Available online at www.sciencedirect.com
SCIENCE DIRECT
Environmental Impact Assessment Review
24 (2004) 695–710
Environmental Impact Assessment Review
www.elsevier.com/locate/eiar

Human exposure, health hazards, and environmental regulations

Anne Steinemann*

Georgia Institute of Technology, Atlanta, GA 30332-0155, USA

Abstract

United States environmental regulations, intended to protect human health, generally fail to address major sources of pollutants that endanger human health. These sources are surprisingly close to us and within our control, such as consumer products and building materials that we use within our homes, workplaces, schools, and other indoor environments. Even though these indoor sources account for nearly 90% of our pollutant exposure, they are virtually unregulated by existing laws. Even pollutant levels found in typical homes, if found outdoors, would often violate federal environmental standards. This article examines the importance of human exposure as a way to understand and reduce effects of pollutants on human health. Results from exposure studies challenge traditional thinking about pollutant hazards, and reveal deficiencies in our patchwork of laws. And results from epidemiological studies, showing increases in exposure-related diseases, underscore the need for new protections. Because we cannot rely solely on regulations to protect us, and because health effects from exposures can develop insidiously, greater efforts are needed to reduce and prevent significant exposures before they occur. Recommendations include the development and use of safer alternatives to common products, public education on ways to reduce exposure, systematic monitoring of human exposure to pollutants, and a precautionary approach in decision-making.

Keywords: Exposure; Pollution; Health; Environment; Regulations; Laws; Indoor air

* Tel.: +1-404-894-6491.
E-mail address: anne.steinemann@arch.gatech.edu (A. Steinemann).

0195-9255/$ – see front matter
doi:10.1016/j.eiar.2004.06.002

1. Pollutant exposure studies: surprising results

Numerous studies on personal exposure to pollutants have revealed some startling results (Wallace, 1987, 1991, 1993; CDC, 2001, 2003; EWG, 2003).

We are regularly exposed to many toxic chemicals and carry them in our bodies, as evidenced by samples of human blood, breath, hair, tissue, and body fluids.

Most of our exposures to these chemicals are not from sources traditionally regulated, such as remote waste sites and factories. Rather, the primary sources are close to us: within our indoor environments, and the personal activities, products, and materials inside those environments.

Of more than several hundred pollutants regulated by federal laws, all but a few are higher indoors than outdoors, due to indoor sources.

The sources of these pollutants are largely unregulated—meaning that our environmental regulations, designed to protect and promote human health, are missing major sources of health risks.

The public is generally unaware of these types of everyday exposures, their health consequences, and the relatively simple and cost-effective actions that could reduce health risks.

2. Exposure science: measuring pollutants that affect humans

The science of human exposure[1] emphasizes an important but often overlooked fact: The pollutants that affect human health are those that come in contact with humans (Ott, 1985). In this way, exposure science differs from traditional approaches to environmental management. Instead of identifying a pollutant source and then trying to trace emissions through the environment to see who or what might be affected, exposure science starts with the receptor of those pollutants—humans. It identifies and measures the pollutants that have reached humans, and then traces the pollutants back to their sources. If a goal of environmental regulations is to protect and promote human health, then we need to address not only the pollutant sources but also the receptors.

Advances in the science of exposure assessment have enabled researchers to measure, with great accuracy, the types, concentrations, durations, and locations of human exposure to environmental pollutants. These advances are technological, such as the use of highly sensitive analytic instruments and portable exposure monitors, and methodological, such as the use of sophisticated probability sampling designs in large-scale field studies (Ott and Roberts, 1998).

[1] An "exposure" is the contact between an agent and a target (Zartarian et al., 1997). In this context, the "agent" is a pollutant, and the "target" is a human.

In the 1980s and 1990s, the U.S. Environmental Protection Agency (EPA) and other researchers conducted the landmark TEAM (total exposure assessment methodology) studies that measured personal exposures to pollutants. These studies monitored more than 3000 participants, in 18 US urban and suburban cities and one Canadian province, for exposure to volatile organic compounds (VOCs) (Wallace, 1987, 1993; Wallace et al., 1991), pesticides (Immerman and Schaum, 1990; Whitmore et al., 1994), carbon monoxide (Akland et al., 1985), particles (Özkaynak et al., 1996a,b; Pellizzari et al., 1993), phthalates (plasticizers) and polycyclic aromatic compounds (PAHs) (Sheldon et al., 1993), among other pollutants. The participants carried around personal exposure monitors that indicated what, how much, and where pollutants were affecting them. In addition, the VOC and CO studies measured breath levels of 2000 participants to detect the chemicals in their bodies (Wallace et al., 1986, 1988).

These studies produced a compelling finding: Most of our exposure to pollutants occurs indoors, and from products that we choose to use. This result contradicted conventional thinking, and conventional regulation, that focus on outdoor sources of emissions rather than indoor and personal sources of exposures.

What and where are these pollutant sources? Studies identified the following:

consumer products, such as air fresheners, deodorizers, cleansers, disinfectants, personal care products, laundry supplies, moth repellants, cosmetics, dry-cleaned clothes, solvents, and pesticides;
building materials and furnishings, such as paints, varnishes, adhesives, solvents, carpets, vinyl flooring, pressed wood products, and combustion appliances;
individual activities, such as bathing and washing in chlorinated water, burning firewood and candles, refueling an automobile tank, and cigarette smoking.

Specific results include the following. VOC exposures indoors are typically 5–50 times higher than outdoors, even in cities with relatively high levels of outdoor pollution and heavy industry. New buildings often contain VOC levels that are hundreds of times higher than outdoor levels (Sheldon et al., 1988; Wallace, 2001). Common VOCs cause both acute and chronic health effects, ranging from sensory irritation and headaches, to neurological damage and cancer (Pierson et al., 1991; Otto et al., 1990; NIH, 2003).

Fragranced and scented products represent significant sources of human exposure to toxic VOCs (Wallace, 2001); specifically, synthetic fragrances that are found in numerous consumer products[2], such as air fresheners, deodorizers,

[2] Paradoxically, and perhaps misleadingly, some fragranced products are labeled ''fragrance-free'' or ''unscented'' because of the addition of a ''masking fragrance,'' another synthetic chemical, to the already fragranced product—without necessarily reducing the toxicity of the product.

laundry and dishwashing detergents, chlorine bleach, dryer sheets, and personal care products such as shampoo, soap, lotions, hairspray, after-shave, nail polish and remover (EPA, 1989b; Sack et al., 1992; Cooper et al., 1992). More than 95% of chemicals used in fragrances are synthetic compounds derived from petroleum, including benzene derivatives, aldehydes and many other known toxics and sensitizers capable of causing cancer, birth defects, central nervous system disorders and allergic reactions (USHR, 1986; NIH, 2003).

Pesticide levels can be 5–10 or more times higher indoors than outdoors, even though some pesticides are used only outdoors (Whitmore et al., 1994). Reasons are that pesticides used outdoors, such as termiticides, can seep indoors or be tracked inside by shoes; plus, pesticides can persist much longer indoors where they are protected from degradation. In addition, past applications of pesticides can persist in the environment and in human bodies for decades after the initial application. For instance, the pesticide dichlorodiphenyltrichloroethane (DDT), which was banned in 1972, was nonetheless found in carpets of 25% of 362 Midwestern homes (Camann et al., 2000). In addition, polycyclic aromatic compound concentrations found in carpet dust of 89% of these homes were more than seven times the Superfund preliminary remediation goal for outdoor soil. This implies that an average urban infant, consuming an average of 100 mg of dust containing 100 ng of benzo(*a*)pyrene a day, could be exposed to an amount equivalent to smoking 2.8 cigarettes a day (Ott and Roberts, 1998).

Partly influenced by the TEAM Study findings, several national and regional task forces attempted to compare a wide selection of environmental risks. The consensus was that the risk from indoor air pollution and consumer products was far greater than most of the other risk factors surveyed, including hazardous waste sites and outdoor air pollution (EPA, 1987, 1988a, 1989a; Omenn, 1997).

The U.S. Centers for Disease Control and Prevention (CDC) recently conducted two nationwide assessments of human exposure to toxic chemicals in air, water, food, soil, dust, and other media (such as consumer products). The First National Report on Human Exposure to Environmental Chemicals (CDC, 2001) presented exposure data for 27 chemicals (lead, mercury, cadmium, and other metals; metabolites of organophosphate pesticides; cotinine; and phthalates). The Second National Report (CDC, 2003) included these 27 chemicals and added 89 more, including polycyclic aromatic hydrocarbons; dioxins, furans, and coplanar polychlorinated biphenyls (PCBs); non-coplanar PCBs; phytoestrogens; organophosphate, organochlorine, and carbamate pesticides; herbicides; pest repellants; and disinfectants.

Results showed some success in dealing with prior problems, such as exposures to lead and environmental tobacco smoke. But they also revealed new problems. One is exposure to phthalates, which virtually all Americans now carry in their bodies. Phthalates are found in numerous consumer products such as soft plastics, pesticides, pharmaceuticals, lotion, children's toys, adhesives, detergents, lubricants, food packaging, soap, shampoo, hairspray, nail polish, and products made from polyvinyl chloride (PVC). Levels of phthalates were highest in children and women of reproductive age, posing risks of developmental and reproductive abnormalities, such as infertility, precocious thelarche (onset of breast development before age eight in girls), sperm damage, and birth defects (Raloff, 2000; NTP, 2000; CERHR, 2000). Another is the prevalence of pesticides and the levels of pesticides, especially in children. Metabolites of the pesticide chlorpyrifos were nearly twice as high in children (age 6–11) than as in adults. Metabolites of the organochlorine pesticide DDT were clearly measurable in young adults ages 12–19, even though they were born after the U.S. ban (CDC, 2002). [For a report on known and potential health effects of chemicals in these CDC studies, see PSR (2003)].

A recent exposure study, led by Mount Sinai School of Medicine in New York, in collaboration with the Environmental Working Group and Commonweal (EWG, 2003), evaluated nine adult subjects; none worked with chemicals, and all were regarded as leading healthy lives. The study found 167 industrial compounds (average of 91 compounds) in the blood and urine of these subjects, including breakdown products from organochlorine and organophosphate pesticides, polychlorinated biphenyls, dioxins, furans, and phthalates. These chemicals are associated with cancer, brain damage, hormonal disruption, birth defects, developmental abnormalities, reproductive system defects, and immune system damage (EWG, 2003; NIH, 2003). Among the chemicals tested, the most prevalent were 77 semivolatile and volatile organic chemicals, present in common consumer products, solvents, cleaners, and paints. None of these 77 compounds was tested in the CDC studies, and each of them was found in at least one subject in this study.

3. Body burden of chemicals and burden of proof

What are the health effects of all these pollutants in our bodies? The magnitudes and multiplicity of health risks may be impossible to assess fully, because we are dealing with mixtures of chemicals, non-monotonic dose–response relationships, cumulative effects, individual susceptibilities, lag time between exposures and effects, and hundreds of documented and potential morbidity and mortality effects (NIH, 2003; PSR, 2003). The complexity of analysis has led to regulatory paralysis, where chemicals are often assumed safe until proven hazardous, placing a perhaps insurmountable burden of proof on the public. Nonetheless, we have another body of evidence.

Rates of diseases with potential links to chemical exposures have been increasing nationwide. Asthma in children under age five has increased by 160% (1980–1994) (CDC, 1998). Autism has increased by 1000% since the mid-1980s (Chakrabati and Fombonne, 2001; Byrd, 2002). Hypospadias, a congenital misplacement of the urinary opening in the penis, has increased by 100% (1968–1993) and now affects one of 125 male babies born (Paulozzi et al., 1997; Baskin et al., 2001). Cancer in children has increased by 26% (1975–1999), with sharp increases in acute lymphocytic leukemia (62%), and brain and nervous system cancers (50%) (NCI, 2002a). Testicular cancer in young men has increased by 85% (1973–1999), and is now the most common cancer in men ages 15–35 (NCI, 2002b, 2000c). If trends continue, breast cancer would affect 25% of the granddaughters of today's young women (NCI, 1997). Further, according to the American Cancer Society, only 5–10% of all cancers can be attributed to inherited factors (ACS, 2001); the rest occur from environmental exposures and other damage throughout our own lifetimes.

Multiple and complex links between pollutant exposures and health effects may have obscured perceptions of risk. Exposures do not always manifest immediate and dramatic health effects; rather, they can cause subtle, gradual, and often irreversible health damage. And even when they do cause immediate effects, there is the troubling tendency to misdiagnose or misattribute common symptoms caused by exposures. For instance, exposure to pesticides can cause acute symptoms that mimic the flu, such as fevers, headaches, nausea, and joint pain, and simultaneously cause chronic damage to the endocrine, neurological, and immune systems (EPA, 2003b; NIH, 2003; Colborn et al., 1993).

Exposures also defy traditional dose–response relationships. Low-level chemical exposures can produce adverse health effects, even below regulatory thresholds and "no effects" levels (ATSDR, 2003; NAS, 2000; Ashford and Miller, 1998). For instance, chlorinated tap water byproducts, trihalomethanes, were linked to increased miscarriages at 75 parts per billion (ppb), even though the maximum contaminant level (MCL) was set at the time at 100 ppb (Waller et al., 1998). The herbicide atrazine is linked to demasculinization of frogs at levels as low as 0.1 ppb, even though the MCL is set at 3 ppb (Hayes et al., 2002).

Further, low-level exposures can be more harmful than high-level exposures of the same pollutant (Schmidt, 2001). Many chemicals, such as endocrine disruptors, exhibit non-monotonic dose–response relationships, meaning that the response (such as an adverse health effect from a chemical exposure) can increase as dose is reduced. One such chemical is bisphenol A, used in products such as plastic water bottles and baby bottles. In a series of studies, low-dose exposure to bisphenol-A caused significant enlargement of the adult prostate weight of mice exposed in the womb, but high-dose exposure produced less or no enlargement (vom Saal et al., 1997; Gupta, 2000).

Thus, we are regularly exposed to hundreds of industrial pollutants, from everyday products and places, that persist in our bodies and in the environment, and that are linked to numerous diseases and health effects. Yet the major sources of these pollutant exposures are not widely recognized, nor covered by environmental laws.

4. The missing coverage in the quilt of laws

Currently, no federal law or agency specifically protects indoor air environments, which is where we spend more than 90% of our time (Klepeis et al., 2001), and which accounts for most of our pollutant exposures. Instead, federal laws concentrate on outdoor pollution, usually media-specific or pollutant-specific. Although the laws address some pieces of indoor air, the responsibilities for those pieces are scattered among more than 20 federal agencies.

A content analysis of 22 major US environmental laws revealed that none mentioned "indoor air" (Steinemann, 2004).[3] Further, no regulation or policy has provided the umbrella coverage needed to address indoor air or, more generally, human exposures to pollutants, which are currently greatest in indoor air environments.[4] Nonetheless, several federal laws have some nexus with indoor air, and could provide the authority, if exercised.

The Clean Air Act of 1970 (CAA)[5] could provide the U.S. EPA the authority to address indoor air quality through the regulation of "ambient air."[6] Yet the original CAA does not define ambient air,[7] and the EPA has limited its interpretation of ambient air to the regulation of "outdoor air."[8] Because of this limited interpretation, the EPA does not currently exercise authority over indoor air pollution under the CAA. The EPA does, however, indirectly address indoor air by the regulation of outdoor air, because outdoor air infiltrates indoors. And the EPA has used its authority under the National Emission Standards for

[3] Although no law specifically mentioned "indoor air," two laws, the Superfund Amendments and Reauthorization Act of 1986 (SARA) and the Safe Drinking Water Act of 1974 (SDWA), referred to indoor air in the context of radon. For SARA context, see Pub. L. 99-499, title IV, Oct. 17, 1986, 100 Stat. 1758, and for SDWA context, see 42 U.S.C. §§ 300g-1. SARA authorized EPA for the first time to carry out research on indoor air pollution, but explicitly forbade regulation of indoor air. A policy office in EPA has done useful work in identifying indoor air pollutant sources (The Inside Story, EPA, 1988c; see also EPA, 1988b).

[4] Although no federal or state requirements address, in a comprehensive way, pollutants within indoor air environments, some regulations address individual pollutants, such as asbestos (15 U.S.C. §§2641–2656, 2000), lead (15 U.S.C. §§2681–2692, 2000), and radon (15 U.S.C. §§2661–2171, 2000).

[5] 42 U.S.C. §§7401–7671(q) (2000).

[6] 42 U.S.C. § 7409(a)(1) (2000).

[7] 42 U.S.C. § 7409 (2000). The foregoing Section of the Clean Air Act provides the EPA Administrator with authority to set national primary and secondary ambient air quality standards, but does not define ambient air.

[8] 40 C.F.R. § 50.1(e) (1991). By regulation, ambient air was defined as "that portion of the atmosphere, external to buildings, to which the general public has access."

Hazardous Air Pollutants (NESHAPS)[9] to ban indoor activities that affect emissions into the atmosphere (such as the spraying of asbestos insulation).[10]

In 1998, standards were passed (pursuant to the CAA) to regulate consumer products if they contribute to at least 80% of the VOC emissions outdoors in areas that violate the National Ambient Air Quality Standards (NAAQS) for ozone.[11] But these standards exempt some of the most significant sources of VOC exposures indoors, such as air fresheners, insecticides, adhesives, and moth-proofing products. Curiously, air fresheners are exempt if they contain more (rather than less) toxic constituents—if they contain at least 98% paradichlorobenzene or at least 98% naphthalene, or if their VOC constituents are 100% fragrance materials.[12]

The Toxic Substances Control Act (TSCA)[13] provides the EPA broad authority to regulate chemicals that present an "unreasonable risk of injury to health or the environment."[14] Yet "unreasonable risk" is not defined in TSCA,[15] and it has been difficult for the EPA to develop the administrative record to meet such a standard, which is a prerequisite to regulation. The EPA can request data from industry only when it can provide evidence that their substance may present an unreasonable risk of injury,[16] or can lead to significant or substantial human exposure,[17] which the EPA generally cannot prove without such additional data from industry. Further, the EPA must treat as confidential much of the industry data submitted under TSCA,[18] further hindering efforts to protect the public. Thus, until scientists have accumulated a body of evidence demonstrating potential harm, which often takes decades, a potentially hazardous chemical can remain on the market (GAO, 1994; EWG, 2003).

[9] See 42 U.S.C. § 7412 (2000). Prior to the 1990 Amendments to the Clean Air Act, EPA listed only eight hazardous air pollutants and established standards for only seven. The 1990 Amendments directed EPA to establish technology-based standards for 189 hazardous substances based on the use of "maximum achievable control technology" (MACT), which may not be less stringent than the average emission limitation achieved by the best performing 12% of existing sources in a similar source category or subcategory. Note that the term MACT does not appear in the CAA Amendments, even though EPA continues to refer to the standards as MACT.

[10] 40 C.F.R.§ 61.146 (2003).

[11] Clean Air Act § 183(e), 42 U.S.C. §7511b(e): National Volatile Organic Compound Emission Standards for Consumer Products, Fed. Reg. 48819–48847 (1998), 40 C.F.R. §§ 59.201–59.214 (2003).

[12] 40 C.F.R. §§ 59.201(c)(1)–(7) (2003).

[13] 15 U.S.C. §§2601–2692 (2000).

[14] 15 U.S.C. §2605(a) (2000).

[15] The legislative history, however, indicates that unreasonable risk involves a "harm versus benefit analysis"—the balancing of the probability that harm will occur and the magnitude and severity of that harm against the effect of a proposed regulatory action on the availability to society of the expected benefits of the chemical substance. See http://www.epa.gov/oppt/newchems/unrerisk.htm.

[16] 15 U.S.C. § 2603(a)(1)(A)(i) (2002).

[17] 15 U.S.C. § 2603(a)(1)(B)(i)(II) (2002).

[18] 15 U.S.C. § 2613(c) (2002).

The Consumer Product Safety Commission, through the Consumer Product Safety Act (CPSA)[19], is directed to protect the public from "unreasonable risks of injury associated with consumer products,"[20] and thus could regulate consumer products that contribute to indoor air pollution and exposures.[21] Yet regulation under the Act is constrained because it relies on voluntary safety standards rather than the promulgation of standards for protection.[22] Regulation is also constrained by a cost-benefit analysis for each attempt at standard-setting by the Commission,[23] and the restrictive definition of a "consumer product" that excludes several primary sources of exposure, such as pesticides and cosmetics.[24]

Moreover, Federal laws do not require manufacturers to disclose all of the ingredients in their products, such as "inert" ingredients in pesticides, and chemicals in mixtures classified as "trade secrets." This exclusion is surprising, considering that undisclosed ingredients often account for more than 95% of the product, and can be even more toxic than the active ingredients (EPA, 2003a,b; EPA, 1991). For example, a study of 85 consumer pesticide products found that 72% contained over 95% inert ingredients, and more than 200 of these inerts were classified as hazardous pollutants in other federal environmental statutes (NY, 1996). As another example, air "fresheners" containing para-dichlorobenzene are not required to list the ingredient, even though it is a registered pesticide and a known rat and mouse carcinogen. Also surprising, a manufacturer of a fragranced product need only list "fragrance" on the label, not the actual chemicals, even though more than 95% of chemicals used in fragrances are known toxics, sensitizers, and carcinogens (USHR, 1986; Fisher, 1998).

Perhaps the most sweeping federal environmental law, the National Environmental Policy Act (NEPA),[25] requires an environmental impact statement (EIS) for federal actions[26] "significantly affecting the quality of the human environment."[27] Yet in the implementation of NEPA, impact assessments have focused on impacts to the environment, rather than impacts on humans. A

[19] 15 U.S.C. §§2051–2084 (2000).

[20] 15 U.S.C. § 2051(a)(3) (2000).

[21] 15 U.S.C. § 2056(a) (2000).

[22] 15 U.S.C § 2056(b) (2000).

[23] 15 U.S.C § 2058(f) (2000).

[24] 15 U.S.C § 2056–57 (2000). "Consumer products" are defined as articles "for sale to a consumer for us in or around a permanent or temporary household or residence, a school, in recreation or otherwise." Certain categories of products, including tobacco, pesticides, motor vehicles, drugs, cosmetics, and other products subject to FDA jurisdiction, are exempt from regulation under the CPSA. § 2052 (a)(1) (2000).

[25] 42 U.S.C. §§4321–4347 (2000).

[26] An activity is "federal" if it requires a permit, a regulatory decision, or funding from a federal agency (40 C.F.R.1508.18b4).

[27] 42 U.S.C. §4332(C) 2000).

nationwide and multi-agency study of EISs (Steinemann, 2000) found that the analysis of human health effects has been sparse, relegated to another environmental statute, or omitted entirely. And these EISs were for proposed actions with potentially significant human health effects, such as pesticide spraying and highway construction.

The Occupational Safety and Health Act (OSH Act),[28] administered by the Occupational Safety and Health Administration (OSHA), regulates occupational environments, but does not protect all employees. For instance, the OSH Act does not cover federal agency employees[29] nor state and municipal government employees unless a state has a plan approved by the OSHA.[30] Even approved state plans are permitted to exclude private sector employees.[31] Efforts to establish exposure limits to toxic substances have generally failed because it is difficult for OSHA to develop the administrative record to demonstrate a "significant risk of material health impairment."[32] Also, under the OSH Act, violations must result in an employee's death in order for the employer to be subject to criminal sanctions.[33] OSHA has tended to focus on single hazards within industrial workplaces (such as large machinery), rather than multiple and often invisible hazards within typical office buildings (such as formaldehyde off-gassing from furnishings). And perhaps the largest regulatory gap, the OSH Act provides no coverage for homes and other non-industrial environments, where many people work.

More generally, environmental laws tend to focus on emissions, rather than human exposures—even though exposures are how pollutants actually contact the human body and affect health. Our laws have successfully reduced outdoor emissions,[34] and those efforts should be continued. But our regulatory lens needs to refocus on total human exposure, from all media. In this approach, units of human exposure could replace source emissions as the regulatory "currency" (Wallace, 1991; Smith, 1988).

[28] 29 U.S.C. § 651 et seq. (1970).

[29] 29 U.S.C. § 652(5). By Presidential Executive Order, federal agencies must maintain an effective safety and health program that meets the same standard as private employers. But federal agencies cannot be fined for violating health and safety standards, except for the U.S. Postal Service, which now falls directly under OSHA's jurisdiction and is treated as a private employer. http://www.osha.gov/as/opa/worker/.

[30] See 29 U.S.C.§652(5) (defining "employer") and § 667 (2000) (describing state plan approval process). States and territories with plans are AK, AZ, CA, CT*, HI, IN, IA, KY, MD, MI, NJ*, NV, NM, NY*, NC, OR, PR, SC, TN, UT, VT, VA, VI, WA, WY and the Virgin Islands*. States and territories marked with an asterisk (*) have OSHA plans approved for state and government employees only.

[31] 29 C.F.R. 1956.

[32] Industrial Union Dept., AFC-CIO v. American Petroleum Inst., 448 U.S. 607 (1980) (plurality opinion) at 614–615.

[33] 29 U.S.C. § 666(e) (2000).

[34] Reductions in principal outdoor air pollutants, over 20 years (1981–2000), include NO2 by 14%, ozone one hour average by 21% and eight hour average by 12%, CO by 61%, SO2 by 50%, lead by 93%, and PM10 by 19% (1991–2000), (EPA, 2002).

Thus, our approach to environmental regulation neglects how pollutants actually reach and affect humans: through exposures (not emissions), through mixtures of pollutants (rather than isolated pollutants), through several media (water, air, land, dust, consumer products, rather than one medium), through several routes (epidermal, ingestion, inhalation, intergenerational, rather than one route), causing multiple health effects (such as damage to the immune, neurological, endocrine, and reproductive systems, in addition to cancer, often the sole regulatory criterion).

What is a solution? The answer is not just regulatory, but also scientific, institutional, and educational. The next section discusses some principles of such an approach.

5. Reducing human exposure: what's needed

The science of exposure assessment can help us to determine what, where, and when pollutants come in contact with humans. The handful of exposure studies, from the EPA TEAM studies through the recent CDC and EWG studies, have shown that our regulations are missing the major sources of pollutant exposures and potential health risks. That is, risks from indoor air pollution, and the consumer products that we choose, are currently far greater than risks from outdoor air and sources traditionally regulated.

Paradoxically, the places that we normally consider "safe" (homes, schools, workplaces, vehicles, public buildings, medical facilities) and the products that we consider "safe" (because they are widely sold and used) are precisely the major sources of pollutant exposures. Yet these sources are virtually unregulated by existing environmental laws.

Fortunately, because many of these exposures are within our control, we can reduce significant health risks through relatively simple and cost-effective actions, such using less toxic consumer products and building materials. Unfortunately, the general public and the medical community are largely unaware of the major sources of pollutant exposures, their health effects, and ways to reduce those risks. Thus, a perilous gap exists between regulation and risk, and between science and public awareness.

What can be done to bridge these gaps? For one, we should have access to accurate and complete information about the chemical ingredients in products, the possible health effects from those chemicals, and the ways to reduce exposures. This would allow consumers to make more informed choices about the products they purchase and use, and if they do use those products, to know how to reduce exposures. This would also provide the data necessary for more effective regulation and protections.

Another important step would be to require more extensive testing, labeling, and evaluation of products before being put on the market, just as currently required for many foods and drugs. As exposure studies have shown, humans are affected by a wide range of non-food and non-pharmaceutical chemicals—chemicals that can cause adverse health effects and that are contained in common products that currently receive little or no pre-market testing in the US.

We should promote the use and production of safer alternatives to common products and practices that pose exposure risks. Such alternatives could provide the same function but with less toxicity, such as personal care products and laundry supplies without synthetic fragrances, paints and varnishes that are low-VOC, and pest control based on integrated pest management rather than synthetic chemical pesticides. Further, using less toxic products and practices can bring additional benefits such as improved performance and productivity, reduced health care costs and liability, and increased profitability. For instance, estimated savings from reducing indoor exposures exceed $100 billion annually, with benefits exceeding costs by tenfold (Fisk, 2001).

We should also take advantage of advances in the science and measurement of exposure; advance that can tell us, with great accuracy, which pollutants are reaching humans and from where. Nationwide exposure monitoring programs, much like ambient air and water monitoring networks currently in place, could provide vital information on how humans are exposed to environmental pollutants. We have vast amounts of epidemiological data, suggesting links between pollutant exposures and illness. To understand and confirm these links, epidemiology can be supplemented with direct measurements of physical, chemical, and biological pollutant exposure.

Yet monitoring exposures is only part of the solution. Given that we have found pollutants in the ''wrong places'' (e.g., pesticides in human breast milk), we need to ask ourselves not only how that exposure occurred, but also why that pollutant is being produced in the first place. Here, a precautionary approach can be usefully applied (Wingspread, 1998). We have evidence that humans can be harmed by substances that are any of the following: persistent, bioaccumulative, carcinogenic, endocrine disrupting, mutagenic, heavy metals, or toxic to immune, endocrine, and neurological systems, among other characteristics. A goal then should be to phase out and significantly reduce the reliance on these types of substances. And rather than waiting until a pollutant is emitted and found in the body, and then trying to assess the resulting harm, we can try to prevent harm in the first place, using what we already know about human exposures.

Acknowledgements

I thank Wayne Ott, Lance Wallace, John Roberts, Peter Montague, Andy Miller, and Ann McCampbell for their very helpful reviews of this manuscript.

References

ATSDR. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polychlorinated Biphenyls (PCBs). http://www.atsdr.cdc.gov/toxprofiles/tp17.html accessed September 12, 2003.

Akland G, Hartwell TD, Johnson TR, Whitmore R. Measuring Human Exposure to Carbon Monoxide in Washington, DC and Denver, CO during the winter of 1982–83. Environ Sci Technol 1985;19:911–8.

American Cancer Society (ACS) Cancer Facts and Figures. http://www.cancer.org/docroot/STT/stt_0_2001.asp?sitearea = STT and Level = 1 accessed September 12, 2003.

Ashford NA, Miller CS. Chemical exposures: low levels and high stakes. New York: Van Nostrand Reinhold; 1998.

Baskin LS, Himes L, Colborn T. Hypospadias and endocrine disruption: is there a connection? Environ Health Perspect 2001;109:1175–82.

Byrd RS. The epidemiology of autism in california: a comprehensive pilot study, California: U.C. Davis M.I.N.D. Institute; 2002.

Camann D, Colt J, Teitelbaum S, Rudel R, Hart R, Gammon M. Pesticide and PAH distributions in house dust from seven areas of the USA. Paper 570 presented at SETAC 2000, Nashville, TN, November 16, 2000.

(CDC) Centers for Disease Control and Prevention. Forecasted state-specific estimates of self-reported asthma prevalence-United States. MMWR Morb Mortal Wkly Rep 1998;47:1022–5.

(CDC) Centers for Disease Control and Prevention. First National Report on Human Exposure to Environmental Chemicals. 2001. http://www.cdc.gov/nceh/dls/report/.

(CDC) Centers for Disease Control and Prevention. Second National Report on Human Exposure to Environmental Chemicals. 2003. http://www.cdc.gov/exposurereport/.

(CERHR) Center for the Evaluation of Risks to Human Reproduction. National Toxicology Program Center for the Evaluation of Risks to Human Reproduction, 2000 [Available online at http://cerhr.niehs.nih.gov/news/phthalates/report.html].

Chakrabati S, Fombonne E. Pervasive developmental disorders in preschool children. JAMA 2001;285:3093–9.

Colborn T, vom Saal FS, Soto AM. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. Environ Health Perspect 1993;101(5):378–84.

Cooper SD, Raymer JH, Pellizzari ED, Thomas KW, Castillo NP, Maewall S. Polar organic compounds in fragrances of consumer products. Final Report, Contract 68-02-4544 U.S. EPA, Research Triangle Park, NC, June 19, 1992.

(EPA) U.S. Environmental Protection Agency. Unfinished Business: A Comparative Assessment of Environmental Problems. Volume I. Overview. US Environmental Protection Agency. Washington, DC. NTIS #PB-88-127048; 1987.

(EPA) U.S. Environmental Protection Agency. Future risk: research strategies for the 1990s. Washington (DC): US Environmental Protection Agency; 1988a.

(EPA) U.S. Environmental Protection Agency. Indoor air quality in public buildings, vols. I and II. Washington (DC): US EPA, Office of Research and Development; 1988b. EPA/600/S6-88/009a,b.

(EPA) U.S. Environmental Protection Agency. The inside story: a consumer's guide to indoor air pollution; 1988c [Washington, DC].

(EPA) U.S. Environmental Protection Agency. Comparing risks and setting environmental priorities: overview of three regional projects. Washington (DC): US Environmental Protections Agency; 1989a.

(EPA) U.S. Environmental Protection Agency. Indoor air pollutants from household sources. EPA 600/X-89-164. Las Vegas (NV): Environmental Monitoring Systems Laboratory; 1989b.

(EPA) U.S. Environmental Protection Agency. Inert Ingredients of Pesticides, Audit report no. E1EPF1-05-0117-1100378, September 27, 1991.

(EPA) U.S. Environmental Protection Agency. U.S. Environmental Protection Agency. Latest

Findings on National Air Quality: 2000. Status and Trends. Research Triangle Park, NC: Office of Air Quality Planning and Standards; 2002. EPA 454/K-01002. http://www.epa.gov/airtrends.

(EPA) U.S. Environmental Protection Agency. Lists of Other (Inert) Pesticide Ingredients http://www.epa.gov/opprd001/inerts/lists.html. 2003a.

(EPA) U.S. Environmental Protection Agency AM. Recognition and Management of Pesticide Poisonings. U.S. Environmental Protection Agency, 2003b. http://www.epa.gov/oppfead1/safety/healthcare/handbook/handbook.htm.

(EWG) Environmental Working Group. Body burden: the pollution in people; 2003. Washington, DC http://www.ewg.org/reports/bodyburden.

Fisher BE. Scents and sensitivities. Environ Health Perspect 1998;106:A594–9 ([November]).

Fisk WJ. Estimates of potential nationwide productivity and health benefits from better indoor environments: an update. In: Spengler JD, McCarthy JF, Samet J, editors. Indoor air quality handbook. New York: McGraw-Hill; 2001. Chapter 4.

(GAO) General Accounting Office. Toxic Substances Control Act: Preliminary Observations on Legislative Changes to Make TSCA More Effective (GAO/T-RCED-94-263). Author; 1994.

Gupta C. Reproductive malformation of the male offspring following maternal exposure to estrogenic chemicals. Proc Soc Exp Biol Med 2000;224:61–8.

Hayes TB, Collins A, Lee M, Mendoza M, Noriega N, Stuart AA, et al. Hermaphroditic, demasculinized frogs after exposure to the herbicide atrazine at low ecologically relevant doses. Proc Natl Acad Sci 2002;99:5476–80.

Immerman FW, Schaum JL. Nonoccupational pesticide exposure study: final report, EPA/600-3-90-003. Washington (DC): U.S. Environmental Protection Agency, 1990.

Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, Behar JV, Hern SC, Engelmann WH. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. J Expo Anal Environ Epidemiol 2001;11:231–52.

(NAS). National Academy of Sciences. Toxicological effects of methylmercury. Washington (DC): Author; 2000.

(NCI) National Cancer Institute. Female breast cancer, SEER cancer statistics review, 1973–1997. Author; 1997. http://seer.cancer.gov/csr/1973_1997/breast.pdf.

(NCI) National Cancer Institute. National cancer institute research on childhood cancers. Author; 2002a. http://cis.nci.nih.gov/fact/6_40.htm.

(NCI) National Cancer Institute. Surveillance, Epidemiology, End-Results (SEER) data base; cancer of the testis, men ages 15–34. Author; 2002b. http://seer.cancer.gov.

(NCI) National Cancer Institute. SEER cancer statistics review, 1973–1999. Author; 2002c. http://www.seer.cancer.gov/csr/1973_1999/sections.html.

NIH. Household products database, material safety data sheets. Author; 2003. http://householdproducts.nlm.nih.gov/health.htm.

(NTS) National Toxicological Program. Center for the evaluation of risks to human reproduction, expert panel review of phthalates, July 14, 2000. Author; 2000. Available at http://-ntp-server.niehs.nih.gov/htdocs/liaison/CERHRPhthalatesAnnct.html.

(NY) New York State. The secret hazards of pesticides: inert ingredients. Office of the Attorney General, Environmental Protection Bureau; 1996. February. http://www.oag.state.ny.us/environment/inerts96.html.

Omenn G. Report on the Presidential/Congressional Commission on Risk Assessment and Risk Management. Volume 1 Framework for Environmental Health Risk Management; Volume 2, Risk Assessment and Risk Management in Regulatory Decision-Making. http://www.riskworld.com/Nreports/1997/risk-rpt/miscinfo/nr7mi002.htm.

Ott WR. Total human exposure: an emerging science focuses on humans as receptors of environmental pollution. Environ Sci Technol 1985;19:880–6.

Ott WR, Roberts JW. Everyday exposure to toxic pollutants. Sci Am 1998;278(2):72–7.

Otto D, Mølhave L, Rose G, Hudnell HK, House D. Neurobehavioral and sensory irritant effects of controlled exposure to a complex mixture of volatile organic compounds. Neurotoxicol Teratol 1990;12:649–52.

Özkaynak H, Xue J, Weker R, Butler D, Koutrakis P, Spengler J. The Particle TEAM (PTEAM) study: analysis of the data vol. 3. (Research Triangle Park, NC, EPA/600/R-95/098).

Özkaynak H, Xue J, Spengler JD, Wallace LA, Pellizzari ED, Jenkins P. Personal exposure to airborne particles and metals: results from the particle TEAM study in Riverside, CA. J Expo Anal Environ Epidemiol 1996b;6:57–78.

Paulozzi LJ, Erickson JD, Jackson RJ. Hypospadias trends in two US surveillance systems. Pediatrics 1997;100:831–4.

Pellizzari ED, Thomas KW, Clayton CA, Whitmore RW, Shores RC, Zelon HS, et al. Particle Total Exposure Assessment Methodology (PTEAM): Riverside, California. Pilot Study vol. 1. Research Triangle Park (NC): NTIS, 1993 (Project Summary. EPA/600/SR-93/050. # PB 93-166957).

PSR. Physicians for social responsibility. bearing the burden: health implications of environmental pollutants in our bodies; 2003. http://www.envirohealthaction.org/environment/biomonitoring/articles.cfm?article_id=164.

Pierson TK, Hetes RG, Naugle DF. Risk characterization framework for noncancer end points. Environ Health Perspect 1991;95:121–9.

Raloff J. Girls may face risks from phthalates. Sci News 2000;158:165 [September 9].

Sack TM, Steele DH, Hammerstrom K, Remmers J. A survey of household products for volatile organic compounds. Atmos Environ 1992;26A:1063–70.

Schmidt CW. The lowdown on low-dose endocrine disruptors. Environ Health Perspect 2001;109(9) [September].

Sheldon LS, Handy RW, Hartwell TD, Whitmore RW, Zelon HS, Pellizzari ED, et al. Indoor Air Quality in Public Buildings vols. I and II. Research Triangle Park (NC): USEPA; 1988. EPA 600/S6-88/009a,b.

Sheldon L, Clayton A, Keever J, Perritt R, Whitaker D. PTEAM: Monitoring of Phthalates and PAHs in Indoor and Outdoor Air Samples in Riverside, California. Vol. 2. Sacramento (CA): Air Resources Board, Research Division; 1993.

Smith KR. Air pollution: assessing total exposure in the United States. Environment 1988;30(8):10–38.

Steinemann A. Rethinking human health impact assessment. Environ Impact Asses Rev 2000;20:627–45.

Steinemann A. Environmental laws and human exposure. In: Ott W, Steinemann A, editors. Human exposure analysis. Boca Raton (FL): CRC Press; 2004. Draft manuscript available from author.

(USHR) U.S. House of Representatives. Neurotoxins: At Home and the Workplace, Report by the Committee on Science and Technology, Report 99-827, Sept. 16, 1986.

vom Saal FS, Timms BG, Montano MM, Palanza P, Thayer KA, Nagel SC, et al. Prostate enlargement in mice due to fetal exposure to low doses of estradiol or diethylstilbestrol and opposite effects at high doses. Proc Natl Acad Sci 1997;94:2056–61.

Wallace LA. The TEAM study: summary and analysis vol. 1. Washington (DC): U.S. EPA; 1987. EPA 600/6-87/002a, NTIS, PB 88-100060.

Wallace LA. Comparison of risks from outdoor and indoor exposure to toxic chemicals. Environ Health Perspect 1991;95:7–13.

Wallace LA. A decade of studies of human exposure: what have we learned. Risk Anal 1993;13:135–9.

Wallace LA. Assessing human exposure to volatile organic compounds. In: Spengler JD, McCarthy JF, Samet J, editors. Indoor air quality handbook. New York: McGraw-Hill; 2001. Chapter 33.

Wallace LA, Pellizzari ED, Hartwell T, Zelon H, Sparacino C, Perritt R, et al. Concentrations of 20 volatile organic compounds in the air and drinking water of 350 residents of New Jersey compared with concentrations in their exhaled breath. J Occup Med 1986;28:603–8.

Wallace LA, Thomas J, Mage D, Ott WR. Comparison of Breath CO, CO Exposure, and Coburn Model Predictions in the U.S. EPA Washington–Denver CO Study. Atmos Environ 1988;22:2183–93.

Wallace LA, Pellizzari E, Wendel C. Total volatile organic concentrations in 2700 personal, indoor, and outdoor air samples collected in USEPA TEAM Studies. Indoor Air 1991;4:465–77.

Waller K, Swan SH, DeLorenze G, Hopkins B. Trihalomethanes in drinking water and spontaneous abortion. Epidemiology 1998;9:134–40.

Whitmore RW, Immerman FW, Camann DE, Bond AE, Lewis RG. Non-occupational exposure to pesticides for residents of two U.S. cities. Arch Environ Contam Toxicol 1994;26(1):47–59.

Wingspread. Wingspread statement on the precautionary principle. Wingspread Conference Center, Racine, Wisconsin, 23–25 January, 1998.

Zartarian VG, Ott WR, Duan NA. Quantitative Definition of Exposure and Related Concepts. J Expo Anal Environ Epidemiol 1997;7(4):411–37.