–102.

Brun, G.L., G.D. Howell, and H.J. ONeill. 1991. Spatial and temporal patterns of organic contaminants in wet precipitation in Atlantic Canada. *Environmental Science and Technology* 25:1249–1261.

Brzuzy, L.P., and R.A. Hites. 1996a. Global mass balance for polychlorinated dibenzo-p-dioxins and dibenzofurans. *Environmental Science and Technology* 30:1797–1804.

———. 1996b. Response to comment on global mass balance for polychlorinated dibenzo-p-dioxins and dibenzofurans. *Environmental Science and Technology* 30:3647–3648.

Burmaster, D., and R. Harris. 1982. Groundwater contamination: an emerging threat. *Technology Review* 85(5):50–61.

Burnett, R. 1994. Crisis management plans for the dioxin reassessment. Memorandum and enclosures from Vinyl Institute executive director to Vinyl Institute (VI) executive board members and VI issues management committee members. Morristown, NJ: Vinyl Institute, September 6.

Burns, T.F. 2000. Comments on LEED CI Draft Proposed Rating System. Morristown, NJ: Vinyl Institute.

Burress, C. 2000. Berkeley group wants toxic ban: city asked to stop dumping of carcinogens. *San Francisco Chronicle*, October 3.

Cameron, K. 1996. Statement of the Fire Brigades Union on PVC in buildings. Surrey, UK, September 30.

Carroll W. 1995. *Is PVC in House Fires the Great Unknown Source of Dioxin?* Washington DC: The Vinyl Institute.

Carroll, W.F., F.E. Borelli, P.J. Garrity, R.A. Jacobs, J.W. Lewis, R.L. McCreedy, and A.F. Weston. 1996. Characterization of dioxins and furans from ethylene dichloride, vinyl chloride, and polyvinyl chloride facilities in the United States: resin, treated wastewater and ethylene dichloride. *Organohalogen Compounds* 27:62–67.

CDC (U.S. Centers for Disease Control). 1999. *Health Consultation: Exposure Investigation Report, Calcasieu Estuary, Lake Charles, Calcasieu Parish, Louisiana*. Washington DC: Department of Health and Human Services.

Chang, D., M. Richards, and G. Huffman. 1988. Studies of POHC DE during simulated atomization failure in a turbulent flame reactor. In: *Land Disposal, Remedial Action, Incineration and Treatment of Hazardous Waste, Proceedings of the Fourteenth Annual Research Symposium*. Cincinnati: U.S. Environmental Protection Agency Hazardous Waste Engineering Laboratory (EPA 600/9–88/021).

Chicago Tribune. 1996. Hazardous lead levels exist in plastic, vinyl miniblinds. July 8:A7.

Chlorine Institute. Production of chlor-alkali chemicals. Washington DC: The Chlorine Institute, 1991.

———. 2000. *Third Annual Report of the Chlorine Institute to U.S. EPA and Environment Canada*. Washington, DC: May.

Christanesen, K., A. Grove, L.E. Hansen, L. Hoffman, A.A. Hensen, K. Pommer, and A. Schmidt. 1990. *Environmental Assessment of PVC and Selected Alternative Materials*. Copenhagen: Danish Ministry of the Environment.

Christmann, W. 1989. Combustion of polyvinyl chloride—an important source for the formation of PCDD/PCDF. *Chemosphere* 19:387–392.

Christmann, W., K.D. Koppel, H. Partscht, and W. Rotard. 1989. Determination of PCDDs/PCDFs in ambient air. *Chemosphere* 19:521–6.

Cleverly, D., M. Monetti, L. Phillips, P. Cramer, M. Heit, S. McCarthy, K. O'Rourke, J. Stanley, and D. Winters. 1996. A time-trends study of the occurrences and levels of CDDs, CDFs and dioxin-like PCBs in sediment cores from 11 geographically distributed lakes in the United States. *Organohalogen Compounds* 28:77–81.

Cohen M, Commoner B, Eisl H, Bartlett P, Dickar A, Hill C, Quigley J, Rosenthal J. Quantitative Estimation of the Entry of Dioxins, Furans, and Hexachlorobenzene into the Great Lakes from Airborne and Waterborne Sources. Flushing, NY: Center for the Biology of Natural Systems, Queens College, City University of New York, 1995.

Colborn, T., D. Dumanoski, and J.P. Myers. 1996. *Our Stolen Future*. New York: Dutton, 1996.

Collins, T. 2001. Towards sustainable chemistry. *Science* 291:48–50.

———. 2000. A call for a chlorine sunset: the hazards of organochlorines require that they be eliminated. *Nature* 406:17–18.

Colon, I., D. Caro, C. Bourdnoy, and O. Rosario. 2000. Identification of phtahalte esters in the serum of young Puerto Rican girls with premature breast development. *Environmental Health Perspectives* 108:895–900.

Costner, P. 1997. *The Burning Question: Chlorine and Dioxin*. Washington DC: Greenpeace USA.

Costner, P., C. Cray, G. Martin, B. Rice, D. Santillo, and R. Stringer. 1995. *PVC: A Principal Contributor to the U.S. Dioxin Burden*. Washington DC: Greenpeace USA.

Cummings, M. 2001. Personal electronic mail communication with B. Walsh of the Healthy Building Network, May 9.

Curry, M.S., M.T. Huguenin, A.J. Martin, and T.R. Lookingbill. 1996. *Contamination Extent Report and Preliminary Evaluation for the Calcasieu Estuary*. Silver Spring, MD: National Oceanic And Atmospheric Administration.

Czuczwa, J.M., and R.A. Hites. 1986. Airborne dioxins and dibenzofurans: sources and fates. *Environmental Science and Technology* 20:195–200.

———. 1985. Historical record of polychlorinated dioxins and furans in Lake Huron Sediments. In: L.H. Keith, C. Rappe, and G. Choudhary, eds. *Chlorinated Dioxins and Dibenzofurans in the Total Environment II*. Boston: Butterworth, 1985:59–63.

———. 1984. Environmental fate of combustion-generated polychlorinated dioxins and furans. *Environmental Science and Technology* 186:444–449.

Czucwa, J.M., B.D. Veety, and R.A. Hites. 1984. Polychlorinated dibenzo-p-dioxins and dibenzofurans in sediments from Siskiwit Lake, Isle Royale. *Science* 226:568–569.

Danish Environmental Protection Agency. 1993. *PVC and Alternative Materials*, English translation. Copenhagen: Danish Environmental Protection Agency.

Davies, F.C.W. 1991. Minimata disease: A 1989 update on the mercury poisoning epidemic in Japan. *Environmental Geochemistry and Health* 13:35–38.

De Santiago, A., and M. Aguilar-Santelises. 1999. Organotin compounds decrease in vitro survival, proliferation and differentiation of normal human B lymphocytes. *Human Experimental Toxicolology* 18:619–24.

de Swart, R.L., P.S. Ross, J.G. Vos, and A.D.M.E. Osterhaus. 1996. Impaired immunity in harbour seals (*Phoca vitulina*) exposed to bioaccumulated environmental contaminants: Review of a long-term feeding study. *Environmental Health Perspectives* 104 (Supplement 4):823–828.

Dellinger, B., P. Taylor, D. Tiery, J. Pan, and C.C. Lee. 1988. Pathways of PIC formation in hazardous waste incinerators. In: *Land Disposal, Remedial Action, Incineration and Treatment of Hazardous Waste, Proceedings of the Fourteenth Annual Research Symposium*. Cincinnati: U.S. EPA Hazardous Waste Engineering Research Laboratory (EPA 600/9–88/021).

DeLorey, D., D. Cronn, and J. Farmer. 1988. Tropospheric latitudinal distributions of CF2Cl2, CFCl3, N20, CH3CCl3, and CCl4 over the remote Pacific Ocean. *Atmospheric Environment*. 22:1481–1494.

Denmark Skatteministeriet. 2001. Taxes on polyvinylchloride and phthalate act. Available at: [www.skm.dk/slutfil,php3?SlutFilId=690].

Deutsch, D.G., and T.D. Goldfarb. 1988. PCDD/PCDF contamination following a plastics fire in a university lecture hall building. *Chemosphere* 12:2423–2431.

DeVito, M.J., L.S. Birnbaum, W.H. Farland, and T. Gasiewicz. 1995. Comparisons of estimated human body burdens of dioxinlike chemicals and TCDD body burdens in experimentally exposed animals. *Environmental Health Perspectives* 103:820–831.

Dewailly, E., P., Ayotte, S. Bruneau, C. Laliberte, D.C.G. Muir, and R.J. Norstrom. 1993. Inuit exposure to organochlorines through the aquatic food chain in Arctic Quebec. *Environmental Health Perspectives* 101:618–620.

Dine, T., M. Luyckx, B. Gressier, et al. 2000. A pharmacokinetic interpretation of increasing concentrations of DEHP in hemodialysed patients. *Medical Engineering and Physics* 22:157–165.

Dow Chemical. 1990. *Waste Analysis Sheet: Heavy Ends from the distillation of Ethylene Dichlorine in Ethylene Dichloride Production*. Plaquemine, LA, February 21.

Downs, P. 2001. Mold and Mildew. Available at: [http://www.paintstore.com/archives/mildew/viny0895.html].

DTI (Danish Technical Institute). 1995. *Environmental Aspects of PVC*. Copenhagen, November.

Duffy, R.M. 1998. Letter to Concord School Board Members. International Association of Fire Fighters. Washington DC, April 14.

Ecocycle Commission of the Government of Sweden. 1994. *PVC: A Plan to Prevent Environmental Impact*. Stockholm: Ecocycle Commission, 104.

Eklund, G., J. Pedersen, and B. Stromberg. 1988. Methane, hydrogen chloride, and oxygen form a wide range of chlorinated organic species in the temperature range 400 C–950 C. *Chemosphere* 173:575–586.

Ema, M., T. Iwase, Y. Iwase, N. Ohyama, and Y. Ogawa. 1996. Change of embryotoxic susceptibility to di-n-butyltin dichloride in cultured rat embryos. *Archives of Toxicology* 70:742–8.

Endo, R. 1990. World demand for PVC (presentation materials). The Second World Chlor-alkali Symposium. Washington, DC, September.

ENDS. 2000. Denmark defies EU opposition to ban lead. *ENDS Daily*, 14 November.

Energy Information Administration, Office of Coal, Nuclear, Electric and Alternate Fuels. 1996. *Electric Power Annual 1995, Volume I*. Washington, DC: U.S. Department of Energy (DOE/EIA-0348(95)/1, Distribution Category UC-950).

Environment Agency. 1997. *Regulation of Dioxin Releases from the Runcorn Operations of ICI and EVC*. Warrington, UK.

EPA (U.S. Environmental Protection Agency). 2000a. Exposure and Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds (review draft). Washington, DC: U.S. EPA Office of Research and Development.

———. 2000b. Integrated Risk Information System, record for Bis (2-ethylhexyl)phthalate. Available at: [www.epa.gov/ncea/iris.htm]. Accessed November.

———. 1998. The Inventory of Sources of Dioxin in the United States (review draft). Washington, DC: U.S. EPA, Office of Research and Development (EPA/600/P–98–002a), 1998.

———. 1994a. Health Assessment Document for 2,3,7,8- tetrachlorodibenzo-p-dioxin and Related Compounds, Volumes I–III, Review Draft. Washington, DC: U.S. EPA, Office of Research and Development (EPA/600/BP–92–001).

———. 1994b. Estimating Exposures to Dioxin-like Compounds, Volumes I–III (review draft). Washington, DC: U.S. EPA, Office of Research and Development (EPA/600/6–88–005).

———. 1990. Standards for owners and operators of hazardous wastes incinerators and burning of hazardous wastes in boilers and industrial furnaces; proposed and supplemental proposed rule, technical corrections, and request for comments. *55 Federal Register 82*, April 27.

———. 1989. Background Document for the Development of PIC Regulations from Hazardous Waste Incinerators. Washington, DC: U.S. Environmental Protection Agency, Office of Solid Waste.

———. 1988. Specific Comments on the Food and Drug Administration's Evaluation on the Environmental Issues Associated with the Proposed Rule on PVC. Washington, DC: U.S. EPA, Office of Federal Activities, May 23.

———. 1987a. National Dioxin Study: Report To Congress. Washington, DC: U.S. EPA, Office of Air Quality Planning and Standards (EPA/530–SW–87–025).

———. 1987b. National Dioxin Study Tier 4—Combustion Sources: Engineering Analysis Report, Washington, DC: U.S. EPA Office of Air Quality Planning and Standards (EPA/450–84—014h).

———. 1985. Health Assessment Document for 2,3,7,8- tetrachlorodibenzo-p-dioxin. Washington, DC: U.S. EPA Office of Health and Environmental Assessment (EPA/600–8–84/014f).

EPA SAB (U.S. Environmental Protection Agency, Science Advisory Board). 1985. Report on the Incineration of Liquid Hazardous Wastes by the Environmental Effects, Transport and Fate Committee. Washington, DC.

European Commission. 2000. Green Paper: Environmental Issues of PVC (COM (2000)469, 26/7/2000). Brussels.

Evers, E. 1989. *The formation of polychlorinated dibenzofurans and polychlorinated dibenzo-p-dioxins and related compounds during oxyhydrochlorination of ethylene*. Amsterdam: University of Amsterdam, Department of Environmental and Toxicological Chemistry.

Evers, E.H.G., K. Ree, and K. Olie. 1988. Spatial variations and correlations in the distribution of PCDDs PCDFs and related compounds in sediments from the river Rhine. *Chemosphere* 17:2271–2288.

Evers, E.H.G., H. Klamer, R. Laane, and H. Govers. 1993. Polychlorinated dibenzo-p-dioxin and dibenzofuran residues in estuarine and coastal North Sea sediments: sources and distribution. *Environmental Toxicology and Chemistry* 12:1583–1598.

Evers, E.H.G., R.W.P.M. Laane, G.J.J. Groeneveld, and K. Olie. 1996. Levels, temporal trends and risk of dioxins and related compounds in the Dutch aquatic environment. *Organohalgen Compounds* 28:117–122.

Fiedler, H., O. Hutzinger, and J. Hosseinpour. 1993. Analysis and remedial actions following an accidental fire in a kindergarten. *Organohalogen Compounds* 14:19–23.

Frankenhaeuser, M., H. Manninen, I. Kojo, J. Ruuskanen, T. Vartiainen, R. Vesterinen, and J. Virkki. 1993. Organic emissions from co-combustion of mixed plastics with coal in a bubbling fluidized bed boiler. *Chemosphere* 27:309–316.

Franklin Associates 1997. Characterization of Municipal Solid Waste in the United States: 1996 Update (EPA-530-R-97-015) Quoted in Hammer Consulting 1998. *It's Perfectly Clear: The Case against PVC Packaging*. Boston: Masspirg Education Fund, April.

Fuhrer, U., and K. Ballschmitter. 1998. Bromochloromethoxybenzenes in the marine troposphere of the Atlantic ocean: a group of organohalogens with mixed biogenic and anthropogenic origin. *Environmental Science and Technology* 32:2208–2215.

Furst, P. 2000. PCDD/PCDFs in human milk: still a matter of concern? *Organohalogen Compounds* 48:111–114.

Geiser, K. 2000. Presentation materials. Organizing Meeting of the Healthy Building Network. Bolinas, CA, June.

German Environment Ministers. 1992. *Impacts on the Environment from the Manufacture, Use and Disposal and Substitution of PVC*. Hamburg: German Joint Federal-State Committee on Environmental Chemicals for the German Environmental Ministers.

Giam, C.S., H.S. Chan, G.S. Neff, and E.L. Atlas. 1978. Phthalate ester plasticizers: a new class of marine pollutant. *Science* 199:419–421.

Giesy, J.P., J.P. Ludwig, and D.J. Tillitt. 1994a. Deformities in birds of the Great Lakes region: assigning causality. *Environmental Science and Technology* 28:128a–134a.

———. 1994b. Dioxins, dibenzofurans, PCBs and wildlife. In: A. Schecter, ed. *Dioxins and Health*. New York: Plenum, 249–308.

Gilbertson, M.G., T. Kubiak, J. Ludwig, and G. Fox. 1991. Great Lakes embryo mortality, edema, and deformities syndrome (GLEMEDS) in colonial fish-eating birds: similarity to chick edema disease. *Journal of Toxicology and Environmental Health* 33:455–520.

Goldspiel, B.R. 1994. Pharmaceutical issues: preparation, administration, stability, and compatibility with other medications. *Annals of Pharmacotherapy* 28:S23–S26.

Goodman, D. 1994. Memorandum: Incineration task force. Washington, DC: Morrisville, NJ, August 22.

Gray, L.E., C. Wolf, C. Lambright, P. Mann, M. Price, R.L. Cooper, and J. Ostby. 1999. Administration of potentially antiandrogenic pesticides (procymidone, linuron, iprodione, chlozolinate, p,p'-DDE, and ketoconazole) and toxic substances (dibutyl- and diethylhexyl phthalate, PCB 169, and ethane dimethane sulphonate) during sexual differentiation produces diverse profiles of reproductive malformations in the male rat. *Toxicology and Industrial Health* 15:94–118.

Great Lakes Water Quality Board. 1987. *1987 Report on the Great Lakes Water Quality*. Windsor, ON: International Joint Commission.

Green, A.E.S. 1993. The future of medical waste incineration. In: A.E.S. Green, ed. *Medical Waste Incineration and Pollution Prevention*. New York: Van Nostrand Reinhold:170–207.

Greenpeace International. 2000. *PVC-Free Future: A Review of Restrictions and PVC-Free Policies Worldwide*. Amsterdam.

Gregor, D., and W. Gummer. 1989. Evidence of atmospheric transport and deposition of organochlorine pesticides and plychlorinated biphenyls in Canadian Arctic snow. *Environmental Science and Technology* 23:561–565.

Guillette, L.J., and D.A.Crain, eds. 1999. *Environmental Endocrine Disruption: An Evolutionary Approach*. London: Taylor and Francis.

Gullett, B.K. 1990. The effect of metal catalysts on the formation of poychlorinated dibenzo-dioxin and polychlorinated dibenzofuran precursors. *Chemosphere* 20:1945–52.

Halonen, I., J. Tarhanen, T. Kopsa, J. Palonen, H. Vilokki, and J. Ruuskanen. 1993. Formation of polychlorinated dioxins and dibenzofurans in incineration of refuse derived fuel and biosludge. *Chemosphere* 26:1869–1880.

Halonen, I., J. Tarhanen, T., Ruokojarvi, K. Tuppurainen, and J. Ruuskanen. 1995. Effect of catalysts and chlorine source on the formation of organic chlorinated compounds. *Chemosphere* 30:1261–1273.

Hamilton-Wentworth (Regional Municipality of Hamilton-Wentworth, Ontario). 1997. Public Health Statement on Plasti-met Fire. Hamilton, Ontario. Available at [http://www.hamilton-went.on.ca].

Hammer Consulting. 1998. *It's Perfectly Clear: The Case against PVC Packaging*. Boston: Masspirg Education Fund, April.

Hanberg, A. 1999. *Compilation of EU Dioxin Exposure and Health Data*. Oxfordshire: AEA Technology plc and European Commission DG Environment.

Hansen, J. 1999. Risk for testicular cancer after occupational exposure to plastics. *International Journal of Cancer* 82:911–912.

Harada, M. 1995. Minimata disease: methyl mercury poisoning in Japan caused by environmental pollution. *Critical Reviews in Toxicology* 25:1–24.

Hardell, L., A. Nasman, C.G. Ohlson, and M. Fredrikson. 1998. Case-control study on risk factors for testicular cancer. *International Journal of Oncology* 13:1200–1303.

Hardell, L., C.G. Ohlson, and M. Frerikson. 1997. Occupational exposure to polybinyl chloride as a risk factor for testicular cancer evaluated in a case control study. *International Journal of Cancer* 73:828–830.

Harris, C.A., P. Henttu, M.G. Parker, and J.P. Sumpter. 1997. The estrogenic activity of phthalate esters in vitro. *Environmental Health Perspectives* 105:802–811.

Hasselriis, F., and L. Constantine. 1993. Characterization of today's medical waste. In: A.E.S. Green, ed. *Medical Waste Incineration and Pollution Prevention*. New York: Van Nostrand Reinhold, 37–52.

Hatanaka, T., T. Imagawa, and M. Takeuchi. 2000. Formation of PCDD/Fs in artificial solid waste incineration in a laboratory-scale fluidizedbed reactor: influence of contents and forms of chlorine sources in high-temperature combustion. *Environmental Science and Technology* 34:3920–3924.

Heindel, J.J.and R.E. Chapin. 1989. Inhibition of FSH-stimulated cAMP accumulation by mono(2-ethylhexyl)phthalate in primary rat Sertoli cell cultures. *Toxicology and Applied Pharmacology* 97:377–385.

Henschler, D. 1994. Toxicity of chlorinated organic compounds: effects of the introduction of chlorine in organic molecules. *Angewandte Chemie* International Edition (English) 33:1920–1935.

Herman-Giddens, M.E., E.J. Slora, R.C. Wasserman, C.J. Bourdony, M.V. Bhapkar, G.G. Koch, and C.M. Hasemeier. 1997. Secondary sexual characteristics and menses in young girls seen in office practice: a study from the Pediatric Research in Office Settings Network. *Pediatrics* 99:505–512.

Hill, G., and J. Holman. 1989. *Chemistry in Context, Third Edition*. London: Nelson Publishers, 266–269.

HSDB (Hazardous Substances Databank). 1997. Bethesda, MD: National Library of Medicine.

Hsieh, S. 2000. Will toxic mold be the next asbestos? *Lawyers Weekly* 853.

Huestis, S.Y., M.R. Servos, and D.G. Dixon. 1997. Evaluation of temporal and age-related trends of chemically and biologically generated 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents in Lake Ontario trout, 1977–1993. *Environmental Toxicology and Chemistry* 16:154–164.

Hutari, J., and R. Vesterinen. 1996. PCDD/F emissions from co-combustion of RDF with peat, wood waste, and coal in FBC boilers. *Hazardous Waste and Hazardous Materials* 13:1–10.

Hutzinger, O. 1986. Dioxin danger: incineration status report. *Analytical Chemistry* 586:633–642.

Hutzinger, O., and H.. Fiedler. 1988. *Formation of Dioxins and Related Compounds in Industrial Processes.* Pilot Study on International Information Exchange on Dioxins and Related Compounds #173. Brussels: NATO Committee on the Challenges of Modern Society.

ICI Chemicals and Polymers, Ltd. 1994. *Report to the Chief Inspector HMIP Authorization AK6039, Improvement Condition, Part 8, Table 8.1, Item 2—Formation of Dioxins in Oxychlorination: Significance for Human Health and Monitoring Proposals.* Runcorn, UK: ICI Chemicals and Polymers Safety and Environment Department, April.

Imajima, T., T. Shono, O. Zakaria, and S. Suita. 1997. Prenatal phthalate causes cryptorchidism postnatally by inducing transabdominal ascent of the testis in fetal rats. *Journal of Pediatric Surgery* 32:18–21.

International Joint Commission. 1992. *Sixth Biennial Report on Great Lakes Water Quality*. Windsor, Ontario.

Ishibashi, N., Y. Yoshihara, K. Nishiwaki, S. Okajima, M. Hiraoka, and J. Shudo. 2000. The effects of organic- and inorganic-chloride in municipal solid wastes on dioxins formation and emission. *Organohalogen Compounds* 46:122–125.

Isosaari, P., T. Kohonen, H. Kiviranta, J. Tuomisto, and T. Vartiainen. 2000. Assessment of levels, distribution and risks of polychlorinated dibenzo-p-dioxins and dibenzofurans in the vicinity of a vinyl chloride monomer production plant. *Environmental Science and Technology* 34:2684–2689.

Jaakkola, J.J., P.K. Verkasalo, and N. Jaakkola. 2000. Plastic wall materials in the home and respiratory health in young children. *American Journal of Public Health* 90:797–799.

Jaakkola, J.J., L. Oie, P. Nafstad, G. Botten, S.O. Samuelsen, and P. Magnus. 1999. Interior surface materials in the home and the development of bronchialobstruction in young children in Oslo, Norway. *American Journal of Public Health* 89:188–192.

Jay, K., and L. Stieglitz. 1995. Identification and quantification of volatile organic components in emissions of waste incineration plants. *Chemosphere* 30:1249–1260.

Jobling, S., T. Reynolds, R. White, M.G. Parker, and J.P. Sumpter. 1995. A variety of environmentally persistent chemicals, including some phthalate plasticizers, are weakly estrogenic. *Environmental Health Perspectives* 103:582–587.

Johansson, N. 1993. PCDD/PCDF and PCB: the Scandinavian situation. *Organohalogen Compounds* 14:337–340.

Johnston, P.A., S. Trondle, R. Clayton, and R. Stringer. 1993. PVC—The Need for an Industrial Sector Approach to Environmental Regulation. GERL Technical Note 04/93. Exeter: University of Exeter Earth Resources Center.

Juttner, I., B. Henkelmann, K.W. Schramm, C.E.W. Steinberg, R. Winkler, and A. Kettrup. 1997. Occurrence of PCDD/F in dated lake sediments of the Black Forest, Southwestern Germany. *Environmental Science and Technology* 31:806–812.

Kaminsky, R., and R.A. Hites. 1984. Octachlorostyrene in Lake Ontario: sources and fates. *Environmental Science and Technology* 18:275–279.

Kanters, M.J., R. Van Nispen, R. Louw, and P. Mulder. 1996. Chlorine input and chlorophenol emission in the lab-scale combustion of municipal solid waste. *Environmental Science and Technology* 30:2121–2126.

Karol, M.H.. Toxicological principles do not support the banning of chlorine. 1995. *Fundamental and Applied Toxicology* 24:1-2.

KemI (*Kemikalienspektionen*). 2000. *PVC—utveckling mot en miljoanpassad PVC*. Translated by Greenpeace International. Stockholm.

KEMI (Swedish Chemicals Inspectorate). 2000. Summary of Report 6/00: *Organotin Stabilizers in PVC—Assessment of Risks and Proposals for Risk Reduction Measures*. Stockholm.

Kerkvliet, N.I. 1994. Immunotoxicology of dioxins and related chemicals. In: A. Schecter, ed. *Dioxins and Health*. New York: Plenum, 199–217.

Khizbullia, F., I. Muslymova, I. Khasanova, L. Chernova, and J. Abdraschitov. 1998. Evaluation of polychlorinated dibenzodioxins and dibenzofurans emissions from vinylchloride-monomer production. *Organohalogen Compounds* 26:225–229.

Kielhorn, J., C. Melber, U. Wahnschaffe, A. Aitio, and I. Mangelsdorf. 2000. Vinyl chloride: still a cause for concern. *Environmental Health Perspectives* 108:579–588.

Kjeller, L.O., and C. Rappe. 1995. Time trends in levels, patterns, and profiles for polychlorinated dibenzo-p-dioxins, dibenzofurans, and biphenyls in a sediment core from the Baltic proper. *Environmental Science and Technology* 29:346–355.

Kjeller, L.O., K.C. Jones, A.E. Johnston, and C. Rappe. 1996. Evidence for a decline in atmospheric emissions of PCDD/Fs in the UK. *Environmental Science and Technology* 30:1398–1403.

———. 1991. Increases in the polychlorinated dibenzo-p-dioxin and -furan content of soils and vegetation since the 1840s. *Environmental Science and Technology* 25:1619–1627.

Kohn, M.C., F, Parham, S.A. Masten, C.J. Portier, M.D. Shelby, J.W. Brock, and J.W. Needham. 2000. Exposure estimates for phthalates. *Environmental Health Perspectives* 108(10).

Kohn, M.C., C.H. Sewall, G.W. Lucier, and C.J. Portier. 1996. A mechanistic model of effects of dioxin on thyroid hormones in the rat. *Toxicology and Applied Pharmacology* 136:29–48.

Kolenda, J., H. Gass, M. Wilken, J. Jager, and B. Zeschmer-Lahl. 1994. Determination and reduction of PCDD/F emissions from wood burning facilities. *Chemosphere* 29:1927–1938.

Kopponen, P., R. Torronen, J. Ruuskanen, J. Tarhanen, T. Vartiainen, and S. Karenlampi. 1992. Comparison of cytochrome P4501A1 induction with the chemical composition of fly ash from combustion of chlorine containing material. *Chemosphere* 24:391–401.

Kramlich, J.C., E.M. Poncelet, R.E. Charles, W.R. Seeker, G.S. Samuelsen, and J.A. Cole. 1989. *Experimental Investigation of Critical Fundamental Issues in Hazardous Waste Incineration*. Research Triangle, NC: U.S. Environmental Protection Agency Industrial Environmental Research Lab (600/2–89–048).

Lahl, U. 1994. Sintering plants of steel industry: PCDD emission status and perspectives. *Chemosphere* 29:1939–1945.

LaKinjd, J.S., C.M. Berlin, and D.Q. Naiman. 2001. Infant exposure to chemicals in breast milk in the United States: what we need to learn from a breast milk monitoring program. *Environmental Health Perspectives* 109:75–88.

Lambright, C., J. Ostby, K. Bobseine, V. Wilson, A.K. Hotchkiss, P.C. Mann, and L.E. Gray. 2000. Cellular and molecular mechanisms of action of linuron: an antiandrogenic herbicide that produces reproductive malformations in male rats. *Toxicological Sciences* 56:389–399.

Leder, A., E. Linak, R. Holmes, and T. Sasano. 1994. *CEH Marketing Research Report: Chlorine/Sodium Hydroxide*. Palo Alto: SRI International.

Lemiuex, P.M. 1997. *Evaluation of Emissions from the Open Burning of Household Waste in Barrels: Volume 1, Technical Report*. Washington, DC: U.S. Environmental Protection Agency Office of Research and Development, Control Technology Center (EPA–600–R–97–134a).

Lenoir, D., A. Kaune, O. Hutzinger, G. Mutzenrich, and K. Horch. 1991. Influence of operating parameters and fuel type on PCDD/F emissions from a fluidized bed incinerator. *Chemosphere* 23:1491–1500.

Liberti, A., G. Goretti, and M.V. Russo. 1983. PCDD and PCDF formation in the combustion of vegetable wastes. *Chemosphere* 12:661–663.

Licis, I.J., and H.B. Mason. 1989. Boilers cofiring hazardous waste: effects of hysteresis on performance measurements. *Waste Management* 9:101–108.

Ligon, W.V., S.B. Dorn, and R.J. May. 1989. Chlorodibenzofuran and chlorodibenzo-p-dioxin levels in Chilean mummies dated to about 2800 years before the present. *Environmental Science and Technology* 23:1286–1290.

Lindqvist, O., K. Johansson,, M. Aastrup, A. Andersson, L. Bringmark, G. Hovsenius, L. Hakanson, A. Iverfeldt, M. Meili, and B. Timm. 1991. Mercury in the Swedish environment: recent research on causes, consequences and corrective methods. *Water Air Soil Pollution* 55:1–32.

Lower Saxony Ministry of Environmental Affairs. 1994. Data report and press release, dioxin data from ICI facility, Wilhelmshaven, Germany, March 22.

Lstibureck, J.W. and J. Carmody. 1994. *Moisture Control Handbook: Principles and Practices for Residential and Small Commercial Buildings.* New York: Van Nostrand Reinhold.

MAFF (Ministry of Agriculture, Food, and Fisheries/Food Safety Directorate). 1995. *Dioxins in PVC food packaging: Food Surveillance Information Sheet Number 59*. London.

Mahle, N., and L. Whiting. 1980. Formation of chlorodibenzodioxins by air oxidation and chlorination of bituminous coal. *Chemosphere* 9:693–699.

Makhijani, A. and K.R. Gurney. 1995. *Mending the Ozone Hole*. Cambridge: MIT Press.

Maloney, E.K., and D.J. Waxman. 2000. Trans-Activation of PPARa and PPARg by structurally diverse environmental chemicals. *Toxicology and Applied Pharmacology* 161:209–218.

Manninen, H., A. Perkio, T. Vartiainen, and J. Ruuskanen. 1996. Formation of PCDD/PCDF: effect of fuel and fly ash composition on the formation of PCDD/PCDF in the co-combustion of refuse-derived and packaging-derived fuels. *Environmental Science and Pollution Research* 3:129–134.

Mark, F. 1994. *Energy Recovery through Co-combustion of Mixed Plastics Waste and Municipal Solid Waste*. Hamburg: Association of Plastics Manufacturers in Europe.

Markowitz, J.S. 1989. Self-reported short-and long-term respiratory effects in PVC-exposed firefighters. *Archives of Environmental Health* 44:30–33.

Markowitz, J.S., E.M. Gutterman, S. Schwartz, B. Link, and S.M. Gorman. 1989. Acute health effects among firefighters exposed to a polyvinyl chloride (PVC) fire. *American Journal of Epidemiology* 129:1023–1031.

Markus, T., P. Behnisch, H. Hagenmaier, K.W. Bock, and D. Schrenk. 1997. Dioxin-like components in incinerator fly ash: a comparison between chemical analysis data and results from a cell culture bioassay. *Environmental Health Perspectives* 105(12):475–481.

Marrack, D. 1988. Hospital red bag waste: an assessment and management recommendations. *Journal of the Air Pollution Control Association* 38:1309–1311.

Martinez, M, J. Diaz-Ferrero, R. Marti, F. Broto-Puig, L. Comellas, and M.C. Rodriguez-Larena. 2000. Analysis of dioxin-like compounds in vegetation and soil samples burned in Catalan forest fires. Comparison with the corresponding unburned materials. *Chemosphere* 41:1927–1935.

Maserti, B.E. and R. Ferrara. 1991. Mercury in plants, soil, and atmosphere near a chlor-alkali complex. *Water, Air, and Soil Pollution* 56:15–20.

Masuyama, H., Y. Hiramatsu, M. Kunitomi, T. Kudo, and P.N. MacDonald. 2000. Endocrine disrupting chemicals, phthalic acid and nonylphenol, activate pregnane X receptor-mediated transcription. *Molecular Endocrinology* 14:421–428.

Mattila, H., T. Virtanen, T. Vartiainen, and J. Ruuskanen. 1992. Emissions of polychlorianted dibenzo-p-dioxins and dibenzofurans in flue gas from co-combustion of mixed plastics with coal and bark. *Chemosphere* 25:1599–1609.

Mayer, C.E. 1998. CPSC won't seek phthalate ban; agency asks that the chemicals not be used in some toys. *Washington Post*, December 2, C16.

McGregor, D.B., C. Partensky, J. Wilbourn, and J.M. Rice. 1998. An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. *Environmental Health Perspectives* 106 (Supplement 2):755–760.

Mears, C.L. 1995. Presentation materials for: Where will the chlorine industry be in ten years? Conference on The Changing Chlorine Marketplace: Business Science and Regulations. New Orleans, April.

Melnick, R.L. 2001. Is peroxisome proliferation an obligatory precursor step in the carcinogenicity of Di(2-ethylhexyl)phthalate (DEHP)? *Environmental Health Perspectives* 109:437–444.

Merkord, J., H. Weber, G. Kroning, and G. Hennighausen. 2000. Antidotal effects of 2,3-dimercapto-propane-1-sulfonic acid (DMPS) and meso-2,3-dimercaptosuccinic acid (DMSA) on the organotoxicity of dibutyltin dichloride (DBTC) in rats. *Human Experimental Toxicology* 19:132–7.

Miller, A.B., D. Bates, T. Chalmers, M.J. Coye, J. Froines, D. Hoel, J. Schwartz, P. Schulte, D.L. Davis, L.M. Poore, and P. Adams. 1991. *Environmental Epidemiology: Public Health and Hazardous Wastes.* Washington, DC: National Academy Press.

National Academy of Sciences, Committee on the Toxicological Effects of Methyl Mercury. 2000. *Toxicological Effects of Methyl mercury*. Washington, DC: National Academy Press.

National Institutes of Health. 1998. U.S. Toxics Release Inventory for 1998. Available at [http://www.medline.nlm.nih.gov].

National Research Council. 2000. *Hormonally Active Agents in the Environment.* Washington, DC: National Academy Press.

National Toxicology Program. 2001. Ninth Report on Carcinogens (Revised January 2001). Washington, DC: U.S. Department of Health and Human Services.

National Toxicology Program Center for the Evaluation of Risks to Human Reproduction. 2000a. *NTP-CERHR Expert Panel Report on Di(2-ethylhexyl)phthalate*. Washington, DC: U.S. Department of Health and Human Services.

———. 2000b. *NTP-CERHR Expert Panel Report on Butylbenzylphthalate*. Washington, DC: U.S. Department of Health and Human Services.

———. 2000c. *NTP-CERHR Expert Panel Report on Di-isononylphthalate*. Washington, DC: U.S. Department of Health and Human Services.

Neubert, R., U. Jacob-Muller, H. Helge, R. Stahlmann, and D. Neubert. 1991. Polyhalogenated dibenzo-p-dioxins and dibenzofurans and the immune system: 2. In vitro effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on lymphocytes of venous blood from man and a non-human primate (Callithrix jacchus). *Archives of Toxicology* 65:213–219.

New York Times. 1998. Toys-R-Us halting sale of some infant products. November 14, C4.

Norstrom, R.J., M. Simon, and D.C.G. Muir. 1990. Polychlorinated dibenzo-p-dioxins and dibenzofurans in marine mammals of the Canadian North. *Environmental Pollution* 66:1–19.

Ohlson, C.G., and L. Hardell. 2000. Testicular cancer and occupational exposures with a focus on xenoestrogens in polyvinyl chloride plastics. *Chemosphere* 40:1277–1282.

Oie, L., L.G. Hersough, and J.O. Madsen. 1997. Residential exposure to plasticizers and its possible role in the pathogenesis of asthma. *Environmental Health Perspectives* 105:972–978.

Olsson, M., B. Karlsson, and E. Ahnland. 1994. Diseases and environmental contaminants in seals from the Baltic and Swedish west coast. *Science of the Total Environment* 154:2317–227.

Ono, M., N. Kanna, T. Wakimoto, and R. Tatsukawa. 1987. Dibenzofurans: a greater global pollutant than dioxins? Evidence from analyses of open ocean killer whale. *Marine Pollution Bulletin* 18:640–643.

Onstot, J., R. Ayling, and J. Stanley. 1987. *Characterization of HRGC/MS Unidentified Peaks from the Analysis of Human Adipose Tissue, Volume I: Technical Approach*. Washington, DC: U.S. Environmental Protection Agency Office of Toxic Substances (560/6–87–002a).

Pacyna, J.M., and J. Munch. 1991. Anthropogenic mercury emission in Europe. *Water, Air, and Soil Pollution* 56:51–61.

Pagnetto, G., F. Campi, K. Varani, A. Piffanelli, G. Giovannini, and P.A. Borea. 2000. Endocrine-disrupting agents on healthy human tissues. *Pharmacology and Toxicology* 86:24–29.

Panda, K.K., M. Lenka, and B.B. Panda. 1990. Monitoring and assessment of mercury pollution in the vicinity of a chlor-alkali plant. *Science of the Total Environment* 96:281–296.

Pandompatam, B., Y. Kumar, I. Guo, and A.J. Liem. 1997. Comparison of PCDD and PCDF emissions from hog fuel boilers and hospital waste incinerators. *Chemosphere* 35:1065–1073.

Papp, R. 1996. Organochlorine waste management. *Pure and Applied Chemistry* 68:1801–1808.

Patton, G.W., M.D. Walla, T.F. Bidleman, and L.A. Barrie. 1991. Polycyclic aromatic and organochlorine compounds in the atmosphere of northern Ellesmere Island. *Canadian Journal of Geophysical Research* 96 (D6):10867–10877.

Pearson, S.D., and L.A. Trissel. 1993. Leaching of diethylhexyl phthalate from polyvinyl chloride containers by selected drugs and formulation components. *American Journal of Hospital Pharmacology* 50:1405–1409.

Peterson, R.E., R.W. Moore, T.A. Mably, D.L. Bjerke, and R.W. Goy. 1992. Male reproductive system ontogeny: effects of perinatal exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. In: T. Colborn and C. Clement, eds. *Chemically-Induced Alterations in Sexual and Functional Development: The Wildlife/Human Connection*. Princeton: Princeton Scientific Publishing, 175–194.

Pitot, H.C., and Y.P. Dragan. 1991. Facts and theories concerning the mechanisms of carcinogenesis. *FASEB Journal* 5:2280–2286.

Plonait, S.L., H. Nau, R.F. Maier, W. Wittfoht, and M. Obladen. 1993. Exposure of newborn infants to di-(2-ethylhexyl)-phthalate and 2-ethylhexanoic acid following exchange transfusion with polyvinyl chloride catheters. *Transfusion* 33:598–605.

Plumacher, J., and P. Schroder. 1993. Accumulation and fate of C2-chlorocarbons and trichloroacetic acid in spruce needles from an Austrian mountain site. *Organohalogen Compounds* 14:301–302.

Portier, C.J., C.D. Sherman, M. Kohn, L. Edler, A. Kopp-Schneider, R.M. Maronpot, and G. Lucier. 1996. Modeling the number and size of hepatic focal lesions following exposure to 2,3,7,8-TCDD. *Toxicology and Applied Pharmacology* 138:20–30.

Ramacci, R., G. Ferrari, and V. Bonamin. 1998. Sources of PCDDs/PCDFs from industrial and municipal waste water discharges and their spatial and temporal distribution in the sediments of the Venice lagoon. *Organohalogen Compounds* 39:91–95.

Rappe, C., L.O. Kjeller, and S.E. Kulp. 1991. Levels, profile and pattern of PCDDs and PCDFs in samples related to the production and use of chlorine. *Chemosphere* 23:1629–1636.

Reijnders, P.J.H. 1986. Reproductive failure in common seals feeding on fish from polluted coastal waters. *Nature* 324:456–457.

Rigo, H., A. Chandler, and W. Lanier. 1995. *The Relationship between Chlorine in Waste Streams and Dioxin Emissions from Combustors*. New York: American Society of Mechanical Engineers.

Rossberg, M., W. Lendle, A. Togel, E.L. Dreher, E. Langer, H. Rassaerts, P. Kleinshmidt, H. Strack, U. Beck, K.A. Lipper, T. Torkelson, E. Loser, and K.K. Beutel. 1986. Chlorinated hydrocarbons. In: W. Gerhartz, ed. *Ullman's Encyclopedia of Industrial Chemistry, Fifth Edition*. New York: VCH Publishers, A6:233–398.

Rudell, R. 2000. Polycycling aromatic hydrocarbons, phtahalates, and phenols. In: Spangler, J.D., McCarthy, J.F., Samet, J.M., eds. *Indoor Air Quality Handbook*. New York: McGraw-Hill, 34.1–34.26.

Rudell, R.A., P.W. Geno, G. Sun, A. Yau, J.D. Spengler, J. Vallarino, and J.G. Brody. Forthcoming. Identification of selected hormonally active agents and animal mammary carcinogens in commercial and residential air and dust samples. *Journal of Air and Waste Management Association*.

Ruuskanen, J., T. Vartiainen, L. Kojo, H. Manninen, J. Oksanen, and M. Frankenhauswer. 1994. Formation of polychlorinated dibenzo-p-dioxins and dibenzofurans in co-combustion of mixed plastics with coal: exploratory principal component analysis. *Chemosphere* 28:1989–1999.

Schaum, J., D. Cleverly, M. Lorber, L. Phillips, and G. Schweer. 1993. Sources of dioxin-like compounds and background exposure levels. *Organohalogen Compounds* 14:319–326.

Schecter, A. 1991. Dioxins in humans and the environment. In: Biological Basis for Risk Assessment of Dioxins and Related Compounds. *Banbury Report* 35:169–214.

Schecter, A., and H. Kessler. 1996. Dioxin and dibenzofuran formation following a fire at a plastic storage warehouse in Binghamton, New York in 1995. *Organohalogen Compounds* 27:187–190.

Schecter, A., A. Dekin, N.C.A. Weerasinghe, S. Arghestani, and M.L. Gross. 1988. Sources of dioxins in the environment: a study of PCDDs and PCDFs in ancient frozen Eskimo tissue. *Chemosphere* 17:627–631.

Schmittinger, P., L.C. Curlin, T. Asawa, S. Kotowski, H.B. Beer, A.M. Greenberg, E. Zelfel, and R. Breitstadt. 1986. Chlorine. In: W. Gerhartz, ed. *Ullman's Encyclopedia of Industrial Chemistry, Fifth Edition*. New York: VCH Publishers, A6:399–480.

Schneider, K.G., and A.S. Keenan. 1997. A documented historical performance of roofing assemblies in the United States. *Proceedings of the Fourth International Symposium on Roofing Technology*. U.S. National Roofing Contractors Association.

SEPA (Swedish Environmental Protection Agency). 1994. *Sample reports 610s09 and 0610s017: PVC suspension/PVC plastic and PVC suspension used to make PVC*. Stockholm: Swedish Environmental Protection Agency, May 5.

SFT (Norwegian State Pollution Control Authority). 1993. Input of organohalogens to the convention area from the PVC industry. Submission to the Oslo and Paris Commissions, Oslo, November.

Simonich, S.L., and R.A. Hites. 1995. Global distribution of persistent organochlorine compounds. *Science* 269:1851–1854.

Socha, A., S. Abernethy, B. Birmingham, R. Bloxam, S. Fleming, D. McLaughlin, and D. Spry. 1997. Plastimet Inc. *Fire, Hamilton, Ontario, July 9–12*, 1997. Ottawa: Ontario Ministry of Environment and Energy.

Solomon K, Bergman H, Huggett R, Mackay D, McKague B. 1993. A Review and Assessment of the Ecological Risks Associated with the Use of Chlorine Dioxide for the Bleaching of Pulp. Washington DC: Alliance for Environmental Technlogy.

SRI International. 1993. *The Global Chlor-Alkali Industry: Strategic Implications and Impacts*. Zurich.

Staley, L., M. Richards, G. Huffman, and D. Chang. 1986. *Incinerator Operating Parameters which Correlate with Performance*. Cincinnati: U.S. EPA Hazardous Waste Engineering Research Laboratory (EPA 600/2–86–091).

Stanley, J.S., R.E. Ayling, P.H. Cramer, K.R. Thornburg, J.C. Remmers, J.J. Breen, J. Schwemberger, H.K. Kiang, and K. Watanabe. 1990. Polychlorinated dibenzo-p-dioxin and dibenzofuran concentration levels in human adipose tissue samples from the continental United States collected from 1971 to 1987. *Chemosphere* 20:895–892.

Svalander, J.R. 1996. Presentation materials on pursuing new horizons: taking PVC beyond the millennium. PVC '96 New Perspectives Conference. Brighton, UK, April.

Svensson, B.G., L. Barregard, G. Sallsten, A. Nilsson, M. Hansson, and C. Rappe. 1993. Exposure to polychlorinated dioxins and dibenzofurans from graphite electrodes in a chloralkali plant. *Chemosphere* 27:259–262.

Takasuga, T., T. Makino, K. Tsubota, and N. Takeda. 2000. Formation of dioxins (PCDDs/PCDFs) by dioxin-free fly ash as a catalyst and relation with several chlorine sources. *Chemosphere* 40:1003–1007.

Tamade, Y., T. Ikeguchi, Y. Yagi, K. Yoneda, and K. Omori. 2000. Generation of dioxins from waste plastics combustion in a fluidized bed incinerator. *Organohalogen Compounds* 46:166–169.

Tatsukawa, R., and S. Tanabe. 1990. Fate and bioaccumulation of persistent organochlorine compounds in the marine environment. In: D.J. Baumgartner, and I.M. Dudall, eds. *Oceanic Processes in Marine Pollution, Volume 6*. Malabar, FL: RE Krieger, 39–55.

Taylor, F. 1957. *A History of Industrial Chemistry*. New York: Abelard Schuman.

Tellus Institute. 1992. CSG/Tellus Packaging Study for the Council of State Governments, U.S. Environmental Protection Agency, and New Jersey Department of Environmental Protection and Energy. Boston.

Theisen, J. 1991. *Untersuchung der Moglichen Umweltgefahrdung Beim Brand Von Kunststoffen (Investigation of Possible Environmental Dangers Caused by Burning Plastics)*. Berlin: German Umweltbundesamt (104–09–222).

Theisen, J., W. Funcke, E. Balfanz, and J. Konig. 1989. Determination of PCDFs and PCDDs in fire accidents and laboratory combustion tests involving PVC-containing materials. *Chemosphere* 19:423–428.

Thomas V, Spiro C. An estimation of dioxin emissions in the United States. *Toxicology and Environmental Chemistry* 50:1-37, 1995.

Thornton, J. 2000. *Pandora's Poison: Chlorine, Health, and a New Environmental Strategy*. Cambridge: MIT Press.

Tittle, W.L. 1995. Presentation materials for: Where will the chlorine industry be in ten years. Conference on The Changing Chlorine Marketplace: Business Science and Regulations. New Orleans, April.

TNO Centre for Technology and Policy Studies. 1996. *A PVC substance flow analysis for Sweden: Report for Norsk-Hydro*. Apeldoorn, Netherlands.

TNO Institute of Environmental and Energy Technology. 1994. *Sources of Dioxin Emissions into the Air in Western Europe*. Brussels: Eurochlor.

Tong, H.Y., M.L.O. Gross, A. Schecter, S.J.Monson, and A. Dekin. 1990. Sources of dioxin in the environment: second stage study of PCDD/Fs in ancient human tissue and environmental samples. *Chemosphere* 20:987–992.

Trenholm, A., and C.C. Lee. 1986. Analysis of PIC and total mass emissions from an incinerator. In: *Land Disposal, Remedial Action, Incineration, and Treatment of Hazardous Waste, Proceed-ings of the Twelfth Annual Research Symposium*. Cincinnati: U.S. Environmental Protection Agency Hazardous Waste Engineering Research Laboratory (EPA 600/9–86/022).

Trenholm, A., and R. Thurnau. 1987. Total mass emissions from a hazardous waste incinerator. In: *Land Disposal, Remedial Action, Incineration, and Treatment of Hazardous Waste, Proceedings of the Thirteenth Annual Research Symposium*. Cincinnati: U.S. Environmental Protection Agency Hazardous Waste Engineering Laboratory (EPA/600/9–87/015).

Trenholm, A., P. Agorman, and G. Jungclaus. 1984. *Performance Evaluation of Full-Scale Hazardous Waste Incinerators, Volume I: Executive Summary*. Cincinnati: U.S. Environmental Protection Agency, Industrial Environmental Research Lab.

Tritscher, A.M., G.S. Clark, and G.W. Lucier. 1994. Dose-response effects of dioxins: species comparison and implications for risk assessment. In: A. Schecter, *Dioxins and Health*. New York: Plenum, 227–248.

Tryphonas, H. 1995. Immunotoxicity of PCBs (Aroclors) in relation to Great Lakes. *Environmental Health Perspectives* 103 (Supplement 9):35–46.

Tukker A., J.L.B. eGroot, and P. van de Hofstadt. 1995. *PVC in Europe: Environmental Concerns, Measures and Markets.* Apeldoorn, Netherlands: TNO Policy Research Group.

Tullos, B.G. 1995. Presentation materials for: The caustic/chlorine relationship. Conference on The Changing Chlorine Marketplace: Business Science and Regulations. New Orleans, April.

UBA (German Federal Office of the Environment). 1992. *Environmental Damage by PVC: An Overview*. Berlin: Umweltbundesamt.

UKDOE (United Kingdom Department of the Environment). 1989. *Dioxins in the Environment: Pollution Paper #27*. London.

United Nations Environment Programme. 2000. *Final Report: UNEP/POPS/INC.4/5—Report of the Intergovernmental Negotiating Committee for an International Legally Binding Instrument for Implementing International Action on Certain Persistent Organic Pollutants on the Work of its Fourth Session*, Bonn, 20–25 March, Geneva.

U.S. Navy. 1986. Military Specification Sheet: Insulation sleeving, electrical, heat shrinable, polyvinyl chloride, flexible, crosslinked and noncrosslinked. MIL–I–23053/2C, May 1.

———. 1979. Military Specification Sheet: Insulation sleeving, electrical, heat shrinkable, polyvinyl chloride, flexible, crosslinked and noncrosslinked. MIL–I–23053/2C, Amendment 1, October 19.

Versar, Inc. 1996. *Formation and Sources of Dioxin-like Compounds: A Background Issue Paper for U.S. Environmental Protection Agency, National Center for Environmental Assessment*. Springfield, VA: Versar.

Vesterinen, R., and M. Flyktmann. 1996. Organic emissions from co-combustion of RDF with wood chips and milled peat in a bubbling fluidized bed boiler. *Chemosphere* 32:681–689.

Vikelsoe, J., and E. Johansen. 2000. Estimation of dioxin emissions from fires in chemicals. *Chemosphere* 40:165–175.

Vinyl Institute. 2000. Vinyl by Design: Vinyl wallcovering. Available at: [http://www.vinylbydesign.com/wall-covering/design.html].

———. 1998. *Vinyl Institute Dioxin Characterization Program Phase I Report*. Morristown, New Jersey: Vinyl Institute.

Visalli, J.R. 1987. A comparison of dioxin, furan and combustion gas data from test programs at three MSW incinerators. *Journal of the Air Pollution Control Association* 3712:1451–1463.

Wagenaar H, Langeland K, Hardman R, Sergeant Y, Brenner K, Sandra P, Rappe C, Tiernan T. Analyses of PCDDs and PCDFs in virgin suspension PVC resin. Organohalogen Compounds 27:72-74, 1996.

Wagener, K.B., C.D. Batich, and A.E.S. Green. 1993. Polymer substitutes for medical grade polyvinyl chloride. In: A.E.S. Green, ed. 1993. *Medical Waste Incineration and Pollution Prevention*. New York: Van Nostrand Reinhold, 155–169.

Wagner, J., and A. Green. 1993. Correlation of chlorinated organic compound emissions from incineration with chlorinated organic input. *Chemosphere* 26:2039–2054.

Wallace, D. 1990. *In the Mouth of the Dragon*. Yonkers: Consumers Union.

Waltermire, T. 1996. Presentation materials for: The vinyl plastics industry. Goldman, Sachs Fourth Annual Chemical Investor Forum. New York, May.

Warren, S. 1998. Toy makers say bye-bye to "plasticizers." *Wall Street Journal*, November 12.

Wienecke, J., H. Kruse, U. Huckfeldt, W. Eickhoff, and O. Wasserman. 1995. Organic compounds in the flue gas of a hazardous waste incinerator. *Chemosphere* 30:907–913.

Wieslander, G., D. Norback, K. Nordstrom, R. Walinder, and P. Venge. 1999. Nasal and ocular symptoms, tear film stability and biomarkers in nasal lavage, in relation to building-dampness and building design in hospitals. *International Archive of Occupational and Environmental Health* 72:451–461.

Wikstrom, E., G. Lofvenius, and C. Rappe. 1996. Influence of level and form of chlorine on the formation of chlorinated dioxins, dibenzofurans, and benzenes during combustion of an artificial fuel in a laboratory reactor. *Environmental Science and Technology* 305:637–1644.

Wilken, M. 1994. *Dioxin Emissions from Furnaces, Particularly from Wood Furnaces*. Berlin: Engineering Group for Technological Environmental Protection for the German Environmental Protection Agency, February.

Wirts, M., W. Lorenz, and M. Bahadir. 1998. Does co-combustion of PVC and other plastics lead to enhanced formation of PCDD/F? *Chemosphere* 37:1489–1500.

Wunderli, S., M. Zennegg, et al. 2000. Determination of polychlorinated dibenzo-p-dioxins and dibenzofurans in solid residues from wood combustion by HRGC-HRMS. *Chemosphere* 40:641–649.

Yasuhara, A., and M. Morita. 1988. Formation of chlorinated aromatic hydrocarbons by thermal decomposition of vinylidene chloride polymer. *Environmental Science and Technology* 22:646-650.

Yoneda, K., Y. Yagi, T. Ikeguchi, Y. Tamade, and K. Omori. 2000. Behaviour of dioxins and precursors in industrial waste incineration. *Organohalogen Compounds* 46:170–173.

The Healthy Building Network is guided by an advisory panel of grassroots activists, health professionals and leaders in the green building movement. For a complete listing, see www.healthybuilding.net/advisors.

The Healthy Building Network is a project of the Institute for Local Self-Reliance.

To get involved, contact the Healthy Building Network at:

www.healthybuilding.net
info@healthybuilding.net

Institute for Local Self-Reliance
2425 18th Street, NW
Washington, DC 20009-2096

phone: (202) 232-4108
fax: (202) 332-0463

ISBN 0-9724632-0-8
90000>
9 780972 463201

This report was printed using soy-based inks on a process chlorine free paper with 100% post-consumer recycled content.

UNION
BUG
FPO