

Exposure from occupational versus other sources

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FISHBEIN L. Exposure from occupational versus other sources. *Scand J Work Environ Health* 1992; 18 Suppl 1:5–16. There is an increasing need to consider the various routes of exposure to individuals in occupational settings, as well as the exposure of the general public to a broad spectrum of carcinogens. The need to consider indoor environments is increasingly recognized in light of the amount of time spent indoors by a large majority of the population and the recognition that levels of certain toxicants are higher indoors than outdoors. This overview highlights (i) a general comparison of exposure to indoor and outdoor levels of 12 volatile organic compounds and (ii) the exposure of workers and the general public to benzene, 1,3-butadiene, formaldehyde, styrene, tetrachloroethylene, phthalate esters, polychlorinated dibenzofurans, radon, and electromagnetic fields.

Key terms: benzene, 1,3-butadiene, bis-(2-ethylhexyl) phthalate, dioxins, electromagnetic fields, formaldehyde, perchlorethylene, phthalate esters, polychlorinated dibenzodioxins, polychlorinated dibenzop-dioxins, polychlorinated dibenzofurans, radon, styrene, 2,3,7,8-tetrachlorodibenzo-p-dioxin, tetrachlorobenzene.

People are potentially exposed to a broad spectrum of inorganic and organic toxicants (frequently to a multitude of chemicals as complex mixtures in the air they breathe, the food they ingest and the water they drink), in addition to being exposed to toxicants in an occupational setting. Many of the individual components of these mixtures are known to be carcinogenic in humans or animals or both. The number and extent of the carcinogens, man-made or natural, in the environment can depend on several factors, including the large increases (primarily in the last half-century) in the production and usage of synthetic organic chemicals, plastics, pesticides and drugs; the production and use of energy; the use of and exposure to natural substances such as lead; and exposure to radon and electromagnetic forces. These toxicants or exposures, whether they be of natural or synthetic origin, encompass a broad range of structural categories extending from the simple to the exotic.

There are about 50 established human carcinogens, of which 30 agents or exposures are from occupational settings. Many more have been identified through animal studies. It is important, both in a holistic sense and for a more definitive risk assessment, to consider estimates of total body burden to these agents not only for those occupationally exposed, but also for the general public as well. It is well recognized that an individual could be exposed via inhalation, ingestion, or dermal contact to many environments (ie, occupational, indoor, and outdoor) concurrently or sequentially.

Outdoor versus indoor exposures

Exposure to environmental carcinogens is generally acknowledged to vary greatly among subpopulations, from the more heavily exposed occupational populations to those living in remote areas lacking obvious sources of pollution. Exposure levels can vary by at least four orders of magnitude among both exposed workers and the general population.

There is increasing recognition that indoor air levels of toxicants may in many instances exceed outdoor air concentrations (1–7). Since most individuals spend approximately 60–90% of their time in indoor environments, this indoor air may constitute a significant source of exposure and has led to increasing concern and apprehension.

In recent years there has been a growing recognition of the presence and diversity of volatile organic compounds in the indoor environment (1–12). The United States (US) Environmental Protection Agency (EPA) has developed what it calls a “total exposure assessment methodology” (TEAM) study to develop methods for measuring a person’s total exposure to pollutants occurring in indoor and outdoor residential environments and the resulting body burdens of toxic and carcinogenic chemicals. Specific methods (Tenax[®] personal monitors) have been developed for assessing 25 chemicals in personal air, outdoor air, drinking water, and breath. To date, data have been collected over the last decade on over 2000 persons representing more than 3 million urban residents in over 10 US cities in five states (1–6). The 12 volatile organic compounds which were intensively studied included the carcinogens benzene, 1,1,1-trichloromethane, trichloroethylene, perchloroethylene, carbon tetrachloride, chloroform, meta- and paradichlorobenzene, and styrene and the noncarcinogenic aromatics ethyl benzene and meta- and para-xylenes. The sali-

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ent findings of the TEAM studies were: (i) levels of pollutants differed by season and hour of day; (ii) for most volatile organic compounds, the indoor concentrations exceeded the outdoor concentrations (about twofold); (iii) when clear indoor sources of emissions existed, the nonoccupational indoor concentrations were at times up to 100 times higher (5–7) (a finding of many other studies as well) (7); (iv) some of the main sources of indoor air emissions proved to be smoking, fuel combustion, consumer products, cooking and building materials, furnishings, solvents, printed materials, and hot showers; (v) personal monitors with adequate populations gave a more accurate estimate of human exposure than stationary monitors; and (vi) a wide variation in exposure among persons with ranges of 1000- or even 10 000-fold were found; if however the population was truncated by deleting 10% from both extremes, the range in exposure was about 10- to 20-fold. Some activities such as smoking, visiting dry cleaners or service stations, and working (ie, in chemical, paint and plastics plants) were associated with significantly elevated exposures and breath levels for some toxicants (ie, benzene, chloroform, perchloroethylene, and styrene).

Benzene

Benzene is a major industrial chemical with a very large global production, primarily in the United States, Western and Eastern Europe, Japan, and the Soviet Union. The annual world-wide production of benzene is estimated at approximately 15 million tons ($13.6 \cdot 10^6$ t, t = metric tons). Approximately 6 million tons ($5.4 \cdot 10^6$ t) of benzene was produced in the United States in 1990, this agent therefore ranking number 16 in the top 50 chemicals produced in the United States. The major occupational sources of benzene include the rubber industry, oil refineries, chemical plants, the shoe manufacturing industry, gasoline storage, shipment, and service stations. Approximately 2–3 million US workers are potentially exposed to benzene. The major environmental sources include hazardous waste sites (found in the majority of US waste sites), service stations, vehicle exhaust fumes, underground storage tanks that leak, waste water from industries

that use benzene, chemical spills, industries (ethyl benzene and styrene production), chemical manufacturing sites, and recovery plants for coke-oven by-products.

The major sources of exposure to benzene by the general public are active and passive inhalation of cigarette smoke (persons weighing 70 kg and smoking 20 cigarettes a day can absorb $7.8 \mu\text{g benzene} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$, driving and personal activities associated with vehicles, pumping gasoline, and the use of certain consumer products (ie, marking pens, paints, glues, rubber products).

In the EPA TEAM studies, the benzene levels in the homes of smokers were 30–50% higher than in the homes of nonsmokers; the median daytime exposures in the fall of 1981 were $21 \mu\text{g} \cdot \text{m}^{-3}$ for smokers and $12 \mu\text{g} \cdot \text{m}^{-3}$ for nonsmokers; the smokers generally had two to four times as much benzene in their breath as the nonsmokers. About 60% of children live in homes with smokers and can have increased exposure to benzene in their early years (3–6).

The range of benzene intake for smoking and non-smoking adults is shown in table 1 (13). The intake values in table 1 do not take into account the excretion (principally exhalation) of absorbed benzene. Nonsmokers living in rural areas have an estimated benzene intake of about $0.3 \text{ mg} \cdot \text{d}^{-1}$, while heavy smokers living in urban areas can receive as much as five times this amount. Occupational benzene can contribute additional intake to these values.

1,3-Butadiene

Butadiene is produced in large quantities as a coproduct in ethylene manufacturing (extracted from a mixed butene stream as crude butadiene). The US production in 1990 was 1.58 million tons ($1.43 \cdot 10^6$ t) ranking it 37th of the top 50 chemicals produced in the United States. This level is approximately similar to production in 1983 in Western European, where it is derived almost exclusively as a coproduct, while 556 000 tons (504 292 t) were produced in Japan in the same year (14). Butadiene is used principally in the production of synthetic polymeric elastomers such as styrene-butadiene rubber and polybutadiene rubber, and about 1600 million pounds ($725.76 \cdot 10^6$ kg) are

Table 1. Estimated daily intake of benzene from the major sources of exposure.

Diet and drink		Respiratory sources			Total intake from all sources (approximate range, μg)
Food ^a (range, μg)	Water ^a (range, μg)	Residential ambient air ^b (range, μg)	Cigarette smoking		
			Packs/day ^c	μg	
100—250	1—5	30—300	0	0	130— 550
			1	600	700—1200
			2	1200	1300—1800

^a On the assumption of complete absorption.

^b With allowance for 50% absorption and on the assumption of a daily respiratory volume of 20 m^3 .

^c Number of packs of 20 cigarettes (on the assumption of an intake of $30 \mu\text{g}$ per cigarette).

used annually in the United States, primarily in the tire industry. Other major applications of butadiene are in the production of acrylonitrile-butadiene-styrene resins, nitrile rubbers, chloroprene and neoprene rubber, and hexamethylenedimine.

The US National Institute for Occupational Safety and Health estimates that 62 000 workers are potentially exposed to butadiene in the United States, principal exposures occurring in consuming plants, terminal facilities, the decontamination and maintenance of process equipment, and the analysis of quality control samples (15). Most modern plants for the production of butadiene monomer and polymer processes are highly automated, and hence operators do not routinely spend much time in the process areas (16).

According to a 1984 EPA survey, emissions to the atmosphere from the US production of 1,3-butadiene were approximately 3.8 million pounds ($1.72 \cdot 10^6$ kg) per year or 0.14% of the amount produced, about 99% of the emissions being due to equipment leaks (17). The largest consumption of 1,3-butadiene in the United States is for the production of styrene-butadiene copolymers, which accounts for over 40% of the total amount of butadiene produced. The EPA has estimated that the total emission of butadiene to the atmosphere from this category is approximately 5 million pounds ($2.27 \cdot 10^6$ kg) a year, or 0.3% of the amount produced (16).

Low levels of butadiene (0.001–0.005 and 0.019–0.030 ppm) have been measured in urban air near industrial sites, and significantly higher levels (0.06–39 ppm) have been detected in occupational settings (14, 15, 18). 1,3-Butadiene has been detected in cigarette smoke and gasoline vapor ($2 \mu\text{g} \cdot \text{m}^{-3}$) (14). It has also been detected in food (ie, at levels of $<0.2 \mu\text{g} \cdot \text{kg}^{-1}$ in retail soft margarine and in plastic tubs which contain margarine at levels of <5 – $310 \mu\text{g} \cdot \text{kg}^{-1}$) (14).

Formaldehyde

Formaldehyde, which is a ubiquitous agent, is produced in very large amounts, principally in North America, Western and Eastern Europe, and Japan. The US production of formaldehyde in 1990 was 3.21 million tons ($2.91 \cdot 10^6$ t) ranking it number 23 in the top 50 chemicals produced. About 60% of the formaldehyde produced is used for plastics and resin manufacture, 22% for intermediates, and 18% for miscellaneous applications (ie, antimicrobials, tissue presentation, embalming). The US Occupational Safety and Health Administration has estimated that in the United States about 107 000 employees are exposed to concentrations of formaldehyde greater than 1 ppm, and about 430 000 employees are exposed to concentrations ranging from 0.5 to 1 ppm. The major sources of formaldehyde are direct emissions from production and use, and secondary sources are primarily the combustion of hydrocarbons from stationary incinerators and mobile sources (vehicle exhaust).

Formaldehyde is also formed in the atmosphere by photochemical reactions involving virtually all classes of hydrocarbon pollutants. During smog episodes (ie, in southern California) in situ production of formaldehyde may be larger than direct emissions.

The principal indoor sources of formaldehyde arise from insulating materials (urea-formaldehyde foam), chipboard, plywood, fabrics, heating and cooking, and cigarette smoke (ie, 60 – $103 \text{ mg} \cdot \text{m}^{-3}$ in mainstream smoke) (19, 20). The contribution of various atmospheric environments to average exposure to formaldehyde is illustrated in table 2 (13). The major route of exposure is via the inhalation of indoor air, releases of formaldehyde from chipboard and other building and furnishing materials comprising the bulk of the exposure. Smoking also contributes significantly to the average exposure (ie, 20 cigarettes a day contributes about $1.0 \text{ mg formaldehyde} \cdot \text{d}^{-1}$) (13). Environmental tobacco smoke contributes 10–25% of the total indoor exposure. The intake of formaldehyde from the ingestion of food (1 – $10 \text{ mg} \cdot \text{d}^{-1}$) can be ignored since formaldehyde in food is not available in free form.

Styrene

The global production of styrene is estimated at 10 million tons ($9.07 \cdot 10^6$ t) per year (21). The US production in 1990 was about 4.0 million tons ($3.63 \cdot 10^6$ t) ranking styrene number 21 in the top 50 chemicals produced. Styrene is principally employed for the production of polystyrene, styrene-butadiene resins, acrylonitrile-butadiene-styrene polymer, styrene-acrylonitrile copolymer, and styrene-butadiene rubber. Approximately 50 000 US workers are considered to be potentially exposed to styrene during its production and use. It is manufactured primarily by the dehydrogenation of purified ethylbenzene and to a much less degree as a coproduct during the manufacture of propylene oxide from ethylbenzene (22).

Table 2. Contribution of various atmospheric environments to the average exposure to formaldehyde.^a

Source	mg · d ⁻¹
Air	
Outdoor air (10% of time)	0.02
Indoor air	
Home (65% of time)	
Conventional	0.5–2.0
Prefabricated (chipboard)	1.0–10.0
Workplace (25% of time)	
Without occupational exposure ^b	0.2–0.8
With $1 \text{ mg} \cdot \text{m}^{-3}$ occupational exposure	5
Environmental tobacco smoke	0.1–1.0
Smoking (20 cigarettes per day)	1.0

^a The contribution of food and water is ignored, but is discussed in the text.

^b On the assumption of normal formaldehyde concentrations in conventional buildings.

Styrene has been detected in the atmosphere in a wide variety of locations, principally due to emission from industrial processes, particularly from the petrochemicals industry. Other sources of styrene in the environment are from vehicle exhaust and from other forms of combustion and incineration (13, 21).

The concentration of styrene in various exposure situations is illustrated in table 3 and shows that occupational exposures by far exceed any other. The concentration of styrene can be around $200 \text{ mg} \cdot \text{m}^{-3}$ (48 ppm) in the reinforced plastics industry and $10 \text{ mg} \cdot \text{m}^{-3}$ (2.4 ppm) in styrene polymerization operations; hence the exposure per workday can amount to 2 g and 100 mg, respectively (13, 21).

For the general population, indoor and outdoor air accounts for the largest exposure. Smokers inhale the highest amounts of styrene (20–48 μg for each cigarette) with a possible nominal intake of 400–960 $\mu\text{g} \cdot \text{d}^{-1}$.

Tetrachloroethylene (perchloroethylene)

The annual production of tetrachloroethylene is estimated at 100 000–250 000 tons (90 700–226 750 t) for Western Europe, 50 000–100 000 tons (45 350–90 700 t) for Eastern Europe and about 55 000 tons (49 885 t) for Japan (13, 23). The US production in 1989 amounted to 237 500 tons (215 413 t). Tetrachloroethylene is used primarily as a solvent in dry cleaning and industrial metal cleaning and degreasing. It is used to a less extent in the manufacture of chlorofluorocarbons, in processing and finishing operations in the textile industry, in grain fumigation, and as an extraction solvent and a heat-exchange fluid (13, 23).

The highest levels of tetrachloroethylene have been found near waste disposal sites and dry-cleaning establishments (ie, at levels of 20–300 $\text{mg} \cdot \text{m}^{-3}$). It has been detected in the air of industrial establishments at

levels of $2 \text{ mg} \cdot \text{m}^{-3}$ (0.3 ppm). It has often been found at higher levels (3–45 ppm) indoors than outdoors (4–7). The main sources of indoor exposures are consumer products and materials (3–6). Table 4 depicts the estimated tetrachloroethylene levels for different types of exposure (13).

Phthalate esters

During the last decade, phthalate esters have been one of the most widely produced classes of chemicals in the world, annual production being estimated at about 2 million tons ($1.8 \cdot 10^6 \text{ t}$) (24). The US production is estimated at about 700 000 tons (634 900 t). Among the phthalate esters, bis-(2-ethylhexyl) phthalate (DEHP) and dibutyl phthalate (DBP) are the most commonly employed, principally as plasticizers in polyvinyl chloride (PVC) (60% weight/weight). They are also widely used in cosmetics, lubricants, floor carpets, tapes, and consumer products such as nail polish.

Occupational exposure to phthalate esters occurs during production and during the addition to plastics (ie, PVC) during calendaring and coating operations (processing of softened plastics). DEHP is generally released to the environment through the weathering of PVC products, whereas phthalate esters are often inhaled as a mist. Occupational air levels of up to $66 \text{ mg} \cdot \text{m}^{-3}$ have been reported for DEHP, with occupational exposure which can amount to $2.0 \text{ mg} \cdot \text{d}^{-1}$ (25).

Typical ambient air concentrations ranging between 0.2 and $20 \text{ mg} \cdot \text{m}^{-3}$ have been measured recently in airborne fallout and air in 14 localities of Sweden. The total deposition of DEHP and DBP on the ground was estimated to be 200 tons (181.4 t) a year, and this level is of a similar order of magnitude as the emissions (24). The fallout rates and the levels of phthalate esters in the air were temperature dependent. In winter when the temperature was low, the atmospheric fallout of phthalate esters was high, while their levels in the air were low.

Exposure to the phthalate esters can also occur via ingestion (ie, leaching from plastic food wraps and water pipes). Levels of up to $68 \text{ mg DEHP} \cdot \text{kg}^{-1}$ in wrapped food have been reported (26).

Polychlorinated dibenzodioxins and polychlorinated dibenzofurans

Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) represent a class of almost planar tricyclic aromatic compounds that exist not as primary products but as unintentional by-products or trace impurities. There are 75 different PCDD and 175 PCDF with varying degrees and location of one to eight chlorine substituents. The various isomers exhibit large differences in toxicity and biological activity. With few exceptions these compounds occur as complex mixtures of isomers and congeners.

Table 3. Relative significance of different routes of exposure to styrene.

Exposure situation	Styrene concentration	Nominal daily intake ^a
Reinforced plastics industry	$200\,000 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	2 g
Styrene polymerization	$10\,000 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	100 mg
Within 1 km of the production unit	$30 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	600 μg
Polluted urban atmosphere	$20 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	400 μg
Urban atmosphere	$0.3 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	6 μg
Indoor air	$0.3\text{--}50 \text{ } \mu\text{g} \cdot \text{m}^{-3}$	6–1000 μg
Polluted drinking water (2 l per day)	$1 \text{ } \mu\text{g} \cdot \text{l}^{-1}$	2 μg
Cigarette smoke (20 cigarettes per day)	$20\text{--}48 \text{ } \mu\text{g} \cdot \text{cigarette}^{-1}$	400–960 μg

^a Calculated on the assumption of a daily breathing volume of 10 m^3 at work or 20 m^3 at home or in an urban atmosphere.

Table 4. Estimated tetrachloroethylene exposure levels in different types of exposure.

Type of exposure	Observed range of concentration	Total volume inhaled or amount consumed per day	Inhalation or ingestion rate (mg · d ⁻¹)
<i>General population</i>			
Inhalation			
Urban	trace to 70 µg · m ⁻³	20 m ³	trace to 1.4
Rural	trace	20 m ³	negligible
Waste disposal sites	400 µg · m ⁻³	20 m ³	≤ 8
Ingestion			
Drinking water	≤ 2 µg · l ⁻¹	2 l	≤ 0.004
Food			≤ 0.160
<i>Occupational group</i>			
Inhalation	≤ 340 mg · m ⁻³	10 m ³	≤ 3400

Of all the congeners of PCDD and PCDF, those with chloro substituents in the 2,3,7, and 8 positions are of special concern because of their toxicity, stability, and persistence. These compounds have been identified in almost all environmental compartments, as well as in human tissue (27–37).

Indeed no toxin more clearly epitomizes the public's dread of chemical contamination in the environment than 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (usually referred to simply as "dioxin"). Its emergence as a potential risk factor for Vietnam veterans who sprayed agent orange and other defoliants has largely supplanted, in some quarters, the concern from the accidental discharge of TCDD from sources such as the chemical accident near Seveso, Italy, in 1976 and several other accidents in plants in The Netherlands, The United Kingdom, Germany, and the United States.

The principal sources of PCDD and PCDF include a variety of industrial, combustion, and diffuse sources (27–41). PCDD and PCDF can arise as impurities or by-products in diverse industrial processes such as the production of chlorophenols (ie, pentachlorophenol), chlorophenoxy herbicides (ie, 2,4-dichlorophenoxyacetic acid and trichlorophenoxyacetic acid), chlorobenzenes (ie, trichlorobenzene, hexachlorobenzene, hexachlorocyclohexane, hexachlorophene), and polychlorinated biphenyls (Aroclors[®], Askarels[®], Kanachlors[®]); the synthesis of aliphatic chlorine compounds; methods involving inorganic chlorochemical processes; and processes utilizing chlorinated catalysts and solvents. Theoretically the production of all polychlorinated aromatic compounds with the use of temperatures above 150°C, alkaline conditions, ultraviolet radiation, or the presence of free halogens or radical initiators can all give rise to PCDD or PCDF formation (29).

Combustion sources such as those from stationary incineration sources including municipal solid waste, hazardous waste, sewage sludge, hospital waste, and metal reclamation plants can all cause the emission of PCDD and PCDF directly into the atmosphere. PCDD and PCDF were initially discovered in the flue gas and fly ash of municipal solid-waste incinerators in 1977

when there was 0.1–0.5 ppm of PCDD and PCDF found in fly ash. Since then PCDD and PCDF have been found world-wide irrespective of the type of incinerator employed and the waste burned (29). Isomer-specific analyses suggest that all possible isomers of PCDD and PCDF can be present in fly ash from the incineration of municipal solid waste. The isomer profiles of many different incinerators are similar (35). High-temperature industrial processes such as those employed in the copper and steel industries can result in the emission of PCDD and PCDF, particularly when copper and iron scrap contaminated with polyvinyl chloride, chlorinated paraffin, or other compounds are processed for reuse. In one study PCDD concentrations of 1–15 pg · m⁻³ were measured in the work environment, and the total emission was estimated to be 0.2–9 µg of PCDD per ton (1 ton = 0.907 t) of scrap iron (34).

Several diffuse sources reported to contribute PCDD and PCDF to the environment include automobile exhaust, private home heating, cigarette smoking, accidental fires (ie, polychlorinated biphenyl transformer fires, PVC fires, warehouse fires), and waste sites (29).

In regard to traffic exhausts, sources of PCDD and PCDF emissions from vehicles using leaded and unleaded gasoline include various chlorine-containing fuel additives, as well as motor oils, which can contain measurable amounts of PCDD and PCDF (34, 39). Usually tetra- and penta-PCDF and hepta- and octa-PCDD have been found in vehicle exhausts with isomer profiles, especially for PCDF, closely resembling those of incineration plants.

High levels of PCDD and PCDF in the range of municipal solid-waste incinerators and also 10 to 100 times higher than the corresponding values for oil burning have been detected in chimney soot from coal and wood burning (29). It should be noted that central heating produces usually 10 times less PCDD and PCDF than individual stoves and ovens.

Recent studies have shown that cigarette smoke contains about 1 pg toxic equivalents · m⁻³, which is lower than the concentrations found in indoor air (1.8 pg · m⁻³) (29).

Dry cleaning has recently been identified as a new source of PCDD and PCDF with various congeners, including 2,3,7,8-substituted congeners, detected in the residues from perchloroethylene distillation (29). The highest concentrations were found in the residues of facilities where only unused new textiles were cleaned before being sold. The cleaning process itself is believed to provide excellent conditions for the formation of PCDD and PCDF [ie, elevated temperatures, the presence of chlorinated solvents, and metals (zippers of copper, nickel, and tin and buckles) which may act as catalysts] (29).

According to several US, Canadian, and Swedish studies, PCDD and PCDF are found in all pulp-bleaching stages using chlorine, the amounts being strongly dependent on the amount of chlorine used per ton of pulp (29, 34, 36). The major PCDD are the hexa-, penta-, and tetra- congeners and a few picograms of 2,3,7,8-TCDD per gram. PCDF are mostly tetra- congeners, 2,3,7,8-tetrachlorodibenzofuran being the dominant isomer. Finished products such as coffee filters, milk cartons, tissues, and tampons have been found to contain small concentrations (a few picograms of the 2,3,7,8-substituted congeners per gram of product) (29, 34, 41).

PCDD and PCDF emitted from a variety of combustion processes can undergo long-distance migration and can enter ultimate environmental sinks such as soils, sediments, oceans, or lakes through dry and wet deposition and vapor uptake (29, 34, 35). Ambient air in urban industrialized areas contains 0.01–10 pg PCDD · m⁻³ and PCDF levels which are five to ten times higher than those found in rural areas. The isomer profiles closely resemble those found in emissions from incineration plants and traffic (34, 35, 42). PCDD and PCDF have been found to be enriched in the food chain (29, 34, 35). For example, the bottom sediment in the Swedish archipelago and the Gulf of Bothnia contains PCDD and PCDF at the parts-per-trillion level, the isomer profiles resembling those of incineration plants and traffic emissions. 2,3,7,8-Substituted PCDD and PCDF have been found in background concentrations of 0.1–100 ppt in fish and seal fat specimens (25, 34, 43). TCDD residues at levels of <25–41 ppt have been found in commercial fish (ie, Great Lakes specimens). Elevated levels of PCDD and PCDF have been found in the sediment and fish downstream from paper pulp mills that use chlorine bleaching (29, 34). The EPA accepts a value of 5000 as a bioconcentration factor for TCDD in fish. TCDD at levels of 5–60 ppt have been found in adipose tissue of beef cattle with access to range land treated with trichlorophenoxyacetic acid (29).

PCDD and PCDF have been detected in human adipose tissue in several industrialized countries (ie, the United States, Germany, Canada, and Japan), as well as in the Peoples Republic of China, Thailand, India, and Vietnam. The PCDD levels vary from 1–10 to 1000 ppt depending upon the degree of chlorination,

while PCDF levels usually range between 1 and 100 ppt (29, 34). The concentrations of PCDD in China were about one-fifth to one-tenth of those measured in Canada and Japan and were for the hexa-, hepta-, and octachlorodibenzodioxins only. In Germany, the human fat concentrations of most PCDD and PCDF isomers are 2–100 ppt, higher concentrations being found only for the hexachlorodibenzodioxins (40–200 ppt) and octadichlorodibenzodioxins (200–110 ppt). It should be noted that concentrations of PCDD and PCDF in persons occupationally exposed can be 10 to 1000 times higher (34, 43). In addition high levels of TCDD were found in the adipose tissue of production workers who produced products contaminated with TCDD (34, 44). Occupationally exposed workers have been found to have dioxin concentrations greater than 1000 ppt even 20 and 30 years after exposure. Levels of over 300 ppt have been found in some Vietnam veterans of the operation known as “Ranch Hand” about 15 years after exposure and in some persons exposed in a chemical plant accident in Seveso who had concentrations as high as 56 000 ppt immediately after the exposure (45). TCDD accumulates preferentially in the body fat of animals and humans, and its half-time in humans is estimated at 5 to 11 years (46, 47). In fatty samples taken from a random collection of young men in the United States in the 1970s, the average concentration of TCDD was between 10.9 and 12.4 ppt, while in a similar study conducted in 1983 by the Centers for Disease Control the average level of TCDD in the general population was about 4 ppt.

Several 2,3,7,8-PCDD and PCDF isomers have been found in human milk at concentrations generally much higher than those found in cow's milk. Individuals of industrialized countries have about 2 ppt of TCDD. Of a total PCDD and PCDF content of 150–450 ppt, more than 50% is octachlorodibenzodioxin. Levels of 20–50 ppt penta- and 20–50 ppt hexachlorodibenzofurans have been detected in human milk. Cow's milk contains the same PCDD and PCDF compounds but at concentrations which are one to two orders of magnitude lower (29). In most countries, breast-fed infants between 0 and 6 months of age have an average daily intake estimated at about 13 pg TCDD · kg⁻¹ body weight or 90 pg TEQ (toxic equivalent) · kg⁻¹ body weight (48).

For the general population food represents the main route of exposure to PCDD and PCDF (29, 34, 48). The total average daily intake in the industrialized countries is estimated at 20 pg TCDD · d⁻¹ · person⁻¹ or 135 pg TEQ · d⁻¹ · person⁻¹ or approximately 0.3 pg TCDD · kg⁻¹ body weight · d⁻¹ or 1.9 pg TEQ · kg⁻¹ body weight · d⁻¹ (48).

Radon

Radon-222 is a radioactive gas that exists everywhere in the atmosphere. It is a member of the radioactive chain of uranium-238 and radon-220 (often referred

to as thoron), whose members are found in all of the materials natural to the biosphere (7, 10, 13, 49, 57). The half-life of radon-222 is 3.8 d, and it decays by alpha particle emission into short-lived isotopes of polonium, lead, bismuth, and thallium (radon daughters). Figure 1 illustrates the decay chain of radon-222 to lead-210, showing the half-life of each isotope and its mode of decay (51). Although radon itself does not pose a substantial health risk, it decays as shown to a series of short-lived chemically active species (particularly ^{218}Po polonium, 3.14 min, and ^{214}Po polonium, 164 μs) that can be attached to the inhaled background aerosol or particle bound form and be deposited in the respiratory tract if inhaled. Subsequent alpha decay irradiates adjacent tissue. Hence exposure to the decay products of radon is associated with an increased risk of lung cancer (13, 49–57).

The largest share of normal background radioactivity is contributed by radon (54%) and, in decreasing order, other natural radiation (27%), medical procedures (14%), other manmade radiation (3%), and nuclear testing (2%) (52). The average concentration of radon gas in the atmosphere at ground level is about $3 \text{ Bq} \cdot \text{m}^{-3}$ with a range of 0.1 (over oceans) to $10 \text{ Bq} \cdot \text{m}^{-3}$. (A becquerel is the unit of activity of a radionuclide and is equal to 27 pCi). The radon daughter concentration in effective equilibrium with $3 \text{ Bq} \cdot \text{m}^{-3}$ of radon gas is of the order of $1\text{--}1.5 \text{ Bq} \cdot \text{m}^{-3}$ equilibrium-equivalent radon (EER) concentration (7, 10, 52).

The concentration of radon varies widely at the global level with high concentrations noted particularly in areas where terrestrial gamma radiation is high (7). As illustrated in table 5, the origins of radon vary, soil being the largest source (52), and annual radon being emission estimated at 2400 million curies ($8880 \cdot 10^{16} \text{ Bq}$). Other contributors of radon include (in decreasing order) ground water, oceans, phosphate residues, uranium mill tailings, coal residues, natural gas emissions, coal combustion, and human exhalation.

Table 6 shows the range of mean concentrations and the relative frequencies of radon daughter EER concentrations exceeding 100, 200, and $400 \text{ Bq} \cdot \text{m}^{-3}$ in dwellings in Sweden, Finland, Norway, the United States, Canada, the United Kingdom, and Germany (10). The mean radon daughter concentrations vary between different countries by as much as a factor of 5. A small fraction of dwellings in each country has concentrations 10 times higher than the national average of that country. In Europe and North America the mean radon daughter concentrations range between 10 and $50 \text{ Bq} \cdot \text{m}^{-3}$. The concentration of radon in indoor air in most buildings is likely to be a multiple of the outdoor concentration (which contributes radon via ventilation air). The main sources of naturally occurring radiation indoors are ground sites and building materials, while minor sources are generally tap water and the domestic gas supply (10, 51, 52).

Prolonged exposures to high levels of radon have

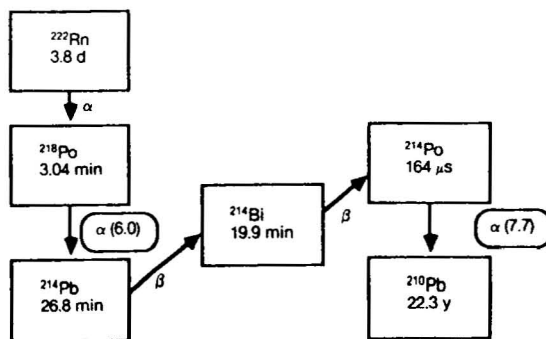


Figure 1. Decay chain of radon-222 to lead-210, showing the half-life of each isotope and its mode of decay. (Rn = radon, Po = polonium, Pb = lead, Bi = bismuth, y = year)

Table 5. Origins of radon.

	Annual radon emissions (millions of curies ^a)
Soil releases	2400
Ground water (potential)	500
Oceans	34
Phosphate residues	3
Uranium mill tailings	2
Coal residues	0.02
Natural gas emissions	0.01
Coal combustion	0.0009
Human exhalation	0.00001

^a 1 Ci = $3.7 \cdot 10^{10} \text{ Bq}$.

Table 6. Distribution of radon daughter equilibrium-equivalent radon concentrations in different countries.

Country	Arithmetic mean (Bq/m ³)	Percentage of dwellings		
		> 100 Bq/m ³	> 200 Bq/m ³	> 400 Bq/m ³
Sweden, Finland, Norway	50	10	3	1
United States	27	5	1	0.1
Canada	25	3	1	0.2
United Kingdom	10	< 0.1	0	0
Germany	25	0.7	0.1	0

been identified as a cause of lung cancer in underground miners (especially uranium miners in Czechoslovakia, the United States, Canada, and Sweden) (10, 49, 55, 58). On the basis of extrapolation from the miner data, it has been postulated that concentrations of radon found in some houses may also result in a substantial lung cancer risk (49, 59–62).

While some correlation studies have reported a positive association between lung cancer mortality and average radon concentrations, others have reported no relationships or a negative association (62). Although several case-referent studies have supported an association between residential radon and lung cancer, most of these studies were small, included actual radon measurements in only a sample of the subject's houses, and did not account for smoking and other risk factors in great detail (63–65).

Estimates of annual cancer deaths from exposure to radon and its daughters from indoor air exposures (particularly in the United States) vary widely and have been the subject of continuing controversy. Using the 1988 "Biological Effects of Ionizing Radiation (BEIR) IV" report, which analyzed data available from different cohorts of uranium miners to estimate radon risks in the home, the EPA originally estimated that the annual number of radon-induced lung cancer deaths ranged from 16 000 to 21 000 (66). The EPA more recently revised the radon health threat to 16 000 deaths per year citing a new National Academy of Sciences study which reanalyzed the miner data (67). Other estimates range from 9000 to 13 000 annual lung cancer deaths from exposure to radon in indoor air exposures. Of the estimated 16 000 deaths, only 5–10% of this mortality rate was projected to occur among nonsmokers (56).

The EPA has established an action level of radon concentration of $4 \text{ pCi} \cdot \text{l}^{-1}$ air ($0.15 \text{ Bq} \cdot \text{l}^{-1}$) for homes and buildings. If a person is exposed to this level of radon and its daughters for a lifetime (70 years), there is a 1–5% chance that that person will die of lung cancer (52). The number of homes above the EPA guideline of $4 \text{ pCi} \cdot \text{l}^{-1}$ ($0.15 \text{ Bq} \cdot \text{l}^{-1}$) may range from 2 million to 10 million (49) although it should be noted that, of the 65 million homes in the United States, only a few thousand have been tested for radon thus far (52).

Electromagnetic field exposures

Electromagnetic fields are created by electrical charges in an alternating current and are generated by anything powered by electricity. They have two components, electrical charges and magnetic waves. All light, radio-waves, and X rays are forms of electromagnetic radiation that differ only in the frequency of their waves. The exposures of concern are power frequency electric and magnetic fields, 50 Hz in Europe and New Zealand and 60 Hz in North America, classified in the extremely low-frequency range of the electromagnetic spectrum. These power sources produce electric and magnetic fields that emit radiation which is non-ionizing, unlike radiation from X rays. (68–70). Electromagnetic field frequencies are several orders of magnitude below the radiowave frequencies and about 16 orders of magnitude below the ultraviolet spectrum, the nearest energy known to cause skin cancer when humans are excessively exposed to sunlight. The electromagnetic fields that most people come in contact with are thus weak. For example, the magnetic field generated by an overhead power line or a video terminal is normally only a few milligauss, or about 1% of the earth's magnetic field. Although the electric field directly below a high-tension power line can be as much as $10 \text{ kV} \cdot \text{m}^{-1}$, the corresponding field induced inside the body is only about $1 \text{ mV} \cdot \text{m}^{-1}$, which is no

larger than the electric fields naturally generated by some cells (68).

The sources and ranges of potential exposures to magnetic and electric fields are summarized in figures 2 and 3 (70). Residential, occupational, and appliance-based electromagnetic fields are potentially important contributors to the total "dose," although most investigators agree that the relative contributions are currently unknown and may vary within a given population (70).

Over the past decade there has been increasing concern related to possible carcinogenic effects associated with occupational and nonoccupational exposures to alternating nonionizing electromagnetic fields of 50 or 60 Hz from electrical power distribution and use (68–78). Occupational exposures are incurred not only in the electric power industry, but wherever electrical equipment is employed, including the use of electric motors, video display terminals, photocopying machines, etc. Nonoccupational exposures are produced primarily in residences, wiring in the home, electric heat, electrical appliances such as electric blankets and hair dryers, as well as exposures produced from outside power lines (70).

Several recent reviews have summarized the epidemiology of electromagnetic fields and cancer (72, 75–78). Five proportional mortality or incidence studies (79–83) and two case-referent studies (83, 84) were summarized and integrated by Savitz & Calle (85). The data from these studies would indicate a tendency for electrical workers to experience an increased frequency of leukemia, enhanced for acute leukemia and especially for acute myeloid leukemia. The electrical worker category groups include radio and telegraph operators (81, 83), power station operators and aluminum reduction workers (79), power and telephone linemen (80), electrical engineers (82, 83), radio and television repairmen and electronic equipment assemblers (84), and amateur radio operators (79, 85, 86). A recent study of the incidence of leukemia in occupations with potential exposure to electromagnetic fields among US navy personnel revealed a statistically borderline excess risk of leukemia for electrician's mates (75).

In the aforementioned studies, the magnitudes of the increase of leukemia were modest (generally less than 50%), but certain subgroups such as telegraph and radio operators, power and telephone linemen, and electrical engineers exhibited twofold or greater relative risks for acute myeloid leukemia (70).

The evidence regarding brain cancer in electrical workers exposed to electromagnetic fields is less extensive although several observations of an association have been reported (70, 87, 88). Evidence of increased brain cancer risk arises from several large case-referent studies (88, 89).

It should be noted that cohort studies have not been as consistently supportive of an association between electrical work and brain cancer or leukemia (70), as

Ambient Background

Within Homes

Away from appliances

Next to appliances

Electric blankets

Distribution/ Subtransmission lines

Edge of right-of-way

Within right-of-way

High voltage transmission lines

Edge of right-of-way

Within right-of-way

Occupational environments

Office

Specialized, high exposure

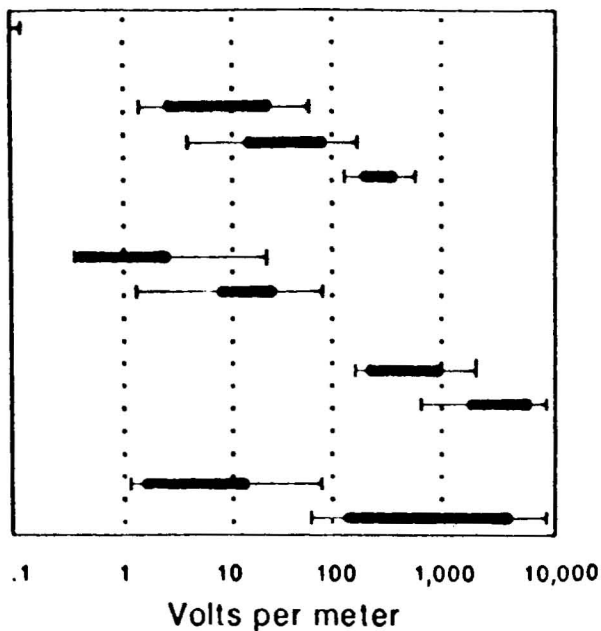
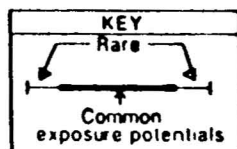


Figure 2. Electric field strengths (60 Hz) in the environment.

Ambient Background

Within Homes

Away from appliances

Next to appliances

Electric blankets

Distribution/ Subtransmission lines

Edge of right-of-way

Within right-of-way

High voltage transmission lines

Edge of right-of-way

Within right-of-way

Occupational environments

Office

Specialized, high exposure

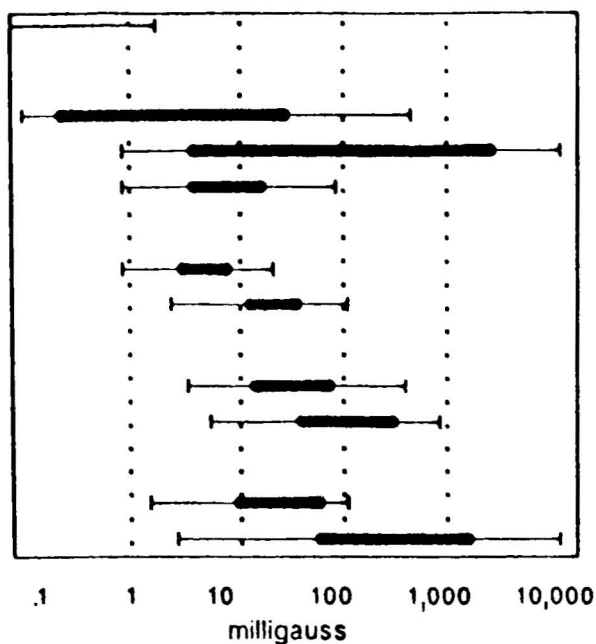
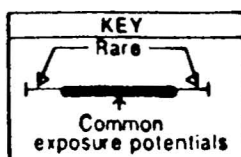


Figure 3. Magnetic field strengths (60 Hz) in the environment.

evidenced with studies of electronic workers (90), thermoelectric power plant workers (91), telecommunications workers (92), and electrical engineers (93), which showed little evidence of increased risks, although the studies were of a limited number of cases. Almost all occupational studies to date appear to suffer from two major defects, namely, a lack of exposure measurements and no evaluation of confounding leukemogenic exposures such as ionizing radiation and benzene (74).

Several recent studies have suggested an increase in cancer from residential exposures to electromagnetic fields. Wertheimer & Leeper (71, 94) initially reported that children who developed leukemia had a higher probability of having lived near high-current, high-voltage (220 kV) transmission lines and had a higher mean length of time near such lines than children free of the disease. They concluded that the children from high-exposure homes were two or three times as likely as those from low-exposure homes to develop cancer, particularly leukemia, lymphomas, and nervous system tumors. A case-referent study from Stockholm also revealed an excess of nervous system tumors and leukemias among children living near visible 200 kV wires carrying a 50-Hz current or in residences with 50-Hz magnetic fields in excess of 3 milligauss (95). However it should be noted that other epidemiologic studies have been unable to establish a strong association between residential proximity to sources of relatively intense electromagnetic fields and an incidence of cancer (96–98). In addition a case-referent study in Los Angeles found no association between the use of electric blankets and the incidence of acute or chronic myelogenous leukemia (99). Studies of adult cancer and electromagnetic field exposures in residences have not been as extensive or as consistent as the childhood cancer studies (70, 100–102).

The mechanisms through which weak electromagnetic fields might cause cancer are not currently known (69, 70). In the absence of gene mutations, damage to deoxyribonucleic acid, or the induction of sister chromatid exchanges in mammalian cells (103), as well as a lack of conclusive animal studies, it has been hypothesized that electromagnetic fields might cause cancer by some form of promotion or growth enhancement rather than through initiation (104–106). It should be mentioned that other biological effects have been noted, namely, cellular changes in hormone levels, protein synthesis, and ion flow across cell membranes (69, 70, 107).

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