Body Burden of Chemicals and Burden of Proof

What are the health effects of all the pollutants in our bodies that we discussed in Rachel's #810? 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 1,000% 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 to 35 (NCI, 2002b). 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% to 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, joint pain, and simultaneously cause chronic damage to the endocrine, neurological, and immune systems (USEPA, 2003; 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 (ASTDR 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.

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 U.S. environmental laws revealed that none mentioned "indoor air" (Steinemann, 2004). 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. Nonetheless, several federal laws have some nexus with indoor air, and could provide the authority, if exercised.

The Clean Air Act of 1970 (CAA) could provide the U.S. EPA the authority to address indoor air quality through the regulation of "ambient air." Yet the original CAA does not define ambient air, and the EPA has limited its interpretation of ambient air to the regulation of "outdoor air." 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 Hazardous Air Pollutants...
such as pesticides and cosmetics.

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. 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.

The Toxic Substances Control Act (TSCA) provides the EPA broad authority to regulate chemicals that present an "unreasonable risk of injury to health or the environment." Yet "unreasonable risk" is not defined in TSCA, 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, or can lead to significant or substantial human exposure, 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, 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).

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

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, 2003). 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), requires an environmental impact statement (EIS) for federal actions "significantly affecting the quality of the human environment." Yet in the implementation of NEPA, impact assessments have focused on impacts to the environment, rather than impacts on humans. A 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), 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, nor state and municipal government employees unless a state has a plan approved by the OSHA. Even approved state plans are permitted to exclude private sector employees. 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." Also, under the OSH Act, violations must result in an employee's death in order for the employer to be subject to criminal sanctions. 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 exposures, 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).

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.
Reduction of 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 handbook 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 as 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 U.S.

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 ten-fold (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.

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* Anne Steinemann holds the title of Professor in the Department of Civil & Environmental Engineering, and in the School of Public Affairs, at the University of Washington in Seattle, where she is also director of the Center for Water and Watershed Studies. This article is a slightly modified version of "Human exposure, health hazards, and environmental regulations," Environmental Impact Assessment Review Vol. 24 (2004), pg. 695-710.

References for Part 2


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