POLYCHLORINATED BIPHENYLS

A Guide for Physicians

THE FACTS:

- Polychlorinated biphenyls (PCBs) were banned from use in the late 1970s; however, low levels of PCBs are ubiquitous in the environment because of their widespread use, persistence, and improper disposal.

- PCBs are highly lipophilic (fat soluble), have long environmental and biologic half-lives, and accumulate in the food chain. As a result, everyone is exposed to a few hundred nanograms of PCBs each day, primarily through the diet. PCBs can be detected in human blood sera, adipose tissue, and breast milk. Since the PCB ban, environmental levels and body burdens have been slowly declining.

- PCBs are considered practically non-toxic upon acute exposure. The primary complaints that were associated with high level, short-term exposures in the workplace were eye and skin irritation.

- Animals chronically exposed to PCBs have shown a variety of effects including dermatologic, hepatic, immunologic, reproductive, and carcinogenic. Since these tests were often carried out at doses thousands of times higher than those to which humans are exposed, their significance to humans is uncertain. Workers exposed to much higher levels of PCBs than the general public have generally not shown adverse health effects from their exposures, with the exception of chloracne and, perhaps, transient liver dysfunction. There is little evidence that exposure to PCBs poses any significant health risk at levels to which most of the population is exposed.

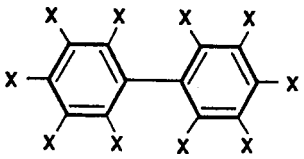
PURPOSE OF THIS GUIDANCE

This document is intended to inform health care providers of the medical issues involved with exposure to PCBs. It summarizes the potential routes of exposure, relevant clinical toxicology, and diagnosis and treatment of symptoms. More detailed information is available from the Illinois Department of Public Health (IDPH) on request. Information contained in this document is based on a review of data available at the time of preparation and is subject to change as new facts come to light. It should not serve as a substitute for careful evaluation of each patient and the exposure history on a case-by-case basis.

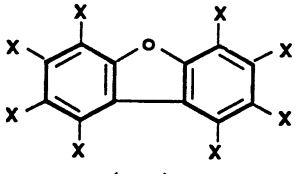
POTENTIAL PATHWAYS OF EXPOSURE

MANUFACTURE AND USE

Polychlorinated biphenyls or PCBs are synthetic halogenated aromatic chemicals, first commercially manufactured in the United States in 1929. The sole U.S. manufacturer was the Monsanto Corporation, which marketed PCBs under the trade name Arochlor. There are 209 possible congeners of PCBs among 10 different isomer classes (monochlorobiphenyl to decachlorobiphenyl). Commercial forms of Arochlor are mixtures of these congeners described by the percentage, by weight, of chlorine. PCB mixtures under names other than Arochlor are of foreign manufacture.



POLYCHLORINATED BIPHENYLS (PCBs)



POLYCHLORINATED DIBENZOFURANS (furans)

PCBs are very stable, highly resistant to extreme conditions of temperature and pressure, and have a high dielectric constant. They found wide use in a variety of industrial applications and consumer goods (Table 1). In the late 1970s, PCBs were banned by the U.S. Environmental Protection Agency (USEPA) as the result of --

- concern over two large cases of accidental poisoning in Asia (Yusho and Yu-cheng) thought to be due to PCBs (it is now believed the toxicity was the result of exposure to polychlorinated dibenzofurans [PCDFs], a pyrolysis product of PCBs),
- the discovery that PCBs could be found at relatively high levels throughout the environment, including in food and the human body, and
- indications from animal tests that chronic toxicity might pose a risk for humans.

TABLE 1 - USES OF PCBs

Insulating and Heat Exchanger Fluids	**Other**	**Additives**
Transformers	*Hydraulic fluids*	*Textiles*
Electrical capacitors	*Plasticizers*	*Inks*
Air conditioners	*Lubricants*	*Caulks*
Television sets	*Cutting oils*	*Paints*
Refrigerators	*Machine oils*	*Carbonless copy paper*
Fluorescent lights		*Flame retardants*
Water well pumps		

OCCUPATIONAL EXPOSURE

PCBs were extensively used in industry for nearly 50 years and, during much of that time, adequate occupational control measures were not used. Workers were often exposed to high concentrations of PCBs both as vapors and liquids. They may also have been exposed to transient high PCB vapors and aerosols during equipment failure. Since PCBs can be absorbed by both the skin and lungs, the occupational exposure may have been substantial and prolonged. A complicating factor is that workers also may have been exposed to other chemicals (most commonly chlorinated benzenes and chlorinated solvents such as trichloroethylene) on the job. Not only do these other chemicals carry health risks of their own, but their solvent properties may have enhanced the absorption of PCBs through the skin.

Despite the PCB ban, the workplace still has a high potential for exposure to PCBs, mainly as the result of maintenance and replacement of old equipment, PCB leaks and spills, and cleanup and disposal of PCBs. Many capacitors and transformers manufactured before the PCB ban are still present in industrial equipment (welding equipment), medical equipment (X-ray machines), and household appliances (refrigerators). The potential for PCB exposure exists in the following occupations:

- Electric cable repair
- Telephone cable repair
- Electroplating/Metal finishing
- Emergency response/Firefighting
- Waste hauling/Disposal
- Janitorial/Maintenance
- Heat exchanger repair
- Paving and Roofing
- Waste oil processing
- Transformer/Capacitors repair
- Pipe fitting/Plumbing

ACCIDENTAL EXPOSURE

The general public and workers may occasionally be exposed to high levels of PCBs and associated compounds as the result of transportation accidents or equipment failure. Other accidental exposure may be caused by seal failure in older submersible water pumps and failure of older ballasts in fluorescent light fixtures.

ENVIRONMENTAL EXPOSURE

Trace levels of PCBs are ubiquitous in the environment as the result of improper disposal, leaks, and spills over the years (Table 2). Of the more than 1 billion pounds of PCBs produced from the late 1920s to the late 1970s, approximately 50 percent is estimated to have been lost to the environment. Due to their low vapor pressure, low water solubility, and affinity for organic matrices, PCBs in the environment are most often associated with soils and sediments. Levels in these environmental media may be significantly elevated where PCB containing-equipment has leaked, where PCB oil was used for dust suppression, or where waste PCBs were disposed.

TABLE 2 - ENVIRONMENTAL OCCURRENCE OF PCBs

Soil/Sediment			
Soils	10-500 μg/kg (U.S. background)	Sediment	98-630 μg/kg (mountain lake)
	50-500,000 + mg/kg (PCB disposal sites)		1,000-13,000 μg/kg (Milwaukee harbor)
Sludge	150-3,100 μg/kg (23 U.S. cities)	Fly Ash	10-1,500 μg/kg (5 city incinerators)
Air			
Ambient	0.5-30 ng/m^3 (urban)	Indoor	229-457 ng/m^3 (Minnesota buildings)
	0.1-2 ng/m^3 (rural)		10,000 ng/m^3 + (school light ballast failure)
	0.02-0.5 ng/m^3 (remote)	Precipitation	10-250 mg/l (urban)
			1-50 ng/l (rural)
			1-30 ng/l (remote)
Water			
Potable	0.05-0.24 μg/l (tapwater - source, Hudson River)	Ground	0.06-1.27 μg/l (New Jersey industrial area)
	0.1-1.4 μg/l (found in finished waster in five of 113 U.S. cities)		0.1 μg/l (found in groundwater supplies in one of 113 U.S. cities)
Surface	0.63-3.3 ng/l (Lake Superior)		
Food			
Plant Foliage	20-30 μg/kg (15 nation survey)	Red Meat	8 μg/kg (FDA study average)
Milk	12-67 μg/kg (FDA survey)	Eggs	72 μg/kg (FDA study average)
Cheese	11 μg/kg (FDA study average)	Poultry	6 μg/kg (FDA study average)
Aquatic Life			
Fish	110-4,900 μg/kg (Lake Ontario)	Shellfish	56 μg/kg (FDA study average)
	700-14,500 μg/kg (Alberta, Canada)	Waterfowl	50-6,300 μg/kg (NY - breast meat)
	3-1,450 μg/kg (Chesapeake Bay)		280-7,850 μg/kg (Spain - breast meat)
	10,200-145,000 μg/kg (Hudson River)		29-12,290 μg/kg (Spain - breast meat)

Despite the low evaporative potential of PCBs, highly contaminated soil may result in a low-level PCB vapor release as well. Once released into the air, PCBs cycle through the environment. Exposure to significant levels of PCBs via vapor or dust are unlikely except for contaminated industrial properties (e.g., junkyards, factories, etc.) or disposal areas. PCBs are also commonly found in the food chain, primarily in animal products, since they are very

lipophilic and bioaccumulate in adipose tissue. PCBs also bioconcentrate in the food chain. Top predators can accumulate much higher levels than originally found in the environment.

Fish. The highest PCB levels in biota are most often found in fish. Subsistence fishermen or avid sport fishermen and their families may be exposed to very high dietary levels of PCBs, primarily through consumption of freshwater fish. Some studies have suggested that sport fishermen may have higher body burdens of PCBs than the general public. Saltwater fish generally do not have high PCB residues. Information on Illinois waters with contamination problems and fish preparation techniques are available through IDPH.

Breast Milk. Given the mobilization of body fat stores during pregnancy and the fat content of breast milk, lactation can serve as a route of excretion for PCBs, thereby exposing infants. Low levels of PCBs are commonly found in the breast milk of women in all walks of life. A significant correlation is observed between plasma levels of PCBs and the PCB level in the breast milk. PCB levels in breast milk drop as breast feeding continues while PCB levels in children rise, eventually surpassing the mother's body burden. The PCB concentration in the children's blood decreases at a constant rate once exposure has ended.

Gardening. Studies suggest PCBs are exceedingly immobile in soil and are not taken up into plants to any great degree. Root crops (e.g., carrots) did show some increase in PCB levels; however, the increase was very slight and the PCBs were confined to the outside layer. For other crops, the main source of contamination would be PCB-contaminated dust settling on the surface of the fruit or vegetable. Peeling, cleaning, and cooking can markedly reduce or eliminate plant residues.

Soil Exposure. In a review of communities exposed to PCBs largely through contaminated soil, 10 out of 12 populations were found to have PCB body burdens no higher than the population background, despite soil PCB levels of up to 130,000 ppm (13%). In the two populations that had slightly higher than expected body burdens, PCB exposure in the workplace and fish consumption were contributing factors. Even gardeners working directly with PCB contaminated soil and sludge have shown no increase in PCB body burdens.

Although exposure to PCB-contaminated soil does not appear to result in significant absorption of PCBs in adults, the hand-to-mouth behavior and soil or dust ingestion can be a significant route of exposure for young children. Children between 1 and 6 years of age consume approximately 50 milligrams of soil each day (children with pica behavior may eat 5 to 10 grams of soil per day). However, studies with lead have shown that outside soil contamination is only slightly associated with increased body burdens, while house dust is a major contributor to exposure. It seems likely that a similar exposure pattern exists for PCBs (tracked-in soil being the source of PCB-contaminated dust indoors). Aggressive housekeeping in areas of high PCB soil contamination can limit exposure.

Diet. For the general population, diet contributes the largest percentage of exposure to PCBs. Although undoubtedly declining since the mid 1970s, the FDA continues to detect PCBs in

samples of various commodities. The highest levels of PCBs are found in fish, followed by shellfish, eggs, milk, cheese, poultry, and red meat. The U.S. Food and Drug Administration limits the amount of PCBs in food, with tolerances ranging from 0.2 to 3 milligrams PCB per kilogram of food (mg/kg or part per million; ppm). In 1983, the general population consumed an average of 350 nanograms of PCBs each day. The levels are probably less today.

CLINICAL TOXICOLOGY

PHARMACOKINETICS

Absorption. The pharmacokinetics of PCBs tend to vary with the different mixtures and isomers and the matrix in which the PCBs are found. PCB mixtures can be readily absorbed by all routes. However, limited dermal exposure to these mixtures does not appear to appreciably increase body burdens. Skin absorption is time dependent. PCBs can be readily removed initially by simple soap and water; however, only 25 percent of the applied dose can be recovered after 24 hours. In the environment, PCBs are most commonly found as contaminants of soil or foods of animal origin, and exposure is primarily through ingestion. PCBs bound to soil or sediment are very poorly absorbed through the skin. This accounts for the lack of increased body burdens in populations exposed to high levels of PCBs in soil.

Distribution. Once absorbed into the body, PCBs have a biphasic distribution. They are initially transported by the blood stream to the liver and muscle and then subsequently redistributed to the adipose tissue. This accumulation is greatest for the more highly chlorinated PCBs (pentachlorobiphenyl and higher), which are less easily metabolized. In general, the partitioning coefficient between fat and blood is 100-200:1 depending on the overall PCB body burden. Partitioning coefficients in other organs (brain:liver:fat - 1:35:81) support the idea that the primary storage depot for PCBs is the adipose tissue. Mobilization of fat stores during rapid weight loss, disease, or pregnancy and lactation can release PCBs from fat into the bloodstream and increase the PCB levels in other tissues. While this may increase circulating PCBs in the short-term, no reports of adverse health effects have been associated with such occurrences. The placenta does not present any barrier to PCB transport to the fetus.

Metabolism. The liver is the primary source of PCB metabolism, with limited metabolism by the lungs and skin. PCBs are hydroxylated to a phenolic compound and excreted as such or as conjugates of glucuronide or sulfate. The efficacy of metabolism is correlated with the number and position of chlorine atoms. Various animal species metabolize PCBs differently (e.g., rodents and dogs metabolize PCBs faster than primates). PCBs are well-known enzyme inducers in animals, stimulating the P-450 mixed function oxidase of the liver, lung, and small intestine. This induction can result in increased metabolism not only of PCBs themselves, but also of other chemicals, medications, or endogenous biomolecules like hormones. This induction is mediated by steroid-like receptor-binding proteins found within the cell cytoplasm, and induction varies widely among various animal species.

Excretion. Excretion of PCBs is generally quite slow and, for reasons not well understood, the presence of the higher chlorinated PCBs seems to inhibit the excretion of the lesser chlorinated compounds. Body burdens tend to increase even at low levels of exposure with higher chlorinated isomers accumulated to the greatest concentration, almost indefinitely. As long as exposure continues, a true steady state is probably not achieved. Higher chlorinated PCBs are metabolized with difficulty. These metabolites are usually excreted in bile while the metabolites of lower chlorinated PCBs are eliminated through both bile and urine. It seems likely that, in the absence of exposure, excretion is also biphasic with an initial rapid excretion followed by a more gradual elimination over a period of years. The half-life of PCBs in rats ranges from 1 to 460 days depending on the degree of chlorination. The half-life of these compounds in man is not well studied, but is thought to be longer than that of animals.

ACUTE TOXICITY

PCBs are not expected to present an acute toxic hazard. The oral dose required to produce death in 50 percent of the animals tested (LD50) ranges from 1 to 10 gm/kg depending on the PCB mixture and animal species tested. For comparison, the oral LD50 for table salt is 4 gm/kg.

Acute effects reported after high exposures typical of the workplace are mild and most commonly involve complaints of eye and skin irritation. Exposures from overheated electrical equipment that released a mist containing PCBs and probably some pyrolysis by-products have resulted in complaints of nausea, eye irritation, sore throat, chest tightness, and headache. The symptoms, however, usually subside within a day. The causative agent(s) for these symptoms could not be identified due to the presence of smoke, co-solvents, and pyrolysis products (dioxins and furans) in addition to PCBs. There are no reports of acute effects from low level environmental exposures.

CHRONIC TOXICITY

PCBs are of greatest potential concern because of their persistence in the body and the possibility of subacute or chronic effects resulting from such long-term exposure. The following information (Table 3) is derived from studies of humans exposed occupationally or from high-dose animal studies.

TABLE 3 - CHRONIC EFFECTS

Dermatologic	Transient dermatitis, edema of skin and eyes; thickening of the skin and fingernails; hyperpigmentation of the skin, nails, conjunctiva and gingiva. Chloracne, characterized by straw-colored cysts and comedones, is the most consistent, though infrequent, finding in occupational exposure. Occurs typically on the periorbital and malar areas of the face; also on the trunk, extremities, genitalia and buttocks. Can be caused by many polyhalogenated aromatic compounds; lesions may be very persistent. Chloracne has not occurred with environmental exposures except in contaminated cooking oil incidents.
Hepatic	Animal studies show increases in serum liver enzyme levels and P-450 enzyme induction. Overt hepatotoxicity occurs at higher levels of exposure. Data in a small number of workers provide evidence for enzyme induction; this was not seen in another cohort with lower exposure levels. Occupational studies show a correlation between serum PCB levels and routine liver enzyme levels, but the levels are generally within the normal range. A single report of asymptomatic hepatomegaly has not been confirmed by other studies. Thus, subclinical enzyme induction and alterations of liver enzymes may occur, although clinical significance is uncertain.
Lipid Metabolism	Many reports of correlation between serum PCBs and serum lipid in exposed individuals, but associations are seemingly unrelated to dose. Also, associations are in individuals with normal serum lipid levels. This association is seen generally within the range of normal lipid values. No correlation found between adipose PCB levels and any serum lipids. Thus, observation may be the result of the affinity of PCBs for the lipid fraction of serum. Similar observations were not reported in animal studies.
Pulmonary	Capacitor workers report respiratory symptoms including upper respiratory tract irritation, wheezing, chest tightness, and cough. Spirometry and chest radiographs do not support respiratory impairment. No correlation between objective measures of pulmonary function and exposure history and/or PCB serum levels. No association between PCBs and pulmonary disease in workers or persons environmentally exposed. No pulmonary effects reported from animal tests. Irritant vapors and/or smoking may cause symptoms in workers.
Immunologic	Animals exposed to high levels of PCBs show immune effects, but results are inconsistent and include both inhibition and potentiation of immune responses. Signs of general toxicity usually present indicate immunotoxicity is a secondary effect. Workers exposed to PCBs show no effect on immunocyte levels, tests of immunocompetence were normal, and no excess morbidity or mortality from infectious diseases has been reported.
Neurologic	PCB workers report subjective symptoms including headache, nervousness, fatigue, insomnia, appetite loss, memory loss, and tingling of the skin. The symptoms do not correlate with exposure and often resolve spontaneously. No excesses in nervous system morbidity and mortality have been found. Nervous system effects are not seen in adult animals.
Reproductive/ Developmental	Lake Michigan fish consumption in pregnant women and umbilical cord blood PCB levels are reported to correlate with lower birth weight, smaller head circumference, and shorter gestational age. Cord blood PCB levels also partially correlate with a measure of visual recognition at 7 months of age and selected tests of short-term visual memory at 4 months of age. A series of investigations suggest that transplacental exposure is associated with poorer psychomotor development during the first year of life and may affect long-term intellectual function.
Carcinogenicity	PCBs do not cause mutation, but at high levels induce tumors (usually liver) in test animals. Only PCBs with a chlorine content of 60 percent (Arochlor 1260) or higher provide consistent evidence of carcinogenesis in animals. PCB mixtures containing 42 percent, 48 percent, or 54 percent chlorine did not increase tumor production significantly. The high doses used in animal tests may alter pharmacokinetics and repair mechanisms resulting in metabolites and damage not seen at lower levels. Worker studies are insufficient to adequately evaluate human carcinogenicity. Selected occupational cohort studies have found excesses of cancers at various sites, including in the hepatobiliar tract. Where excesses have been reported, dose response relationships have often not been evident. Environmental exposure to PCBs has not provided evidence of increased cancer risk. USEPA considers PCBs a probable human carcinogen. A recent study found higher PCB levels in breast tissue of women with breast cancer compared to those without; a causal relationship has not been established.
Other	No cardiovascular, hematopoietic, renal, or gastrointestinal effects have been reported or are anticipated.

In summary, a wide variety of toxicologic responses have been observed in various animal species exposed to high doses of PCBs. In humans, chloracne is the most consistent finding,

resulting from high dose occupational exposure. Other serious effects have not been shown. The apparent absence of toxic effects in humans, in spite of the animal data, is consistent with the conclusions of several recent reviews of PCB toxicology. The divergence in the findings reported for animal and human studies may result from several factors including species differences in susceptibility to PCBs or the high doses often used in animal tests, some of which are far greater than that encountered in even the highest of workplace exposures. Confidence in the apparent absence of PCB-related diseases in humans should be strengthened by the observation that this information is based primarily on studies of individuals with long-term exposure to very high levels of PCBs in industries where PCB use spanned 50 years. Further data are needed to adequately determine human carcinogenicity.

CLINICAL AND LABORATORY EVALUATION

PHYSICAL EXAMINATION

A thorough occupational and environmental history should always be taken to identify exposures that may be important to patient care. In cases of acute exposure, the primary symptoms are likely to be mucous membrane and skin irritation. Chloracne would be unlikely to occur following low exposure, its onset following higher exposure may be delayed, and a follow-up visit after several weeks may be advisable. Hepatomegaly should be considered, but must be interpreted in light of other possible risk factors for liver disease.

The physical examination should include a dermal and hepatic evaluation. In cases of acute exposure, the major reports have been of transient eye, skin, and respiratory irritation as well as other subjective symptoms. The major dermal effects of PCB exposure (e.g., chloracne) are unlikely to manifest themselves at all in cases of low exposure or immediately in the cases of high exposure. Elevated liver enzymes may be a sensitive indicator of PCB exposure although past accidental exposure has not necessarily found a measurable effect in those exposed. The absence of observable dermal and hepatic effects, therefore, cannot be used to rule out exposure and a follow-up visit is indicated some weeks after the exposure to ensure health status. Chronic exposure has not indicated overt toxicity or major effects aside from dermal involvement. Subjective neurologic symptoms reported often resolve spontaneously or after a change in work practices. Reports of hepatic involvement are not confirmed in epidemiologic studies, but signs and symptoms associated with case reports of hepatic involvement have included weight loss, anorexia, nausea, vomiting, jaundice, and abdominal pain.

LABORATORY EVALUATION

PCB body burdens depend on the route and length of exposure, place of residence, age, gender, and, possibly, alcohol consumption. Blood sera are most commonly used to determine body burdens, but PCBs can also be found in adipose tissue, breast milk, urine, feces, and semen.

TABLE 4 - PCB BODY BURDEN - UNITED STATES (1985)

	General Population		Workers	
	Average	Range	Average	Range
Serum	7 ppb	ND-20 ppb[1]	172 ppb	ND-2,530 ppb[2]
			18.2 ppb[3]	ND-424 ppb[3]
Adipose (100-250 times sera levels)	< 1 ppm	ND-3 ppm[4]	5 ppm	ND-700 ppm
Breast Milk (10-100 times sera levels)				
Whole Breast Milk	45 ppb	< 10-150 ppb	400 ppb	< 100-1500 ppb[5]
Milk Fat	1-2 ppm	0.5-2 ppm[6]	10 ppm	5-60 ppm

ND - not detected.

[1] 95 percent of U.S. population (1985) had PCB serum levels below 20 ppb. High serum lipid levels may result in artificially elevated PCB levels due to the lipophilicity of PCB.

[2] The highest PCB serum level in a worker is reported as 7,500 ppb.

[3] Measured five years after exposure ended.

[4] National Human Adipose Tissue Survey (NHATS): PCB adipose levels > 3 ppm are declining, but prevalence of detectable levels are increasing.

[5] The highest reported PCB level in whole milk is 2,160 ppb.

[6] PCBs in the milk fat of a woman residing downwind of a municipal incinerator was measured at 4.3 ppm.

Although body burdens of PCBs can be determined, analysis is expensive, time-consuming, and not generally recommended because body burdens are generally not useful in diagnosis. Analysis of adipose tissue requires removal of sufficient subcutaneous fat to allow analysis. Neither serum nor breast milk analyses are warranted unless exposure has been chronic or severe. The levels of PCBs in tissue do not correlate to any known or suspected health condition and, therefore, serve as little more than an indication of past exposure. The presence of PCBs in serum or fat does not call for any specific intervention. PCB levels generally detected in breast milk are not necessarily an indication that breast feeding should be avoided. If unusual PCB exposure is suspected and testing of PCB levels is believed necessary, such testing should be carried out as a congener-specific analysis by a certified testing laboratory using procedures developed by the U.S. Centers for Disease Control and Prevention.

Liver function tests may be a sensitive measure of PCB exposure in the absence of dermal involvement; however, the interpretation may be questionable given their nonspecificity. Other laboratory measures of organ system function (e.g., immune system) are not currently validated for diagnosis of PCB toxicity. The alleviation of patient concerns or complaints is best addressed by a thorough history and physical examination, with attention to the dermatologic and hepatic systems, and follow-up as necessary.

TREATMENT

There is no treatment indicated for either acute or chronic PCB exposure other than supportive care. If PCBs are splashed into the eyes, irrigate with warm water immediately for at least 15 minutes and follow with an ophthalmic evaluation. In other acute exposures, promptly remove clothing and decontaminate skin with soap and water, washing the area for at least 15 minutes. Be careful that emergency responders or medical staff do not contaminate themselves or their surroundings as PCB cleanup is costly and time-consuming. Use of gloves and disposable plastic will prevent cross-contamination. If PCB fluid is ingested, induce vomiting immediately unless the patient is unconscious or the fluid is known or suspected to contain co-solvents that may be aspirated into the lungs. Gastric lavage may be performed in a medical facility until gastric washings are clear. Activated charcoal has no proven benefit, but is not contraindicated. Most reported symptoms from acute exposure resolve without intervention once patients are removed from exposure. Periodic follow-ups with attention to hepatic function and dermal lesions may be considered.

Treatment of chronic exposure to PCBs is again nonspecific and based on symptoms experienced. Skin lesions may be biopsied. Chloracne is treated with measures commonly employed for acne vulgaris once exposure has ended or by referral to a dermatologist for recalcitrant cases. Since PCBs may affect the liver, avoidance of other hepatoxic agents should be recommended. Medical follow-ups should be conducted periodically as necessary.

There are no known methods of reducing body burdens of PCBs nor does their toxicity seem to warrant the attempt. So-called "detoxification" techniques have occasionally been suggested or employed in attempts to purge the body of PCB (and other persistent contaminants) lipid stores. These most often involve some combination of exercise, sauna, special diets or nutritional supplements, massage or other activities thought to increase PCB excretion from the body. There is no credible evidence that such treatments reduce the body burden of PCBs, or have any real medical benefit. Crash diets risk mobilizing PCB fat stores and at least temporarily increase the dose to other organs or possibly to nursing infants.

The toxicity of PCBs, and strategies to reduce or prevent chronic exposure, should be discussed with the patient. This may allay some fears. The uncertainty regarding carcinogenicity must be acknowledged. A simplified PCB brochure for the general public is available from IDPH.

REGULATORY GUIDELINES

Table 5 details current regulatory standards or guidelines regarding PCBs. For questions regarding the interpretation of this information or for assistance in interpreting the results and significance of environmental, occupational, or biomedical tests, contact IDPH.

TABLE 5 - REGULATORY STANDARDS OR GUIDELINES

Soil, residential	10 mg/kg	USEPA
Soil, industrial	25 mg/kg	USEPA
Interior surfaces	10 $\mu g/ft^2$	USEPA
Air, workplace	1 mg/m^3 (42% Cl)	OSHA
Air, workplace	0.5 mg/m^3 (54% Cl)	OSHA
Air, workplace	0.001 mg/m^3	NIOSH
Air, ambient	None	--
Water, drinking	0.5 $\mu g/l$	USEPA
Water, ambient	0.001 $\mu g/l$	USEPA
Food	0.2-3 mg/kg	FDA
Food	2 mg/kg (fish)	FDA
Paper/Plastic packaging	10 mg/kg	FDA

For more information concerning PCBs or for referral information, contact --

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