PCB'S AND OTHER PERSISTENT ORGANIC CONTAMINANTS

What are Chlorinated compounds?

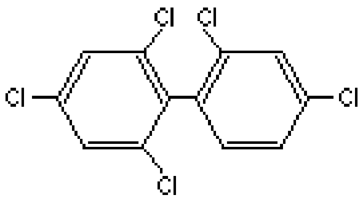
Chlorine and its compounds

Chlorines, benzene rings and dioxins

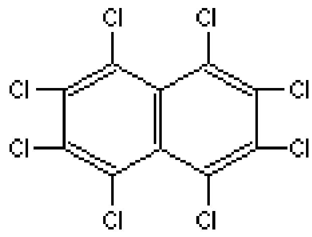
Halogenated Aromatic Compounds

Structure, Manufacture and Purpose

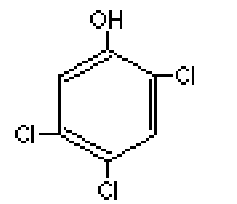
Some Examples:



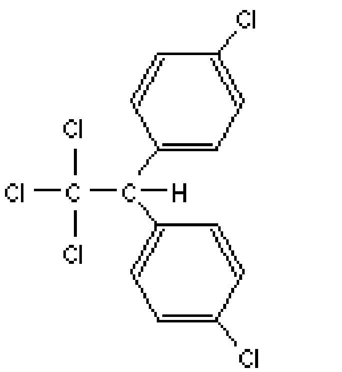
A PCB (polychlorinated biphenyl)



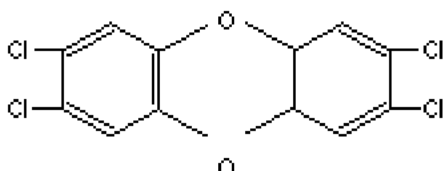
The fully chlorinated ingredient in polychlorinated naphthalene (PCN).



2,4,5-Trichlorophenol or Dowicide 2 (fungicide, wood preservative and sanitizing agent).



Dichlorodiphenyltrichloroethane (DDT)



2,3,7,9-TCDD (the most toxic of the 75 known dioxins)

Uses of chlorinated compounds:

- food additives, paints, adhesives, plastics (PVC - polyvinyl chloride)
- chlorine is a powerful bleaching agent and disinfectant
- used in water supply since kills viruses, bacteria and algae
- chlorinated compounds useful as solvents
- pesticides : DDT, 2,4-D and PCP

The chlorination contraversy: health *vs.* economics

Problems:

- large number of uses therefore large number of ways to enter environment
- persistent, lipophilic and bioaccumulate

Polychlorinated Aromatic Compounds (PCAs):

- carbon compounds with chlorine as the functional group
- changes physical properties of compound
- increases stability (persistence in environment)

- lipophilic (stays in fatty tissues of organism)
- PCA -> PCA: Receptor Actions -> Effects -> Responses (arylhydrocarbon receptor is most common)
- includes polychlorinated napthalenes (PCNs), polychlorinated biphenols (PCBs), and dioxins
- PCBs - used in transformers, hydraulic fluids, plastics, adhesives, lubricants and flame retardants.
- PCNs - have many properties in common with DDT

The mechanism of toxicity of chlorinated compounds
Mechanisms of PCB toxicity and carcinogenesis

PERSISTENT CHLORINATED ORGANICS IN THE ENVIRONMENT

1. General introduction to transfer of toxic organic chemicals
The critical processes which eventually affect the fate of persistent toxic organic chemicals in the Great Lakes are those which operate across interfaces between major ecosystem compartments. The major interfaces are:

Land-Water

Point sources are the connecting channels of the lower Great Lakes - Niagara River and Detroit-St. Clair rivers. Non-point sources are ground water and tributaries to the lakes.

Air-Water

The atmosphere acts as both a source of toxic organic chemicals - in the form of wet-precipitation, and as a sink - the water release volatile chemicals to the atmosphere.

Sediment-Water

The sediment affect the fate of persistent toxic organic chemicals in three ways:

- Sorption-desorption

- Burial (into bottom sediments)

- Sedimentation/resuspension

Nutrient-Water

Increased nutrient increase productivity, which affect the toxic organic chemicals in three processes - sedimentation, degradation and bioaccumulation.

A more flexible, toxic organic-chemical oriented sampling of aquatic media should aim at providing not only a monitor of conditions, but a more readily interpretable database of bioavailability and bioaccumulation processes.

2. Great Lakes - toxic chemical exposure and effects

1. 1960-1970
    - discovery of persistent organic chemicals (eg. DDE, methylmercury, PCBs) which bioaccumulate
    - Great Lakes fish-eating birds are suffering reproductive problems

2. 1970s'
   - hundreds of persistent trace contaminant derivative discovered
   - human health concerns arose first for the sale of commercial fish, then for the consumption of sport fish and finally for drinking water
3. 1980s'
   - it was discovered that the surface interaction of the lakes with the atmosphere was linked for toxic chemicals, therefore it was necessary to consider chemical effects in all ecosystem pathways (air, water, soil, sediment and biota) and exposure routes, rather than just effects in one aqueous ecosystem compartment
4. 1986
   - humans were found to be contaminated with Great Lakes' contaminants;
   - discovery of the major exposure route -- **FOOD** !!! Therefore it is necessary to realize that these chemicals not just bioaccumulate, but they cycle through the environment.

3. PCB Toxicity in Relation to Animal and Human Studies and Regulations for Control

- *Animal studies* :
  - Similar symptoms of PCB toxicity include: reproductive dysfunction, increased mortality, neurological dysfunctions, altered neuromuscular reflexes (Rhesus monkeys showed symptoms most similar to humans exposed)
- *Human studies* :
  - Occupational workers experienced "burning skin", skin lesions, chloracne
  - 2 accidental exposure events in history: both related to contaminated rice oil consumption in Japan and Taiwan.
  - individuals exposed showed facial swelling, chloracne, increased skin pigmentation
  - children born of exposed mothers had lower IQs, growth retardation, premature births, diminished sensory and motor nerve conduction
  - PCBs transferred from mother to fetus through "placental transfer" and later from mother to child through breast feeding
- *PCB regulation* :
  - FDA established temporary tolerance levels of PCBs in food, many have been lowered since their original establishment, due to human concerns
  - modern guideline sets a limit of 1 mg/kg per day for fish alone, but this limit is exceeded by Great Lakes fish eaters
- *Great Lakes Fish* :
  - Guidelines to eating sport fish: federal guideline by 1987 for the commercial sale of fish is 2 ppm.
  - smaller fish have lower levels of PCBs than larger fish due to lower levels of body fat and less exposure time
  - 1992: Senate introduced legislation that commercially caught fish will be subject to mandatory federal inspection

- Levels of PCBs have been declined continuously since the 1960s but are nowhere near "safe" levels yet

4. Modelling of effect of PCBs in the open ocean

- *Open Ocean:*
  - final sink for contaminants
  - far away from human activities, distribution of pollutants uniform to simplify modelling
  - marine mammals are most endangered species to long term accumulation and toxicity of PCBs (low metabolic potency, large lactational transfer over generations and top of food chain)
- *Purpose of modelling:*
  - important to make clear the distribution, behaviour, fate of chemicals that threaten living beings in the environment and to establish safety measures in production,use and disposal
  - focus research on representative compounds and study in detail from wide range of temporal and spatial scope on a global scale
- *Why PCBs* ?
  - large production and world wide use lead to presence in all global enviromental compartments
  - many accidental contamination and harmful impacts have occurred
  - continuous release (from use in close system expected)
  - data available for modelling
  - case 1: no destruction of PCBs now under use carried out in future
  - case 2: 50% PCBs will be treated within 10 Years
  - look at mesotrophic, eutrophic and oligotrophic zones

For the next few decades, PCB levels will:

Case 1:			
	Eutrophic	Mesotrophic	Oligotrophic
Surface water	decrease	decrease	increase
Marine mammal	increase	increase	increase
Case 2:			
surface water	decrease	decrease	constant
Marine mammal	increase*	increase*	increase*

* total values in case 2 are expected to be less that of Case 1, but still increasing

For more details see the following:
Ecosystem Surprise: Toxic Chemical Exposure and Effects in the Great Lakes (by D.J. Hallett), from 'Toxic Contaminations In Large Lakes -chronic effects of toxic contaminants in large lakes', 1988. (reviewed by Marion Lai)
Factors affecting sources and fate of persistent toxic organic chemicals: examples from the Laurentian

Great Lakes (by Allan, R.J.), from 'Aquatic Ecotoxicology: Fundamental Concepts and Methodologies, 1989. (reviewed by Shauna Hu)
PCBs on the Globe: Possible Trends of Future Levels in the Open Ocean Environment (edited by Schmidtke) from: 'Toxic contamination in Large Lakes - Sources, fate and controls of toxic contaminants' . (reviewed by Joanna Wilson).
Perspectives on Human Health Concerns from Great Lakes Contaminants (by Sonzogi & Swain), from 'Toxic contaminants in the Great Lakes', 1984. (reviewed by Martha Lai).