Persistent Bioaccumulative Toxicants (PBT)

Participant Guide

Acknowledgments

The Midwest Consortium developed this material under cooperative agreement number U45 ES06184 from the National Institute of Environmental Health Sciences. Several member institutions of the Midwest Consortium contributed to the development of this program. See http://med.uc.edu/eh/academics/training/mwc for a listing of contacts at each member institution of the Midwest Consortium for additional information about our organization and other training. We encourage you to comment on these materials. Please give your suggestions to those teaching the program in which you are now enrolled, or forward them to the Midwest Consortium for Hazardous Waste Worker Training, University of Cincinnati, P.O. Box 670056, Cincinnati, Ohio 45267-0056 or click on 'contact us' at http://med.uc.edu/eh/academics/training/mwc.

Warning

The material was prepared for use by experienced instructors in the training of persons who want information about chemicals released into their environment. Users are cautioned that the subject is constantly evolving. Therefore, the material may require additions, deletions, or modifications to incorporate the effects of that evolution occurring after the date of this material preparation. All web links are active as of April 2018; if you find an error, please inform the facilitator so that it can be updated.

Disclaimer

This training is intended raise awareness of residents and workers to chemicals from workplaces and other uses that may pose a health risk, consistent with the OSHA Hazardous Waste Operations and Emergency Response (HAZWOPER). The program covers sources of exposure in your community and methods to use to find more information and reduce exposures in your home or workplace and those of neighbors and coworkers. It does not provide the necessary hazard recognition and protective skills required to work in hazardous waste remediation or emergency response or perform emergency response activities. To undertake any of these activities, additional training is necessary. For further information about this matter, consult the training facilitator.

Table of Contents

WELCOME AND INTRODUCTION

You are here because you are concerned about the quality of your environment and how its quality affects your health and the health of your family and your community. The U.S. Environmental Protection Agency (EPA) in cooperation with international agencies has identified a group of chemical hazards that are toxic, stay in the environment for long periods of time, and accumulate in the food chain posing risks to human health and ecosystems. These chemicals are referred to as PBT chemicals. These chemicals are:

- **Persistent**— do not readily break down in the environment and therefore stay in the environment for a long time
- **Bioaccumulative**— may be stored in wildlife, humans and plants as a result of the uptake from the surrounding environment (e.g., from water or air), or as a result of one organism consuming another that is lower on the food chain (i.e. people eat contaminated fish)
- **Toxic**— can harm the environment and people

Program Objectives

After this program you will be better able to:

- Recognize PBT chemicals and access the priority PBT list
- Describe how PBT chemicals spread through the environment and into the food chain
- Determine if exposure to PBTs occurs in your community
- Select strategies to reduce exposure

Since World War II, chemical manufacturing has increased such that we now manufacture about 500 billion pounds of chemicals annually. We are exposed to as many as 80,000 chemicals in our daily lives. These compounds are known as "xenobiotics", which means "foreign compounds of no nutritional value". Classes of these are listed in Table I.

Groups of Chemicals to which humans are exposed

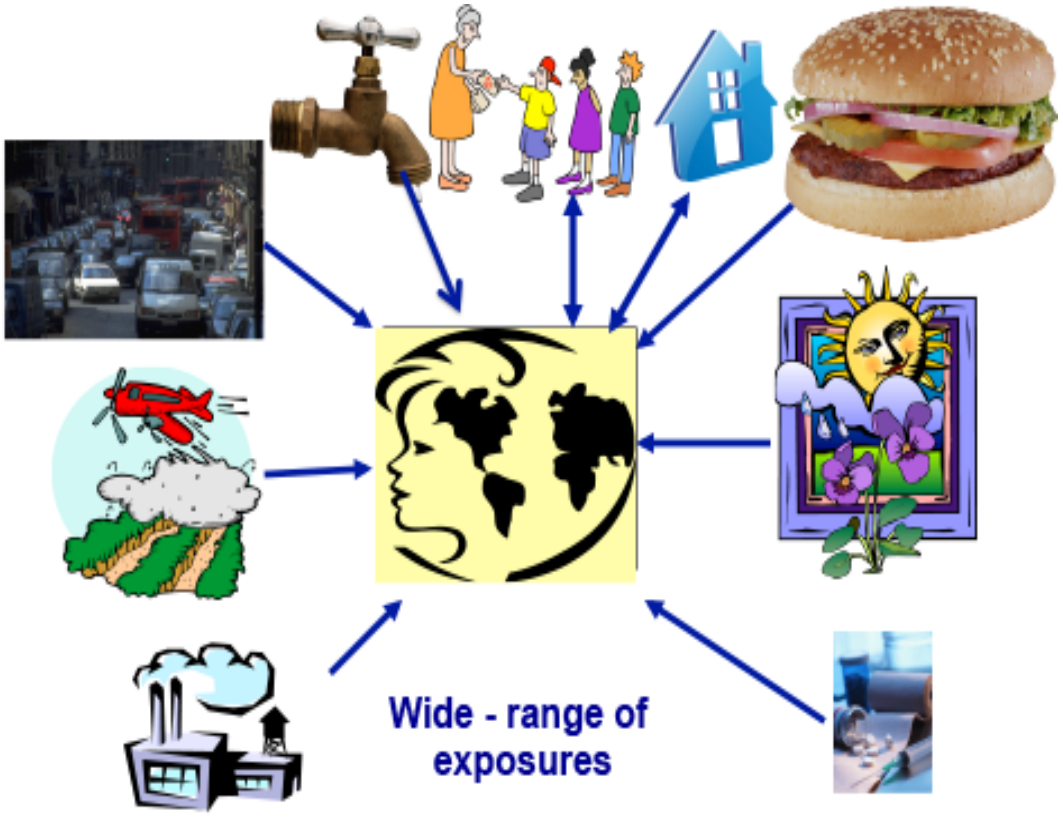
Xenobiotic	Approximate Number of Compounds
Pesticides	1,500
Active Drugs	4,000
Drug Additives	2,500
Food Additives	5,500
Other Chemicals	70,000

In addition to pesticides, drugs, and food or drug additives, this includes approximately 70,000 other chemicals, such as plasticizers, glues, solvents, oils, petroleum products and paints. These chemicals have made our lives easier in many aspects but can have a harmful effect on human health; most have not been tested for safety (https://www.nrdc.org/issues/toxic-chemicals).

Some of these chemicals are PBTs and may be stored in wildlife, humans and plants as a result of the uptake from the surrounding environment (e.g., from soil, water or air), or as a result of one organism consuming another that is lower on the food chain. PBTs work their way up the food chain by accumulating in the fat of animals. The concentration of PBT chemicals increases as they rise in the food chain. When PBT

chemicals are present in low levels at the bottom of the food chain, they may pose a significant risk to health when we eat animals higher up in the food chain. A good example is fish consumption by humans. Often these compounds are stored in fat of animals and are referred to as lipophilic (fat loving) or hydrophobic (water hating) compounds.

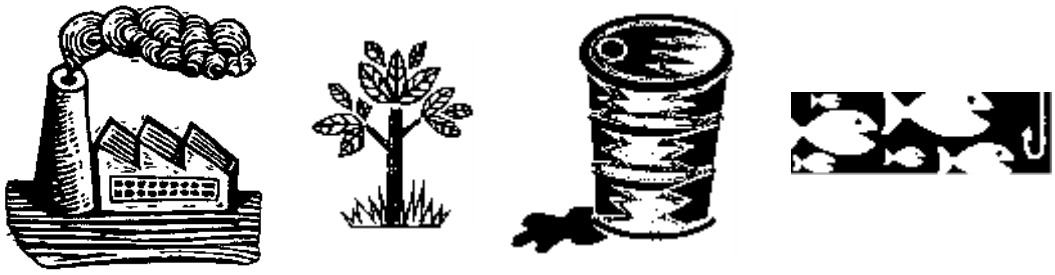
These compounds are also often referred to as Persistent Organic Pollutants (POPs). Sometimes POP and PBT are used interchangeably.



We want you to participate in the program. Please **ask questions** about anything that you do not understand and/or anything you would like to discuss in more detail.

How Chemicals Enter, Change, and Move Throughout the Environment

Pollutants move between different parts of the environment—air, water, and soil. This is called "*Fate and Transport*" of chemicals. Some PBTs have been found in surface water, ground water, sediments, fish, wildlife, human breast milk and foodstuffs (fatty foods such as butter, meat, and milk products) around the world.



Chemicals enter the air and can be deposited on soil and in waterways. On the ground livestock may ingest the chemicals, which are on the surface or chickens may eat insects that have picked up some of the chemical. Spills or releases of chemicals on land may be washed into the water supply by runoff. Fish may ingest the chemicals, and other organisms or humans may eat the fish.

Water and PBTs

Water pollution of PBTs occurs by runoff from contaminated soils, deposition from the air, releases by man-made operations into the water, and deposition onto the soil. Chemicals and microorganisms in the water can chemically change pollutants.

Many of the pollutants come from us as consumers. A good example of this is the chemical Triclosan. Triclosan is a widely used antibacterial agent that is used in consumer products. In the natural environment, Triclosan can be converted to forms of dioxin (https://www.sciencenews.org/blog/science-public/new-source-dioxins-clean-hands). In addition, Triclosan itself has toxic actions on animals and possibly in humans. (http://articles.mercola.com/sites/articles/archive/2012/08/29/triclosan-in-personal-care-products.aspx). Triclosan and 18 other sanitizers have been banned in consumer hand soaps in the US, effective September 2017 (https://www.federalregister.gov/documents/2016/09/06/2016-21337/safety-and-effectiveness-of-consumer-antiseptics-topical-antimicrobial-drug-products-for).

Minnesota was the first state in the US to take action to limit use of triclosan (http://www.webmd.com/news/20140520/minnesota-soap-triclosan). This chemical is classified as a POP and is being phased out in the European Union (http://chemicalwatch.com/18783/eu-authorities-back-triclosan-ban-in-various-products).

Air and PBTs

Air pollution by PBTs occurs by emission from manufacturing operations, combustion of fuels, spills of volatile materials, or application of pesticides. Pollutants in the air can be chemically changed by exposure to sunlight, water, and various sources of oxygen in the air.

Soil and PBTs

Pollution of the soil by PBTs occurs by improper disposal of waste chemicals, spills, deposition from the air and water, or through application of pesticides.

Example: Heavy Metals

Several heavy metals such as mercury can become bonded to soil particles. Heavy metals often accumulate in the top layer of the soil and are therefore accessible for uptake by the roots of crops.

Heavy metals often are adsorbed onto the surfaces of particulates suspended in water. The particles settle on the bottom of lakes and become buried in an anaerobic zone of sediment.

Soil contamination may still be present from agricultural uses of organic mercury compounds to kill fungi. This resulted in these compounds contacting the soil where they are broken down; the mercury becomes trapped as insoluble compounds in clay and organic material in the soil. Mercury is then leached into water systems by natural processes. Anaerobic bacteria can convert the soil-bound ionic mercury (Hg^{2+}) into methylmercury (CH_3Hg^{+}), which is soluble in water and is taken up by phytoplankton and benthic organisms in the sediment. These organisms are eaten by fish, which store methyl mercury.

Soil may be contaminated with lead-arsenic insecticides used for many years in orchards. See: https://www.opb.org/news/article/contaminated-soil-lingers-where-apples-once-grew/.

Example: Organic Pollutants

Waste PCB-containing transformers often were dumped into landfills, and their PCB content was allowed to leak into the ground.

Microorganisms in the soil actually break down pesticides such as DDT to other toxic compounds: DDD and DDE.

Living Systems and PBTs

Plants and animals that live in the water take up many toxic compounds from the water. The concentration actually increases as the compounds move up the food chain, from one-celled animals or plants to fish, to birds that eat the fish, and then to people.

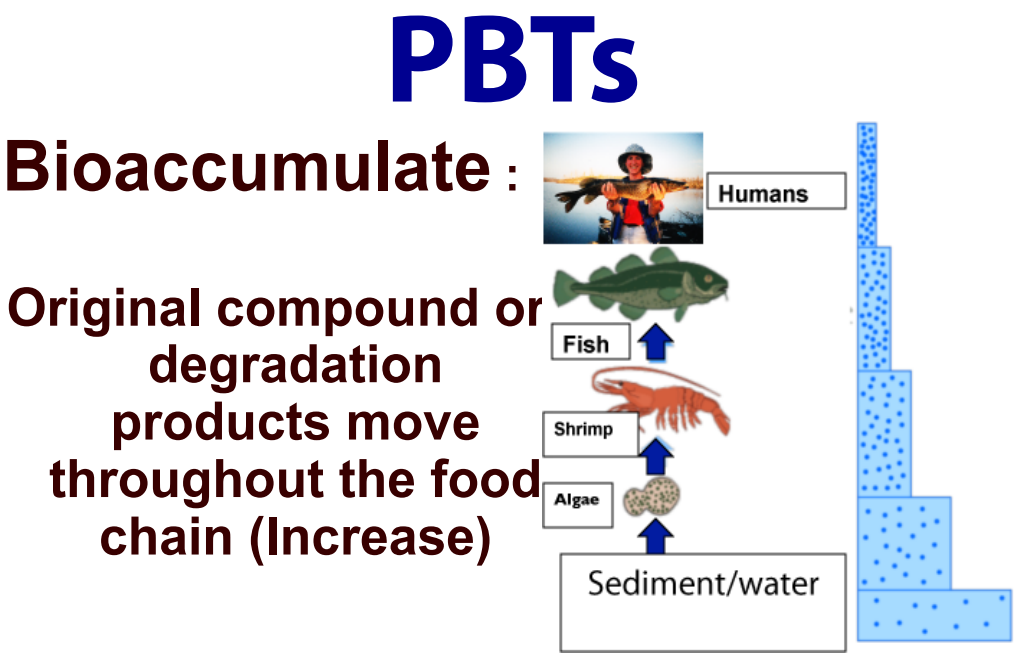
This principle is illustrated by the following levels of DDT that were found in the Long Island Sound:

0.000003 ppm[1] in the water
0.04 ppm in the plankton
0.5 ppm in the minnows
2.00 ppm in the needlefish that swim in these waters
25 ppm in the birds that feed on the fish

[1] ppm is parts per million. Examples of 1 ppm: 1 teaspoon in 1300 gallons, 1 inch in 16.7 miles

PBTs are often stored in fat of organisms, and have been found in breast milk, blood and fat of humans.

Notice in the figure below how the dots become more concentrated as you go further up the food chain.



PBTs Bioaccumulate in the food chain

PBTs—WHAT ARE THEY?

Objectives

After completion of this section, you should be able to:

- Recognize PBTs
- Access the EPA PBT website
- Identify classes of PBTs

The first goal is to learn about the PBT listing of chemicals. Below is the current EPA list of 16 chemicals.

Aldrin / Dieldrin	Mirex
Benzo(g,h,i)perylene	Methoxychlor
Chlordane	Octachlorostyrene
Heptachlor	Pendimethalin
Hexachlorobenzene	Pentachlorobenzene
Isodrin	Polychlorinated biphenyl (PCBs)
Lead	Toxaphene
Mercury	Trifluralin

EPA also lists five categories of compounds:

- Dioxin and dioxin-like compounds - These compounds include polychlorinated furans as well. There are 75 different congeners of polychlorinated dioxins, and 135 different forms of polychlorinated furans.
- Hexabromocyclododecane (HBCD)
- Lead Compounds, including Alkylated lead
- Mercury compounds – Metallic and methylated forms of mercury
- Polycyclic aromatic compounds (PACs) – Products of combustion, such as Benzo(a)pyrene and polycyclic aromatic hydrocarbons (PAHs)

Modified from: https://www.epa.gov/toxics-release-inventory-tri-program/persistent-bioaccumulative-toxic-pbt-chemicals-covered-tri

Access the EPA PBT website: https://www.epa.gov/toxics-release-inventory-tri-program/persistent-bioaccumulative-toxic-pbt-chemicals-rules-under-tri.

Some of these pollutants may be very familiar, such as lead. Various forms of lead were used as pigments for centuries, because they give stable, brilliant colors. White lead was extensively used until the middle of the twentieth century as a component of white paint and contributed to lead poisoning among young children; older buildings continue to pose a potential of lead paint exposures. Some lead is still used in exterior paints, which could result in the soil around the house becoming contaminated. Tetraethyl lead was used as a fuel additive to prevent engine knock in the United States until 1996, adding to lead in soil along roadways and where vehicles idled.

High concentrations of lead today are found near lead smelters, and was the focus of popular press reports (see Alison Young and Peter Eisler, Some neighborhoods dangerously contaminated by lead fallout, USA Today, http://usatoday30.usatoday.com/news/nation/story/2012-04-20/smelting-lead-contamination-soil-testing/54420418/1). See summary here: https://www.dhs.wisconsin.gov/lead/sources.htm

Sources and uses of selected PBT Chemicals

PBT	Source	Uses
Polychlorinated dioxins and furans	Incineration, byproduct of chlorinated compound synthesis	None
Mercury	Incineration, lightbulbs, batteries, natural	Electronics
DDT, DDE, DDP, Aldrin, Dieldrin, chlordane	Fish, plants, water waste sites	Pesticides
Polycyclic aromatic compounds: Benzo(a)pyrene, Benzo(g,h,i)perylene	Combustion	None
Hexachlorobenzene	Agriculture, fish	Pesticide
Lead compounds	Fuels, soils, batteries	Ammunition, paint, industrial uses
Mirex	Waste disposal sites, fish	Pesticide
PCBs	Waste disposal sites, transformers, fish	Heat Transfer

PBTs can be put into several broad categories:

Categories of PBTs

Group	Examples
Pesticides	Aldrin, Dieldrin, Chlordane, Hexachlorobenzene, Toxaphene, Mirex
By-Products and Contaminants	Polycyclic aromatic hydrocarbons [Benzo(a)pyrene; Benzo(g,h,i)perylene]; Octachlorostyrene; Polychlorinated dioxins and furans; Hexachlorobenzene.
Metals	Lead, mercury compounds
Dielectric Fluid	Polychlorinated biphenyls (PCBs)

Matching/Completion Exercise

Fill in the blanks below. Work in small groups, to discuss choices.

PBT	USES	SOURCES	CLASSIFICATION
Mercury	Electronics		
	Dielectric fluid for transformers		Synthetic organic compound
	Pesticide banned in 1972	Hazardous waste sites	
		Fuel additive	Organic metal compound
	None	Combustion	

- 92% of PBTs leave factories in products
- more than 151 tons of mercury was added to products
- there is no requirement to label products that contain PBTs

ARE PEOPLE IN YOUR COMMUNITY EXPOSED?

In order to be a danger to residents in your community, the PBT must be present and people have to come in contact with it by breathing it in, eating it, drinking it or getting it on their skin.

How can you find out if there are exposures in your community? In this part of the program, mapping and electronic data resources will be used to find some of the answers.

Objectives

After completion of this section, you should be able to:

- Identify possible sources of PBTs in your community
- Describe how people may be exposed

Exercise—Map Potential Sources

Go to Google Maps (https://www.google.com/maps/@44.9405554,-93.223559,13z) and download a map of your community, which will be used for the exercises in this section.

On the map, mark possible locations of these PBTs, such as:

- Hazardous Waste Sites
- Brownfields
- Power plants
- Hospitals
- Waterways
- Water treatment plants
- Large manufacturing facilities

Use EPA mapping tools, as needed to identify locations of the above types of sources. See http://www.epa.gov/myenvironment/.

Exercise—Match PBTs to Exposure Sources

Are these chemicals in your community? Think about your home, neighborhood, and community. Where could exposure to PBTs occur? Complete the table below. Then mark each on your map, using a different color for each of the four PBT groups

PBT	Exposure Sources
Example: Mercury	Hospital, Fish Dump site

Exercise—More information needed

Is the table above complete, or are there other possible sources for which you need more information?

For each group of PBTs, list the questions you need to answer to make the table of sources more complete.

Pesticides: Used in your home?

Used on crops, lawns or gardens in the community or upstream?

Metals:

By-products:

Dielectric Fluid:

Brainstorm in groups about how to find the answers.

Internet: Where would you look? What search words would you use?

Observations: For what would you look?

Ask questions: Whom would you ask?

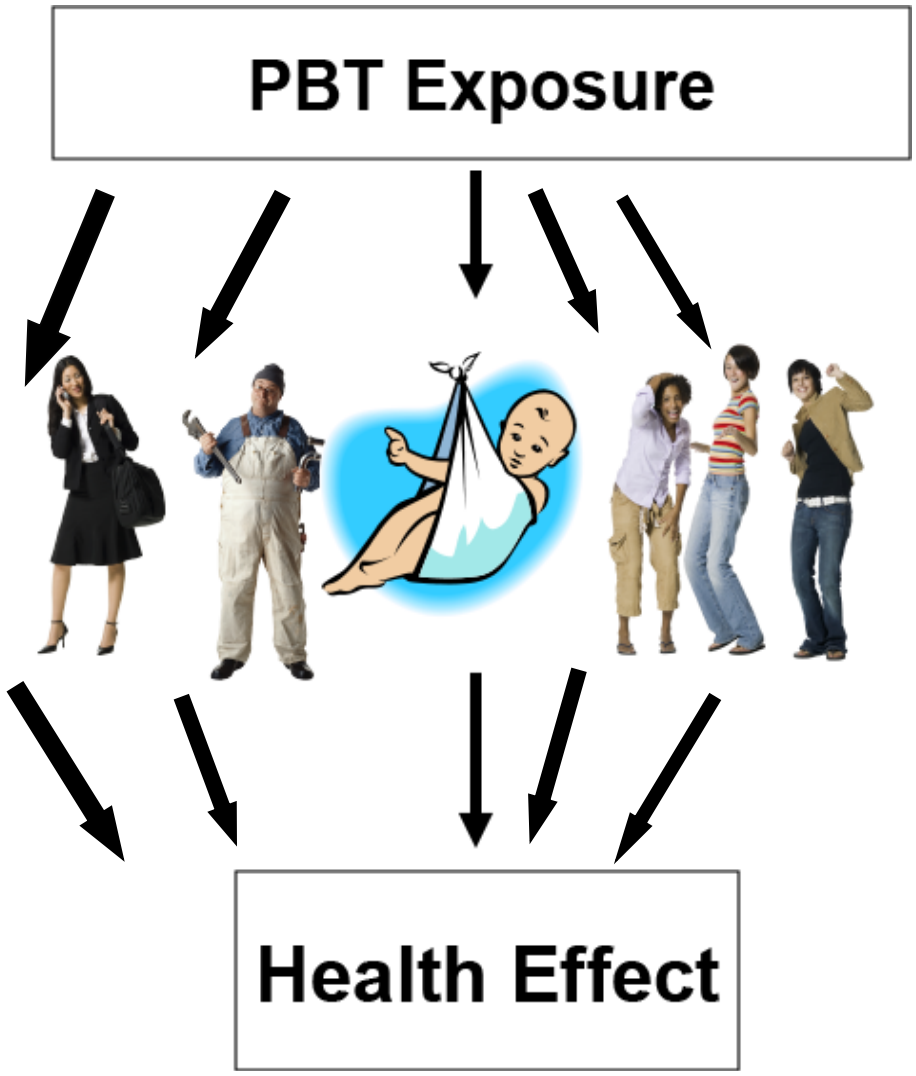
HEALTH EFFECTS

In this section you will learn about health effects that may occur due to PBT exposure. Recognizing the potential for an exposure that could result in a health effect is the first step in avoiding it.

Objectives

After completion of this section, you should be able to:

- Describe the types of health effects that may occur from exposure
- Describe how the body can react to PBTs
- Provide reasons the health of children is at greater risk from exposure compared with adults



Exposure can occur at any age, during work and leisure.

How do PBTs Enter the Body?

Chemicals can enter our bodies through skin or eye contact, ingestion, inhalation, or injection.

Skin/Eye Contact: If you come into physical contact with some types of chemicals, they may just irritate your skin or eyes; and/or they may be absorbed into your body through the skin or eyes. Chemicals more easily enter if the skin is cut or abraded.

Ingestion: If you smoke or eat after being exposed, chemicals on your hands may enter your body through ingestion. Eating contaminated food or water is also a source of exposure.

Inhalation: Chemicals can enter your body when you breathe. Liquid droplets and dusts may also be inhaled.

Injection: If you have any type of puncture wound, it is possible that whatever caused the puncture wound could be contaminated or the material in the device that punctured the skin could be toxic. Examples are needle sticks or pressure against the skin that causes puncture such as a pressurized line that breaks the skin on contact.

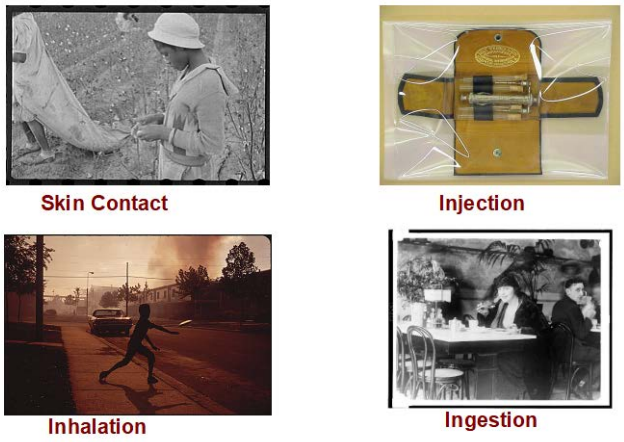


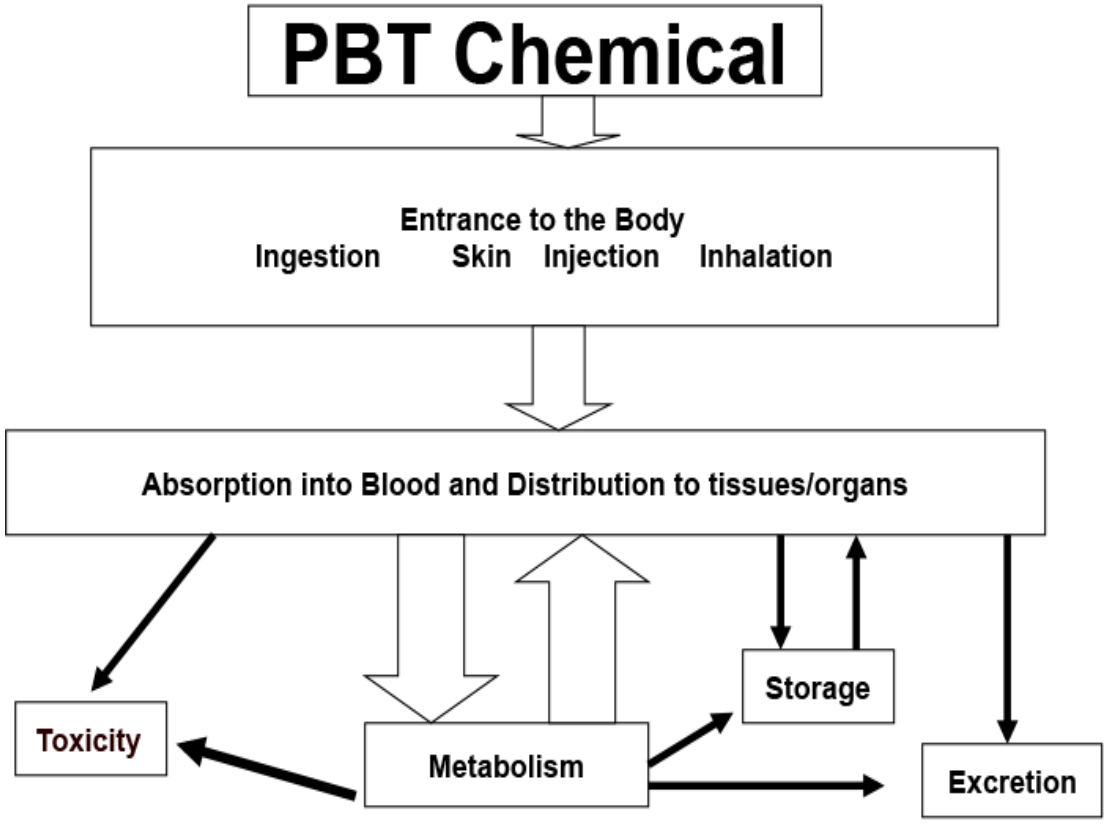
Figure 2. Means of Exposure to PBT chemicals.

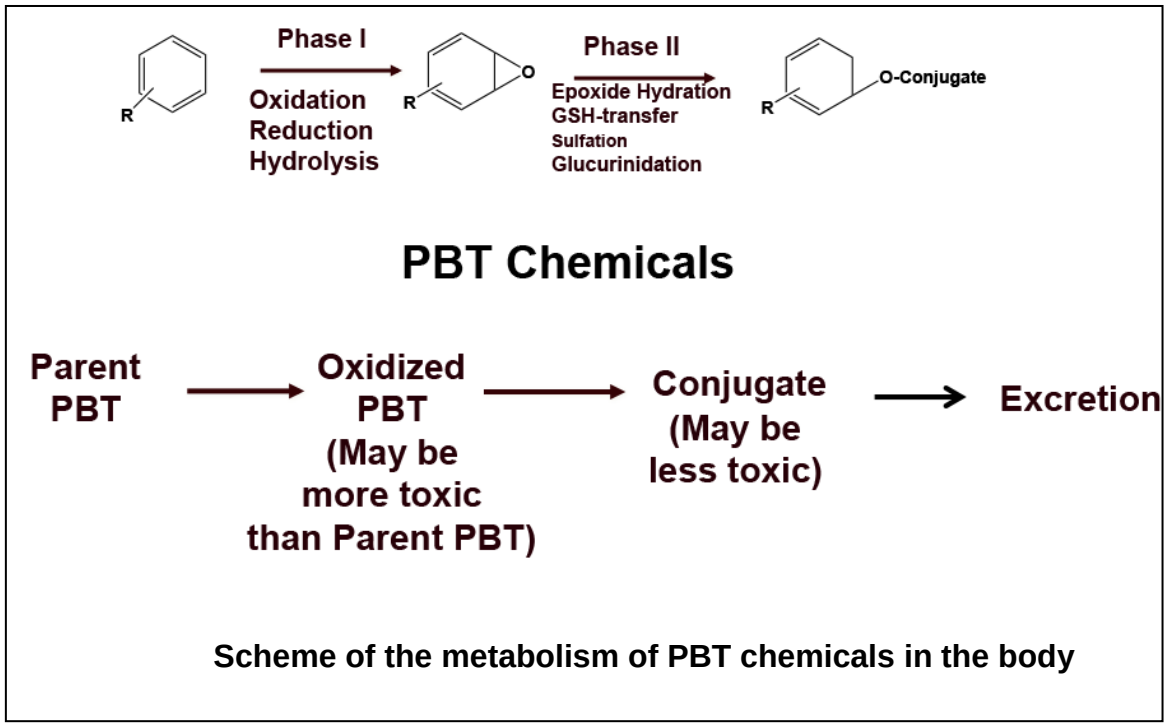
Once in the body, PBT chemicals circulate in the blood stream to various organs, where the chemical may be stored, or delivered to an organ such as the liver to be metabolized to become more toxic or less toxic or excreted.

An example of this transport and metabolism is Benzo(a)pyrene, which is metabolized to a more reactive compound called an epoxide. The epoxide can react with DNA to cause a mutation. If this mutation occurs in a so-called onco- (or cancer-causing) gene, a process is initiated that could lead to the formation of a malignant tumor months or years in the future. Sometimes, the PBT can be metabolized into a less reactive material by adding a natural metabolite to it (these include sugars, sulfates, amino acids, or a compound called glutathione) making the PBT more water- soluble so it can be eliminated by excretion.

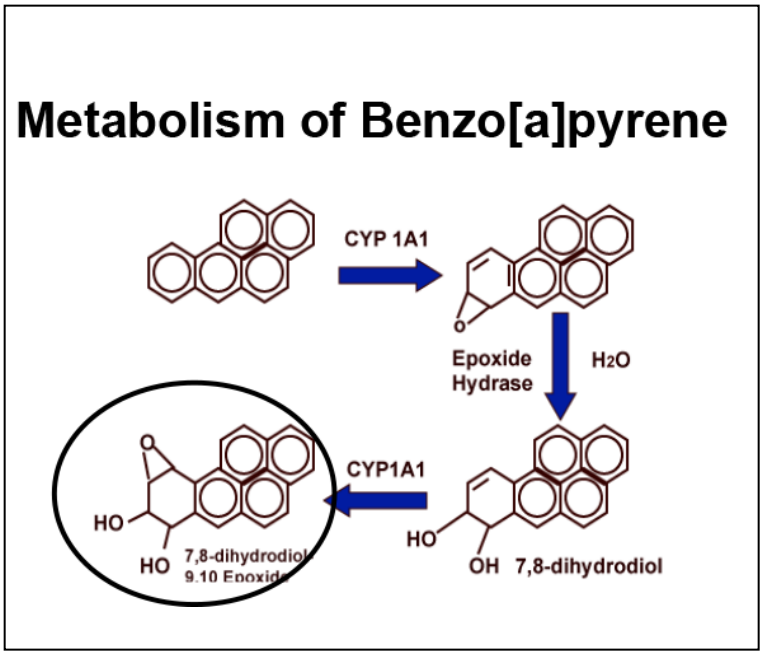
When PBT chemicals interact with macromolecules (DNA, proteins, large carbohydrates) a toxic response occurs. Sometimes the insult can be repaired, but it can lead to clinical disease.

This is illustrated on the next three figures: an overview of exposure uptake and fate; diagram of metabolism, details of Benzo(a)pyrene metabolism.

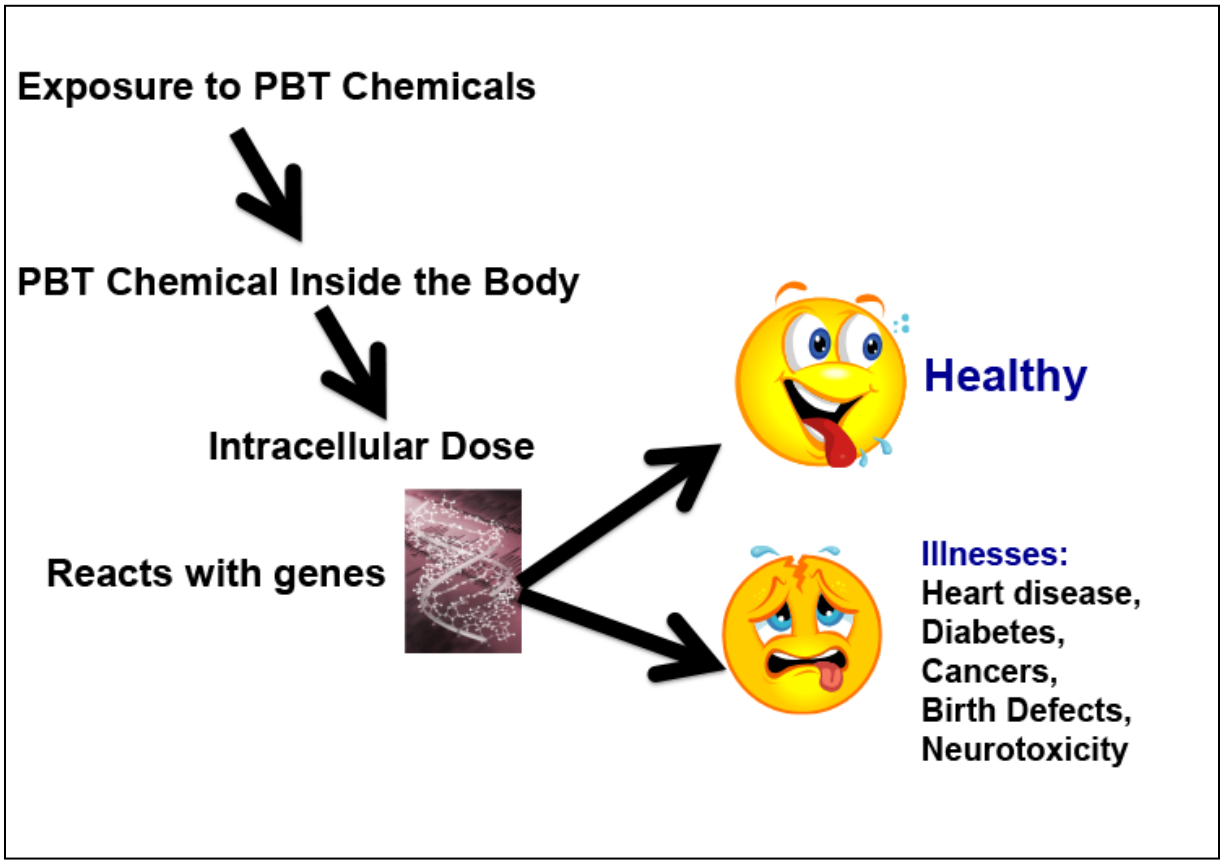




Scheme of the metabolism of PBT chemicals in the body



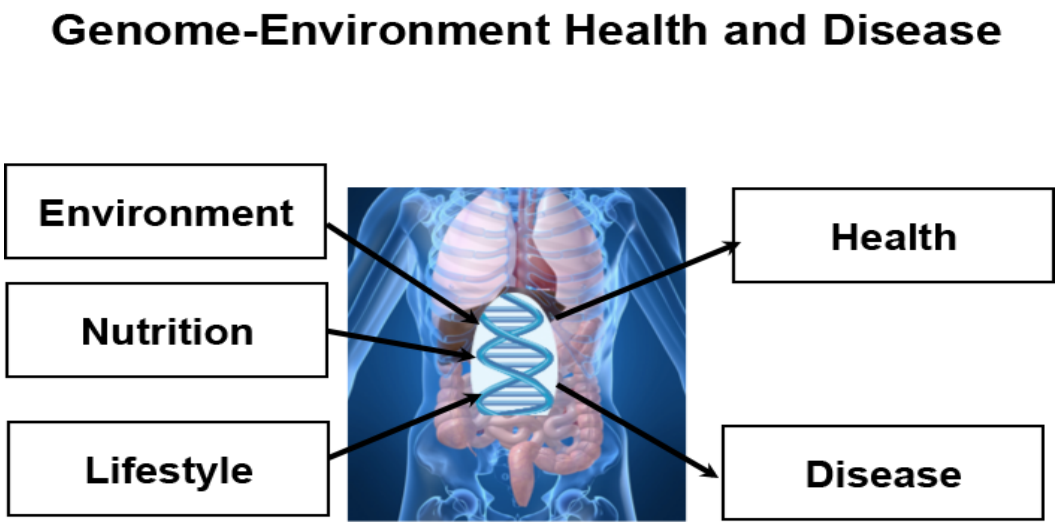
What are Some Effects?



As shown above, the concentration of the chemical is known as the intracellular dose, which may be stored in fat, or react with macromolecules, such as DNA to cause disease. Alternatively, the chemical may be metabolized to a less harmful form and excreted.

PBT exposure is responsible for many health effects. These can include diseases such as cancers of various kinds, birth defects, obesity, heart disease, diabetes, or learning deficits, such as ADHD (Attention Deficit Hyperactivity Disorder), or can program an infant in the womb to develop a disease later in life. This is called Developmental Origins of Adult Health and Disease (DOHaD).

The question is: How does this happen?



PBTs in our environment interact with our genes and sometimes can affect the way the genes are expressed. This is called epigenetics. Epigenetics literally means above the gene. That is, it is not a mutation such as the beginning stages of cancers or some birth defects, but it changes the way our genes are expressed. You may know that the DNA in each of our cells is exactly the same. Then how is it that an eye is expressed in the head and not on our arm? This is regulated by the DNA expression pattern. Environmental PBT chemicals can alter gene expression resulting in a diseased state can change this pattern.

When Will Effects Appear?

Effects of chemicals may be considered acute and/or chronic.

Acute: Develops quickly, usually after exposure to high concentrations of a hazardous substance. Example: Contact with alkyl-lead can cause headache and insomnia.

Chronic: Takes a long time to develop or requires long exposures, usually at low concentrations. Example: Exposure to Benzo[a]pyrene and other PBT products of combustion can result in a cancer after many years of exposure.

Where Will Effects Appear?

Reactions to chemical exposure may be local or systemic.

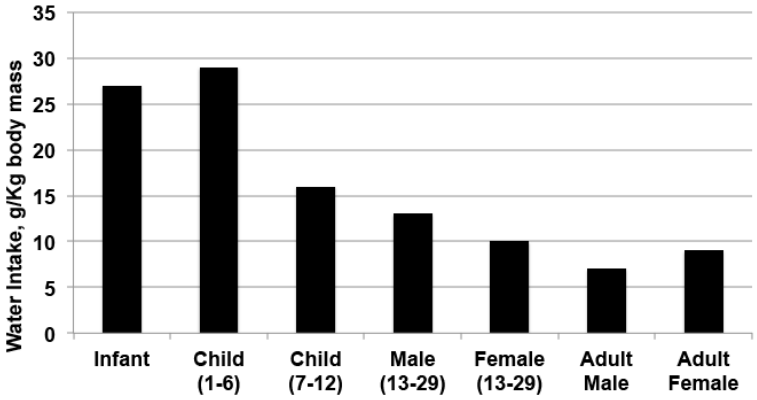
Local: The reaction develops where the substance enters the body or comes into direct contact with parts of the body. Example: Substances in tar that may contain polycyclic aromatic hydrocarbons (PAHs) can cause skin burns.

Systemic: The reaction develops at some place other than the point of contact. Ingestion or inhalation of pesticides such as DDT may cause liver damage.

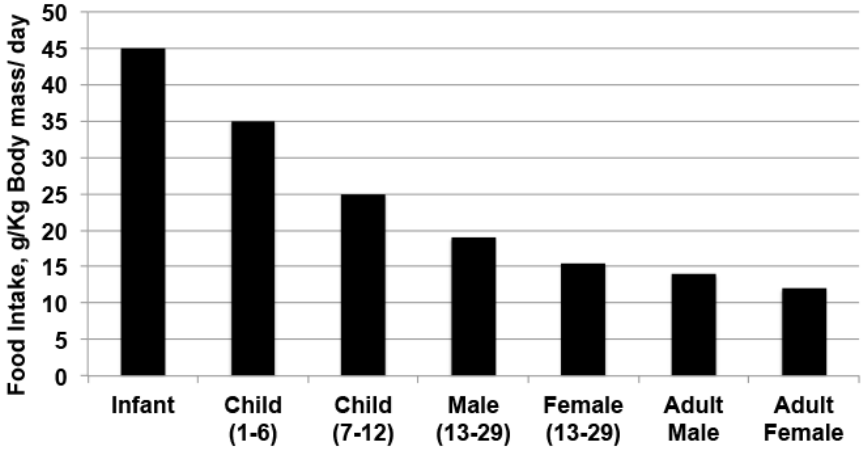
Children Are at Greater Risk

One of the major concerns about PBT exposure is the effect of these chemicals on children. Children are particularly at risk from environmental exposures because:

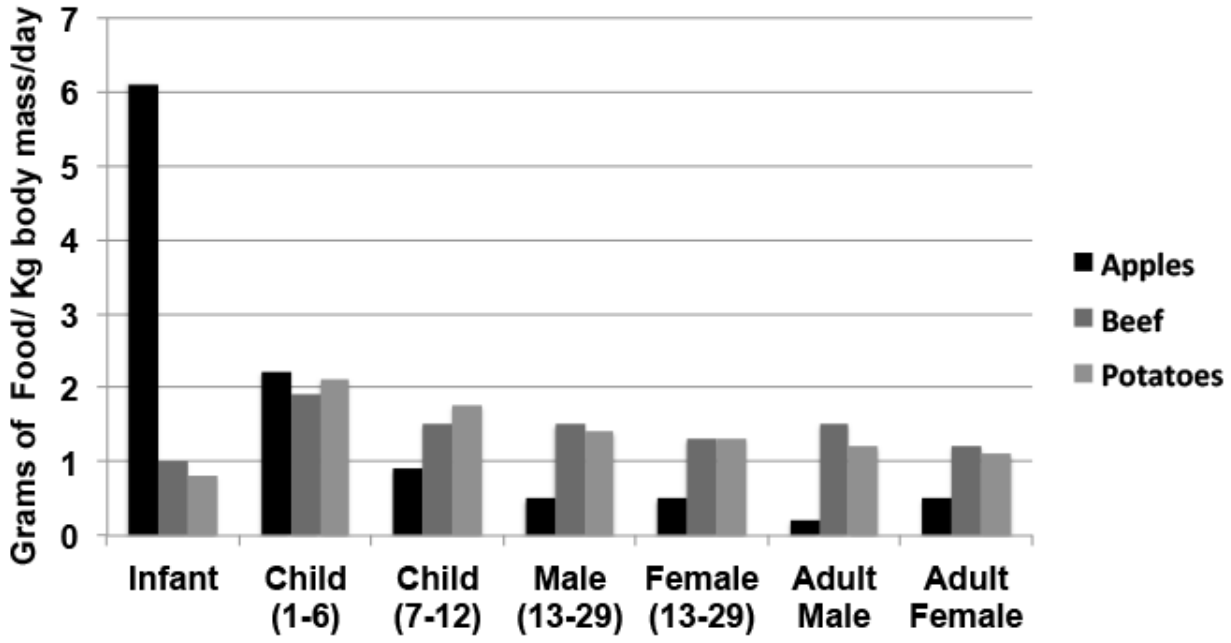
- Children's systems are still developing so they may be more affected by exposure.
- Children drink proportionately more fluids including water and eat more food and breathe more air because of the smaller body size. For example, a child has a larger lung volume-to-body mass ratio than adults, so a child playing catch outside in polluted air has a higher exposure for his/her body mass than the adult. The types of foods consumed also changes with age.
- See three figures below as illustration:



Infants and young children take in a proportionately larger quantity of water than adolescents and adults (grams of water per kilogram of body weight). Source: Health 2012.



Differences in food intake throughout the human lifespan. Infants and younger children have proportionately higher food intake than adolescents and adults. Source: Health 2012.



Differences in the pattern of food intake throughout the lifespan. Infants consume much higher levels of apples than children and older people. Apples may be contaminated with various pesticides. Sources: Health 2012; Hurley 2013.

Children have not developed avoidance behavior and have natural curiosity and a tendency to explore, which may result in exposure risks adults can more easily avoid. One example is hand-to-mouth activity among the young that can result in more exposure to potentially contaminated toys, dust and soil. Curiosity may result in exploring under the counter or in a garage, finding household chemicals, garden chemicals, and other potentially hazardous substances.

Of further concern is how exposure to PBT compounds while the child is *in utero* or early postnatally may cause diseases that appear much later in life. This is known as Developmental Origins of Adult Health and Disease (DOHaD). We now know that exposure to some chemicals during specific times of uterine development can lead to a reprogramming that predisposes the child to later disease. Examples of concern are childhood obesity and early onset of diabetes, early puberty, decreasing age of onset of hormonal tumors (breast, ovarian) and increased fibroid prevalence.

Children may be in closer proximity to some environmental exposures compared with adults. For example, the breathing zone for adults is around four to six feet above the floor, but for children the breathing zone is much closer to the floor, posing an increased risk of higher exposure to chemicals that are tend to be at lower levels, either because the lower level is the source or the chemical is heavier than air. Chemicals off gassing from carpet or flooring is one example (Bearer 1995).

Endocrine Disrupting Compounds

Some of the PBT chemicals are endocrine disrupting chemicals (EDCs) or endocrine disruptors. EDCs are chemicals that interfere with endocrine (or hormone) systems in animals and humans. The signals given *via* the endocrine system are tightly regulated. However, when we are exposed to PBTs that have endocrine actions, the body's signal system is fooled. Endocrine disruptions are new instructions that distort normal function; when exposure occurs in the womb, development can be altered. Some hormone active agents include Environmental Estrogens, such as PCBs (a PBT), PBDFs (polybrominated dibenzofuran), and Bisphenol A (BPA). Dioxins are anti-estrogens, Fenitrothion, which is an antiandrogen, and the pesticide Methoprene is an anti-vitamin A (retinoid) compound.

The PBT compounds that are endocrine disruptors are part of a diverse group of industrial, pharmaceutical consumer, and agricultural chemicals that come into contact with humans and wildlife that have the capacity to mimic or obstruct hormone functions. An interesting book on endocrine disruption is *Our Stolen Future*, by Theo Colburn, Dianne Dumanoski, and J. P. Myers (available from many booksellers, and can be down loaded as a pdf at http://www.ourstolenfuture.org/basics/bookbasics.htm).

Some example endocrine disruptors are listed in the table below:

Pesticides		Industrial Compounds
2,4-D	Alachlor	Benzene
Hexachlorobenzene	Tributyl Tin	Heavy Metals
Zyram	Methoprene	PCBs, PBDFs
Endosulfan	DDT, DDE, DDD	Dioxins
Atrazine		Phthalates, Bisphenol A

Some xenobiotics and phytoestrogens (compounds from plants that mimic estrogens) also have estrogenic activity as shown in the table below:

Xenobiotics	Phytoestrogens
Parabens	Zearalanol
Methoxychlor	Coumestrol
Benzo(a)pyrene	Isoflavones
Chlordane	Genistein
Nonylphenol	Lignans (seeds and grains)
Triclosan	Matairesinol

Some of the effects seen in wildlife include abnormally short penis length in alligators; Vitellogenin (an egg protein) in male fish; hermaphroditism (organism has both male and female reproductive organs) in frogs; lactating male bats. In humans, effects include lowered sperm counts, increase in hormonal cancers, hypospadias (abnormal male urinary canal) and gynecomastia (noncancerous increase in breast tissue) in boys.

Exercise: Health Effects Review

Use what you have learned to fill in the sentences below.

1. Exposure to substances containing _____ will likely affect the learning and memory of young children.

2. Eating imported foods from countries, which still use, the pesticide _____ could cause you to ingest this substance.

3. High exposure to PCBs can cause damage to the _____, _____ and _____.

4. Ingestion of shellfish or inhalation from spills are ways to you can be exposed to compounds of the metal _____.

5. Residents of homes that have been sprayed with _____ to kill termites are at risk for exposure to this substance.

6. _____, _____, _____ and _____ are well known for their ability to damage the nervous system.

7. _____, _____ and _____ are three substances that can be passed from the mother to the infant by breast milk.

STRATEGIES for REDUCING EXPOSURE

Objective

After completion of this section, you should be able to:

- Identify a strategy to reduce PBT exposure

The strategies for avoiding or reducing an exposure can be very specific to your community. For example, the first approach to keeping children out of an old shed where PBT pesticides were stored may be to fence it off or board it up while the community develops a budget for a more permanent solution; in another town, it might be decided to have the structure dismantled and the material put into a hazardous waste site. Individual resident and community strategies will differ.

This part of the program offers an opportunity to discuss strategies for you or your community. Because eating fish is an important source of exposure to some PBTs, the Midwest Consortium has developed a Fish Consumption Fact Sheet, shown on the next pages.

Limiting intake of fish is an example of avoiding exposure to yourself, but not reducing the amount in the environment. Other strategies include reducing the toxic release to the environment or eliminating use of the toxic material through substitution.

Fish Consumption Fact Sheet

Health Benefits

Fish are nutritious and good to eat. When properly prepared, fish provide a diet high in protein and low in saturated fats. Many doctors suggest that eating one-half pound of fish each week helps prevent heart disease. Fish eaten often provide valuable vitamins and minerals, high-quality protein, and beneficial oils that are low in saturated fat. The health benefits of fish can be gained and the consumption of unwanted contaminants can be reduced by following the guidelines in the advisories available in each state (See below).

The Issue

Because of the concern about pollution in Rivers and the Great Lakes, a fish advisory program has been in place since the 1970s. The 2010 fish consumption advisory is based on the work of public health, water, and fisheries experts from the eight Great Lake states and the Canadian province of Ontario. For up-to-date local information see: https://www.epa.gov/choose-fish-and-shellfish-wisely/fish-and-shellfish-advisories-and-safe-eating-guidelines.

Chemicals from man-made sources have entered the waterway system, including polychlorinated biphenyls, pesticides, and heavy metals. Fish take in the chemicals from the water they live in and the food they eat. The advisory is in place because some of the fish contain contaminants at levels suspected to produce adverse health effects. These contaminants can build up in the body to concentrations that may cause serious health problems.

Using the best available scientific evidence, the experts have determined how much fish is safe to eat based on the amount of chemicals that may be found in the fish. Health officials believe that the guidelines in the advisory will protect the most sensitive people, including women of childbearing age, children, and developing fetuses, from the possible health effects of consuming contaminated fish.

Avoid Exposure and Reduce Health Risks

The advisory is intended to help people make educated decisions about where to fish, what type of fish to eat, and how to limit the amount and frequency of fish consumed. By following the advisory, people can enjoy the health benefits of fish and reduce the consumption of unwanted contaminants. To avoid exposure to these chemicals and reduce the health risks of eating these fish, some general recommendations should be followed:

- Choose fish from bodies of water not listed on the advisory.
- Eat the fish listed as less likely to be contaminated.
- Choose smaller fish within a species and choose lean (lower-fat) fish.
- Choose fish that don't eat other fish.
- Properly clean and prepare fish in a way that will reduce skin, fatty material, and dark meat consumption. This includes cutting off the skin and fat before baking, broiling, grilling, roasting, boiling, or poaching the meat.
- Eat no more than one meal per week of fish and eat smaller servings of the fish.

For the most up-to-date advice concerning the fish consumption advice in your area, contact your local health department or Department of Natural Resources (DNR) office. Fish advisories in States within the Midwest Consortium (MWC) and neighboring areas where MWC residents may fish or from which fish may be readily available follow:

State/area	Website for Fish Advisory
Illinois	http://www.idph.state.il.us/envhealth/factsheets/fishadv.htm
Indiana	http://www.in.gov/isdh/23650.htm
Kentucky	http://fw.ky.gov/Fish/Pages/Fish-Consumption-Advisories.aspx
Michigan	http://www.michigan.gov/mdch/0,4612,7-132--258927--,00.html
Minnesota	http://www.health.state.mn.us/divs/eh/fish/
New York	http://www.health.state.ny.us/nysdoh/environ/fish.htm
Ohio	http://www.epa.state.oh.us/dsw/fishadvisory/index.aspx
Ontario	https://www.ontario.ca/page/eating-ontario-fish-2017-18
Penn.	http://www.dep.pa.gov/Business/Water/CleanWater/WaterQuality/FishConsumptionAdvisory/Pages/default.aspx

Tenn.	https://www.tn.gov/news/2007/4/25/updated-fish-consumption-advisories-issued-for-tennessee.html
Wisconsin	http://dnr.wi.gov/topic/Fishing/Consumption/

Exercise—Matching Strategies to Sources

Brainstorm in small groups regarding how to reduce the exposures you have identified on your map or in discussions during the program. Examples are given. Use this format to record ideas for your community.

Situation	Strategy
Household/Farm Pesticides	Local or state government
	Example: dispose of during pesticide collection days
Mercury in fish	Fish where designated
	Cook according to recommendations
Identify if water is contaminated	Contact Dept. of Natural Resources

_____

_____

_____

_____

Exercise—Act to Reduce Exposure

Use your map and what you have learned as you complete the next exercise: Green, it's not just a color! Your instructor will distribute the materials for this.

CLOSING AND EVALUATION

Key Point: PBTs are: Persistent, Bioaccumulative, Toxic

Resources are appended to this manual that may be useful to you in the future.

Thank you for participating in this program.

This is an opportunity to ask any questions you may have, or to discuss how the knowledge and skills learned can be used at work.

Finally, we ask that you take 10 minutes to complete the program evaluation forms. These are important for improving the program. The Midwest Consortium does take your comments seriously and has made changes in content and the skill exercises based on feedback. Your comments are anonymous.

We hope to see you at another Midwest Consortium program in the future.

If you completed a Plan, someone from the training center will follow up to discuss your progress on your planned actions to reduce exposure to PBTs. Thank you in advance for taking a few minutes at that time to let us know then to let us know if the program facilitated a change that you planned. Any barriers you experienced in completing your plan will also help us in future training programs.

RESOURCES

To provide some background information on PBTs (both those listed by EPA and other chemicals with similar properties) fact sheets on the following are provided:

Bisphenyl-A (BPA)
Pesticides (Introduction, DDT, Chlordane, Mirex)
Halogenated Compounds—Dioxins and Furans
Polychlorinated Biphenyls (PCBs)
Polybrominated Diphenyl Ethers (PBDEs)
Metals (Mercury and compounds, alkyl lead)
Polycyclic Aromatic Hydrocarbons (PAHs)

See other web resources shown in the Web Resources section.

A brief introduction to the Toxic Release Inventory (TRI) is provided.

References cited in this guide and in the following fact sheets are the final resource shown, Bibliography.

Bisphenol-A (BPA)

Bisphenol-A (BPA) is a chemical that was synthesized in 1905 as an estrogenic compound. BPA was designed as an estrogen replacement drug to give to women who could not otherwise synthesize estrogen. It was soon recognized however that this compound had very weak estrogenic properties. BPA was replaced on the market by a compound called diethylstilbesterol (DES). DES was a much stronger estrogen and was widely used in women to prevent miscarriages. As many women know, DES did not work as predicted and daughters of women who took the drug had many problems. It is estimated that 5 to 10 million people in the United States were exposed to DES from 1938 to 1971. Women who were exposed to DES before birth (in utero) are known as DES daughters. They are at risk for developing clear cell carcinomas of the vagina and cervix, reproductive tract structural differences, pregnancy complications, and infertility. Although DES daughters appear to be at highest risk for clear cell cancer in the teens and early 20s, these cases have been recorded in DES daughters into their 30s and 40s. DES sons are at an increased rate for non-cancerous epididymal cysts. If you were exposed to DES, consult the CDC DES webpage https://www.cdc.gov/des/index.html Based on the large body of scientific work on DES, and observation of feminization of some wildlife species, a new area of science called endocrine disruption was opened.

That brings us back to the case of BPA. Organic chemists are very brilliant people. And based on chemical properties of BPA it was soon discovered that it could polymerized to make a nice hard clear plastic substance. The plastic compound is known as polycarbonate plastic. Polycarbonate plastic are made into plastic bottles, including baby bottles, compact disks, safety equipment, and medical devices. The epoxy resins containing BPA are used as coatings in food cans bottle tops and water supply pipes. BPA can also be found in thermal paper products that include some cash register and ATM receipts. They are also used in sealants and composites in dentistry that could contribute to BPA exposure in humans. BPA is widespread in the environment, and it has been estimated that greater than 90% of Americans are exposed to BPA. The wide exposure of BPA to the American population has caused great concern for environmental health scientists. Exposure to BPA in utero, early childhood and adolescents is associated with increased obesity observed in those populations (Carwile and Michels 2011; Trasande et al. 2012). Other studies show that exposure to BPA in utero is associated with early onset of type 2 diabetes (Young and Yu 2009). An important aspect of these studies is that they show that in addition to reproductive and sexual function effects, endocrine disrupting chemicals can affect other endocrine diseases such as obesity and diabetes. It leads us to consider whether the current obesity epidemic we are observing in the population may have an important environmental component.

We can reduce exposure to BPA by using containers that are not made of polycarbonate. Whenever possible use glass, porcelain, or stainless steel (with no BPA in the liner). Don't wash polycarbonate plastic containers in the dishwasher with harsh detergents. When these containers are scratched by vigorous washing, BPA is released into the water or food stored in the container. Never microwave food in polycarbonate plastic food containers. Avoid canned food, eat fresh or frozen foods instead. For infants, use bottles that are BPA free.

Pesticides

The Impact of Pesticides

Since the advent of the *Green Revolution*, in the late 1960s, pesticides have become more widely used in agriculture. Later, chemical pesticides became commonplace in landscaping, and in our homes. There is no biological classification of “pest". We define pests as plant, animals, insects that can cause deleterious effects, especially compounds that destroy agriculture products.

Pesticides:

Chemicals to control unwanted forms of life - Pests

- **Insecticides**
  - **Insects,**
  - **spiders**
- **Herbicides**
  - **Plants**
  - **Weeds**
- **Fungicides**
  - **Fungi**
  - **Molds**
- **Repellents**
  - **Deet**
  - **Citronella**

Organo-phosphate pesticide compounds are reversibly acutely neurotoxic. Metabolites of organophosphate compounds can be more toxic than the parent compound. These metabolites are called “oxons” and are highly toxic to humans. The route of exposure is typically through contact with skin. The formulations of organophosphate pesticides are packaged with various solvents, which in their own right may be highly toxic and more toxic than the pesticide. Organophosphate pesticides can come in various formulations and a variety of solvents. Toxicity may vary, but acute symptoms include headache, nausea, vomiting, muscle weakness, twitching, diarrhea, siezures, and pulmonary edema (Strong et al. 2004).

Chlorinated hydrocarbon (organochlorine) insecticides, solvents, and fumigants are used globally. This group of compounds containing carbon, hydrogen, and chlorine comprise this group of pesticides. Organochlorine pesticides are very effective at killing insects, but unfortunately have dramatic toxic actions in humans and other animals. Many have been universally banned because of their unacceptably slow degradation and subsequent bioaccumulation and toxicity (Stockholm Convention. Available at http://chm.pops.int/default.aspx). The toxicity of these agents varies according to their

molecular size, volatility, and effects on the central nervous system (CNS). In general, they cause either CNS depression or stimulation, depending upon the agent and dose (Bhalla and Thami 2004).

After exposure to these compounds, they usually reside in fatty tissue and have long half lives in humans. Organochlorines are typically absorbed orally and by inhalation. Transdermal absorption is variable. For example, DDT is poorly absorbed across skin, but cyclodienes readily cross skin barriers (Mortonsen 1986; Reigart and Roberts 1999).

Common Chlorinated Pesticides

dichloro diphenyl trichloroethane	*chlordane*	*endrin*
dienochlor	*heptachlor*	**methoxychlor**
chlorobenzilate	*benzene hexachloride*	*chlorodecone*
mirex	**lindane**	*dieldrin*
dicofol	*toxaphene*	**endosulfan**

Endosulfan Sprayer

Note that the person shown in the photo above is not using any protective equipment while spraying the pesticide. The effectiveness of pesticides is not very long lasting. There is much concern about exposure to pesticides in the agricultural environment. About 27% of pesticide applicators store pesticides in their homes, and about 94% of clothing worn for pesticide work is washed in the same machine as other laundry. However, agriculture is not the only place where we see exposure to pesticides. We use a large volume of pesticides in our homes as shown below.

Pesticides used in Homes and Gardens in the USA

Pesticide	Tons x 10^{-6}	Type	Toxicity (EPA)	Environmental toxicity
2,4-D	3150 – 4050	Herbicide	Slight - high	Birds, Fish, Insects
Glyphosate	2250 - 3600	Herbicide	Moderate	Birds, Fish, Insects
Dicamba	900 - 1800	Herbicide	Slight	Aquatic
Diazanon	900 - 1800	Insecticide	Moderate	Birds, Fish, Insects
Chloropyrifos	900 - 1800	Insecticide	Moderate	Birds, Fish
Carbaryl	900 - 1800	Insecticide	Moderate - high	Fish, Insects
Dacthal	450 - 1350	Herbicide	Low	Birds, Fish

From: Heynan et. Al., In the Nature of Cities, Routlege, 2006

The average life span of effectiveness of pesticides is about 10 years before the pest starts to show resistance.

Appearance of Pesticide Resistance

Pesticide	Introduction	Resistance
2,4-D	1945	1954
Dalapon	1953	1962
Atrazine	1958	1968
Picloram	1963	1988
Trifluralin	1963	1988
Triallate	1964	1987
Dicofop	1980	1987
ALS Inhibitors	1982	1987

From: S. R. Palumbi "The Evolution Explosion", Norton Press, 2001

The pesticides listed are: 2,4-D = 2,4-Dichlorophenoxyacetic acid, a common systemic pesticide/herbicide used in the control of broadleaf weeds. It is one of the most widely used herbicides in the world and is the third most commonly used herbicide in North America. 2,4-D is used for broadleaf weed control in agricultural and nonagricultural settings, and it is registered for use in both terrestrial and aquatic environments. Major sites include pasture and rangeland, residential lawns, roadways, and cropland. It has little effect on grasses. 2,4-D is an Auxin, a plant hormone that appears to work by causing uncontrolled cell division in vascular tissue. Abnormal increases in cell wall plasticity, biosynthesis of proteins, and production of ethylene occur in plant tissues following exposure, and these processes are responsible for uncontrolled cell division, so the weeds die of over growth. Crops treated with 2,4-D include field corn, soybeans, spring wheat, hazelnuts, sugarcane, and barley. To find a list of pesticide products containing 2,4-D, visit https://nepis.epa.gov/Exe/ZyNET.exe/2000U3UD.TXT?ZyActionD=ZyDocument&Client=EPA&Index=Prior+to+1976&Docs=&Query=&Time=&EndTime=&SearchMethod=1&TocRestrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&IntQFieldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A%5Czyfiles%5CIndex%20Data%5C70thru75%5CTxt%5C00000004%5C2000U3UD.txt&User=ANONYMOUS&Password=anonymous&SortMethod=h%7C-&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i425&Display=hpfr&DefSeekPage=x&SearchBack=ZyActionL&Back=ZyActionS&BackDesc=Results%20page&MaximumPages=1&ZyEntry=1&SeekPage=x&ZyPURL

Dalapon is an herbicide used to control grasses in a wide variety of crops, including fruit trees, beans, coffee, corn, cotton and peas. It is also registered for use in a number of non-crop applications such as lawns, drainage ditches, along railroad tracks, and in industrial areas.

Atrazine is one of the most widely used agricultural pesticides in the U.S.; Atrazine may be applied before and after planting to control broadleaf and grassy weeds. It is used primarily on corn, sorghum, and sugarcane, and is applied most heavily in the Midwest. Atrazine is used to a lesser extent on residential lawns, particularly in Florida and the Southeast. Atrazine is an endocrine disruptor, which works in the pathway of estrogen synthesis to increase the levels of that hormone. Studies have demonstrated that atrazine is an endocrine-disruptor e in fish, amphibians, and reptiles. Atrazine also induces mammary and prostate cancer in laboratory rodents. There are correlations between atrazine and similar reproductive cancers in humans. Atrazine as a risk factor in endocrine disruption in wildlife and reproductive cancers in laboratory rodents and humans (Fan et al. 2007; Vandenberg et al. 2012).

Picloram is a systemic herbicide used for general woody plant control. It also controls a wide range of broad-leaved weeds, but most grasses are resistant. A chlorinated derivative of picolinic acid, Picloram is in the pyridine family of herbicides.

Trifluralin is an herbicide used on grass to control broadleaf weeds. It is also used on some crops and vegetables, flowers, and shrubs.

Dicofop is a restricted use herbicide used on some crops (wheat, barley) and on golf courses. This herbicide suppresses grass weeds. Diclofop-methyl is classified as a likely carcinogen
https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/fs_PC-110902_1-Sep-00.pdf

ALS inhibitors are herbicides that inhibit acetolactate synthase (ALS), the enzyme common to the biosynthesis of the branch-chain amino acids (valine, leucine, and isoleucine), affect many species of higher plants as well as bacteria, fungi, yeasts, and algae. The novel mechanism of action attributed to ALS inhibitors, their effect on the reproduction of some plant species, their potency at extremely low concentrations, and the rapid evolution of resistance to these herbicides in some plants and microorganisms are characteristics that set ALS inhibitors apart from their predecessors. This class of chemicals affects seedling growth. Older plants exhibit varied signs of malformation, stunting, and reduced seed production. These herbicides are so potent that they can affect plants at levels that are undetectable by any standard chemical protocol. Weeds quickly become resistant to ALS inhibitors, presumably because these herbicides have a single mode of action and because many have long residual activity (Whitcomb 1999).

DDT https://www.epa.gov/ingredients-used-pesticide-products/ddt-brief-history-and-status

DDT (dichloro-diphenyl-trichloroethane) was used to combat malaria, typhus, and the other insect-borne human diseases among both military and civilian populations and for insect control in crop and livestock production, institutions, homes, and gardens. DDT's wide use in the United States and other countries led to the development of resistance by many insect pest species. In the 1970’s the naturalist Rachael Carson noted that the wide application of DDT was resulting in softening egg shells such that the birds were not hatching. Ms. Carson loved to hear the bird songs every spring and envisioned a time when there would not be bird songs. Her book, *Silent Spring* was instrumental in starting the environmental movement in the United States, and was a major factor in the 1972 banning of DDT.

Although it is no longer used or produced in the United States, we continue to find DDT in our environment. Other parts of the world continue to use DDT in agricultural

practices and in disease-control programs. Therefore, atmospheric deposition is the current source of new DDT contamination in our Great Lakes. DDT and its metabolites, DDE and DDD, are persistent, bioaccumulative and toxic (PBT) pollutants targeted by EPA.

Although DDT was banned in 1972, it can take more than 15 years to break down in the environment. Fish consumption advisories are in effect for DDT in many waterways including the Great Lakes ecosystem.

DDT is not acutely toxic but has been implicated in a number of health effects, including, and liver damage. Some data are in the literature that DDT exposure may be linked to breast cancer depending on the amount of DDT and the age of exposure (Cohn et al. 2007). DDT is an endocrine disruptor and is related to reproductive success in animal species and humans, and damage to the reproductive system (Mahalingaiah et al. 2012).

We can be exposed to DDT by eating contaminated fish and shellfish, by consuming imported food directly exposed to DDT, and eating crops grown in soil contaminated with DDT. Infants may be exposed through breast milk.

Although DDT was banned in the United States many years ago, DDT is still present in our environment. DDT in soil can be absorbed by some growing plants and by the animals or people who eat those plants. Fish and shellfish absorb DDT in water. When DDT is used outside the United States, we may be exposed because of atmospheric deposition, or runoff from land that was sprayed with DDT.

The World Health Organization (WHO) has approved the use of DDT in areas of the world with high levels of malaria. The spraying in that case is mainly indoors rather than areal.

Chlordane

The term chlordane actually refers to a complex mixture of chlordane isomers, other chlorinated hydrocarbons and by-products. In most temperate climates, only the two chlordane isomers: alpha and gamma chlordane, generally persist.

Chlordane was used in the United States from 1948 to 1978 as a pesticide on agricultural crops, lawns, and gardens and as a fumigating agent. In 1978, EPA canceled the use of chlordane on food crops and phased out other above-ground uses for the next 5 years. From 1983 to 1988, chlordane’s only approved use was to control termites in homes. The pesticide was applied underground around the foundation of

homes. In 1988, all approved uses of chlordane in the United States were terminated; however, manufacture for export still continues. Chlordane was banned in the United States because laboratory mice that were fed chlordane over long periods of time had a higher incidence of liver cancer than untreated mice. These results raised concerns about chlordane's ability to cause cancer in humans. Chlordane was also found to stay in the environment and build up in animal and fish fat. There was a concern that people may be exposed to this insecticide by eating food contaminated with chlordane, including fish, shell-fish, dairy, meat and poultry products. Chlordane is a persistent, bioaccumulative, and toxic (PBT) pollutant targeted by EPA.

Everyone in the United States has been exposed to low levels of chlordane due to its wide spread use. Because chlordane is bioaccumulative, it builds up in our food chain and becomes more concentrated as it moves up our food chain to humans and other wildlife. Fish consumption advisories for some species are in effect for chlordane in the Great Lakes ecosystem. Chlordane remains in our food supply because it was commonly used on crops in the 1960’s and 1970’s.

Chlordane is no longer made in the U.S. for export. Formulations containing chlordane are available internationally for termite control and wood treatment.

Although Chlordane is no longer made in the United States, it may be found in particles in the water column, or in soils that had been treated with Chlordane, or soils around wooden structures that were treated for termites. The EPA sets the maximum level of chlordane allowed in drinking water. This "maximum contaminant level" for chlordane has been set at no more than 2 micrograms of chlordane per liter of drinking water (one liter is approximately one quart). The Food and Drug Administration (FDA) and the U.S. Department of Agriculture (USDA) monitor the levels of chlordane and its breakdown products in domestic and imported foods.

Hexachlorobenzene (HCB) is a white crystalline solid, which was commonly used as a pesticide until 1965. In the past, HCB was also used as a fungicide to protect seeds of wheat and for a variety of industrial purposes. HCB is a persistent, bioaccumulative, and toxic (PBT) pollutant targeted by EPA.

There is concern about HCB because it is a PBT and stays in our environment for a long time and contaminates our food chain. HCB can cause severe health problems for humans and other wildlife.

Harmful effects resulting from HCB exposure include, damage to bones, kidneys, and blood cells. This compound can also cause immune system dysfunction and can lower survival rates of newborn children. HCB is teratogenic and is an endocrine disruptor.

We are exposed to HCB in a variety of ways:

- Infants exposed through breast milk
- During pregnancy, unborn children can be exposed through the mother's blood stream
- By eating foods such as meat and poultry if those animals are exposed from contaminated feed
- By drinking dairy products where the cattle have been exposed through their feed
- By eating contaminated fish and shellfish
- Inhaled in urban air

Although HCB is no longer directly used, it is still found in our environment as a by-product of certain activities and because of past use.

Past Uses

- To make fireworks and ammunition
- To manufacture synthetic rubber
- Used as a fungicide to protect wheat and other seeds

Potential Environmental Sources

- By-product when making other chlorine-containing compounds
- Found in water sediments
- By-product when manufacturing some pesticides
- Use of HCB-contaminated pesticides
- Found in chlorination treatment of process water and wastewater
- Incineration of municipal and hazardous wastes
- By-product when making chemical solvents (chemicals used to dissolve other chemicals)

Mirex

The insecticide Mirex was used for 16 years (1962–1978) in the Southeastern United States to control the imported fire ant. Eight of the infested states border on the Atlantic Ocean or the Gulf of Mexico or both, making fire ant control programs an issue in the use and management of estuaries. Mirex as a control chemical for fire ants became controversial when it was found to be highly toxic to a variety of marine crustaceans, including commercially important species of shrimps and crabs. Extensive public

hearings were held during the period 1973–1976. All uses of Mirex were cancelled in 1978. This chemical is usually seen as a snow-white crystalline solid, is odorless, and does not burn easily. When Mirex does break down, it turns into photomirex that also can have harmful health effects. Mirex has been listed as a persistent, bioaccumulative, and toxic (PBT) pollutant targeted by EPA. Mirex is not broken down in the body and is stored in our fat.

Mirex is classified by the EPA as a probable carcinogen in humans. This chemical also has harmful effects on stomach and intestines, damages the liver and kidneys, harms the eyes and thyroid gland, causes damage to the nervous system and the reproductive system, and may be related to miscarriages. Mirex is also an endocrine disruptor.

We are exposed to Mirex in a number of ways. The most common exposure in infants is probably through breast milk. Older children, adolescents, and adults may be exposed to Mirex by eating contaminated fish and shellfish.

Although the sale, distribution, and use of Mirex is prohibited in the United States, it is still found in our environment.

Past Uses

- Widely used in the Southeast to kill fire ants
- Commonly used as a fire retardant in plastics, rubber, paint, paper, and electrical goods

Potential Sources to Our Environment include

- contaminated bottom sediments in waterways
- surface water contamination
- sediments

Organophosphate pesticides are not considered PBTs but are widely used and pose a threat to human health. It is estimated that about 50% of all pesticide exposure is from organophosphates. Organophosphate compounds are neuro toxic, and have been used in at least one terrorism attack in Japan (http://www.opcw.org/news/article/the-sarin-gas-attack-in-japan-and-the-related-forensic-investigation/).

Organophosphates are clear and colorless and have little or no odor.
Organophosphates can be inhaled or absorbed through the skin. They are acutely toxic but may have long-term effects as well.

Halogenated Compounds—Dioxins and Furans

Dioxin (2,3,7,8-TPCDD)

The term Dioxin is commonly used to refer to a family of congeners that all share a similar chemical structure. This family includes 75 of the polychlorinated dibenzo dioxins (PCDDs), and 135 of the polychlorinated dibenzo furans (PCDFs). PCDDs and PCDFs are not commercial chemical products but are trace level unintentional byproducts of most forms of combustion and several industrial chemical processes. TCDD was a byproduct of 2,4,5-Trichlorophenoxyacetic acid (2,4,5-T), an herbicide also known as *Agent Orange* that was used widely during the Vietnam War era. Dioxin levels in the environment have been declining since the early seventies and have been the subject of a number of federal and state regulations and cleanup actions; however, current exposure levels still remain a concern.

There is great concern about the toxicity of dioxins because of its reputation as the most toxic compound ever synthesized (http://www.ejnet.org/dioxin/). Because dioxins are widely distributed throughout the environment in low concentrations, are persistent and bioaccumulated, most people have detectable levels of dioxins in their tissues. These levels, in the low parts per trillion, have accumulated over a lifetime and will persist for years, even if no additional exposure were to occur. This background exposure is likely to result in an increased risk of cancer and is uncomfortably close to levels that can cause subtle adverse non-cancer effects in animals and humans.

Dioxins have been characterized by EPA as likely to be human carcinogens and are anticipated to increase the risk of cancer at background levels of exposure. Although many studies implicate dioxins as potential carcinogens, there is also a large body of evidence that dioxin exposure is also related to other complex diseases, possibly by its endocrine disruption ability (Lee et al. 2006).

In 1997 the International Agency for Research on Cancer classified 2,3,7,8, TPCDD, the best studied member of the dioxin family, a known human carcinogen. It is suggested that 2,3,7,8 TCDD accounts for about 10% of our background dioxin risk.

At body burden levels 10 times or less above those attributed to average background exposure, adverse non-cancer health effects have been observed both in animals and, to a more limited extent, in humans. In animals these effects include changes in hormone systems, alterations in fetal development, reduced reproductive capacity, and immunosuppression. Effects specifically observed in humans include changes in

markers of early development and hormone levels. At much higher doses, dioxins can cause a serious skin disease in humans called chloracne.

Human exposure to dioxins result in a change of the sex ratio (Mocarelli et al. 2000) spontaneous abortion (Schnorr et al. 2001), endometriosis (Bruner-Tran and Osteen 2010), and various teratogenic actions (Thackaberry et al. 2005).

Dioxins are commonly detected in air, soil, sediments and food. Dioxins are not very water-soluble. Dioxins are transported primarily through the air and are deposited on the surfaces of soil, buildings and pavement, water bodies, and the leaves of plants. Most dioxins are introduced to the environment through the air as trace products of combustion. The principal route by which dioxins are introduced to most rivers, streams and lakes is soil erosion and storm water runoff from urban areas. Industrial discharges can significantly elevate water concentrations near the point of discharge to rivers and streams. Major contributors of dioxin to the environment include:

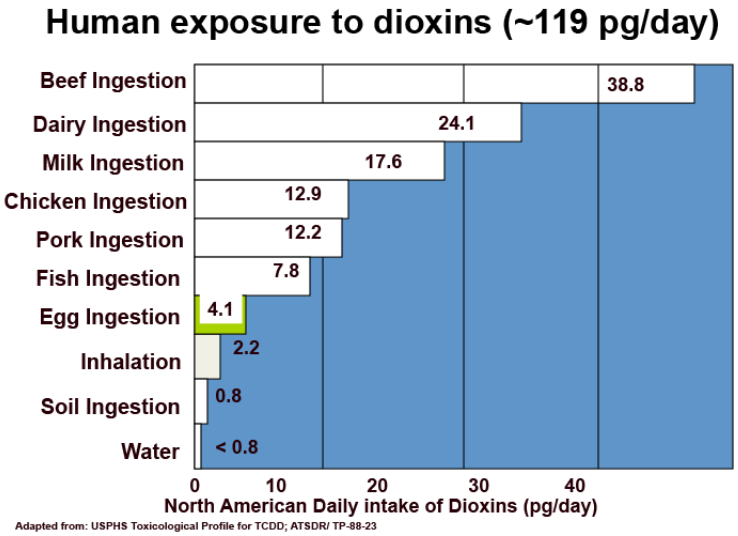
- Incineration of municipal solid waste.
- Incineration of medical waste.
- Secondary copper smelting.
- Forest fires.
- Land application of sewage sludge.
- Cement kilns.
- Coal fired power plants.
- Residential wood burning.
- Chlorine bleaching of wood pulp.
- Backyard burning of household waste may also be an important source.
- Vinyl chloride manufacture and disposition.

Most of us receive almost all of our dioxin exposure from food, specifically from the animal fats associated with eating beef, pork, poultry, fish, as well as milk and dairy products (see the figure below). Most of us get these foods through the commercial food supply. Since most of the meats and dairy products we consume are not produced locally but have been transported hundreds or thousands of miles, the majority of our dioxin exposure does not come from dioxin sources within our own community. Additionally, because we are all being exposed from the same national food supply, we are all receiving a similar exposure with the main difference between individuals being individual food preferences.

Important exceptions to this pattern of general population exposure are individuals who, over an extended period of time, eat primarily locally grown meat, fish or dairy products

that have significantly greater dioxin levels than those found in the commercial food supply. Individuals in this situation receive greater exposure and are at greater risk than the general population. These elevated dioxin food levels can be the result of nearby local sources or from past contamination of soil or sediments. Another example of elevated exposure is nursing infants; however, health experts generally agree the overall benefits to infants of nursing far outweigh potential risks.

Working in industries involved in producing certain pesticides containing PCDDs as impurities, working at paper and pulp mills, or operating incinerators.



The most noted health effect in people exposed to large amounts of 2,3,7,8-TPCDD is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other skin effects noted in people exposed to high doses of 2,3,7,8-TPCDD include skin rashes, discoloration, and excessive body hair. Changes in blood and urine that may indicate liver damage also are seen in people. Exposure to high concentrations of PCDDs may induce long-term alterations in glucose metabolism and subtle changes in hormonal levels.

In certain animal species, 2,3,7,8-TPCDD is especially harmful and can cause death after a single exposure. Exposure to lower levels can cause a variety of effects in animals, such as weight loss, liver damage, and disruption of the endocrine system. In many species of animals, 2,3,7,8-TPCDD weakens the immune system and causes a decrease in the system's ability to fight bacteria and viruses. In other animal studies, exposure to 2,3,7,8-TPCDD has caused reproductive damage and birth defects. Some animal species exposed to PCDDs during pregnancy had miscarriages and the

offspring of animals exposed to 2,3,7,8-TPCDD during pregnancy often had severe birth defects including skeletal deformities, kidney defects, and weakened immune responses.

Several studies suggest that exposure to 2,3,7,8-TCDD increases the risk of several types of cancer in people. Animal studies have also shown an increased risk of cancer from exposure to 2,3,7,8-TCDD.

The World Health Organization (WHO) has determined that 2,3,7,8-TPCDD is a human carcinogen. The Department of Health and Human Services (DHHS) has determined that 2,3,7,8-TPCDD may reasonably be anticipated to cause cancer.

Polychlorinated Biphenyls (PCBs)

There are no known natural sources of PCBs in our environment. PCBs are either oily liquids or solids, are colorless to light yellow, and have no smell or taste. Because they do not easily burn and are good insulators, PCBs have been used widely as coolants and lubricants. PCBs are persistent, bioaccumulative and toxic (PBT) pollutants that have been targeted by EPA. There are no current uses for PCBs.

PCBs do not break down in our environment and can have severe health effects on humans. PCBs in the air eventually return to our land and water by settling or from runoff in snow and rain. In our water, PCBs build up in fish and can reach levels hundreds of thousands of times higher than the levels in water. Fish consumption advisories are in effect for PCBs in all five of the Great Lakes. PCBs are the leading chemical risk from fish consumption.

Some of the known toxicities associated with exposure to PCBs include

- Possible carcinogenesis
- Damage the stomach
- Skin irritation
- Liver and Kidney damage
- Thyroid gland injuries
- Endocrine disruption

During the 1960s, mink farmers in the Great Lakes region fed their mink fish from Lake Michigan tributaries that had been contaminated with PCBs. These ranch mink suffered severe outcomes including high mortality rates and reproductive failure. The mink breeding industry in this region experienced great economic hardship as a result of the animals' failure to successfully reproduce (http://books.google.com/books?id=MuV3G5Zjn1oC&pg=PA265&lpg=PA265&dq=lake+michigan+PCBs+1960+commercial+mink+ranchers&source=bl&ots=Vrgcu3puEx&sig=gVQwLRvzSEzvIHhj8OXB1sLWjmk&hl=en&sa=X&ei=whuBU4ilONGtyASA8oKgCQ&ved=0CCgQ6AEwAA, - v=onepage&q=lake michigahttp://books.google.com/books?id=MuV3G5Zjn1oC&pg=PA265).

We can be exposed to PCBs by eating contaminated fish and shellfish (Hites et al. 2004). PCBs may be present in milk, meat, and other fatty products. Infants may be exposed through breast milk.

It is also possible to be exposed by breathing indoor air in buildings where electrical equipment contains PCBs.

Manufacturing of PCBs was stopped in the Unites States in 1977 because they were found to build up in our environment and cause harmful effects. However, we can still find them in our environment, especially in our lakes, rivers, and streams. PCBs may be found in the environment at

- Poorly maintained hazardous waste sites containing PCBs
- Illegal/improper dumping of PCB wastes such as transformer fluids
- Leaks or releases from electrical transformers containing PCBs
- Improper disposal of PCB-containing consumer products
- Old microscope oil and hydraulic fluids
- Old television sets and refrigerators, lighting fixtures, electrical devices, or appliances containing PCB capacitors made before 1977.
- Sediments in the bottom of lakes, river, or our ocean constantly release small amounts of PCBs into the environment.
- Generated in municipal and industrial incinerators from the burning of organic wastes.

Polybrominated Diphenyl Ethers (PBDEs)

Polybrominated diphenyl ethers (PBDEs) are man-made chemicals found in plastics, used in a variety of consumer products to make them difficult to burn. Very little is known about the health effects of PBDEs in humans, but effects have been reported in animals. PBDEs have been found in any of the 1,657 current or former National Priority List (NPL) sites identified by the EPA.

PBDEs are flame – retardant chemicals that are used in plastics and foam products to make them difficult to burn. There are different kinds of PBDES; some have only a few bromine atoms attached, while some have as many as 10 atoms of bromine attached to the central molecule.

PBDEs exist as mixtures of similar chemicals called congeners. Because they are mixed into plastics and foams rather than bound to them, PBDEs can leave the products that contain them and enter the environment.

- PBDEs enter the air, water, and soil during their manufacture and use in consumer products
- In air, PBDEs can be present as particles, but eventually settle to soil or water.
- Sunlight can degrade some PBDEs.
- PBDEs do not dissolve easily in water but stick to particles and settle to the bottom of bodies of water.
- Some PBDEs can accumulate in fish but usually at low concentrations.

How might I be exposed to PBDEs?

- The concentration of PBDEs in human blood, breast milk, and body fat indicate that most people are exposed to low levels of PBDE.
- You may be exposed to PBDEs from eating foods or breathing air contaminated with PBDEs.
- Workers involved in the manufacture of PBDEs or products that contain PBDEs may be exposed to higher levels than the general population.
- Occupational exposures can occur in people who work in enclosed spaces where PBDE-containing products are repaired or recycled.

There is no definite information on health effects of PBDEs in people. Rats and mice that ate food with moderate amounts of PBDEs for a few days had effects on the thyroid gland. Those that ate smaller amounts of PBDEs for weeks or months had effects on the thyroid and the liver. Large differences in effects are seen between highly brominated and less brominated PBDEs in animal studies.

Preliminary evidence suggests that high concentrations of PBDEs may cause neurobehavioral alterations and affect the immune system in animals. We do not know whether PBDEs can cause cancer in humans. Rats and mice that ate food with dibromodiphenyl ether (one type of PBDE) throughout their lives developed liver tumors. Based on this evidence the EPA has classified dibromodiphenyl ether as a possible human carcinogen. PBDEs with fewer bromine atoms than dibromodiphenyl ether are listed by the EPA as not classifiable as to human carcinogenicity because of the lack of human and animal cancer studies.

Children are exposed to PBDEs in generally the same way as adults, mainly by eating contaminated food. Because PBDEs dissolve readily in fat they can accumulate in breast milk and may be transferred to babies and young children. Exposure to PBDEs in the womb and through nursing has caused thyroid effects and neurobehavioral alterations in newborn animals, but not birth defects. It is not known whether PBDEs can cause birth defects in children (Birnbaum and Staska 2004).

Families can minimize exposure to these chemicals by discouraging children from playing in the dirt near hazardous waste sites. Children should also be discouraged from eating dirt and encouraged to wash their hands frequently. Don't use synthetic, such as polyester, bed clothing for children.

People who are exposed to PBDEs at work should shower and change clothes before going home each day. Work clothes should be stored and laundered separately from the rest of the family's clothes.

There are clinical tests that can detect PBDE's in blood, body fat and breast milk. These tests can tell whether you have been exposed to high levels of the chemicals but cannot tell the exact amount or type of PBDE you have been exposed to, or whether harmful effects will occur. Blood tests are the easiest and safest for detecting recent exposures to large amounts of PBDEs. These tests are not routinely available at your doctor's office, but samples can be sent to laboratories that have the appropriate equipment.

The EPA requires that companies that transport, store, or dispose of p-bromodiphenyl ether (a particular PBDE compound) follow the rules and regulations of the federal hazardous waste management program. The EPA requires that industry tell the National Response Center each time 100 pounds or more p-bromodiphenyl ether are released to the environment (ATSDR 2004).

Metals

Mercury and Compounds

Mercury is a toxic metal and a natural element, commonly seen as a shiny, silver-white, odorless liquid metal. Mercury is a persistent, bioaccumulative, and toxic (PBT) pollutant.

Mercury affects the nervous system. Methyl mercury is a chemical species that bioaccumulates in fish. Fish consumption advisories are in effect for mercury in thousands of lakes and rivers, including much of the Great Lakes ecosystem.

Much of the toxicity of mercury was discovered after an exposure to the population of Minamata, Japan (Hachiya 2006). The major concern with mercury exposure is neurotoxicity (Rice et al. 2014).

- May cause cancer.
- Damages the stomach and large intestine.
- Permanently damages the brain and kidneys.
- Permanently harms unborn children.
- Can cause lung damage, increased blood pressure and heart rate.

We are exposed to mercury

- By eating contaminated fish and shellfish.
- Unintentional mercury spills.
- Incinerators and facilities burning Hg-containing fuels (i.e. coal or other fossil fuels, mercury-containing wastes).
- Unborn children can be exposed through the mother’s blood and infants may be exposed through breast milk.

Current Uses of mercury include

- Production of some batteries
- Thermostats and cameras
- Cathode tubes
- Calculators and small appliances
- Medical laboratory chemicals
- Catalyst in production of urethane polymers for plastics
- Cathode in electronic production of chlorine and caustic soda

- Mercury vapor lamps
- Thermometers and Barometers
- Switches (electrical)
- Hearing aids
- Common household cleaners

Mercury is used in the following ways:

- Manufacturing activities—mining (especially gold mining) and smelting
- Wastewater entering lakes, rivers, etc.
- Waste from dental cavity-filling material
- Emissions from coal burning
- Combustion of various fuels
- Mixed waste incineration
- Medical waste incineration
- Old paint

Alkyl-Lead

Alkyl-lead compounds are man-made compounds in which a carbon atom of one or more organic molecules is bound to a lead atom. Tetraethyllead [TEL] and Tetramethyl lead [TML] compounds are the most common alkyl-lead compounds. Alkyl-lead compounds are used as a fuel additive to reduce “knock” in combustion engines. They were banned from use in automobile fuels but are still in limited use today in the United States.

In the body, alkyl-lead compounds are distributed through the blood to “soft tissues” particularly the liver, kidneys, muscles, and brain. Alkyl-lead is a predominant type of organic lead compound and is much more bioavailable and toxic than inorganic lead. Exposures to humans can result in lead poisoning.

Initial symptoms of alkyl-lead poisoning include:

- Anorexia
- Insomnia
- Tremor
- Weakness,
- Fatigue
- Nausea and vomiting
- Mood shifts such as aggression or depression

- Impairment of memory
- ADHD

In the case of acute alkyl-lead poisoning, possible health effects include:

- Mania
- Convulsions
- Delirium
- Fever
- Coma
- Death

Lead poisoning can result from the ingestion or inhalation of inorganic lead compounds emitted as exhaust through the combustion process (as a direct result of the use of alkyl-lead in gasoline).

The alkyl-lead problem in the U.S. was solved by banning its use in gasoline. However, there are still some limited uses of alkyl-lead in racing, piston driven aircraft, and recreational marine fuels (http://www.epa.gov/pbt/pubs/alkylaction.htm).

Potential sources to our Environment

- Piston driven aircraft gasoline
- Auto racing gasoline
- Recreational marine gasoline

Polycyclic Aromatic Hydrocarbons (PAHs)

Benzo(a)pyrene - B(a)P

Benzo(a)pyrene (B(a)P) is a member of a class of compounds known as polycyclic aromatic hydrocarbons (PAHs) which generally occur as complex mixtures and not as single compounds. PAHs are primarily by-products of incomplete combustion. These combustion sources are numerous, including natural sources such as wildfires, industrial processes, transportation, energy production and use, food preparation, smoking tobacco, and disposal activities such a as open trash burning.

B(a)P along with other PAHs are suspected of causing cancer in humans. It is bioaccumulative, does not break down easily in our environment, and is subject to long range air transport.

B(a)P is harmful to human health. B(a)P is a carcinogen in humans (http://www.foreffectivegov.org/epa-scientists-deem-benzo-a-pyrene-cancer-causing-chemical), and causes skin disorders in humans and animals and harmful developmental and reproductive effects. Additionally, B(a)P is an endocrine disruptor in marine species (Tian et al. 2013).

We are exposed to B(a)P In the home by breathing air contaminated by smoke from fireplaces, wood stoves, furnaces burning coal or oil and from food preparation.

Eating meats and fish that have been smoked or charbroiled

Smoking tobacco products

Inhaling vehicle exhaust – Especially diesel exhaust

Inhaling fumes from working with coal tar and asphalt

Working near charbroiling and high temperature frying equipment

Working in coal coking operations and other industrial operations such as asphalt and aluminum production

B(a)P has no specific uses. It is generated by various combustion sources, including wood and tobacco smoke. B(a)P is also a component or contaminant of such materials as tar and asphalt.

Potential sources of PAHs in our environment include:

- Wildfires and prescribed burnings
- Primary aluminum production
- Coke ovens
- Residential wood stoves
- Burning of scrap tires
- Open trash burning
- On-road vehicles
- Asphalt roofing manufacturing
- Industrial boilers
- Meat charbroiling
- Wood smoke
- Tobacco smoke

Web Resources

EPA—Mercury, www.epa.gov/mercury

Contents: General information

Actions

Fish advisories

Technical Information

Wisconsin Mercury Source Book—"A Guide to Help your Community Identify and Reduce Releases of Elemental Mercury," http://infohouse.p2ric.org/ref/04/03851/section1.pdf

Contents:Background on mercury.

How to draft a community mercury reduction plan.

Sources of mercury by sector.

Toxic Release Inventory Latest Data Release, http://www.epa.gov/tri/

Community Information on EPA Superfund Program, http://www.epa.gov/superfund/community/index.htm

Health and Safety and Risk Assessment as applicable to Superfund, http://www.epa.gov/superfund/health/index.htm

Map of Superfund Sites in your community, http://www.epa.gov/superfund/sites/

EPA Lead Program: Books, Brochures and Posters, http://www.epa.gov/lead/pubs/leadpdfe.pdf

Testing Your Home for Lead: In paint, dust and soil (EPA 747-K-00-01), July 2000 (http://www.epa.gov/lead/pubs/leadtest.pdf)

Lead in Your Home: A Parent's Reference Guide (EPA 747-B-98-002), June 1998 (http://www.epa.gov/lead/pubs/leadrev.pdf)

EPA National List of Fish Advisory, http://www.epa.gov/ost/fish

Environmental Education for Kids, http://www.epa.gov/kids/

EPA Environmental Education Center, http://www.epa.gov/teachers

National Institute of Environmental Health Sciences (NIEHS): Support and Community Outreach Programs, http://www.niehs.nih.gov/health/index.cfm

What is the Toxics Release Inventory?

The Toxics Release Inventory (TRI), published by the U.S. EPA, is a valuable source of information regarding toxic chemicals that are being used, manufactured, treated, transported, or released into the environment (http://www.epa.gov/tri/).

Two rules, Section 313 of the Emergency Planning and Community Right-To-Know Act (EPCRA) and Section 6607 of the Pollution Prevention Act (P2), mandate that a publicly accessible toxic chemical database be developed and maintained by the U.S. EPA. This database, known as the Toxics Release Inventory (TRI), contains information concerning waste management activities and the release of toxic chemicals by facilities that manufacture, process, or otherwise use said materials on the land, air and water.

Under the Pollution Prevention Act (P2) of 1990, TRI collects information to track industry progress in reducing waste generation and moving towards safer waste management alternatives. Companies can report on progress of pollution prevention. https://www.epa.gov/p2.

The National Policy included in the P2 Act puts forward these principles;

- Pollution should be prevented or reduced at the source whenever feasible;
- Pollution that cannot be prevented should be recycled in an environmentally safe manner whenever feasible;
- Pollution that cannot be prevented or recycled should be treated in an environmentally safe manner whenever feasible; and
- Disposal or other release into the environment should be employed only as a last resort and should be conducted in an environmentally safe manner.

Manufacturing facilities must report to TRI if:

- The facility is in a specific industrial sector (e.g., manufacturing, mining, electric power generation),

- Ten or more full-time equivalent employees, work at the facility

- The Facility Manufactures or processes more than 25,000 pounds of a TRI-listed chemical or otherwise uses more than 10,000 lbs. of a listed chemical in a given year.

Manufacturing facilities include: chemical, petroleum refining, primary metals, fabricated metals, paper, plastics, and transportation equipment. Federal facilities have been required to report since 1994, regardless of their SIC classification.

The TRI database includes information on:

- Chemicals released into the local environment during the preceding year
- Quantity of each chemical released into the air, water, and land in a particular year
- Quantity of the chemicals that were transported away from the reporting facility for disposal, treatment, recycling, or energy recovery
- How the chemical wastes were treated at the reporting facility
- The efficiency of waste treatment
- Pollution prevention and chemical recycling activities. However, the law does not cover toxic chemicals that reach the environment from non-industrial sources, such as dry cleaners or auto service stations

Reported releases are annual estimates. Thus, there are limitations to the reporting base. Persistent, Bioaccumulative, Toxic chemicals (PBTs) have lower reporting thresholds:

- More than 100 lbs. of Aldrin, Lead, Lead Compounds, Methoxychlor, PACs, Pendimethalin, Tetrabromobisphenol A, Trifluralin
- More than 10 lbs. of Benzo(g,h,i)perylene, Chlordane, Heptachlor, Hexachlorobenzene, Isodrin, Mercury, Mercury Compounds, Octachlorostyrene, Pentachlorobenzene, PCBs, Toxaphene
- More than 0.1 grams of Dioxin and Dioxin-like Compounds

A Public “Report Card”

- TRI is a public “report card” for the industrial community, creating a powerful motivation for waste reduction.
- The annual accounting of the nation’s management of industrial toxic chemical wastes is a valuable source of information for concerned individuals and communities.

- Citizens can use TRI to evaluate local facilities through comparisons to determine how toxic chemicals are used and, with other information, evaluate potential health risks for their community.
- Organizations can use TRI information as a starting point for constructive dialogue with manufacturing businesses in the area.

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