

THE LEGAL CONTROL OF INDOOR AIR POLLUTION†

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I. INTRODUCTION TO INDOOR AIR POLLUTION

The Clean Air Act (CAA) has three principal programs aimed at protecting human health: (1) an ambient air quality program aimed at controlling emissions of six pollutants and their precursors that are released in large quantities; (2) a hazardous air pollution program aimed at controlling approximately 189 chemicals that are released from major stationary sources; and (3) a program to control emissions from new motor vehicles and fuels. All three programs focus on the outdoor or ambient air. Yet indoor air pollution, which can create greater risks to human health,¹ is usually subject only to minimal and desultory control. The work environment is the only place where indoor air is regulated in any meaningful way. This situation may slowly be changing as the health risks from air pollution in the non-industrial workplace become better known. Thus, standards developed under the Occupational Safety and Health Act (OSH Act) may

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¹ Risk studies consistently rank indoor air quality as a top environmental risk to public health. See OFFICE OF POLICY, PLANNING AND EVALUATION, U.S. ENVTL. PROTECTION AGENCY, COMPARING RISKS AND SETTING ENVIRONMENTAL PRIORITIES 44 (1989); see also R. Bruce Dickson, *Regulation of Indoor Air Quality: The Last Frontier of Environmental Regulation*, 8 NAT. RESOURCES & ENV'T 20 (1994).

affect the development of environmental law. Moreover, the increased concern for preventing air pollutants from being released, rather than just requiring end-of-the-pipe controls, is resulting in the slow extension of the OSH Act's preventive approach. However, indoor air pollution control has no consistent regulatory approach; rather, a substance is identified that is responsible for a health problem and an ad hoc program is developed to deal with it. This lack of consistency has led to a variety of federal agencies and federal laws that regulate indoor air pollution.

This Article examines some of the major types of indoor air pollution and aims to identify the public health-oriented laws and regulations that are used to control indoor air pollution in non-occupational settings where the law is in its early stages of development. It also examines material from the occupational health field, particularly the OSH Act, that is relevant to the control of non-occupational indoor air pollution. This Article does not deal with the tort or workers' compensation laws that may be utilized by those injured due to exposure to indoor air pollution; it does provide information for tort practitioners concerning relevant federal statutory law that may be germane to establishing the standard of care or other elements of a toxic tort cause of action.

Indoor air pollutants are found in residential and commercial buildings, and industrial facilities, at levels that adversely affect human health.² Most indoor environments have some form of air pollution and many have a significant air quality problem.³ Pollutants come from sources such as: fuels used for heating—oil, gas, kerosene, coal or wood; building materials and furnishings;⁴ products for household cleaning and maintenance; products used for personal care or hobbies;

² "A growing body of scientific evidence has indicated that air within homes and other buildings can be more seriously polluted than the outdoor air in even the largest and most industrialized cities." U.S. Env'tl. Protection Agency & U.S. Consumer Prod. Safety Comm'n, *The Inside Story: A Guide To Indoor Air Quality* (last modified Apr. 2, 1997) <<http://www.epa.gov/iaq/pubs/insidest.html>> [hereinafter *EPA Inside Story*]; see also U.S. Consumer Prod. Safety Comm'n, U.S. Env'tl. Protection Agency, and the American Lung Ass'n, *What You Should Know About Combustion Appliances and Indoor Air Pollution* (last modified Mar. 31, 1997) <<http://www.epa.gov/iaq/pubs/combust.html>> [hereinafter *Combustion Appliances*]. For indoor air quality information, products, and services, a specialized website can be accessed at <<http://www.envirovillage.com/>>.

³ See *EPA Inside Story*, *supra* note 2; see generally Grace C. Guiffreda, *The Proposed Indoor Air Quality Acts of 1993: The Comprehensive Solution to a Far-Reaching Problem?*, 11 PACE ENVTL. L. REV. 311 (1993).

⁴ One concern is a variety of toxic volatile organic compounds (VOCs), e.g. formaldehyde, released into the home from glues, resins, and preservatives used to manufacture items such as

pets; tobacco use; cooling systems and humidification devices; and ambient air pollution sources including radon, pesticides, automobile emissions and other pollutants.⁵

Indoor air pollution may pose a greater danger to health than pollution of ambient air⁶ because people spend up to ninety percent of their time indoors.⁷ Groups that potentially are more likely to be adversely affected by air pollution, such as infants, the elderly, and the infirm, are indoors nearly all the time.⁸ Even low concentrations of air pollutants can be injurious to long-term health⁹ because exposure to indoor pollutants is more frequent and more prolonged than is ambient air exposure.¹⁰ Thus, a pound of pollution released indoors is usually more damaging to health than that amount released outdoors.¹¹ It is even more dangerous to children, who breathe more air for their weight than adults and whose lungs and immune systems are still developing.¹²

The impact of individual pollutants depends on a number of factors such as toxicity, concentration,¹³ duration of exposure, and sensitiv-

synthetic flooring, carpets, drapes, furniture, and upholstery, or that are present in paneling, particleboard, and plywood building materials. See ANDREW N. DAVIS & PAUL E. SCHAFFMAN, *THE HOME ENVIRONMENTAL SOURCEBOOK* 107, 110 (1996).

⁵ See *EPA Inside Story*, *supra* note 2.

⁶ See *id.*

⁷ See *id.*

⁸ See Ken Sexton, *An Inside Look at Air Pollution*, 19 EPA J., Oct.-Dec. 1993, at 9.

⁹ See Kirk R. Smith, *Taking the True Measure of Air Pollution*, 19 EPA J., Oct.-Dec. 1993 at 6; see also OFFICE OF PROGRAM POL'Y ANALYSIS AND GOV'T ACCOUNTABILITY, REP. NO. 96-32: REVIEW OF FLORIDA'S INDOOR AIR QUALITY PROGRAMS (1997) [hereinafter Rep. No. 96-32]; *Combustion Appliances*, *supra* note 2.

¹⁰ EPA estimates that indoor air pollution levels are two to five times higher than those found outside. See DAVIS & SCHAFFMAN, *supra* note 4, at 109. Further, concentrations of chemicals indoors are often ten times greater than outdoors and maximum indoor exposures are at least a hundred times greater than maximum outdoor exposures. See Andrew Kopon, Jr. & Joseph C. Gergits, *Indoor Environment: Regulatory Developments and Emerging Standards of Care*, 62 DEF. COUNS. J. 47, 47 (1995).

¹¹ See Smith, *supra* note 9, at 7.

¹² See generally PAUL MILLER, NORTHEAST STATES FOR COORDINATED AIR USE MANAGEMENT, *THE LONG-RANGE TRANSPORT OF OZONE AND ITS PRECURSORS IN THE EASTERN UNITED STATES* 1 (1997).

¹³ "The primary factors which determine the concentration of indoor air pollutants are: (1) indoor source emission rates; (2) the air exchange rate; (3) pollution concentrations in outdoor air; and (4) pollution removal or chemical transformation rates." Andrew J. Harrison, Jr., *An Analysis of The Health Effects, Economic Consequences and Legal Implications of Human Exposure to Indoor Air Pollutants*, 37 S.D. L. REV. 289, 292-93 (1991-1992) (citing U.S. ENVTL. PROTECTION AGENCY, REPORT TO CONGRESS ON INDOOR AIR QUALITY: VOLUME II: ASSESSMENT AND CONTROL OF INDOOR AIR POLLUTION, 1-8 (1989) [hereinafter EPA REPORT]); see also DADE W. MOELLER, *ENVIRONMENTAL HEALTH* 26 (1992).

ity of those exposed.¹⁴ Some emission sources, including carpeting,¹⁵ pressed wood products, and some household products, emit pollutants at high levels immediately after installation. Over time, these emissions, called "off-gassing," gradually decrease.¹⁶ Other sources emit pollutants in relation to the occupant's activities. For instance, environmental tobacco smoke (ETS) and some cleaning solvents emit pollutants only during and shortly after the period of use; however, ETS does consist of semi-volatile tars which stick to surfaces and may later off-gas.

Insufficient ventilation, resulting in poor air exchange, can intensify indoor air pollution.¹⁷ During the energy-conscious 1970s, Americans made their buildings more energy-efficient by "weatherizing" them, thereby reducing the flow of outside air into the buildings.¹⁸ Energy conservation features, such as commercial wall and roof insulation, thermal windows, and sophisticated heating and cooling systems, affect air exchange rates and thermal characteristics, both of which affect the level of pollutants.¹⁹ Heating and cooling systems are important not only for the role they play in air exchange but also because they may emit combustion gases and distribute them throughout the building. Heat also speeds chemical reactions involving pollutants and increases off-gassing. An energy-efficient house, with energy saving ventilation equipment that maintains adequate air exchange rates, would cost about fifteen percent more to build with non-emitting materials than a comparable conventional house.²⁰

The amount and quality of outdoor air entering the home is significant in determining the concentration and distribution of indoor air pollutants.²¹ Outdoor air enters a home through infiltration, natural

¹⁴ See *Combustion Appliances*, *supra* note 2.

¹⁵ Hundreds of VOCs may be released from carpeting, including benzene, xylene, ethylbenzene, and 4-PC, a chemical used for stain resistance and in latex backings. See DAVIS & SCHAFFMAN, *supra* note 4, at 110; see also U.S. Env'tl. Protection Agency, *Carpet and Indoor Air Quality* (last modified Apr. 3, 1997) <<http://www.epa.gov/iaq/pubs/carpet.html>>.

¹⁶ See DAVIS & SCHAFFMAN, *supra* note 4, at 110.

¹⁷ See Office of Air and Radiation, U.S. Env'tl. Protection Agency, *Fact Sheet: Ventilation and Air Quality In Offices* (last modified Apr. 2, 1997) <<http://www.epa.gov/iaq/pubs/ventilat.html>> [hereinafter *Ventilation*]. The federal government currently has no standards for ventilation, and it is therefore regulated by local building codes which may address concerns other than indoor air quality. See *id.*; Guiffrida, *supra* note 3, at 314-15.

¹⁸ See Guiffrida, *supra* note 3, at 333; see also *EPA Inside Story*, *supra* note 2.

¹⁹ See Harrison, *supra* note 13, at 295.

²⁰ See DAVIS & SCHAFFMAN, *supra* note 4, at 111; see also *Indoor Air: Interiors May Pose Worse Threat Than Outdoors*, GREENWIRE, May 27, 1997, (page unavail.), available in LEXIS, News Library, Curnws File.

²¹ See *Ventilation*, *supra* note 17; see also *EPA Inside Story*, *supra* note 2.

ventilation through open windows and doors, and heating and cooling systems designed to introduce outdoor air and vent indoor air.²² Generally, ambient air is less polluted than the air in homes or commercial buildings.²³ However, poorly designed ventilation systems may not introduce sufficient outdoor air into a building, or may introduce polluted outdoor air through air intakes installed in poorly chosen locations.²⁴ For example, air intakes located near areas heavily used by cars and trucks can introduce vehicular exhaust into the ventilation system.²⁵

While in effectively "weatherized" homes the air exchange rate varies from 0.2 to 0.3 changes per hour, there can be 2.0 changes per hour in older or poorly constructed homes; the average in the U.S. is about 0.5 to 1.0 changes per hour.²⁶ Air exchange rates in commercial buildings range from 0.29 to 1.73 changes per hour with a mean of 0.94.²⁷ The air exchange rate necessary to "cleanse" indoor air of pollutants harmful to human health varies depending upon building design and the types of pollutants that are present. Filtering mechanisms can be used to increase pollutant removal. After construction of environmental control systems, proper maintenance is important to avoid system failures or the buildup of microbial contamination.²⁸

To counteract the negative effects of "weatherizing" homes, some laws include directives to consider indoor air quality. For example, the Energy Conservation and Production Act,²⁹ enacted to reduce energy demand through development of energy-efficient residential and commercial buildings, directs the Department of Energy to consider the impact of such energy-efficient options on habitability and on persons,³⁰ and to achieve a balance between a healthy environment and energy conservation.³¹ The National Manufactured Housing Construction and Safety Standards Act of 1974 requires the Department of Housing and Urban Development to promulgate standards for the

²² See *EPA Inside Story*, *supra* note 2.

²³ See Tony Bartelme, *Bad Air May Make Your Children Ill*, POST AND COURIER, May 18, 1997, at A1, available in LEXIS, News Library, Curnws File.

²⁴ See *Ventilation*, *supra* note 17; see also *EPA Inside Story*, *supra* note 2.

²⁵ See *EPA Inside Story*, *supra* note 2.

²⁶ See Harrison, *supra* note 13, at 294 (citation omitted).

²⁷ See *id.* (citing EPA REPORT, *supra* note 13, at 1-8).

²⁸ See *id.* at 294 (citing *EPA Inside Story*, *supra* note 2); see also *Ventilation*, *supra* note 17.

²⁹ Energy Conservation and Production Act, Pub. L. No. 94-385, 90 Stat. 1125 (1976); 12 U.S.C. § 1701z-8 (1994); 15 U.S.C. § 787 (1994); 42 U.S.C. §§ 787-790h (1994); 42 U.S.C. §§ 6801-6892 (1994).

³⁰ 42 U.S.C. § 6851 (1997).

³¹ 42 U.S.C. § 6863(b)(2)(A) (1997).

construction and safety of manufactured housing, including health and safety features related to indoor air.³²

The measurable economic impacts of indoor air pollution include: (1) materials and equipment costs; (2) direct medical costs; and (3) lost productivity.³³ Indoor air pollution can soil or permanently damage equipment, necessitating expensive cleaning or repair and possibly premature replacement.³⁴ Air pollutants that harm equipment and machinery include sulfur oxides, nitrogen oxides, ozone, particulates, and acidic gases.³⁵ Damage to electronic equipment may also occur due to the salt content of particulates.³⁶ Microbial growth may damage both equipment and buildings.³⁷ Costs associated with equipment damage include maintenance costs, repair and replacement expenditures, and reduced service life.³⁸ Direct medical costs include the expenses related to doctor visits, increased hospital visits,³⁹ hospital care, surgery, medication,⁴⁰ psychological counseling and employee sick days.⁴¹ These costs are high; the health care costs of smoking-related illnesses alone are estimated to be about \$50 billion a year.⁴² Indoor air pollution also increases the medical costs of treating pre-existing diseases.⁴³

³² National Manufactured Housing Construction and Safety Standards Act, Pub. L. No. 93-383, Title VI, 88 Stat. 700 (1974); 42 U.S.C. §§ 5401-5426 (1997).

³³ See ARNOLD W. REITZE, JR., AIR POLLUTION LAW 425 (citing EPA REPORT, *supra* note 13, at 5-1). EPA estimates that poor indoor air quality costs Americans tens of billions of dollars per year in medical expenses and lost productivity. See *Sears Announces National Rollout Plans for New Home Environment Center; Retailer Responds to Consumer Demand for Increased Health and Safety Measures in The Home*, PR NEWswire, May 28, 1997 (page unavail.), available in LEXIS, News Library, Curnws File.

³⁴ See REITZE, *supra* note 33, at 425 (citing EPA REPORT, *supra* note 13, at 5-1).

³⁵ See *id.* (citing EPA REPORT, *supra* note 13, at 5-5).

³⁶ See *id.* (citing EPA REPORT, *supra* note 13, at 5-5).

³⁷ See *id.* (citation omitted).

³⁸ See *id.* (citation omitted).

³⁹ See REITZE, *supra* note 33, at 425 (citing EPA REPORT, *supra* note 13, at 5-10).

⁴⁰ See *id.* (citation omitted).

⁴¹ See *id.* (citation omitted).

⁴² See Tara Parker-Pope, *The Settlement: Facts About the Global Tobacco Business*, WALL ST. J., June 23, 1997, at B1. Allergy to house dust mites and cats increases the risk of childhood asthma fourfold to sixfold. The annual cost associated with asthma in the U.S. is estimated to be more than \$6.2 billion. Andrew M. Pope, *Indoor Allergens: A Report*, 19 EPA J., Oct.-Dec. 1993, at 13.

⁴³ For example, EPA reported that asthmatic children who live in households with a smoker average 3.09 hospital emergency room visits per year, whereas asthmatic children in smokeless homes average 1.83 annual visits. The estimated annual costs on a national scale were approximated at \$157 to \$226 million per year. EPA REPORT, *supra* note 13, at 5-11 (citing David Evans et al., *The Impact of Passive Smoking on Emergency Room Visits of Urban Children with*

The costs associated with lost productivity may have the most significant impact on the economy. Such costs include lost productivity⁴⁴ because of fatigue, eye irritation, and headaches.⁴⁵ The Environmental Protection Agency (EPA) reported that in 1989, the annual cost to the economy because of productivity losses caused by radon-triggered lung problems may have added up to approximately \$1991 billion dollars (using 1986 dollar valuations).⁴⁶ The agency further reported that a New England survey estimated that the average productivity losses were fourteen minutes per day, in addition to 0.6 annual sick days each year. Applying these estimates to the white-collar work force, EPA estimated annual lost productivity costs of roughly \$60 billion dollars.⁴⁷

Expenditures to reduce lost productivity due to indoor air pollution appear justified on a monetary basis.⁴⁸ For example, EPA stated that the additional construction costs of providing twenty cubic feet per minute (cfm) per person of ventilation rather than five cfm/person would be fifty cents per square foot;⁴⁹ the costs of renovating existing buildings to increase ventilation would be higher. However, EPA reported that the necessary expenditures are offset by increases in worker productivity of only one percent.⁵⁰ Ideally, the value of an individual's pain and suffering, and the lost opportunity costs for those caring for the afflicted individual, also should be considered when evaluating the costs of indoor air pollution abatement.⁵¹

Asthma, 135 AM. REV. OF RESPIRATORY DISEASES 567, 567-72 (1987)); see also Geoffrey Cowley et al., *Why Ebonie Can't Breathe*, NEWSWEEK, May 26, 1997, at 58 (hospital admissions for asthma rise 20 to 30 percent during periods of severe air pollution); Lee Bowman, *Doctor Bills Run Higher For Kids of Smoker Moms*, CHI. SUN-TIMES, Mar. 24, 1997, at 17. EPA has estimated the medical costs for excess cancer caused by indoor air pollution to range from \$188 million to \$1.375 billion nationwide. See Curtis Haymore & Rosemarie Odom, *Economic Effects of Poor IAQ*, 19 EPA J., Oct.-Dec. 1993, at 28.

⁴⁴ See Harrison, *supra* note 13, at 323 (citing EPA REPORT, *supra* note 13, at 5-1, 5-2).

⁴⁵ See *id.* (citing EPA REPORT, *supra* note 13, at 5-13).

⁴⁶ See *id.* (citing EPA REPORT, *supra* note 13, at 5-14, Exhibit 5-6).

⁴⁷ See *id.* (citing EPA REPORT, *supra* note 13, at 5-15).

⁴⁸ See *id.* (citing EPA REPORT, *supra* note 13, at 5-15, 5-16); see also Alan J. Heavens, *Sick Haven*, SANTA FE NEW MEXICAN, May 25, 1997, at 11.

⁴⁹ See REITZE, *supra* note 33, at 426 (citing EPA REPORT, *supra* note 13, at 5-16, 5-17).

⁵⁰ See *id.* (citation omitted).

⁵¹ See *id.* (citation omitted).

II. THE ROLE OF EPA

Currently, no federal program applies to air quality in private homes,⁵² although the Occupational Safety and Health Administration (OSHA), EPA, and many state agencies regulate indoor air quality under various programs.⁵³ The CAA provides very little protection for those exposed to indoor air pollution. The CAA improves indoor air indirectly through its programs to lower the concentrations of air pollution in the outdoor or ambient air.⁵⁴ For example, CAA programs, such as the asbestos demolition work practices⁵⁵ and EPA's proposed regulation of Volatile Organic Compound (VOC) emissions from many consumer products, may improve indoor air quality.⁵⁶ But the modest efforts of EPA to control indoor air pollution are scattered among a variety of statutes, usually on a pollutant-specific basis.⁵⁷

EPA's broadest power to regulate indoor air pollutants is found in the 1976 Toxic Substances Control Act (TSCA).⁵⁸ TSCA gives EPA the authority to regulate chemicals from "cradle to grave" as well as broad powers to require testing, control, record-keeping, and reporting for chemicals and chemical mixtures.⁵⁹ EPA can require industry to test old and new chemicals⁶⁰ and may regulate the introduction of new chemicals into commerce.⁶¹ TSCA's broad record-keeping and reporting provision goes beyond the authority granted under other en-

⁵² There are, however, some federal regulations that affect indoor air quality. For example, HUD regulations require mobile home manufacturers to comply with certain indoor air pollutant level standards. See *Manufactured Home Construction and Safety Standards*, 58 Fed. Reg. 54,975 (1993) (codified at 24 C.F.R. pt. 3280); 57 Fed. Reg. 6420 (1992) (codified at 24 C.F.R. pt. 3280); 49 Fed. Reg. 31,996 (1985) (codified at 24 C.F.R. pt. 3280); 48 Fed. Reg. 37,136 (1983) (codified at 24 C.F.R. pt. 3280).

⁵³ There are more than 20 federal agencies with some responsibility for indoor air quality, some because of their statutory responsibilities and some because they are included among the regulated entities. An Interagency Committee of Indoor Air Quality (CIAQ) coordinates activities. It is co-chaired by EPA, the Consumer Product Safety Commission, the Department of Energy, the National Institute for Occupational Safety and Health, and the Occupational Safety and Health Administration. See Bob Axelrad, *Improving IAQ: EPA's Program*, 19 EPA J., Oct.-Dec. 1993, at 14, 17.

⁵⁴ 42 U.S.C. §§ 7401-7671q (1994).

⁵⁵ See National Emission Standards for Hazardous Air Pollutants; Asbestos NESHAP Revision, 55 Fed. Reg. 48,406 (1990) (codified at 40 C.F.R. pt. 61).

⁵⁶ VOC controls are discussed *infra* Section IV.F.

⁵⁷ See REITZE, *supra* note 33, at 427-31.

⁵⁸ Toxic Substances Control Act (TSCA), Pub. L. No. 94-469, 90 Stat. 2003 (1976) (current version at 15 U.S.C. §§ 2601-2692 (1994)).

⁵⁹ See generally TSCA §§ 2-412, 15 U.S.C. §§ 2601-2692.

⁶⁰ TSCA § 4, 15 U.S.C. § 2603 (1994).

⁶¹ TSCA § 5, 15 U.S.C. § 2604 (1994).

vironmental statutes.⁶² TSCA, however, provides that chemicals regulated under other acts will not normally be regulated under TSCA unless the EPA Administrator determines that such regulation is in the public interest.⁶³ TSCA specifically exempts pesticides, tobacco, some nuclear material, alcohol, food, drugs, and cosmetics.⁶⁴ It also contains an imminent hazard provision that allows EPA to take court actions against chemicals that threaten immediate harm.⁶⁵ TSCA provides EPA with authority to: (1) require manufacturers and processors to develop data on the effects of chemical substances and mixtures on health and the environment and (2) regulate chemical substances and mixtures that present an unreasonable risk of injury to human health and the environment.⁶⁶ EPA is authorized to apply a number of sanctions ranging from requiring labeling with instructions or warnings to prohibiting or limiting the manufacture, processing, or distribution of a chemical.⁶⁷ Thus, EPA may regulate the manufacture, processing, distribution, use, or disposal of toxic chemical substances.⁶⁸ TSCA has specific programs to deal with asbestos,⁶⁹ indoor radon abatement,⁷⁰ lead exposure reduction,⁷¹ and the control of polychlorinated biphenyls.⁷² However, it is difficult for EPA to develop the administrative record that would meet the "unreasonable risk" test which is the prerequisite for regulation.⁷³

Other sources of statutory authority that EPA can use to regulate indoor air include the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA),⁷⁴ the Comprehensive Environmental Response, Com-

⁶² TSCA § 8, 15 U.S.C. § 2607 (1994).

⁶³ TSCA § 9(b), 15 U.S.C. § 2608(b) (1994). TSCA's relationship to other environmental laws is not clear. This subject is explored in MCKENNA & CUNEO, L.L.P., TSCA HANDBOOK 251 (3d ed. 1997).

⁶⁴ TSCA § 3(2)(B), 15 U.S.C. § 2602(2)(B) (1994).

⁶⁵ TSCA § 7, 15 U.S.C. § 2606 (1994).

⁶⁶ TSCA § 5, 15 U.S.C. § 2604.

⁶⁷ TSCA § 6, 15 U.S.C. § 2605 (1994).

⁶⁸ *Id.*

⁶⁹ TSCA §§ 201-216, 15 U.S.C. §§ 2641-2656 (1994); *see, e.g.*, Asbestos; Manufacture, Importation, Processing, and Distribution in Commerce Prohibitions, 54 Fed. Reg. 29,460 (1989) (codified at 40 C.F.R. pt. 763). The asbestos regulation was rejected in *Corrosion Proof Fittings v. EPA*, 947 F.2d 1201 (5th Cir. 1991).

⁷⁰ TSCA §§ 301-311, 15 U.S.C. §§ 2661-2671 (1994).

⁷¹ TSCA §§ 401-412, 15 U.S.C. §§ 2681-2692 (1994).

⁷² TSCA § 6(e), 15 U.S.C. § 2605(e); *see, e.g.*, Disposal of Polychlorinated Biphenyls; Import for Disposal, 61 Fed. Reg. 11,096 (1996) (codified at 40 C.F.R. pts. 9, 761).

⁷³ *See* REITZE, *supra* note 33, at 427-28 (citing EPA REPORT, *supra* note 13, at 8-6).

⁷⁴ Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), 7 U.S.C. §§ 136-136y (1994).

pensation and Liability Act (CERCLA)⁷⁵ and the Safe Drinking Water Act (SDWA).⁷⁶

FIFRA may protect indoor air because some pesticides regulated under the act affect indoor environments. EPA has the authority under FIFRA to regulate indoor air pollution by banning or limiting pesticide use.⁷⁷ EPA considers the removal from the market of chlor-dane, a termiticide, and mercury, used as a mildewcide, to be accomplishments that improve indoor air quality.⁷⁸ The Food Quality Protection Act of 1996 (FQPA),⁷⁹ which amends FIFRA, may require EPA, when making food safety decisions for a single chemical, to consider exposures from traces of a pesticide in drinking water and exposures from uses around the home; FQPA may indirectly reduce some indoor air pollutants by leading manufacturers to withdraw minor-use pesticides from the market to avoid the high cost of the testing required for reregistration.⁸⁰

CERCLA provides minimal control over indoor air. It is concerned primarily with the control of releases to the environment. "Environment" is defined to include releases to the ambient air.⁸¹ Moreover, the term release excludes "(A) any release which results in exposure to persons solely within a workplace, with respect to a claim which such persons may assert against the employer of such persons."⁸² Nevertheless, CERCLA was amended in 1986⁸³ to require EPA to report to Congress on federal efforts to address indoor air problems.⁸⁴ The amendment also required EPA to coordinate its efforts with other federal agencies, which led to the creation of the Interagency Committee on Indoor Air Quality.⁸⁵

⁷⁵ 42 U.S.C. §§ 9601-9675 (1994).

⁷⁶ 42 U.S.C. §§ 300f-300j-26 (1994).

⁷⁷ See generally FIFRA §§ 1-31, 7 U.S.C. §§ 136-136y (1996); Guiffrida, *supra* note 3, at 328-29.

⁷⁸ See Axelrad, *supra* note 53, at 16.

⁷⁹ Food Quality Protection Act, Pub. L. No. 104-170, 110 Stat. 1489 (1996).

⁸⁰ See *EPA Declines to Extend Irodione Fungicide Use on Cotton, Citing New Law*, Daily Env't Rep. (BNA) at D6 (May 5, 1997); see generally Allison D. Carpenter, Note, *Impact of the Food Quality Protection Act of 1996*, 3 ENVTL. LAW. 479 (1996).

⁸¹ Comprehensive Environmental Response Compensation and Liability Act (CERCLA) § 101(8), 42 U.S.C. § 9601(8) (1994).

⁸² CERCLA § 101(22), 42 U.S.C. § 9601(22). There are other exclusions for engine exhaust, nuclear materials, etc. that could also be used to avoid application of CERCLA to the indoor environment. See *id.*

⁸³ Superfund Amendments and Reauthorization Act (SARA), Pub. L. No. 99-499, 100 Stat. 1613 (1986).

⁸⁴ See Guiffrida, *supra* note 3, at 320. EPA reported to Congress in 1989. *Id.*

⁸⁵ See Axelrad, *supra* note 53, at 17. This Committee holds quarterly meetings in Washington, D.C., and functions as the primary federal coordination mechanism for indoor air. See *id.* The

CERCLA established the Agency for Toxic Substances and Disease Registry (ATSDR) within the Public Health Service⁸⁶ to perform research, health status surveys, and "screening programs to determine relationships between exposure to toxic substances and illness."⁸⁷ "The Administrator of ATSDR may perform health assessments for releases or facilities where individual persons or licensed physicians provide information that individuals have been exposed to a hazardous substance, for which the probable source of such exposure is a release."⁸⁸

Under the SDWA,⁸⁹ EPA may regulate contaminants that "may have any adverse effects on the health of persons."⁹⁰ Radon is found in the drinking water supplies of some areas of the country, as are other pollutants, including pesticides. The SDWA's language gives EPA authority to set standards for indoor air pollutants coming from the water supply.⁹¹ Many VOCs that can enter the air through volatilization of water have been regulated.⁹² The SDWA also has regulations dealing with heavy metals including lead.⁹³ The 1996 Amendments to the SDWA⁹⁴ do not have a significant effect on indoor air

Committee's members include EPA, Department of Energy, Department of Health and Human Services, Department of Labor, Department of Defense, Department of the Interior, Department of State, Department of Transportation, General Services Administration, Department of Housing and Urban Development, National Aeronautics and Space Administration, National Institute of Standards & Technology, Consumer Product Safety Commission, and the Tennessee Valley Authority. See INTERAGENCY COMMITTEE ON INDOOR AIR QUALITY, U.S. ENVTL. PROTECTION AGENCY, No. 402K-95005, CURRENT FEDERAL INDOOR AIR QUALITY ACTIVITIES i-vii (1995). The Committee's most recent effort is geared toward producing and implementing "an action plan that will increase EPA's commitment and attention to human health indoors in the 21st century." AGENDA: QUARTERLY MEETING OF THE INTERAGENCY COMMITTEE ON INDOOR AIR QUALITY (Apr. 30, 1997).

⁸⁶ CERCLA § 104(i), 42 U.S.C. § 9604(i) (1994).

⁸⁷ CERCLA § 104(i)(1)(E), 42 U.S.C. § 9604(i)(1).

⁸⁸ CERCLA § 104(i)(6)(B), 42 U.S.C. § 9604(i)(6)(B). But note that the definition of "release" limits the applicability of this provision. See CERCLA § 101(22), 42 U.S.C. § 9601(22),

⁸⁹ 42 U.S.C. §§ 300(f)-300(j) (1988) (*amended by the Safe Drinking Water Act Amendments of 1996*, 42 U.S.C. §§ 300(f) to 300(j)-25 (1996)).

⁹⁰ 42 U.S.C. § 300(g)-1(b)(3)(A).

⁹¹ See generally 42 U.S.C. §§ 300(g)-1 to 300(h)-7 (1996).

⁹² See Axelrad, *supra* note 53, at 16. EPA set standards for 18 VOCs as of 1993 and more were planned to be promulgated. See Office of Air and Radiation, U.S. Env'tl. Protection Agency, *Targeting Indoor Air Pollution, EPA's Approach and Progress* (visited June 23, 1997) <<http://www.epa.gov/iaq/pubs/targetng.html>>.

⁹³ See Maximum Contaminant Levels Goals and National Primary Drinking Water Regulations for Lead and Copper, 56 Fed. Reg. 26,460 (1991) (codified at 40 C.F.R. pts. 141, 142). EPA proposed revisions to its lead and copper rule on April 12, 1996. See Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper, 61 Fed. Reg. 16,348 (1996) (to be codified at 40 C.F.R. pts. 141, 142) (proposed Apr. 12, 1996).

⁹⁴ 42 U.S.C. §§ 300(f) to 300(j)-25 (1996).

pollution,⁹⁵ although a provision of the amendments provides a new process and schedule for radon regulation.⁹⁶

III. THE WORKING ENVIRONMENT

Early federal occupational protection efforts focused on protecting seamen, railroad workers, and miners from the dangers associated with those occupations.⁹⁷ In 1936, the Walsh-Healey Public Contracts Act directed the Department of Labor to ensure that federal contractors met minimum health and safety standards.⁹⁸ In 1969, the Federal Coal Mine Health and Safety Act significantly increased the federal role in protecting the well-being of miners.⁹⁹ In 1970, Congress enacted the comprehensive Occupational Safety and Health Act (OSH Act).¹⁰⁰ Prior to 1970, the control of workplace pollutants was primarily based on the use of voluntary standards. These standards were promulgated by organizations such as the American Conference of Governmental Industrial Hygienists (ACGIH) which publishes Threshold Limit Values (TLVs) that are supposed to be the maximum concentrations of pollutants without adverse effects due to inhalation exposure.¹⁰¹ The American Standards Association (ASA), now the American National Standards Institute (ANSI), also sets standards; however, it no longer sets chemical exposure standards.¹⁰²

Indoor air in the workplace is subject to regulation under the OSH Act. The OSH Act applies to most private sector businesses.¹⁰³ However, workplaces that are regulated by specific health and safety laws, the Atomic Energy Act of 1954¹⁰⁴ and the Federal Coal Mine Health and Safety Act,¹⁰⁵ for example, are not covered. The OSH Act covers federal employees because of an executive order,¹⁰⁶ but does not cover

⁹⁵ See William E. Cox, *Evolution of the Safe Drinking Water Act: A Search for Effective Quality Assurance Strategies and Workable Concepts of Federalism*, 21 WM. & MARY ENVTL. L. & POL'Y REV. 69, 105 (1997).

⁹⁶ 42 U.S.C. § 300g-1(b)(13). See also *infra* Section IV.(B)(3).

⁹⁷ See NICHOLAS A. ASHFORD & CHARLES C. CALDART, *TECHNOLOGY, LAW, AND THE WORKING ENVIRONMENT* 91 (1996).

⁹⁸ 41 U.S.C. §§ 35-45 (1994).

⁹⁹ See generally 30 U.S.C. §§ 801-960 (1994).

¹⁰⁰ 29 U.S.C. §§ 651-678 (1994).

¹⁰¹ See John C. Dernbach, *The Unfocused Regulation of Toxic and Hazardous Pollutants*, 21 HARV. ENVTL. L. REV. 1, 29 (1997).

¹⁰² *Id.* at n.143.

¹⁰³ 29 U.S.C. §§ 652(5), 653-654(a) (1994).

¹⁰⁴ 42 U.S.C. §§ 2011-2297g-4 (1994).

¹⁰⁵ 30 U.S.C. §§ 801-960 (1994).

¹⁰⁶ Exec. Order No. 11,807, 39 Fed. Reg. 35,559 (1974) (superseding Exec. Order No. 11,612, 36 Fed. Reg. 13,891 (1971)).

state or municipal workers unless a state is operating under a plan approved by OSHA that provides such coverage.¹⁰⁷ Control of air pollution exposure under the OSH Act can be based on the "general duty" provision of section 5(a)(2) or on a specific occupational health standard. The general duty provision imposes a duty to furnish a place of employment "free from recognized hazards that are likely to cause death or serious physical harm" to employees.¹⁰⁸ A hazard is "recognized" if it is a condition that is (a) common knowledge or generally recognized in the particular industry in which it occurs, and (b) detectable (1) by means of the senses or (2) by being widely recognized as a hazard in the industry, with generally known and accepted tests for its existence.¹⁰⁹ The general duty clause may be applicable to an employer even if a specific standard is also applicable and the employer has observed the specific standard.¹¹⁰ Specific standards under the OSH Act are interim, permanent, and temporary emergency standards.¹¹¹ Interim standards are federal standards from other acts and national consensus standards existing at the time the OSH Act was implemented.¹¹² Permanent standards replace or augment interim standards.¹¹³ Emergency Temporary Standards (ETS) may be issued if there is a grave danger to employees.¹¹⁴ The ETS provision is not an interim relief measure, but is an extraordinary power to be used only in limited situations where a grave danger exists, and even then is to be exercised delicately.¹¹⁵ Use of ETS requires balancing health protection against economics.¹¹⁶ It is not easy to meet the legal requirements in order to use an ETS to protect workers from air pollutants.¹¹⁷

OSHA adopted thousands of national consensus standards.¹¹⁸ About 400 of the standards concerned health rather than safety. These in-

¹⁰⁷ See 29 U.S.C. § 667 (1994).

¹⁰⁸ 29 U.S.C. § 654(a).

¹⁰⁹ See 33 C.F.R. § 142.4(c) (1996).

¹¹⁰ See *International Union, United Auto., Aerospace & Agric. Implement Workers of Am. v. General Dynamics Land Sys. Div.*, 815 F.2d 1570, 1576 (D.C. Cir. 1987).

¹¹¹ See 29 U.S.C. § 655(a)-(c) (1994).

¹¹² See 29 U.S.C. § 655(a). "National consensus standard" is defined at 29 U.S.C. § 653(9) (1994).

¹¹³ See 29 U.S.C. § 655(b).

¹¹⁴ See *id.*

¹¹⁵ See *Public Citizen Health Research Group v. Aucther*, 702 F.2d 1150, 1155 (D.C. Cir. 1983) (quoting *Florida Peach Growers Ass'n v. United States Dep't of Labor*, 489 F.2d 120, 129 (5th Cir. 1974)).

¹¹⁶ See *Florida Peach Growers*, 489 F.2d at 129-30.

¹¹⁷ See *generally Asbestos Info. Ass'n/North Am. v. OSHA*, 727 F.2d 415 (5th Cir. 1984).

¹¹⁸ See 29 C.F.R. § 1910.2(g) (1997).

cluded TLVs recommended by ACGIH, and exposure levels for toxic substances recommended by ANSI.¹¹⁹ The most important standard may be the ANSI/American Society of Heating, Refrigeration and Air-Conditioning Engineers (ASHRAE) Standard 62-1989 dealing with ventilation in commercial, institutional, and residential buildings.¹²⁰ OSHA has the authority to issue standards reasonably necessary to eliminate or diminish a risk of material health impairment.¹²¹ OSHA has promulgated standards that set exposure limits for about twenty-nine substances.¹²² In addition, OSHA imposes work practices on hazardous materials¹²³ and has provisions specifying personal protective equipment.¹²⁴ There are provisions for process safety management of highly dangerous chemicals and for emergency response to problems involving hazardous waste operations.¹²⁵

OSHA's efforts to create permanent standards to strengthen the consensus standards have not been successful. To promulgate new standards for toxic materials or harmful physical agents the Secretary of Labor must set standards that: (1) are feasible; (2) are based on the best available evidence; and (3) protect employees from material impairment of health or functional capacity even if the employees are regularly exposed for their working life.¹²⁶ The U.S. Supreme Court, in *Industrial Union Department v. American Petroleum Institute*, interpreted these requirements to make it difficult, expensive, and slow for OSHA to promulgate new or revised standards.¹²⁷ The Court held that there must be a significant risk of harm that would be reduced or eliminated by a feasible standard.¹²⁸ The *American Petro-*

¹¹⁹ See ASHFORD, *supra* note 97, at 103; see also *AFL-CIO v. OSHA*, 965 F.2d 962, 968 (11th Cir. 1992).

¹²⁰ In August 1996 the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) submitted a draft proposed standard to the American National Standards Institute (ANSI) Board of Standards Review (BSR). The proposed standard BAR/ASHRAE 62-1989R, Ventilation for Acceptable Indoor Air Quality, will update industry standards concerning ventilation. Among the changes are increased ventilation necessary to dilute pollutants from building furnishings and materials. ASHRAE is located at 1791 Tullie Circle, NE, Atlanta, Ga. 30329-2305; (404) 636-8400.

¹²¹ See *Industrial Union Dep't, AFL-CIO v. American Petroleum Inst.*, 448 U.S. 607, 639 (1980).

¹²² 29 C.F.R. § 1910.1000 (1997).

¹²³ 29 C.F.R. §§ 1910.101-1910.120 (1997).

¹²⁴ 29 C.F.R. §§ 1910.132-1910.138 (1997).

¹²⁵ 29 C.F.R. §§ 1910.119-1910.120 (1997).

¹²⁶ 29 U.S.C. § 655(b)(5) (1994).

¹²⁷ See *Industrial Union Dep't, AFL-CIO v. American Petroleum Inst.*, 448 U.S. 607, 628 (1980) (benzene standard). The benzene standard is found at 29 C.F.R. § 1910.1028.

¹²⁸ See *American Petroleum Inst.*, 448 U.S. at 613-15.

leum Institute case also made it very difficult for OSHA to create a generic cancer policy; therefore, many of the assumptions that produced the proposed generic cancer policy were suspect.¹²⁹ In 1981, a final rule deleted provisions of the generic cancer policy that were inconsistent with the U.S. Supreme Court decision.¹³⁰ OSHA published amendments to the generic cancer policy in 1981,¹³¹ but then withdrew them.¹³² In 1982, OSHA issued an advance notice of proposed rulemaking,¹³³ and in 1986 published its intent to revise its generic cancer policy by January 1987.¹³⁴ The proposal, however, was withdrawn in August 1990.¹³⁵

OSHA issued an Air Contaminants Standard in 1989 that amalgamated 428 specific and individual substance exposure limits.¹³⁶ The rule would have reflected updates of 212 TVLs issued by ACGIH and would have added 164 substances that were previously unregulated.¹³⁷ However, in 1992, the U.S. Court of Appeals for the Eleventh Circuit vacated the entire standard based on OSHA's overall flawed approach.¹³⁸ The Eleventh Circuit rejected the standard because OSHA had failed to show in the administrative record the necessary significant risk of material impairment and feasibility for each substance.¹³⁹ However, the court did find that irritation severe enough to seriously threaten employee health and job performance constitutes material impairment, even if transitory.¹⁴⁰ The following year, OSHA reinstated its pre-1989 standards¹⁴¹ with two additions for formalde-

¹²⁹ OSHA issued its proposed generic cancer policy on October 4, 1977. Identification, Classification and Regulation of Toxic Substances Posing a Potential Occupational Carcinogenic Risk, 42 Fed. Reg. 54,147 (1977) (codified at 29 C.F.R. pt. 1990).

¹³⁰ See Identification, Classification and Regulation of Potential Occupational Carcinogens; Conforming Deletions, 46 Fed. Reg. 4889 (1981) (codified at 29 C.F.R. pt. 1990).

¹³¹ See Identification, Classification and Regulation of Potential Occupational Carcinogens; Proposed Amendments, 46 Fed. Reg. 7402 (1981) (to be codified at 29 C.F.R. pt. 1990) (proposed Jan. 23, 1981).

¹³² See Identification, Classification and Regulation of Potential Occupational Carcinogens, Withdrawal of Proposed Amendments, 46 Fed. Reg. 19,000 (1981) (codified at 29 C.F.R. pt. 1990).

¹³³ See Identification, Classification and Regulation of Potential Occupational Carcinogens, 47 Fed. Reg. 187 (1982) (to be codified at 29 C.F.R. pt. 1990) (proposed Jan. 5, 1982).

¹³⁴ See Semiannual Agenda of Regulations, 51 Fed. Reg. 38,560 (1986).

¹³⁵ See Semiannual Agenda of Regulations, 55 Fed. Reg. 44,680 (1990).

¹³⁶ See Air Contaminants, 54 Fed. Reg. 2332 (1989) (codified at 29 C.F.R. § 1910.1000).

¹³⁷ See *id.*

¹³⁸ See *AFL-CIO v. OSHA*, 965 F.2d 962, 975 (11th Cir. 1992).

¹³⁹ See *id.* at 986-87.

¹⁴⁰ See *id.* at 975.

¹⁴¹ See Air Contaminants, 58 Fed. Reg. 35,338, 35,340 (1993) (codified at 29 C.F.R. pt. 1910).

hyde¹⁴² and methylenedianiline.¹⁴³ Thus, "all but 28 of the toxic and hazardous substances now regulated under OSHA derive primarily from the 1968 ACGIH recommendations."¹⁴⁴ In April 1994, OSHA proposed a comprehensive standard to regulate indoor air quality.¹⁴⁵ The provisions were to apply to all indoor "nonindustrial work environments," with all worksites covered by provisions dealing with control of environmental tobacco smoke. The proposal required employers to develop and implement a written indoor air quality compliance plan, implement controls for specific contaminants and their sources, limit degradation of indoor air quality (as defined by ASHRAE 62), inform and train employees, and meet record-keeping requirements.¹⁴⁶ However, OSHA has not yet promulgated a final rule.¹⁴⁷

Section 18 of the OSH Act provides for federal law to preempt state law when OSHA has promulgated a health standard.¹⁴⁸ But states can develop their own standards if OSHA approves their occupational health plans.¹⁴⁹ The states and territories with occupational safety and health plans are: Alaska, Arizona, California, Connecticut (for state and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Nevada, New Mexico, New York (for state and local government employees only), North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, the Virgin Islands, Washington, and Wyoming.¹⁵⁰

A major weakness of the OSH Act is its penalty provisions. Under the OSH Act, violations must result in an employee's death in order for the employer to be subject to criminal sanctions. The penalty for a willful violation is a \$10,000 fine and imprisonment for up to six months for a willful violation.¹⁵¹ OSHA, however, usually fails to punish employers.¹⁵² Nevertheless, the OSH Act and its regulations still

¹⁴² 29 C.F.R. § 1910.1048 (1997).

¹⁴³ 29 C.F.R. § 1910.1050 (1997).

¹⁴⁴ Dernbach, *supra* note 101, at 31.

¹⁴⁵ See Indoor Air Quality, 59 Fed. Reg. 15,968 (1994) (codified at 29 C.F.R. pts. 1910, 1915, 1926, 1928).

¹⁴⁶ See *id.*

¹⁴⁷ See Semiannual Agenda of Regulations, 61 Fed. Reg. 21,934 (1997).

¹⁴⁸ 29 U.S.C. § 667 (1994).

¹⁴⁹ *Id.*

¹⁵⁰ See Occupational Exposure to Methylene Chloride, 62 Fed. Reg. 1494 (1997) (to be codified at 29 C.F.R. pts. 1910, 1915, 1926).

¹⁵¹ 29 U.S.C. § 666(e) (1994).

¹⁵² See generally Wallace N. Davidson III et al., *Are OSHA Penalties Effective?*, Bus. & Soc'y Rev., Jan. 1995, at 25-28.

represent the most comprehensive effort to deal with indoor air pollution in the United States legal system.

IV. INDOOR AIR POLLUTION CONTAMINANTS

Among the most significant indoor air pollutants are ETS, radon and radon progeny, biological contaminants, asbestos, lead, VOCs¹⁵³ (including polycyclic aromatic hydrocarbons (PAHs)),¹⁵⁴ pesticides, and combustion byproducts. The health effects of these pollutants include irritation of the eyes, nose and throat; headaches; dizziness and fatigue; emphysema and other respiratory diseases; heart disease; cancer; chronic organ damage; and acute toxicity.¹⁵⁵

Health effects often are noticed only after repeated exposures or after long periods of time.¹⁵⁶ Moreover, the effects of pollutants can vary substantially from person to person; some people are genetically predisposed to react adversely to pollution exposure.¹⁵⁷ Symptoms of diseases such as asthma, hypersensitivity pneumonitis, and humidifier fever, usually are intensified by exposure to indoor air pollutants.¹⁵⁸ Certain portions of the human population are more susceptible to the adverse effects of pollution exposure, including the young, the elderly, and those affected by weakened physiological defense mechanisms, such as reduced resistance to pulmonary irritants, reduced resistance to infections, or heightened sensitivity to pollutants.¹⁵⁹ Because they are more likely to be indoors, these people usually have greater

¹⁵³ EPA has identified over 900 VOCs in indoor environments. See Harrison, *supra* note 13, at 321 n.297 (citing EPA REPORT, *supra* note 13, at 3-6). VOCs are emitted from construction materials, structural components, furnishings, cleaners and solvents, personal care products, insecticides and pesticides, electrical equipment, and combustion sources. See John D. Spengler, *Sources and Concentrations of Indoor Air Pollution*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE 33, 46 (Jonathan M. Samet & John D. Spengler eds., 1991). VOC sources can be controlled or eliminated in a number of ways including prohibiting indoor smoking, reducing the use of deodorizers, and removing pesticides and solvents from indoors. See Lance A. Wallace, *Volatile Organic Compounds*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE 252, 265 (Jonathan M. Samet & John D. Spengler eds., 1991). VOC sources cause a variety of health effects ranging from sensory irritation to behavioral, neurotoxic and hepatotoxic effects. *Id.*

¹⁵⁴ Polycyclic aromatic hydrocarbons (PAHs) are found in ETS, and in emissions from kerosene heaters and wood stoves. Health effects include cancer and cardiovascular effects. See Harrison, *supra* note 13, at 321 n.294 (citing EPA REPORT, *supra* note 13, at 3-2, Exhibit 3-1). PAHs are "probable human carcinogens." See *id.* (citing EPA REPORT, *supra* note 13, at 4-15).

¹⁵⁵ See EPA *Inside Story*, *supra* note 2; see also Harrison, *supra* note 13, at 320-21 (citing EPA REPORT, *supra* note 13, at 3-4).

¹⁵⁶ See EPA *Inside Story*, *supra* note 2.

¹⁵⁷ See *id.*

¹⁵⁸ See *id.*

¹⁵⁹ See Harrison, *supra* note 13, at 292; see also EPA *Inside Story*, *supra* note 2.

exposure to indoor air pollutants than the general population does.¹⁶⁰ This complicates efforts to quantify the potential of pollutants to cause adverse health effects, and the synergistic effects of multiple pollutants and exposure paths further complicate the evaluation of health effects.

Scientific knowledge generally is insufficient to know the levels of exposure where adverse health effects begin or to make accurate quantitative risk determinations.¹⁶¹ Some animal and human health studies have supplied data adequate to quantify the relationship between exposure to specific pollutants and the severity of the expected health effect.¹⁶² Also, some data is available from studies of indoor air pollution in industrial settings.¹⁶³ Epidemiologic studies have linked exposure to certain pollutants to morbidity or mortality in human populations, but this area of science is still in its infancy.¹⁶⁴ Some diseases, however, are associated with very specific air pollutants. They include: byssinosis—cotton dust; mesothelioma and asbestosis—asbestos; coal workers' pneumoconiosis—coal dust; lead poisoning; and diseases associated with heavy metal poisoning.¹⁶⁵ Such diseases have been the health problems most frequently associated with efforts to control occupational exposure to air pollution. The Black Lung Benefits Act¹⁶⁶ provides monetary benefits to coal miners who are totally disabled due to pneumoconiosis caused by employment in the nation's coal mines.¹⁶⁷ The Act also provides benefits for surviving dependents of miners whose deaths were due to pneumoconiosis.¹⁶⁸

¹⁶⁰ See *EPA Inside Story*, *supra* note 2 (people with respiratory or cardiovascular diseases are especially susceptible); see also Rep. No. 96-32, *supra* note 9, (infants, young children, and the elderly are especially vulnerable).

¹⁶¹ See REITZE, *supra* note 33, at 395.

¹⁶² See *id.*

¹⁶³ See *id.*

¹⁶⁴ See *id.*

¹⁶⁵ See generally U.S. DEP'T OF HEALTH, EDUC., AND WELFARE, *OCCUPATIONAL DISEASES: A GUIDE TO THEIR RECOGNITION* (W. M. Gafafer ed., 1967).

¹⁶⁶ 30 U.S.C. §§ 901-945 (1994).

¹⁶⁷ See Arnold W. Reitze, Jr., *Federal Compensation for Vaccination Induced Injuries*, 13 B.C. ENVTL. AFF. L. REV. 169, 211-14 (1986).

¹⁶⁸ See *id.*; see also John S. Lopatto, III, *The Federal Black Lung Program: A 1983 Primer*, 85 W. VA. L. REV. 677 (1983).

A. *Environmental Tobacco Smoke (ETS)*¹⁶⁹

1. Sources of ETS

It is widely accepted that the direct inhalation of tobacco smoke is detrimental to human health.¹⁷⁰ Cigarette smoking is claimed to result in over 400,000 deaths in the United States each year.¹⁷¹ Interest in the harmful effects of ETS on nonsmokers increased significantly in the 1980s. Federal regulation of this public health problem has been minimal, although this situation may be changing.¹⁷² The lack of federal efforts to deal with this massive loss of life can be explained by the opposition to control of tobacco from the people involved in businesses related to the tobacco industry, including tobacco growers, manufacturers, distributors, and advertisers. A Price Waterhouse survey estimated that there were some 200,000 tobacco-growing and manufacturing jobs in 1990, in addition to 468,000 suppliers, wholesalers and retailers who depended on the industry.¹⁷³ These people are concerned that regulation of the tobacco industry would have serious economic repercussions for them; this business involved \$46.6 billion in consumer spending in 1996.¹⁷⁴ The tobacco industry's freedom from serious governmental control can perhaps also be explained by its generous contributions to Congress. Common Cause reported that tobacco industry contributions in 1995–1996 of just the “soft money,”

¹⁶⁹ ETS consists of mainstream (exhaled) and sidestream (emissions from a smoldering cigarette) smoke. See Spengler, *supra* note 153, at 41.

¹⁷⁰ Worldwide tobacco is responsible for 2.6 percent of the planet's disease burden. It is projected to increase to nine percent by the year 2020 based on increased smoking rates. This will make tobacco use the world's leading cause of disease. See Fred Hiatt, *The Smoking Gun—On Sale Overseas*, WASH. POST, May 26, 1997, at A19 (citing a ten volume study sponsored by the World Health Organization, the World Bank, and the Harvard School of Public Health authored by Christopher J.L. Murray and Alan D. Lopez).

¹⁷¹ AMERICAN CANCER SOC'Y, QUESTIONS ABOUT SMOKING, TOBACCO, AND HEALTH 13 (1996) [hereinafter QUESTIONS]. Tobacco use accounts for 30 percent of all cancer deaths in the United States and almost 180,000 Americans die from cardiovascular disease caused by smoking. See *id.* at 2.

¹⁷² As this Article was being completed, the newspapers, on nearly a daily basis, carried articles on the efforts of the Food and Drug Administration to regulate the nicotine content of cigarettes. See, e.g., Sandra Torry & John Schwartz, *Tobacco Negotiations Hit Stumbling Blocks; Lawsuit Restrictions, FDA Regulation at Issue*, WASH. POST, May 31, 1997, at A9; John Schwartz & Sandra Torry, *Tobacco Pact Calls for Strict Federal Controls*, WASH. POST, June 21, 1997, at A1.

¹⁷³ See Yumiko Ono, *Smoke Rings: If Cigarette Industry Coughs, Remote Areas Expect to Catch Cold*, WALL ST. J., Oct. 26, 1995, at A1.

¹⁷⁴ See Parker-Pope, *supra* note 42, at B1.

that is, money to be used for party activities, amounted to \$557,421 for the Democratic congressional committees and \$1,980,370 for the Republican congressional committees. The Philip Morris Corporation gave over \$1,088,770 to Republicans and \$430,268 to Democrats.¹⁷⁵

According to EPA, some 50 million citizens¹⁷⁶ smoke approximately 600 billion cigarettes, 4 billion cigars and 11 billion pipefuls of tobacco each year.¹⁷⁷ The burning of 20,000 tons of tobacco indoors each year¹⁷⁸ is believed to cause harmful health effects for nonsmokers forced to inhale ETS indoors. ETS contains the same harmful chemicals as the smoke that smokers inhale and may contain larger amounts of some cancer-causing substances.¹⁷⁹ ETS increases the concentration of some indoor air pollutants, such as Respirable Suspended Particles (RSP), benzene, acrolein, nitrogen oxides and carbon monoxide.¹⁸⁰

2. Health Effects of ETS

ETS is composed of irritating gases and carcinogenic tar particles, including the addictive drug nicotine, forty-three known or suspected carcinogens, and over 400 other toxins.¹⁸¹ Some of the toxic chemicals found in ETS are carbon monoxide, carbon dioxide, nicotine, carcinogenic tars, sulfur dioxide, ammonia, nitrogen dioxide, vinyl chloride, hydrogen cyanide, formaldehyde, radionuclides, benzene, nitrosamines, aromatic hydrocarbons, benzo[a]pyrene, and arsenic.¹⁸² These chemicals are released as the result of the incomplete burning of tobacco.¹⁸³ When a human is exposed to ETS, tar particles collect in the branching points of the lungs and the smaller particles collect in

¹⁷⁵ See Charles R. Babcock, *Parties' Congressional Campaign Committees Took in Millions in 'Soft Money' in 1995-96*, WASH. POST, Mar. 17, 1997, at A15.

¹⁷⁶ See QUESTIONS, *supra* note 171, at 10.

¹⁷⁷ See Michael K. Mahoney, Comment, *Coughing Up the Cash: Should Medicaid Provide for Independent State Recovery Against Third-Party Tortfeasors Such as the Tobacco Industry?*, 24 B.C. ENVTL. AFF. L. R. 233, 235 (1996).

¹⁷⁸ See Smith, *supra* note 9, at 8-9. This is a significant reduction from the 467,000 tons reported a few years earlier. See U.S. ENVTL. PROTECTION AGENCY, INDOOR AIR FACTS: ENVIRONMENTAL SMOKE, INDOOR AIR FACTS No. 5 (1989) [hereinafter ETS FACTS].

¹⁷⁹ See QUESTIONS, *supra* note 171, at 9-10.

¹⁸⁰ See Stanton A. Glantz & Richard A. Daynard, *Health Hazards of Secondhand Smoke*, 27 TRIAL, June 1991, at 38.

¹⁸¹ See QUESTIONS, *supra* note 171, at 4; see also ETS FACTS, *supra* note 178; EPA *Inside Story*, *supra* note 2. Burning tobacco smoke has been identified as the source of over 4,500 compounds, 50 of which are known or suspected carcinogens. See Spengler, *supra* note 153, at 41.

¹⁸² See ETS FACTS, *supra* note 178; see also QUESTIONS, *supra* note 171, at 4.

¹⁸³ See Harrison, *supra* note 13, at 298.

the air sacs.¹⁸⁴ Researchers have studied the level of cotinine¹⁸⁵ in the urinary content of both smokers and nonsmokers to determine the number of persons exposed to ETS. The results indicate that ETS affects nearly nine out of ten nonsmokers as measured by cotinine levels in the blood.¹⁸⁶ EPA has concluded that ETS is a Group A (known human) carcinogen.¹⁸⁷ EPA estimated in 1992 that as many as 3,000 nonsmokers die from lung cancer associated with ETS each year.¹⁸⁸

ETS is recognized as the cause of harmful health effects in both children and adults.¹⁸⁹ Children experience significant respiratory problems because of exposure to ETS, including chronic wheezing, coughing, and sputum production,¹⁹⁰ aggravation of asthmatic conditions,¹⁹¹ acute respiratory illness in early childhood,¹⁹² chronic middle ear infections; increased hospitalization rates for bronchitis and pneu-

¹⁸⁴ See Glantz & Daynard, *supra* note 180, at 37.

¹⁸⁵ In the body, nicotine breaks down into cotinine and can be detected and measured in saliva, blood, and urine. The presence of cotinine in nonsmokers indicates exposure to ETS. See ETS FACTS, *supra* note 178.

¹⁸⁶ See U.S. Dep't of Health and Human Services, HHS News, *Exposure to Second-Hand Smoke Widespread* (last modified Dec. 1996) <<http://www.cdc.gov/nchwww/releases/96news/nrsmoke.htm>>.

¹⁸⁷ See U.S. Env'tl. Protection Agency, *Secondhand Smoke* (last modified Apr. 21, 1997) <<http://www.epa.gov/iaq/pubs/etsbro.html>> at 1 of 5 [hereinafter *Secondhand Smoke*]. See also U.S. ENVTL. PROTECTION AGENCY, HEALTH EFFECTS OF PASSIVE SMOKING: ASSESSMENT OF LUNG CANCER IN ADULTS AND RESPIRATORY DISORDERS IN CHILDREN 1-3 (1990) [hereinafter PASSIVE SMOKING REPORT]. Although this document was released as a review draft, it was later approved by EPA's Science Advisory Board. See *SAB Gives Nod to EPA Passive Smoking Study, Paving Way for Regulation*, INSIDE EPA, May 3, 1991, at 14. In a settlement in 1997, Liggett Group, Inc., a major U.S. tobacco company, publicly acknowledged that cigarettes are addictive and cause cancer. See *Cigarette Firm to Acknowledge Cancer Link*, BALTIMORE SUN, Mar. 20, 1997, at 7A.

¹⁸⁸ See U.S. ENVTL. PROTECTION AGENCY, RESPIRATORY HEALTH EFFECTS OF PASSIVE SMOKING: LUNG CANCER AND OTHER DISORDERS 1 [hereinafter ETS REPORT]; see also Rep. No. 96-32, *supra* note 9.

¹⁸⁹ See Jonathan M. Samet et al., *Environmental Tobacco Smoke*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE, 131-32 (Jonathan M. Samet & John D. Spengler eds., 1991); see also *Secondhand Smoke*, *supra* note 187 (EPA estimates that ETS is responsible for between 150,000 and 300,000 lower respiratory tract infections in infants and children under 18 months annually).

¹⁹⁰ The children of smoking parents may experience a 20 to 80 percent increase in wheezing, coughing, and sputum production. See ETS FACTS, *supra* note 178.

¹⁹¹ See *Secondhand Smoke*, *supra* note 187 (between 200,000 and 1,000,000 asthmatic children's conditions are made worse by ETS).

¹⁹² Children whose parents smoke are more likely to suffer from pneumonia or bronchitis in the first two years of life than children who live in smoke-free households. See QUESTIONS, *supra* note 171, at 10.

monia;¹⁹³ reduced lung development; low birth weight; and decreased attained height.¹⁹⁴

For adults, an increased risk of lung cancer in nonsmokers is attributed to ETS.¹⁹⁵ Potential health effects also include increased lower respiratory infections, increased respiratory symptoms, reduced lung function, increased risk for and exacerbation of asthma, increased risk of cardiovascular disease and nonrespiratory cancers, and earlier onset of menopause.¹⁹⁶ ETS can cause irritation of the eyes, nose, throat, and lower respiratory tract; headaches; nausea; and dizziness.¹⁹⁷ Some research has indicated that exposure to ETS may cause brain tumors and cervical cancer.¹⁹⁸ Other health effects result from exposure during pregnancy and include harm to the fetus.¹⁹⁹ Individuals with pre-existing health conditions often notice exacerbated symptoms due to ETS exposure.²⁰⁰ Moreover, the cumulative effects of exposure to ETS and other workplace pollutants, such as asbestos, are of significant importance.²⁰¹ ETS constituents may interact with other chemical pollutants to yield increased adverse health effects.²⁰² Scientists have also demonstrated that exposure to radon and ETS creates a synergistic effect that may result in lung cancer.²⁰³

¹⁹³ See U.S. Envtl. Protection Agency, *Setting The Record Straight: Secondhand Smoke is A Preventable Health Risk* (last modified Apr. 3, 1997) <<http://www.epa.gov/iaq/pubs/strsfs.html>> [hereinafter *Setting the Record Straight*] (150,000 to 300,000 children under 18 months get these diseases from breathing ETS).

¹⁹⁴ See Harrison, *supra* note 13, at 298 (citing ETS FACTS, *supra* note 178).

¹⁹⁵ ISAAC TURIEL, *INDOOR AIR QUALITY AND HUMAN HEALTH* 75 (1985) (citation omitted).

¹⁹⁶ See Samet, *supra* note 189, at 132; see also ETS FACTS, *supra* note 178.

¹⁹⁷ See QUESTIONS, *supra* note 171, at 10.

¹⁹⁸ See generally James L. Repace & Alfred H. Lowery, *Predicting the Lung Cancer Risk of Domestic Passive Smoking*, 136 AM. REV. RESPIRATORY DISEASE 1308 (1987) (citing Takeshi Hirayama, *Cancer Mortality in Nonsmoking Women With Smoking Husbands Based on a Large-Scale Cohort Study in Japan*, 13 PREVENTIVE MED. 680 (1984)). The studies were cited by Glantz & Daynard, *supra* note 180, at 38.

¹⁹⁹ See Glantz & Daynard, *supra* note 180, at 38 (citing Esther M. John et al., *Prenatal Exposure to Parents' Smoking and Childhood Cancer*, 133 AM. J. EPIDEMIOLOGY 123 (1991)); see also Samet, *supra* note 189, at 149. Several studies established a link between parental smoking and sudden infant death syndrome—children of smoking parents have a twofold increased risk of dying. See QUESTIONS, *supra* note 171, at 10.

²⁰⁰ See REITZE, *supra* note 33, at 398.

²⁰¹ See Glantz & Daynard, *supra* note 180, at 38 (citing WORLD HEALTH ORGANIZATION, *WORLD NO-TOBACCO DAY 7* (1991)).

²⁰² See Harrison, *supra* note 13, at 299-300.

²⁰³ See Glantz & Daynard, *supra* note 180, at 38 (citing Olav Axelsson et al., *Indoor Radon Exposure and Active and Passive Smoking in Relation to the Occurrence of Lung Cancer*, 14 SCANDINAVIAN J. WORK ENV'T & HEALTH 286 (1988)).

3. Lung Cancer

The Surgeon General and the National Academy of Sciences concluded in a 1986 report that smoking may cause lung cancer in both smokers and nonsmokers.²⁰⁴ Today, smoking is considered to be responsible for about eighty-three percent of lung cancers.²⁰⁵ Nonsmokers exposed to ETS for many years are susceptible to lung cancer and other cancers. EPA concluded in its 1990 draft report on the health effects of ETS that "passive smoking is causally associated with lung cancer in adults."²⁰⁶ According to the National Research Council, the risk of lung cancer for spouses of smoking partners is thirty percent greater than for spouses of nonsmoking partners.²⁰⁷ EPA reported the results of several studies involving nonsmokers and individuals exposed to ETS that estimated annual lung cancer death rates in passive smokers to range from twelve to 5,200; however, the majority of estimates fell between 2,500 and 5,200.²⁰⁸ EPA, in its 1992 report on the effects of ETS, attributed to ETS 1,500 lung cancer deaths per year in women who have never smoked, 500 lung cancer deaths per year in men who have never smoked, and 1,000 lung cancer deaths per year in former smokers of both sexes.²⁰⁹ Two independent studies completed since the publication of EPA's report support at least some of EPA's findings concerning the increased risk of lung cancer due to exposure to ETS.²¹⁰ Some evidence suggests that ETS may increase

²⁰⁴ See W. Allen Crawford, *Air Pollution, Environmental Tobacco Smoke and Lung Cancer*, 38 J. AIR & WASTE MGMT. ASS'N 1386, 1389 (1988); (quoting U.S. SURGEON GEN., PUB. HEALTH SERVICE, U.S. DEP'T OF HEALTH & HUMAN SERVICES, *THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING: A REPORT OF THE SURGEON GENERAL* 13, 96 (1986)); see also NATIONAL RESEARCH COUNCIL, *ENVIRONMENTAL TOBACCO SMOKE* (1986).

Note that just one year earlier, the Surgeon General only mentioned passive smoking briefly in his 1985 report on "Smoking in the Workplace." As a result, he suffered strong criticism from the AFL/CIO. See Crawford, *supra*.

²⁰⁵ See QUESTIONS, *supra* note 171, at 2.

²⁰⁶ See ETS FACTS, *supra* note 178.

²⁰⁷ See QUESTIONS, *supra* note 171, at 10; see also ETS FACTS, *supra* note 178; *Setting the Record Straight*, *supra* note 193.

²⁰⁸ See REITZE, *supra* note 33, at 399 (citing Repace & Lowery, *supra* note 198 (estimating 4,700 lung cancer deaths per year in lifelong nonsmokers and ex-smokers aged 35 years or older)). These and other studies were cited in REITZE, *supra* note 33, at 399 (citing EPA REPORT, *supra* note 13, at 4-20 to 4-23); see also Secondhand Smoke, *supra* note 187 (estimating 3,000 cancer deaths in nonsmokers each year from ETS).

²⁰⁹ See ETS REPORT, *supra* note 188.

²¹⁰ See generally Ross C. Brownson et al., *Passive Smoking and Lung Cancer in Nonsmoking Women*, 82 AM. J. PUB. HEALTH 1525 (1992) (claiming that long-term exposure to ETS increased the risk of lung cancer in women who never smoked).

the chance of cancer at other sites in the body, including brain tumors and risk of childhood rhabdomyosarcoma.²¹¹

The tobacco industry, however, is doing its best to limit the damage caused by the public uproar over ETS. One of the industry's major efforts in the realm of ETS has been its attempt to discredit EPA's 1992 report on passive smoking.²¹² To that end, the tobacco industry filed suit against EPA; EPA's attempt to have the suit dismissed did not succeed.²¹³ In its suit, the tobacco industry claimed that EPA violated the Radon Gas and Indoor Air Research Act of 1986, the Administrative Procedure Act, and the guarantee of due process provided by the Fifth Amendment of the Constitution by publishing the 1992 report.²¹⁴

Notwithstanding limited success against EPA, the tobacco industry recently suffered a major defeat. On April 25, 1997, federal court Judge Jim Osteen ruled in *Coyne Beahm v. FDA*,²¹⁵ that the FDA may regulate tobacco products as drugs.²¹⁶ Although the industry has appealed the decision, it faces serious obstacles in having the decision overturned.²¹⁷ If the ruling is upheld, the tobacco industry will be subject to FDA's authority to direct almost every aspect of tobacco production.²¹⁸

4. Heart Disease

ETS aggravates, and possibly causes, heart disease.²¹⁹ The evidence linking ETS to heart disease was based on thirteen epidemiological studies indicating a thirty percent increase in the risk of death due to heart disease in nonsmokers exposed to ETS compared to nonsmokers not exposed to ETS.²²⁰ ETS is responsible for other physiological

²¹¹ See Samet, *supra* note 189, at 149.

²¹² See generally Steven Bayard & Jennifer Jinot, *Environmental Tobacco Smoke: Industry's Suit*, 19 EPA J., Oct.-Dec. 1993, at 20 (EPA defending its report).

²¹³ See *Flue Cured Tobacco Coop. Stabilization Corp. v. EPA*, 857 F. Supp. 1137, 1145 (M.D.N.C. 1994).

²¹⁴ See *id.* at 1140.

²¹⁵ See generally *Coyne Beahm, Inc. v. FDA*, 958 F. Supp. 1060 (M.D.N.C. 1997).

²¹⁶ See *id.* at 1084. However, the court also said that FDA cannot regulate cigarette advertising and promotions. See *id.*

²¹⁷ See Bob Van Voris, *Tobacco: And Now the Appeal*, NAT'L L.J., May 12, 1997, at A1.

²¹⁸ See *id.*

²¹⁹ See ETS FACTS, *supra* note 178.

²²⁰ See Glantz & Daynard, *supra* note 180, at 40 (citing Martha L. Slattery et al., *Cigarette Smoking and Exposure to Passive Smoke Are Risk Factors for Cervical Cancer*, 261 JAMA 1593, 1593-98 (1989)).

effects that increase the risk of heart disease, including impairment of platelet function, damage to the inside lining of arteries, and interference with oxygen delivery and use by cells.²²¹ ETS may also depress cellular respiration and enhance fatty deposits on vessel walls.²²²

The most important research effort may be the study of 32,000 nurses by Ichiro Kawachi, of the Harvard Medical School, and his colleagues.²²³ This long-term study, released on May 20, 1997, found that high exposure to secondhand smoke produces a relative risk of coronary heart disease of 1.91 compared to nonsmoking women.²²⁴ This near doubling of the risk of heart disease is estimated to translate into 60,000 additional deaths each year in the United States that are attributable to ETS.²²⁵ This estimate is twenty times the deaths from lung cancer attributable to ETS as stated in the controversial 1992 passive smoking report.²²⁶

5. Control of ETS

The number of smokers and pattern of smoking determine ETS concentrations. Short-term concentrations may be very high in bars, restaurants, automobiles, and homes.²²⁷ Physical separation of smokers in common air space reduces, but does not eliminate, exposure to ETS.²²⁸ Ventilation also reduces, without eliminating, the presence of ETS but cannot control ETS to within an acceptable level of risk.²²⁹ Generally, ventilation techniques are insufficient to remove the pollutants that build up in buildings where smoking is allowed.²³⁰ Smoking may be limited to smoking areas only; however, the successful exclusion of ETS from other areas of a home or a building requires

²²¹ See Glantz & Daynard, *supra* note 180, at 40.

²²² See *id.*

²²³ See John Schwartz, *Secondhand Smoke Linked to Increased Heart Attack Rate*, WASH. POST, May 20, 1997, at A2.

²²⁴ See *id.*

²²⁵ See *id.*

²²⁶ See ETS REPORT, *supra* note 188.

²²⁷ See Spengler, *supra* note 153, at 44.

²²⁸ See EPA Inside Story, *supra* note 2.

²²⁹ See generally James L. Repace & Alfred H. Lowrey, *An Indoor Air Quality Standard for Ambient Tobacco Smoke Based on Carcinogenic Risk*, 85 N.Y. STATE J. OF MED. 381 (1985).

²³⁰ See EPA Inside Story, *supra* note 2; see also Edward R. Lipinski, *Breathe Easy*, CHI. TRIB., Feb. 1, 1997, at 13 (running a number of large fans also can increase energy bills). The 1981 ASHRAE standards for indoor air quality recommend introduction of five cubic feet of outside air per minute per occupant (cfm/occ) in smoke-free office buildings and 20 cfm/occ where smoking is permitted in order to control only the odor from tobacco smoke. See ETS FACTS, *supra* note 178.

effective confinement through depressurization and direct venting to the outside.

The only completely effective means of eliminating ETS from the indoor environment is to prohibit smoking indoors.²³¹ There are many state and local smoking laws and ordinances that restrict or prohibit smoking in public places.²³² Gradually, the U.S. is taking action to prevent this form of indoor air pollution.²³³ In 1993, the Smoke-Free Environment Act of 1993²³⁴ was proposed, but not enacted. This would have prohibited smoking in most workplaces.²³⁵ In May 1994, Mississippi filed the first lawsuit seeking reimbursement for smoking-related claims paid by Medicaid.²³⁶ In 1994, a law was passed authorizing the state of Florida to sue cigarette manufacturers to recover Medicaid funds used to treat persons with tobacco-related illnesses.²³⁷ This law was upheld by the Florida Supreme Court.²³⁸ As of June 1997, thirty states had sued the tobacco industry to recover the Medicaid costs of treating smoking-related diseases.²³⁹ Negotiations were being conducted in an effort to reach a settlement, but the issues of private tort liability involving class actions in seventeen states, as well as the desire by the tobacco industry to have an agreement with the Food and Drug Administration concerning its regulatory efforts, were hampering settlement.²⁴⁰

²³¹ The right of nonsmokers to a smoke-free environment was discussed as early as 1883. See David Dudley Field, *The Smoke-Nuisance*, in 2 SPEECHES, ARGUMENTS AND MISCELLANEOUS PAPERS OF DAVID DUDLEY FIELD 285 (A.P. Sprague ed., 1884).

²³² See 41 C.F.R. § 101-20.105-3 (1997); QUESTIONS, *supra* note 171, at 11. Today, laws in 49 States and the District of Columbia restrict or prohibit smoking in public places. QUESTIONS, *supra* note 171, at 11. Title 41 of the C.F.R. currently regulates smoking in public places. See 41 C.F.R. § 101-20.105-3.

²³³ See Fran Du Melle, *Laws Protecting Nonsmokers*, 19 EPA J., Oct.-Dec. 1993, at 21.

²³⁴ H.R. 3434, 103rd Cong. (1993).

²³⁵ *Id.*

²³⁶ See Mark Curriden, *Litigants Talk Tobacco: Immunity Among Issues that Could Hinder Settlement*, A.B.A. J., June 1997, at 20.

²³⁷ See *U.S. Supreme Court Will Not Review Ruling Allowing Florida to Sue Cigarette Firms*, 11 Toxics L. Rep. (BNA) at 1143 (Mar. 19, 1997).

²³⁸ See *Agency for Health Care Admin. v. Associated Indus. of Fla.*, 678 So.2d 1239, 1257 (Fla. 1996).

²³⁹ See *States News Briefs, Florida*, June 11, 1997, available in LEXIS, Allnws Library, CURNWS File.

²⁴⁰ The nation's major tobacco companies agreed on June 20, 1997, to submit to federal control of cigarettes. See 12 Toxics L. Rep. (BNA) at 105 (June 25, 1997); see, e.g., John Schwartz & Sandra Torry, *Tobacco Pact Calls for Strict Federal Controls*, WASH. POST, June 21, 1997, at A1. Seven individuals have been described as "essential" to providing the ideas, evidence, and strategies that changed the balance of power between cigarette companies and their antagonists which eventually led to this settlement. See Alix M. Freedman & Suein L. Hwang, *Leaders of*

Many fast food chains now ban smoking in their restaurants.²⁴¹ Many federal worksites, including the White House, are smoke-free.²⁴² Congress has banned smoking on all domestic airplane flights of six hours or less.²⁴³ OSHA proposed a ban in 1994 on smoking in open areas at every workplace.²⁴⁴ OSHA has been sued unsuccessfully six times in an effort to force it to regulate tobacco smoke in the workplace.²⁴⁵ In the most recent case, the U.S. Court of Appeals for the District of Columbia Circuit held that the timetable for regulating tobacco smoke set out in EPA's 1980 Cancer Policy was discretionary.²⁴⁶ As of June 1997, OSHA has not issued a final rule concerning ETS.²⁴⁷

The Americans with Disabilities Act (ADA) may provide a method for limiting or eliminating ETS in the workplace.²⁴⁸ The ADA requires that an employer make a reasonable accommodation to the known disabilities of an employee who requests such an accommodation.²⁴⁹ Thus, individuals who suffer from respiratory conditions or illnesses that limit their ability to breathe may request an accommodation that would reduce ETS exposure in the workplace.²⁵⁰ In one case,²⁵¹ the

the Pact; How Seven Individuals With Diverse Motives Halted Tobacco's Wars, WALL ST. J., July 11, 1997, at A1, A8. The seven are: Jeffrey Nesbit, Michael T. Lewis, Jeffrey Wigand, Walt Bogdanich, Bennett LeBow, Dick Morris, and Grady Carter. *See id.* The settlement may be impacted by the U.S. Supreme Court's decision in which the Court upheld a Third Circuit decision rejecting a \$1.3 billion settlement involving claims of asbestos-related injuries. *See Amchem Prods. v. Windsor*, 117 S. Ct. 2231, 2252 (1997).

²⁴¹ *See* Kopon & Gergits, *supra* note 10, at 48.

²⁴² *See* QUESTIONS, *supra* note 171, at 11.

²⁴³ 49 U.S.C. § 41706 (1994).

²⁴⁴ *See* Indoor Air Quality, 59 Fed. Reg. 15,968 (1994) (to be codified at 29 C.F.R. pts. 1910, 1915, 1926, 1928).

²⁴⁵ *See Federal Appeals Court Again Declines To Force OSHA To Regulate Workplace Smoke*, 11 Toxics L. Rep. (BNA) at 774 (Dec. 11, 1996).

²⁴⁶ *See* Action on Smoking and Health v. Department of Labor, 100 F.3d 991, 994 (D.C. Cir. 1996).

²⁴⁷ *See* Unified Agenda, Long Term Actions, 62 Fed. Reg. 21,976, 21,978 (1997); Semiannual Agenda of Regulations, 2 Fed. Reg. 21,934 (1997).

²⁴⁸ *See* Adam T. Klien, *Litigating Tobacco Cases Pursuant to the Americans With Disabilities Act of 1990*, 10 Mealey's Litig. Rep.: Tobacco at 1 (Nov. 1, 1996).

²⁴⁹ *See id.*

²⁵⁰ *See id.*

²⁵¹ *See generally* Staron v. McDonalds Corp. and Burger King Corp., 51 F.3d 353 (2d Cir. 1995); *see also* Muller v. Costello, No. 94-CV-842, 1996 WL 191977 (N.D.N.Y. Apr. 16, 1996). *But see generally* Bell v. Elmhurst Chicago Stone Co., 919 F. Supp. 308 (N.D. Ill. 1996), *def.'s mot. for summ. j. denied* 957 F. Supp. 1025 (N.D. Ill. 1997) (employer cannot be charged with battery for allowing other people in the workplace to smoke); Homeyer v. Stanley Tulchin Assocs., Inc., 91 F.3d 959 (7th Cir. 1996) (dismissal of suit under ADA for exposure to tobacco smoke).

Court of Appeals for the Second Circuit held that a complete ban on smoking is a reasonable modification and not an undue hardship on an employer.²⁵² However, a total ban on smoking may not be required by the ADA if a court determines that there are other sufficient measures to protect employees that are less burdensome on an employer.²⁵³

While outside the scope of this Article, an important case concerning ETS began in early June 1997. Because it involved nonsmoking airline flight attendants, the tobacco industry was not able to use the "freedom-of-choice" defense. Thirty named plaintiffs and a class of 60,000 present and former flight attendants, who never smoked, sued the major U.S. cigarette manufacturers in the Florida Circuit Court in Miami. The case is important because it settled when the four cigarette manufacturers agreed to pay \$300 million to fund scientific research into smoking-related diseases.²⁵⁴

B. Radon

1. Sources of Radon

Radon is a colorless, odorless, tasteless, radioactive gas, which human senses cannot detect.²⁵⁵ It is produced by the natural decay, in several steps, of uranium-238.²⁵⁶ Radon is the immediate decay product of radium-226.²⁵⁷ Soil and rock are the major sources of radioactive material that produce radon because radon gas migrates through soil

²⁵² See Klien, *supra* note 248.

²⁵³ See *id.*; see also *Harmer v. Virginia Elec. & Power Co.*, 831 F. Supp. 1300, 1306 (E.D. Va. 1993); *Vickers v. Veterans Admin.*, 549 F. Supp. 85, 89 (W.D. Wash. 1982).

²⁵⁴ See Donald P. Baker, *Nonsmoker's Suit Presents New Challenge for Industry*, WASH. POST, May 31, 1997, at A3; see also *Four Cigarette Makers To Pay \$300 Million for Research In Deal With Flight Attendants*, 12 TOXICS L. REP. (BNA) at 551 (Oct. 15, 1997) (citing *Broin v. Philip Morris Cos.*, No. 91-49738 CA (22) (Fla. Cir. Ct.) (settlement Oct. 10, 1997)).

²⁵⁵ See REITZE, *supra* note 33, at 402 (citing U.S. GEN. ACCOUNTING OFFICE, REPORT TO THE PENNSYLVANIA CONGRESSIONAL DELEGATION, HOUSE OF REPRESENTATIVES, AIR POLLUTION: HAZARDS OF INDOOR RADON COULD POSE A NATIONAL HEALTH PROBLEM 11 (1986) [hereinafter U.S. GAO]).

²⁵⁶ See generally U.S. ENVTL. PROTECTION AGENCY, RADON ENVIRONMENTAL BACKGROUND (1989) [hereinafter RADON]; U.S. ENVTL. PROTECTION AGENCY & U.S. DEP'T OF HEALTH AND HUMAN SERVICES, A CITIZEN'S GUIDE TO RADON: WHAT IS IT AND WHAT TO DO ABOUT IT (1986) [hereinafter EPA CITIZEN'S GUIDE]; Janet I. Moore, *The Radon Review: The Federal and State Governments' Responses to Indoor Radon Contamination*, 7 TEMP. ENVTL. L. & TECH. J. 39, 39 (1988).

²⁵⁷ See David Dearing, *Radon Litigation: An Even View of Homeowner's Potential Causes of Action*, 20 CUMB. L. REV. 825, 826 n.9 (1990) (citing Carolyn M. Shuko, *Radon Gas: Contractor Liability for an Indoor Health Hazard*, 12 AM. J.L. & MED. 241, 242 (1986)). Uranium-238 decays

and rock, and enters buildings through cracks in the foundations, gaps around drainage pipes, and other openings.²⁵⁸ Outside, radon is diluted in the atmosphere; however, radon that enters buildings can accumulate to high concentrations.²⁵⁹ Water passing through soil and rock absorbs radon. If water contains high radon levels, exposure occurs directly by drinking the water, as well as from the release of radon into the air, especially if the water temperature is elevated as during showering, doing laundry, boiling water, or running the dishwasher.²⁶⁰

Several factors determine the amount of radon that enters a building from the soil. These include the amount of radium present in the soil beneath the building,²⁶¹ soil permeability, the type of building construction, the condition of the building foundation, and the airtight quality of the building.²⁶² Some building materials, including bricks and concrete, emit radon.²⁶³ Additionally, radon may enter buildings through the ground water.²⁶⁴ Natural gas is another source of indoor

in several steps over a period of four billion years into radium-226. Radium 226 decays with a half-life of 1620 years to radon 222. Radon 222 decays with a half-life of about 3.82 days to polonium-218 and then decays to lead-214, bismuth-214, polonium-214 and finally to lead-206, a stable element. During the decay process, radiant energy is released until the elements reach lead-206. The tiny particles of these elements attach to dust particles or smoke particles, enter and lodge in the lungs and continue to decay. These particles are referred to as "radon daughters." See Jonathan M. Samet, *Radon, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE* 323 (Jonathan M. Samet & John D. Spengler eds., 1991). Radon's isotopes, 219-Rn, 220-Rn, and 222-Rn, are the most dangerous, and each emits alpha particles during their half-life of 3.82 days.

²⁵⁸ See REITZE, *supra* note 33, at 402; RADON, *supra* note 256, at 2.

²⁵⁹ See RADON, *supra* note 256, at 2.

²⁶⁰ See U.S. GAO, *supra* note 255, at 11; Dearing, *supra* note 257, at 825-26. When water is at room temperature, as much as 30 percent of the radon can diffuse into indoor air. Note that generally 10,000 pCi/l of water results in diffusion of 1 pCi/l of air in normal household water usage. Of all secondary sources of radon, groundwater is the most significant. See C. Richard Cothorn, *Radon in Drinking Water*, WATER WORKS ASSOC. J., Aug. 1, 1986, at 7 [hereinafter Cothorn I]. Approximately one to seven percent of the fatalities due to radon exposure are the result of radon released from drinking water sources. See generally C. Richard Cothorn et al., *Drinking Water Contribution to Natural Background*, 50 HEALTH PHYSICS 33 (1986).

²⁶¹ Homes built on granitic rock ledges containing radium are more likely to test higher. See DAVIS & SCHAFFMAN, *supra* note 4, at 145. For example, in Connecticut, many homes tested at 20 pCi/l or higher. See *id.*

²⁶² See Harrison, *supra* note 13, at 303 (citing U.S. GAO, *supra* note 255, at 11). The presence of a home on the soil creates a pressure gradient and therefore an upward driving pressure of gas. See Spengler, *supra* note 153, at 55.

²⁶³ See ISAAC TURIEL, *INDOOR AIR QUALITY AND HUMAN HEALTH* 34 (1985). These building materials may contain large amounts of radioactive substances. See *id.* Examples include phosphate slag used as aggregate for concrete foundations in homes in Idaho from 1962 to 1977 and concrete containing radium in Sweden. See *id.*

²⁶⁴ See RADON, *supra* note 256, at 2. Radon in water sources is not a problem where surface water is used as the drinking water source. See *id.*

radon, although its combustion is considered a minor contributor.²⁶⁵ Other sources include emanation from oceans, phosphate residues, uranium tailings, coal residues, and coal combustion.²⁶⁶

In the 1930s, radon's role in lung cancer was identified for those suffering from "miner's disease," a disease that was first recognized in central Europe during the late 1500s.²⁶⁷ Epidemiology studies were first conducted on miners in the 1950s in the United States.²⁶⁸ As a result of an Atomic Energy Commission (AEC) study at a mine in Grand Junction, Colorado, the AEC set an occupational health standard for radon of 100 pico curies per liter (pCi/l) of air; the International Commission on Radiological Protection set the standard at 10 pCi/l of air.²⁶⁹ Radon did not become a concern for the population-at-large until the 1960s when it was discovered that homes had been built atop wastes from uranium mines (uranium mill tailings).²⁷⁰ These homes had significantly elevated levels of radon in the indoor environment.²⁷¹

Radon is recognized today as a significant indoor health threat.²⁷² Congress established a goal of reducing indoor radon to the level of outdoor radon.²⁷³ Almost all American homes contain at least some radon.²⁷⁴ EPA estimated that up to 6 million American homes have annual average radon levels above four pCi/l of air,²⁷⁵ which is EPA's

²⁶⁵ See U.S. GAO, *supra* note 255, at 11.

²⁶⁶ See Cothorn I, *supra* note 260, at 7.

²⁶⁷ See David J. Hanson, *Radon Tagged as Cancer Hazard by Most Studies, Researchers*, 67 CHEM. & ENGINEERING NEWS, Feb. 6, 1989, at 7.

²⁶⁸ See REITZE, *supra* note 33, at 403.

²⁶⁹ *Id.* "Pico curies per liter is a measure of radon decompositions (a measure of radioactivity) occurring per second per liter of air." *Id.*

²⁷⁰ See *id.* at 403-04.

²⁷¹ See *id.* at 404 (citing Hanson, *supra* note 267, at 7). "Later, a similar increase in radon levels was found in houses built on other land reclaimed from mining operations, particularly phosphate mines in Florida." REITZE, *supra* note 33, at 404 n.88 (quoting Hanson, *supra* note 267, at 7).

²⁷² See *id.* at 404 (citing John H. Harley, *Radioactive Emissions and Radon*, 57 BULL. N.Y. ACAD. MED. 883 (1981)). "The dose to the bronchial epithelium delivered by the alpha-emitting daughters of radon-222 is the highest radiation dose received by man from natural sources." *Id.* at 404 n.89 (quoting Harley, *supra*, at 883).

²⁷³ See *id.* (citing Harley, *supra* note 272, at 883).

²⁷⁴ See *id.*

²⁷⁵ See Model Standards and Techniques for Control of Radon in New Residential Buildings, 59 Fed. Reg. 13,402 (1994). Average annual indoor levels range from one to two pCi/l. Outdoor levels average below one. See *id.* If radon screening measurements exceed four pCi/l, but not 20 pCi/l, EPA recommends follow-up measurements within 12 months in the general living areas of the house. If the measurements originally exceeded 20 pCi/l, EPA recommends a more intensive follow-up. See REITZE, *supra* note 33, at 404 n.91 (citing EPA REPORT, *supra* note 13, at 2-17).

"action level."²⁷⁶ EPA reported in 1989 that twenty-six percent of the homes it had tested had radon levels above the "action level," which is a risk level for lung cancer of two percent/lifetime.²⁷⁷

2. Health Effects of Radon

The primary health effect of radon is the increased risk of developing lung cancer.²⁷⁸ Radon is second only to smoking as a cause of lung cancer.²⁷⁹ "Of the indoor air pollutants in many homes, radon is currently the one posing the greatest threat to health."²⁸⁰ EPA estimated that 7,000 to 30,000 lung cancer deaths in the United States each year may be attributable to radon exposure.²⁸¹ Risk is dependent upon radon concentration and duration of exposure.²⁸² Risk increases as radon concentration and length of exposure increase.²⁸³

Even low-level exposure to radon for extended periods of time can cause adverse health effects due to the effective delivery of alpha particles into the body.²⁸⁴ Radon gas is inhaled and exhaled. However, the radon progeny can become lodged in the lungs where they continue emitting radiation.²⁸⁵ Moreover, unattached radon progeny have deposition frequencies of 100 percent, whereas a much smaller per-

²⁷⁶ See Model Standards and Techniques for Control of Radon in New Residential Buildings, 59 Fed. Reg. 13,402.

²⁷⁷ See OFFICE OF AIR & RADIATION, U.S. ENVTL. PROTECTION AGENCY, SURVEY RESULTS AND RECOMMENDATIONS 6 (1989). See also Interview with Richard Cothorn, EPA employee (July 16, 1997).

²⁷⁸ See U.S. GAO, *supra* note 255, at 18. Radon is classified as a group "A" carcinogen. See Karen Hoyden Curtin, *Indoor Radon: A Blameless Cause*, 3 BUFF. ENVTL. L.J. 181, 191 (1995); see also National Primary Drinking Water Regulations, Radionuclides, 56 Fed. Reg. 33,050 (1991) (codified at 40 C.F.R. pts. 141, 142).

²⁷⁹ See RADON, *supra* note 256, at 2; see also Med Access, *Indoor Air Pollution: An Introduction for Health Professionals, Two Long-Term Risks: Asbestos and Radon* (visited July 2, 1997) <http://www.medaccess.com/environ/ind_air/prof_09.htm>. Radon gas seeping into homes causes 14,000 lung cancer deaths per year. See Henry A. Waxman, *The View From Congress*, 19 EPA J., Oct.-Dec. 1993, at 38.

²⁸⁰ Crawford, *supra* note 204, at 1389.

²⁸¹ See Rep. No. 96-32, *supra* note 9.

²⁸² See RADON, *supra* note 256, at 1.

²⁸³ See *id.* The highest level of radon occurred in 1984 in Pennsylvania where a home had a radon level of 2,700 pCi/l, equal to smoking about two hundred packs of cigarettes a day. See DAVIS & SCHAFFMAN, *supra* note 4, at 145.

²⁸⁴ See Hanson, *supra* note 267, at 9.

²⁸⁵ See *id.* Radon progeny, not the gas itself, cause injury because radon gas does not remain in the lungs for a sufficient time. See Cothorn I, *supra* note 260, at 13.

centage of radon progeny attached to dust particles remain in the lungs.²⁸⁶

Extrapolating the results of mining studies to lower radon exposure levels, most researchers agree that radon poses a health risk even at low levels.²⁸⁷ Excess lung cancers were detected among miners in at least fifteen studies conducted in the United States, Canada, and other countries.²⁸⁸ One study involving 4,146 uranium miners exposed to radon found 173 lung cancer deaths above normal after eliminating lung cancers attributable to smoking.²⁸⁹ Researchers have estimated that the risk of lung cancer in the general population attributable to radon exposure is from one to five percent to ten to forty percent.²⁹⁰ One estimate attributed forty percent of lung cancers in nonsmoking women to their lifetime exposure to radon.²⁹¹

Persons exposed to one working level month of radon have an increased risk of developing lung cancer equal to 1.5 times that of persons only exposed to background levels.²⁹² Studies estimate 130 to 730 additional annual lung cancer deaths per million occur to individuals exposed to one working level month.²⁹³ Calculations using EPA's risk assessment model indicate that "radon exposure in single-family homes may cause twenty thousand lung cancer deaths annually in the

²⁸⁶ See Cothorn I, *supra* note 260, at 13.

²⁸⁷ However, at least one researcher has found that household concentrations of 1.5 pCi/l creates about the same risk of lung cancer for individuals as that of having an accident in the home. See Hanson, *supra* note 267, at 13 (statement of Anthony V. Nero, who researches for the Indoor Environment Program at Lawrence Berkeley Laboratory of the University of California, Berkeley). Allegedly, excess risk occurs when household levels are 20 pCi/l or higher. See *id.* Exposure levels of 10–20 pCi/l over a lifetime can equal exposure detected where miners were found to have high cancer rates. See *id.*

²⁸⁸ See U.S. GAO, *supra* note 255, at 18.

²⁸⁹ See *id.*

²⁹⁰ See Crawford, *supra* note 204, at 1390. The difference seems to be due to the uncertainty of the compounding factor of smoking. See Cothorn Interview, *supra* note 277.

²⁹¹ See Edward P. Radford, *Radon Daughters and Lung Cancer*, 313 N. ENG. J. MED. 1610, 1610–11 (1985).

²⁹² See *Radon Cancer Risk Greater Than Thought, Especially for Smokers, NAS Study Says*, 18 Env't Rep. (BNA) No. 37, at 1997 (Jan. 8, 1988) [hereinafter *Radon Cancer Risk*]. A working level month (WLM) is the amount of exposure equivalent to working 170 hours in an environment with 100 pCi/l of air. See Samet, *supra* note 257, at 325.

²⁹³ See *Radon Cancer Risk*, *supra* note 292, at 1997. Excess lung cancers have been attributed to lifetime exposures of radon at 80 WLM (1 WLM = 200 pCi/l). One study found excess lung cancers due to lifetime exposures as low as 40 WLM. See U.S. GAO, *supra* note 255, at 18. Researchers must be careful in comparing data from studies on miners to the exposure incurred in the home environment because the same level of exposure in each environment may not lead to identical dosages of alpha radiation to the lungs. See Samet, *supra* note 257, at 329.

United States.”²⁹⁴ One EPA survey of 130 schools in sixteen states showed that in nineteen percent of the classrooms there were radiation levels of more than four pCi/l and in three percent there were levels in excess of twenty pCi/l.²⁹⁵

The National Academy of Sciences stated that cigarette smokers exposed to radon are ten times more likely to die from lung cancer due to the synergistic effects of radon and smoking.²⁹⁶ Although the results of studies are inconsistent, the effects of combined exposure to radon and smoking appear to be synergistic and multiplicative.²⁹⁷ Several studies reviewed by Jonathan M. Samet showed an association between exposure to radon and incidence of, or mortality from, lung cancer.²⁹⁸

3. Control of Radon

The National Academy of Sciences believes that the cancer risk from radon decreases after exposure has ceased; risk declines by fifty percent about fifteen years after exposure is terminated.²⁹⁹ Thus, remediation efforts to reduce radon exposure are beneficial. Control of radon involves sealing cracks and other openings in slabs, basement walls and floors; ventilating crawl spaces; installing sub-slab ventilation systems or heat recovery ventilators (air-to-air heat exchangers); and treating radon-contaminated well water through aeration or filtration using a granulated-activated charcoal filter.³⁰⁰ Other treatment methods include basement pressurization and application of suction to drain tiles.³⁰¹ Combinations of methods of control are often

²⁹⁴ Model Standards and Techniques for Control of Radon in New Residential Buildings, 59 Fed. Reg. 13,402 (1994).

²⁹⁵ See Michael Weisskopf, *EPA Finds Excessive Radon In Schools, Urges Nationwide Tests*, WASH. POST, Apr. 21, 1989, at A3.

²⁹⁶ See *Radon Cancer Risk*, *supra* note 292, at 1997. When two agents are considered synergistic, there is an increased effect when the agents are both present, thus the combined effect exceeds the sum of the independent effects. See Samet, *supra* note 257, at 333. Note, however, that these analyses are primarily based on epidemiologic studies of underground miners. See *id.* at 334.

²⁹⁷ See Samet, *supra* note 257, at 333–34. When the interaction of two stimuli is multiplicative, the combined effect of the stimuli equals the product of their independent effects. See *id.* at 333.

²⁹⁸ See *id.* at 335–37.

²⁹⁹ See *Radon Cancer Risk*, *supra* note 292, at 1998.

³⁰⁰ See REITZE, *supra* note 33, at 406 (citing EPA REPORT, *supra* note 13, at 6–4).

³⁰¹ *Id.* at 406–07 (citing EPA REPORT, *supra* note 13, at 6–5). The most effective method of radon removal is subslab ventilation or depressurization. In this method, a contractor inserts pipes through the concrete basement slab. Radon is pulled through the pipes by a fan and vented directly outdoors. EPA says that the method can cut radon concentrations by 90% or more.

used. The appropriate mitigation technique and the degree of success may fluctuate by season.³⁰² Generally, mitigation work is expensive, although in some cases a simple basement fan may be sufficient.³⁰³

Radon can be removed from water by carbon filtration through Granulated-Activated Charcoal ("GAC") treatment or by aeration.³⁰⁴ GAC treatment, which filters radon-bearing water through a special charcoal filter, is about eighty-five to ninety-nine percent efficient and is less costly than aeration.³⁰⁵ Aeration systems, which force radon gas from the water by spraying or using air bubbles to vent it outside the home, can achieve ninety-five to ninety-nine percent radon reduction.³⁰⁶

Radon measurements should be taken to evaluate radon concentrations in the home. Measurements of four pCi/l or higher (the EPA "action level") should be followed by additional testing to assure an accurate evaluation and, if necessary, mitigation measures designed to reduce the level of radon in the home. Radon testing can be performed by professionals or by homeowners who purchase radon detection kits and later have the devices examined by a laboratory.³⁰⁷ If

Hanson, *supra* note 267, at 10-11. These systems may cost \$1500 or more. See DAVIS & SCHAFFMAN, *supra* note 4, at 148. Ludwig and Turner pointed out that the most effective control should combine reduction of outside pressure with sealing the slab and walls. See Jerry F. Ludwig & William Turner, *Control Strategies*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE 351, 369 (Jonathan M. Samet & John D. Spengler eds., 1991).

³⁰² See U.S. GAO, *supra* note 255, at 26-30.

³⁰³ EPA estimated costs to homeowners of \$1,500-\$3,300 for installation of sub-slab ventilation, \$2,300-\$5,500 for installation of wall ventilation, \$700-\$1,300 for installation of drain tile ventilation, and \$300 for furnace ventilation. See U.S. GAO, *supra* note 255, at 31. Note that these figures were based on the costs of incomplete radon mitigation projects conducted on 18 homes in April 1986.

³⁰⁴ See DAVIS & SCHAFFMAN, *supra* note 4, at 153.

³⁰⁵ See *id.*

³⁰⁶ See *id.*

³⁰⁷ Two types of radon detection kits are available. Short-term kits are canisters which contain activated charcoal. The device collects radon progeny over two to four days. The device is sent to a laboratory where gamma rays from radon progeny lead-214 and bismuth-214 are counted. Concentration is determined by considering the radon progeny count and the length of exposure. Costs for radon tests range from \$25 to \$200 depending on the size of the home, number of samples collected, and whether it is a do-it-yourself or professional test. See *id.*

A special polymer film designed to count ("track") alpha particles constitutes the long-term test or "track-etch monitor." After exposure for three months to a year in the home, a lab etches the film with sodium hydroxide and electronically counts the alpha particle "tracks." This information is used to calculate the concentration of radon. See Hanson, *supra* note 267, at 10.

For more radon testing information call EPA's RCP program (202-233-9370) or the Radon Hotline (800-505-RADON). See also U.S. ENVTL. PROTECTION AGENCY, No. 402-R-93-003, HOME BUYER'S AND SELLER'S GUIDE TO RADON (1993); U.S. ENVTL. PROTECTION AGENCY, No. 402-K92-003, CONSUMER'S GUIDE TO RADON REDUCTION (1992); U.S. ENVTL. PROTECTION

it is determined that radon concentrations equal or exceed the four pCi/l "action level," a professional should be consulted to determine the appropriate remediation methods.

EPA's efforts to reduce the public's exposure to radon began in 1986 with the publication of information on radon control.³⁰⁸ In 1987, EPA and the National Association of Homebuilders published guidance for builders.³⁰⁹ EPA published its first technical guidance in 1988 and updated it in 1991.³¹⁰ In 1994, EPA promulgated final standards for the control of radon in new residential buildings.³¹¹ These standards require passive control systems to be used in areas of high radon potential,³¹² which are defined as Zone 1 of maps prepared by EPA with the assistance of the U.S. Geological Survey and state geologists.³¹³

EPA also oversees two voluntary programs: the Radon Measurement Proficiency (RMP) program and the Radon Contractor Proficiency (RCP) program.³¹⁴ The RMP program determines the accuracy of radon detectors and the RCP program evaluates radon mitigation companies.³¹⁵ These programs are often run at the state level.³¹⁶

AGENCY, No. 520/1-89-027, REDUCING RADON RISKS (1992); U.S. DEP'T OF HEALTH AND HUMAN SERVICES AND CENTERS FOR DISEASE CONTROL, U.S. ENVTL. PROTECTION AGENCY, No. 402-K92-001, A CITIZEN'S GUIDE TO RADON (2d ed. 1992).

³⁰⁸ See Model Standards and Techniques for Control of Radon in New Residential Buildings, 59 Fed. Reg. 13,402 (1994) (citing U.S. ENVTL. PROTECTION AGENCY, No. EPA-86-004, A CITIZEN'S GUIDE TO RADON (1986) and U.S. ENVTL. PROTECTION AGENCY, No. EPA-86-005, RADON REDUCTION METHODS, A HOMEOWNER'S GUIDE (1986)). EPA was given responsibility to gather information, coordinate research, and assess mitigation programs by the Radon Gas and Indoor Air Quality Research Act of 1986 (Title IV of the Superfund Amendments and Reauthorization Act), 42 U.S.C. § 7401 (1997)). See Curtin, *supra* note 278, at 200-01.

³⁰⁹ See *id.* (citing U.S. ENVTL. PROTECTION AGENCY, No. EPA-87-009, RADON REDUCTION IN NEW CONSTRUCTION, AN INTERIM GUIDE (1987)).

³¹⁰ See *id.* (citing U.S. ENVTL. PROTECTION AGENCY, No. EPA/600/8-88/087, RADON-RESISTANT RESIDENTIAL NEW CONSTRUCTION (1988) and U.S. ENVTL. PROTECTION AGENCY, No. EPA/625/2-91/032, RADON-RESISTANT CONSTRUCTION TECHNIQUES FOR NEW RESIDENTIAL CONSTRUCTION: TECHNICAL GUIDANCE (1991)).

³¹¹ See *id.*

³¹² See Model Standards and Techniques for Control of Radon in New Residential Buildings, 59 Fed. Reg. 13,403 (1994).

³¹³ See *id.*

³¹⁴ See DAVIS & SCHAFFMAN, *supra* note 4, at 148; see also EPA Reports State Radon Survey Results, PR NEWswire, Oct. 17, 1990, (page unavail.), available in LEXIS, Envirn Library, Allnws file.

³¹⁵ See DAVIS & SCHAFFMAN, *supra* note 4, at 148.

³¹⁶ See *id.*; see, e.g., Ed Bas, *Radon Mitigation: It's Still a Hot, Cold Market for Mechanical*

In the 1988 amendments to TSCA, also known as the Indoor Radon Abatement Act, Congress stated its long-term goal for indoor radon levels to be no higher than outdoor levels (about 0.4 pCi/l).³¹⁷ Pursuant to the Indoor Radon Abatement Act, EPA promulgated the State Indoor Radon Grants (SIRG) program.³¹⁸ Also in 1988, EPA and the U.S. Surgeon General issued an advisory recommending that all homes be tested for radon.³¹⁹ Pursuant to the requirements of the Indoor Radon Abatement Act, the *Model Standards and Techniques For Control of Radon in New Residential Buildings* was promulgated as a final standard on March 21, 1994.³²⁰

The 1996 SDWA Amendments require the withdrawal of any national primary drinking water regulation (NPDWR) previously proposed for radon, and prescribe a process and schedule for establishing a new NPDWR.³²¹ The statute requires a proposed NPDWR to be promulgated by August 6, 1999, and a final maximum contaminant level goal (MCLG) and NPDWR to be promulgated by August 6, 2000.³²² The statute also requires EPA to perform a risk assessment as part of this effort.³²³ If the maximum contaminant level (MCL) developed as part of this process would result in drinking water's contribution to radon in indoor air to have a concentration above the national average concentration in outdoor air, then EPA is to promulgate an alternative MCL for radon.³²⁴ The alternative MCL is to use the national average concentration of radon in the outdoor air as the target concentration for drinking water's contribution to radon in the indoor air.³²⁵

However, despite radon's recognized health risks, the public's reaction has been one of apathy and disinterest. Several reasons have been suggested for this attitude including: scientific illiteracy and ambiguity of risk; resistance to controls imposed on activities within the home; feelings of invulnerability; burnout from the never-ending

Contractors, 188 AIR CONDITIONING, HEATING & REFRIGERATION NEWS, Apr. 19, 1993, at 3 (mentioning Michigan's EPA-approved Radon Contractor Proficiency Program).

³¹⁷ TSCA § 301, 15 U.S.C. § 2661 (1994).

³¹⁸ State Indoor Radon Grants, 54 Fed. Reg. 36,857 (1989).

³¹⁹ See Guiffreda, *supra* note 3, at 316.

³²⁰ See *Model Standards and Techniques for Control of Radon in New Residential Buildings*, 59 Fed. Reg. 13,402 (1994).

³²¹ See 42 U.S.C. § 300g-1(b)(13)(A) (1994).

³²² See 42 U.S.C. § 300g-1(b)(13)(D). A MCLG is a nonenforceable goal.

³²³ See 42 U.S.C. § 300g-1(b)(13)(B).

³²⁴ See 42 U.S.C. § 300g-1(b)(13)(F). MCL is defined at 42 U.S.C. § 300f(3).

³²⁵ See 42 U.S.C. § 300g-1(b)(13)(G)(i).

list of environmental problems; and conflicting information provided by a variety of sources.³²⁶ Because of lack of interest in the radon threat, the public may be reluctant to mitigate; forget to put out purchased testing devices; be unwilling to test; fail to relate housing prices and high radon levels; and show decreasing interest and participation in radon control services.³²⁷ Thus, radon control may be thwarted regardless of governmental control programs. However, increasingly the sale of homes is contingent on radon testing. With the five percent turnover of homes per year, within ten years half the homes in the U.S. will be tested—and hopefully mitigated.

C. *Biological Contaminants*

Biological contaminants have been estimated by some experts to be the most significant source of indoor air pollution and to affect tens of millions of people in the United States.³²⁸ Airborne biological contaminants include a diverse group of organisms and biological matter originating from many sources. They are present in all indoor and outdoor environments and include viable agents, such as viruses, fungi, amoebae, algae and bacteria;³²⁹ and non-viable agents, such as house dust mite fecal pellets, cockroach feces, insect and arachnid dried hulks and body parts, animal danders, nonviable remains of molds and spores, dried animal excretions, and pollens.³³⁰

1. Health Effects of Biological Contaminants

Emissions of biological contaminants (biogenic aerosols) may be toxic, pathogenic, or allergenic.³³¹ Mycotoxins are produced by molds and may cause direct toxic effects, immunosuppression, gastrointes-

³²⁶ See C. Richard Cothorn, *Widespread Apathy and the Public's Reaction to Information Concerning the Health Effects of Indoor Air Radon Concentrations*, 6 CELL BIOLOGY & TOXICOLOGY 315, 315-22 (1990).

³²⁷ See *id.* For example, only five percent of U.S. homes were tested for radon in 1990, and less than three percent of homeowners whose homes had levels above 4 pCi/l took action to reduce radon exposure. See *id.* at 315.

³²⁸ See DAVIS & SCHAFFMAN, *supra* note 4, at 47.

³²⁹ See Harrison, *supra* note 13, at 318 (citing EPA REPORT, *supra* note 13, at 3-13) see generally David N. Weissman & Mark R. Schuyler, *Biological Agents and Allergic Disease*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE 285 (Jonathan M. Samet & John D. Spengler eds., 1991).

³³⁰ EPA *Inside Story*, *supra* note 2; see also Weissman & Schuyler, *supra* note 329, at 288-89.

³³¹ See REITZE, *supra* note 33, at 420 (citing EPA REPORT, *supra* note 13, at 3-13).

tinal lesions, hematopoietic suppression, suppression of reproductive function, anorexia, lassitude, and nausea.³³²

Some non-building related, human- or animal-transmitted pathogens found in indoor environments cause diseases including influenza, chicken pox, measles, and pulmonary tuberculosis.³³³ Infectious bacteria sometimes colonize in humidifiers and air conditioners and result in the spread of diseases, such as Legionnaire's Disease³³⁴ and Pontiac Fever.³³⁵ Three common respiratory allergies occur in some persons as a result of exposure to biologic indoor air pollutants: allergic rhinitis,³³⁶ bronchial asthma,³³⁷ and hypersensitivity pneumonitis.³³⁸ A small subset of the population is also affected by allergenic agents that cause allergic or hypersensitive reactions.³³⁹

2. Control of Biological Contaminants

Air conditioners, heating and ventilation systems, humidifiers, dehumidifiers, and refrigerator drip pans are fertile breeding grounds for mold, mildew, and other microorganisms.³⁴⁰ Wet areas or materials, such as musty furniture and carpets,³⁴¹ and wet basements and walls,

³³² See *id.* at 420–21. Non-specific symptoms may be produced by trichocena mycotoxins. *Id.* Some mycotoxins are highly systemic poisons. See *id.*

³³³ See *id.*

³³⁴ The bacterium that causes Legionnaire's Disease is *Legionella pneumophila*. Legionnaire's Disease affects the lungs, the gastrointestinal tract, the kidneys and the central nervous system. See REITZE, *supra* note 33, at 421 n.238 (citing EPA REPORT, *supra* note 13, at 3–14). An estimated 20,000 new cases of Legionnaire's Disease are reported per year in the United States. See Robert E. Geisler, *The Fungusamongus: Sick Building Survival Guide*, 8 ST. THOMAS L. REV. 511, 512–13 (1996).

³³⁵ Pontiac Fever, also caused by *Legionella pneumophila*, is a short-term disease and is not fatal. The disease is characterized by fever, chills, headache, and muscle ache. Other symptoms include coughing, sore throat, chest pain, nausea, and diarrhea. See REITZE, *supra* note 33, at 421 n.239 (citing EPA REPORT, *supra* note 13, at 3–14).

³³⁶ Allergic rhinitis is also known as "hay fever." See Weissman & Schuyler, *supra* note 329, at 287.

³³⁷ Bronchial asthma is characterized by narrowing of the bronchioles, hypersecretion of thick mucous, wheezing, shortness of breath, and coughing. See REITZE, *supra* note 33, at 421 n.243 (citing EPA REPORT, *supra* note 13, at 3–15).

³³⁸ Hypersensitivity pneumonitis is an acute immune reaction which includes production of IgE antibodies, cellular hypersensitivity and formation of interstitial granulomas. Inflammatory cells fill and cause destruction of the alveoli eventually resulting in irreversible pulmonary fibrosis, pulmonary failure and death. See *id.* at n.244 (citing EPA REPORT, *supra* note 13, at 3–15); see also Weissman & Schuyler, *supra* note 329, at 287–88.

³³⁹ See REITZE, *supra* note 33, at 421 (citing EPA REPORT *supra* note 13, at 3–15).

³⁴⁰ See DAVIS & SCHAFFMAN, *supra* note 4, at 48. After the organisms form in these appliances, the appliances themselves may act to distribute the microorganisms.

³⁴¹ If not kept clean, carpets can be a "sink" for dust mites and other biological contaminants. See *id.* at 110.

also provide ideal conditions for growth.³⁴² Preventing biological contaminants indoors is best accomplished by preventing the buildup of warm, moist conditions favorable to the growth of microorganisms. Relative humidity levels in most homes should be between thirty and fifty percent.³⁴³ A vacuum cleaner with a special filter, known as a high-efficiency particulate air (HEPA) filter, may be necessary to remove biocontaminants from carpets, furniture and draperies.³⁴⁴ Heating/air conditioning systems should be professionally cleaned every three to five years.³⁴⁵ Biocontaminant precaution includes installation of exhaust fans in kitchens, bathrooms, and dryers; ventilating attics and crawl spaces; conducting regular maintenance on humidifiers and air conditioning systems; removing water-damaged items; and keeping the house clean.³⁴⁶ Room or whole-house cleaning units can be installed, although their effectiveness is questionable.³⁴⁷ Air exchangers also provide clean outside air and are useful in energy-efficient homes.³⁴⁸ Currently, there is no federal program applicable specifically to biocontaminants.

3. Tuberculosis

a. *Introduction/Sources*

Infectious diseases are responsible for at least thirty-two percent of global mortality and kill more people than cancer, heart disease, and cerebrovascular disease combined.³⁴⁹ One major and currently prevalent building-related infectious disease is tuberculosis (TB). TB is one of the oldest recognized human diseases³⁵⁰ and remains a serious health problem today.³⁵¹ The Greek physician Hippocrates described

³⁴² See *EPA Inside Story*, *supra* note 2; see also DAVIS & SCHAFFMAN, *supra* note 4, at 48.

³⁴³ See DAVIS & SCHAFFMAN, *supra* note 4, at 49.

³⁴⁴ See *id.*

³⁴⁵ See *id.* at 50.

³⁴⁶ See *EPA Inside Story*, *supra* note 2.

³⁴⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 49.

³⁴⁸ See *id.*

³⁴⁹ See Anne E. Platt, *Confronting Infectious Diseases*, in STATE OF THE WORLD 1996, 114, 115 (Lester Brown ed., 1996). Of the 51 million deaths in the world in 1993, 20% were due to communicable diseases, but 99% of these deaths occurred in developing countries. See Brad Wye & Dita Smith, *North-South Gap in Death*, WASH. POST, Mar. 22, 1997, at A20.

³⁵⁰ See Patricia C. Kuszler, *Balancing The Barriers: Exploiting and Creating Incentives to Promote Development of New Tuberculosis Treatments*, 71 WASH. L. REV. 919, 922 (1996). In 1900, deaths were more common from diseases like TB than cancer. See *Science, Not 'Fear,' Should Be Key to Setting Public Health Priorities*, ACSH Official Says, 38 FOOD CHEM. NEWS, Feb. 26, 1996 (page unavail.).

³⁵¹ Estimates predict ninety million new TB cases by the end of the decade. See Kuszler, *supra*

it and named it "phthisis," meaning "to melt away."³⁵² By the mid-nineteenth century it was called consumption.³⁵³ The disease was also known as the "white plague."³⁵⁴ In 1882, Robert Koch discovered the bacillus responsible for the tubercle lesions found in the bones and tissues of patients with consumption.³⁵⁵ TB is the infectious disease with the world's third highest incidence, causing more adult deaths worldwide than any other,³⁵⁶ and is responsible for nearly three million deaths each year worldwide.³⁵⁷ The World Health Organization (WHO) declared a global health emergency in 1993 due to the resurgence of TB.³⁵⁸ In 1994, 8.8 million people contracted active TB.³⁵⁹ Approximately 1.7 billion people, one third of the world's population, are infected with the predominant TB organism,³⁶⁰ although most people never get an active infection.³⁶¹ Although worldwide active TB leveled off in 1996 at about eight million new cases a year, it is still one of the world's most serious diseases.³⁶²

note 350, at 920. At least 30 million people are expected to die from TB in the coming years. *See id.*; *see also* World Health Organization, *Groups at Risk* <http://www.who.ch/programmes/gtb/tbrep_96/execsum.htm> [hereinafter *Risk*].

³⁵² Kuszler, *supra* note 350, at 922 (citation omitted).

³⁵³ *See id.*

³⁵⁴ *Id.* at 920.

³⁵⁵ *See* FELISSA L. COHEN & JERRY D. DURHAM, *TUBERCULOSIS: A SOURCEBOOK FOR NURSING PRACTICE* 8 (1995).

³⁵⁶ *See id.* at 29; *see also* National Institute of Allergy and Infectious Diseases, *NIAID Fact Sheet*, Apr. 1995. <<http://www.niaid.nih.gov/factsheets/tb.htm>> [hereinafter *NIAID Fact Sheet*]. In 1995, more people died of TB than in any other year. *See Risk*, *supra* note 351.

³⁵⁷ *See* Platt, *supra* note 349, at 116; *see also* Kuszler, *supra* note 350, at 920; *Global Spread of Tuberculosis is Leveling Off, WHO Says*, BALTIMORE SUN, Mar. 20, 1997, at 19A [hereinafter *Global Spread*].

³⁵⁸ *See Risk*, *supra* note 351. This was the only time an infectious disease was a global health emergency. *See* World Health Organization, *Tuberculosis* (Mar. 1996) (Fact Sheet No. 104) (revised) <http://www.who.ch/programmes/gtb/wtbd/day/facts_1e.htm>.

³⁵⁹ *See* Platt, *supra* note 349, at 284.

³⁶⁰ *See NIAID Fact Sheet*, *supra* note 356. It is estimated that five percent of the U.S. population tests positive for TB. *See New OSHA Standards for Protecting Health Care Workers From TB Will Be Published for Comment*, 39 BLUE SHEET, Sept. 25, 1996, at 12-14 [hereinafter BLUE SHEET]. Further, 4.4 million people worldwide are infected with TB and HIV. *See NIAID Fact Sheet*, *supra* note 356. In the U.S., approximately 100,000 HIV-infected people also have TB. *See id.*

³⁶¹ *See generally* Platt, *supra* note 349.

³⁶² *See* Curt Suplee, *Global Tuberculosis Epidemic Leveling Off*, WASH. POST, Mar. 20, 1997, at A24.

There are currently ten to fifteen million people in the U.S. infected with TB.³⁶³ About ten percent will develop active TB.³⁶⁴ There was a fourteen percent increase in reported active TB cases in the U.S. between 1985 and 1993,³⁶⁵ but the number of active TB cases declined by seven percent between 1995 and 1996.³⁶⁶ Increased TB rates have been attributed to factors such as the HIV/AIDS co-epidemic;³⁶⁷ social conditions such as poverty, crowding, and poor nutrition; deinstitutionalization of the mentally ill; increasing costs of medication; decreased TB control funding; increased immigration from high prevalence TB countries; and transmission of TB in settings with high risk.³⁶⁸ Deterioration of the health care infrastructure is also a factor.³⁶⁹

While deaths from TB are concentrated in the developing countries,³⁷⁰ the domestic public health concerns over this disease have increased as multiple-drug resistant TB, with a mortality rate above fifty percent, has emerged in the United States.³⁷¹ In New York City, in 1992, more than half of the new cases of TB were resistant to at least one of the major drugs used to control the disease.³⁷² To treat an active case of drug-resistant TB in the United States costs twenty times the approximately \$10,000 it takes to treat an active case of

³⁶³ See Amy Goldstein & Curt Suplee, *District TB Rate Bucks Downward Trend*, WASH. POST, Mar. 25, 1997, at A3 [hereinafter *District TB*].

³⁶⁴ See *id.*

³⁶⁵ See NIAID Fact Sheet, *supra* note 356; see generally Walter V. Reid, *Biodiversity and Health: Prescription for Progress*, ENV'T, July 1995, at 12 (number of TB cases was declining six percent per year until 1985).

³⁶⁶ See *District TB*, *supra* note 363, at A3. In the U.S. there were 22,860 cases of active TB in 1995; in 1996, the number decreased to 21,327. See generally Thomas R. Frieden et al., *Tuberculosis in New York City—Turning the Tide*, 333 NEW ENG. J. MED. 229 (1995).

³⁶⁷ HIV-positive persons are 40 times more likely to contract and develop TB than those who are HIV-negative. See Rosemary G. Reilly, *Combating the Tuberculosis Epidemic: The Legality of Coercive Treatment Measures*, 27 COLUM. J.L. & SOC. PROBS. 101, 104 (1993).

³⁶⁸ Medical authorities have attributed prison overcrowding to the TB resurgence. In 1988, the TB rate in state and federal prisons was 75 per 100,000 people, compared to 14 cases per 100,000 people in the general population. See Mark Watts, *Prison HVAC Design: The Total Concept Approach*, 9 HEATING, PIPING, AIR CONDITIONING, Sept. 1996, at 49.

³⁶⁹ See COHEN & DURHAM, *supra* note 355, at 31.

³⁷⁰ For example, in 1993, 41.5 percent of deaths were attributed to infectious disease in developing countries while only 1.2 percent of deaths were caused by infectious disease in developed countries. Three-fourths of the world's TB is concentrated in 13 countries: Bangladesh, Brazil, China, Ethiopia, India, Indonesia, Mexico, Pakistan, the Phillipines, Thailand, Russia, South Africa, and Zaire. See Suplee, *supra* note 362, at A24.

³⁷¹ See Reid, *supra* note 365.

³⁷² See Frieden, *supra* note 366, at 229.

non-drug-resistant TB.³⁷³ WHO estimates that fifty million people worldwide are infected with drug-resistant TB.³⁷⁴

TB is a chronic, communicable disease³⁷⁵ caused by bacteria, particularly *Mycobacterium (M.) tuberculosis*.³⁷⁶ On average, of those people infected with *M. tuberculosis*, ten percent develop active TB.³⁷⁷ The ninety percent who do not develop active TB are not infectious to others.³⁷⁸ Transmission of TB occurs person to person through microscopic airborne particles or droplets (droplet nuclei) that are contaminated with *M. tuberculosis* bacteria.³⁷⁹ These droplets are produced when someone with infectious TB coughs, sneezes, speaks, sings, laughs, or yells.³⁸⁰ Droplets with TB bacilli are small enough to bypass the natural defenses of upper respiratory passages.³⁸¹ Infection begins when the bacilli reach the air sacs of the lungs, where they multiply.³⁸² More than eighty percent of TB cases occur in the lung.³⁸³ The droplets can remain suspended in the air.³⁸⁴ People in the same air space as those with infectious TB³⁸⁵ then inhale the droplets;³⁸⁶ the bacteria then multiplies. Heating, ventilation, and air conditioning

³⁷³ See generally Carolyn Dunbar, *Los Alamos Rolls Out Technology Transfer Project for Radiology*, 15 HEALTH MGMT. TECH. 42 (Nov. 1994).

³⁷⁴ See Suplee, *supra* note 362, at A24.

³⁷⁵ The early symptoms of TB are: weight loss, fever, night sweats, and loss of appetite. Later symptoms include lesions, chest pain, coughing, bloody sputum, shortness of breath, and dull aching chest pain or tightness. See COHEN & DURHAM, *supra* note 355, at 7. The death rate of untreated patients is between 40 and 60 percent; however, TB can be cured more than 90 percent of the time. See *NIAID Fact Sheet*, *supra* note 356. With proper treatment, most people fully recover from TB. However, in recent years, TB has become resistant to common drugs, making treatment more difficult. See COHEN & DURHAM, *supra* note 355, at 67. People treated with drugs for at least two weeks are usually not infectious, although daily doses may be necessary for up to one year. See generally Boyd A. Byers, *TB or not TB: OSHA Updates Enforcement Policy for Exposure to TB in the Workplace*, 2 KAN. EMPLOYMENT LAW LETTER 1 (Jan. 1996). More than 80 percent of TB cases occur in the lungs. See COHEN & DURHAM, *supra* note 355, at 5.

³⁷⁶ See COHEN & DURHAM, *supra* note 355, at 3-5.

³⁷⁷ On average, people have a 50 percent chance of TB infection if they spend eight hours a day for six months living or working with someone with active TB. See *NIAID Fact Sheet*, *supra* note 356.

³⁷⁸ See *id.*

³⁷⁹ See COHEN & DURHAM, *supra* note 355, at 5.

³⁸⁰ See *id.*

³⁸¹ See *NIAID Fact Sheet*, *supra* note 356.

³⁸² See *id.*

³⁸³ See COHEN & DURHAM, *supra* note 355, at 5; see also *NIAID Fact Sheet*, *supra* note 356. TB may also occur in the larynx, lymph nodes, kidney, bone, and brain.

³⁸⁴ See COHEN & DURHAM, *supra* note 355, at 5.

³⁸⁵ TB transmission usually occurs only after prolonged exposure to someone with active TB because most infected people expel few bacilli. See *NIAID Fact Sheet*, *supra* note 356.

³⁸⁶ See COHEN & DURHAM, *supra* note 355, at 5.

systems can spread the disease.³⁸⁷ TB may also be transmitted through ingestion of contaminated food or drink or through direct inoculation,³⁸⁸ but it is not likely to be transmitted through personal items merely touched by those with TB.³⁸⁹ Most people who inhale TB bacterium do not become infected with active TB, but the risk of acquiring it increases with the length of time the susceptible person shares air space with the person with active TB.³⁹⁰ Other factors that affect transmission include the characteristics of the droplets, the environmental conditions to which the droplets are exposed,³⁹¹ and the circumstances under which the contact with the new host occurs.³⁹² Certain groups have a higher prevalence of TB infection including homeless persons, prison inmates, alcoholics, the elderly, and injecting drug users.³⁹³

b. Regulation/Control

New York City was the leader in devising a public health approach to TB.³⁹⁴ New York City's plan to control TB became the model for public health authorities throughout the United States.³⁹⁵ Most states passed laws to codify the TB public health strategy.³⁹⁶

³⁸⁷ See Ian Warpole, *Is Your Office Making You Sick? Sick Building Syndrome*, 15 AM. HEALTH, July 1996, at 66.

³⁸⁸ See COHEN & DURHAM, *supra* note 355, at 5.

³⁸⁹ See NIAID Fact Sheet, *supra* note 356.

³⁹⁰ See COHEN & DURHAM, *supra* note 355, at 5-6. TB transmission occurs most frequently in crowded environments such as hospitals, prisons, and shelters (where HIV-infected persons make up a growing proportion of the population). A long airplane flight may also subject a passenger to TB by recirculating air. See Alan Goodman, *Home Sweet Home: A Potential Source of Many Ills*, 31 EXEC. HEALTH'S GOOD HEALTH REP., May 1995, at 4.

³⁹¹ Further, certain occupations may be associated with elevated risk because the environment is favorable for transmission. See BLUE SHEET, *supra* note 360, at 12-14. These environments include health care facilities, homeless shelters, prisons, laboratories, and facilities housing animals. See *id.* Also, occupations with impoverished, unskilled workers frequently have elevated TB rates. See *id.*

³⁹² See COHEN & DURHAM, *supra* note 355, at 17. Infection with HIV is the greatest risk factor for developing TB. See *id.* at 23. Pesticides can also play a role in TB infection by compromising the body's ability to fight infection. See Janet Raloff, *Pesticides May Challenge Human Immunity*, SCI. NEWS, Mar. 9, 1996, at 149.

³⁹³ See COHEN & DURHAM, *supra* note 355, at 37. Moreover, people whose lungs have been affected by inhalation of dusts show increased susceptibility. See Adrian Budgen, *The T&N Experience: Lessons To Be Learned From Armley?*, 11 Mealey's Litig. Rep.: Asbestos (page unavail.) (Sept. 20, 1996).

³⁹⁴ See Kuszler, *supra* note 350, at 924 (citation omitted).

³⁹⁵ See *id.*

³⁹⁶ See *id.*

The most important measure to prevent TB transmission is adequate ventilation.³⁹⁷ Another major preventative measure is identifying infected individuals early and treating them with drug therapy;³⁹⁸ one TB vaccine, BCG, is widely used.³⁹⁹ Other preventative measures include: covering the mouths of TB patients when coughing or sneezing; using UV light to kill the TB bacterium;⁴⁰⁰ using filters, respirators, and masks to protect healthy people; and isolation of TB patients. On October 17, 1997, OSHA published in the *Federal Register* a proposed rule establishing an occupational health standard for tuberculosis.⁴⁰¹ The rule proposes several prevention and control measures to be used where employees are exposed to TB, including the use of respirators, procedures for early identification and treatment of infection, isolation of infectious individuals, and medical follow-up for workers who are infected through occupational exposure.

M. tuberculosis is difficult to study in the laboratory, which can hamper TB research.⁴⁰² Recently, however, there have been some fledgling efforts to renew research into anti-tuberculosis agents.⁴⁰³ Several types of vaccines are being researched.⁴⁰⁴

Funding for TB programs has been inadequate. Costs of treating TB in 1991 were estimated at \$703.1 million and are expected to grow to as much as \$2.2 billion by 2000.⁴⁰⁵ From 1962 to 1990, budgets allocated by the Centers for Disease Control (CDC) and the National Institute of Allergy and Infectious Diseases (NIAID) for TB research

³⁹⁷ See NIAID Fact Sheet, *supra* note 356.

³⁹⁸ See COHEN & DURHAM, *supra* note 355, at 8. The following people should be considered for preventative therapy: close contacts of people infected with TB, people with HIV, injection drug users, foreign-born people from countries where TB is common, low-income groups, and residents of long-term care facilities. See NIAID Fact Sheet, *supra* note 356. The five major drugs available to treat TB are: streptomycin, isoniazid, pyrazinimide, ethambutol, and rifampin. See Kuszler, *supra* note 350, at 934 n.93.

³⁹⁹ See *id.* at 947. The protection of this vaccine ranges from zero to eighty percent. See P.E.M. Fin, *Variation in Protection by BCG: Implications of and for Heterologous Immunity Bacillus Calmette-Guerrin*, LANCET, Nov. 18, 1995, at 1339.

⁴⁰⁰ In 1996, researchers experimented with UV light to destroy airborne viruses and bacteria, including TB. See generally *Research at the Electric Power Institute Says Ultraviolet Light Cleans Water, Destroys Viruses, and Purifies Air*, 7 ENVTL. LABORATORY WASH. REP., July 22, 1996.

⁴⁰¹ Occupational Exposure to Tuberculosis, 62 Fed. Reg. 54,160 (1997) (to be codified at 29 C.F.R. pt. 1910).

⁴⁰² See NIAID Fact Sheet, *supra* note 356.

⁴⁰³ See Kuszler, *supra* note 350, at 947-48.

⁴⁰⁴ See *id.*; see also M. Harboes, *Novel Developments: Vaccines Against Tuberculosis*, VACCINE WKLY., Apr. 1, 1996 (page unavail.).

⁴⁰⁵ See generally E. Brown et al., *Health-Care Expenditures for Tuberculosis in the United States*, ISS ARCHIVES INTERNAL MED. 1595 (1995).

dwindled.⁴⁰⁶ However, from 1990 to 1995, there were large increases in spending.⁴⁰⁷ NIAID supports more than one hundred research projects related to TB.⁴⁰⁸ In fiscal year 1995, it devoted \$31 million to TB research, more than an eight-fold increase since 1991.⁴⁰⁹ However, the amount of money designated for TB by NIAID now is essentially returning to zero.⁴¹⁰ In 1994, the CDC announced a plan to spend \$125 million per year on upgrading measures to counter TB; however, Congress only appropriated \$7.7 million and the plan was scaled back.⁴¹¹ Internationally, WHO also has committed only a small budget to deal with the TB threat.⁴¹² In 1994, it provided six million dollars for TB control, of which only fourteen percent was from WHO's budget.⁴¹³ Further, there are numerous obstacles that make comprehensive treatment of TB patients worldwide virtually impossible, including an inadequate international public health infrastructure, prohibitively high cost of treatment, and outmoded and inadequate drugs and vaccines.⁴¹⁴

There has been some limited litigation concerning TB. Recent cases regarding TB include:

- (a) a 1996 case, where New York's highest court struck down a lower court's finding that a shelter program for the homeless with HIV failed to provide minimum health protections against TB;⁴¹⁵ and
- (b) a 1996 North Carolina Court of Appeals ruling that an employee who contracted TB from a co-worker is not entitled to workers' compensation benefits.⁴¹⁶

⁴⁰⁶ See generally BLUE SHEET, *supra* note 360.

⁴⁰⁷ CDC spent about \$140 million per year; NIAID's budget went up to about \$15-25 million. See *id.*

⁴⁰⁸ See NIAID Fact Sheet, *supra* note 356.

⁴⁰⁹ See *id.*; see generally BLUE SHEET, *supra* note 360.

⁴¹⁰ See generally BLUE SHEET, *supra* note 360.

⁴¹¹ See Christina Kent, *A Long-Neglected System Strains to Respond to a Rising Threat*, AM. MED. NEWS, Jan. 8, 1996, at 9.

⁴¹² See Kuszler, *supra* note 350, at 939-40.

⁴¹³ See *id.* at 940.

⁴¹⁴ See *id.* at 937-38.

⁴¹⁵ See *Mixon v. Grinker*, No. 123 (N.Y. June 11, 1996); see generally *Court Overturns Ruling on Housing of Homeless with HIV*, 11 AIDS POL'Y & LAW 1 (1996).

⁴¹⁶ See *Higgs v. Southeastern Cleaning Serv.*, 470 S.E.2d 337, 340-41 (N.C. Ct. App. 1996); see generally *WOMBLE CARLYLE SANDRIDGE & RICE, PLLC, Is TB Transmitted by Co-Worker Compensable?*, 6 N.C. EMPLOYMENT L. LETTER, June 1996, at 1.

For more TB information, a web site on TB has been established by the National Tuberculosis Center at the University of Medicine and Dentistry of New Jersey (UMDNJ), in Newark.⁴¹⁷

D. Asbestos

Asbestos is a group of mineral fibers⁴¹⁸ commonly used in past years in building products because it possesses qualities of flexibility, strength, and durability and is resistant to heat and corrosion.⁴¹⁹ Asbestos can be found in over 2,000 products.⁴²⁰ Many commercial buildings, older homes, and schools contain asbestos products such as roofing and flooring materials,⁴²¹ textiles, papers, filters and gaskets, cement, pipes, coating materials, thermal and acoustic insulation,⁴²² and textured paints.⁴²³ School buildings are most likely to contain asbestos in spray-applied fireproofing, pipe and boiler insulation, acoustical and decorative insulation, and floor and ceiling tile.⁴²⁴

⁴¹⁷ <<http://www.umdj.edu/ntbc.>>.

⁴¹⁸ See *EPA Inside Story*, *supra* note 2; see also Radon Detection Systems, *Asbestos* (last modified Feb. 14, 1996) <<http://www.abwam.com/grossing/refasbes.htm>>; Clinton L. Hach, *Avoid Environmental Obstacle*, 37 TRANSP. & DISTRIBUTION, Apr. 1996, at 54. "Asbestos' includes chrysolite, amosite, crocidolite, tremolite asbestos, anthophyllite asbestos, actinolite asbestos, and any of these chemicals that has been chemically treated and/or altered." 29 C.F.R. § 1910.1001(b) (1997).

⁴¹⁹ See Gerald W. Boston, *Toxic Apportionment: A Causation and Risk Contribution Model*, 25 ENVTL. L. 549, 553 (1995); see also Michael M. Stahl & David J. Kling, *Asbestos In Buildings*, TRIAL, Aug. 1990, at 28.

⁴²⁰ See Steven J. Murdzia, *Gaining an Understanding of the New OSHA Asbestos Rule*, 3 CONN. ENVTL. COMPLIANCE UPDATE 1 (1995); see also U.S. GEN. ACCOUNTING OFFICE, INDOOR AIR POLLUTANTS: AN EMERGING HEALTH PROBLEM 8 (1980).

⁴²¹ See Med Access, *Indoor Air Pollution: An Introduction for Health Professionals* <http://www.medaccess.com/ind_air/prof_09.html> [hereinafter Med Access]. For example, 9" x 9" vinyl floor tiles in pre-1980 homes contain asbestos about 90 percent of the time and 12" x 12" vinyl tiles about 50 percent of the time. See DAVIS & SCHAFFMAN, *supra* note 4, at 45.

⁴²² See DAVIS & SCHAFFMAN, *supra* note 4, at 45-46; see also Spengler, *supra* note 153, at 52-53; Med Access, *supra* note 421.

⁴²³ See *EPA Inside Story*, *supra* note 2. As many as 733,000 buildings in the United States may contain asbestos. This figure does not include buildings with fewer than ten units. See Spengler, *supra* note 153, at 52.

⁴²⁴ See Stahl & Kling, *supra* note 419, at 28.

1. Health Effects of Asbestos

Asbestos exposure occurs through absorption through the skin⁴²⁵ or through inhalation⁴²⁶ or ingestion⁴²⁷ and is transported throughout the body by blood and by the lymphatic systems.⁴²⁸ Based on human epidemiological data, EPA listed asbestos as a Group A (known) human carcinogen.⁴²⁹ Asbestos exposure has been closely linked with asbestosis,⁴³⁰ lung cancer,⁴³¹ and mesothelioma.⁴³² Asbestos fibers that are ingested are associated with stomach or gastrointestinal cancer.⁴³³ The majority of people afflicted with these diseases developed them as a result of occupational exposure to asbestos; however, these diseases also have resulted from exposure to asbestos brought home from the workplace in clothing and equipment.⁴³⁴

⁴²⁵ Asbestos may cause severe skin irritation. See Lawrence S. Kirsch, *Behind Closed Doors: Indoor Air Pollution and Government Policy*, 6 HARV. ENVTL. L. REV. 339, 356-57 (1982).

⁴²⁶ Not all fibers become lodged in the lungs; some are removed in the same manner as are other foreign particles. However, some fibers do remain in the lungs or migrate to the heart, blood or lymphatic systems. See *id.* at 357 n.146. Asbestos may cause fibrosis of the heart cavity which is the formation of fibrous bodies within the lungs, heart, heart cavity or lung cavity. See *id.* at 357 n.149.

⁴²⁷ See *id.* at 356-57.

⁴²⁸ See *id.* at 357.

⁴²⁹ See Harrison, *supra* note 13, at 312 (citing EPA REPORT, *supra* note 13, at 4-15); see also Asbestos Worker Protection; Asbestos-Containing Materials in Schools; Proposed Amendments, Part IV, 59 Fed. Reg. 54,746, 54,750 (1994) (codified at 40 C.F.R. pt. 763). In 1994, over 17,000 lung cancer deaths were attributed to inhalation of carcinogens, such as asbestos, in the workplace. See Rep. No. 96-32, *supra* note 9.

⁴³⁰ See Med Access, *supra* note 421. Asbestosis is a condition resulting from scarring of the lungs with fibrous tissue as a result of the inhalation of asbestos fibers. See Stahl & Kling, *supra* note 419, at 28. Asbestosis is a leading cause of death for workers exposed to asbestos. However, some workers exposed to asbestos do not become impaired. See Kirsch, *supra* note 425, at 357 n.148.

⁴³¹ See Med Access, *supra* note 421.

⁴³² See *id.* at 1 of 3. Mesothelioma results from formation of tumors in the lungs due to the presence of asbestos fibers. It is the cancer of mesothelial cells lining the lung and heart cavity. See *id.*

⁴³³ See DAVIS & SCHAFFMAN, *supra* note 4, at 42.

⁴³⁴ Asbestosis and mesothelioma have been observed in the non-occupational setting such as persons living in the same house as exposed workers. See Kirsch, *supra* note 425, at 356-57 n.143 (citing Anderson et al., *Asbestosis Among Household Contacts of Asbestos Factory Workers*, 330 ANNALS N.Y. ACAD. SCI. 387 (1979)).

2. Control of Asbestos

Asbestos in existing construction does not create a public health problem if it remains fixed in the materials;⁴³⁵ people must come in contact with the fibers to be affected.⁴³⁶ Nonfriable asbestos-containing materials, e.g., floor tiles, will not normally release fibers and thus are considered less hazardous;⁴³⁷ friable asbestos products are the building materials most likely to release fibers. These products are easily reduced to powder and, as a result, are most susceptible to disturbance or damage.⁴³⁸ Solid materials containing asbestos are more likely to release fibers if they are subjected to grinding, vibrating, cutting, or sanding.⁴³⁹ Once asbestos fibers are released, they may remain airborne for many hours.⁴⁴⁰ As a result, the activities of a building's owners and occupants play a significant role in determining the concentration of asbestos fibers.⁴⁴¹

If a building has asbestos-containing materials, the owner should consult a professional to determine whether the asbestos is aged or damaged and whether it should be removed.⁴⁴² Friable asbestos products should immediately be repaired or removed to prevent a release.⁴⁴³ Asbestos-containing materials in good condition should not be

⁴³⁵ See Spengler, *supra* note 153, at 54.

⁴³⁶ See Stahl & Kling, *supra* note 419, at 29.

⁴³⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 41. "Friable" asbestos is asbestos that has "deteriorated or sustained physical injury such that the cohesion of the material . . . is inadequate, or which . . . lacks fiber cohesion." Asbestos-Containing Materials in Schools, 52 Fed. Reg. 41,826, 41,829 (1987) (codified as amended at 40 C.F.R. pt. 763, subpt. F).

⁴³⁸ See Asbestos-Containing Materials in Schools, 52 Fed. Reg. at 41,829.

⁴³⁹ See Kirsch, *supra* note 425, at 356. For example, vinyl flooring will not release fibers when cleaned but may release fibers if sanded, drilled, filed, or scraped. See DAVIS & SCHAFFMAN, *supra* note 4, at 43-44.

⁴⁴⁰ Asbestos fibers are microscopic in size and are also very light. See Stahl & Kling, *supra* note 419, at 28.

⁴⁴¹ Contact the Consumer Product Safety Commission (CPSC) (800-638-CPSC) or the EPA's Asbestos Hotline (202-534-1404) for more asbestos information.

⁴⁴² See EPA *Inside Story*, *supra* note 2. A home asbestos inspection may cost \$300 to \$500, or an extra \$100 to \$300 if added onto a full home inspection. Lab analysis for asbestos costs \$20 to \$35 per sample. See DAVIS & SCHAFFMAN, *supra* note 4, at 46. Asbestos-abatement contractors typically charge \$4 per square foot to remove vinyl flooring (tiles and linoleum), \$4 to \$5 per square foot to remove ceiling tiles, \$10 to \$15 per square foot to remove textured ceiling paint or plaster, up to \$20 per linear foot to remove asbestos pipe insulation, and \$30 per square foot to remove furnace insulation. The cost to hire a licensed contractor to remove and dispose of asbestos materials around a furnace, for example, could be \$1,000 to \$2,000. See *id.* at 42.

⁴⁴³ See DAVIS & SCHAFFMAN, *supra* note 4, at 43. With multifamily homes, for example, state and federal rules require landlords and building managers to survey for and repair or remove friable asbestos containing materials that could lead to exposure. See *id.* at 44.

cut or otherwise disturbed.⁴⁴⁴ If removal is appropriate, it should be done by trained and properly equipped professionals.⁴⁴⁵

The presence of asbestos in schools has received much attention from both Congress and federal agencies. EPA first regulated asbestos in schools in 1982 through regulations issued under TSCA section 6(a).⁴⁴⁶ A loan and grant program for schools with severe asbestos contamination was created under the Asbestos School Hazard Abatement Act.⁴⁴⁷ In 1986 Congress enacted TSCA amendments, known as the Asbestos Hazard Emergency Response Act (AHERA), which mandated the steps schools must take to protect students and school employees.⁴⁴⁸ Under these amendments, schools must: (1) inspect for both friable and nonfriable asbestos-containing materials; (2) prepare and submit a management plan to the governor of the state in which the school is located and make the plan available to parents of students and to school employees; and (3) determine and conduct appropriate actions to minimize the risk of exposure.⁴⁴⁹

EPA issued regulations under AHERA on October 30, 1987.⁴⁵⁰ EPA also promulgated regulations in 1986 and 1987 to protect asbestos workers involved in state or local government asbestos abatement projects that are not covered by either the OSH Act, a state plan approved by OSHA, or a state asbestos regulation that is comparable to or more stringent than EPA regulations.⁴⁵¹

⁴⁴⁴ See *id.* at 43; see also *EPA Inside Story*, *supra* note 2.

⁴⁴⁵ See *EPA Inside Story*, *supra* note 2; see also DAVIS & SCHAFFMAN, *supra* note 4, at 43. Unless proper precautions are instituted, the removal process may itself result in the release of fibers. See Kirsch, *supra* note 425, at 358. In addition, removal by an owner of a building may be illegal. In Nevada, an apartment building owner was criminally convicted for illegally removing asbestos from his buildings and violating the federal asbestos law. See DAVIS & SCHAFFMAN, *supra* note 4, at 44; see also *In re Seneca Asbestos Removal & Control, Inc.*, No. CAA-010A-1993 (EPA EAB 1997) (asbestos abatement consultant held liable for CAA Section 112 violations for improper removal); *Agency Considering Whether to Appeal ALJ Ruling on Asbestos Removal Liability*, Daily Env't Rep. (BNA) at B-3 (Jan. 31, 1997).

⁴⁴⁶ See Asbestos; Friable Asbestos-Containing Materials in Schools; Identification and Notification, 47 Fed. Reg. 23,360 (1982) (codified at 40 C.F.R. pt. 763).

⁴⁴⁷ Asbestos Hazard Emergency Response Act (AHERA), 20 U.S.C. §§ 4011-4021 (1984), current version at 15 U.S.C. § 2656 (1994).

⁴⁴⁸ TSCA §§ 201-215, 15 U.S.C. §§ 2641-2656 (1994).

⁴⁴⁹ See Stahl & Kling, *supra* note 419, at 28-29. AHERA does not mandate removal; it mandates the development of a management plan. See *id.* at 30. Stahl and Kling have reported that EPA estimated that 94 percent of schools have complied with AHERA. See *id.*

⁴⁵⁰ See Asbestos-Containing Materials in Schools, 52 Fed. Reg. 41,826 (1987) (codified as amended at 40 C.F.R. pt. 763, subpt. F).

⁴⁵¹ See Toxic Substances, Asbestos Abatement Projects, 51 Fed. Reg. 15,722 (1986) (codified at 40 C.F.R. pt. 763); see also Asbestos Abatement Projects; Worker Protection, 52 Fed. Reg.

Another significant federal action under TSCA⁴⁵² was EPA's issuance of regulations to phase out all the use of asbestos in approximately ninety-four percent of all asbestos-containing products that were manufactured in the United States.⁴⁵³ On October 18, 1991, the U.S. Court of Appeals for the Fifth Circuit vacated and remanded most of this rule for procedural failures and because EPA had not met the TSCA section 6(a) mandate to promulgate the least burdensome regulation that would adequately protect human health.⁴⁵⁴ On November 5, 1993, EPA lifted the ban on eight categories of asbestos-containing products and continued the ban on six categories of products using asbestos, including all new uses of asbestos.⁴⁵⁵ This was a fact-finding initiative⁴⁵⁶ that was followed by a technical amendment to bring the regulations in line with the court decision.⁴⁵⁷ On June 28, 1994, EPA banned asbestos-containing flooring felt and new uses of asbestos after August 27, 1990; asbestos-containing commercial paper, corrugated paper rollboard or specialty paper were banned effective August 26, 1996.⁴⁵⁸

Asbestos also is regulated pursuant to section 112 of the CAA.⁴⁵⁹ Section 112 controls emissions of hazardous air pollutants through the implementation of National Emission Standards for Hazardous Air Pollutants (NESHAPs).⁴⁶⁰ Asbestos was one of the few substances regulated under the pre-1990 CAA section 112.⁴⁶¹ Regulations issued after the 1990 CAA amendments significantly increase the legal re-

5618 (1987) (codified at 40 C.F.R. pt. 763, subpt. G). The 1986 rule extends OSHA protection to such employees. The 1987 rule replaces the 1986 rule and aims to ensure that public and private sector employees have similar levels of protection.

⁴⁵² See 15 U.S.C. §§ 2605, 2607(c) (1994).

⁴⁵³ See 40 C.F.R. § 763.160 (1996). A final rule was published in July 1989 banning the manufacture, importation, processing, and distribution in commerce of most asbestos containing products. See Asbestos; Manufacture, Importation, Processing, and Distribution in Commerce Prohibitions, 54 Fed. Reg. 29,460 (1989) (codified at 40 C.F.R. pt. 763). The rule did not ban the six percent of asbestos products that did not create a high risk of exposure and for which reasonably priced alternatives were unavailable. See *id.*

⁴⁵⁴ See *Corrosion Proof Fittings v. EPA*, 947 F.2d 1201, 1215 (5th Cir. 1991).

⁴⁵⁵ See Asbestos Manufacture, Importation, Processing, and Distribution Prohibitions, 58 Fed. Reg. 58,964 (1993) (codified at 40 C.F.R. pt. 763).

⁴⁵⁶ See *id.*

⁴⁵⁷ See Technical Amendment in Response to Court Decision on Asbestos; Manufacturer, Importation, Processing, and Distribution Prohibition, 59 Fed. Reg. 33,208 (1994) (codified at 40 C.F.R. pt. 763).

⁴⁵⁸ See 59 Fed. Reg. 33,209 (1994) (codified at 40 C.F.R. § 763.165).

⁴⁵⁹ 42 U.S.C. § 7412(b)(1) (1994).

⁴⁶⁰ The NESHAPs for asbestos are located at 40 C.F.R. §§ 61.140–61.157 (1997).

⁴⁶¹ See REITZE, *supra* note 33, at 270.

quirements applicable to asbestos abatement programs.⁴⁶² For instance, asbestos is now measured by a percent of area measurement using polarized light microscopy instead of by weight.⁴⁶³ The new regulations also expand the definition of "owner or operator," increase notification requirements,⁴⁶⁴ and include disposal requirements similar to those in RCRA.⁴⁶⁵ On July 28, 1995, EPA clarified its asbestos air toxic standard.⁴⁶⁶ Residential buildings with four or fewer dwelling units are exempt from the asbestos rule, even if they are safety hazards or public nuisances under local law.⁴⁶⁷ Multiple small buildings located on the same site and under common ownership or control are not exempt if they are demolished or renovated.⁴⁶⁸ Residential structures that are demolished as part of a commercial or public project also are not exempt.⁴⁶⁹

Under federal law, any material containing more than one percent asbestos is considered to be a regulated "asbestos-containing material," or ACM.⁴⁷⁰ EPA and OSHA have set permissible exposure levels in public buildings and for worker exposure, and require labeling of products containing asbestos in order to reduce asbestos exposure.⁴⁷¹ Federal regulations set forth work standards, i.e., use of safety equipment is mandated for employees when working where friable asbestos is present in buildings prior to demolition or major remodeling.⁴⁷² The asbestos NESHAP regulation was interpreted when the United States Court of Appeals for the Sixth Circuit decided *United States v. Midwest Suspension and Brake* on March 27, 1995.⁴⁷³ The case was a civil action brought against a brake shoe rehabilitation business for

⁴⁶² See National Emissions Standards for Hazardous Air Pollutants; Asbestos NESHAP Revision, 55 Fed. Reg. 48,406 (1990) (codified at 40 C.F.R. pt. 61).

⁴⁶³ See *id.*

⁴⁶⁴ For example, one provision requires EPA be notified of any plan to renovate a structure containing asbestos "as early as possible." 40 C.F.R. §§ 61.145(b)(1), 61.145(b)(3)(iii) (1997).

⁴⁶⁵ See National Emission Standards for Hazardous Air Pollutants; Asbestos NESHAP Revision, 55 Fed. Reg. 48,406 (1990) (codified at 40 C.F.R. pt. 61).

⁴⁶⁶ See Asbestos NESHAP Clarification of Intent, 60 Fed. Reg. 38,725 (1995) (codified at 40 C.F.R. pt. 61).

⁴⁶⁷ See *id.* at 38,735.

⁴⁶⁸ See *id.*

⁴⁶⁹ See *id.*

⁴⁷⁰ See Occupational Exposure to Asbestos, 59 Fed. Reg. 40,964, 41,057 (1994) (codified at 29 C.F.R. pts. 1910, 1915, 1926).

⁴⁷¹ See *id.*; see also Occupational Exposure to Asbestos; Corrections, 60 Fed. Reg. 33,974 (1995) (codified at 29 C.F.R. pts. 1910, 1915, 1926).

⁴⁷² See 29 C.F.R. § 1926.58 (1997).

⁴⁷³ See *U.S. v. Midwest Suspension & Brake*, 49 F.3d 1197, 1206 (6th Cir. 1995).

violation of the NESHAP for asbestos and an administrative order issued by EPA.⁴⁷⁴ Midwest supplies brakes and other parts for heavy duty trucks.⁴⁷⁵ Its brake operation includes the collection and rehabilitation of used brake shoes for resale.⁴⁷⁶ An EPA inspection found emissions of asbestos, waste disposal that released asbestos, and asbestos in the shop floor dust.⁴⁷⁷ EPA issued an administrative order requiring Midwest to comply with the regulatory "no visible emission requirement."⁴⁷⁸ Subsequent inspections of Midwest found that asbestos violations continued.⁴⁷⁹ This led the United States to bring a civil judicial action.⁴⁸⁰ The district court found for the government and ordered Midwest to pay a \$50,000 civil penalty.⁴⁸¹

Midwest appealed the district court order, claiming it was exempt under 40 C.F.R. § 61.149(a) because it was primarily an installer.⁴⁸² The U.S. Court of Appeals for the Sixth Circuit refused to allow this defense because it was first raised two years after the defendants amended their pleadings; thus, they failed to proceed with due diligence.⁴⁸³ Midwest then claimed it was not "fabricating" friction products containing commercial asbestos as required by 40 C.F.R. § 61.149(a) and, therefore, it was not "processing" asbestos.⁴⁸⁴ The appellate court upheld the district court's finding that Midwest's "cutting" and "altering" brake shoes was a process that constitutes "fabrication" within the meaning of the Act.⁴⁸⁵ Midwest next argued that dust emissions were insufficient proof of a violation because the inspector did not see asbestos particles with the naked eye.⁴⁸⁶ The asbestos NESHAP defines visible emissions as emissions detectable without the aid of instruments.⁴⁸⁷ The appellate court again upheld the district court's determination that visible emissions means visible

⁴⁷⁴ See *id.* at 1200.

⁴⁷⁵ See *id.*

⁴⁷⁶ See *id.*

⁴⁷⁷ See *id.*

⁴⁷⁸ See *Midwest Suspension & Brake*, 49 F.3d at 1200; see also 40 C.F.R. § 61.152(b) (1997).

⁴⁷⁹ See *Midwest Suspension & Brake*, 49 F.3d at 1200.

⁴⁸⁰ See *id.* at 1201.

⁴⁸¹ See *id.*

⁴⁸² See *id.*

⁴⁸³ See *id.* at 1202.

⁴⁸⁴ See *Midwest Suspension & Brake*, 49 F.3d at 1202.

⁴⁸⁵ *Id.* at 1203.

⁴⁸⁶ See *id.* at 1204.

⁴⁸⁷ See 40 C.F.R. § 61.141 (1997).

dust containing asbestos fibers that are impossible to observe with the naked eye.⁴⁸⁸ The court then upheld the \$50,000 civil penalty.⁴⁸⁹

3. OSHA Requirements

OSHA has regulated asbestos exposure since 1971.⁴⁹⁰ Approximately 1.3 million workers in construction and general industry are exposed to asbestos,⁴⁹¹ but initially the OSHA asbestos exposure regulations only applied to general industry. The first asbestos Permissible Exposure Limit (PEL), based on the national consensus standard, was 12.0 fibers per cubic centimeter (f/cc).⁴⁹² On December 7, 1971, this level was reduced to 5 f/cc using OSHA's ETS authority.⁴⁹³ It was not challenged and became a permanent standard in June 1972 through normal notice-and-comment procedures.⁴⁹⁴ In 1975, OSHA attempted to reduce the PEL to 0.5 f/cc, but its approach was rejected by the U.S. Supreme Court.⁴⁹⁵ In 1976, OSHA reduced the standard to 2 f/cc.⁴⁹⁶ On November 4, 1983, OSHA lowered the PEL for asbestos to 0.5 f/cc using its ETS authority.⁴⁹⁷ In 1984 the U.S. Court of Appeals for the Fifth Circuit held that OSHA did not invoke its ETS powers properly and struck down the regulation.⁴⁹⁸

OSHA revised the permanent asbestos standard from 2 to 0.2 f/cc in 1986.⁴⁹⁹ The OSHA standard for general industry occupational exposure to asbestos of 0.2 f/cc was extended on June 17, 1986, to the construction injury.⁵⁰⁰ The standard was generally upheld in *Building and Construction Trades Department v. Brock* but resulted in nine

⁴⁸⁸ See *Midwest Suspension & Brake*, 49 F.3d at 1204.

⁴⁸⁹ See *id.* at 1205.

⁴⁹⁰ See *Asbestos Info. Ass'n v. OSHA*, 727 F.2d 415, 418 (5th Cir. 1984).

⁴⁹¹ See Rep. No. 96-32, *supra* note 9.

⁴⁹² See *Asbestos Info. Ass'n*, 727 F.2d at 418 n.6; 20 U.S.C. § 655(a) (1994).

⁴⁹³ See *Asbestos Info. Ass'n*, 727 F.2d at 418 n.6.

⁴⁹⁴ See *id.* at 418.

⁴⁹⁵ See *Industrial Union Dep't, AFL-CIO v. American Petroleum Inst.*, 448 U.S. 608, 608 (1980).

⁴⁹⁶ Occupational Safety and Health Standards, 29 C.F.R. pt. 1910 (1997).

⁴⁹⁷ See Occupational Exposure to Asbestos, 48 Fed. Reg. 51,086 (1983) (codified at 29 C.F.R. pt. 1910).

⁴⁹⁸ See *Asbestos Info. Ass'n*, 727 F.2d at 417.

⁴⁹⁹ See Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite, 51 Fed. Reg. 22,612 (1986) (codified at 29 C.F.R. pts 1910, 1926).

⁵⁰⁰ See *id.*

issues being remanded to OSHA.⁵⁰¹ OSHA removed some minerals from the coverage of the asbestos standards on June 8, 1992.⁵⁰²

OSHA published a final rule concerning occupational exposure to asbestos on August 10, 1994.⁵⁰³ The final rule amends the OSHA standards issued on June 17, 1986,⁵⁰⁴ for occupational exposure to asbestos in general industry and in the construction industry.⁵⁰⁵ There is also a separate standard covering occupational exposure to asbestos in the shipyard industry.⁵⁰⁶ The effective date of these amendments is October 11, 1994.⁵⁰⁷ The standards specify various start-up dates.⁵⁰⁸ Major revisions to these standards include a reduced time-weighted-average PEL of 0.1 f/cc for all asbestos work, and a new classification scheme for asbestos construction and shipyard industry work that ties mandatory work practices to work classification.⁵⁰⁹ There now is a presumptive asbestos identification requirement for building materials containing "high hazard" asbestos, limited notification requirements for employers using unlisted compliance methods in high risk asbestos abatement work, and mandatory methods of control of asbestos during brake and clutch repair.⁵¹⁰

The asbestos rules applicable to owners of buildings built before 1981 became effective on October 1, 1995.⁵¹¹ This regulation, nearly two hundred pages long, has been expanded by two sets of OSHA clarifications and corrections.⁵¹² The construction standard applies to almost any activity that disturbs material containing asbestos, or that is presumed to contain asbestos, in commercial buildings.⁵¹³ The regulations require specific work practices to be followed that are designed

⁵⁰¹ See generally *Building & Constr. Trades Dep't v. Brock*, 838 F.2d 1258 (D.C. Cir. 1988).

⁵⁰² See *Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite*, 57 Fed. Reg. 24,310 (1992) (codified at 29 C.F.R. pts. 1910, 1926).

⁵⁰³ See *Occupational Exposure to Asbestos*, 59 Fed. Reg. 40,964 (1994) (codified at 29 C.F.R. pts. 1910, 1915, 1926).

⁵⁰⁴ See 51 Fed. Reg. at 22,612; see also 29 C.F.R. § 1910.1001 (1997).

⁵⁰⁵ See 29 C.F.R. § 1926.1101 (1997).

⁵⁰⁶ See 29 C.F.R. § 1915.1001 (1997).

⁵⁰⁷ See 59 Fed. Reg. at 40,964.

⁵⁰⁸ See *id.*

⁵⁰⁹ See *id.*

⁵¹⁰ See *id.*

⁵¹¹ See *id.*

⁵¹² See *Occupational Exposure to Asbestos; Corrections, Part III*, 60 Fed. Reg. 33,974 (1995) (codified at 29 C.F.R. pts. 1910, 1915, 1926); *Occupational Exposure to Asbestos; Final Rule Amendments*, 60 Fed. Reg. 50,411 (1995) (codified at 29 C.F.R. pts. 1915, 1926); *Occupational Exposure to Asbestos; Tremolite, Anthophyllite, and Actinolite*, 61 Fed. Reg. 43,454 (1996) (codified at 29 C.F.R. pts. 1910, 1915, 1926).

⁵¹³ See 59 Fed. Reg. 41,019 (1994).

to prevent the release to the air of asbestos fibers.⁵¹⁴ Workers exposed to airborne asbestos levels of 0.1 f/cc must be protected through the use of personal protection gear according to OSHA and EPA regulations.⁵¹⁵

Most of the revisions in the amended standards are the final response to an order of the Court of Appeals for the District of Columbia Circuit, which upheld the 1986 standards but remanded certain issues for reconsideration.⁵¹⁶ OSHA previously made changes in response to the court order on December 14, 1989,⁵¹⁷ and February 5, 1990.⁵¹⁸ OSHA issued a notice correcting and clarifying certain of these provisions on June 29, 1995.⁵¹⁹ OSHA further corrected and clarified the construction and shipyard employment standards on September 29, 1995, but did not amend the general industry standards.⁵²⁰ The amendments became effective October 1, 1995.⁵²¹ The Building and Construction Trades Department of the AFL-CIO challenged these job-related asbestos standards in the D.C. Circuit.⁵²² The American Petroleum Institute filed a motion to intervene, arguing that a victory by the union could have a direct effect on companies that are members of the API.⁵²³ In late 1995, the D.C. Circuit transferred the case and two related cases to the Fifth Circuit.⁵²⁴

E. Lead

Humans have used lead for thousands of years because it has a number of desirable characteristics; however, they also have been

⁵¹⁴ See James B. Witkins, *Owners Face New Asbestos Rules*, LEGAL TIMES, Oct. 16, 1995, at S27; see also 29 C.F.R. § 1910.1001 (1997).

⁵¹⁵ 29 C.F.R. § 1926.58 (1997).

⁵¹⁶ See generally *Building & Constr. Trades Dep't v. Brock*, 838 F.2d 1258 (D.C. Cir. 1988).

⁵¹⁷ See *Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite*, 54 Fed. Reg. 52,024 (1989) (codified at 29 C.F.R. pts. 1910, 1926).

⁵¹⁸ See *Occupational Exposure to Asbestos*, 55 Fed. Reg. 3724 (1990) (codified at 29 C.F.R. pts. 1910, 1926).

⁵¹⁹ See *Occupational Exposure to Asbestos; Corrections*, 60 Fed. Reg. 33,974 (1995) (codified at 29 C.F.R. pts. 1910, 1915, 1926).

⁵²⁰ See *Occupational Exposure to Asbestos*, 60 Fed. Reg. 50,411 (1995) (codified at 29 C.F.R. pts. 1915, 1926).

⁵²¹ See *id.*

⁵²² See generally *Building & Constr. Trades Dep't v. OSHA*, No. 95-1584, 1995 WL 791559 (D.C. Cir. Dec. 14, 1995).

⁵²³ See *Chemical Safety: API Seeks to Intervene in BCTD's Asbestos Challenge*, Daily Env't Rep. (BNA) at A-3 (Dec. 13, 1995).

⁵²⁴ See generally *Building & Constr. Trades Dep't*, 1995 WL 791559, at *1.

aware for nearly as long of the fact that it poses human-health risks.⁵²⁵ Its toxicity was first reported by Eberhard Gochel in 1697.⁵²⁶ Nevertheless, it continued to be used as a food additive, as a glaze, and in pipes; thus human injury continues to the present day.⁵²⁷ Today, lead-based paint is the primary source of the indoor air health hazard created by the use of lead.⁵²⁸ From the turn of the century, lead was used as an ingredient in many oil-based paints because it improved the adherence, brightness, and durability of the paint.⁵²⁹ Two-thirds of the houses built before 1940, one-third of those built between 1940 and 1960, and some homes built after 1960 were painted with lead-based paints.⁵³⁰ The use of lead in paint produced for residential use was prohibited in 1978 because lead paint flakes off of walls and later is inhaled or ingested by children.⁵³¹ Exposure to lead also occurs when lead-based paint is removed by scraping, sanding, or open flame burning.⁵³² Other sources of lead found in indoor environments include lead use in activities such as soldering and stained glass artwork.⁵³³ Contaminated air, drinking water, food, soil, and dust⁵³⁴ provide additional avenues of exposure to humans.⁵³⁵ Cigarette smoke is also a source of lead.⁵³⁶ Other significant sources of lead exposure may occur from ambient air. Lead is controlled as a criteria pollutant under the CAA,⁵³⁷ but less attention is given to lead compared with the other criteria pollutants. Automobile exhaust from vehicles using leaded

⁵²⁵ See Occupational Exposure to Lead, 43 Fed. Reg. 52,952 (1978) (codified at 29 C.F.R. pt. 1910).

⁵²⁶ See generally Josef Eisinger, *Sweet Poison*, 105 NATURAL HIST. 48 (1996).

⁵²⁷ See *id.*

⁵²⁸ See Donald E. Lively, *The Diminishing Relevance of Rights: Racial Disparities in the Distribution of Lead Exposure Risks*, 21 B.C. ENVTL. AFF. L. REV. 309, 315 (1993). The other major source of risk is drinking water.

⁵²⁹ See DAVIS & SCHAFFMAN, *supra* note 4, at 115.

⁵³⁰ See National Primary and Secondary Ambient Air Quality Standards for Lead, 43 Fed. Reg. 46,246 (1978) (codified at 40 C.F.R. pt. 50).

⁵³¹ See U.S. Env'tl. Protection Agency, *Lead Poisoning and Your Children* (visited Nov. 23, 1997) <<http://www.epa.gov/ledweb00/pubs/lead.html>>; see also Lively, *supra* note 528, at 315.

⁵³² See EPA *Inside Story*, *supra* note 2.

⁵³³ See U.S. Dept. of Health and Human Serv., Public Health Serv., Centers for Disease Control and Prevention, *Preventing Lead Poisoning in Young Children* (last modified Oct. 1991) <<http://www.cdc.gov/nceh/pubcatns/1994/cdc/books/plpsn/plpsn0-3.htm>>.

⁵³⁴ Recently, CPSC issued a warning concerning lead dust hazard from deteriorating cheap vinyl miniblinds. See DAVIS & SCHAFFMAN, *supra* note 4, at 116.

⁵³⁵ See *id.*

⁵³⁶ See Agency for Toxic Substances and Disease Registry, *Public Health Statement: Lead* (visited July 7, 1997) <<http://atsdrl.atsdr.cdc.gov:8080/ToxProfiles/phs8817.html>>.

⁵³⁷ Clean Air Act § 109, 42 U.S.C. § 7407 (1994); 40 C.F.R. § 50.12 (1997) (setting out the national primary and secondary ambient air quality standards for lead).

gasoline was previously one of the major sources of lead exposure; however, lead is no longer a gasoline additive.⁵³⁸ Today most ambient air lead exposure is due to industries that release lead. For example, battery recycling plants release lead-containing acid mist; lead is also found in their exhaust plumes.⁵³⁹ The United States produces twenty percent of the world's lead; most of it is used to produce storage batteries.⁵⁴⁰

1. Health Effects of Lead

Lead particles generally enter the body through inhalation or ingestion. Lead is especially toxic because it accumulates in the blood and soft tissues of the body and is absorbed by the bones.⁵⁴¹ Lead trapped in the bone structure does not present a health threat.⁵⁴² Lead can be released, however, when bones are broken, when a person is bedridden, or as a result of bone disease.⁵⁴³ During pregnancy, lead that accumulates in the mother can harm the fetus because the calcium in the mother's bones is the fetus's source of calcium.⁵⁴⁴

Lead affects virtually every system of the body. It can damage the brain, kidneys, peripheral nervous system, and red blood cells and may cause high blood pressure.⁵⁴⁵ While it is harmful to individuals of all ages, lead exposure can be especially damaging to children, fetuses,

⁵³⁸ See generally Arnold W. Reitze, Jr., *The Regulation of Fuels and Fuel Additives Under Section 211 of the Clean Air Act*, 29 TULSA L.J. 485 (1994).

⁵³⁹ See Mark Jaffe, *The Lethal Legacy of Lead Poisoning: Long After a Battery Plant Shuts Down, Contamination Lingers In Soil and Bones*, WASH. POST, May 28, 1991, at Z7. On July 31, 1991, the U.S. Department of Justice launched a crackdown on several industrial sources of lead. The Justice Department filed 24 cases in federal district courts, and EPA issued 12 administrative orders. These actions were directed against several major industrial lead sources including battery, ceramic, and electronic firms. See *Enforcement: Justice, EPA, Launch Crackdown on Lead, File 36 Actions Using Six Environmental Laws*, 22 Env't Rep. (BNA) at 827 (Aug. 2, 1991). Only one of these cases has been decided. See generally *United States v. NL Indus. Inc.*, 936 F. Supp. 545 (S.D. Ill. 1996). In *United States v. NL Industries* the judge held that the federal Superfund law does not give federal courts jurisdiction to enjoin an ongoing environmental cleanup. See *id.* at 563; see also *Illinois Judge Denies City's Move to Enjoin Cleanup of CERCLA Site*, 5 Mealey's Litig. Rep.: Lead (page unavail.) (Sept. 5, 1996). For a complete list of all of the cases, see *EPA, Justice Department Form Joint Effort in Lead Cases*, 1 Mealey's Litig. Rep.: Lead 1 (Oct. 3, 1991).

⁵⁴⁰ See Agency for Toxic Substances and Disease Registry, *Public Health Statement; Lead* (1990) <<http://atsdr1.atsdr.cdc.gov:8080/ToxProfiles/phs8817.html>>.

⁵⁴¹ See DAVIS & SCHAFFMAN, *supra* note 4, at 116.

⁵⁴² Jaffe, *supra* note 539, at Health 7.

⁵⁴³ See *id.*

⁵⁴⁴ See *id.*

⁵⁴⁵ See *EPA Inside Story*, *supra* note 2.

and women of childbearing age.⁵⁴⁶ This increased sensitivity to lead exposure is attributed to several factors. Children absorb lead more readily and their tissues are more sensitive.⁵⁴⁷ A child's lower body weight reduces the amount of lead necessary to generate higher concentrations at lower exposures.⁵⁴⁸ Children are more likely to encounter lead because they are inclined to place items in their mouths, including sources of lead such as paint chips.⁵⁴⁹ Lead contamination in children may lead to "delays in physical and mental development, lower IQ levels, shortened attention spans, and increased behavioral problems."⁵⁵⁰ Due to the potential for significant harmful effects on health, the control of lead exposure among children is particularly important.

Lead poisoning is a "silent disease" because its effects may occur gradually and imperceptibly. Often there are no obvious symptoms of lead poisoning. "Blood-lead levels as low as 10 ug/dL (micrograms per deciliter) have been associated with learning disabilities, growth impairment, permanent hearing and visual impairment, and other damage to the brain and nervous system. In large doses, lead exposure can cause brain damage, convulsions, and even death."⁵⁵¹ Lead exposure before or during pregnancy can alter fetal development and cause miscarriages. Recent studies have identified previously unrecognized effects, leading to increasing concern about blood-lead levels once thought to be safe.⁵⁵² Since 1978, the CDC has lowered the blood-lead level of concern from sixty ug/dL to ten ug/dL.⁵⁵³

In 1991, the Secretary of Health and Human Services said lead poisoning was the "number one environmental threat to the health of children in the United States."⁵⁵⁴ The percentage of children with elevated blood-lead levels has declined over the last twenty years, but

⁵⁴⁶ See Final Rule, Lead; Requirements for Disclosure of Known Lead-Based Paint and/or Lead-Based Paint Hazards in Housing, 61 Fed. Reg. 9064, 9065 (1996) (codified at 40 C.F.R. pt. 745; 24 C.F.R. pt. 35). Approximately 15 percent of American children have elevated blood-lead levels. See Lively, *supra* note 528, at 312.

⁵⁴⁷ See *EPA Inside Story*, *supra* note 2.

⁵⁴⁸ See U.S. ENVTL. PROTECTION AGENCY, ENVIRONMENTAL HAZARDS IN OUR SCHOOL: A RESOURCE HANDBOOK 26.27 (1990).

⁵⁴⁹ See *EPA Inside Story*, *supra* note 2.

⁵⁵⁰ *Id.*

⁵⁵¹ *Id.*

⁵⁵² See Lead; Requirements for Disclosure of Known Lead-Based Paint and/or Lead-Based Paint Hazards in Housing, Final Rule, 61 Fed. Reg. 9065 (1996).

⁵⁵³ See *id.*

⁵⁵⁴ *Id.*

millions of children have blood-lead levels high enough to threaten their health.⁵⁵⁵ Lead is assigned the highest value possible under the Hazard Ranking System (HRS) used under CERCLA because of its high toxicity without a demonstrated threshold below which it causes no adverse health effects.⁵⁵⁶

2. Control of Lead

Lead is regulated as a criteria pollutant in the atmosphere with a standard set at not more than 1.5 ug/m³ (micrograms per cubic meter) of air based on a three month average.⁵⁵⁷ There is no generally applicable indoor standard for lead.

Lead-based paint (LBP) is probably the most prevalent source of indoor lead exposure in older homes.⁵⁵⁸ If LBP in a home is not cracking or peeling, it should be left undisturbed. However, if the paint is damaged and may result in exposure, corrective action is necessary. When LBP is being removed, all non-essential workers should leave the area and workers should wear protective gear.⁵⁵⁹ A dry scraper, belt sander, propane torch, or heat gun should never be used to remove LBP;⁵⁶⁰ paint chips or dust should not be cleaned up with a household vacuum.⁵⁶¹ Costs to remove LBP completely can be \$15,000 to \$20,000 or more for a single-family home or \$10,000 per unit in multifamily housing—these costs can exceed the value of a home.⁵⁶² These estimates do not include costs for temporary housing during the lead removal process or the disposal costs.

⁵⁵⁵ See *id.* Note that in a 1988 study by CDC, EPA reported that lead levels in the blood of African-American children under the age of five greatly exceeded levels in similarly-aged white children in the same cities. See AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY, CDC, *THE NATURE AND EXTENT OF LEAD POISONING IN CHILDREN IN THE U.S.: A REPORT TO CONGRESS* (1988); see also Michael Fisher, *Environmental Racism Claims Brought Under Title VI of the Civil Rights Act*, 25 ENVTL. L. 285, 298 (1995) (stating that people of color have greater chance of being exposed to health-threatening work environments than whites).

⁵⁵⁶ See *RSR Corp. v. EPA*, 102 F.3d 1266, 1267 (D.C. Cir. 1997).

⁵⁵⁷ See National Primary and Secondary Ambient Air Quality Standards for Lead, 43 Fed. Reg. 46,246 (1978) (codified at 40 C.F.R. pt. 50).

⁵⁵⁸ Generally, it should be expected that a pre-1960 home contains significant amounts of lead-based paint; a home built before 1978 also is more likely to contain lead-based paint unless it has been renovated. See DAVIS & SCHAFFMAN, *supra* note 4, at 123.

⁵⁵⁹ See generally LEAD-BASED PAINT HAZARDS ASSESSMENT AND MANAGEMENT (Vincent M. Coluccio ed., 1994).

⁵⁶⁰ See OSHA, U.S. DEPT OF LABOR, OSHA No. 93-47, LEAD EXPOSURE IN CONSTRUCTION 353-80 (1993).

⁵⁶¹ See DAVIS & SCHAFFMAN, *supra* note 4, at 119.

⁵⁶² See *id.* at 117.

LBP may be an issue when insuring or financing a home. Most insurance policies contain specific lead exclusions; even where there is no specific exclusion, lead may be excluded through a broad pollution exclusion clause.⁵⁶³ Further, many banks and mortgage companies require lead tests *before* they will provide financing.⁵⁶⁴ A lessor of a home or apartment containing LBP may be exposed to liability based on tort if a tenant or the tenant's child ingests lead.⁵⁶⁵ Several states have enacted statutes that create a private cause of action against property owners who fail to abate hazards created by lead.⁵⁶⁶

Federal, state,⁵⁶⁷ and local governments have responded to the problem of lead-based paint by enacting lead-based paint abatement laws.⁵⁶⁸ In the 1950s and 1960s, cities began regulating lead-based paint use, which several cities banned entirely.⁵⁶⁹ In 1971, Congress passed the Lead-Based Paint Poisoning Prevention Act (LPPPA).⁵⁷⁰ Among other things, the LPPPA prohibited the use of lead-based paint in housing for which the federal government provided financing or construction support.⁵⁷¹ The federal government adopted both a "health approach" and a "housing approach" in the LPPPA.⁵⁷² Several states also adopted abatement laws, but they are based on the health approach.⁵⁷³ HUD then passed regulations banning the use of lead-

⁵⁶³ See *id.* at 118.

⁵⁶⁴ See *id.*

⁵⁶⁵ See *Lanthier v. Feroletto*, 654 N.Y.S.2d 531, 531 (N.Y. App. Div. 1997) (plaintiff failed to show landlord had actual or constructive notice of lead-based paint for a period of time so that in the exercise of reasonable care it should have been corrected); see generally ARNOLD & PORTER, 8 ENVTL. LAW IN NEW YORK 101 (Matthew Bender 1997).

⁵⁶⁶ See, e.g., Lead Poisoning Prevention and Control, LA. REV. STAT. ANN. § 40:1299.29 (West, WESTLAW through 1996 Exec. Sess. & Reg. Sess.); ME. REV. STAT. ANN. tit. 22, § 1326 (West, WESTLAW through 1996 Sess.); MASS. GEN. LAWS ANN. ch. 111, § 199 (Lawyers' Coop. Publ'g, LEXIS through 1996 Sess.); VA. CODE ANN. § 36-107.1 (Michie, LEXIS through 1997 Supp.).

⁵⁶⁷ See Jennifer Tiller, *Easing Lead Paint Laws: A Step in the Wrong Direction*, 18 HARV. ENVTL. L. REV. 265, 268 (1994).

⁵⁶⁸ See Michele Gilligan & Deborah Ann Ford, *Investor Response to Lead-Based Paint Abatement Laws: Legal and Economic Considerations*, 12 COLUM. J. ENVTL. L. 243, 244, 259-78 (1987). What is allowable as proper abatement varies from state to state. See DAVIS & SCHAFFMAN, *supra* note 4, at 120.

⁵⁶⁹ The cities of Chicago, Cincinnati, New Haven, New York, Philadelphia, St. Louis, and Washington, D.C. banned it. See DAVIS & SCHAFFMAN, *supra* note 4, at 121.

⁵⁷⁰ See 42 U.S.C. §§ 4821-4846 (1994).

⁵⁷¹ See Gilligan & Ford, *supra* note 568, at 262.

⁵⁷² See *id.* at 267-68.

⁵⁷³ See *id.* at 268. As of 1994, states with required abatement of lead hazards were Arkansas, California, Connecticut, Delaware, Florida, Illinois, Iowa, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Minnesota, Missouri, New Hampshire, New Jersey, New York, North

based paint in HUD-associated housing the following year.⁵⁷⁴ The Consumer Product Safety Commission (CPSC) banned lead in house paint beginning in 1978; specifically, the CPSC banned the use of paints with more than 0.06 percent lead by weight.⁵⁷⁵ In 1983 HUD was directed to restructure its lead-based paint program in *Ashton v. Pierce*.⁵⁷⁶ In 1988 Congress amended LPPPA⁵⁷⁷ and on April 8, 1990, HUD promulgated interim guidelines to implement the Act.⁵⁷⁸

In 1992, Congress passed the Residential Lead-Based Paint Hazard Reduction Act (1992 Act).⁵⁷⁹ This Act amends the LPPPA and adds sections 401 through 408 to TSCA.⁵⁸⁰ It applies to the sale or lease of housing constructed before the phaseout of residential lead-based paint use in 1978.⁵⁸¹ The new law requires EPA and HUD to promulgate joint regulations for disclosure of any known lead-based paint or any known lead-based paint hazards in target housing offered for sale or lease.⁵⁸² Specifically, section 1018 requires the following activities before a purchaser or lessee is obligated under a contract to purchase or lease target housing: (1) sellers and lessors must provide purchasers and lessees with a lead hazard information pamphlet, as developed under section 406(a) of TSCA; (2) sellers and lessors must disclose the presence of known lead-based paint and/or lead-based paint hazards in such housing and provide purchasers and lessees with any lead hazard evaluation report available to the seller or lessor; (3) sellers must permit purchasers ten days to conduct a risk assessment or inspection for the presence of lead-based paint hazards; and (4) sales contracts must include an attached lead warning statement and acknowledgement, signed by the purchaser.⁵⁸³

Carolina, South Carolina, Rhode Island, and Wisconsin. See DOUG FARQUHAR, LEAD POISONING PREVENTION: A GUIDE FOR LEGISLATORS, Table 1 (1994).

⁵⁷⁴ See Lead-Based Paint Poisoning Prevention in HUD-Associated Housing and Federally Owned Property to be Sold for Residential Habitation, 41 Fed. Reg. 28,875 (1976) (codified at 24 C.F.R. § 35).

⁵⁷⁵ See Lead-Containing Paint and Certain Consumer Products Bearing Lead-Containing Paint, Final Rule, 42 Fed. Reg. 44,191 (1977) (codified at 16 U.S.C. § 1303).

⁵⁷⁶ *Ashton v. Pierce*, 723 F.2d 70, 70 (D.C. Cir. 1983).

⁵⁷⁷ 42 U.S.C. §§ 300j-21 to 300j-26 (1994).

⁵⁷⁸ See Lead Based Paint: Interim Guidelines for Hazard Identification and Abatement in Public and Indian Housing, 55 Fed. Reg. 14,556 (1990).

⁵⁷⁹ This act was Title X of the Housing and Community Development Act, Pub. L. No. 102-550, 106 Stat. 3887 (1992) (codified at various sections of Titles 15 and 42 of U.S. Code).

⁵⁸⁰ See 15 U.S.C. §§ 2681-2688 (1994).

⁵⁸¹ See 42 U.S.C. §§ 4851b(27), 4852d (1994).

⁵⁸² See 42 U.S.C. § 4851b(27) (defining target housing).

⁵⁸³ See 42 U.S.C. § 4851b(1). Lead-based paint and its health hazard to children are discussed

Final regulations implementing part of the 1992 Act were published on March 6, 1996.⁵⁸⁴ These regulations require sellers and landlords of pre-1978 housing to:

Disclose the presence of known lead-based paint or lead-based paint hazards in housing and provide the buyer/tenant any available information (including all records, reports, and test data); provide disclosure and acknowledgement language containing a "Lead Warning Statement" (must be worded precisely as set forth in the regulations); provide the buyer or tenant with an EPA/HUD-approved lead hazard information pamphlet; and allow the buyer ten days to conduct a lead-based paint inspection or risk assessment before becoming obligated to purchase the house.⁵⁸⁵

Real estate agents must also ensure that sellers and landlords comply with the Act's requirements.⁵⁸⁶ Sellers and landlords, and their real estate agents, can be liable for triple damages, legal and expert fees, and court costs, not to mention possible fines and even imprisonment, for nondisclosure under the new law.⁵⁸⁷ Sellers and landlords must keep the required records and disclosure and acknowledgment contract for three years.⁵⁸⁸ Be aware that sellers and landlords of housing built *after* 1978 still may have LBP disclosure obligations if they have knowledge of the presence of LBP.⁵⁸⁹ The law does not force sellers to inspect or test a home for lead before a sale or to fix a lead hazard, if discovered. The purchaser is responsible for financing any inspection or risk assessment.⁵⁹⁰ Many state and local laws are similar to or more stringent than the 1992 Act.⁵⁹¹ Some states also have LBP regulations governing property condition disclosures; home inspections and testing; lead-poisoning prevention, screening/testing, control, and follow-

in a law review article that questions the economic efficiency of state and federal regulation. See generally Thomas J. Miceli et al., *Protecting Children from Lead-Based Paint Poisoning: Should Landlords Bear the Burden?*, 23 B.C. ENVTL. AFF. L. REV. 1 (1995).

⁵⁸⁴ See Lead; Requirements for Disclosure of Known Lead-Based Paint and/or Lead-Based Paint Hazards in Housing, 61 Fed. Reg. 9064 (1996) (codified at 40 C.F.R. pt. 745; 24 C.F.R. pt. 35); see also Nicolas M. Kublicki, *Heavy Metal: Residential Lead Rule*, 10 PROB. & PROP., Sept.-Oct. 1996, at 39.

⁵⁸⁵ Final Rule, Lead; Requirements for Disclosure of Known Lead-Based Paint and/or Lead-Based Paint Hazards in Housing, 61 Fed. Reg. 9064-9088 (1996).

⁵⁸⁶ See 42 U.S.C. § 4852d(a)(4) (1994).

⁵⁸⁷ 61 Fed. Reg. at 9077, 9078, 9085.

⁵⁸⁸ 61 Fed. Reg. at 9076, 9081, 9088.

⁵⁸⁹ See *id.*

⁵⁹⁰ 61 Fed. Reg. at 9077.

⁵⁹¹ DAVIS & SCHAFFMAN, *supra* note 4, at 122.

up in poisoning incidents; training, licensing, and certification of inspectors and contractors; and abatement options.⁵⁹²

The LBP hazard pamphlet required by TSCA section 406(a) was available as of August 1, 1995.⁵⁹³ The regulations required by TSCA section 403 were not included in the March 6, 1996 promulgation, but an interim guidance document was issued on September 11, 1995.⁵⁹⁴ The regulations required by TSCA section 402 were proposed on September 2, 1994,⁵⁹⁵ and finalized August 8, 1996.⁵⁹⁶

Two other statutes help to control lead in drinking water. Amendments to the Safe Drinking Water Act control lead through a prohibition on the use of lead pipes, solder, or flux.⁵⁹⁷ The statute mandates that the use of "any pipe, solder, or flux, which is used after June 19, 1986, in the installation or repair of - (A) any public water system, or (B) . . . residential or non-residential facility . . . shall be lead free."⁵⁹⁸ The second statute is the Lead Contamination Control Act of 1988 (LCCA).⁵⁹⁹ Under this statute no "drinking water coolers"⁶⁰⁰ may be sold in interstate commerce unless they are "lead free."⁶⁰¹ The LCCA also requires a recall of drinking water coolers with lead-lined tanks.⁶⁰² Finally, the Act requires that remediation programs be developed and implemented for school drinking water systems.⁶⁰³ EPA regulations limit lead in drinking water to 0.015 milligrams per liter.⁶⁰⁴

⁵⁹² See generally Miceli, *supra* note 583, at 1; FARQUHAR, *supra* note 573; Tiller, *supra* note 567, at 266-67.

⁵⁹³ See Lead Hazard Information Pamphlet; Notice of Availability, 60 Fed. Reg. 39,167 (1995). Since 1995, several states have expressed desire to seek approval to use their own pamphlets in lieu of this federal pamphlet. EPA and HUD encourage states to seek approval for their own pamphlets. See Final Rule, Lead; Requirements for Disclosure of Known Lead-Based Paint and/or Lead-Based Paint Hazards in Housing, 61 Fed. Reg. 9064, 9071 (1996) (codified at 40 C.F.R. pt. 745; 24 C.F.R. pt. 35).

⁵⁹⁴ See Guidance on Identification of Lead-Based Paint Hazards, 60 Fed. Reg. 47,248 (1995).

⁵⁹⁵ See Lead; Requirements for Lead-Based Paint Activities, 59 Fed. Reg. 45,872 (1994) (codified at 40 C.F.R. pt. 745).

⁵⁹⁶ See Lead; Requirements for Lead-Based Paint Activities in Target Housing and Child-Occupied Facilities, 61 Fed. Reg. 45,778 (1996) (codified at 40 C.F.R. pt. 745).

⁵⁹⁷ See 42 U.S.C. § 300g-6 (1994).

⁵⁹⁸ 42 U.S.C. § 300g-6(a)(1).

⁵⁹⁹ See 42 U.S.C. § 300j-21 (1988).

⁶⁰⁰ See 42 U.S.C. § 300j-23. "The term 'drinking water cooler' means any mechanical device affixed to drinking water supply plumbing which actively cools water for human consumption." 42 U.S.C. § 300j-21(1).

⁶⁰¹ "Lead free" means that no portion of a drinking water cooler which comes in contact with water may contain more than eight percent lead, and the interior of any drinking water cooler tanks may not be composed of more than two percent lead. 42 U.S.C. § 300j-21(2).

⁶⁰² 42 U.S.C. § 300j-22.

⁶⁰³ 42 U.S.C. § 300j-24.

⁶⁰⁴ See Drinking Water Regulations: Maximum Contaminant Level Goals and National Pri-

3. Lead Under the OSH Act

Workers in the following industries may be exposed to lead: battery manufacturing and repair; demolition/renovation of old homes or other structures; automobile assembly, auto body and radiator repair; secondary lead smelting (recovery of lead from batteries); lead scrap smelting or metal founding; inorganic chemical manufacturing; ammunition manufacturing; brick making; cable making and splicing; cutlery manufacturing; fish sinker manufacturing; jewelry making; plastic manufacturing; pottery making; roofing; glass and stained glass manufacturing; welding; law enforcement; plumbing; rubber manufacturing; and ship building.⁶⁰⁵

In 1978 OSHA issued a rule to protect workers from airborne lead exposure.⁶⁰⁶ Because lead is not carcinogenic, OSHA could not rely on any policy that assumes there is no safe level for lead.⁶⁰⁷ The agency amassed voluminous evidence of specific harmful effects at various air-lead levels.⁶⁰⁸ The agency then promulgated a standard not only to prevent overt early symptoms of the disease but subclinical effects as well.⁶⁰⁹ Although the lead standard was significantly more stringent than earlier standards, it was upheld for all but a few industries in *United Steelworkers of America, AFL-CIO-CLV v. Marshall*.⁶¹⁰ The OSH Act's lead regulations include Medical Removal Protection (MRP) that includes requirements for medical monitoring and, if necessary, removal of the worker from high-exposure workplaces without loss of wages or seniority.⁶¹¹

On June 3, 1993, OSHA's interim standard for lead in the construction industry went into effect.⁶¹² This brought the construction industry's legal responsibilities in line with the General Industry Standard that applies to all industrial settings except construction and agricultural workers.⁶¹³ Construction work is defined to include construction,

mary Drinking Water Regulations for Lead and Copper, 56 Fed. Reg. 26,460 (1991) (codified at 40 C.F.R. pts. 141, 142).

⁶⁰⁵ See COLUCCIO, *supra* note 559, at 13.

⁶⁰⁶ See Occupational Exposure to Lead, 43 Fed. Reg. 52,952 (1978) (codified at 29 C.F.R. § 1810), with minor amendments at Occupational Exposure to Lead; Administrative Stay; Reconsideration; Corrections, 44 Fed. Reg. 5446 (1979) (codified at 29 C.F.R. pt. 1910.1025).

⁶⁰⁷ See ASHFORD & CALDART, *supra* note 97, at 151.

⁶⁰⁸ See 43 Fed. Reg. at 52,954-63.

⁶⁰⁹ See *id.* at 52,963-64.

⁶¹⁰ See *United Steelworkers of America, AFL-CIO-CLV v. Marshall*, 647 F.2d 1189, 1311 (D.C. Cir. 1980).

⁶¹¹ See 29 C.F.R. § 1926.62 (1997).

⁶¹² See 29 C.F.R. § 1926.62(p).

⁶¹³ See 29 C.F.R. § 1910.25 (1997).

alteration and/or repair, including painting and decorating.⁶¹⁴ It includes demolition, removal or encapsulation, salvage, and cleanup activities.⁶¹⁵ A permissible exposure limit (PEL) was set at 50 ug/m³, averaged over an eight-hour period, and an action level⁶¹⁶ was set at 30 ug/m³, which is the same as the General Industry Standard that applies to all industrial workers.⁶¹⁷

F. VOCs

Volatile Organic Compounds (VOCs)⁶¹⁸ are released as gases from many items in the home, including wood finishes, paints, lacquers, thinners, adhesives, and consumer products like rug and oven cleaners, particleboard furniture, pesticides, home furnishings, perfumes, and skin lotion.⁶¹⁹ Architectural coatings (paints and finishes) account for nine percent of all VOC product emissions.⁶²⁰ In 1987, EPA found that VOC concentrations are consistently higher indoors than out—up to ten times higher.⁶²¹ Exposure to VOCs can produce a variety of adverse health effects including skin rash, eye and upper respiratory irritation, nasal congestion and inflammation, nose bleeds, headache, nausea, vomiting, fatigue, and dizziness.⁶²² Some VOCs are mutagenic, teratogenic, or carcinogenic.⁶²³

VOC levels are affected by the design of a building and the materials used in its construction.⁶²⁴ Airflow rates, the amount of intro-

⁶¹⁴ See 29 C.F.R. § 1926.62(a).

⁶¹⁵ See *id.*

⁶¹⁶ An action level is the point at which something needs to be done to correct or eliminate the presence of lead. The likelihood of exposure above the action level requires an air monitoring program. Medical surveillance may be required. See 29 C.F.R. § 1926.62(j).

⁶¹⁷ 29 C.F.R. § 1910.1025.

⁶¹⁸ VOCs are a subset of hydrocarbons that are reactive and participate in atmospheric chemical reactions. EPA has determined that some VOCs have negligible photochemical reactivity and excludes them in a list at 40 C.F.R. § 51.100(s) (1997).

⁶¹⁹ See Spengler, *supra* note 153, at 46-48.

⁶²⁰ See National Volatile Organic Compounds Emission Standards for Architectural Coatings, 61 Fed. Reg. 32,729, 32,731 (1996) (codified 40 C.F.R. at pt. 59).

⁶²¹ See Mike McClintock, *Indoor Air Quality; Limiting Exposure to Pollutants and Allergens*, WASH. POST, Jan. 16, 1997, at T7.

⁶²² See Warpole, *supra* note 387, at 66; see also Kenneth H. Hudnell et al., *Exposure of Humans to a Volatile Organic Mixture, I, Behavioral Assessment: A Selection of Papers from Indoor Air '90 Concerning Health Effects Associated with Indoor Air Contaminants*, 47 ARCHIVES OF ENVTL. HEALTH, Jan. 1992, at 23-24.

⁶²³ See THE NATIONAL RESEARCH COUNCIL, THE NATIONAL ACADEMY OF SCIENCES, VAPOR-PHASE ORGANIC POLLUTANTS 201 (1976).

⁶²⁴ See Indoor Air Quality, 59 Fed. Reg. 15,968, 15,985 (1994) (codified at 29 C.F.R. pts. 1910, 1915, 1926, 1928).

duced outside air,⁶²⁵ office occupancy density, and the degree of partition use in open offices also are factors that determine VOC levels.⁶²⁶ Elevated temperatures increase VOC emissions off-gassing from building materials and furnishings.⁶²⁷ Changes in building use can lead to pollution buildup unless care is exercised to ensure renovations are properly designed.⁶²⁸

There is almost no general regulation of VOCs in indoor air by EPA. But many of the CAA's controls aimed at reducing ambient air levels of VOCs may reduce indoor air pollution levels as well. For example, EPA is required by CAA section 183(e)(3)(A) to list categories of consumer products that account for eighty percent of the VOC emissions in areas that violate the NAAQS for ozone.⁶²⁹ Such products are required to reduce emissions by using "best available controls."⁶³⁰ EPA can carry out this mandate by regulating any activity that results in the emission into the ambient air of VOCs from consumer products.⁶³¹

EPA proposed regulations on April 2, 1996,⁶³² that would set nationwide standards on VOC emissions from twenty-four consumer products.⁶³³ EPA expected to promulgate final rules in the fall of 1997.⁶³⁴ The rules have been delayed by litigation related to the rulemaking process brought by paint manufacturers.⁶³⁵ EPA also proposed na-

⁶²⁵ See Marian C. Marbury & James E. Woods, Jr., *Building-Related Illnesses*, in *INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE* 306, 308-09 (Jonathan M. Samet & John D. Spengler eds., 1991).

⁶²⁶ See *id.* at 317.

⁶²⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 110.

⁶²⁸ See generally McClintock, *supra* note 621, at T07. When building-use patterns change due to increased occupancy levels, additional heat and pollutant sources, such as computers and lights, are added and thereby tax the HVAC system. Partitions greatly add to this scenario. See Marbury & Woods, *supra* note 625, at 317.

⁶²⁹ 42 U.S.C. § 7511b(e)(3)(A) (1990).

⁶³⁰ See 42 U.S.C. § 7511b(e)(1)(A).

⁶³¹ Clean Air Act (CAA), § 183(e)(3)(A), 42 U.S.C. § 7511b(3)(A). Consumer of commercial product is defined at CAA § 183(e)(1)(B), 42 U.S.C. § 7511b(e)(1)(B).

⁶³² See National Volatile Organic Emission Standards for Consumer Products, 61 Fed. Reg. 14,531 (1996). This was only a notice.

⁶³³ The consumer products proposed to be regulated include: air fresheners, automotive windshield washer fluids, bathroom and tile cleaners, carburetor and choke cleaners, cooking sprays, dusting aides, engine degreasers, fabric protectants, floor polishes and waxes, furniture maintenance products, general purpose cleaners, glass cleaners, hairsprays, hair mousses, hair styling gels, household adhesives, insecticides, laundry prewash, laundry starch products, nail polish removers, oven cleaners, shaving creams, aerosol antiperspirants, and aerosol deodorants. *Final Rules Cutting VOCs From Products, Paints Set for Release This Fall, EPA Says*, 28 Env't Rep. (BNA) at 264 (June 6, 1997).

⁶³⁴ See *id.*

⁶³⁵ See *id.*

tional emission standards for VOCs in fifty-five types of architectural coatings on June 25, 1996.⁶³⁶ These standards were expected to take effect in April 1997; however, the proposed compliance date was delayed until January 1, 1998.⁶³⁷ In addition, there are proposed regulations for automobile refinish coatings.⁶³⁸

Some VOCs are controlled through programs aimed at specific chemicals; other VOC emissions are controlled through regulation of specific products. Regulations to control hazardous air pollutants by industry classifications, for example, can reduce a product's potential for being an indoor air pollution source.⁶³⁹ Two categories—formaldehyde and pesticides—are discussed below. Combustion gases, discussed *infra* Section IV.G., and ETS discussed *supra* Section IV.A also involve VOCs. But the substance-by-substance approach used in the limited regulatory programs to control potential indoor air pollutants increasingly is being rejected by litigants who focus on entire buildings where a large number of chemicals in low concentrations are found in constantly changing mixtures. Because of the absence of regulatory programs, the law of indoor air quality is being shaped by common-law-based toxic tort claims that often involve suits against virtually everyone who has anything to do with building ventilation.⁶⁴⁰ This topic is discussed *infra* Section IV.I.—Building Sickness.

1. Formaldehyde

One VOC that has been the subject of considerable concern as an indoor air pollutant is formaldehyde. Formaldehyde (HCHO) is a colorless, pungent smelling, water soluble gas, usually derived from methyl alcohol, which is found in hundreds of products.⁶⁴¹ Formaldehyde is an industrial chemical that usually is produced by catalytic

⁶³⁶ See National Volatile Organic Compound Emission Standards for Architectural Coatings, 61 Fed. Reg. 32,729 (1996) (reproposed and corrected on Sept. 3, 1996); 61 Fed. Reg. 46,410 (1996) (reopened for public comment Oct. 8, 1996); 61 Fed. Reg. 52,735 (1996) (to be codified at 40 C.F.R. pt. 59).

⁶³⁷ See National Volatile Organic Compound Standards for Architectural Coatings, 61 Fed. Reg. 52,735 (1996) (to be codified at 40 C.F.R. pt. 59).

⁶³⁸ See National Volatile Organic Compound Standards for Automobile Refinish Coatings, 61 Fed. Reg. 19,005 (1996) (to be codified at 40 C.F.R. pt. 59) (proposed Apr. 30, 1996).

⁶³⁹ See, e.g., National Emission Standards for Hazardous Air Pollutants for Flexible Polyurethane Foam Production, Proposed Rule, 61 Fed. Reg. 68,406 (1996) (to be codified at 40 C.F.R. pt. 63).

⁶⁴⁰ For a discussion of some unreported cases see R. Bruce Dickson, *Regulation of Indoor Air Quality: The Last Frontier of Environmental Regulation*, 9 NAT. RESOURCES & ENV'T 20 (1994).

⁶⁴¹ See RANDOM HOUSE DICTIONARY OF THE ENGLISH LANGUAGE 557 (1966).

oxidation of methanol.⁶⁴² It has four basic uses: as an intermediate chemical used to produce resins; as an intermediate chemical used to produce industrial chemicals; as a bactericide or fungicide; and as a component of consumer items.⁶⁴³ Three types of resins account for about fifty-nine percent of total formaldehyde consumption.⁶⁴⁴ Nearly one-third is used to produce other chemicals.⁶⁴⁵ Over sixty percent of urea formaldehyde resin production was used to make particleboard and plywood in 1977.⁶⁴⁶ Formaldehyde resin may be released from these products over their useful life. The resin is also used in decorative laminates, paper, foundry sand molds, paints, and coating products.⁶⁴⁷ Small amounts are used in other consumer products, such as cosmetics, shampoos, and glue.⁶⁴⁸ Two percent is used in textile treatments to obtain durable press properties in fabric.⁶⁴⁹ About sixty to eighty-five percent of all apparel fabric is finished with formaldehyde-containing resins.⁶⁵⁰ Because apparel manufacturing is the sixth largest industry in the United States, this is the use that is the major source of worker exposure.⁶⁵¹ A formerly significant source of formaldehyde was the formaldehyde emitted from urea-formaldehyde foam insulation (UFFI), which developed during the energy-conscious 1970s and early 1980s.⁶⁵² Approximately 500,000 homes contain UFFI, although the substance is no longer used in new products.⁶⁵³

⁶⁴² See AMERICAN CHEMICAL SOCIETY, CHEMISTRY IN THE ECONOMY 32 (1973).

⁶⁴³ See Occupational Exposure to Formaldehyde, Final Rule, 57 Fed. Reg. 22,290 (1992) (codified at 29 C.F.R. pt. 1910).

⁶⁴⁴ See 57 Fed. Reg. at 22,297.

⁶⁴⁵ See *id.*

⁶⁴⁶ See *id.*

⁶⁴⁷ See 59 Fed. Reg. 22,290.

⁶⁴⁸ See 57 Fed. Reg. at 22,291.

⁶⁴⁹ See *id.*

⁶⁵⁰ See *id.*

⁶⁵¹ See *id.*

⁶⁵² See DAVIS & SCHAFFMAN, *supra* note 4, at 88-89; see also TURIEL, *supra* note 195, at 16. UFFI, a wet foam material, was pumped under pressure into walls through small holes, where it hardened to form a layer of effective, inexpensive insulation. After installation, UFFI released significant amounts of formaldehyde into the air. See DAVIS & SCHAFFMAN, *supra* note 4, at 88.

⁶⁵³ See Spengler, *supra* note 153, at 49. In February 1982, the Consumer Product Safety Commission (CPSC) determined that UFFI presented an unreasonable health hazard and attempted to ban its sale and use as an insulator. See Ban of Urea-Formaldehyde Foam Insulation; Extension of Time, and Denial of Requests for Additional Comment Period, 47 Fed. Reg. 6436 (1982) (codified at 16 C.F.R. pt. 1306); Urea-Formaldehyde Foam Insulation; Proposed Ban; Denial of Petition, 46 Fed. Reg. 11,188 (1981) (codified at 16 C.F.R. pt. 1306). The Fifth Circuit overturned the attempted ban. See *Gulf South Insulation v. United States Consumer Prod. Safety Comm'n*, 701 F.2d 1137, 1150 (5th Cir. 1983). Effectively, however, the controversy surrounding UFFI ended its use. See Spengler, *supra* note 153, at 49.

Household sources of formaldehyde include smoking,⁶⁵⁴ unvented fuel-burning appliances,⁶⁵⁵ floor coverings,⁶⁵⁶ fabrics,⁶⁵⁷ and other consumer products.⁶⁵⁸ The primary sources of formaldehyde in homes are pressed wood products, such as particleboard, hardwood plywood paneling, and medium density fiberboard.⁶⁵⁹ Many of these materials are made using urea-formaldehyde resins.⁶⁶⁰ Today, plywood and particleboard used in prefabricated and mobile homes must conform to specified formaldehyde emission limits set by the Department of Housing and Urban Development.⁶⁶¹ Potential sources of formaldehyde in office buildings include insulation, new furniture and furnishings, carpets, carbonless copy paper, and tobacco smoke.⁶⁶² Fortunately, emission levels created by products containing formaldehyde decrease as the materials age.⁶⁶³

a. *Health Effects of Formaldehyde*

The health effects of formaldehyde include eye, nose and throat irritation; wheezing and coughing; fatigue; skin rash; severe allergic reactions; and possibly cancer.⁶⁶⁴ Exposure pathways include inhalation, ingestion, and dermal absorption.⁶⁶⁵ Exposure levels of 0.1 parts

⁶⁵⁴ "Cigarette smoke contains as much as 40 parts per million of formaldehyde." Kirsch, *supra* note 425, at 352 n.93; see also Spengler, *supra* note 153, at 48.

⁶⁵⁵ These include gas stoves and kerosene heaters. See Spengler, *supra* note 153, at 48.

⁶⁵⁶ The use of formaldehyde in carpets has greatly decreased in the past ten years. See DAVIS & SCHAFFMAN, *supra* note 4, at 110.

⁶⁵⁷ Textiles contain formaldehyde to reduce creasing, crushing, shrinking, and flammability. See Kirsch, *supra* note 425, at 353 n.105.

⁶⁵⁸ Some common consumer products containing formaldehyde including grocery bags, waxed paper, facial tissues, paper towels, and disinfectants. See Kirsch, *supra* note 425, at 353 n.106.

⁶⁵⁹ See EPA *Inside Story*, *supra* note 2.

⁶⁶⁰ See *id.* Urea formaldehyde insulation was banned by the United States Consumer Products Safety Commission on February 22, 1982. Ban of Urea-Formaldehyde Foam Insulation, 47 Fed. Reg. 14,366 (1982) (codified at 16 C.F.R. pt. 1306).

⁶⁶¹ See Manufactured Home Construction and Safety Standards, 49 Fed. Reg. 31,996 (1984) (codified at 24 C.F.R. pt. 3280).

⁶⁶² See Marian C. Marbury & Robert A. Krieger, *Formaldehyde*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE 223, 225 (Jonathan M. Samet & John D. Spengler eds., 1991).

⁶⁶³ See *id.*

⁶⁶⁴ See EPA *Inside Story*, *supra* note 2. Formaldehyde causes cancer in animals and may cause cancer in humans. See *id.* For a thorough discussion of the human health effects of formaldehyde, see Marbury & Krieger, *supra* note 662, at 228-45. See also Kathleen M. Rest & Nicholas A. Ashford, *Regulation and Technical Options: The Case of Occupational Exposure to Formaldehyde*, 1 HARV. J.L. & TECH. 63, 80 (1988). EPA estimates that 10 to 20 percent of the U.S. population is particularly susceptible to formaldehyde effects. See McClintock, *supra* note 621, at T07.

⁶⁶⁵ See Marbury & Krieger, *supra* note 662, at 226.

per million (ppm) may cause difficulty in breathing, and high concentrations may trigger asthma attacks.⁶⁶⁶ At the 0.1 ppm level, the gas can be smelled.⁶⁶⁷ This level of 0.1 ppm is commonly used as a guideline in homes.⁶⁶⁸ For example, the American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) recommends that indoor formaldehyde concentrations inside the home be no greater than 0.1 ppm.⁶⁶⁹ OSHA's formaldehyde exposure standard is 0.75 ppm for an eight hour time-weighted average, with a Short-Term Exposure Limit of 2 ppm for 15 minutes.⁶⁷⁰

Because UFFI was installed during the 1970s and 1980s, formaldehyde levels in UFFI houses dropped with the passage of time and health complaints sharply diminished as well.⁶⁷¹ However, concentrations as low as 0.05 parts per million can irritate the eyes.⁶⁷² EPA has classified formaldehyde as a probable human carcinogen.⁶⁷³ However, scientists do not yet agree on human carcinogenicity as a result of low-level exposure or on the ability of formaldehyde to cause respiratory sensitization.⁶⁷⁴

b. *Control of Formaldehyde*

The best way to prevent formaldehyde exposure is to avoid products that contain formaldehyde.⁶⁷⁵ Other ways include: (1) coating formaldehyde-containing products with polyurethane to prevent emissions; (2) maintaining moderate indoor temperature and humidity levels;⁶⁷⁶ (3) maintaining adequate ventilation;⁶⁷⁷ and (4) using exte-

⁶⁶⁶ See *EPA Inside Story*, *supra* note 2.

⁶⁶⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 89. Formaldehyde is a "sensitizer," a chemical that, after exposure, requires higher and higher levels to be detected at all. See *id.*

⁶⁶⁸ See *id.*

⁶⁶⁹ See *id.*

⁶⁷⁰ See *id.* See also Occupational Exposure to Formaldehyde, 57 Fed. Reg. 22,290 (1992) (codified at 29 C.F.R. pt. 1910). OSHA's formaldehyde exposure standard is 0.75 ppm for an 8-hour time-weighted average, with a Short Term Exposure Limit of 2 ppm for 15 minutes. See *id.* For formaldehyde levels in commercial buildings, OSHA generally receives complaints when the formaldehyde concentration reaches a level between approximately 0.45 and 0.60 ppm. See *id.* Some people, about 20 percent of the population, are highly sensitive even at very low levels. See *id.*

⁶⁷¹ See DAVIS & SCHAFFMAN, *supra* note 4, at 89.

⁶⁷² See *id.*

⁶⁷³ See REITZE, *supra* note 33, at 419 (citing EPA REPORT, *supra* note 13, at 4-14).

⁶⁷⁴ Marbury & Krieger, *supra* note 662, at 224. Household products with one percent formaldehyde are considered strong sensitizers under the Federal Hazardous Substances Act and must have a cautionary label. See Rest & Ashford, *supra* note 664, at 79-80.

⁶⁷⁵ See *EPA Inside Story*, *supra* note 2.

⁶⁷⁶ Formaldehyde emissions are accelerated by heat and high humidity. See *id.*

⁶⁷⁷ See *id.*

rior-grade plywood and particleboard indoors rather than interior-grade materials.⁶⁷⁸ A drastic step is to remove urea-formaldehyde foam insulation from a home.⁶⁷⁹

In 1972, OSHA first regulated worker exposure to formaldehyde with a permissible exposure level (PEL) of 3 ppm based on the risk of eye, skin and respiratory irritations.⁶⁸⁰ In 1981, the United Auto Workers and thirteen other unions petitioned OSHA to issue an emergency temporary standard based on new research that indicated formaldehyde might be a human carcinogen.⁶⁸¹ On December 4, 1987, OSHA issued a comprehensive regulation governing occupational exposure to formaldehyde with a PEL of 1 ppm as an eight hour time-weighted average (TWA), and established a 2 ppm 15-minute short-term exposure limit (STEL).⁶⁸² It also included an "action level" of 0.5 ppm measured as an eight hour TWA and provisions for employee monitoring, medical surveillance and other requirements.⁶⁸³ The standard was challenged by both industry and labor.⁶⁸⁴ The U.S. Court of Appeals for the District of Columbia Circuit affirmed most of the rule on June 9, 1989, but remanded the final standard for OSHA to explain its refusal to lower the standard below 1 ppm and its failure to include a medical removal protection requirement.⁶⁸⁵

On May 27, 1992, OSHA responded to the D.C. Circuit's remand with a final rule that lowered the permissible exposure level for formaldehyde to 0.75 ppm on an eight hour TWA.⁶⁸⁶ The amendments also added medical removal protection provisions to supplement the existing medical surveillance requirements and the hazard communi-

⁶⁷⁸ Exterior grades contain less formaldehyde than interior grades. See DAVIS & SCHAFFMAN, *supra* note 4, at 112.

⁶⁷⁹ See TURIEL, *supra* note 195, at 27. However, money need not be spent to remove UFFI material since most homes with UFFI no longer have risky levels of formaldehyde. See DAVIS & SCHAFFMAN, *supra* note 4, at 89. Removal of UFFI is extremely disruptive to a home and costly. Removal can require tearing down walls, for example; it is typically a last resort measure. See *id.* at 90. Further, the benefits of removing UFFI are uncertain. See *id.*

⁶⁸⁰ 29 C.F.R. § 1910.1000(b) (1997).

⁶⁸¹ See *generally* International Union, UAW v. Donovan, 590 F. Supp. 747 (D.D.C. 1984), *aff'd* 756 F.2d 162 (D.C. Cir. 1985).

⁶⁸² See Occupational Exposure to Formaldehyde, 52 Fed. Reg. 46,168 (1987) (codified at 29 C.F.R. pts. 1910, 1926).

⁶⁸³ See 57 Fed. Reg. at 22,290 (codified at 29 C.F.R. pt. 1910).

⁶⁸⁴ See, e.g., International Union, UAW v. Pendergrass, 878 F.2d 389, 390-91 (D.C. Cir. 1989).

⁶⁸⁵ See *id.* at 400-01.

⁶⁸⁶ See Occupational Exposure to Formaldehyde, 57 Fed. Reg. 22,290 (1992) (codified at 29 C.F.R. pt. 1910).

cation requirements.⁶⁸⁷ It was the first negotiated rulemaking involving OSHA.⁶⁸⁸

There are currently no federal or state requirements concerning formaldehyde levels in the indoor air of homes.⁶⁸⁹ EPA considered regulating formaldehyde under Section 4(f) of TSCA,⁶⁹⁰ but never did.⁶⁹¹ In 1982, CPSC banned the use of urea-formaldehyde foam insulation in residences and schools; this ban was later overturned.⁶⁹² In 1984, HUD issued regulations regarding formaldehyde emissions from pressed wood products that are used in manufactured homes and required that plywood and particleboard emit no more than 0.2 ppm and 0.3 ppm, as measured by a specified air chamber test, respectively.⁶⁹³ The rule was upheld by a U.S. Court of Appeals.⁶⁹⁴

At the state level, more than half of the states have some type of *mandatory* property disclosure law or seller disclosure law.⁶⁹⁵ Problems that must be disclosed are generally those considered to be "material defects."⁶⁹⁶ Some states require sellers and landlords to determine if formaldehyde insulation (UFFI) is present and to disclose levels of formaldehyde gas in the indoor air.⁶⁹⁷

Formaldehyde is no longer a high priority regulatory item for the relevant federal agencies because the public is no longer concerned with its dangers. Formaldehyde emissions have lowered significantly as a result of the HUD standard, the increased air flow that is designed into buildings, the reduction in emissions from manufactured

⁶⁸⁷ See *id.*

⁶⁸⁸ For a history of the proposed rule, see Occupational Exposure to Formaldehyde; Response to Court Remand, 56 Fed. Reg. 32,302 (1991) (codified at 29 C.F.R. pt. 1910).

⁶⁸⁹ See DAVIS & SCHAFFMAN, *supra* note 4, at 91.

⁶⁹⁰ 15 U.S.C. § 2603(f) (1994); see Formaldehyde; Determination of Significant Risk, 49 Fed. Reg. 21,870 (1984) (codified at 40 C.F.R. pt. 765).

⁶⁹¹ See Toxic and Hazardous Substances Control; Formaldehyde Termination of Regulatory Investigation Concerned With Occupational Exposure, 51 Fed. Reg. 9469 (1986) (codified at 40 C.F.R. pt. 765).

⁶⁹² See *Gulf S. Insulation v. Consumer Prod. Safety Comm'n*, 701 F.2d 1137, 1140 (5th Cir. 1983).

⁶⁹³ See Manufactured Home Construction and Safety Standards, 49 Fed. Reg. 31,996 (1984) (codified at 24 C.F.R. pt. 3280).

⁶⁹⁴ See *New Mexico v. United States Dep't of Hous. & Urban Dev.*, 1987 WL 109007, at *6 (10th Cir. 1987) (unreported disposition).

⁶⁹⁵ See DAVIS & SCHAFFMAN, *supra* note 4, at 17. To obtain a copy of a state's law and the disclosure form, contact a real estate agent or state consumer protection office. See *id.* at 18.

⁶⁹⁶ See *id.* at 13-14; see, e.g., DEL. CODE ANN. tit. 6, § 2572 (1996); 765 ILL. COMP. STAT. ANN. 77/25 (West 1996). Examples of material defects include the presence of formaldehyde insulation, radon, lead-based paint, or asbestos. See DAVIS & SCHAFFMAN, *supra* note 4, at 14, 18.

⁶⁹⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 17; see, e.g., MASS. GEN. LAWS. ANN. ch. 255, § 12I (Law/Co-op. 1996); 10 M.R.S. § 1482 (1996).

wood products due to changes in industry practices, and the substitution of gypsum wallboard for plywood in construction.⁶⁹⁸

2. Pesticides

Many, but not all, of the most dangerous pesticides are VOCs. Pesticides were used in approximately three-fourths of U.S. households in 1996.⁶⁹⁹ Thus, pesticide exposure occurs indoors as a result of use indoors, but also as a result of introduction of contaminated soil or dust and emissions from pesticides stored indoors.⁷⁰⁰ Pesticides are a major source of public concern because of their known toxicity, widespread use, persistence in the environment, and possible associations with delayed health effects.⁷⁰¹ The health effects of pesticides include irritation to the eyes, nose and throat; damage to the central nervous system and kidneys; cancer,⁷⁰² and even death.⁷⁰³ The active ingredients and some inert ingredients are usually dangerous to human health. Household use of pesticides generally includes the application of insecticides, termiticides and fungicides. Some pesticides are so dangerous that EPA now prohibits their use or requires that they be applied only under specified conditions.⁷⁰⁴ The simplest means of reducing exposure to pesticides, absent non-use, is to use them cor-

⁶⁹⁸ See generally Sara Thurin Rollin, *Particleboard Makers Urge EPA to Halt Formaldehyde Probe, Refuse Peer Review*, 20 Chem. Reg. Rep. (BNA) No. 10 at 329 (June 7, 1996).

⁶⁹⁹ See *EPA Inside Story*, *supra* note 2.

⁷⁰⁰ See *id.* One example of a pesticide contaminating the air of homes is chlordane, which contaminates the air of approximately 75 percent of homes built before March 1988. Chlordane enters the air by infiltration under the home, attic contamination, exterior contamination, accidental spills, indoor application, and soil contamination. See *How to Remove Chlordane from the Indoor Air*, <<http://www.chem-tox.com/repair>>; see also *Many Illnesses Suspected for People Living in Chlordane Pesticide Treated Homes* (visited July 2, 1997) <<http://www.chem-tox.com/chlordane>>.

⁷⁰¹ See Ketty Modbed et al., *Occupational Health Problems Among Migrant and Seasonal Farm Workers*, 157 W. J. MED. 367, 369 (1992).

⁷⁰² See *EPA Inside Story*, *supra* note 2; see also MARGIE T. SEARCY, *A GUIDE TO TOXIC TORTS* 23-37 to 23-40 (1995).

⁷⁰³ In its 1997 *Special Report on Endocrine Disruption: An Effects Assessment and Analysis Document*, EPA stated that while persistent chemicals might be responsible for hormone-mediated illnesses in humans, a causal link generally has not been established. The report, focusing on endocrine disruption, includes mainly pesticides that are potential hormone disruptors. See *EPA Report Says Causal Relationship Between Illness, Exposure Not Established*, 11 *Toxics L. Rep.* (BNA) at 1144 (Mar. 19, 1997).

⁷⁰⁴ EPA has banned the use of the pesticides aldrin, chlordane, and dieldrin. Heptachlor may be used only by utility companies as a means of controlling fire ants found in underground cable boxes. See *EPA Inside Story*, *supra* note 2; see generally *Environmental Defense Fund v. United States Env'tl. Protection Agency*, 548 F.2d 998 (D.C. Cir. 1976) (chlordane/heptachlor); *Environmental Defense Fund v. United States Env'tl. Protection Agency*, 465 F.2d 528 (D.C. Cir. 1972) (aldrin/dieldrin).

rectly.⁷⁰⁵ Pesticides should be used in well-ventilated areas, if and only if alternative methods, such as biological pesticides or frequent washing of indoor plants or pets, are not viable options.⁷⁰⁶

The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)⁷⁰⁷ was enacted to assure that the use of pesticides, in compliance with labeling instructions, will not cause "unreasonable adverse effects" to humans or the environment.⁷⁰⁸ Further, FIFRA gives EPA the authority to require submission of pesticide-specific data and to restrict the distribution and use of the pesticide. Some pesticides are used indoors. EPA regulates these indoor air pollutants by banning or limiting their use and by establishing directions for safe use.⁷⁰⁹ Using its authority under FIFRA, EPA successfully banned commercial use of chlordane, dieldin, aldrin, and hetachlor, and imposed labeling requirements on other household pesticides.⁷¹⁰

G. Combustion Byproducts

During the combustion process indoor air pollutants may be produced due to unvented combustion byproducts.⁷¹¹ Combustion gases of concern include carbon dioxide (CO₂), carbon monoxide (CO), nitrogen dioxide (NO₂), sulfur dioxide (SO₂), formaldehyde, and VOCs.⁷¹² A common source of CO buildup in homes is motor vehicle exhaust fumes that enter the home from a garage.⁷¹³ Particulate emissions, which can include carcinogenic particulates, are a problem⁷¹⁴ for those using unvented kerosene and gas space heaters,⁷¹⁵ woodstoves, fireplaces,⁷¹⁶ and gas stoves.⁷¹⁷

⁷⁰⁵ See *EPA Inside Story*, *supra* note 2.

⁷⁰⁶ See *id.*

⁷⁰⁷ 7 U.S.C. §§ 136–136y (1994).

⁷⁰⁸ *Id.*

⁷⁰⁹ See Harrison, *supra* note 13, at 333 (citing EPA REPORT, *supra* note 13, at 8–7); see also Guiffrida, *supra* note 3, at 328.

⁷¹⁰ See Guiffrida, *supra* note 3, at 329 (citation omitted).

⁷¹¹ See Spengler, *supra* note 153, at 37.

⁷¹² See DADE W. MOELLER, ENVIRONMENTAL HEALTH 22 (1992).

⁷¹³ See DAVIS & SCHAFFMAN, *supra* note 4, at 52.

⁷¹⁴ See *EPA Inside Story*, *supra* note 2; see also DAVIS & SCHAFFMAN, *supra* note 4, at 52.

⁷¹⁵ Approximately 11 percent of the U.S. population uses gas or kerosene heaters. See Spengler, *supra* note 153, at 39. Particulate matter is also emitted by gas and kerosene heaters. See *id.*

⁷¹⁶ Wood stoves are found in six percent of homes and fireplaces are found in 19 percent of homes. See *id.*

⁷¹⁷ See *EPA Inside Story*, *supra* note 2. Wood stoves, fireplaces, and unvented kerosene space

1. Health Effects of Combustion Gases and Particulates

Carbon monoxide is a colorless, odorless gas⁷¹⁸ that binds readily with hemoglobin and prevents delivery of oxygen throughout the body.⁷¹⁹ Low concentrations of CO may only cause fatigue in most people, but persons with chronic heart disease may experience increased chest pain.⁷²⁰ As concentrations and duration of exposure to CO increase, the health effects are more significant.⁷²¹ They may include headaches, dizziness, weakness, nausea, confusion, and disorientation.⁷²² CO can also harm fetuses, impair perception and thinking, slow reflexes, and cause drowsiness.⁷²³ Unconsciousness and death may also result.⁷²⁴ Thousands of people each year are affected by CO; many confuse the symptoms with those of the flu or food poisoning.⁷²⁵

Nitrogen dioxide impairs pulmonary defense mechanisms and changes ventilatory function.⁷²⁶ It may also cause lung damage and increase respiratory infections in young children.⁷²⁷ Children and those with asthma or other respiratory diseases are particularly sensitive.⁷²⁸ Sulfur dioxide (SO₂) at high exposure levels alters the lungs' defense mechanisms and aggravates existing respiratory and cardiovascular

heaters also emit polycyclic aromatic hydrocarbons. Acid aerosols may also be emitted by kerosene heaters. *See id.*

⁷¹⁸ CO is produced by the incomplete combustion of carbon-based fuels. *See* David B. Coultas & William E. Lambert, *Carbon Monoxide*, in *INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE* 187 (Jonathan M. Samet & John D. Spengler eds., 1991).

⁷¹⁹ *See id.*

⁷²⁰ *See id.* at 193.

⁷²¹ *See id.* at 187.

⁷²² *See EPA Inside Story*, *supra* note 2.

⁷²³ *See* F. Kaid Benfield, *Running on Empty: The Case for a Sustainable National Transportation System*, 25 ENVTL. L. 651, 655 (1995).

⁷²⁴ Several categories of persons are more susceptible to the effects of carbon monoxide. These include elderly people and those with anemia and heart or respiratory disease. People who need high levels of available oxygen may be more sensitive to the adverse effects of carbon monoxide. *See EPA Inside Story*, *supra* note 2.

⁷²⁵ *See* DAVIS & SCHAFFMAN, *supra* note 4, at 53.

⁷²⁶ *See* THE NATIONAL RESEARCH COUNCIL, NATIONAL ACADEMY OF SCIENCES, *NITROGEN OXIDES* 271 (1977).

⁷²⁷ *See* DAVIS & SCHAFFMAN, *supra* note 4, at 53; *see also* Jonathan M. Samet, *Nitrogen Dioxide in Indoor Air Pollution: A Health Perspective* 170 (Jonathan M. Samet & John D. Spengler eds., 1991).

⁷²⁸ The effects of nitrogen dioxide are reviewed in National Ambient Air Quality Standards for Nitrogen Dioxide: Proposed Decision, 60 Fed. Reg. 52,874 (1995) (codified at 40 C.F.R. pt. 50) (proposed Oct. 11, 1995).

disease.⁷²⁹ VOCs include many substances and previously have been discussed.⁷³⁰

Respirable particles can cause eye, nose, and throat irritation; respiratory infections; and bronchitis.⁷³¹ The foremost concern is premature death from respiratory diseases (including cancer) and heart attacks.⁷³² Other pollutants, such as radon daughters, may attach to inhaled respirable particles and may lodge in the lungs, causing irritation or damage.⁷³³

2. Control of Combustion Gases and Particulates

Several methods of controlling combustion gases are available, including removing the source, careful and proper operation to reduce emissions,⁷³⁴ providing sufficient ventilation, proper maintenance,⁷³⁵ and use of exhaust vents and fans that vent emissions directly outdoors.⁷³⁶ If there is a combustion source in the home, it is prudent to have a CO detector.⁷³⁷ EPA's standard for CO in ambient air is nine ppm for an eight-hour day;⁷³⁸ OSHA's standard is thirty-five ppm.⁷³⁹

In the U.S., local building codes regulate the installation and use of furnaces, woodstoves, and fireplaces, and some form of permit or approval is often required.⁷⁴⁰ Some cities, e.g., Chicago, require use of CO detectors in new single-family homes and in older homes with oil or gas furnaces.⁷⁴¹ Combustion of gases produces the fine particles, smaller than 2.5 microns (um), that are the subject of EPA's proposed

⁷²⁹ See OFFICE OF AIR QUALITY PLANNING AND STANDARDS, U.S. ENVTL. PROTECTION AGENCY, NO. 454/R-96-005, NATIONAL AIR QUALITY AND EMISSION TRENDS REPORT 29 (1996).

⁷³⁰ See *supra* Section IV.F.

⁷³¹ See *EPA Inside Story*, *supra* note 2.

⁷³² See JOHN E. BLODGETT ET AL., CONGRESSIONAL RESEARCH SERVICE, AIR QUALITY: EPA'S PROPOSED NEW OZONE AND PARTICULATE MATTER STANDARDS, NO. 97-8, 6 (1997).

⁷³³ See *EPA Inside Story*, *supra* note 2. Respirable particles may lead to lung cancer. See *id.*

⁷³⁴ See *id.* For example, flame color indicates excess CO is being released from a gas appliance. If the flame is yellow tipped—and not blue—the burner should be adjusted. See DAVIS & SCHAFFMAN, *supra* note 4, at 56.

⁷³⁵ See Kirsch, *supra* note 425, at 351; see also *EPA Inside Story*, *supra* note 2.

⁷³⁶ See *EPA Inside Story*, *supra* note 2, at 19.

⁷³⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 55 (referring to July 1995 issue of *Consumer Reports* which reviews brands and models).

⁷³⁸ 40 C.F.R. pt 50 (1997).

⁷³⁹ See Air Contaminants, 54 Fed. Reg. 2332, 2651 (1989) (codified at 29 C.F.R. pt. 1910).

⁷⁴⁰ See DAVIS & SCHAFFMAN, *supra* note 4 at 55; see, e.g., PITTSBURGH, PA. MUN. CODE § 747.03(B), 1007.02 (1993).

⁷⁴¹ See generally AGENDA: QUARTERLY MEETING OF THE INTERAGENCY COMMITTEE ON INDOOR AIR QUALITY (Apr. 30, 1997) (referring to Chicago local ordinance requiring CO detectors).

new fine particle standard that was published in the *Federal Register* on December 13, 1996.⁷⁴² However, EPA and OSHA currently have no indoor air standards applicable to residential properties. The use of wood as a heating fuel has increased since 1970.⁷⁴³ Generally, the combustion of wood for use in heating or cooking is incomplete, and as a result, nitrogen oxides, sulfur oxides, organic compounds, carbon monoxide, and particulates are emitted.⁷⁴⁴ EPA regulates woodstoves through the use of new source performance standards.⁷⁴⁵

H. *Electromagnetic Fields*

The invisible lines of force that surround any electrical device constitute an electromagnetic field (EMF).⁷⁴⁶ EMFs consist of an electric field and a magnetic field; electric fields are produced by the presence of electrical charges, whereas magnetic fields are produced by the movement of those charges (i.e., when there is an electric current flow).⁷⁴⁷ Electromagnetic fields are a form of radiation.⁷⁴⁸

⁷⁴² See National Ambient Air Quality Standards for Particulate Matter: Proposed Decision, 61 Fed. Reg. 65,638 (1996) (to be codified at 40 C.F.R. pt. 50).

⁷⁴³ See generally Marian C. Marbury, *Wood Smoke*, in INDOOR AIR POLLUTION: A HEALTH PERSPECTIVE (Jonathan M. Samet & John D. Spengler eds., 1991).

⁷⁴⁴ See *id.*

⁷⁴⁵ See Standards of Performance for New Stationary Sources; New Residential Wood Heaters, 53 Fed. Reg. 5860 (1988) (codified at 40 C.F.R. pt. 60). EPA twice published corrections to this final rule in April 1988. See Standards of Performance for New Stationary Sources; New Residential Wood Heaters; Correction, 53 Fed. Reg. 12,009 (1988); 53 Fed. Reg. 14,888 (1988). The development of the NSPS is covered in William Funk, *When Smoke Gets in Your Eyes: Regulatory Negotiation and The Public Interest—EPA's Woodstove Standards*, 18 ENVTL. L. 55 (1987).

⁷⁴⁶ See NATIONAL INST. OF ENVTL. HEALTH SCIENCES AND U.S. DEP'T OF ENERGY, NO. DOE/EE-0040, QUESTIONS AND ANSWERS ABOUT ELECTRIC AND MAGNETIC FIELDS ASSOCIATED WITH THE USE OF ELECTRIC POWER 5 (1995) [hereinafter QUESTIONS AND ANSWERS]; see also Craig T. Liljestrand, *EMFs and the Potential for Injury: Real Danger or Overreaction?*, 62 DEF. COUNS. J. 400, 400-01 (1995).

⁷⁴⁷ See Liljestrand, *supra* note 746, at 401. Thus, an appliance that is plugged in, and therefore connected to a source of electricity, has an electric field even when the appliance is turned off. To produce a magnetic field, however, the appliance not only must be plugged in, but also must be operating, so that the current is flowing. See *id.*; see generally Daniel Wartenberg, *EMFs: Cutting Through the Controversy*, 111 PUB. HEALTH REP. 204, 204-17 (1996).

⁷⁴⁸ See U.S. ENVTL. PROTECTION AGENCY, NO. 402-R-92-009, QUESTIONS AND ANSWERS ABOUT ELECTRIC AND MAGNETIC FIELDS 2 (1992) [hereinafter U.S. EPA]; see also John Weiss, *The Power Line Controversy: Legal Responses to Potential Electromagnetic Field Health Hazards*, 15 COLUM. J. ENVTL. L. 359, 362 (1990); see generally Rufus C. Young, Jr. & Craig S. Gunther, 1995 Update: *Electromagnetic Fields and Their Land Use Implications*, CAL. ENVTL. L. REP. 75 (1994).

EMFs can be characterized by their frequency and wavelength:⁷⁴⁹ the amount of energy in an EMF increases as wavelength becomes smaller and frequency increases.⁷⁵⁰ The electromagnetic spectrum covers a large range of frequencies expressed in cycles per second, or Hertz (Hz).⁷⁵¹ Alternating current used in homes, offices, and factories operates at the low end of the frequency spectrum at sixty Hz;⁷⁵² hence, its EMF radiation also has a frequency of sixty Hz.

Electric and magnetic fields have different properties and possible different biological effects.⁷⁵³ The electric fields associated with EMFs do not easily penetrate the body; they may be blocked by earth, trees, or buildings and are thought to have little effect on humans.⁷⁵⁴ In contrast, magnetic fields readily penetrate the body and have the ability to modify the biological functioning of living organisms.⁷⁵⁵

Electric field strength is commonly measured in units of volts per meter or kilovolts per meter.⁷⁵⁶ The strength of a magnetic field is most commonly measured in units of gauss (G) or milligauss (mG).⁷⁵⁷ Electric field strength is dependent on voltage,⁷⁵⁸ while magnetic field strength is dependent on current.⁷⁵⁹ Both electric and magnetic fields weaken with increasing distance from the source.⁷⁶⁰ Magnetic field

⁷⁴⁹ See U.S. ENVTL. PROTECTION AGENCY, No. 402-R-92-008, *EMF IN YOUR ENVIRONMENT* (1992) [hereinafter EMF].

⁷⁵⁰ See Zack Mansdorf, *EMF: Media Hype or Real Hazard?*, *OCCUPATIONAL HAZARDS*, Mar. 1994, at 31.

⁷⁵¹ See *QUESTIONS AND ANSWERS*, *supra* note 746, at 5.

⁷⁵² See Liljestrand, *supra* note 746, at 400. Alternating currents do not continuously move in the same direction; they "alternate" back and forth. In the U.S., electric power alternates at a rate of 60 times each second, known as 60 Hertz (Hz) or 60 cycle power. See *QUESTIONS AND ANSWERS*, *supra* note 746, at 5; see also Mansdorf, *supra* note 750, at 32; U.S. EPA, *supra* note 748, at 2.

⁷⁵³ See *QUESTIONS AND ANSWERS*, *supra* note 746, at 5.

⁷⁵⁴ See David A. Savitz et al., *Methodological Issues in the Epidemiology of Electromagnetic Fields and Cancer*, 11 *EPIDEMIOLOGY REVS.* 59, 63 (1989).

⁷⁵⁵ For this reason, residential exposures to electric fields are not predicted by external electric power lines because these electric fields are blocked from entering the residences. Therefore, effects of residential exposure from external power lines are reflective of magnetic rather than electric fields. See *id.*

⁷⁵⁶ See Weiss, *supra* note 748, at 362.

⁷⁵⁷ See *id.* at 362; EMF, *supra* note 749, at 31. This a measure of magnetic flux density. See Weiss, *supra* note 748, at 362.

⁷⁵⁸ Changes produce electric fields. A stronger electric field occurs where a higher voltage is present because the higher voltage produces more charges; the charges produce electric fields. See *EMF's* (visited Dec. 4, 1997) <<http://www.xpert.net/grossing.refemf.html>>.

⁷⁵⁹ See Bette Hileman, *Health Effects of Electromagnetic Fields Remain Unresolved*, 71 *CHEM. & ENGINEERING NEWS*, Nov. 8, 1993, at 18.

⁷⁶⁰ See U.S. EPA, *supra*, note 748, at 6; see also Mansdorf, *supra* note 750, at 32; Liljestrand, *supra* note 746, at 401.

strength generally decreases by the square of the distance from the source.⁷⁶¹ With a power line, magnetic field strength is determined by the distance from the line, and the type of power line producing the EMF.⁷⁶² In a room, the strength of a field depends upon the number and kinds of sources, how far away they are, how many are operating, and their power rating.⁷⁶³

Another type of electromagnetic radiation is known as electromagnetic pulse (EMP) radiation. EMP is a broad-band electromagnetic pulse below 10^{15} Hz associated with nuclear explosions.⁷⁶⁴ Like EMF, EMP is also a form of non-ionizing radiation; however, unlike electromagnetic fields, which are given off in constant waves, EMP is pulsed.⁷⁶⁵ There is concern regarding EMP, particularly in the military, due to its unique ability to affect large areas.⁷⁶⁶ There have been several cases brought concerning EMP.⁷⁶⁷

One type of EMF non-ionizing radiation is the radio frequency (RF) radiation emitted by radio and communication (R/C) towers and other communications equipment.⁷⁶⁸ The major environmental impact of R/C towers, however, is aesthetic—people consider the towers to be “visual pollution.”⁷⁶⁹

⁷⁶¹ See generally Wartenberg, *supra* note 747; see also Curt Suplee, *Power Line Hazard Called Small*, WASH. POST, Nov. 1, 1996, at A4.

⁷⁶² For example, EMF levels twenty feet away from a typical power line will be less than 100 mg; at three hundred feet, EMF levels will be under 2 mg. Compare this to an EMF field produced one inch from a hair dryer which ranges from 60 to 20,000 mg, or one foot from a microwave oven which may be 40 to 80 mg. See DAVIS & SCHAFFMAN, *supra* note 4, at 211.

⁷⁶³ The standard American home has a background magnetic field level of .1 mG to 4 mG. See U.S. EPA, *supra* note 748, at 4.

⁷⁶⁴ EMP is a burst of electromagnetic energy resulting from the interaction between gamma rays released by the explosion and electrons whose orbits are disturbed by those gamma rays. See Yonkel Goldstein, *The Failure of Constitutional Controls Over War Powers in the Nuclear Age: The Argument for a Constitutional Amendment*, 40 STAN. L. REV. 1543, 1544 (1988).

⁷⁶⁵ EMP is a single large pulse of electromagnetic energy. See Gerhard Albert Steubben, *Electromagnetic Pulse Radiation: An Overview of Military Applications, Health Risks, and Regulatory Options* (1992) (unpublished LL.M. dissertation, The George Washington University Law School) (on file with author), at 16.

⁷⁶⁶ See *Conservation Law Found. v. United States Dep't of Air Force*, No. 87-1871-K, 1987 WL 46370, at *1 (D. Mass. Nov. 23, 1987); see also *Wisconsin v. Weinberger*, 745 F.2d 412, 414 (7th Cir. 1984); *Institute for Policy Studies v. United States Dep't of Air Force*, 676 F. Supp. 3, 4 (D.D.C. 1987).

⁷⁶⁷ In a ground-breaking case, in 1990, Boeing settled a personal injury suit for \$500,000 in which the plaintiff claimed to have contracted leukemia from EMP radiation. See Roland A. Giroux, *Daubert v. Merrell Dow: Is This Just What the EMF Doctor Ordered?*, 12 PACE ENVTL. L. REV. 393, 435 (1994).

⁷⁶⁸ See DAVIS & SCHAFFMAN, *supra* note 4, at 297.

⁷⁶⁹ *Id.*

1. Sources of Electromagnetic Fields

EMF radiation, produced by the use of electricity, permeates our environment⁷⁷⁰ with low-frequency, low-energy, electric and magnetic fields. Electromagnetic fields found in buildings are potentially important contributors to EMF exposure.⁷⁷¹

Inside buildings, the major EMF sources are common electrical devices including computers, microwave ovens, and cellular phones. Exposures in residences are produced by outside power lines,⁷⁷² wiring in the home, electric heat, electric hot water heaters, water pipes to which the home electrical system has been grounded, electrical appliances, and other electric devices.⁷⁷³ Occupational exposures are created wherever electrical equipment is used, including electric motors, photocopying machines, and video display terminals.

There are also natural sources of EMFs including the earth's magnetic field;⁷⁷⁴ thunderstorms; lightning; and even the human body, which produces electrical fields in making the heart and nervous system work.⁷⁷⁵ The variety and ubiquity of sources of EMFs in the environment make it difficult to assess exposure and health impacts of EMF.

⁷⁷⁰ EMFs produce low frequency, non-ionizing radiation rather than the high frequency ionizing radiation, produced by radon and other radioactive materials, which is a *known* source of molecular alteration and genetic mutation. See John F. Cahill, *An Introduction to the Indoor Pollution Problem*, 40 PRAC. LAW. 27, 51 (1994); see also Mandsorf, *supra* note 750, at 31; U.S. EPA, *supra* note 748, at 2; EMF, *supra* note 749, at 7, 8.

⁷⁷¹ On average, residences and offices expose people to 60 Hertz fields of about .1 to 3 mG; by comparison, the Earth's magnetic field is 500 mG. See Suplee, *supra* note 761, at A4; see also Joseph Mercola, *Preventive Environmental Medicine: Are EMFs Hazardous to Our Health?* (visited July 11, 1997) <<http://alt.medmarket.com/members/reiddds/herbplus/info/herb2art.html>> [hereinafter *Preventive*].

⁷⁷² Power (transmission) lines are the biggest *nearby* source of EMFs for home owners. There are over 600,000 miles of high voltage power lines in the United States exposing millions of people to EMFs every day. See DAVIS & SCHAFFMAN, *supra* note 4, at 211.

⁷⁷³ See Roy F. Krieger & Michael E. Withey, *EMF and the Public Health*, 9 NAT. RESOURCES & ENV'T 3, 3 (1994). Large magnetic fields have been discovered in residences. There is, however, little data available concerning the contribution of in-home electrical wiring and grounding practices on magnetic fields. However, household appliances are usually on for short periods of time and, thus, exposure is limited. See generally Weiss, *supra* note 748, at 363.

⁷⁷⁴ The earth's magnetic field permits compasses to work. See Weiss, *supra* note 748, at 361; see also Liljestrand, *supra* note 746, at 401; U.S. EPA, *supra* note 748, at 4 (discussing earth's magnetic field being 500 mg).

⁷⁷⁵ See Liljestrand, *supra* note 746, at 401.

2. Health Effects of Electromagnetic Fields

EMF exposure once was believed to be harmless; however, this view is no longer the consensus among experts.⁷⁷⁶ The question remains, however, whether EMFs cause significant human health problems. The potential health effects of EMFs have been debated since World War II military personnel who were exposed to powerful radar systems and video screens began to experience health problems they attributed to EMF exposure.⁷⁷⁷ In the U.S., the EMF health debate became a general topic of concern in 1979 when a study concluded that children with leukemia in the Denver area were more likely to have had homes near electric power lines.⁷⁷⁸ Since then, there have been many worldwide studies of the effects of EMF exposure, some of which are mentioned in the text that follows.

Throughout the 1980s, EPA reviewed studies addressing the carcinogenicity of EMFs. In 1990, public anxiety concerning EMF exposure began to escalate when Paul Brodeur authored a series of articles in the *New Yorker* that became a book on EMFs entitled *CURRENTS OF DEATH*.⁷⁷⁹ In 1990, EPA published a draft report that concluded "several studies showing leukemia, lymphoma and cancer of the nervous system in children exposed to magnetic fields from residential 60-Hz electrical power distribution systems . . . show a consistent pattern of response which suggests a causal link."⁷⁸⁰ Today, public concerns continue regarding the potential threats of invisible EMF radiation.

In general, public health concerns focus on EMF produced by: electric transmission and distribution facilities (especially high-voltage power lines),⁷⁸¹ electric appliances, household wiring, and industrial machinery. Other potentially harmful sources of EMFs include

⁷⁷⁶ See Young & Gunther, *supra* note 748, at 2.

⁷⁷⁷ See *Electromagnetic Fields Pose Little Threat, Research Council Finds*, Daily Env't Rep. (BNA) at A-2 (Nov. 4, 1996) [hereinafter *Little Threat*]; see also Cahill, *supra* note 770, at 50.

⁷⁷⁸ See Krieger & Withey, *supra* note 773, at 4; QUESTIONS AND ANSWERS, *supra* note 746, at 12; see generally Kathiann M. Kowalski, *Can EMFs Hurt You?*, 22 CURRENT HEALTH 30 (1995).

⁷⁷⁹ See Suplee, *supra* note 760, at A4. See generally PAUL BRODEUR, *CURRENTS OF DEATH: POWER LINES, COMPUTER TERMINALS AND THE ATTEMPT TO COVER UP THEIR THREAT TO YOUR HEALTH* (1989).

⁷⁸⁰ See U.S. ENVTL. PROTECTION AGENCY, No. EPA/600/6-90/005B, *EVALUATION OF THE POTENTIAL CARCINOGENICITY OF ELECTROMAGNETIC FIELDS 1-7* (1990)

⁷⁸¹ It is now widely believed that EMFs from power lines are too low to cause genetic damage. See Anne D. Walling, *Risk of Cancer Associated with Exposure to Power Lines; Tips from Other Journals*, 55 AM. FAMILY PHYSICIAN, Feb. 15, 1997, at 944; see also *EMF's: Experts Downplay*

cellular telephones, police radar guns, hair dryers, electric blankets, computers, television sets, and microwave ovens. Researchers do not know at what levels EMF exposure can be considered "safe" or "unsafe."⁷⁸² While many household and office appliances generate electromagnetic fields, most are regarded by the public as being "safe," possibly because most exposures are of short duration. While EMF strengths diminish rapidly with distance from the source, some research has shown that biological effects of EMF exposure that appear at certain levels of exposure will disappear at higher levels, only to reappear at still higher levels. One major problem with EMF research is that scientists and researchers do not know what harmful effects they are looking for, and they do not know what aspect of field exposure causes a harmful effect—extended exposure, for example, may not be the harmful factor.⁷⁸³ Often, when health effects are found, scientists are unable to replicate the results in other studies, and studies frequently contradict each other.⁷⁸⁴

Because we are constantly exposed to electromagnetic radiation from the use of electricity, it is unlikely that EMFs are a significant hazard at normal exposure levels.⁷⁸⁵ Most of the studies done in more than two decades of research indicate an increase in risk of illness due to EMF exposure.⁷⁸⁶ All forms of non-ionizing radiation (including EMF) are capable of causing damage by heating body tissues.⁷⁸⁷ However, there is no conclusive proof that low frequency non-ionizing radiation can produce the adverse health effects created by ionizing radiation, which is a known source of molecular alteration and genetic mutation.⁷⁸⁸ EMFs from 60-Hz power cannot break apart human cells.⁷⁸⁹ However, there is some evidence that EMFs can create weak electric currents in the bodies of people and animals,⁷⁹⁰ and these weak

Link Between Power Lines, Cancer, GREENWIRE, Nov. 1, 1996, (page unavail.), available in LEXIS, Envirn Library, Allnws File.

⁷⁸² EPA has stated: "The bottom line is that there is no established cause and effect relationship between EMF exposure and cancer or other disease. For this reason, we can't define a hazardous level of EMF exposure" U.S. EPA, *supra* note 748, at 9.

⁷⁸³ See Mansdorf, *supra* note 750, at 32.

⁷⁸⁴ See generally Kowalski, *supra* note 778, at 30.

⁷⁸⁵ See Mansdorf, *supra* note 750, at 32.

⁷⁸⁶ See Krieger & Withey, *supra* note 773, at 46. By comparison, carcinogens such as asbestos and tobacco smoke result in risk increases as much as forty times normal. See *id.*

⁷⁸⁷ See Mansdorf, *supra* note 750, at 31.

⁷⁸⁸ See QUESTIONS AND ANSWERS, *supra* note 746, at 9.

⁷⁸⁹ See *id.*

⁷⁹⁰ See *id.* Scientists argue that a cell may respond to the induced EMF current as a signal,

currents may affect natural processes that occur at the cellular and molecular levels.⁷⁹¹

The purported adverse health effects of EMFs are the result of involuntary exposure, and usually are delayed in their manifestation—factors that contribute to increased anxiety. Effects depend on several parameters such as strength, direction, and rate of change of the field, as well as time of the exposure.⁷⁹² Adverse effects may also depend on genetic differences among people so that only small subpopulations may be affected. While EMFs can produce a variety of biological effects on the human body, in most cases it is not clear how EMFs produce these effects.⁷⁹³ The many published investigations about whether exposure to EMFs results in adverse health effects, particularly leukemia and other cancers,⁷⁹⁴ provide no clear answers. There is cause for concern, and the general agreement is that better information is needed.⁷⁹⁵

Society's main concern about EMF exposure involves its correlation to cancer. A 1990 EPA draft report evaluating the potential carcinogenicity of EMFs recommended that they be classified as a Class B carcinogen.⁷⁹⁶ However, because of significant criticism from the scientific community, EPA noted there was insufficient data to determine a cause and effect relationship between cancer and exposure to EMFs.⁷⁹⁷ Some subsequent studies supported this view,⁷⁹⁸ but other studies raise the possibility that EMFs may present some risks, in particular by encouraging the growth of cancer cells.⁷⁹⁹ Epi-

detectable even though currents from 60-Hz EMS are weaker than natural currents of the body. *See id.*

⁷⁹¹ *See* Cahill, *supra* note 770, at 51; *see also* Hileman, *supra* note 759, at 29–30. However, currents from 60-Hz EMFs are weaker than the natural currents in the body and, therefore, some scientists argue that it is impossible for EMFs to have any important biological effects. The currents EMFs induce in living organisms are approximately 1,000 times smaller than the electric currents that animals produce normally in their brain cells and in the nerve trains that trigger heart muscles to beat. *See* Suplee, *supra* note 761, at A4.

⁷⁹² *See* EMF, *supra* note 749, at 24.

⁷⁹³ *See* QUESTIONS AND ANSWERS, *supra* note 746, at 8–9.

⁷⁹⁴ Note that while most of the recent research on possible adverse health effects from EMFs has concentrated on magnetic fields, a 1996 study suggested that experts should pay more attention to electric fields in the future. *See Little Threat*, *supra* note 777, at A-2.

⁷⁹⁵ *See* QUESTIONS AND ANSWERS, *supra* note 746, at 1.

⁷⁹⁶ *See generally* Preventive, *supra* note 771.

⁷⁹⁷ *See id.*

⁷⁹⁸ In 1995, for example, attempts to replicate findings linking EMF exposure to cancer failed completely, rebutting the decade-long work of two scientists. *See* Gary Taubes, *Another Blow Weakens EMF-Cancer Link*, *SCI.*, Sept. 29, 1995, at 1816–17.

⁷⁹⁹ *See* Marilitz Dizon, *Naturally Healthy*, *BUS. DAILY*, Nov. 25, 1996 (page unavail.); *see*

demiological studies have suggested that a link may exist between EMF exposure and certain types of cancer, primarily leukemia,⁸⁰⁰ brain cancer,⁸⁰¹ lymphoma, and breast cancer.⁸⁰² Even if 60-Hz EMFs do not cause cancer, some scientists suspect that they might promote growth of tumors that start some other way.⁸⁰³ Studies suggest that EMFs may slow production of melatonin, a hormone believed to fight cancer.⁸⁰⁴

EMF exposure experiments also have suggested that magnetic fields can affect brain cells, cause drops in hormone levels and other chemicals that the body manufactures, and produce functional changes in isolated cells and tissues.⁸⁰⁵ In addition, animal studies have shown effects on the central nervous system and increased incidence of skin tumors in mice. Questions also have been raised about increased risk of birth defects⁸⁰⁶ and chronic depression.

In 1993, a panel commissioned by the U.S. Department of Labor reviewed more than one thousand EMF studies and reported that "there is no convincing evidence . . . to support the contention that exposures to EMFs generated by such sources as household appliances, video display terminals, and local power lines are demonstrable health hazards."⁸⁰⁷

In 1994, two studies indicated that people with high (occupational) exposure to EMFs are at least three times as likely to develop Alzhe-

generally P. Brown, *Shocking Safety Concerns*, 348 LANCET 959 (1996); Winifred Conkling, *Shocking Charges; How Electromagnetic Fields Affect Health*, 12 AM. HEALTH 50 (1993).

⁸⁰⁰ In 1996, a study linked exposure to electric fields to higher-than-expected rates of leukemia. This study is in contrast to a 1995 study that found no correlation between EMFs and leukemia in electrical utility workers, although the workers did have about twice the death rate from brain cancer as the national average. See *Surprising EMF Study Result*, 57 OCCUPATIONAL HAZARDS 26 (Mar. 1995).

⁸⁰¹ See *id.*

⁸⁰² See Krieger & Withey, *supra* note 773, at 3; see also QUESTIONS AND ANSWERS, *supra* note 746, at 1, 20; Bette Hileman, *Findings Point to Complexity of Health Effects of Electric, Magnetic Fields*, 72 CHEM. & ENGINEERING NEWS, July 18, 1994, at 27.

⁸⁰³ See generally Kowalski, *supra* note 778, at 30.

⁸⁰⁴ See *id.*

⁸⁰⁵ See Mansdorf, *supra* note 750, at 32; see also Liljestrand, *supra* note 746, at 402. EMFs may cause calcium atoms in nerve cells to lose electrons and become charged ions, which may cause the cells to die, or EMFs may alter the flow of calcium ions from cell to cell. See James A. Haught, *Power Peril: Alzheimer's Link Ominous*, THE CHARLESTON GAZETTE, Aug. 3, 1994, at 4A.

⁸⁰⁶ A 1991 study found no connection between the use of video display terminals (VDT's) and miscarriages. See Susan Okie, *No Link Found Between VDT's, Miscarriages*, WASH. POST, Mar. 14, 1991, at A1.

⁸⁰⁷ See DAVIS & SCHAFFMAN, *supra* note 4, at 212.

imer's disease as those without significant exposure.⁸⁰⁸ In 1995, a panel of the National Council on Radiation Protection and Measurements (NCRP) concluded that some health effects linked to EMF appear real and warrant steps to reduce EMF exposure.

In 1995, after tracking the link between EMFs and cancer for seven years, the American Physical Society issued a policy statement saying that the public has overreacted and that studies have not shown a connection between EMF and cancer.⁸⁰⁹ Reviews by EPA, the Australian Minister of Health, the National Radiological Protection Board of the United Kingdom, the Danish Ministry of Health, the Swedish National Electrical Safety Board and a number of state-sponsored reviews,⁸¹⁰ have concluded that although evidence of EMF causing cancer is suggestive, it does not show EMF causes cancer.⁸¹¹

The National Research Council (NRC) stated in its 300-page report, *Possible Health Effects of Exposure to Residential Electric and Magnetic Fields*, released in 1996, that the kinds of EMFs generated in and around the average American home pose no discernible hazard to human health.⁸¹² The panel found that the only cause for concern was a "weak but statistically significant association between proximity to high-voltage electrical transmission lines and childhood leukemia."⁸¹³

⁸⁰⁸ See Thomas H. Maugh II, *Studies Link EMF Exposure to Higher Risk of Alzheimer's*, L.A. TIMES, July 31, 1994, at A3; see generally *Further Study Needed on EMF/Alzheimer's Link*, 1991 U.S. Total AD Cost \$67 Bil., USC Researchers Report, THE BLUE SHEET, Aug. 24, 1994, at 12-13; E. Sobel et al., *Occupations With Exposure to Electromagnetic Fields: A Possible Risk Factor for Alzheimer's Disease*, 142 AM. J. EPIDEMIOLOGY 515 (1995). In comparison, the cancer risk associated with EMFs is believed to be 1.5 to 1.8 times as high among those with occupational exposure. See Haught, *supra* note 805, at 4A.

⁸⁰⁹ See American Physical Society, *APS Council Adopts Statement on EMFs and Public Health* (July 1995) <<http://aps.org/cpsnews/articles/11209.html>>.

⁸¹⁰ See QUESTIONS AND ANSWERS, *supra* note 746, at 61.

⁸¹¹ See *id.* at 26.

⁸¹² See *Little Threat*, *supra* note 777, at A-2; see also Suplee, *supra* note 761, at A4; *Sound Science, Sound Energy Policy: A Senator Reflects*, RESOURCES, Winter 1997, at 16. The group examined seventeen years of research and more than 500 published studies.

⁸¹³ Suplee, *supra* note 761, at A4. However, the panel could not relate that finding to the effects of EMFs and concluded that further study may be needed. *Id.*; see also *Little Threat*, *supra* note 777, at A-2. This study followed a publication in October 1996, from Finland, which found that residential EMFs "do not seem to be related to the risk of overall cancer in adults." Suplee, *supra* note 761, at A4. The National Cancer Institute recently published the results of a major study on the effect of power line EMFs on children, supplying additional evidence that these EMFs have little, if any, effect on causing cancer in children. See Curt Suplee, *No Greater Cancer Risk is Found in Children Living Near Power Lines: Federal Study Tries to Shed Light on High-Voltage Debate*, WASH. POST, July 3, 1997, at A3.

Although there is uncertainty as to the adverse health effects of EMFs, in some instances, EMF has positive applications. For example, EMF radiation from a long low-frequency radio antenna apparently stimulated growth in nearby trees and river algae.⁸¹⁴ EMFs are also used to mend broken bones.⁸¹⁵

3. Control of Electromagnetic Fields

a. Regulation

Four federal agencies that regulate EMF emissions or emitting devices are: the Department of Labor, the Department of Health and Human Services, the Federal Communications Commission,⁸¹⁶ and the Consumer Product Safety Commission.⁸¹⁷ No federal or state standards have been set governing exposure to electromagnetic fields, and no laws regulate EMFs, although a few bills aimed at setting federal standards and increasing EMF research and public information funding have been proposed.⁸¹⁸ It is unlikely that the federal government will do more than fund research in the next few years because the scientific evidence is not yet adequate to rationally establish limits. In addition, efforts to control EMF exposure would be expensive. Three organizations, the International Commission on Non-Ionizing Radiation Protection, the American Conference of Governmental Industrial Hygienists, and the American National Standards Institute, have developed standards or guidelines for 60-Hz EMF exposure.⁸¹⁹ OSHA

⁸¹⁴ See *EMF Good for Trees?*, SCI., Jan. 27, 1995, at 451.

⁸¹⁵ See Hileman, *supra* note 759, at 32. See also *Electromagnetic Fields* (last modified Feb. 14, 1996) <<http://www.abwam.com/grossing/refemt.html>> [hereinafter EF]. Researchers reported results that EMFs have a beneficial effect on bone healing for fractures or hip problems that resist repair with ordinary treatment. In many places, use of pulsed EMF to help repair difficult-to-heal fractures is accepted procedure. But some physicians have questioned whether EMFs provide any benefit above that provided by plaster casts alone. See *id.*

⁸¹⁶ For example, the FCC set maximum allowable exposure at the base of a R/C tower containing high-powered transmitters by requiring a worst-case approximation of RF levels that assumes all antennas are transmitting simultaneously on all channels at full power. The FCC excludes cellular towers from the requirement to perform RF exposure calculations because of their relatively low power. See DAVIS & SCHAFFMAN, *supra* note 4, at 298. EPA also has a program on EMFs but does not have much of a budget to follow the issue; currently, there are no plans to propose a rule regarding residential power lines. See *Little Threat*, *supra* note 777, at A-3.

⁸¹⁷ See Young & Gunther, *supra* note 748, at 4.

⁸¹⁸ See, e.g., H.R. 1982, 103rd Cong. (1993); H.R. 1494, 103rd Cong. (1993); H.R. 3953, 102nd Cong. (1981).

⁸¹⁹ See QUESTIONS AND ANSWERS, *supra* note 746, at 29; Mansdorf, *supra* note 750, at 32.

has set a standard for radio frequency EMF in the 10 MHz to 100 GHz range,⁸²⁰ but does not have an EMF standard applicable to power lines.

Internationally, some EMF standards have been established. For example, the United Kingdom derived national magnetic limits from guidelines issued by WHO in 1984.⁸²¹ In January 1990, the International Radiation Protection Association issued interim standards based upon those guidelines.⁸²² Worldwide EMF regulations are set based on biological effects that occur at high EMF levels, on currents known to be induced in the body by magnetic fields, or on eliminating the effects that can be detected near some power lines.⁸²³

Most EMF control efforts have been at the state and local levels.⁸²⁴ Policies are not uniform. Recent actions by states and localities range from minimal controls to bans on the construction of transmission lines and similar projects.⁸²⁵ Seven states have safety-based limits on electric field strength.⁸²⁶ Two states, Florida and New York, have imposed magnetic field strength limits.⁸²⁷ Suffolk County, New York, and San Francisco, California, have guidelines limiting continuous work at video display terminals (VDTs).⁸²⁸ These laws generally are not based on scientific determinations concerning the safe level of magnetic field exposure, but rather on the assumption that the status quo is to be maintained. In addition, the California Public Utilities

⁸²⁰ See 29 C.F.R. § 1910.97(a)(2)(i) (1997).

⁸²¹ EF, *supra* note 815.

⁸²² See *id.*

⁸²³ See Hileman, *supra* note 759, at 33.

⁸²⁴ Many states, including California, Colorado, Connecticut, Florida, Illinois, New Jersey, New York, Ohio, Rhode Island, Texas, and Wisconsin, have some kind of EMF standard, guideline, or policy. Many require utilities to avoid populated areas or places like schools or daycare centers when planning power lines. New York and Florida are the only states that have specific EMF standards for new and upgraded power lines—they set allowable EMF levels at the edge of the right-of-way. These are not health-based exposure standards but rather are based on averages for existing power lines. See DAVIS & SCHAFFMAN, *supra* note 4, at 214; see also QUESTIONS AND ANSWERS, *supra* note 746, at 28.

⁸²⁵ For example, Florida, New York, and the City Commission of Brentwood (a suburb of Nashville, Tennessee) passed limitations on magnetic fields. In Tennessee and Michigan, legislation was proposed that would impose a moratorium on new construction of certain power lines, while similar legislation was narrowly defeated in Rhode Island. See Scott H. Strauss & Susan M. Bernard, *Power and the People*, 8 ENVTL. F., Nov.-Dec. 1991, at 13-14.

⁸²⁶ See Weiss, *supra* note 748, at 360. These states are Florida, Minnesota, Montana, New Jersey, New York, North Dakota, and Oregon. See *id.* at 380; see also QUESTIONS AND ANSWERS, *supra* note 746, at 28.

⁸²⁷ Strauss & Bernard, *supra* note 825, at 13.

⁸²⁸ See EF, *supra* note 815.

Commission has adopted interim policies implementing no-cost and low-cost steps to reduce EMF levels, and is working to develop EMF guidelines.⁸²⁹

EMF also is regulated in a piecemeal manner by the courts. As a result of the universal nature of EMF exposure and the public's distrust of technology, the issue of EMF exposure is an emerging area of tort litigation. There are primarily four areas where EMF litigation has developed: 1) owners of private property are bringing suits to recover for diminution in market value of their property (i.e., eminent domain, condemnation, inverse condemnation);⁸³⁰ 2) groups and communities are bringing suits to block proposed new developments such as the building of power lines;⁸³¹ 3) individuals exposed to EMF are seeking compensation;⁸³² and 4) employees are bringing worker's compensation suits for alleged injuries due to EMF exposure. Utilities, however, have been defending lawsuits effectively, alleging that

⁸²⁹ See Young & Gunther, *supra* note 748, at 5.

⁸³⁰ Courts take three views with respect to power line condemnation cases. See generally Willsey v. Kansas City Power & Light Co., 631 P.2d 268 (Kan. Ct. App. 1981). In August of 1996, the California Supreme Court affirmed a lower court ruling against plaintiffs' claims that alleged EMF fields emitted from electric power lines had caused them emotional distress, made their home uninhabitable, and destroyed its market value. See San Diego Gas & Elec. Co. v. Superior Court, 920 P.2d 669, 705 (Cal. 1996). In December of 1996, a New York appeals court held that land owners do not have an inverse condemnation claim against an electric utility company for diminution in property value caused by electromagnetic power lines. See Reiss v. Consolidated Edison Co., 650 N.Y.S.2d 480, 482 (N.Y. App. Div. 1996); see also *No Recovery For Diminished Property Value Allegedly Caused by Power Company's Lines*, 11 Toxics L. Rep. (BNA) at 863 (Jan. 15, 1997).

⁸³¹ See, e.g., *City Opposes Utah Power Line Plan Due to Aesthetics, EMF Concerns*, UTIL. ENV'T REP. at 6 (July 19, 1996).

⁸³² Note that according to an Illinois appeals court, the FDA has primary jurisdiction over cellular telephone claims alleging that the manufacturer failed to warn users about harm from electromagnetic waves. See Verb v. Motorola, Inc., 672 N.E.2d 1287, 1293 (Ill. App. Ct. 1996); see also *FDA Has Exclusive Jurisdiction Over Claims Alleging Health Risks From Cellular Phones*, 11 Toxics L. Rep. (BNA) at 797 (Dec. 18, 1996). EMF litigation focusing on illness caused by cellular phones is a promising new area. But see Ward v. Motorola, Inc., No. S97C0483, 1997 Ga. LEXIS 336 (Ga. Mar. 7, 1997) (declining to review suit dismissing action alleging that EMF from cellular phone caused brain tumor). See Richard C. Reuben, *Utility Power Plays*, ABA J., Dec. 1996, at 18-19. In 1997, a Florida appeals court ruled that a power company had no duty to warn of potential health risks from electromagnetic fields. See generally Glazer v. Florida Power & Light, 689 So.2d 308 (Fla. Ct. App. 1997); *Power Company Had No Duty to Warn Couple of Health Risks From EMF in Plumbing Lines*, 11 Toxics L. Rep. (BNA) at 1059 (Feb. 26, 1997).

Probably the largest EMF verdict awarded was in 1996, when a New Jersey jury refused to find that EMF caused a homeowner's cancer but still awarded \$500,000 for the negligent infliction of emotional distress. See Altoonian v. Atlantic City Elec. Co., No. L-1342-91, 1996 N.J. LEXIS 651 (N.J. Mar. 28, 1996).

power lines cause cancer in people who live nearby.⁸³³ As a result, litigation in this area has decreased.⁸³⁴

Between 1985 and 1994, over 100 EMF suits had been filed, and an article reported that in 1992 alone, 201 challenges to utility projects were based on EMF concerns.⁸³⁵ As of 1993, there were twenty-five EMF personal injury cases before the courts. In 1996, a total of seventy-nine EMF cases were currently pending in the U.S., a majority of which were property damage suits.⁸³⁶ The existence of a causal connection between EMFs and cancer generally is accepted in EMF cases, although a clear link between EMFs and cancer has not yet been established.⁸³⁷ The potential for EMF cases becoming common tort actions will depend, in part, on the restrictions imposed by courts concerning the use of questionable scientific evidence based on the *Daubert* test.⁸³⁸ In an important case, the California Supreme Court ruled that the California Public Utilities Commission has exclusive jurisdiction over claims of alleged injuries from EMF emitted by power lines.⁸³⁹ The effect of this decision is to prevent tort actions for alleged EMF injuries from being brought in California trial courts.⁸⁴⁰

b. Research

Because EMF has the potential to affect virtually everyone, the EMF issue has led to an increase in private and government-funded research. By 1990, over one hundred EMF studies had been conducted worldwide, and at least two dozen epidemiological studies

⁸³³ See Reuben, *supra* note 832, at 18.

⁸³⁴ See *id.* In addition, the slowdown is attributed to the difficulty in establishing a causal link between EMF and physical harms and also to the high costs associated with bringing a case. See *id.*

⁸³⁵ See Giroux, *supra* note 767, at 394.

⁸³⁶ See *EMF Litigation Swells*, WORLD INSURANCE REP., Sept. 20, 1996, (page unavail.), available in LEXIS, News Library, Curnws File.

⁸³⁷ See generally Gabriella Krockmalnic, *An Update on Indoor Air Quality Issues*, 3 MASS. ENVTL. COMPLIANCE UPDATE (Oct. 1995).

⁸³⁸ See *Daubert v. Merrell Dow Pharm. Inc.*, 509 U.S. 579 (1993); see also *Joiner v. General Elec. Co.*, 78 F.3d 524 (11th Cir. 1996), *cert. granted*, *General Elec. Co. v. Joiner*, 65 U.S.L.W. 3629 (Mar. 17, 1997) (No. 96-188); Richard M. Bernstein, 'Daubert' Revisited: *The Proper Standard of Review*, 11 TOXICS L. REP. (BNA) at 1389 (May 14, 1997). The U.S. Supreme Court rejected a petition for certiorari in an indoor air pollution case involving carpet adhesive fumes. See *Guilbeau v. W.W. Henry Co.*, 85 F.3d 1149 (5th Cir. 1996), *cert. denied* (No. 96-27) (Jan. 21, 1997).

⁸³⁹ See *San Diego Gas & Elec. Co. v. Superior Court*, 920 P.2d 669, 687 (Cal. 1996).

⁸⁴⁰ See *California High Court Says Utility Commission Has Exclusive Jurisdiction Over EMF Lawsuits*, 11 TOXICS L. REP. (BNA) at 396 (Sept. 4, 1996).

indicated a link between EMFs and serious human health problems.⁸⁴¹ There are now over two hundred worldwide studies⁸⁴² under way or planned involving EMF.⁸⁴³

The federal government has conducted EMF studies for almost twenty years, involving more than a dozen federal government agencies.⁸⁴⁴ Several states, including New York and California, also have sponsored EMF-related research.⁸⁴⁵ In the U.S., public and private funds support EMF research.⁸⁴⁶ The federal government has spent over \$60 million on EMF research.⁸⁴⁷ EPA has reported that the priorities for EMF research should be: (1) cancer effects, biophysical mechanisms and human exposure assessment; (2) productive and nervous system effects; and (3) immune system effects and control technology.⁸⁴⁸

To accelerate EMF research efforts in the U.S., specifically to determine how EMF affects human cells, the Energy Policy Act of 1992⁸⁴⁹ included a provision for a five-year, \$65 million program of EMF Research and Public Information Dissemination (RAPID), supported by both federal and matching non-federal funds and coordinated by the Department of Energy and the National Institute of Environmental Health Sciences.⁸⁵⁰ As directed by this Act, nine federal agencies formed an Electric and Magnetic Fields Interagency Committee in 1993 to conduct research and inform the public on

⁸⁴¹ See *Preventive*, *supra* note 771.

⁸⁴² At least twenty-two countries are conducting the EMF research. See QUESTIONS AND ANSWERS, *supra* note 746, at 31.

⁸⁴³ See Krieger & Withey, *supra* note 773, at 4; see also QUESTIONS AND ANSWERS, *supra* note 746, at 30. These include epidemiologic, laboratory, environmental, and engineering studies. Many of the studies involve cancer development. They are sponsored by federal and local government agencies and private organizations, including electric utilities and appliance manufacturers. See *id.*

⁸⁴⁴ See QUESTIONS AND ANSWERS, *supra* note 746, at 31. Examples of EMF research by federal agencies include: 1) Department of Defense—radio frequency/breast cancer studies; 2) Department of Energy—exposure assessment; 3) Department of Transportation—high speed and maglev trains; 4) EPA—radiofrequency guidelines and research, and 5) Department of Health and Human Services—cellular phones, electric blankets, video display terminals, medical devices, health effects, and risk assessment. See *id.*

⁸⁴⁵ See *id.* For example, in 1988, California enacted a law requiring utilities to spend \$2 million on EMF research. See Weiss, *supra* note 748, at 379.

⁸⁴⁶ See QUESTIONS AND ANSWERS, *supra* note 746, at 31.

⁸⁴⁷ See U.S. EPA, *supra* note 748, at 20.

⁸⁴⁸ See *id.* at 12.

⁸⁴⁹ 42 U.S.C. § 13478 (1994).

⁸⁵⁰ See QUESTIONS AND ANSWERS, *supra* note 746, at 30; see also U.S. Dep't of Energy & Nat'l Inst. of Env'tl. Health Sciences, *Progress Report on the Electric and Magnetic Fields Research and Public Information Dissemination Program* (visited July 8, 1997) <http://www.niehs.nih.gov/emfrapid/RepToCongress/INT_RPT.html> at 3 of 16.

EMFs.⁸⁵¹ The Committee's final report is scheduled for release on September 30, 1997.⁸⁵²

On June 4, 1996, WHO announced an international five-year study to assess the health effects of exposure to EMFs.⁸⁵³ WHO expects to publish four monographs in the Environmental Health Criteria Series. The project is funded through contributions by interested governments and institutions. Representatives of twenty-three countries and six international organizations participated in the first preparatory meeting of the EMF project in May 1996.⁸⁵⁴

c. *Exposure Reduction*

Many experts feel that exposure to EMF should be limited, particularly at night.⁸⁵⁵ Although it would be impossible to avoid EMFs completely,⁸⁵⁶ there are ways to reduce exposure to EMFs. The recommended course of action is "prudent avoidance,"⁸⁵⁷ which is the pursuit of no-cost or minimal-cost strategies to reduce EMF exposures.⁸⁵⁸ Some of these strategies are as easy as increasing the distance between yourself and the EMF source. Magnetic fields can also be reduced by enclosing a source in certain types of metal, but this is not practical for many EMF sources.

EMF levels can be measured using a portable device known as a "gaussmeter."⁸⁵⁹ These devices can be purchased (for \$100 to \$200) or

⁸⁵¹ The nine federal agencies which form the committee are Department of Energy (DOE), National Institute of Environmental Health Sciences, EPA, Department of Defense, OSHA, National Institute of Standards and Technology, Department of Transportation, Rural Electrification Administration, and Federal Energy Regulatory Commission. DOE administers the program. See *Federal Agencies Begin Five-Year Study on EMF*, 2 Mealey's Litig. Reps.: Toxic Torts at 1 (Apr. 1, 1993).

⁸⁵² See *id.*

⁸⁵³ See *WHO Launches Study of EMFs, Health Effects*, 5 Mealey's Emerging Toxic Torts at 5 (June 14, 1996).

⁸⁵⁴ See *id.*

⁸⁵⁵ See Wartenberg, *supra* note 747; see generally Frederica Templeton, *A Healthy House: Addressing House-Related Health Hazards*, COUNTRY LIVING, June 1996, at 66.

⁸⁵⁶ There is no simple way to block EMFs indoors because the fields are generated by the electrical system and devices that are indoors. See U.S. EPA, *supra* note 748, at 16.

⁸⁵⁷ This term was coined by Professor M. Granger Morgan of the Department of Engineering and Public Policy at Carnegie Mellon University in a 1989 report. See U.S. CONGRESS OFFICE OF TECHNOLOGY ASSESSMENT, BIOLOGICAL EFFECTS OF POWER FREQUENCY ELECTRIC AND MAGNETIC FIELDS: BACKGROUND PAPER (1989). See Krieger & Withey, *supra* note 773, at 3; see also EF, *supra* note 815; Young & Gunther, *supra* note 748, at 15.

⁸⁵⁸ See *Electro-Magnetic Fields* (visited July 2, 1997) <<http://www.greenbuilder.com/Sourcebook/Emf.html>>. Because scientists continue to debate whether EMFs are a health hazard, it is not clear how much should be done to reduce exposure.

⁸⁵⁹ See DAVIS & SCHAFFMAN, *supra* note 4, at 215.

rented (for under \$75) or an inspector can do the measurement as part of a home inspection. However, currently there is no simple way to use the results because there are no accepted and established safe standards in the U.S.⁸⁶⁰

I. *Building Sickness*

The oil crises of the 1970s created the impetus for Americans to modify homes and buildings to conserve energy.⁸⁶¹ Due to the "weatherization" of buildings, combined with the use of synthetic building materials, cleaning and pest control products, office machines, and central heating and air conditioning, the level of indoor air pollutants increased. While the amount of indoor air pollution increased, the air exchange in buildings decreased. Thermal control became the dominant determinant of system design, and adequate air flow became an incidental consideration.⁸⁶² Moreover, operation and maintenance failures resulted in air exchange systems not functioning as designed.⁸⁶³ This led to claims that buildings were making people sick. Illnesses apparently related to work environments have focused on the workplace using the scientifically inaccurate but descriptive term "building sickness."⁸⁶⁴ To assist in the evaluation process, EPA has established three classifications of acute building sickness: building-related illness; sick building syndrome; and multiple chemical sensitivity.⁸⁶⁵

⁸⁶⁰ See *id.* For more information on EMF, the EPA and EMF Research and Information Dissemination ("RAPID") Program runs an EMF hotline at 800-EMF-2383. In Washington, D.C., call 484-1803. (FAX (703) 821-8236; local (703) 442-8934.) Another source for EMF information is Microwave News, at (212) 517-2800.

⁸⁶¹ The Arab states first cut off petroleum shipments to the United States in 1973. This led to legislation to move the nation to energy self-sufficiency (which failed to achieve its goal) and to private sector fuel conservation efforts in response to the dramatic increase in the costs of petroleum-based products. See Arnold W. Reitze, Jr., *Environmental Policy—It Is Time For A New Beginning*, 14 COL. J. ENVTL. L. 111, 139 (1989).

⁸⁶² See Hal Levin, *IAQ: Whose Responsibility?*, 19 EPA J., Oct.-Dec. 1993, at 34.

⁸⁶³ See *id.*

⁸⁶⁴ See generally Andrew Blum, *Sick Building Syndrome: Structures Face Legal Scrutiny Over Illnesses*, NAT'L L.J., Jan. 25, 1988, at 1; Charles Edward Anderson, *Sick-Building Syndrome: Suits Increase for Indoor Pollution Despite Absence of Favorable Verdict*, A.B.A. J., Dec. 1990, at 17.

"Since the early 1970s, outbreaks of work-related health complaints have occurred in large numbers in a wide variety of nonindustrial workplaces such as hospitals, schools, and office buildings." Marbury & Woods, *supra* note 625, at 306.

⁸⁶⁵ See Marbury & Woods, *supra* note 625, at 307.

1. Building-Related Illness (BRI)

Building-related illness includes illness due to "exposure to the building air, where symptoms of illness, including infection, fever, and clinical signs of pathology, are identified and an airborne pathway for the stressor is recognized."⁸⁶⁶ According to Marian C. Marbury and James E. Woods, Jr., nosocomial infections, humidifier fever, hypersensitivity pneumonitis, Legionnaire's Disease,⁸⁶⁷ and symptoms of chemical exposure are the most prevalent forms of building-related illness.⁸⁶⁸ The symptoms of building-related illness may not subside when exposure ends.⁸⁶⁹ Susceptibility to building-related illness is influenced by factors such as the patient's age and immune system status.⁸⁷⁰ One health incident associated with indoor air problems from carpets occurred at EPA's Washington, D.C., office in 1987, when hundreds of employees became sick after new carpeting was installed, allegedly because of 4-PC, a component of carpet backing.⁸⁷¹

2. Sick Building Syndrome

In commercial buildings, an indoor air pollution problem often is referred to as "sick building syndrome."⁸⁷² Sick building syndrome refers to acute health and discomfort effects (or symptoms) experienced by a substantial percentage of a building's occupants⁸⁷³ as a

⁸⁶⁶ *Id.* In other words, a particular building problem has to be linked to a particular illness. See generally Geisler, *supra* note 334, at 511.

⁸⁶⁷ In 1976, "Legionnaire's Disease" was added to our vocabulary when the first reported occurrence of BRI caused 182 cases of pneumonia, including twenty-nine deaths, which occurred at a hotel in Philadelphia. See Geisler, *supra* note 334, at 512.

⁸⁶⁸ These health effects are the result of exposure to bioaerosols, bacteria, and chemical or biologic substances. See Marbury & Woods, *supra* note 625, at 307.

⁸⁶⁹ See *id.* at 307-08. These symptoms include cough, chest tightness, fever, chills, and muscle aches.

⁸⁷⁰ Building-related illness is traceable to specific sources in buildings; often it is linked to bacteria found in the ventilation system. See Gene J. Heady, Comment, *Stuck Inside These Four Walls: Recognition of Sick Building Syndrome Has Laid the Foundation to Raise Toxic Tort Litigation to New Heights*, 26 TEX. TECH L. REV. 1041, 1042 n.6 (1995).

⁸⁷¹ See Guiffida, *supra* note 3, at 316-17. Another interesting indoor air pollution case involved inmates of a Massachusetts prison claiming that exposure to airborne asbestos fibers throughout the prison facility violates their Eighth Amendment right to be free from cruel and unusual punishment. In a procedural skirmish, a federal district court judge held that the complaint stated a cause of action and the case could move forward. See generally Gonyer v. McDonald, 874 F. Supp. 464 (D. Mass. 1995).

⁸⁷² See Mary Rose Kornreich, *Minimizing Liability for Indoor Air Pollution*, 4 TUL. ENVTL. L.J. 61, 61 (1990). In 1991, OSHA estimated this affected 1.2 million commercial buildings and up to 70 million workers. See also Guiffida, *supra* note 3, at 315.

⁸⁷³ See Office of Air and Radiation, U.S. Env'tl. Protection Agency, *Indoor Air Facts No. 4*

result of exposure to a number of indoor air pollutants, although no specific pollutant is identified.⁸⁷⁴ WHO estimates that nearly thirty percent of new and remodeled buildings worldwide may be afflicted with indoor air quality problems that may lead to sick building syndrome.⁸⁷⁵ Unlike building-related illness, the symptoms of sick building syndrome generally occur when the affected people enter the building and dissipate when they exit.⁸⁷⁶ Moreover, the occupants exhibit no specific illness or etiology. Instead, the affected occupants exhibit a spectrum of symptoms including irritation of the eyes, nose, throat and skin; neurotoxic symptoms, including mental fatigue and headaches; runny nose; dry cough; bronchial asthma; odor and taste complaints; and rashes and itches.⁸⁷⁷ Generally, there is no identification of any single exposure factor or specific cause.⁸⁷⁸ Part of the difficulty in determining the cause results from: the large number of indoor air pollutants potentially present in a building; the additive or synergistic effect of these pollutants; variation in temperature and relative humidity, noise and lighting; work related and non-work psychosocial stresses; and the varying physical sensitivity of the occupants.⁸⁷⁹

(revised): *Sick Building Syndrome* (last modified Apr. 2, 1997) <<http://www.epa.gov/iaq/pubs/sbs.html>> [hereinafter *SBS*]. Sick building syndrome may affect as many as 30–40% of a building's occupants. See also Guiffrida, *supra* note 3, at 315 n.12; Geisler, *supra* note 334, at 514–15. The WHO requires that more than 20% of a building's occupants complain of the symptoms before it will classify the problem as sick-building syndrome. See Geisler, *supra* note 334, at 515.

⁸⁷⁴ The term "sick building syndrome" originated with WHO in 1982. See Geisler, *supra* note 334, at 514. "[S]ick-building syndrome is characterized by an increased prevalence of certain nonspecific symptoms in more than 20 percent of the work force." See Marbury & Woods, *supra* note 625, at 308.

⁸⁷⁵ See *EPA Inside Story*, *supra* note 2.

⁸⁷⁶ See *Med Access, Indoor Air Pollution: An Introduction for Health Professionals: Sick Building Syndrome* <http://www.medaccess.com/ind_air/prof_08.html>.

⁸⁷⁷ See REITZE, *supra* note 33, at 409 (citing EPA REPORT, *supra* note 13, at 3–10); see also 59 Fed. Reg. 15,970 (1994) (WHO characterizes SBS by eight noninclusive symptoms). Usually, if no cause is associated with these complaints, they are thrown into the SBS category. See Geisler, *supra* note 334, at 515 (citation omitted). Between ten and twenty-five million occupants in 800,000 to 1.2 million commercial buildings in the U.S. are estimated to exhibit these symptoms. Heady, *supra* note 870, at 1054 (citing Richard Menzies et al., *The Effect of Varying Levels of Outdoor-Air Supply on the Symptoms of Sick Building Syndrome*, 328 NEW ENG. J. MED. 821 (1993)).

⁸⁷⁸ Note that cancer-causing agents, such as asbestos and formaldehyde, are not considered causes of sick building syndrome. See Marbury & Woods, *supra* note 625, at 308; see also *SBS*, *supra* note 873. A specific cause is found in only twenty-five percent of sick buildings investigated. See generally Heady, *supra* note 870, at 1041. Once a cause is determined, an SBS may be later described as a BRI. See Geisler, *supra* note 334, at 517.

⁸⁷⁹ See REITZE *supra* note 33, at 409 (citing EPA REPORT, *supra* note 13, at 3–10). "Pollutant mixtures may play an important role in causing acute symptoms associated with the 'acute

Although medical science has yet to determine some of the specific causes of sick building syndrome symptoms, researchers believe "that sick-building syndrome is multifactorial in nature, and the physical, psychological, and biologic factors must all be considered."⁸⁸⁰ Three primary causes are: (1) indoor air pollutant sources;⁸⁸¹ (2) poorly designed, maintained or operated ventilation systems;⁸⁸² and (3) unanticipated or poorly planned uses of the building.⁸⁸³ EPA has set forth a number of causes of sick building syndrome, including poor lighting, noise, vibration, thermal discomfort, and psychological stress.⁸⁸⁴ Remedying a sick building problem can be costly.⁸⁸⁵ Recognition of sick building syndrome and regulation of indoor air quality likely will result in increased toxic tort litigation.⁸⁸⁶

3. Multiple Chemical Sensitivity (MCS)⁸⁸⁷

Some people have developed an acute sensitivity to chemicals in the environment.; affected persons suffer reactions upon exposure to en-

building syndrome." *Id.* at 3-8. Note also that building-related illness and sick building syndrome problems may occur at the same time in a single building. See Marbury & Woods, *supra* note 625, at 308.

⁸⁸⁰ See Marbury & Woods, *supra* note 625, at 319; see also SBS, *supra* note 873.

⁸⁸¹ Commonly found pollutant sources in offices include tobacco smoke, asbestos, formaldehyde, organics, biological contaminants, and pesticides. See *EPA Inside Story*, *supra* note 2.

⁸⁸² Inadequate ventilation can result from poorly planned energy efficiency controls, blocked vents or return flows and improperly located air intakes. Ventilation systems can spread biological contaminants growing in cooling towers, humidifiers, dehumidifiers, air conditioners or ductwork. See *id.* When the capabilities of HVAC systems are taxed, pollutant levels rise and contribute to SBS. See Marbury & Woods, *supra* note 625, at 317.

⁸⁸³ Specialized uses of portions of the building, such as dry cleaning stores and print shops, can lead to introduction of pollutants. See *EPA Inside Story*, *supra* note 2. Improperly renovated portions of a building to be used (not originally) as office space often have inadequate ventilation. See *id.*

⁸⁸⁴ See *id.* at 27 of 41.

⁸⁸⁵ See Heady, *supra* note 870, at 1046-51 for examples of cases where remedial work was done.

⁸⁸⁶ See *id.* at 1042.

⁸⁸⁷ Although there is no widely-accepted clinical definition of MCS, one possible definition includes the following:

1. The disorder is acquired in relation to some documentable environmental exposure(s), insult(s), or illness(es);
2. symptoms include more than one organ system;
3. symptoms recur and abate in response to predictable stimuli;
4. symptoms are elicited by exposures to chemicals of diverse structural classes and toxicologic modes of action;
5. symptoms are elicited by exposures that are demonstrable (albeit at low levels);
6. exposures that elicit symptoms must be significantly below exposures known to cause adverse human response;
7. no single widely available test of organ system function can explain symptoms. See Harri-

vironmental chemical levels that normally would not affect the average individual.⁸⁸⁸ There are basically two phases in multiple chemical sensitivity (MCS) cases: an initial sensitization stage and a triggering stage.⁸⁸⁹ Substances that cause initial sensitization include pesticides, solvents, combustion products, and indoor air pollutants.⁸⁹⁰ Following this sensitization, affected persons suffer symptoms unrelated to the initial sensitizing event from exposure to chemicals such as tobacco smoke, gasoline, traffic exhaust, cleaning agents, after-shave lotion, hair spray, and perfumes.⁸⁹¹ Symptoms of persons with multiple chemical sensitivity include: fatigue, dizziness, headaches, respiratory problems, chest pain, muscle aches, memory loss, depression, irritability, nausea, and gastrointestinal problems.⁸⁹² Scientists have disagreed over the existence and etiology of this disorder,⁸⁹³ however, MCS is starting to be recognized as a clinical illness. MCS is not considered a curable disease.⁸⁹⁴ Alleged treatments range from combinations of drugs to diets to saunas. A common treatment for MCS is avoidance of pollutants, although this sometimes isolates a person from society. No MCS treatment has been validated in clinical trials.⁸⁹⁵

Although the medical community has neither defined nor created a test for MCS,⁸⁹⁶ state and federal agencies are beginning to regard MCS as an illness.⁸⁹⁷ There have been several large research projects undertaken involving MCS. For example, in 1993, Congress directed the Agency for Toxic Substances and Disease Registry to use some of its budget for research on low-level chemical sensitivities.⁸⁹⁸ An-

son, *supra* note 13, at 310 n.164 (citing EPA REPORT, *supra* note 13, at 3-11). MCS is known by several other names, including Chemical AIDS, Environmental Disease, and 20th Century Disease.

⁸⁸⁸ See *id.*; see also Frank L. Mitchell, *Multiple Chemical Sensitivities: Where Are We?*, 5 Mealey's Litig. Rep. (page unavail.) (May 17, 1996). MCS can be described as a breakdown of the immune or nervous system due to an overload of offending agents. See Michael Fumento, *People with "Multiple Chemical Sensitivity" Are Definitely Suffering. The Question Is, Why?*, REASON, June 1996, at 20.

⁸⁸⁹ See Kelly Corbett, Comment, *Multiple Chemical Sensitivity Syndrome: Occupational Disease or Work-Related Accident?*, 24 B.C. ENVTL. AFF. L. REV. 395, 401 (1997).

⁸⁹⁰ Symptoms develop after exposure to high levels of environmental chemicals, or after continuous and repeated exposure to lower levels of chemicals. See *id.*

⁸⁹¹ See *id.*

⁸⁹² See *id.*

⁸⁹³ For examples of views of medical authorities disbelieving MCS see Fumento, *supra* note 888, at 20.

⁸⁹⁴ Most physicians consider a 50 percent treatment success rate as excellent. See Mitchell, *supra* note 888.

⁸⁹⁵ See Fumento, *supra* note 888, at 20.

⁸⁹⁶ See Corbett, *supra* note 889, at 401.

⁸⁹⁷ See Mitchell, *supra* note 888.

⁸⁹⁸ See Corbett, *supra* note 889, at 400.

other federal study relating to MCS is taking place at the EPA laboratories in North Carolina.⁸⁹⁹ In Washington State, a 1994 law established a \$1.4 million MCS research fund.⁹⁰⁰ In addition, the Americans with Disabilities Act (ADA)⁹⁰¹ may require that the special needs of MCS sufferers be accommodated by employers under the ADA.⁹⁰² In 1991, regulators of the ADA ruled that MCS can be considered a disability on a case-by-case basis.⁹⁰³ Employers are concerned that these MCS claims for reasonable accommodation will be burdensome.⁹⁰⁴ However, in the MCS case brought under the ADA, the claimant did not receive an award.⁹⁰⁵ In 1997, a federal court in Maryland allowed a plaintiff to proceed with her action alleging that carbonless copy paper caused her MCS.⁹⁰⁶

The United States Department of Housing and Urban Development recognizes MCS as a disability.⁹⁰⁷ The Social Security Administration also recognizes MCS, on an individual basis, as a disability for which benefits can be paid.⁹⁰⁸ Other agencies, including EPA, the Food and Drug Administration, the Department of Education, and OSHA have issued policy statements to deal with MCS claims.⁹⁰⁹

Because of the lack of federal or state statutory protection for those suffering from MCS, most cases involve the use of common law tort remedies.⁹¹⁰ While this topic is not within the scope of this Article, one case is worth mentioning because it involves the agency that is primarily responsible for regulating air pollution.⁹¹¹ In 1990, EPA was involved in a suit by Washington, D.C., headquarters employees who

⁸⁹⁹ See Mitchell, *supra* note 888, at 793.

⁹⁰⁰ See *id.* at 771.

⁹⁰¹ 42 U.S.C. §§ 12.101–12.213 (1994).

⁹⁰² See Ed Bas, *Scope, Risk of Indoor Air Quality is Widespread Experts Say; People's Susceptibility to Problems Depends on Age, Genetics, Health, Heredity*, 198 AIR CONDITIONING, HEATING & REFRIGERATION NEWS, July 1, 1996, at 19.

⁹⁰³ See Mitchell, *supra* note 888; see generally Ronald E. Gots, *Multiple Chemical Sensitivities—Public Policy*, 33 J. OF TOXICOLOGY: CLINICAL TOXICOLOGY 111 (1995).

⁹⁰⁴ See Gots, *supra* note 903, at 804.

⁹⁰⁵ See generally Whillock v. Delta Air Lines, Inc., 926 F. Supp. 1555 (N.D. Ga. 1995); Mitchell, *supra* note 888; see also M.S. Lieberman et al., *Multiple Chemical Sensitivity: An Emerging Area of Law*, TRIAL, July 1995, at 22–32.

⁹⁰⁶ See *Helsinki v. Appleton Papers Inc.*, No. JFM-93-3784 (D. Md. 1997). Summary judgment was granted in suits by three Virginia residents with similar claims because of a difference in the Maryland and Virginia statutes of limitations. See *Court Allows Maryland Woman's Claim, Dismisses Virginia Plaintiffs on Limitations*, 11 TOXICS L. Rep. (BNA) at 1088 (Mar. 5, 1997).

⁹⁰⁷ See *Chemical Safety: State, Federal Agencies Said to Regard Multiple Chemical Sensitivity as Illness*, Daily Env't Rep. (BNA) at A-6 (Nov. 8, 1995).

⁹⁰⁸ See *id.*

⁹⁰⁹ See generally Fumento, *supra* note 888.

⁹¹⁰ See Heady, *supra* note 870, at 1063–87.

⁹¹¹ See *District of Columbia Judge Overturns Verdict for Four Plaintiffs with Chemical*

claimed EPA's leased building had given them MCS, among several other diseases and illnesses.⁹¹² Nearly 200 EPA employees were injured, and about seventy to eighty made formal complaints to EPA about feeling sick.⁹¹³ A lawsuit was brought by nineteen EPA employees against the building owners claiming injury from airborne irritants following renovations to the building.⁹¹⁴ To foster a settlement, five plaintiffs were selected for an initial trial.⁹¹⁵ In 1993, after a two-month trial, one plaintiff was awarded \$232,000 for physical injury.⁹¹⁶ The jury found the other four plaintiffs suffered somatic harm, but had no physical injuries, and awarded them damages ranging from \$119,000 to \$232,000.⁹¹⁷ The lawsuit cost approximately \$3 million.⁹¹⁸ However, the D.C. Superior Court later threw out the verdict and granted judgment notwithstanding the verdict (JNOV) in favor of the building owners on the grounds that the four plaintiffs did not have serious and verifiable injuries.⁹¹⁹ These first five plaintiffs have appealed and are still in the court system, while the other fourteen plaintiffs await trial.⁹²⁰ A small number of EPA employees chose to seek worker's compensation⁹²¹ rather than participating in a lawsuit.⁹²² Other EPA employees who were sick did not seek compensation.⁹²³ Some employees now work at home or have left EPA due to their illness.⁹²⁴ Further options explored by these employees include dis-

Sensitivity, 10 Toxics L. Rep. (BNA) at 827 (Dec. 20, 1995) (citing *Bahura v. S.E.W. Investors*, No. 90-CA-10594 (D.C. Super. Ct. Dec. 7, 1995)) [hereinafter *Judge Overturns*]. Four earlier tort suits brought by federal workers from the Department of Agriculture for injuries alleged to be caused by the glue used to install carpet at the agency were dismissed by a federal judge in Kansas in 1990. See *Cases Against Carpet, Glue Makers Dismissed; Federal Workers Failed To Establish Defects*, 5 Toxics L. Rep. (BNA) at 945 (Jan. 2, 1991).

⁹¹² See *Judge Overturns*, *supra* note 911, at 827-28. The EPA building was also the subject of an EPA-sponsored study. See Brian Leaderer, *Investigating Sick Buildings*, 19 EPA J., Oct.-Dec. 1993, at 25.

⁹¹³ Interview with Richard Cothorn, EPA employee (Apr. 25, 1997).

⁹¹⁴ There were twenty plaintiffs, until one plaintiff dropped out of the case. See *id.*

⁹¹⁵ See *Judge Overturns*, *supra* note 912, at 827.

⁹¹⁶ See Cothorn Interview, *supra* note 913.

⁹¹⁷ See *Judge Overturns*, *supra* note 912, at 827-28.

⁹¹⁸ See Cothorn Interview, *supra* note 913.

⁹¹⁹ See *id.*

⁹²⁰ See *id.*

⁹²¹ See Corbett, *supra* note 889, at 398. (further discussion and examples of MCS workers' compensation cases).

⁹²² See Cothorn Interview, *supra* note 913.

⁹²³ See *id.*

⁹²⁴ See *id.*

ability retirement or premature retirement, but these options may impose significant hardships on the employees.⁹²⁵

V. CONCLUSION

EPA has not been effective or successful in its efforts to control indoor air pollution. However, there has been neither public nor congressional support in any significant measure for an indoor air pollution program. For this reason a call for a comprehensive indoor air pollution law or program would be unrealistic. What we presently have in the way of indoor air pollution controls is as much as we are likely to have in the foreseeable future. The environmental practitioner, therefore, needs to be aware of the ad hoc approach to indoor air pollution control and the variety of legal tools that may be available to deal with such problems. Indoor air pollution issues are addressed, on a pollutant-specific basis, by using tort law, workers compensation, state environmental law, local government ordinances, occupational safety and health laws, disability compensation laws, and scattered sections of the federal environmental laws implemented by EPA.

⁹²⁵ See *id.*