# Long-term Effects of Bioaccumulation in Ecosystems

Jaakko Paasivirta

Department of Chemistry, University of Jyväskylä, Finland, P.O. Box 35, SF-40351 Jyväskylä, Finland

Extensive damage to organisms and declines in wildlife populations have been observed together with long-term bioaccumulation and biomagnification of persistent xenobiotic chemicals. Heavy metals, especially organic or biomethylated mercury, lead, cadmium and organic tin compounds have caused environmental damage through bioaccumulation on a local scale. Effects on wildife caused by bioaccumulation of persistent organochlorine compounds are more widespread. However, the causal relationship between a biomagnified compound and the long-term effects have been established in only a few cases. Metabolic transformations, and occurrence of several toxic contaminants together in many cases, complicate evaluations of the sources of long-term effects. Environmental fate, exposure of biota and biomagnification of a chemical can be predicted by modelling from its properties and from ecological, geological and climatic conditions of the recipient environment. Model predictions can be refined by experimental factors obtained from results of the field studies. Empirical estimates of hazardous bioaccumulation or biomagnification are obtained from field analyses of different trophic levels. Trend analyses of biomagnified contaminants and their effects can be utilized in prognosis of future development and in evaluation of the need for further action to protect the environment and human health.



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## List of Symbols and Abbreviations

| | |
|---|---|
| ACE | aromatic chloroethers |
| AHH | aryl hydrocarbon hydroxylase |
| ALAD | aminolevulinic acid dehydratase |
| BCF | bioconcentration factor |
| Bp | boiling point |
| CHL | chlordanes |
| CYMD | chlorocymenenes |
| CYMS | chlorocymenes |
| DBT | dibutyltin |
| DDD | tetrachlorodiphenylethane (1,1-dichloro-2,2-bis(4-chlorophenyl) ethane) |
| DDE | dichloro-diphenyl-dichloroethene (1,1-dichloro-2,2-bis (4-chlorophenyl)ethene) |
| DDT | dichloro-diphenyl-trichloroethane (1,1,1-trichloro-2,2-bis (4-chlorophenyl)ethane) |
| dw | dry weight |
| EI | eggshell (thickness) index |
| EROD | etoxyresorufin-O-deethylase |
| ESM | eyed stage mortality (of hatching fish juveniles) |
| FERM | fertilizing mortality (of fish eggs) |
| GCOL | fish egg colour |
| fw | fresh weight (wet tissue) |
| HCBz | hexachlorobenzene |
| HCH | hexachlorocyclohexanes |
| $k_B$ | biodegradation rate, |
| $k_H$ | hydrolysis rate, |
| $k_P$ | photodegradation rate |
| LIND | $\gamma$-HCH |
| lw | lipid weight (in fat) |
| MBT | monobutyltin |
| MFO | mixed fuction oxydases |
| $\delta^{15}N$ | nitrogen isotope ratio |
| OCC | organochlorine compound |
| OCS | octachlorostyrene |
| P | vapour pressure |
| PBA | polybromoanisoles |
| PBB | polybromobiphenyls |
| PBDE | polybromodiphenyl ethers |
| PCA | polychloroanisoles (compounds) or principal component analysis (statistical treatment) |
| PCB | polychlorobiphenyls |
| PCBA | polychlorobiphenyl anisoles |
| PCBOH | polychlorobiphenylols |
| PCDD | polychlorodibenzo-*p*-dioxins |
| PCDE | polychlorodiphenyl ethers |

| | |
|---|---|
| PCDF | polychloridibenzofurans |
| PCC | toxaphene (polychlorinated camphene, TOX) |
| PCN | polychloronaphthalenes |
| PCPA | polychlorophenoxyanisoles |
| PCT | polychloroterphenyls |
| PCV | polychloroveratroles |
| PeCP | pentachlorophenol |
| POP | persistent organic pollutant |
| QSAR | quantitative structure-activity relationship |
| RPCBB | alkyl polychlorobibenzyls |
| RPCFL | alkyl polychlorofluorenes |
| RPCN | alkyl polychloronaphthalenes |
| RPCPH | alkyl polychlorophenanthrenes |
| S | solubility in water |
| SCHL | sum of chlordane residues (CHL) |
| SDDT | DDE + DDD + DDD |
| SPCB | total PCB content |
| TBT | tributyltin |
| TBTO | bis(tributyltin) oxide |
| TCDD | 2,3,7,8-tetrachlorodibenzo-*p*-dioxin |
| 345TCG | 3,4,5-trichloroguaiacol |
| 246TCP | 2,4,6-trichlorophenol |
| TeCG | tetrachloroguaiacol |
| TeCC | tetrachlorocatechol |
| TeCP | 2,3,4,6-tetrachlorophenol |
| TEF | toxic equivalency factor (potency compared to TCDD) |
| TEQ | toxic equivalent (concentration or load compared to TCDD) |
| TML | tetramethyl lead |
| TotHg | total mercury content |
| TOX | toxaphene (PCC) |
| TPT | triphenyltin |
| YSM | yolk sac mortality (of juvenile fish) |

## 1 Introduction

Man-made chemicals have caused deaths of wildlife populations due to serious dumpings, industrial discharges and accidental spills. In many cases the causal linkage between certain toxic chemical and damage has been obvious. In many other cases epidemic deaths of animal populations or vegetation has been suspected to be caused by an acute exposure to chemicals but not verified. Ecological damage from chronic exposure is even more difficult to explain. Long-term studies on the occurrence of anthropogenic chemicals in the environment, their effect potencies and their monitoring in connection with specific episodes have, however given some specific answers. These results justify the conclusion that some persistent anthropogenic chemicals are causing eco-

logical damage such as the decline of populations through breeding losses or developmental damage as a result of bioaccumulation and biomagnification in the environment.

## 2
## Observed Effects from Bioaccumulation

### 2.1
### Mercury

Mercury is mobilized in the environment mainly from sources related to human activities [1, 2]. Its ecotoxic effects were first observed in Sweden during the period 1948–65, where excess mortality of seed-eating birds was found to be caused by methyl mercury used as seed dressing fungicide [3, 4]. Serious human mass poisonings from seafood in the early 1950s in Minamata and the early 1960s in Niigata were due to alkylmercury discharged from chemical manufacturing plants being bioaccumulated in fish and crustacea consumed by people [5]. Symptoms, e. g. visual field impairment, unsteadiness, frequent falls, circling movements, convulsions and death, were also observed in cats of Minamata and in swine fed with alkylmercury-dressed grain in the USA [6, 7].

A large human catastrophe (6530 hospitalized, 459 died) took place in the winter 1971–1972 in Iraq, where people ate homemade bread prepared from wheat seed that had been treated with methylmercurial fungicide [6, 7]. A food chain transfer of alkylmercury occurred in Mexico in 1969, where a farmer and five of his neighbours fed treated grain to hogs and then ate the contaminated pork. The pigs died or developed blindness, and several family members suffered methylmercury poisoning [6, 7].

In the environment mercury circulates as vaporized element $Hg^0$, as inorganic mercury salts (mainly $HgCl_2$), as dimethyl mercury ($H_3C$-Hg-$CH_3$) and as monomethyl mercury (Cl–Hg–$CH_3$) [2]. The latter organic mercury compounds are significantly more toxic than elemental or inorganic mercury. While inorganic mercury is methylated by microbia in terrestrial and aquatic solids to the persistent, lipophilic methylmercury, which is bioaccumulating at a high rate and being enriched in the food chain, serious risk of long term damage to humans and wildlife is expected from mercury releases. The investigations carried out during 1965–1975 showed that enriched methyl mercury concentrations in fish were widespread on a global scale [2, 4]. In addition to local industrial discharges, dredging and especially reservoir construction mobilized mercury which then bioaccumulated and biomagnified in aquatic species [6, 8]. In La Grande 2 reservoir, mercury levels as high as 3000 ng $g^{-1}$ fw were measured in fish [9]. Further bioaccumulation of mercury in reservoir areas was considered to form a threat to loons, bald eagles, ospreys and other predator species [10].

Humans, cats and birds are sensitive to mercury poisoning but fish are not. The lifetime of methyl mercury in fish (400–1000 days) is significantly longer than in mammals (in humans 70–76 days, small mammals only few days) [6, 11]. Mercury in fish is more than 90% in methylated form. Accordingly, the

most important environmental mercury hazard is the bioaccumulated methyl mercury in seafood eaten by humans and animals. There are numerous examples of elevated mercury levels in high fish consumers, but not many reported toxic symptoms in these populations [6].

Wild mammals collect mercury via their food: in southern Ontario in 1973–74 fischerotter, marten and mink had high (330–710 ng $g^{-1}$ fw) but fox, raccoon and skunk low (50–99 ng $g^{-1}$ fw) total mercury contents in muscles [12]. This corresponds well with the different feeding habits of species: more fish-eating animals get higher mercury contamination. Any toxic effects from mercury were not reported in mammals in rural areas. Near contamination sources, however, small mammals have shown developmental disturbances (genetic aberrations and asymmetry) thought to be due to elevated mercury levels [13]. Marine mammals at the top of the aquatic food chain could be expected to accumulate harmful levels of mercury. Like some terrestrial mammals, however, they seem to metabolize organic mercury to inorganic mercury. This adaptation effect prevents toxic consequences and is perhaps the result of evolution during long periods of geological time [14]. A review of Hg accumulation in organs of wild terrestrial mammals in relation to dietary habits, sex and age was published in 1986 [15].

## 2.2 Other Heavy Metals

Besides mercury, lead (Pb), cadmium (Cd) and tin (Sn) are the most hazardous heavy metals which can bioaccumulate to toxic levels [1, 6, 16]. Industrially manufactured organolead and organotin compounds are emitted from traffic and other technical uses, and can be serious bioaccumulating ecotoxicants. Both organic and inorganic lead compounds are bioaccumulating. Microbial biomethylation takes place in the environment with lead and tin, but not with cadmium [1, 16].

Lead is converted in nature to tetramethyl lead (TML) which is bioavailable to such a degree that 10–24% of the total lead content in fish muscle consists of TML [1, 6]. Accordingly, environmental hazard from TML is not as great as from methyl mercury [6]. Bioaccumulation of lead compounds has caused human sickness and ecologial damage. Aqueous emissions of alkyl lead has been a source of mass mortality for water birds [17–19], and ingestion of lead shot has been connected to increased avian mortality [20, 21]. Reduction of the enzyme delta-ALAD has been observed as a biomarker of lead intoxication [22]. Wood ducks near mining and smelting sites collected lead up to 8 µg $g^{-1}$ levels in their blood and 14 µg $g^{-1}$ in their livers. The Pb concentrations correlated negatively with ALAD and with nesting success, showing population damage as a result of lead bioaccumulation [23]. Lead shot has also contaminated soil in shooting ranges. Transformation of pellets to bioavailable forms of lead has been shown to cause high concentrations of lead and toxic effects in exposed small mammals [24, 25]. In the aquatic ecosystem, significant bioaccumulation of lead causing adverse effects seems to take place only near point sources of heavy lead pollution [26]. Restriction of the use of leaded gasoline and changing to other

metals for shot has already greatly reduced the overall environmental hazard from lead pollution.

The toxic threat from bioaccumulating cadmium was demonstrated by human "itai-itai" disease in Japan 1947. Industrial discharge of cadmium in the Jintsu River area was exposing humans fatally (more than 100 deaths) via contaminated drinking water [1, 6, 20]. Cadmium has both acute and long-term toxicity to mammals because it is not eliminated but instead accumulates in the liver, kidneys and bones [20]. Inorganic cadmium accumulates in biota because it binds tightly to sulfur-containing proteins such as metallothionein [27]. Bioconcentration factors for cadmium from water to some insects, snails and amphipods are as high as 90 000 [28]. Cadmium-metallothionein is stored in hepatopancreas of crustaceans: extraordinarly high Cd concentrations were measured in hepatopancreas and green glands of lobsters near a lead smelter [29]. Record high levels measured for Cd were in scallops, being 200–500 μg $g^{-1}$ fw in whole organisms and 2000 μg $g^{-1}$ dw in hepatopancreas [20, 30]. According to present literature, ecotoxic effects of bioaccumulated cadmium are local incidences and not of global concern.

Organic tin compounds used in stabilizers, pesticides and marine antifouling paints cause local or regional ecological problems [6]. Inorganic tin is biomethylated similar to mercury and lead [16, 31]. Bioconcentration factors (BCF) from water to fish were 1800 for bis(tributyltin) oxide (TBTO) [32]. Tributyltin (TBT) and triphenyltin (TPT) compounds had BCF of 50–600 to fish muscle and up to 5000 to liver and kidney [33]. In a marina contaminated with organotin compounds, BCF values of 5000–60 000 from water to blue mussels were measured. In this field study, the half life time for depuration of organic and total Sn were 40 and 25 days, respectively [34]. Bioaccumulation of TBT from sediments to deposit-feeding clams up to toxic levels has been observed [35]. Bioaccumulated tin causes shell-thickening in oysters [36], and sterility in juvenile and imposex (the growth of a penis and vas deferens in females) in adult dog-whelks [37]. Neurotoxic influence of TBTO bioaccumulation in fish has been indicated [38]. Algae seem to be able to collect relatively high amounts of TBT, but also degradate it to less toxic dibutyltin (DBT) and monobutyltin (MBT) compounds [39]. Accumulation of TBT and TPT in red sea bream was observed to take place more by direct uptake from water and less by dietary intake. Compared to PCB and methyl mercury, assimilation efficiency and the percentage retention of organotin compounds were low [40].

### 2.3 Other Metals

Aluminium, iron, zinc, chromium and copper are common metallic elements that are strongly bioaccumulating in their salt form. Their increased release from soil due to acid precipitation has toxic effects on plants and aquatic organisms in lakes [6, 16]. However, biomagnification of these elements is, in general, not significant. Therefore, their long-term effects in the environment are less likely to be connected to bioaccumulation than those of persistent lipophilic pollutants. However, aluminium is of great public concern because of its impli-

cation in several human disorders such as Alzheimer's disease and senile dementia [41]. These chronic effects support the irreversible accumulation of aluminium in certain tissues.

## 2.4 Organochlorine Compounds

Very much higher amounts of organohalogen compounds are produced in nature than manufactured, used and discharged in human activities [41–43]. But the anthropogenic organohalogens, especially organochlorine compounds (OCC), are responsible for all wide scale ecological damage associated with bioaccumulation of organic halogen compounds. OCCs of human origin often occur locally at high concentrations, while natural OCCs are diluted in the terrestrial and aquatic compartments. Human originated OCCs enter the natural environment in accidents, but more commonly from industrial discharge, pesticide and preservative usage, urban waste and especially from chlorination and combustion processes [44]. In general, covalently bound chlorine increases persistency and lipophilicity of an organic molecule, and thus enhances bioaccumulation and biomagnification to toxic levels.

The harmful ecological effects of organochlorine compounds were first shown by the decline in certain bird populations in areas where organochlorine pesticides, especially DDT, were heavily used, as Rachel Carson revealed in her book *Silent Spring* in 1962. A scientific explanation for this decrease in reproduction was found in the eggshell thinning effect of DDE, which is the major persistent metabolite of DDT [45]. In addition to DDT and its metabolites, persistent residues of many organochlorine pesticides have been accumulated in food chains globally [44]. They include aldrin, chlordane, lindane, heptachlor, dieldrin (persistent metabolite of aldrin), toxaphene and mirex [46]. Their bioaccumulative potential and various observed acute and chronic toxic effects on animals including estrogenity and teratogenity has led to the banning or severe restriction of their use in both industrialized and developing countries [47].

Hexachlorobenzene (HCBz) occurs in the environment in amounts that are orders of magnitude higher than its production for fungicidal usage and technical fluids [6]. Therefore, discharges as unwanted by-product and combustion products are major sources of environmental HCBz [48, 49]. Acute toxicity of HCBz is small, but its chronic effect causing hepatic porphyria in mammals is severe. Consuming treated seeds caused an epidemic of HCBz-induced porphyria cutanea tarda in Turkey from 1955 to 1959 involving 3000–5000 people with a mortality of 10% [48]. Biomagnification rate of HCBz in the aquatic-terrestrial food chain is similar to that of DDE [6].

Polychlorinated biphenyls (PCB) as environmental contaminants were first found in Baltic seals and fish [50]. PCBs are industrial products, about one millon tons being manufactured from 1929 to 1987 for use in electrical equipment, in closed power and heat transfer systems, as plasticizers, binders, paint, copy-paper additives, adhesives etc. [6]. Their extreme persistency and lipophilicity caused their biomagnification to high levels, which were associated with cases of damage to the reproduction of sea-lions in California [51], seals [52] and

mink [53] in the Baltic sea, area and birds in the Lower Great Lakes of North America [54]. The effects on embryos and juveniles of wild birds were the same as chick oedema disease which killed millions of broilers in the USA in 1957 due to contamination of their food by leaked PCB used as a heat transfer liquid [55]. Later, the symptoms were associated with pyrolysis products of PCBs [56–58], as were the human catastrophes in Japan (1968) and Taiwan (1979), where people consumed rice-oil which was contaminated with heated PCB [44].

The extremely toxic TCDD (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) and related compounds first became known as the source of chloracne in industrial workers exposed during chlorine and chlorophenol production [59, 60]. After several documented occupational mass poisonings in industry manufacturing chlorophenols, polychlorodibenzo-*p*-dioxins (PCDD) and polychlorodibenzofurans (PCDF) were also found in emissions of thermal processes, industrial discharges, pesticides and preservatives [61]. They are widespread in the environment and have a high bioaccumulation power [62]. Dioxins have been included in notorious incidents. Herbicide 245-T used extensively as a leaf-dropping agent in the Vietnam war contained tens of ppm of TCDD. Accidental release of TCDD from production of 2,4,5-trichlorophenol to the environment followed by mass deaths of animals and contamination of people in Seveso, Italy, 1976 triggered great public concern about dioxins [63]. Chick oedema disease [55], Yusho oil poisoning in Japan and Yu Cheng disease in Taiwan from heated PCB contamination was found to be due to toxic PCDFs formed in the pyrolysis of PCBs [56–58]. PCDFs were most probably formed from ortho-OH substituted PCBs formed as major products during pyrolysis in the presence of oxygen [58].

The toxic effects of PCDDs and PCDFs were associated with their structure-related metabolism. Compounds having chlorine in lateral positions 2,3,7,8 of the dioxin or furan molecule (seven PCDDs and ten PCDFs out of a total of 210 structures possible; see Fig. 1) were found to fit closely to cytosolic receptors (Ah) of mixed function oxydase (MFO) enzymes which oxidize xenobiotics such as PAHs [64]. Because the substrates of MFO also include steroid hormones, certain vitamins, fatty acids and bile acids [65], the induction of MFO enzymes by dioxins and related compounds is associated with their long term toxic impact on both wildlife and humans [66, 67]. Binding of PCDDs and PCDFs to Ah receptors leads to hepatic MFO induction (e.g. AHH or EROD) which parallels dermal toxicity, thymic atrophy, reproductive effects, teratogenity, hepatoxicity and carcinogenity [63, 68]. However, hormone-like behaviour of TCDD and other dioxins is also demonstrated by their anti-tumour-promoting activity. Consequently, they are studied as potential cancer drugs [69].

Use of MFO induction potency as a measure of dioxin-like toxicity [70] is confused by the fact that many planar aromatic and heteroaromatic naturally formed compounds are also strongly bound to Ah receptors and potent MFO inducers [71]. However, toxic potency as equivalency factors (TEF) related to TCDD is generally used in emission control and toxic load estimation of dioxins and related compounds. TEFs are based on MFO-inductions, immunotoxicity and other biological response measurements and evaluated by expert groups. For each chemical in sample (food, tissue or emission) is calculated a toxic load

value TEQ = TEF × concentration. Total TEQ load, based on the assumption that the effects are additive, is calculated as the sum of the TEQs of each compound [72, 73].

Structural similarity with toxic PCDDs and PCDFs explains the same MFO-induction and toxic effects of certain PCBs and TCDD. The structures of dioxin-like toxic PCDDs, PCDFs and PCBs are illustrated in Fig. 1. When chlorine substitution in the PCB molecule is in meta (3,3′,5,5′) and para (4,4′) positions, the molecule seeks its lowest energy configuration in the plane [74, 75]. The non-ortho chlorine substituted "coplanar" PCBs, such as 3,3′,4,4′-tetrachlorobiphenyl (PCB77), 3,3′,4,4′,5-pentachlorobiphenyl (PCB126) and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB169) are the most toxic of the PCB congeners [74–77]. Mono-ortho coplanar PCBs, such as 2,3′,3′,4,4′-pentachlorobiphenyl (PCB105), 2,3′,4,4′,5-pentachlorobiphenyl (PCB118) and 2,3,3′,4,4′,5-hexachlorobiphenyl (PCB156) are also MFO inducers and could be taken into TEQ evaluations. The TEF approach can been extended to bromo analogues of PCDDs, PCDFs and PCBs, and also to polychlorodiphenyl ethers (PCDE) [77, 78]. The TEFs for PCDDs, PCDFs and PCBs have been evaluated internationally by toxicology expert groups [79, 80]. A bioaccumulation estimate for PCDDs, PCDFs and PCBs from Baltic wildlife analysis results [81–83] as TEQ loads is illustrated in Fig. 2.

Further bioaccumulating OCCs which are suspected, but less frequently observed in the field, to have harmful effects in ecosystems are polychloronaphthalenes (PCN) [84–87], polychloroterphenyls (PCT) [87–92], octachlorostyrene (OCS) [87, 93–98] and hexachlorobutadiene (HCBD) [96–100]. The major persistent and bioaccumulating OCCs discharged from bleaching of pulp or from chlorodisinfection of water are alkylaromatic chlorohydrocarbons – chlorocymenes (CYMS), chlorocymenenes (CYMD), alkyl polychlorobibenzyls

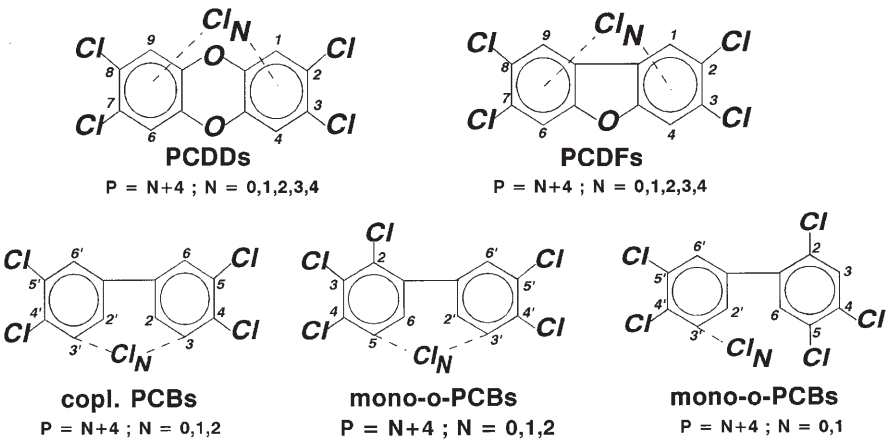


**Fig. 1.** Structures of the most toxic polychlorodibenzo-*p*-dioxins (PCDDs; seven compounds), polychlorodibenzofurans (PCDFs; ten compounds) and polychlorobiphenyls (three coplanar and and six mono-ortho-substituted PCBs)

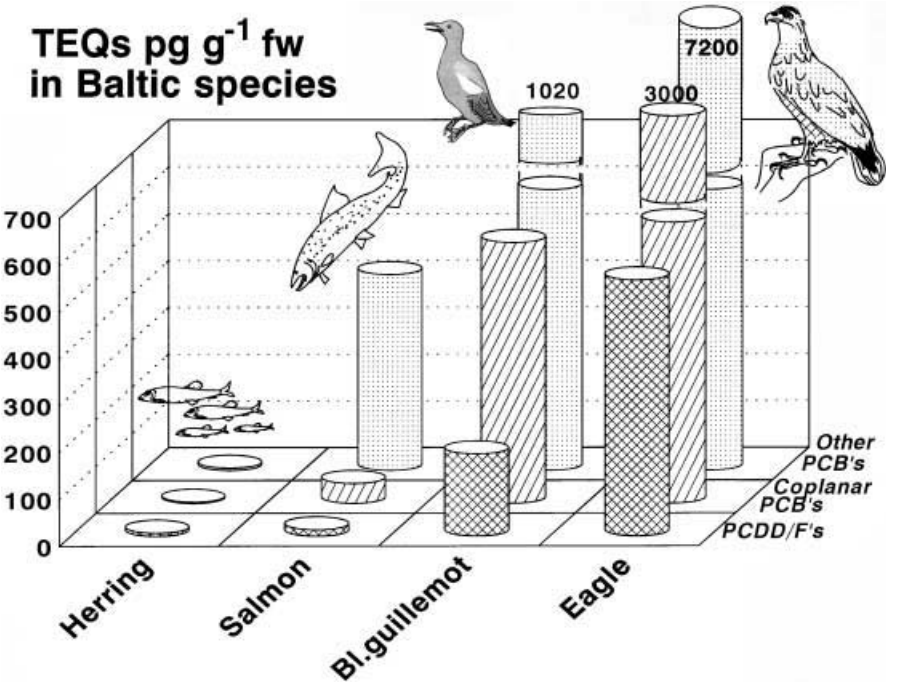


**Fig. 2.** Average contents of PCDDs, PCDFs and PCBs as toxic TCDD equivalents (TEQs) in Baltic sea animals in the 1980s [81–83]

(RPCBB), alkyl polychlorophenanthrenes (RPCPH), alkyl polychloronaphthalenes (RPCN) and alkyl polychlorofluorenes (RPCFL). Their bioaccumulation potency has been shown but their toxic effects have not been much studied to date [101–114].

In addition to PCDDs and PCDFs, some other groups of aromatic chloroethers (ACE) are of environmental concern [115, 116]. Biomethylation products of chlorophenols, chlorocatechols and chloroguaiacols – chloroanisoles (PCA) and chloroveratroles (PCV) – are well known as extremely potent off-flavours [117–119]. Although the observed tainting effects of PCAs and PCVs are local incidences from point source discharges, PCAs occur as globally distributed pollutants [120].

Major phenolic impurities of chlorophenol products, polychlorinated phenoxyphenols (PCPP) [121–125], transfer in the environment to their biomethylation products, polychlorophenoxyanisoles (PCPA) [126], which have been detected in Baltic wildlife and fish liver oil [115, 127, 128]. PCPAs also occur as minor neutral impurities in tetrachlorophenol preservative Ky-5 made by chlorination of phenol [129].

Other phenolic impurities in chlorophenol formulations are polychlorobiphenylols (PCBOH) [123] which are metabolites of PCBs [130] and also major products of the air-pyrolysis of PCBs [58]. Biomethylation products of PCBOHs

and further metabolites of PCBs, polychlorobiphenyl anisoles (PCBA) [130], have been identified in fish liver oil and in Baltic whitetailed eagles [115, 127]. Polychlorodiphenyl ethers (PCDE) are the most abundant neutral impurities of technical chlorophenol formulations [121, 123]. The main PCDE congeners in Ky-5 were identified via model substance syntheses by Humppi [124, 129]. Additionally, a number of highly chlorinated PCDEs were synthesized and studied by Nevalainen [131–133] and Kurz [134] with their coworkers. PCDEs are bioaccumulating and enriched in biosphere as are PCBs, although their background levels are lower [82, 83, 115, 116, 134–137].

From the bromo-analogues of OCCs, bromoanisoles (PBA) occur and bioaccumulate in the marine environment [138, 139]. Bromoanisoles and the corresponding chloroanisoles, at least the 2,4,6-trisubstituted ones, can be, in great part, products of natural halogenation of anisoles or phenols [43, 140]. Fire retardant mixture of polybrominated biphenyls (PBB) seriously contaminated livestock and humans in an accidental poisoning of animal food in Michigan [141, 142]. Another fire retardant group of polybromodiphenyl ethers (PBDE) has caused widespread pollution of aquatic wildlife [143]. The concentrations of PBDEs in cod liver were highest in the southern and lowest in the northern North Sea and decreased over the time 1977–87, showing a point source pollution which had a decreasing trend [98].

## 3
## Predicted and Observed Bioaccumulation in the Environment

### 3.1
### Exposure Models

To assess the potential exposure of humans and the environment to chemical substances, mathematical modelling is needed to reduce the need of time-consuming and expensive analyses from field samples [144–146]. The models handle the major environmental processes in compartments of air, water, solids and plants. In soil/plant systems leaching, run-off and plant uptake, and in aquatic systems bioaccumulation are the major pathways of chemicals leading to contamination of food and drinking water of higher animals and humans [145, 147]. Steady state multimedia mass balance models are most popular for estimating exposure of biota in different compartments, starting from known emissions [146].

The environmental fate of the discharged chemical is predicted with modelling from the known properties of the compound and verified by analyses in the environment [146, 147]. A preliminary fate modelling only needs a limited number of properties of the chemical: molecular mass, water solubility (S), vapour pressure (P) in environmental temperatures, and bioconcentration factors (BCF) and rates of hydrolysis ($k_H$), photodegradation ($k_P$) and biodegradation ($k_B$) in the compartments (air, water, soil/sediment) of the model environment. Instead of expensive testing [148], some of these properties can be roughly estimated from readily available properties [149] such as octanol-water partition coefficient (S and BCF from Kow) or boiling point (P from Bp).

Degradation rates must be measured in the environment or under similar laboratory conditions. For the series of similar compounds, however, evaluation of the rate constants can also be done by semiempirical calculations from accumulated data using quantitative structure-activity relationships (QSAR) [149, 150].

The multimedia models include the main processes in the environment which influence the fate of the chemical. Transport processes determine the distribution, and transformation processes the persistence of the chemical in the environment. The main transport processes incorporated in models are 1) advection which transports the chemical in dissolved, gaseous, condensed or particulate phases, 2) dispersion as a result of turbulence and molecular diffusion, 3) volatilization determining air-water and soil-air transfers, 4) adsorption on soils and sediments, 5) bioaccumulation, 6) water phase heterogenous transport, particle settling, resuspension, sedimentation and sediment mixing, and 7) air phase heterogenous transport by wet and dry deposition. The main transformation phenomena modeled are 1) biodegradation, 2) hydrolysis, 3) phototransformation, and 4) speciation by dissociation to charged species and complex formation [151].

The multimedia models can be classified into four different levels [146, 147, 151]. In the level I model, equilibrium is assumed, and transformation of the chemical is excluded. Output consists of the relative concentrations (equilibrium distribution) of the substance in compartments of an environment. The compartments are air, water, soil, sediment, suspended sediment and fish (biota).

In the level II model, equilibrium is also assumed, but transformation and advection are taken into account. In addition to the steady-state concentrations, reaction and advection rates and residence times are obtained. The concentrations obtained are arbitrary, calculated from assumed total emission rate, but their ratios are characteristic to the environment and compound chosen.

The level III model gives similar output as level II, but with greater precision and in non-equilibrium conditions. Estimates of chemical quantities, concentrations and lifetimes in four compartments (air, water, soil and sediment) are obtained. Concentration in fish is given (as in Level II) only based on partition between biota (lipid) and water.

Level IV models assume non-steady state. They predict the time needed for the chemical to reach steady state when the releases are changed [146, 151].

In addition to multimedia models, a number of models for fate of chemicals with reduced numbers of compartments, like models for air, water/rivers/watercourses, soil/groundwater, air/plants, soil/plants etc., are widely used [145, 151]. Normally these models handle real environments as do multimedia models. The latter, however, also use hypothetical “generic” environments for preliminary estimation of the environmental hazard potential of a chemical [146]. Microcomputer ”toolbox” CemoS [152, 153] is an integrated system used to simulate distribution of a chemical substance from continuous releases to air, water and soil from both diffuse and point sources in multimedia environment systems by transport to plants and by movement in the food chain. CemoS consists of the following nine models:

| | |
|---|---|
| AIR | a one-dimensional box model for boundary surface releases to air |
| BUCKETS | a scoop chain model for transport in soil |
| CHAIN | a model for the food chain with three trophic levels |
| LEVEL1 | a multimedia model (Mackay level 1) for equilibrium distribution assuming that the chemical is fully persistent |
| LEVEL2 | a multimedia model (Mackay level 2) for steady state equilibrium distribution including advections and transformations |
| PLANT | a box model for uptake in plants |
| PLUME | a three-dimensional steady state model for point sources to air |
| SOIL | a one-dimensional model for vertical transport in soil |
| WATER | a one-dimensional stationary state box model for point sources in flowing waters |

Bioaccumulation is estimated from the modeled concentration of chemical in air, water or solids depending on habitat of the exposed biota. Bioaccumulation models [145, 146, 154, 155] normally predict the concentration in the first trophic level, in producers (phytoplankton, plants), by the bioconcentration factor (BCF) which can be estimated as a function of Kow (bioconcentration by lipid/media partitioning). In aquatic systems, most simply: BCF = Lipid fraction × Kow; Concentration in producer = BCF × Concentration in water. Also, BCF for uptake of chemical by plants from air to leaves/needles and from soil to roots is linearly dependent on lipid content in plant tissue [145]. Bioaccumulation to higher trophic levels (herbivores, carnivores) takes place not only by partitioning, but also by biomagnification uptake via food and elimination by excretion and metabolism [154, 155]. Concentration of the chemical is also decreased due to the dilution effect from the growth of the animal [154]. All these processes can be successfully included in programs of the predictive environmental fate models [146].

### 3.2
### Model Results Compared with Environmental Levels

A simple prediction of environmental fate can be made by a multimedia fugacity model for a hypothetical unit environment of 1 $km^2$ area [156]. An example of application is prediction of the levels of common organic pollutants HCH, 2,3,4,6-tetrachlorophenol (TeCP), DDT and chlordanes (CHL, sum of chlordane residues, *cis*-chlordane as representative molecule) in Bay of Bothnia fish [157]. The results are listed in Table 1. In level I distribution the importance of water and air media in distribution of HCH and dominance of solids for the others is clearly seen. Level II calculation, including bioconcentration, gave residence times and relative concentrations which could be compared with those observed.

The results give a rough approximation of the exposure of biota as a basis of environmental hazard from bioaccumulation. Deviation of the actual levels indicates that the very lipophilic substances DDT and CHL are accumulated in fish not only by lipid/water partitioning, but also via food. More accurate estimation of concentrations in fish can be obtained from predicted levels in water and suspended solids by biomagnification models including uptake by food

**Table 1.** Level I and II modelling results for organochlorines in Bay of Bothnia environment [157]

| | HCH | TeCP | DDT | CHL |
|---|---|---|---|---|
| Level I | | | | |
| % in Air | 30.4 | 8.88 | 0.45 | 9.72 |
| % in Water | 47.9 | 18.1 | 0.46 | 3.06 |
| % in Sediment | 21.6 | 73.0 | 99.1 | 87.2 |
| Level II | | | | |
| Assumed daily input to $km^2$ (kg) | 0.868 | 2.00 | 0.042 | 0.050 |
| Residence time (days) | 37 | 5 | 7233 | 395 |
| Predicted concentration in fish (mg $l^{-1}$) | 0.203 | 0.232 | 6.08 | 0.341 |
| Observed level in salmon 1982–85 (ng $g^{-1}$ lw) | 4.7 | 5.0 | 436 | 27.4 |
| Relative predicted concentration | 1 | 1.14 | 30 | 1.7 |
| Normalized observed level | 1 | 1.06 | 93 | 5.8 |

and elimination processes, e.g. by those of Thomann [154] or Clark et al. [155, 158].

A further modelling of the fate of lindane (LIND = $\gamma$-HCH), chlordanes (CHL as above) and toxaphene (TOX or PCC) was performed using the program FATEMOD [147]. It is a modification of the Mackay GENERIC program [146], which contains estimations at levels I, II and III.

The environments were the boreal Bay of Bothnia and a fictious Southern Sea which has sizes and fluxes the same but average temperature (25 instead of 2 C) and organic carbon fractions different (significantly lower) compared to the Bay of Bothnia. The values of emissions for level III estimation were derived by trial modelling to give approximately the same concentrations as observed in air, water or fish at the Bay of Bothnia [159–162]. Then, the same emissions were used in modelling the Southern Sea case. Some modelling results are shown in Table 2. According to the model, bioaccumulation of LIND and CHL was slightly higher and that of TOX about the same in the Bay of Bothnia compared to the "Southern Sea" [147].

Two specific models for estimation of the fate of discharged chemicals in watercourses were tried in a pulp mill recipient in Äänekoski, Central Finland [147]. The model EXWAT was developed for the characterization of the transport and fate of a chemical in surface water bodies at steady state [163, 164]. It is a box model with two compartments: fluid and sediment. The processes considered were 1) deposition and resuspension of suspended matter, 2) partitioning of chemicals between water and suspended matter in the fluid and between pore water and benthic sediment solids, 3) ionization equilibrium, 4) exchange between pore and fluid water as driven by dispersion, 5) sediment burial, 6) volatilization, 7) degradation, and 8) bioconcentration. PPEFF model is a three-segment version of the Quantitative Water-Air-Soil-Interaction (QWASI) fugacity model [146, 165].

Both EXWAT and PPEFF models could be readily applied to the Äänekoski watercourse. For EXWAT, the 18-km long region downstreams from the discharge point can be divided into five segments each containing 1-km long bo-

**Table 2.** FATEMOD results for Bay of Bothnia compared with similar more southern area and observed average levels in Bay of Bothnia. Ca, Cw, Csed and Cb are the concentrations in air, water, sediment and biota (fish), respectively [147]

| Area | Bay of Bothnia | | | Southern Sea | | |
|---|---|---|---|---|---|---|
| Compound | LIND | CHL | TOX | LIND | CHL | TOX |
| Level I | | | | | | |
| % in Air | 1.75 | 0.003 | 0.0005 | 1.75 | 8.64 | 1.60 |
| % in Water | 87.6 | 6.15 | 2.03 | 87.6 | 14.13 | 4.95 |
| % in Sediment | 10.4 | 91.6 | 95.7 | 10.4 | 84.3 | 93.4 |
| Level II | | | | | | |
| Res.time h | 509 | 1274 | 4669 | 152 | 338 | 1264 |
| Level III | | | | | | |
| Emission kg $h^{-1}$ | | | | | | |
| to air | 0.055 | 0.10 | 0.005 | 0.055 | 0.10 | 0.005 |
| to water | 4.00 | 0.36 | 1.355 | 4.00 | 0.36 | 1.335 |
| Ca pg $m^{-3}$ | 29.4 | 64 | 3.2 | 66.7 | 93.0 | 12.5 |
| Cw ng $L^{-1}$ | 2.3 | 0.25 | 0.65 | 0.84 | 0.15 | 0.63 |
| Csed ng $g^{-1}$ fw[a] | 0.010 | 0.033 | 0.78 | 0.00043 | 0.015 | 0.073 |
| Cb ng $g^{-1}$ fw | 0.72 | 3.4 | 21.6 | 0.27 | 2.2 | 26.7 |
| Observed | | | | | | |
| Ca [159] | 30 | 3 | | | | |
| Cw [160] | 1.5 – 2.3 | | | | | |
| Cb Salmon [b] | 1.72 | 34.3 | 134.8 | | | |
| Cb Trout [b] | 0.76 | 3.12 | 21.5 | | | |

[a] Conc. in fresh sediment; Csed dw is approximated by division by 0.37.
[b] From Paasivirta and Rantio [161] and Paasivirta et al. [162].

xes. The model gives concentrations of the chemical in each of the 18 boxes and in each compartment (water, suspended solids, sediments and biota). In the PPEFF run, a three-box version (Lake Kuhnamo, River Kapeenkoski and Lake Vatia) could be applied.

Three pulp-mill originated chlorophenolics, 2,4,6,-trichlorophenol (246TCP), 3,4,5-trichloroguaiacol (345TCG) and tetrachlorogiaiacol (TeCG) were modelled for two time periods – August 1986 and March 1987 – when their concentrations in discharge and in environmental samples had been intensively analyzed. Examples of the modelled and observed data are presented inTable 3. Assuming that other necessary environment and compound parameters for model were reasonably true independent data, degradation rates were to be fitted by the model to give the best agreement of measured and modelled concentration in water of all 18 boxes.

Model predictions of the concentrations in fish were in fair agreement with observed levels in pike (Table 3). In the case of EXWAT, this might be a coincidence, because lipid partition should cause lower levels (fat percent in pike muscles is only about 0.5), but this was perhaps compensated by food uptake by this predatory species. In the case of PPEFF, various uptake, growth and metabolism mechanisms are included in the model, and the fish concentration result was selected for the "large piscivores" class.

**Table 3.** Modelled (EXWAT and PPEFF) and observed concentrations in water and in fish (Pike, *Esox lucius*) at the Äänekoski watercourse. Sample places KUH, KAP, VAT and KUU are 2, 7, 15 and 18 km downstream of the discharge. Value of pH of water at all sampling places was 6.5 [147]

| Compound | 246 TCP | | 345 TCG | | TeCG | |
|---|---|---|---|---|---|---|
| Time | Aug 86 | Mar 87 | Aug 86 | Mar 87 | Aug 86 | Mar 87 |
| Temp. °C | 16 | 1 | 16 | 1 | 16 1 | |
| k [a] | 0.063 | 0.058 | 0.087 | 0.08 | 0.077 | 0.070 |
| t1/2 d | 11.0 | 12.0 | 7.97 | 8.66 | 9.00 | 9.90 |
| Discharge g $d^{-1}$ | 227 | 271 | 600 | 718 | 584 | 699 |
| Waterflow $m^{-3}$ $s^{-1}$ | 60 | 38 | 60 | 38 | 60 38 | |
| **Conc. in water µg $l^{-1}$** | | | | | | |
| KUH EXWAT | .035 | .062 | .093 | .162 | .057 | .090 |
| *KUH obs.* | *.038* | *.100* | *.085* | *.362* | *.091* | *.104* |
| KUH PPEFF | .044 | .084 | | | | |
| KAP EXWAT | .024 | .036 | .062 | .093 | .019 | .021 |
| *KAP obs* | *.014* | *.051* | *.026* | *.050* | *.010* | *.043* |
| VAT EXWAT | .016 | .021 | .039 | .048 | .007 | .006 |
| *VAT obs* | *.019* | *.012* | *.036* | *.057* | *.010* | *.013* |
| KUU EXWAT | .014 | .019 | .034 | .043 | .007 | .006 |
| *KUU obs* | *.017* | *.008* | *.036* | *.026* | *.010* | *.008* |
| KUU PPEFF | .035 | .051 | | | | |
| **Conc. in fish ng $g^{-1}$** | | | | | | |
| KUH EXWAT | 3.75 | 5.61 | 6.9 | 10.2 | 9.2 | 9.7 |
| *KUH obs.* | *6.98* | *4.70* | *8.0* | *9.9* | *2.0* | *3.2* |
| VAT EXWAT | 2.51 | 3.25 | 4.3 | 5.3 | 3.6 | 3.0 |
| *VAT obs.* | *6.99* | *2.52* | *8.0* | *4.0* | *3.0* | *1.5* |
| KUU EXWAT | 2.22 | 2.94 | 3.8 | 4.8 | 3.3 | 2.8 |
| *KUU obs.* | *2.52* | *3.42* | *11.9* | *7.1* | *3.2* | *1.9* |
| KUU PPEFF | 1.48 | 11.0 | | | | |

[a] Degradation rate constant (k $d^{-1}$) in water and solids fitted by the model.

More accurate model estimations of biomagnification are complicated by the dependence of the bioaccumulation process on the lipid/water distribution ratio expressed as log Kow. When log Kow < 5, only partitioning is important. Food chain biomagnification is well predictable for compounds having log Kow 5–7, as shown by comparison of the calculated concentrations in top predators and those observed in the field. When log Kow is > 7, food chain effects are sensitive to the chemical assimilation efficiency and phytoplankton BCF [154].

While the bioconcentration (BCF) factor from the primary producer is fairly well modelled from simple lipid partitioning, the models of biomagnification to higher levels must consider ingestion from food and elimination and dilution by growth mechanisms. One model of food ingestion mechanism assumes that the biomagnification occurs in the organism's tissue after the lipophilic xenobiotic has been transferred there from intestine coassimilated with lipid [166]. However, other laboratory and field studies support an alternative, the fugacity model of Gobas et al. [155, 158], where the intestinal absorption is controlled by

the chemical diffusion of the xenobiotic molecule which is leaving the lipid before transfer through the intestinal wall. This model explains not only biomagnification in fish but the biomagnification process from mammalian mother to baby during breast feeding. For example, the PCB concentrations in mother's and embryo's blood lipid are equal at the birth, but then during lactation period (24 months for humans), PCB in infant blood lipid increases by a factor of two, but in mother's blood lipid decreases to one third [158, 167, 168]. Accordingly, baby is on the higher trophic level related to mother with biomagnification rate for PCB as high as 5.5 [158]. This lactation enrichment model could, in addition to diet and poor metabolism suggested, explain the very high bioaccumulation rates of dieldrin, PCB and DDT residues from low concentrations in water to marine cetaceans [169].

Environmentally hazardous chemicals are not only locally discharged (direct emissions) at the geographical region modelled; their long-range transport must also be considered. In particular, some persistent organic pollutants (POPs) occur at significant levels far away from their sources due to atmospheric transport. Arctic POP pollution, bioaccumulation and food chain enrichment is in great part due to global atmospheric transport [170–180]. The process of transport is successfully explained by the model of Wania and

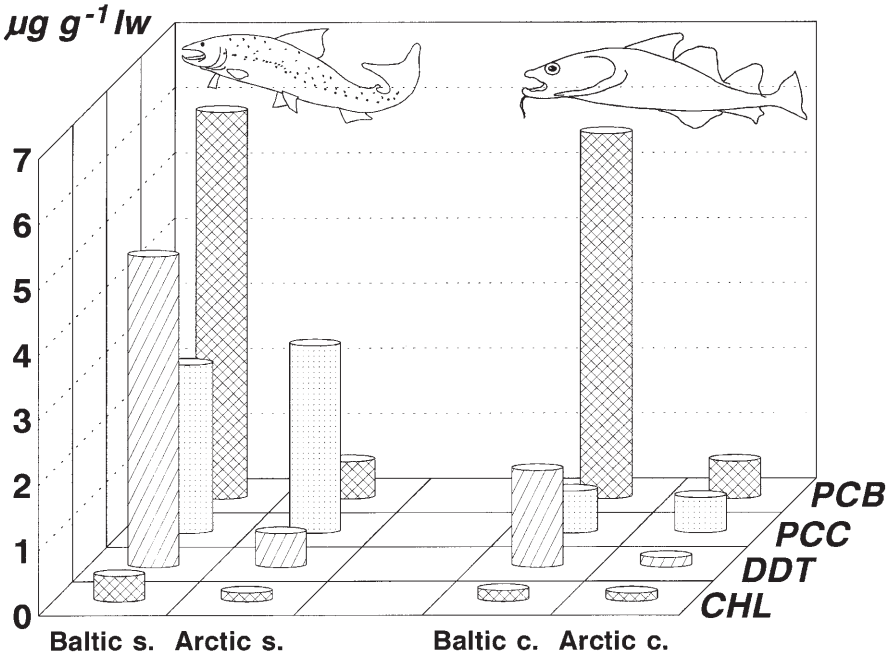


**Fig. 3.** Contents of polychlorobiphenyls (PCB), toxaphene (PCC), DDT residues and chlordane residues (CHL) in lipid of salmon muscle (s.) from Simojoki River (Baltic) and Teno River (Arctic), and in lipid of cod livers (c.) from Gulf of Finland (Baltic) and Vestertana fjord (Arctic) [175]

Mackay which incorporates the theory of global fractionation and cold condensation [181]. The model explains why toxaphene (PCC) residues in the Arctic ecosystem are at the same level as in more temperate areas [175, 182, 183]. Examples of toxaphene and other chlorohydrocarbon concentrations in Baltic and Arctic fish [183] are illustrated in Fig. 3.

## 4
## Case Studies in the Field

### 4.1
### Aquatic-Terrestrial Food Chain Bioaccumulation

Contents of xenobiotics in food chains of the freshwater lakes in Finland were intensively studied in the 1970s and their trends followed in the 1980s [184–190]. The levels of DDT residues and PCB were low, near background, but the mercury levels were elevated due to industrial discharges which had been stopped in 1968. These xenobiotics all biomagnify significantly (Fig. 4).

Concentrations of PCB, SDDT and mercury in adult fish-eating birds were orders of magnitude higher than in local fish [184, 187]. Study of eggs and juveniles of these bird species eliminated the influence of contaminants collected by adult birds [188]. Residues in eggs were an additional burden to the chick, which collected more biocides from food and diluted them by growth.

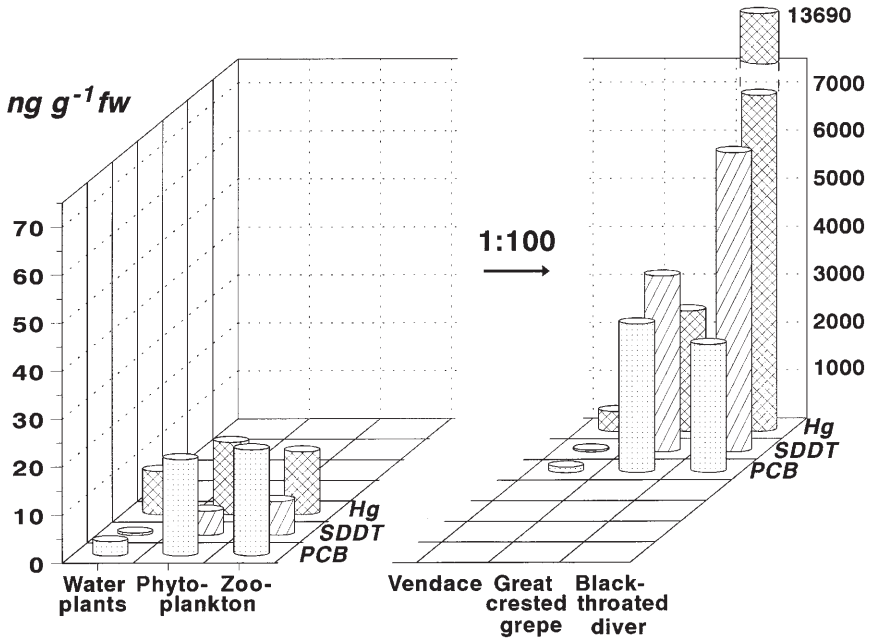


**Fig. 4.** Average concentrations in different trophic levels of the Lake Päijänne, Finland, in 1972–74 [184–187]

Therefore, instead of concentrations in eggs and juveniles, total amounts in specimens gave a better estimate of bioaccumulation of xenobiotics from food (fish) to the chicks (Fig. 5). During the nesting period the amount of pollutant was increasing in juveniles of two species for SDDT, five species for PCB, and for total mercury (TotHg) in all seven species studied. The relative biomagnification power of different compounds, however, could not be obtained from the amount ratios, but one must consider the different contents in food. A relative enrichment factor ($E_{rf}$) based on increase of the amount in juveniles was defined as

$$E_{rf} = \frac{A_{juv} - A_{egg}}{C_{ven}} \quad (1)$$

where $A_{juv}$ = amount in juvenile, $A_{egg}$ = amount in egg, and $C_{ven}$ = concentration in vendace (main food of the chicks) [6, 157].

In cases where biomagnification was observed ($A_{juv} > A_{egg}$), variation in $E_{rf}$ values between species were for mercury 0.1–1.14, for DDE 0.74–4.55 and for PCB 0.33–641. This great variation must be due to different food compositions (in addition to vendace) and metabolism of the species.

The method of comparing total amounts of egg and juvenile specimens has been used in a three-step terrestrial food chain study [191]. From 15 organochlorine compounds studied, concentrations of some PCB congeners, *p,p′*-DDE and hexachlorobenzene, indicated the highest biomagnification rates from oak

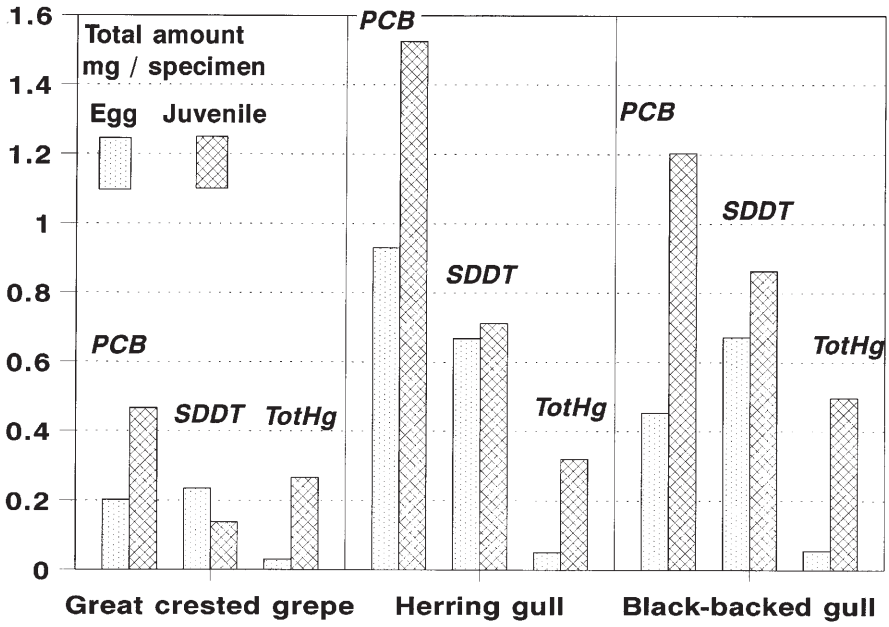


**Fig. 5.** Average total amounts in eggs and juveniles of three species of fish-eating bird at Lake Päijänne, Finland in 1972–74 [188]

leaves to caterpillars and further to birds (great tit specimens). Bird eggs had higher concentrations than juvenile birds. However, the amounts in eggs were lower than those in juveniles for PCB101, PCB138, PCB153 and PCB180 (numbering according to Ballschmiter et al. [192]), and for *p,p′*-DDE. The most abundant biomagnifying organochlorines in this study, PCB153 and PCB138, have been suggested to be estrogenic agents which might be responsible for impaired sperm mobility and, consequently, for the pollution originated nonfertility in the human population [193].

### 4.2
### Empirical Estimates of Biomagnification

Comparison of contents at different trophic levels on a fresh weight basis is the simplest empirical estimate of biomagnification of a xenobiotic. One way to describe the estimate is to show the portions of the compounds as percentages of the combined contents at each trophic level [190, 194].

Comparison of the content distributions (Fig. 6) indicates the most significant biomagnification to tetrachloroguaiacol (TeCG), total mercury (TotHg, mainly methyl mercury) and hexachlorobenzene (HCBz). Biomagnification of 2,4,6-trichlorophenol, PCB, 4,5,6-trichloroguaiacol and DDT residues is also clear: if lipid weight basis had been used, levels in pike would be significantly higher than those in roach. Only 2,3,4,6-tetrachlorophenol, pentachlorophenol (PeCP) and tetrachlorocatechol (TeCC) showed no biomagnification from

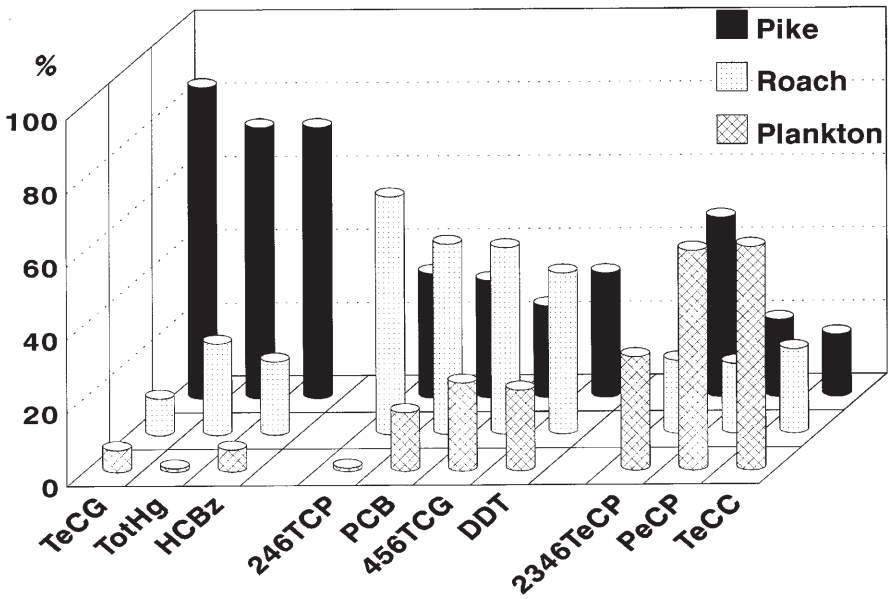


**Fig. 6.** Distributions of the concentrations in three trophic levels in lakes of central Finland [190, 194]

plankton to roach. In the environment, they were frequently buried in sediment [194].

A four trophic level estimation of biomagnification power can be constructed from concentrations of persistent organic pollutants (POP) analyzed in herring, salmon, seals and eagles in Gulf of Finland and Gulf of Bothnia areas of the Baltic Sea in 1985 – 1989 [161, 195, 196]. The species in comparison are not exact representatives of the food chain except that Baltic herring is the main food of salmon. However, the averages of the observed concentrations (Table 4) can be used to rank POPs according their environmental hazard. Comparisons of potential hazard as biomagnification power is illustrated for fresh weight (fw) data in Fig. 7 and for lipid weight (lw) data in Fig. 8.

In the above comparisons the PCB, DDT and chlordane (SCHL) residues show high biomagnification. In addition, biomagnification rates of HCBz are relatively high. The dioxin-like toxic POP congeners showed high biomagnification rates in the order PCB169 > PCB126 > 23478PeCDF > PCB77 > 2378TeCDF. They must be considered as serious candidates to cause ecological damages in Baltic biota. The biomagnification of PCB105, HCHs and toxaphene components (PCC) seemed to be low or negligible according to experience of these four species.

Kubiak et al. measured the bioconcentration factors from spottail shriners to Forsters tern as 0.17, 64 and 176 for PBB77, PCB126 and PCB169, respectively [197]. These ratios have very much the same trend as C(eagle)/C(herring) ratios shown in Fig. 8. The high biomagnification of extremely toxic coplanar PCB126 and PCB169 has been demonstrated to form one of the most significant long-

**Table 4.** Average concentrations ng $g^{-1}$ in Baltic wildlife 1985 – 1989

| Expl. | Herring | | Salmon | | Seal | | Eagle | |
|---|---|---|---|---|---|---|---|---|
| Lipid % | 8.10 | | 3.66 | | 69.8 | | 28.9 | |
| | lw | fw | lw | fw | lw | fw | lw | fw |
| SPCB | 1030 | 83.4 | 4243 | 155 | 254000 | 177292 | 848000 | 245000 |
| SDDT | 770 | 62.4 | 3254 | 119 | 27200 | 19990 | 88000 | 25400 |
| SCHL | 43.9 | 3.56 | 147 | 5.38 | 1100 | 768 | 8620 | 2490 |
| HCBz | 57.0 | 12.4 | 153 | 5.60 | 230 | 161 | 2900 | 838 |
| PCC | 545 | 44.1 | 2058 | 75.3 | 80 | 55.9 | < 10 | < 3 |
| $\alpha$-HCH | 111 | 8.99 | 70 | 2.56 | 90 | 62.8 | < 10 | < 3 |
| $\gamma$-HCH | 71.5 | 5.79 | 37 | 1.35 | 20 | 13.9 | < 10 | < 3 |
| PCB77[a] | 1.55 | .126 | 14.7 | .538 | 3.59 | 2.51 | 246 | 71.1 |
| PCB105[a] | 17.4 | 1.41 | 73.0 | 2.67 | 129 | 90.0 | 140 | 40.5 |
| PCB126[a] | .159 | .0129 | 1.75 | .0641 | 2.31 | 1.61 | 176 | 50.9 |
| PCB169[a] | .014 | .00113 | 0.622 | .0228 | 1.30 | 0.907 | 68.9 | 19.9 |
| 2378TeCDF | .0041 | .000332 | 0.238 | .00871 | .199 | 0.139 | < 1 | < 0.3 |
| 23478PeCDF | .0087 | .000705 | 0.165 | .00604 | .194 | 0.135 | 1.8 | 0.520 |

Herring = *Clupea harengus*; Salmon = *Salmo salar*; Seal = grey seal (*Halichoerus grypus*).
Eagle = whitetailed eagle (*Haliaetus albicilla*).
SPCB = sum of the main PCB congeners; SDDT = *p,p′*-DDT + *p,p′*-DDE +*p,p′*-DDD.
SCHL = sum of the chlordane residues; PCC = toxaphene.
[a] Ballschmiter numbering [192].

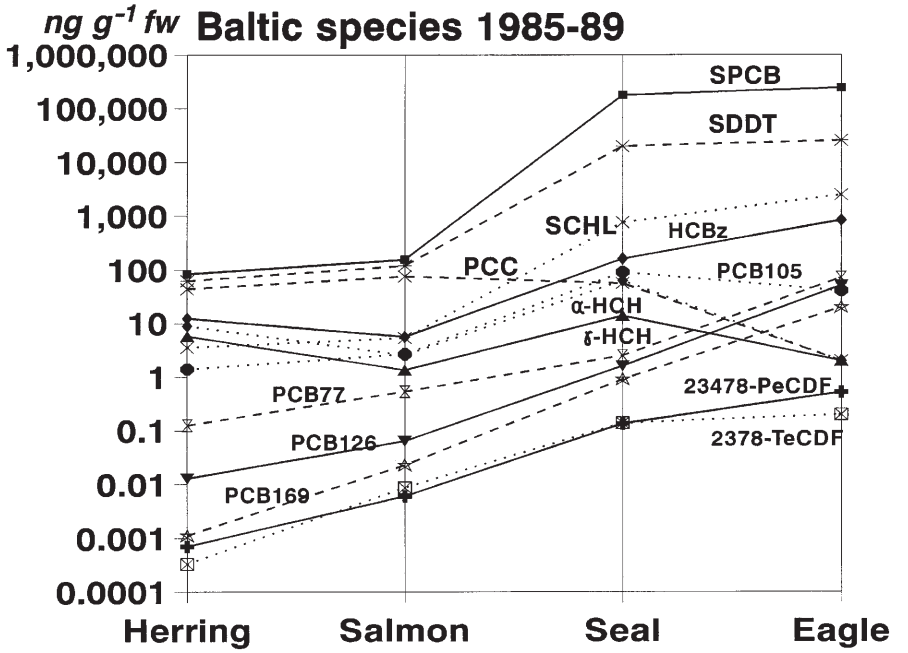


**Fig. 7.** Average concentrations in fresh muscles of Baltic species shown in logarithmic scale (Table 5)

term hazards to biota globally [198, 199]. They are more persistent than the main PCB congeners and are therefore enriched in the food chain and stay in top consumers longer than PCBs, on average. Although their effect on wildlife cannot fully be isolated from the coaccumulated toxic chloropesticides, PCDDs and PCDFs and other less studied OCCs, there are wide indications of their significant role in reproductive damage, especially among predatory birds [47, 197, 198].

Stable isotope ratios present a novel diagnostic tool to estimate biomagnification, because they are dependent on trophic position of the species. A useful index of trophic level of the organism is the stable nitrogen isotope ratio:

$$\delta^{15}\mathrm{N} = [(^{15}\mathrm{N}/^{14}\mathrm{N}_{\mathrm{sample}} \div {}^{15}\mathrm{N}/^{14}\mathrm{N}_{\mathrm{atmosphere}}) - 1] \times 1000 \qquad (2)$$

$\delta^{15}$N is readily measurable by mass spectrometry and it has been found to correlate significantly and positively with food chain length and with concentration of lipophilic xenobiotic. Differences in levels of organochlorine in top predator fish from different waters in the same region can be explained by the different lengths of their food chains measured as $\delta^{15}$N and supported with observations in the field [177, 200–202].

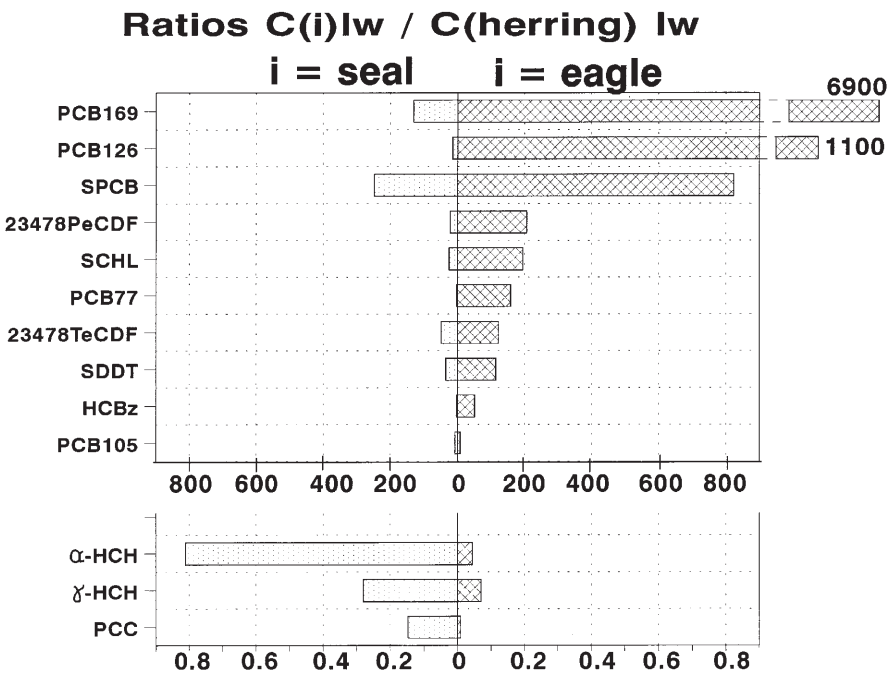


**Fig. 8.** Biomagnification power estimated as concentrations in lipid of seal and eagle normalized to those in herring (calculated from the data in Table 5)

### 4.3
### Trends of Biomagnified Contaminants and Ecotoxic Effects

Follow-up of time trends is important in assessing possible future hazards of POP pollution. For OCCs in Baltic wildlife, the most useful monitoring data thus far have been obtained from analyses of fish, especially herring and salmon samples. Mother salmon of the natural population breeding in the Simojoki River (Bay of Bothnia, Finland) were analyzed extensively during 1988–1992 [82, 179]. These salmon collect OCCs and other pollutants from their prey during their feeding migration in winter along the Baltic and Gulf of Bothnia. The number of samples was sufficient to detect significant time trends by statistics. Most common OCCs – HCHs, oxychlordane, toxaphene (PCC) and SPCB – showed very significant decreases during 1988–1992. In contrast, dioxin-like toxic PCDDs, PCDFs and coplanar PCBs indicated increasing or no trends.

Colour (GCOL) of the eggs and offspring survival (FERM = fertilizing mortality; ESM = eyed stage mortality and YSM = yolk sac mortality) of the same Simojoki river salmon were also recorded. During the study years, YSM increased dramatically, showing a great upward jump in 1991. Similarly, since 1974 YSM has occurred in many salmon populations spawning in Swedish rivers flowing to the Baltic sea [203], and the epidemics were named M74 syndrome.

The data on Simojoki salmon 1988–1992 allowed the use of statistics to study the possible connection of OCC pollution and YSM. Principal component analysis (PCA; Fig. 9) shows that dioxin-like contaminants, especially 1,2,3,7,8-penta-CDF, 2,3,4,7,8-penta-CDF and PCB126 could have a significant role in the occurrence of YSM. In addition, 2,3,7,8-tetra-CDF, PCB77, PCB169, oxychlordane and hexachlorobenzene could participate in the effect. The paler the orange colour (GCOL) of the eggs, the higher is YSM.

The hypothesis of dioxin-like POPs as an original cause of YSM is supported by the fact that exposure to TCDD and similarly toxic chloroaromatic substances (PCDDs, PCDFs and coplanar PCB congeners) has been shown to cause mortality of fish (other species than Baltic salmon) fry in the yolk sac phase of development [204, 205]. Wide studies also show the connection of YSM with thiamine deficiency [206]. This and the decrease of GCOL possibly arise from the influence of dioxin-like contaminants on the metabolism producing vitamin-degradating ezymes. The TEQ approach based on fish TEFs measured for early juvenile mortality [205] support the significance of 2,3,4,7,8-penta-CDF and PCB126 as pollutants behind YSM [82].

Other known reproductive damage from biomagnification is the extinction of some bird populations due to the eggshell thinning effect, which was asso-

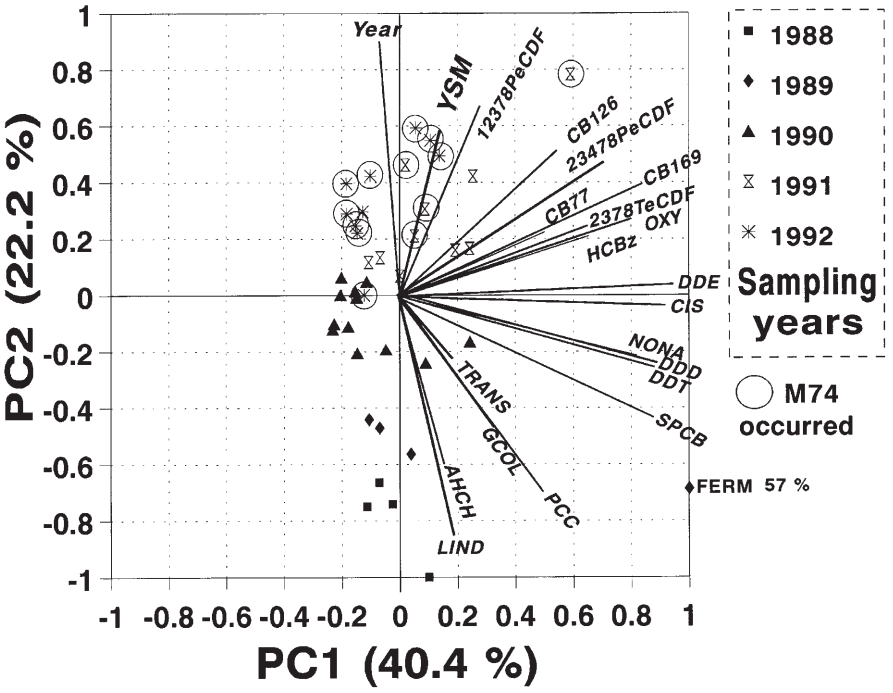


**Fig. 9.** Biplot from the PCA of concentrations in lipid of 40 samples together with sampling year, egg colour (GCOL) and yolksac mortality. Sample scores are shown as *points*, variable loadings as *vectors*

ciated with DDE [45, 207]. The effect is characterized by an eggshell index (EI) defined by

$$\mathrm{EI} = \frac{\text{Weight of the shell (mg)}}{\text{Breadth (mm)} \times \text{Length (mm)}} \tag{3}$$

As an example, DDE contents in the membrane lipids of peregrine falcon (*Falco peregrinus*) eggs collected at different times and from a variety of areas correlate significantly with EI. The linear regression done by this author [6] from data of Peakall and Kiff [208] is illustrated in Fig. 10.

The eggshell thinning effect has been observed as the reason for the low nesting success of Baltic whitetailed eagles in the 1970s [209, 210]. Eggs from the time before DDT usage had EI values of 3.1–3.2, but in the 1970s this was 2.6–2.8.The average DDE concentration in lipid of ten addled eagle eggs collected in 1974–1978 was 452 µg $g^{-1}$ [210]. Further addled eggs (together with whitetailed eagles found dead and their prey) were collected and studied in 1980–1985. These eggs had slighly lower DDE contents and their EIs were a little increased compared to eggs from 1974–1978. Simultaneously, the nesting success of Baltic whitetailed eagles has improved [211, 212].

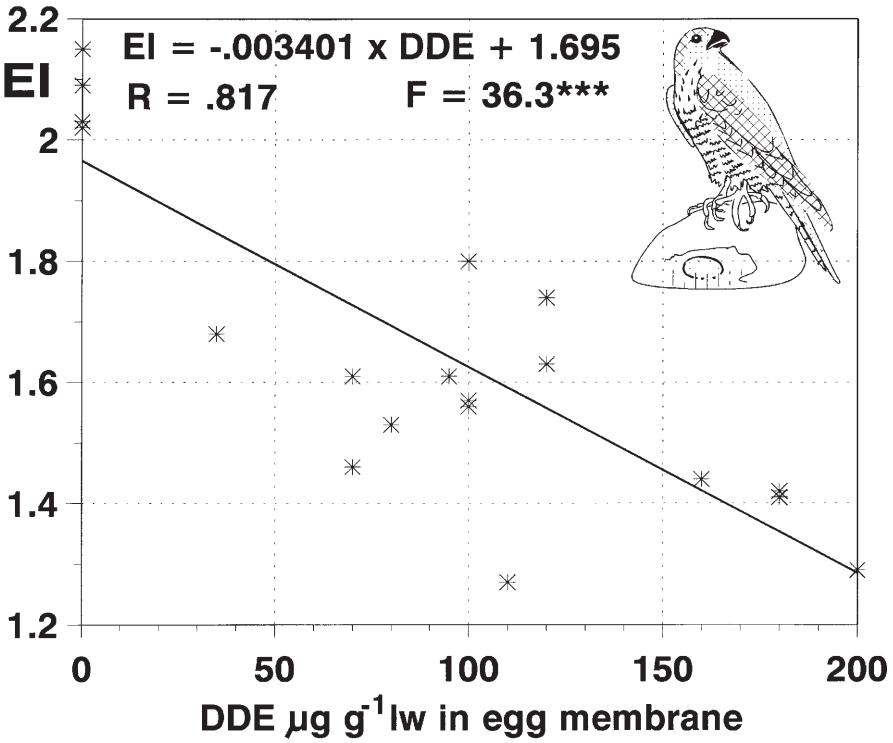


**Fig. 10.** Dependence of eggshell index (EI) on DDE in membrane lipid of peregrine falcon eggs [6]

Chick oedema disease has been demonstrated in many bird species as result of biomagnification of dioxin-like pollutants [55]. The first symptoms: bloody swelling of embryos and chicks associated with poor hatching success, were observed in herring gull colonies of the lower Great Lakes of North America [54]. Bioaccumulation of chloropesticides, PCBs, PCDDs and PCDFs had led to high concentrations in eggs. Injection and follow-up experiments revealed that 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), the most toxic dioxin, was the dominating factor of disease in herring gulls, which were insensitive to chloropesticides and to purified commercial PCB. In eggs collected in the early 1970s from a gull colony in Scotch Bonnett Island, over 1000 ppt (pg $g^{-1}$) concentrations of TCDD were present. A steep decreasing trend from 1971 to 1981 in TCDD levels, which went down to 50–80 ppt was observed. The chick oedema disease was not seen any more in 1975–1981, when the TCDD level in eggs decreased below 500 ppt [54]. Similar trends of chick oedema disease and levels of TCDD in eggs of the herring gull colonies of Lake Ontario indicate that the bioaccumulation of this single compound was the main cause of breeding damage there.

Analyses of trends and prognosis of future development of bioaccumulated pollutants are important to protect man and the environment against future damage. A predictive method was developed during monitoring of merury in pike of different areas of Lake Päijänne, Finland, after industrial discharge of mercury had been restricted in 1968. From Hg analysis results from different sizes of fish over several years, empirical equations (Eqs.(4)–(6)) were developed by multiple regression for mercury content as function of weight of the fish and year of sampling [213]:

$$\text{TIIRINSELKÄ TotHg} = 0.1094 \times \text{WEIGHT}^{0.327} \times (\text{YEAR} - 1970)^{-0.242} \quad (4)$$

$$\text{RISTINSELKÄ TotHg} = 0.0593 \times \text{WEIGHT}^{0.474} \times (\text{YEAR} - 1970)^{-0.332} \quad (5)$$

$$\text{TEHINSELKÄ TotHg} = 0.1758 \times \text{WEIGHT}^{0.408} \times (\text{YEAR} - 1970)^{-0.525} \quad (6)$$

Validity of the prognosis was investigated in 1981 by collecting ten pike from Ristinselkä, analyzing Hg from their muscles, and calculating by linear regression the observed TotHg for 1-kg pike. The predicted content was 0.70 and the observed was 0.69 mg $kg^{-1}$. Then the prognosis was revised, adding the new data to previous data to give [190]

$$\text{RISTINSELKÄ TotHg} = 0.0650 \times \text{WEIGHT}^{0.460} \times (\text{YEAR} - 1970)^{-0.331} \quad (7)$$

A second validation was done in 1987 by analyzing another ten pike from Ristinselkä. Predicted TotHg for 1-kg pike then was exactly the same as the observed value, 0.61 mg $kg^{-1}$ [6]. Accordingly, this method seems to be very useful in planning and managing future fishery economics in new reservoirs, which are normally rich in fish but have mercury mobilization and bioaccumulation problems after the first few years [8, 214, 215].

## 5 Summarizing Conclusions

It is certain that bioaccumulation of persistent chemicals as a consequence of human activities has caused long-term adverse effects in ecosystems. However, in most cases coaccumulation of many toxic substances has made identification of the damaging agents difficult. Cause-effect relationships from bioaccumulation of mercury, lead, cadmium and tin compounds on the local scale have been well demonstrated in numerous cases. Persistent organochlorines (OCC) have obviously caused regional scales of ecological damage, but distinction between the different OCC compounds as major factors behind the damage has not often been achieved. As a result of restriction of release, levels of OCCs in wildlife in many industrialized areas have declined during the 1970s and 1980s. Coinciding with this decline, populations of many wildlife species have been recovered.

## References


1. Hutzinger O (ed) (1980) Handbook of environmental chemistry, vol 3 part A. Anthropogenic compounds, Springer, Berlin Heidelberg New York
2. Gavis J, Ferguson JF (1972) Water Res 6:989
3. Johnels AG, Westermark T (1969) Mercury contamination of the environment of Sweden. In: Chemical fallout, chap 10. Thomas, Springfield
4. Lindqvist O, Jernelöv A, Johansson K, Rohde H (1984) Mercury in the Swedish environment, global and local sources. National Swedish Environment Protection Board Report snv pm 1816
5. Environment Agency, Japan (1975) Studies on the Health effects of alkylmercury in Japan
6. Paasivirta J (1991) Chemical ecotoxicology, L-366. Lewis, Chelsea, MI
7. Mitra S (1986) Mercury in the ecosystem. Trans Tech Publications, Aedermannsdorf, Switzerland
8. Potter L, Kidd D, Standiford D (1975) Environ Sci Technol 9:41
9. Brouard D, Demers C, Lalumiere R, Schetagne R, Verdon R (1990) Evolution of mercury levels in fish of the La Grande hydroelectric complex. Quebec. Summary Report, Hydro Quebec (Montreal) and Schooner (Quebec), PQ
10. Rimmer CC (1992) Amer Birds 46:216
11. Nordberg GF, Skerfving S (1972) Metabolism. In: Friberg L, Vostal J (eds) Mercury in the environment. CRC Press, Cleveland, p 29
12. Frank R, Van Hove Holdrinet M, Suda P (1979) Bull Environ Contam Toxicol 22:500
13. Pankakoski E, Koivisto I, Hyvärinen H (1992) Acta Zool Fennica 91:135
14. Wren C, MacCrimmon H, Frank R, Suda P (1980) Bull Environ Contam Toxicol 25:100
15. Wren CD (1986) Environ Res 40:210
16. Hutzinger O (ed) (1980) Handbook of environmental chemistry, vol 1, part A. The natural environment and biogeochemical cycles. Springer, Berlin Heidelberg New York
17. Bull KR, Avery WJ, Freestone P, Hall JR, Osborn D, Cooke AS, Stowe T (1983) Environ Pollut 31A:239
18. Osborn D, Eney WJ, Bull KR (1983) Environ Pollut 31A:261
19. Wilson KW, Head PC, Jones PD (1986) Wat Sci Tech 18:171
20. Ramade F. (1987) Ecotoxicology. Wiley, Chichester, UK
21. Eisler R (1988) Lead hazards to fish, wildlife, and invertebrates: a synoptic review. Biological Report 85(1.14), US Fish and Wildlife Service, Laurel, MD

22. Dieter MP, Finley MT (1979) Environ Res 19:127
23. Blus LJ, Henny CJ, Hoffman DJ, Grove RA (1993) Ecotoxicology 2:139
24. Jörgensen SS, Willems M (1987) Ambio 16:11
25. Ma W (1989) Arc Environ Contam Toxicol 18:617
26. Hodson PV, Whittle DM, Wong PTS, Borgmann U, Thomas RL, Chau YK, Nriagu JO, Hallett DJ (1984) Lead contamination of the Great Lakes and its potential effects on aquatic biota. In: Ngiaru JO, Simmons MS (eds) Toxic contaminants in the Great Lakes. Wiley, New York, p 335
27. Kägi JHR, Nordberg M (eds) (1979) Metallothionein. Birkhäuser, Basel
28. Spehar RL, Anderson RL, Fiandt JT (1978) Environ Pollut 15:195–208
29. Ray S, McLeese DW, Burridge LE (1981)Mar Pollut Bull 12:383
30. Bryan GW (1976) Some aspects of heavy metal tolerance in aquatic organisms. In: Lockwood AP (ed) Effects of pollutants on aquatic organisms. Cambridge University Press, Cambridge, pp 7–34
31. Cadd GM (1993) FEMS Microbiol Rev 11:297
32. Ward GS, Gramm GC, Parrish PR, Trachman H, Schlesinger A (1981) ASTM Spec Tech Publ 737:183
33. Tsuda T, Nakanishi H, Aoki S, Takebayashi J. (1986) Toxicol Environ Chem 12:137
34. Zuolian C, Jensen A (1989) Mar Pollut Bull 20:281
35. Langston WJ, Burt GR (1991) Mar Environ Res 32:61
36. Cosson R, Amiard-Triquet C, Grandier-Vazeille X (1989) Oceanis 15:411
37. Bryan GW, Gibbs PE, Burt GR, Hummerstone LG (1987) J Mar Biol Assoc 67:525
38. Triebskorn R, Koehler H-R, Flemming J, Braunbek T, Negele R-D, Rahmann H (1994) Aquat Toxicol 30:189
39. Saint-Louis R, Pelletier E, Marsot P, Fournier R (1994) Water Res 28:2533
40. Yamada H, Tateishi M, Takayanagi K (1994) Environ Toxicol Chem 13:1415
41. Lewis TE (1989) Environmental chemistry and toxicology of aluminium,.Lewis, Chelsea, MI
42. Gribble GW (1992) J Nat Prod 55:1353
43. Grimwall A, de Leer EBW (eds) (1995) Naturally-produced organohalogens. Kluwer, Dordrecht
44. Paasivirta J (1988) Wat Sci Tech 20:119
45. Ratcliffe DA (1967) Nature 215:208
46. Smith AG (1991) Chlorinated hydrocarbon insecticides. In: Hayes WJ, Laws ER (eds) Handbook of pesticide toxicology classes of pesticides 2:731
47. Detzell E, Doull J, Giesy J, Mackay D, Munro I, Williams G (1994) Interpretative review of the potential adverse effects of chlorinated organic chemicals on human health and the environment. Regul Toxicol Pharmacol 20, part 2, pp 1–1056
48. Courtney KD (1979) Environmental Research 20:225
49. Ahling B, Brjørseth (1978) Chemosphere 7:799
50. Jensen S (1966) New Scientist 32:612
51. Bowes GW, Simoneit BR, Burlingame AL, de Lappe BW, Risebrough RW (1973) Environ Health Persp 5:191–198
52 Helle EM, Olsson M, Jensen S (1976) Ambio 5:261
53 Kihlström JE, Olsson M, Jensen S (1976) NORDFORSK Milövårdsekreteriatet Publikation 2:567
54 Gilbertsson M (1983) Chemosphere 12:357
55. Firestone D (1973) Environ Health Persp 5:59
56. Vos JG, Koeman JH, van der Maas LH, ten Noever de Brauw MC, Vos RH (1970) Food Cosmet Toxicol 8:625–633
57. Buser RH, Bosshardt HP, Rappe C (1978) Chemosphere 7:109
58. Paasivirta J, Herzschuh R, Humppi T, Kantolahti E, Knuutinen J, Lahtiperä M, Laitinen R, Salovaara J, Tarhanen J, Virkki L (1985) Envir Health Persp 60:269
59. Sandermann W, Stockmann H, Casten R (1957) Chem Ber 90:690
60. Kimmig J, Schulz KH (1957) Dermatologia 115:540

61. Hutzinger O, Fiedler H (1989) Chemosphere 18:23
62. Rappe C, Andersson R, Bergqvist P-A, Brohede C, Hansson M, Kjeller L-O, Lindström G, Marklund S, Nygren M, Swanson SE, Tysklind M, Wiberg K (1987) Chemosphere 16:1603
63. Hutzinger O, Frei RW, Merian E, Pocchiari F (eds) (1982), Chlorinated dioxins and related compounds. Impact on the environment. Pergamon, Oxford
64. Poland A, Clover E, Kende AS (1976) J. Biol. Chem. 251:4936
65. Conney AH, Kuntzman R (1971) Metabolism of normal constituents by drug-metabolizing enzymes in liver microsomes, In: Brodie BB, Gillette J (eds) Concepts in biochemical pharmacology. Springer, Berlin Heidelberg New York, p 401
66. McKinney JD (1981) Environmental health chemistry. Ann Arbor Science Publ, Ann Arbor, MI
67. Tanabe S (1988) PCB problems in the future: foresight from current knowledge. Environ Pollut 50:5–28
68. Kimbrough RD, Jensen AA (eds) (1989) Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products, 2nd edn. Elsevier, Amsterdam
69. Ueda S, Fujiwara A, Umemura T, Tokuda H (1995) Organohalogen Compounds 21:209
70. Safe S (1987) Chemosphere 16:791
71. Gillner M, Fernström B, Gustafsson J-Å, Bergman J, Cambillau C (1986) Chemosphere 15:1673
72. Barnes DG, Bellin J, Cleverly D (1986) Chemosphere 15:1895
73. Nordisk Expertgrupp (1988) Nordisk Dioxinriskbedömning. Nordisk Ministerråd. NORD 49
74. McKinney JD, Singh P (1981) Chem-Biochem Interact 33:271
75. McKinney JD, Chae K, McConnell EE, Birnbaum LS (1985) Ann NY Acad Sci 320:179
76. McKinney JD, Chae K, McConnell EE, Birnbaum LS (1985) Environ Health Persp 20:57
77. Safe S (1984) CRC Crit Rev Toxicol 13:319
78. Safe S (1992) Chemosphere 25:61
79. Ahlborg UG, Brouwer A, Fingerhut MA, Jacobson JL, Jacobson SW, Kennedy SW, Kettrup AAF, Koeman JH, Poiger H, Rappe C, Safe SH, Seegal RF, Tuomisto J, van der Berg M (1992) Eur J Pharmacol – Envir Toxicol Pharmacol Section 228:179
80. Ahlborg U, Becking GC, Birnbaum RS, Brouwer A, Derks HJGM, Feeley M, Golor G, Hanberg A, Larsen JC, Liem AKD, Safe SH, Schlatter C, Waern F, Younes M, Yrjänheikki E (1994) Chemosphere 28:1049
81. Bergqvist P-A, Bergek S (1989) Chemosphere 19:513
82. Paasivirta J, Vuorinen PJ, Vuorinen M, Koistinen J, Rantio T, Hyötyläinen T, Welling L (1995) Organohalogen Compounds 25:355
83. Koistinen J, Koivusaari J, Nuuja I, Paasivirta J, (1995) Chemosphere 30:1671
84. Jakobsson E (1994) Synthesis and analysis of chlorinated naphthalenes. Biological and environmental implications. PhD thesis, Stockholm University
85. Hanberg A, Waern F, Asplund L, Haglund E, Safe S (1990) Chemosphere 20:1161
86. Engwall M, Jakobsson E, Brunström B (1994) Arch Toxicol 68:37
87. De Boer J (1995). Analysis and biomonitoring of complex mixtures of persistent halogenated micro-contaminants. PhD thesis, Amsterdam University. DLO-Netherlands Institute for Fisheries Research, The Netherlands
88. Hale RC, Graeves J, Gallagher K, Vadas GG (1990) Environ Sci Technol 24:1727
89. Freudenthal J, Greve PA (1973) Bull Environ Contam Toxicol 10:108
90. Zitko V, Hutzinger O, Jamieson WD, Choi PMK (1973) Bull Environ Contam Toxicol 10:200
91. Renberg L, Sundström G (1978) Chemosphere 6:477
92. Jensen AA, Jörgensen KF (1983) Sci Total Environ 27:231
93. Kaminsky R, Hites RA (1984) Environ Sci Technol 18:275
94. Griffiths RW (1991) Hydrobiologia 219:143
95. Chu I, Villeneuve DC, Secours VE, Valli VE, Leeson S, Shen SY (1986) Fundam Appl Toxicol 6:69
96. Oliver BG (1987) Environ Sci Technol 21:785

97. Oliver BG, Niimi AJ (1988) Environ Sci Technol 22:388
98. De Boer J (1989) Chemosphere 18:2131
99. Pereira WE, Rostad CE, Chiou CT, Brinton TI, Barber LB II, Demcheck DK, Demas CR (1988) Environ Sci Technol 22:772
100. USEPA (1989) Hexachlorobutadiene: drinking water health advisory. NTIS Report PB91-160 663
101. Bjørseth A, Lunde G, Gjs N (1977) Acta Chem Scand B 31:797
102. Kuokkanen T (1989) Chlorocymenes and chlorocymenenes: persistent chlorocompounds in spent bleach liquors of kraft pulp mills, Deparment of Chemistry, University of Jyväskylä, Research Report 32
103. Rantio T (1992) Chemosphere 25:505
104. Paasivirta J, Koistinen J, Kuokkanen T, Maatela P, Mäntykoski K, Paukku R, Rantalainen A-L, Rantio T, Sinkkonen S, Welling L (1993) Chemosphere 27:447
105. Rantio T, Koistinen J, Paasivirta J (1996) Bioaccumulation of pulp chlorobleaching-originated aromatic chlorohydrocarbons in recipient watercourses. In: Servos MR, Munkittrick KR, Carey JH, Van der Kraak GJ (eds) Environmental fate and effects of pulp and paper mill effluents. Sr. Lucie Press, Delray Beach, FL, p 341
106. Nevalainen T, Koistinen J (1990) Organohalogen componds 4:361
107. Nevalainen T (1995) Synthesis of chlorinated alkylbibenzyls and 9-chlororetene, Chemosphere 30:847
108. Koistinen J, Nevalainen T, Tarhanen J (1992) Environ Sci Technol 26:2499
109. Koistinen J (1992) Chemosphere 24:559
110. Koistinen J, Paasivirta J, Lahtiperä M (1993) Chemosphere 27:149–156
111. Koistinen J, Paasivirta J, Nevalainen T, Lahtiperä M (1994) Chemosphere 28:1261–1277
112. Hodson PV (1996) Mixed function oxygenase induction by pulp mill effluents: advances since 1991. In: Servos MR, Munkittrick KR, Carey JH, Van der Kraak GJ (eds), Environmental fate and effects of pulp and paper mill effluents, Sr. Lucie Press, Delray Beach, FL, p 349
113. Bjørseth A, Carlberg GE, Møller M (1979) Sci Total Environ. 11:197
114. Koistinen J, Paasivirta J, Nevalainen T, Lahtiperä M (1994) Chemosphere 28:2139
115. Paasivirta J, Tarhanen J, Soikkeli J (1986) Chemosphere 15:1429
116. Paasivirta J, Koistinen J (1994) Chlorinated ethers. In: Kiceniuk JW, Ray S (eds) Analysis of contaminants in edible aquatic resources, chap 6. Book II: Organics. VCH Publ, New York, p 411
117. Curtis RF, Land DG, Griffiths NM, Gee M, Robinson D, Peel JL, Dennis C, Gee JM (1972) Nature 235:223–224
118. Paasivirta J, Klein P, Knuutila M, Knuutinen J, Lahtiperä M, Paukku R, Veijanen A, Welling L, Vuorinen M, Vuorinen PJ (1987) Chemosphere 16:1231
119. Paasivirta J, Rantalainen A-L, Welling L, Herve S, Heinonen P (1992) Wat Sci Tech 25:105–113
120. Schreitmüller J, Ballschmiter K (1995) Environ Sci Technol 28:207
121. Firestone D, Ress J, Brown NL, Barron RP, Damico JN (1972) J Assoc Offic Anal Chem 55:85
122. Jensen S, Renberg L (1972) Ambio 1:1–4
123. Paasivirta J, Lahtiperä M, Leskijärvi T (1982) Experiences of structure analyses of chlorophenol dimers and trimers found in different samples. In: Hutzinger O, Frei RW, Merian E, Pocchiari F (eds) (1982) Chlorinated dioxins & related compounds. Impact on the environment. Pergamon, Oxford, p 191
124. Humppi T (1985) Synthesis, identification and analysis of dimeric impurities of chlorophenols. Department of Chemistry, University of Jyväskylä, Research Report 23
125. Humppi T, Knuutinen J, Paasivirta J (1984) Chemosphere 13:1235
126. Humppi T (1985) Chemosphere 14:523
127. Paasivirta J, Tarhanen J, Juvonen B (1987) Chemosphere 16:1787
128. Paasivirta J, Heinola K, Humppi T, Karjalainen A, Knuutinen J, Mäntykoski K, Paukku R, Piilola T, Surma-Aho K, Tarhanen J, Welling L, Vihonen H, Särkkä, J (1985) Chemosphere 14:469

129. Humppi T, Heinola K (1985) J.Chromatogr 331:410
130. Sundström G, Hutzinger O, Safe S (1976) Chemosphere 5:267
131. Nevalainen.T (1995) Polychlorinated diphenyl ethers: synthesis, NMR spectroscopy, structural properties, and estimated toxicity. Department of Chemistry, University of Jyväskylä, Research Report 50
132. Nevalainen T, Koistinen J, Nurmela, P (1994) Environ Sci Technol 28:1341
133. Hu J, Kolehmainen E, Nevalainen T, Kauppinen R (1994) Chemosphere 28:1069
134. Kurz J, Ballschmiter K (1995) Fresenius J Anal Chem 351:98
135. Becker M, Phillips T, Safe S (1991) Toxicol Environ Chem 33:189
136. Koistinen J, Vuorinen PJ, Paasivirta J (1993) Chemosphere 27:2365
137. Koistinen J, Paasivirta J, Suonperä M, Hyvärinen H (1995) Environ Sci Technol 29:2541
138. Miyazaki T, Kaneko S, Horii S, Yamagishi T (1981) Bull Environm Contamin Toxicol 26:577
139. Atlas E, Sullivan K, Giam CS (1986) Atm. Environ. 20:1217
140. Grimwall A, Borén H (1995) Preprints of papers presented at the 210th ACS National Meeting, Chicago, IL, August 20–24, 1995, 35(2):294
141. Anonymous (1975) Feed contaminant in farmers' blood. Chem Eng News 24:7
142. Sundström G, Hutzinger O, Safe S (1976) Chemosphere 5:11
143. Andersson Ö, Blomkvist B (1981) Chemosphere 10:1051
144. Suter GW II.(ed) (1993) Ecological risk assessment. L785. Lewis, Chelsea, MI
145. Calamari D (ed) (1993) Chemical exposure prediction. Lewis Publ, Roca Baton, FL
146. Mackay D (1991) Multimedia environmental models. The fugacity approach. L-242. Lewis, Chelsea, MI
147. Paasivirta J, Sinkkonen S, Rantio T, Calamari D, Di Guardo A, Matthies M, Trapp S (1995) Modelled and observed fate of selected organochlorines in the Nordic environment. In: Munawar M, Luotola M (eds) The contaminants in the Nordic ecosystem: the dynamics, processes and fate. SPB Academic Publ, Amsterdam, pp 11–24
148. OECD (1981) Guidelines for testing of chemicals. OECD, Paris
149. Lyman WJ, Reehl WF, Rosenblatt DH (1990) Handbook of chemical property estimation methods. American Chemical Society, Washington, DC
150. Hermens JLM, Oppenhuizer A (eds) (1991) QSAR in environmental toxicology, Elsevier, Amsterdam
151. ECETOC (1992) Estimating environmental concentrations of chemicals using fate and exposure models. European Centre for Ecotoxicology and Toxicology of Chemicals, Technical Report 50, Brussels
152. Trapp S, Matthies M (1996) Dynamik von Schadstoffen-Umweltmodellierung mit CemoS, Springer, Berlin Heidelberg New York
153. Baumgarten G, Reiter B, Scheil S, Schwartz S, Wagner J-O (1995) CemoS Handbuch für ProgrammeVersion 1.0. University of Osnabrück, Germany
154. Thomann, RV (1989) Environ Sci Technol 23:699
155. Clark KE, Gobas FAPC, Mackay D (1990) Environ Sci Technol 24:1203
156. Neely WB, Mackay D (1982) Evaluative model for estimating environmental fate. In: Dickson KL, Maki AW, Cairns J (eds) Modeling the fate of chemicals in the aquatic environment. Ann Arbor Science Publ, Ann Arbor, MI
157. Paasivirta J (1990) Organohalogen Compounds 1:367
158. Gobas FAPC, McCorquodalle JR, Haffner GD (1993) Environ Toxicol Chem 12:567–576
159. Bidleman TF, Wideqvist U, Jansson B, Söderlund R (1987) Atmos Environ 21:641–654
160. Gaul H (1992) ICES Mar Sc Symp 195:110
161. Paasivirta J, Rantio T (1991) Chemosphere 22:47
162. Paasivirta J, Rantio T, Koistinen J, Vuorinen PJ (1993) Chemosphere 27:2011
163. Brüggemann R, Münzer B (1987) EXWAT Multicompartiment Modell für den Transport von Stoffen in Oberflächenwässern, GSF-Bericht 33/87, Neuherberg
164. Matthies M, Trapp S (1988). Environmental models for exposure and hazard assessment of chemicals. In: Lokke H, Tyle H, Bro-Rasmussen F (eds) 1st European Conference on Ecotoxicology, October 17–19, 1988, Copenhagen, Denmark, p 420

165. Mackay D, Southwood JM (1992) Modelling the fate of organochlorine chemicals in pulp mill effluents. Report of the Pulp and Paper Centre of the University of Toronto
166. Vetter RD, Carey MC, Patton JS (1985) J Lipid Res 26:428
167. Masuda Y, Kagawa R, Kuroki H, Kuratsune M, Yoshimura T, Take I, Kusuda M, Yamashita F, Hayashi M (1978) Food Cosmet Toxicol 16:543
168. Kodama H, Ota H (1980) Arch Environ Health 35:95
169. Morris RJ, Law RJ, Allchin CR, Kelly CA, Fileman CF (1989) Mar Pollut Bull 20:512
170. Ballschmiter K (1992) Angew Chem Int Edn Engl 31:487
171. Pacyna J, Oehme M (1988) Atmos Environ 22:243.
172. Patton GW, Hinkley DA, Walla MD, Bidleman TF (1989) Tellus 41B:243
173. Patton GW, Walla MD, Bidleman TF, Barrie LA (1991) J Geophys Res 96D:10 867
174. Hargrave BT, Vass WP, Erickson PE, Fowler BR (1988) Tellus 40B:480
175. Gregor DJ, Gummer W (1989) Environ Sci Technol 23:561
176. Bidleman TF, Patton GW, Walla MD, Hargrave BT, Vass WP, Erickson PE, Fowler BR, Scott V, Gregor DJ (1989) Arctic 42:307
177. Muir DCG, Norstrom RJ, Simon M (1988) Environ Sci Technol 22:1071
178. Muir DCG, Wagemann R, Hargrave BT, Thomas DJ, Peakall DB, Norstrom RJ (1992) Sci Total Environ 122:75
179. Paasivirta J, Koistinen J, Rantio T, Vuorinen PJ (1994) Organohalogen Compounds 20:529
180. Bright DA, Duschenko WT, Grundy S, Reimer KJ (1995) Sci Tot Environ 160/161:265
181. Wania F, Mackay D (1993) Ambio 22:10
182. Wania F, Mackay D (1993) Chemosphere 27:2079
183. Paasivirta J, Rantio T, Koistinen J, Vuorinen PJ (1993) Chemosphere 27:2011
184. Särkkä J, Hattula M-L, Paasivirta J, Janatuinen J (1978) Holarctic Ecol 1:326
185. Hattula M-L, Janatuinen J, Särkkä J, Paasivirta J (1978) Environ Pollut 15:121
186. Hattula M-L, Särkkä J, Janatuinen J, Paasivirta J, Roos A (1978) Environ Pollut 17:19
187. Särkkä J, Hattula M-L, Janatuinen J, Paasivirta J, Palokangas R (1978) Pestis Monit J 12:26
188. Paasivirta J, Särkkä J, Pellinen J, Humppi T (1981) Chemosphere 10:787
189. Paasivirta J, Särkkä J, Aho M, Surma-Aho K, Tarhanen J, Roos A (1981) Chemosphere 10:405
190. Paasivirta J, Särkkä J, Surma-Aho K, Humppi T, Kuokkanen T, Marttinen M (1983) Chemosphere 12:239
191. Winter S, Streit B (1992) Chemosphere 24:1765
192. Ballschmiter K, Bacher R, Mennel A, Fischer R, Riehle U, Swerev M (1992) J High Resolut Chromatogr 15:260–270
193. Bush B, Bennett AH, Snow JT (1986) Arch Environ Contam Toxicol 15:333
194. Paasivirta J, Särkkä J, Leskijärvi T, Roos A (1980) Chemosphere 9:441
195. Bergqvist P-A, Bergek S (1989) Chemosphere 19:513
196. Paasivirta J, Paukku R (1989) Chemosphere 19:1551
197. Kubiak TJ, Harris HJ, Smith LM, Scwartz TR, Stalling DL, Trick JA, Silfo L, Docherty DE, Erdman TC (1989) Arch Environ Contam Toxicol 18:706
198. Tanabe S, Kannan N, Subramanian An, Watanabe S, Tatsukawa R (1987) Environ Pollut 47:147–163
199. Tarhanen J, Koistinen J, Paasivirta J, Vuorinen PJ, Koivusaari J, Nuuja I, Kannan N, Tatsukawa R (1989) Chemosphere 18:1067
200. Schindler DW, Kidd KA, Muir DCG, Loclkhart WL (1995) Sci Tot Environ 160/161:1
201. Kidd KA, Schindler DW, Muir DCG, Lockhart WL, Hesslein RH (1995) Science 269:240
202. Rasmussen JB, Rowan DJ, Lean DRS, Carey JH (1990) Can J Fish Aquat Sci 47:2030
203. Norrgren L, Andersson T, Bergqvist P-A, Björkelund I (1993) Environ Toxicol Chem 12:2065
204. Helder Th (1982) Effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) on early life stages of two fresh-water fish species. In: Hutzinger O, Frei RW, Merian E, Pocchiari F (eds) Chlorinated dioxins and related compounds, impact on the environment. Pergamon, Oxford, pp 455–462

205. Walker MK, Peterson RE (1991) Aquat Toxicol. 21:219
206. Naturvårdsverket (1995) Second Workshop on Reproduction Disturbances in Fish, 20–23 November 1995, Stockholm, Sweden, Mimeograph
207. Ratcliffe DA (1970) J Appl Ecol 7:67
208. Peakall DB, Kiff LF (1979) Ibis 121:200–204
209. Odsjö T, Johnels AG (1972) Äggskalförtunning hos några svenska fågelarter. Report to the National Swedish Environmental Protection Board 1972-2-14, Stockholm (mimeographed)
210. Koivusaari J, Nuuja I, Palokangas R, Finnlund M (1980) Environ Pollut 23:41
211. Paasivirta J, Kääriäinen H, Paukku R, Surma-Aho K, Koivusaari J, Nuuja I, Palokangas R (1985) Chlorohydrocarbons and mercury in whitetailed eagles, their eggs and food. In: Paasivirta O (ed) Ministry of Environment, Finland, Division of Environmental Protection, Publication A3, p 27 (in Finnish)
212. Koivusaari J, Nuusa I, Paasivirta J, unpublished data
213. Paasivirta J, Särkkä J, Aho M, Surma-Aho K, Tarhanen J, Roos A (1981) Chemosphere 10:405
214. Abernathy AR, Cumbie PM (1977) Bull Environ Contam Toxicol 17:595
215. Surma-Aho K, Paasivirta J, Rekolainen S, Verta M (1986) Chemosphere 15:353–372