

Update on the Environmental Health Impacts of
Polyvinyl Chloride (PVC) as a Building Material:
Evidence from 2000-2004

a commentary for the U.S. Green Building Council

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OVERVIEW

In December 2000, the Healthy Building Network and the Center for Maximum Potential Building Materials submitted to the U.S. Green Building Council (USGBC) a briefing paper that summarized the environmental health effects of polyvinyl chloride (PVC) building materials[1]. Since that time, the USGBC's deliberations over PVC have continued and evolved. This report, therefore, represents an update of the scientific evidence. It is intended to serve as a reader's guide to some of the most important documents, reports, and data submitted on behalf of the Healthy Building Network to the Technical and Scientific Advisory Committee (TSAC) in response to its November 2003 solicitation for further evidence.

An impressive amount of research has been generated on the health effects of PVC since 2000. Taken as a whole, these studies substantially reinforce and extend the concerns that were raised at that time. This report covers a fraction of the many dozens of reports that the Healthy Building Network is submitting to the TSAC for its consideration. A complete annotated listing of those reports is available on the Healthy Building Network website (<http://www.healthybuilding.net>).

Highlighted in the discussion that follows are--

- 1) reports linking the creation of chlorine gas to significant releases of mercury by chlor-alkali facilities;
- 2) reports demonstrating multiple cancer threats to PVC workers, including a newly documented link between PVC dust and lung cancer;

- 3) data documenting the constant contamination of communities located near PVC facilities with vinyl chloride, a known carcinogen;
- 4) reports on the endocrine-disrupting properties of PVC plasticizers, including evidence for DNA damage in human sperm at background levels;
- 5) reports linking gaseous emissions from vinyl materials in home and offices to respiratory symptoms in workers and asthmatic symptoms in children;
- 6) reports documenting the inability of all known methods of disposal (incineration, landfilling, recycling) to safely and inexpensively manage PVC waste.

TSAC's solicitation for evidence comes at a critical time in the public awareness of PVC. Three years ago, knowledge about the environmental health problems associated with vinyl was held mainly by environmental researchers, members of the medical community, and firefighters. Now, the hazards of PVC are widely understood by the members of the concerned public—sometimes for very chilling reasons. The incineration and collapse of the World Trade Center in 2001, for example, sent a dioxin-filled plume over lower Manhattan. The hazards of burning vinyl were subsequently discussed on FOX News[2], and Senator Jon Corzine introduced the Chemical Security Bill, which identified the untracked production and transport of toxic chemicals—including the industrial feedstocks for PVC—as threats to homeland security[3]: The release of the documentary film “Blue Vinyl” likewise brought the cancer deaths of PVC workers into the public eye and inspired many green building projects. One is the faith-based initiative Building in Good Faith, which has vowed to eliminate PVC from construction projects involving places of worship[4].

Two different international accords, the European Union's REACH proposal and the United Nation's Stockholm Convention, have shined a global spotlight on PVC. REACH is a harmonized policy on chemicals that is still being deliberated by the European Parliament and Commission. It would require the registration of all toxic chemicals, including the class-A carcinogen vinyl chloride, and any hormone-disrupting additives[5]. The Stockholm Convention, which goes into effect in 2005, aims to reduce and eliminate releases of persistent organic pollutants (POPs) to air, water, land, and product. For unintentionally produced POPs, such as PVC-generated dioxin, the Convention requires that "consideration should be given to alternatives." More specifically, each signatory country shall "promote the development and, where it deems appropriate, require the use of substitute or modified materials, products and processes to prevent the formation and release of the chemicals"[6].

Not surprisingly, then, many downstream companies and investors are also beginning to distance themselves from PVC. Sony, Ricoh, Hewlett-Packard, Nikon, Seiko Instruments, General Motors, and Honda have all recently pledged to eliminate completely or significantly reduce PVC in their products; Honda intends to create a recyclable, PVC-free car by 2010[7].

Socially responsible investment firms, too, have issued warnings about PVC. Here, for example, is the description of PVC provided to clients by Domini investment group:

PVCs are environmentally hazardous throughout their lifecycle (production, use, and disposal). Dioxin, a known human carcinogen, is created during the production of PVC feedstocks, as well as when PVCs are burned in waste incinerators. Among other things, dioxin has been linked to endocrine disruption, reproductive abnormalities, neurological problems, and infertility in humans and animals. In addition, large amounts of chemicals called "phthalates" are used to

manufacture PVC products. A commonly used phthalate plasticizer called diethylhexyl-phthalate (DEHP) is a probably reproductive toxicant, as well as a toxicant of the liver and kidney....

PVCs are...extensively used in building materials such as furniture and floor coverings. We will support resolutions asking companies to report on the risks, financial costs, and benefits, and environmental and health impacts of the continued use of PVCs in these types of products[8].

I. UPDATE ON DANGERS ASSOCIATED WITH PRODUCTION

A. The Mercury Connection

The first step in the PVC production process is the generation of elemental chlorine. This gas is created in chlor-alkali facilities by running an electrical current through salty brine in the presence of a catalyst. The result is caustic soda (sodium hydroxide) and chlorine. Nine chlor-alkali facilities in the United States still use mercury as their catalyst, a 50-year-old technology. Mercury-cell chlor-alkali facilities produce only ten percent of the nation's chlorine but, in so doing, contribute hugely to annual atmospheric emissions of mercury. All together, 3,000 tons of mercury are currently in use by these plants.[9].

In the last few months, mercury-cell chlor-alkali plants have made headline news because of the disconnect between the amount these plants report to the Toxics Release Inventory (TRI) as their annual mercury releases and transfers (15 tons) and the amount consumed in manufacturing (30 tons). This is a difference of 15 tons of mercury. There exists an even greater disparity between the TRI figure and amount of new mercury purchased by these plants (130 tons in 2002) This is a difference of 115 tons[10]. What happened to all this mercury? This question has been taken up by state and federal regulators as well as environmental watchdog organizations. In December 2003, the

National Resources Defense Council along with the Sierra Club sued the EPA for changes in regulations that they contend fail to address the question of where all the missing mercury is. The EPA itself admitted in the Federal Register that the fate of mercury consumed in these facilities "remains somewhat of an enigma"[11]. If, in fact, the missing mercury is being seeded into the air as fugitive emissions, as many suspect, then the contribution of chlorine manufacturing to the mercury loading of the atmosphere far exceeds that of the coal-burning power plants, which, heretofore, have been presumed to be the number one source of atmospheric mercury. (All together, coal-burning power plants in the United States release 50 tons of mercury into the air each year.)

In 2000, the Vinyl Institute asserted that 20 percent of the chlorine produced in the United States by the mercury process is used to produce vinyl[12]. If so, then the production of PVC is directly responsible for the annual of release into the atmosphere of at least six tons, and as much as 26 tons, of mercury.

What makes the PVC-mercury link even more significant is the emerging evidence for neurological damage among children at very low levels of mercury exposure.

When elemental mercury from chlorine manufacturing plants is released to the atmosphere as a vapor, it can be carried long distances before returning back to earth. Once it does, methylating bacteria quickly convert the metal into an organic form, methylmercury, which is a powerful brain poison as well as a bioaccumulating, persistent pollutant. From here, it is quickly siphoned up the food chain, reaching its highest levels in fish and seafood. The EPA now estimates that 630,000 infants are born *each year* in

the United States at risk for neurological damage from exposures to methylmercury (that is, infants who receive *in utero* doses of methylmercury equal to or greater than the EPA's established reference dose for safety)[13]. In other words, one in every six U.S. babies now comes into the world with prenatal mercury exposures known to be associated with an increased risk for developmental harm to the brain. The adverse effects of prenatal mercury exposure include deficits in memory, attention span, motor control, and the ability to learn.

Other recent studies on methylmercury reveal evidence for widespread exposure as well as serious health effects at background dosages. The Centers for Disease Control estimates that 8 percent of U.S. women have body burdens of methylmercury that exceed the EPA's recommended reference dose[14]. A study of children on the Faroe Islands who were exposed to methylmercury from their mother's seafood consumption during pregnancy showed deficits in brain stem functioning that persisted into adolescence. The researchers concluded that "the neurotoxic effects from intrauterine methylmercury exposures are irreversible"[15]. A 2002 study published by a team of researchers from Johns Hopkins University found a link between mercury body burden and risk for heart attack. The researchers concluded that the ongoing contamination of fish with mercury "diminishes the cardioprotective effect of fish intake"[16].

These and other studies have prompted the Food and Drug Administration, as well as the Environmental Protection Agency, to issue more stringent advisories for fish consumption for women and children—including, as of 2004, restrictions on eating canned tuna. Even these new, stricter dietary advisories have been challenged by a leading health, consumer, and environmental groups as insufficiently protective[17].

The European Union's Food Safety Authority also warned, in February 2004, that mercury exposure from typical levels of fish consumption places women and children at risk[18]. In the same month, the European Commission released a report about the impact of chlor-alkali plants on mercury flow in Europe and the world[19].

B. Effects on Workers

Once chlorine is generated and combined with carbon to form ethylene dichloride (more on this intermediate product below), the next major step in the PVC manufacturing process is the synthesis of vinyl chloride monomer. The ability of vinyl chloride to cause angiosarcoma of the liver is well known. New studies demonstrate risks for other occupational cancers as well as non-cancer diseases.

A 2003 Italian study found among PVC workers significantly increased mortality from all causes of death, all tumors, lung cancer, lymphomas, leukemias, and liver cirrhosis[20]. This study is important because the authors used other blue-collar workers as the internal reference group. That is, rather than compare PVC workers to members of the general public, the authors compare probably exposed workers with unexposed workers, which is a less-biased method.

A 2003 study of workers in a PVC plant in Louisville, Kentucky found strong associations with angiosarcoma and vinyl chloride exposure. This study also uncovered a cluster of brain cancers that could not be associated with vinyl chloride exposure *per se* but which was generally associated with having worked in a PVC facility[21]. This

unexplained cluster remains of interest because the Italian study also found brain cancers among its PVC workers.

A 2003 study from Taiwan reports on an interaction between vinyl chloride exposure and Hepatitis B infection (HBV). Workers without a history of chemical exposure but who were infected with HBV had a four-fold increase in angiosarcoma. Workers free of the viral infection but with vinyl chloride exposure experienced a 26-fold increase in angiosarcoma. However, the highest risk was found among workers both exposed to the virus and to vinyl chloride: the risk of these workers for liver cancer was elevated by a factor of 396. (Smoking, alcohol consumption, and medical history were all accounted for.) This study shows that vinyl chloride is a more powerful liver carcinogen than hepatitis B, which is a well-known cause of liver cancer. This study also demonstrates a synergistic interaction between vinyl chloride and HBV that resembles that seen between tobacco smoke and asbestos exposure.[22]

The next step in PVC manufacture is the polymerization of vinyl chloride monomer. This process creates a fine powder, which is handled by workers known as PVC baggers. Recent studies have documented increased rates of lung cancer and other pulmonary diseases within this group of workers.

A 2003 Italian study of 1,668 PVC workers found that baggers were exposed to high levels of respirable PVC dust. These workers, who were not exposed to vinyl chloride monomer, suffered from an increased risk of lung cancer associated with exposure to this dust. Age and smoking were controlled for[23].

Possible mechanisms for the link between PVC dust exposure and lung cancer are suggested by two animal studies that investigated lung changes in rats exposed to PVC dust. Funded by the plastics industry itself, this set of studies, also published in 2003, found evidence for a PVC-induced acute inflammatory reaction in the lung as well as more persistent alterations in the pulmonary immune profile. These changes were evident whether chemical additives coating the PVC particles had been washed off or not. The authors conclude, "Our findings suggest that immunologic mechanisms are directly or indirectly involved in the pathogenesis of lung changes after exposure to PVC dusts"[24].

C. Effects on Communities

The ability of vinyl chloride monomer to drift beyond the factory fence line remains a primary issue. However, the public health problems start even before vinyl chloride is manufactured. The material created as an intermediate between chlorine gas and vinyl monomer is called ethylene dichloride (EDC), a substance that is classified as a possible carcinogen and is notoriously capable of leaching into groundwater. In September 2003 in Botany, Australia, EDC was found during monitoring of deep groundwater near the chemicals firm Orica, which manufactured EDC for the PVC industry. Further testing revealed that three fingers of EDC are now moving towards Botany Bay[25].

In February, 2002, a PVC plant in Saugus, California was raided by multiple federal agencies under the direction of the FBI as part of an investigation of claims that the company had repeatedly released toxic chemicals and faked air emissions data as part

of an organized cover-up. Among other falsifications, the EPA discovered that the plant's air emissions had routinely exceeded quarterly limits, in spite of claims to the contrary. Ten months later, the company ceased its PVC resin-manufacturing operation[26].

In Plaquemine, Louisiana, a trailer park that is home to 300 residents was forced to close in 2003 when vinyl chloride was detected in their drinking water. Residents there report an excess of miscarriages. The poisoning of the Plaquemine aquifer launched a criminal investigation by state and federal authorities and a front page story in the *New York Times* [27].

During that same month, a newspaper investigation of air emissions from plants in Louisville, Kentucky revealed that residents were being exposed to toxic chemicals, including vinyl chloride, that greatly exceeded EPA's health-based limits [28]. Chemist Wilma Subra has studied company and public health records of air quality in the neighborhood of the Louisville plants, as well as around a range of PVC plants in Louisiana, and found consistent patterns of exposure to school children, seniors, and other area residents to vinyl chloride, dioxin, and other toxic chemicals exceeding regulated health standards[29].

Threats to communities surrounding PVC plants include those in eastern Europe. Testimony by Children of the Earth before the European Commission at a 2000 public hearing on PVC revealed a long history of grievous problems at one such plant in the Czech Republic. They ranged from crop deaths caused by chlorine gas releases to dioxin-contaminated ash from fires and explosions[30].

All together, the data from so-called "fence-line studies" indicate that release of toxic chemicals from PVC facilities and the threats that these releases pose to the surrounding communities are not the result of a few isolated accidents or a few bad actors within an otherwise well-regulated industry. Rather, they are result of routine operations of an inherently dangerous industrial process that is also, by its very nature, prone to frequent accidents and upsets. According to all available data, vinyl chloride monomer production results in a constant exposure of neighbors to VCM and/or EDC[31].

II. UPDATE ON DANGERS ASSOCIATED WITH USE

The smell of PVC is apparent to anyone who has bought a new shower curtain and hung it in a bathtub. And a 2002 study has shown that a single vinyl shower curtain can, in fact, raise indoor air toxics concentrations for longer than a month[32].

In addition to the volatilization of organic chemicals in PVC products, threats to human health associated with the use of polyvinyl chloride building materials can originate from degradation of the vinyl material itself. There is also growing concern about the endocrine-disrupting potential of the phthalate plasticizers used to make PVC pliable.

A. Threats Associated with Degradation

A 2003 Finnish study investigated a high incidence of adult-onset asthma among employees working in an office building. Rates of asthma in this workplace were nine times higher than that among Finnish workers similarly employed. Researchers

discovered that degraded vinyl floor covering had released volatile organic chemicals such as 3-ethyl-1-hexanol and 1-butanol, into indoor air as well as underlying concrete slabs. When the floor covering was removed, VOC levels in indoor air decreased as did the prevalence of workers' symptoms. Indeed, after the removal of vinyl flooring, several employees found they no longer needed asthma medication at all[33].

Other studies from around the world corroborate these findings. Damp PVC flooring and carpeting have been shown to degrade indoor air quality through release of volatile organic compounds[34]. A study of more than 10,000 Swedish children found that the combination of floor moisture and PVC flooring significantly increased asthmatic symptoms[35].

B. Threats Associated with Phthalate Emissions from PVC Building Materials

There are many kinds of phthalates. Some are used primarily in cosmetics and fragrances. Others are used for printing inks, pesticides, or pharmaceutical products. Di(2-ethylhexyl)phthalate (DEHP) is the main plasticizer for PVC. DEHP is an animal carcinogen, an endocrine disrupter, and a developmental toxicant, with the male reproductive system considered the system most sensitive to the effects of DEHP.

Research on health effects of phthalates has exploded during the last four years. This report references only those phthalates immediately relevant to PVC building materials. Such studies fall into one of three categories: studies documenting the release of phthalates into the environment from vinyl building materials; studies documenting human exposures; and studies documenting human health effects from such exposures.

1. Release of Phthalates into Indoor Air

A 2004 study from Japan measured phthalate levels in samples of indoor air from 27 different houses in Tokyo. Levels were surprisingly high in all homes, reaching their highest levels in newly constructed buildings. The authors conclude, "This research indicated that exposure to phthalate esters through inhalation or air from the indoor environment is as important as dietary intake of phthalate esters, and can contribute to daily intake to a much greater extent than has been assumed hitherto"[36].

A 2004 study from Denmark measured phthalate emissions from different materials. The highest levels of phthalates were emitted from wax-covered polyolefine flooring. However, this phthalate was dibutylphthalate (DBP), which the National Toxicology Center identifies as a less toxic substance than DEHP. (DBP is "of minimal concern" for potential effects on human development and "of negligible concern" for effects on the adult reproductive system. By contrast, the National Toxicology Program has expressed "serious concern" about the possible harm of DEHP exposure to the reproductive tract of developing male fetuses. More on this below.) The polyolefine floor released no DEHP. By contrast, PVC materials did emit DEHP as well as DBP[37].

These results support those of a pilot study published in 2002 which identified vinyl flooring and vinyl wall décor as the source of DEHP in dust particles in a child's room[38].

2. Human Exposure to Phthalates

In 2003, the Centers for Disease Control released the results of its extensive body burden survey, which attempts to measure concentrations of common chemical

contaminants in a representative sample of the U.S. population. The CDC survey found nearly ubiquitous exposure to phthalates among all age, ethnic and gender categories. Levels of DEHP were highest in children[39]. This study corroborated a smaller CDC study, published in 2000, which also detected phthalate metabolites in the urine of nearly every individual in a U.S. reference population[40].

Two recent German studies also report on phthalate levels in human urine. The first study, published in 2003, uncovered strikingly high levels of DEHP in the urine of residents in the southern city of Erlangen. All subjects tested had some level of DEHP metabolite in their urine. More than ten percent had values greater than the “tolerable daily intake” limit (TDI) used by European Union, and nearly one-third of subjects exceeded the U.S. EPA’s reference dose (RfD). The authors concluded, “We are not aware of any other environmental contaminant for which the TDI and RfD are exceeded to such an extent within the general population”[41].

The second German study, carried out by the same team of researchers, was published in 2004. In this study, investigators compare levels of DEHP metabolites in the urine of nursery school children, their parents, and their teachers. They found that children’s exposures to DEHP were roughly double that of adults. “Routes of the ubiquitous exposure to DEHP remain indistinct[42].”

3. Human Health Effects from Phthalate Exposure

A number of pre-2000 studies highlighted the connection between phthalate environments and asthma or other bronchial obstruction problems in children. Much of

the work on phthalates in the last four years has focused on understanding the connection between phthalates and developmental concerns.

A review published in 2003 by the environmental committee of the American Academy of Pediatrics looked at prenatal risks created by DEHP exposure during pregnancy and concluded there were not enough human studies to conclude that phthalates are safe[43].

In July 2000, the National Toxicology Program's Center for the Evaluation of Risks to Human Reproduction concluded its evaluation of seven phthalate esters at an expert panel meeting in Virginia. All phthalates were ranked as to their level of concern. All but DEHP received marks of "low, minimal, or negligible." DEHP was not only singled out as a substance of "serious concern" for the possibility of its adverse effects to the developing reproductive system of boy babies, but the panel also expressed concern current estimated adult exposures to DEHP might be sufficient to adversely affect male fetuses in pregnant women. They also expressed concern about apparently higher exposure levels in infants and children[44].

In the same year, a team of researchers in Puerto Rico documented a correlation between high DEHP levels in young girls and premature breast development. Puerto Rican girls with dramatically early breast development (with an average age of 31 months) had seven time more DEHP in their blood than a matched group of girls without early breast development. "This study suggests a possible association between plasticizers with known estrogenic and antiandrogenic activity and the cause of premature breast development in a human female population"[45].

Associations have also been found between DEHP exposure and DNA damage in the sperm of adult males. As part of a 2003 study in Boston, researchers collected urine and semen samples from 168 subfertile men with no known occupational exposure to DEHP reported. Increasing levels of DEHP metabolite in urine were associated with decreasing levels of genetic integrity in sperm cells. "In conclusion, this study represents the first human data to demonstrate that urinary MEP [a DEHP metabolite], at environmental levels, is associated with increased DNA damage in sperm[46]."

III. UPDATE ON DANGERS ASSOCIATED WITH DISPOSAL

Recent studies on the disposal problems presented by PVC confirm an earlier established truth: there is no safe way to get rid of it, and no good way to recycle it. At the end of its life span, PVC dies one of four deaths: it is buried in a landfill; it is burned in an incinerator (and then its ashes are buried in a landfill); it is "downcycled;" or it burns up in a fire that is either set accidentally or, as in the case of arson or terrorism, on purpose. 

Perhaps the most telling commentary on PVC's disposal woes is found on the website of the tiny Canadian town of East Gwinlimbury, whose official mascot is a beaver swimming in a lake. East Gwinlimbury boasts a very enthusiastic Environmental Services Advisory Committee. Among other tasks the committee runs an annual Non-Blue Box Plastics Collection Day to recover plastic trash that is otherwise not picked up at curbside. Gladly accepted are Styrofoam, packing materials, and ice cream tubs.

However, “we cannot accept PVC since these material (grey sewer pipe, Venetian blinds etc.) cannot be safely recycled.”

The difficulty of recycling PVC waste was taken up in a 2000 study for the European Commission. Recycling of PVC, according to this report, has technical limits, including the fact that recycled materials are not equivalent to virgin PVC. Moreover, many vinyl products contain lead and cadmium as stabilizers, and these heavy metals can become dispersed during the recycling process and contaminate the recyclates. Examining future trends in PVC disposal in Europe, the study concludes that the costs of recycling PVC will remain high, necessitating financial assistance. “As a consequence, the mechanical recycling [of PVC] must be subsidized, i.e. the additional costs compared to waste disposal must be borne by society, industry, or waste owners.” The authors estimate these costs at between 90-290 million Euro per year. In spite of these subsidies, however, “mechanical recycling is not qualified to contribute significantly to the management of PVC post-consumer wastes in the next decades, reaching at most 18 percent of total PVC waste arising. This means that the major part of future PVC waste volumes has to be recovered or disposed or in other ways...”[47].

One other way is landfilling. This is also an imperfect solution. Another 2000 report for the European Commission reports on the behavior of PVC in landfills. This study estimates that 28 percent of the total amount of lead in municipal waste landfills comes from PVC products; however, the migration of lead and other heavy metals out of landfilled PVC could not be quantified. The report describes phthalate emissions in landfill gases as well as leachate. It concludes—

“There is no evidence that the release of additives will come to a standstill. Thus, it is expected that this process will last for a very long time....The technical guarantee for landfill bottom liners and pipes for leachate collection is restricted to 80 years. Emissions resulting from the presence of PVC in landfills are likely to last longer than the guarantee of the technical barrier”[48].

Incineration is the other alternative. Another 2000 report for the European Commission examines this option. It notes that the high chlorine content of PVC places a high demand on the use of alkaline reagents in the air pollution control systems of incinerators. The additional costs of these reagents amount to 165 Euro per ton of rigid PVC. But since these costs are spread across all materials sent to the incinerator, the additional expense of burning PVC is currently paid by the incineration of other materials: “This effectively subsidizes PVC waste incineration.” The advantage of incineration, says this report, is that it destroys phthalates that would otherwise leach out of a landfill. On the other hand, incineration frees up lead and cadmium so that the ash that is subsequently buried from PVC incineration is more likely to contain mobile heavy metals than the original PVC matrix. The authors conclude—

“...it is likely that there will be benefits to be gained from diverting PVC away from incineration, particularly toward recycling, though there are clearly very finite limits to what can be recycled. There are also economic limits for separation of PVC mingled with other types of waste, Whatever the future for

PVC, this problem will remain with us for many years as a consequence of the large stock of long-lived PVC products currently in use..."[49].

Blazes other than those that occur inside the ovens of incinerators also destroy PVC building materials. Among the dangers when PVC burns in open fires are dioxin generation, the formation of hydrochloric acid mist, and the generation of thick, choking smoke. In September 2002, a wildfire in California's Santa Clarita Valley claimed 25 acres of brush and dry creek bed full of PVC piping. The resulting black smoke closed roads and shut down train service[50]. A 1997 fire in a plastics plant in Hamilton, Ontario consumed 400 tons of PVC, triggered the evacuation of 700 residents, and generated so much hydrochloric acid that the metal on nearby fire trucks melted[51]. More than 200 firefighters who fought that blaze later filed claims that it destroyed their health[52]. In March 2004, one of these firefighters, Bob Shaw, died of cancer of the esophagus, and both his union as well as his personal physicians attributed his death to the 1997 PVC fire. (Nevertheless, Shaw's claim for compensation was rejected by the Workplace Safety and Insurance Board, on the grounds that esophageal cancer has not been definitively linked to the occupation of firefighting.)[53].

The dioxin created by burning vinyl in the World Trade Center inferno was a topic taken up in a February 2004 report on the health and environmental consequences of that disaster.[54] The following month, the World Health Organization called for more protection for the developing brains of children against a variety of environmental toxicants, including dioxin. Its comprehensive study on the environmental threats to

children's health will be presented this June at a Budapest conference entitled "The Future for Our Children"[55].

SUMMARY

All together, data from the past four years indicates that PVC poses serious threats to environmental health at every stage of its existence. Its production contributes to the ongoing contamination of fish and seafood with methylmercury. Its manufacture and assembly is linked to lung cancer, as well as liver cancer, in workers. PVC plants routinely poison neighboring communities. The use of PVC as a building material contributes to the degradation of indoor air and is linked to respiratory symptoms in children and office workers. The plasticizers with which it is treated pose clear threats, at background level, to fetal development of the male reproductive tract and may also damage sperm cells in adult males. At the end of its life, PVC waste creates intractable disposal problems because it is expensive and unsafe to burn, it releases hazardous chemicals into groundwater and air when buried, and is not cheaply or easily recycled.

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