Chemical Dynamics in Fresh Water Ecosystems

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6 MODELING THE ACCUMULATION AND TOXICITY OF ORGANIC CHEMICALS IN AQUATIC FOOD CHAINS

INTRODUCTION

For the management of contaminants in rivers and lakes, it is important to be able to predict the impacts of a discharge of a single and/or multiple chemicals in terms of chemical concentrations and toxic effects in aquatic biota. Building such a predictive capability requires knowledge of the transport of the chemical from its origin into the organism and of the interaction of the chemical with crucial target sites in the organism. This knowledge can then be captured in computer models, which when peer reviewed, represents the best available knowledge for practical use in water quality management. Further research will improve these models and provide a better predictive ability and management.

Several chapters in this volume discuss the factors that control the water and sediment concentrations in aquatic systems and formulate models that can be used for predicting water and sediment concentrations from chemical discharges. This chapter addresses the uptake and bioaccumulation of organic chemicals from the water and sediments into single organisms and in entire food chains. It summarizes the current state of knowledge regarding the mechanism of chemical uptake and bioaccumulation in various aquatic organisms and it presents a model to predict the accumulation of organic substances in aquatic food chains. It is further shown how this model can be used to assess toxic effects in fish and other aquatic organisms.

We will demonstrate the ability of this "food chain" model to predict chemical concentrations in aquatic food chains by applying the model to experimental data for Lake Ontario. Since the model only requires a small set of basic, and readily accessible data to characterize the food chain, the model is believed to be a practical tool for contaminant management on an "ecosystem" level.

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BIOAVAILABILITY

One of the most important factors controlling the uptake and bioaccumulation of organic chemicals in aquatic organisms, is the concentration of the chemical in the water that can be absorbed by the organism from the water (e.g., via the gills in fish). In particular for very hydrophobic chemicals, the concentration of absorbable or bioavailable chemical is often only a fraction of the total chemical concentration in the water. This fraction is usually referred to as the Bioavailable Solute Fraction (BSF), or simply the bioavailability (Landrum et al. 1985, Black and McCarthy 1988, Gobas et al. 1989a).

The bioavailability of organic chemicals in natural waters is largely determined by the interaction of the chemical with organic carbon-containing materials, which occur both in particulate and in dissolved form. There remains controversy about the state of the chemical when sorbed to the organic matter. The most generally held current belief is that the chemical is dissolved in a solid solution form in, or on, a sponge-like matrix of organic matter, which is too large to permeate through biological membranes (e.g., the gill membrane). Sorption is generally viewed as an equilibrium partitioning of the chemical between the water and the organic matter (OM) in the water column (Karickhoff 1984)

$$\text{Dissolved Chemical} + \text{OM} \Leftrightarrows \text{Chemical} - \text{OM} \qquad (1)$$

which can be expressed by a partition coefficient K_{oc} (l/kg):

$$K_{oc} = C_{WB} / \left(C_{WD} \cdot [OM]\right) \qquad (2)$$

where C_{WB} (µg/l) is the concentration of "bound" or "sorbed" chemical, i.e., $(C_{WT} - C_{WD})$, C_{WD} is the concentration of dissolved chemical (µg/l) and [OM] is the concentration of organic matter in the water (kg/l). Based on the hypothesis that only chemical in true solution is bioavailable, the bioavailability can be defined as the ratio of the truly dissolved chemical concentration in the water C_{WD} (µg/l) and the total chemical concentration in the water C_{WT}(µg/l), i.e.,

$$BSF = C_{WD} / C_{WT} \qquad (3)$$

Since, it has been suggested that within experimental error, K_{oc} equals K_{ow}/d_{oc} (DiToro 1985), the following expression can be derived by substituting Equation 2 in Equation 3 to estimate the bioavailability of organic chemicals in natural waters.

$$BSF = 1 / \left(1 + K_{ow} \cdot [OM] / d_{oc}\right) \qquad (4)$$

where

d_{oc} = the density of organic carbon (kg/L)

Typical concentrations of organic matter in natural waters vary between approximately 10^{-6} to 10^{-7} kg/l. If for example, the organic matter content of the water is 10^{-6} kg/l, Equation 4 predicts a bioavailability for a chemical with a log K_{ow} of 6 of 50%. This means that only half of the chemical concentration in the water can be absorbed by organisms, whereas the other half is in a nonabsorbable form.

CHEMICAL UPTAKE AND BIOACCUMULATION IN BENTHIC INVERTEBRATES

It is often convenient to view the uptake and bioaccumulation of hydrophobic organic substances in benthic invertebrates as the result of an equilibrium partitioning of the chemical between the lipids of the organism, the organic carbon fraction (OC) of the sediment, and the interstitial (or pore) water (Shea 1988, Gobas et al. 1989b).

$$C_B \cdot d_L / L_B = C_s \cdot d_{oc} / OC = K_{LW} \cdot C_P \quad (5)$$

where C_B is the chemical concentrations in the benthic invertebrates (μg/kg wet weight), C_S is the concentration in the sediments (μg/kg dry weight), C_P is the truly dissolved chemical concentration in the pore water (μg/l water); L_B is the lipid fraction of the benthos (kg lipid/kg organism), d_L is the density of the lipids of the benthos (kg/l), OC is the organic carbon fraction of the sediments (kg organic carbon/kg organism), d_{oc} is the density of the organic carbon fraction of the sediments (kg/l), and K_{LW} is the lipid-water partition coefficient.

An interesting aspect of this model is that the organism/sediment concentration ratio C_B/C_S is only dependent on organism and sediment characteristics, namely L_B, d_L, OC and d_{oc}. The nature and properties of the chemical (e.g., K_{ow}) are not important. In other words, if the equilibrium assumption applies in the field, C_B/C_S should be approximately similar for organic chemicals, namely $L_B \cdot D_{oc}/OC \cdot d_L$, or simply L_B/OC since d_L and d_{oc} are approximately the same. If, for example, L_B is 6% and OC is 2%, then the concentration in the benthic invertebrates is approximately three times higher than the concentration in the sediments.

This simple equilibrium model ignores the physiological and time-dependent processes of chemical exchange between the organism, the sediments and the interstitial and overlying water. Alternative kinetic models, notably by Landrum (e.g., Landrum et al. 1992, this volume), achieve a greater detail by treating

independently chemical uptake from water (i.e., via the gills), uptake from ingestion of sediment associated mater (i.e., via the gastrointestinal tract), and elimination to the water (i.e., via the gills), elimination into egested "faecal" matter (via the gastrointestinal tract) and elimination by metabolic transformation (i.e., for metabolizable chemicals). From a physiological point of view, this kinetic model is more correct, but it relies on laboratory measurements of several rate constants. Presently, these measurements are difficult to make, which may explain the poor agreement of the kinetic model with observed field data (Landrum et al. 1992, Gobas et al. 1989b). With continued research, the predictability of the kinetic model is likely to improve. In anticipation of better models, we adopt the simpler sediment-organism equilibrium model for benthic invertebrates. The applicability of this model can be demonstrated by comparing model predictions to field data. The results of such a comparison are listed in Table 1, which compiles results for chemicals varying in log K_{ow} from 2.7 to 8.3, for various species of benthic invertebrates, i.e., *Tubifex tubifex, Limnodrilus hofmeisteri, Pontoporeia affinis* (data from Oliver and Niimi 1988 and Fox et al. 1983), *and Hexagenia limbata* (data from Gobas et al. 1989b). The data in Table 1 indicate that chemical concentrations in benthic invertebrates and in sediments are approximately equal if they are expressed on respectively a lipid (i.e., as C_B/L_B) and on an organic carbon (i.e., as C_S/OC) basis; i.e., the benthos/sediment concentration ratio or BSR (i.e., $C_B \cdot OC/L_B \cdot C_S$) is 0.98, with a standard deviation of a factor of 3 (n = 203). This is in satisfactory agreement with a model, which predicts a value of approximately 1.0.

CHEMICAL UPTAKE AND BIOACCUMULATION IN AQUATIC MACROPHYTES

Various studies have shown that the uptake and bioaccumulation of chemicals in submerged aquatic macrophytes and phytoplankton are largely controlled by chemical exchange between the organism and the water (Geyer et al. 1984, Mallhot 1987, Gobas et al. 1991). Growth of the individual organism or population growth can also play a role, since an increase in the organism's weight or volume has a "diluting" effect on the chemical concentration in the organism. The following model represents this:

$$dC_A / dt = k_1 \cdot C_{WD} - k_2 \cdot C_A - k_G \cdot C_A \qquad (6)$$

where C_A (μg/kg) is the chemical concentration in the organism and C_{WD} (μg/l) is the bioavailable concentration in the water. k_1 (l/kg/d) andk_2 (1/d) are the first-order rate constants for respectively chemical uptake from the water and chemical elimination to the water. k_G (1/d) is the rate constant for growth, thus assuming that growth can be described by a first-order rate constant. This model has the following steady-state solution:

Table 1. Observed Benthos/Sediment Concentration Ratios (BSR).

Tubifex tubifex and *Limnodrilus hoffmeisteri*, Lake Ontario[a]

Chemical	log K_{ow}	BSR	Chemical	log K_{ow}	BSR
PCB-28	5.80	1.11	PCB-146	6.90	0.48
PCB-18	5.60	3.78	PCB-141	6.90	0.44
PCB-22	5.60	1.89	PCB-128	7.00	0.99
PCB-26	5.50	16.20	PCB-151	6.90	0.66
PCB-33	5.80	9.72	PCB-132	7.30	0.44
PCB-17	5.60	5.40	PCB-156	6.90	0.39
PCB-25	5.50	5.40	PCB-136	6.70	1.16
PCB-24	5.50	2.70	PCB-180	7.00	0.83
PCB-32	5.80	4.73	PCB-187	7.00	1.38
PCB-66	5.80	0.49	PCB-170	6.90	0.65
PCB-70	5.90	1.10	PCB-183	7.00	1.22
PCB-56	6.00	0.53	PCB-177	7.00	0.54
PCB-52	6.10	0.68	PCB-174	7.00	0.26
PCB-47	5.90	0.81	PCB-178	7.00	0.32
PCB-44	6.00	0.81	PCB-171	6.70	0.57
PCB-74	6.10	2.20	PCB-203	7.10	0.43
PCB-49	6.10	0.86	PCB-201	7.50	0.34
PCB-64	6.10	0.69	PCB-194	7.10	0.66
PCB-42	5.60	0.69	PCB-209	8.26	0.34
PCB-53	6.10	9.45	ppDDE	5.70	0.36
PCB-40	5.60	0.96	ppDDT	5.80	0.18
PCB-46	6.00	3.60	mirex	6.89	0.60
PCB-45	6.00	0.74	γ-chlordane	2.78	0.26
PCB-101	6.40	0.77	α-BHC	3.81	1.98
PCB-84	6.10	0.63	lindane	3.80	0.27
PCB-118	6.40	0.77	HCBD	4.80	0.09
PCB-110	6.40	0.55	OCS	6.20	0.61
PCB-87	6.50	2.43	HCB	5.47	0.09
PCB-105	6.40	0.70	QCB	5.03	0.07
PCB-95	6.40	0.95	TeCB-1,2,3,5	4.65	0.04
PCB-85	6.20	0.44	TeCB-1,2,4,5	4.51	0.10
PCB-92	6.50	1.22	TeCB-1,2,3,4	4.75	0.03
PCB-82	6.20	0.47	TCB-1,3,5	4.02	0.21
PCB-91	6.30	0.57	TCB-1,2,4	3.98	0.02
PCB-99	6.60	0.98	TCT-2,4,5	4.72	0.09
PCB-153	6.90	0.81	TCT-2,3,6	4.80	0.17
PCB-138	7.00	0.56	PCT	6.20	0.27
PCB-149	6.80	0.73			

Table 1. (continued)

Pontoporeia affinis, Lake Ontario[a]

Chemical	log K_{ow}	BSR	Chemical	log K_{ow}	BSR
PCB-28	5.80	1.59	PCB-170	6.90	2.34
PCB-18	5.60	2.16	PCB-183	7.00	3.77
PCB-22	5.60	1.53	PCB-177	7.00	2.12
PCB-26	5.50	22.50	PCB-174	7.00	1.66
PCB-33	5.80	5.58	PCB-178	7.00	1.80
PCB-17	5.60	4.50	PCB-171	6.70	1.94
PCB-25	5.50	5.40	PCB-185	7.00	1.53
PCB-24	5.50	5.85	PCB-173	7.00	1.97
PCB-32	5.80	3.83	PCB-203	7.10	0.55
PCB-66	5.80	0.59	PCB-201	7.50	0.51
PCB-70	5.90	1.25	PCB-194	7.10	0.88
PCB-56	6.00	0.68	PCB-195	7.10	0.98
PCB-52	6.10	0.79	PCB-206	7.20	0.09
PCB-47	5.90	1.05	PCB-209	8.26	0.07
PCB-44	6.00	1.02	PCB-31	5.70	1.59
PCB-74	6.10	2.73	PCB-27	5.80	5.85
PCB-49	6.10	1.31	PCB-76	6.00	1.25
PCB-64	6.10	0.80	PCB-60	5.90	0.68
PCB-42	5.60	0.88	PCB-81	6.10	0.68
PCB-53	6.10	10.80	PCB-48	6.10	1.05
PCB-40	5.60	0.93	PCB-97	6.60	2.75
PCB-46	6.00	5.80	PCB-182	7.00	1.50
PCB-45	6.00	1.39	PCB-190	7.00	2.34
PCB-101	6.40	1.23	PCB-196	7.50	0.55
PCB-84	6.10	1.03	ppDDE	5.70	0.64
PCB-118	6.40	1.26	ppDDD		0.07
PCB-110	6.40	0.68	ppDDT	5.80	0.43
PCB-87	6.50	2.75	mirex	6.89	0.35
PCB-105	6.40	1.08	photomirex		0.97
PCB-95	6.40	1.41	γ-chlordane	2.78	2.49
PCB-85	6.20	0.74	α-BHC	3.81	12.60
PCB-92	6.50	1.19	lindane	3.80	2.97
PCB-82	6.20	0.84	HCBD	4.80	0.12
PCB-91	6.30	0.92	OCS	6.20	0.56
PCB-99	6.60	1.08	HCB	5.47	0.16

Table 1. (continued)

Pontoporeia affinis, Lake Ontario[a]

Chemical	log K_{ow}	BSR	Chemical	log K_{ow}	BSR
PCB-153	6.90	1.62	QCB	5.03	0.14
PCB-138	7.00	1.56	TeCB-1,2,3,5	4.65	0.07
PCB-149	6.80	1.22	TeCB-1,2,4,5	4.51	0.12
PCB-146	6.90	0.83	TeCB-1,2,3,4	4.75	0.17
PCB-141	6.90	1.46	TCB-1,3,5	4.02	0.11
PCB-128	7.00	0.72	TCB-1,2,4	3.98	0.09
PCB-151	6.90	1.39	TCB-1,2,3	4.04	0.18
PCB-132	7.30	0.70	TCT-2,4,5	4.72	0.50
PCB-156	6.90	1.67	TCT-2,3,6	4.80	0.70
PCB-136	6.70	7.46	PCT	6.20	0.41
PCB-180	7.00	3.32			
PCB-187	7.00	1.50			

Hexagenia limbata, Lake St. Clair[b]

Chemical	log K_{ow}	BSR
QCB	5.03	0.28
HCB	5.45	0.28
OCS	6.29	0.74
PCB-101	6.40	0.92
PCB-87	6.50	1.08
PCB-118	6.40	0.82
PCB-153	6.90	1.42
PCB-138	7.00	1.08
PCB-180	7.00	1.24

Tubifex tubifex and *Pontoporeia affinis*, Lake Ontario[c]

Chemical	log K_{ow}	BSR	BSR
TCB-1,3,5	4.02	3.34	2.11
TCB-1,2,4	3.98	2.35	1.76
TCB-1,2,3	4.04	2.09	1.86
HCBD	4.80	1.42	1.45

Table 1. (continued)

***Tubifex tubifex and Pontoporeia affinis* Lake Ontario[c]**

Chemical	log K_{ow}	BSR	BSR
TeCB-1,2,4,5	4.51	3.31	1.79
TeCB-1,2,3,4	4.75	2.33	2.35
QCB	5.03	5.03	5.99
HCB	5.47	10.50	8.31

Note: The concentration in the benthos is expressed on a lipid weight basis, the concentration in the sediment is expressed on an organic carbon basis.

[a] Oliver and Niimi 1988.
[b] Gobas et al. 1989b.
[c] Fox et al. 1983.

$$BCF = C_A / C_{WD} = k_1 / \left(k_2 + k_G\right) \tag{7}$$

where the ratio of the concentration in the organism and that in the water is often referred to as the bioconcentration factor BCF. If k_G is of the same magnitude or larger than k_2, the size of the growth factor k_G can have a significant effect on the bioconcentration factor. However, unless macrophyte populations are in a rapid growth phase (e.g., at certain times in the spring or summer), k_G may be small compared to k_2, which simplifies BCF to k_1/k_2. Since lipids are usually the predominant site for bioaccumulation of hydrophobic substances, BCF can be satisfactorily approximated by the chemical's octanol-water partition coefficient K_{ow}, giving

$$BCF = C_A / C_{WD} = k_1 / k_2 = L_A \cdot K_{ow} \tag{8}$$

where L_A is the lipid content of the macrophytes (Gobas et al. 1991 for aquatic plants, Geyer et al. 1984 for phytoplankton).

CHEMICAL UPTAKE AND BIOACCUMULATION IN ZOOPLANKTON

Due to their small size and large area/volume ratio, uptake and bioaccumulation of organic chemicals in zooplankton are predominantly due to chemical ex-

change between the organism and the water (Clayton et al. 1977). A similar process has been discussed for aquatic macrophytes, and the following model can be proposed to estimate chemical concentrations in zooplankton C_Z (μg/kg):

$$BCF = C_Z / C_{WD} = k_1 / k_2 = L_Z \cdot K_{ow} \tag{9}$$

where

L_Z = lipid content of the zooplankton

CHEMICAL UPTAKE AND BIOACCUMULATION IN FISH

Fish absorb chemicals directly from the water, i.e., via the gills and through the consumption of food, i.e., via the gastrointestinal tract (Bruggeman et al. 1981). Other uptake routes such as chemical absorption via the skin are usually considered to be insignificant. Chemical loss or elimination can occur via the gills to the water (i.e., essentially the reverse process of chemical uptake from the water), via egestion of faecal matter or as a result of metabolic transformation. The following equation combines these processes in an overall flux equation, describing the net flux of chemical into the fish as the sum of all of the uptake and loss fluxes:

$$\begin{aligned} d\left(V_F \cdot C_F\right) / dt = {} & k_1 \cdot V_F \cdot C_{WD} - k_2 \cdot V_F \cdot C_F + k_D \cdot V_F \cdot C_D \\ & - k_E \cdot V_F \cdot C_F - k_M \cdot V_F \cdot C_F \end{aligned} \tag{10}$$

where

C_{WD} = dissolved chemical concentration in the water (μg/l)
C_D = chemical concentration in the food (μg/kg)
C_F = chemical concentration in the fish (μg/kg fish)
V_F = weight of the fish (kg)

The 'k's are first order rate constants: k_1 for uptake from the water via the gills (l/kg·day); k_2 for elimination via the gills to the water (1/day); k_D for chemical uptake from food (kg food/kg fish/day); k_E for elimination by faecal egestion (1/day) and k_M for metabolic transformation of the chemical (in 1/day).

If fish growth is insignificant (V_F is constant), the steady-state solution of this model is

$$C_F = \left(k_1 \cdot C_{WD} + k_D \cdot C_D\right) / \left(k_2 + k_E + k_M\right) \tag{11}$$

where

$k_1/(k_2 + k_E + k_M)$ = bioconcentration factor
$k_D/(k_2 + k_E + k_M)$ = biomagnification factor

Various authors have suggested that fish growth can have a considerable effect on bioaccumulation factors and concentrations in fish, in particular for chemicals of high K_{ow} (Thomann and Connolly 1984, Clark et al. 1990). From a modeling perspective, the effect of growth can be treated by varying V_F in Equation 10 with time (Gobas et al. 1989a). However, in that case a simple steady-state solution does not exist. To incorporate fish growth into the model and to allow for a simple steady-state solution to be applied, the effect of growth can be introduced in terms of a rate constant k_G, i.e., $dV_F/V_F \cdot dt$, which has units of 1/day.

If this simplification for fish growth is introduced into the fish bioaccumulation model, the flux equation becomes

$$dC_F / dt = k_1 \cdot C_{WD} - k_2 \cdot C_F + k_D \cdot C_D - k_E \cdot C_F - k_M \cdot C_F - k_G \cdot C_F \quad (12)$$

and the steady-state mass balance equation is

$$C_F = \left(k_1 \cdot C_{WD} + k_D \cdot C_D\right) / \left(k_2 + k_E + k_M + k_G\right) \quad (13)$$

where

$k_1/(k_2 + k_E + k_M + k_G)$ = bioconcentration factor
$k_D/(k_2 + k_E + k_M + k_G)$ = biomagnification factor

From Equation 13 it follows that to estimate steady-state chemical concentrations in fish, information is required of the values of k_1, k_2, k_D, k_E, k_M, and k_G for different chemicals and fish species.

Gill Uptake

The rate at which chemicals are absorbed by fish via the gills is expressed by the gill uptake rate constant k_1 which has units of l/kg·day. The gill uptake rate is the combined process of the gill ventilation rate G_V (m^3/day) and the diffusion rate of the chemical across the gills (Gobas and Mackay 1987). The extent to which chemicals that enter the gill compartment by gill ventilation are actually absorbed by the organism is usually expressed by the gill uptake efficiency E_W. The uptake rate constant k_1 then follows as

$$k_1 = E_W \cdot G_V / V_F \quad (14)$$

Studies of the relationship between k_1 and K_{ow} and between E_w and K_{ow} in various fish species have shown that (1) k_1 and E_w increase with K_{ow} if log K_{ow} is low (< 4.5 to 5), (2) k_1 and E_w are constant if K_{ow} is large (between 5 to 7), and (3) k_1 and E_w drop with increasing K_{ow} for chemicals with extremely high K_{ow} (log K_{ow} above 7) (McKim et al. 1985, Gobas et al. 1986, Gobas and Mackay 1987). Based on these observations, a two-phase resistance model has been suggested, which assumes that gill uptake involves transport in aqueous and in lipid or membrane phases (Gobas and Mackay 1987). The resulting equations for k_1 and E_w are

$$1 / k_1 = (V_F / Q_W) + (V_F / Q_L) / K_{ow} \tag{15}$$

$$1 / E_W = (G_V / Q_W) + (G_V / Q_L) / K_{ow} \tag{16}$$

where Q_w and Q_L are transport parameters with units of l/day that represent the transport rates in the aqueous and the lipid phases of the fish. In essence, Equation 15 and Equation 16 demonstrate that uptake and elimination tend to be controlled by transport in the lipid phases if the chemical's K_{ow} is low. But with increasing K_{ow}, chemical transport in the aqueous phases of the fish becomes more important and ultimately dominates the kinetics, resulting in a constant k_1 and E_w with K_{ow}. This model satisfactorily describes the behavior of k_1 and E_w for most chemicals but it does not explain the drop of k_1 and E_w with K_{ow} for chemicals with extremely high K_{ow}. It is believed that the observed drop for high K_{ow} substances is not due to reduced gill uptake but the result of a reduced bioavailability and/or experimental errors associated with the difficult water concentration measurements (Gobas and Mackay 1987).

Equation 15 demonstrates that to estimate k_1, data are required for the chemical's K_{ow}, the weight of the fish V_F and the water and lipid phase transport parameters Q_w and Q_L. The following relationship between Q_w and V_F has been derived from experimental data and can be used to estimate Q_w from the weight of the fish (Gobas and Mackay 1987):

$$Q_w = 88 \cdot 3 \cdot V_F^{0.6(\pm 0.2)} \tag{17}$$

Because of insufficient data, a similar weight-dependent relationship for Q_L can not be derived at present. However, it appears from the available data that Q_L is approximately 100 times smaller than Q_w (Gobas and Mackay 1987). In particular for chemicals with a high bioconcentration potential (i.e., high K_{ow}), Q_L can therefore often be ignored and an accurate value is not needed.

Equations 15 and 17 provide a simple method to estimate k_1 from K_{ow} and the weight of the fish. For example, in a 0.25 kg fish, Q_w is 38.4 l/day and Q_L is approximately 0.384 l/day, thus giving a k_1 of 154 $l \cdot kg^{-1} \cdot d^{-1}$ for a chemical with a log K_{ow} of 6.

Gill Elimination

The rate at which chemicals are being eliminated by fish via the gills is expressed by the gill elimination rate constant k_2 which has units of 1/day. Models for the chemical elimination from the fish to the water via the gills are closely related to models for the chemical uptake rate constant since elimination is in essence the reverse process of gill uptake. The ratio of k_1 and k_2 can be viewed as the chemical's partition coefficient between the fish lipids and water, which can be approximated by $L_F \cdot K_{ow}$.

$$k_1 / k_2 = L_F \cdot K_{ow} \tag{18}$$

where

L_F = lipid content of the fish, i.e., the ratio of the lipid weight V_L (g) and weight V_F (g) of the fish, V_L/V_F. After substitution of Equation 15 into Equation 18, it follows that

$$1 / k_2 = \left(V_L / Q_W\right) \cdot K_{ow} + \left(V_L / Q_L\right) \tag{19}$$

where Q_w and Q_L are the same as in Equation 15 and Equation 16. Equation 19 predicts that k_2 is relatively constant if K_{ow} is low, and drops with increasing K_{ow} for chemicals of higher K_{ow}.

Metabolic Transformation

The rate at which chemicals are being metabolized in fish is expressed by the metabolic transformation rate constant k_M which has units of 1/day. Presently, there are few models that can be used to estimate k_M for organic substances in fish or other organisms. This is a serious knowledge gap, in particular when the purpose of the model is to estimate the bioaccumulation tendency of new chemicals for which no information regarding metabolic transformation exists.

However, if information regarding the metabolic transformation is available, an appropriate value for k_M can be estimated. For example, if the chemical's half-life $t_{1/2}$ is five years, a k_M of $0.693/t_{1/2}$ or 0.00038 d^{-1} can be estimated. This value may be small compared to k_2 or k_E, which makes its precise value irrelevant. So, if a chemical is known to be persistent, it is often possible to assume that k_M is zero, without affecting the model calculations significantly.

Dietary Uptake

The rate at which chemicals are absorbed by fish from the diet, i.e., via the gastrointestinal tract, is expressed by the dietary uptake rate constant k_D which

has units of kg food/kg fish/day. The dietary uptake rate is the combined process of the food ingestion rate F_D (kg food/day) and the diffusion rate of the chemical across the intestinal wall and the faecal egestion rate F_E (Gobas et al. 1988). The extent to which chemical in the diet is actually absorbed by the organism can be expressed by the dietary uptake efficiency E_D, which is related to k_D by

$$k_D = E_D \cdot F_D / V_F \tag{20}$$

Although there is a considerable variability in the data, it has been shown that E_D is approximately 0.5 for chemicals with a log K_{ow} less than 6. With further increasing log K_{ow} (i.e., above 6), E_D shows a tendency to drop with increasing K_{ow} (Gobas et al. 1988). These observations can be explained by a two-phase resistance model for dietary uptake, which assumes that dietary uptake involves transport in aqueous and in lipid or membrane phases (Gobas et al. 1988). From this model it follows that:

$$1 / E_D = 5.3(\pm 1.5) \cdot 10^{-8} \cdot K_{ow} + 2.3(\pm 0.3) \tag{21}$$

Based on the work of Weininger (1978) a simple model based on fish bioenergetics can be used to estimate the feeding rate F_D as a function of temperature T and the fish's body weight V_F as follows:

$$F_D = 0.022 \cdot V_F^{0.85} \cdot \exp(0.06 \cdot T) \tag{22}$$

Equations 20 to 22 provide a simple method to estimate k_D from K_{ow}, the weight of the fish and temperature. For example, a 0.25 kg fish at 10°C has a feeding rate of approximately 12 g food/day or 0.012 kg food/day. If the food contains a chemical with a log K_{ow} of 6, E_D is approximately 42% and k_D is 0.42 · 0.012/0.25 or 0.021 kg food/kg fish/day.

Elimination by Fecal Egestion

The rate at which chemicals are being eliminated by egestion of fecal matter, i.e., via the gastrointestinal tract, is expressed by the fecal elimination rate constant k_E which has units of kg feces/kg fish/day. Presently, there are few data that can be used to estimate k_E. However, the available data suggest that the fecal egestion rate is approximately three to five times lower than the ingestion rate (Gobas et al. 1988, 1989a). We thus suggest that

$$k_E = 0.25 \cdot k_D \tag{23}$$

and assume that k_E is related to K_{ow} and the feeding rate in a similar manner as

k_D. The four times lower value of k_E is believed to be due to the effect that as a result of food digestion, the egestion rate is lower than the feeding rate. In addition, fecal matter may have a lower affinity for hydrophobic chemicals than the more organic rich food phase. The two effects combined cause k_E to be smaller than k_D, resulting in a concentration increase in the gastrointestinal tract (i.e., magnification). This increase in chemical concentration in the gastrointestinal tract due to food digestion provides the concentration gradient that is required for net uptake of the chemical into the fish, thus causing the concentration in the fish to exceed that in the consumed food (i.e., biomagnification) as long as gill elimination and metabolic transformation are small (Gobas et al. 1992). The ratio k_D/k_E is the predominant factor in the model that causes biomagnification in the food chain.

Growth

The rate of fish growth can have a significant effect on the steady-state concentration in the fish. Fish growth results in an increase of the fish volume and a drop in concentration if uptake is too slow to compensate the reduction in chemical mass per volume. The following generalized growth equations have been suggested by Thomann et al. (this volume) and are believed to give an adequate representation of the magnitude of fish growth:

$$k_G = 0.00251 \cdot V_F^{-0.2} \quad \text{for temperatures around } 25^\circ\text{C} \tag{24}$$

$$k_G = 0.000502 \cdot V_F^{-0.2} \quad \text{for temperatures around } 10^\circ\text{C} \tag{25}$$

FOOD-CHAIN ACCUMULATION

So far we have discussed chemical bioaccumulation in individual organisms. However, as a result of the feeding relationships, the chemical concentration in the predator is related to that in its prey. The trophodynamics thus play a role in the transfer of chemicals through the food chain and the accumulation of chemicals in the organisms of the food chain (Oliver and Niimi 1988, Thomann and Connolly 1984). In terms of describing and modeling this process we can combine the submodels for individual organisms to form food chains if feeding relationships can be defined. To include the effect of feeding interactions on the chemical concentration in the fish, we can add food preference factors P_i to Equation 13 to give

$$C_F = \left(k_1 \cdot C_{WD} + k_D \cdot \Sigma P_i \cdot C_{D,i}\right) / \left(k_2 + k_E + k_M + k_G\right) \tag{26}$$

where $\Sigma P_i \cdot C_{D,i}$ represents the composition of the fish's diet. P_i is the fraction of the fish's diet that consists of component i with concentration of $C_{D,i}$. If for example, the fish's diet consists of 20% phytoplankton, 70% benthic invertebrates and 10% small fish, then P_i is respectively 0.2, 0.7 and 0.1, adding up to 1, and $C_{D,i}$ is respectively C_A, C_B and C_F for small fish.

Since chemical uptake in phyto- and zooplankton is predominantly from the water, dietary accumulation (or biomagnification) can usually be ignored. Food consumption by phyto- and zooplankton may thus contribute insignificantly to the accumulation of chemicals in the food chain.

MODELING TOXICITY

Toxic effects resulting from chemical exposure can be expected to occur when the concentration of the chemical in the organism reaches a certain threshold level. If these threshold levels can be defined, it is possible to estimate toxic effects in biota from the chemical concentrations in the fish. For example, several studies have shown that for a large group of hydrophobic organic substances (often referred to as "narcotics") an internal concentration in the organism of approximately 1 to 3 mmol/kg causes acute lethality (McCarthy 1986, Abernethy and Mackay 1988, Van Hoogen and Opperhuizen 1988, Gobas et al. 1991). This concentration of approximately 2.0 mmol/kg appears to apply to several species of organisms including various species of fish, benthic invertebrates and plants, and to a number of hydrophobic inert substances such as alcohols, ketones, chlorinated aromatics, and alkanes. It is believed that this concentration represents a "minimal" or "basic" toxicity of a chemical substance. In other words, if a chemical does not act by a specific mode of toxic action, it exerts acute lethality at an internal concentration of approximately 2.0 mmol/kg. This suggests that many hydrophobic organic chemicals have a similar activity at the target site (i.e., a similar toxicity) and that the extent of the toxic impact is directly related to the concentration in the organism. Differences in observed LC_{50}s and "sensitivities" between various organisms are therefore considered to be a reflection of the rates of chemical uptake in the organisms, causing the threshold levels to be reached at different times in different organisms.

Because this mode of toxicity appears to be chemical independent, it is not surprising that several studies have demonstrated that the toxicity of mixtures of these simple hydrophobic organics is additive (Hermens 1989). Consequently, it can be suggested that acute lethality occurs if the sum of the chemical concentrations in the organisms reaches the threshold level. From a modeling perspective this means that the occurrence of acute lethality can be treated as the sum of the toxic contributions of the individual chemical components, i.e., $\Sigma X_i / V_F$, where X_i is the amount of each chemical i in the mixture (in mmol) and V_F is the volume of the organism (in kg). When $\Sigma X_i / V_F$ reaches the toxic threshold level(s), acute lethality is predicted.

An interesting feature of this model is that it is possible to determine which component of a mixture of chemicals in water or sediment exerts the greatest toxic impact. This is the chemical which causes the highest concentration in the organism (i.e., the nearest to the toxic threshold level). This information may be important when setting priorities for chemical clean up. Another convenient aspect of this modeling approach is that LC_{50} values for individual chemicals in various organisms are not needed to make predictions of chemical toxicity in a variety of organisms.

MODEL APPLICATION

To test the food chain model, we applied the model to the Lake Ontario food chain. This was possible thanks to the extensive collection of data on chemical concentrations in the most important species in lake Ontario (Oliver and Niimi 1988). We refer to these original studies for a complete account of the observed data. The model considers phytoplankton, zooplankton (i.e., *Mysis relicta*), two benthic invertebrate species, (i.e., Oligochaetes (*Tubifex tubifex*) and *Pontoporeia affinis*), and four fish species, i.e., sculpins (*Cottus cognatus*), alewifes (*Alosa pseudoharengus*), smelt (*Osmerus mordax*), and a composite group of 60 large size salmonid species, including Lake Trout (*Salvelinus namaycush*), Rainbow trout (*Salmo Gairdneri*), and Coho Salmon (*Oncorhynchus velinus namaycush*). To compare the model predictions to field observations, data regarding lipid contents and weights of the individual organisms were taken from the study of Oliver and Niimi (1988). Data for feeding preferences were taken from Flint (1986). Based on experimental observations that approximately 50% of the total concentration of PCBs in Lake Ontario is in a sorbed state (IJC 1991), a value of $2.5 \cdot 10^{-7}$ kg/l was chosen for the organic matter content of Lake Ontario water, representing a C_{WD} that is 50% of C_{WT} for a chemical with a log K_{ow} of 6.6. A summary of the data that were used in the model are listed in Table 2.

Typical results of the model-data comparison are graphically illustrated in Figure 1 for phyto- and zooplankton, in Figure 2 for benthic invertebrates and in Figure 3 for various species of fish. Each of these model-data comparisons consist of data for approximately 60 organic substances, including several PCB congeners, DDT, DDE, chlorobenzenes, mirex, octachlorostyrene, hexachlorobutadiene, lindane, and others. Typical results are also presented in Table 3, which summarizes predicted and observed concentrations of total PCBs in various organisms of the Lake Ontario food chain. Figure 4 further illustrates the good agreement with observed data. The only exception is that observed concentrations in phyto- and zooplankton are higher than predicted. The reason for this is unknown, but it is possible that sampling difficulties and small sample numbers ($n = 3$ for phytoplankton and $n = 2$ for zooplankton) contribute to the poor fit. The apparent underestimation of the observed concentrations in phyto- and zooplankton does not appear to affect the quality of fit for the fish species.

Table 2. A Summary of the Input Parameters that are used in the Food Chain Model Calculations.

Water temperature: 8°C
Organic content of the water: 0.00000025 kg/l
Organic carbon content of the sediments: 2%
Density of lipids: 0.9 kg/l
Density of organic carbon: 0.9 kg/l
Metabolic transformation rate constant: 0 d^{-1}

Species characteristics:
Phytoplankton:
Lipid content 0.5%

Zooplankton: *Mysids*
Lipid content 5.0%

Benthos 1: *Pontoporeia*
Lipid content 3.0%

Benthos 2: *Oligochaetes*
Lipid content 1.0%

Fish 1: Sculpin
Weight: 5.4 g; lipid content: 8.0%
Diet: 18% zooplankton, 82% *Pontoporeia*

Fish 2: Alewife
Weight: 32 g; lipid content: 7.0%
Diet: 60% zooplankton, 40% *Pontoporeia*

Fish 3: Smelt
Weight: 16 g; lipid content: 4.0%
Diet: 54% zooplankton; 21% *Pontoporeia*; 25% sculpins

Fish 4: Salmonids
Weight: 2150 g; lipid content: 16%
Diet: 10% sculpin, 50% alewife, 40% smelt

This may be an indication that actually observed concentrations for phyto- and zooplankton may have exceeded the real values.

A sensitivity analysis showed that contaminant concentrations in all fish species are more sensitive to changes in concentration in the sediments than in

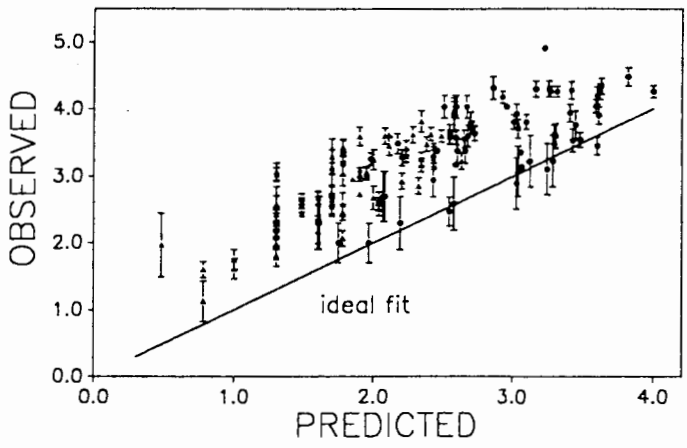

Figure 1. Logarithms of observed concentrations (ng/kg) of various organochlorines in phytoplankton (▲) and in zooplankton (●) versus model predicted concentrations. The solid line represents the ideal fit.

the water. This indicates that, in general, concentrations in Lake Ontario fish are more responsive to sediment than to water concentrations.

CONCLUSIONS

This study presents a model for the chemical distribution in aquatic food chains. The model consists of a series of equations for the chemical uptake, elimination, and bioaccumulation in fish, aquatic macrophytes, benthos, and zooplankton. Each of these "submodels" has been tested individually and represents the current state of understanding regarding the mechanism of chemical bioaccumulation. The food chain model combines these expressions and the combined model is shown to be in good agreement with observed data for the Lake Ontario food chain.

This ability to predict chemical distribution and food chain transfer of organic substances in real food chains provides an important tool for the management of contaminants on an "ecosystem" level. The model is simple since it only requires basic data to characterize the food chain, such as organism weights, lipid

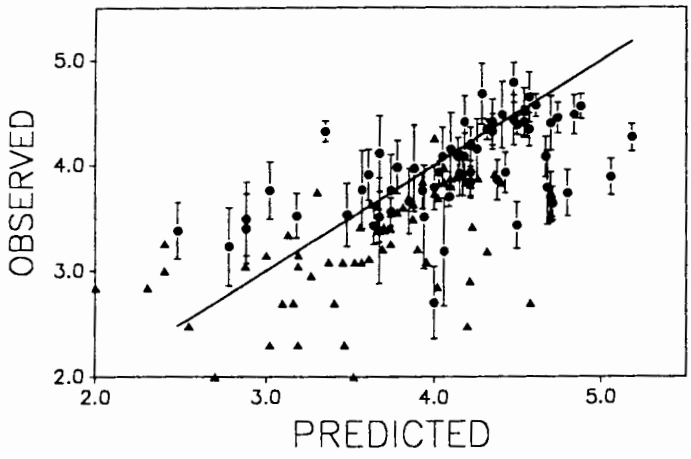

Figure 2. Logarithms of observed concentrations (ng/kg) of various organochlorines in Oligochaetes (▲) and in *Pontoporeia* (●) versus model predicted concentrations. The solid line represents the ideal fit.

contents, and trophic interactions. Thus with little effort, predictions of chemical concentrations in various organisms can be made from concentrations in water and sediments. The model predictions are believed to be accurate within a factor of two to three.

Recently, models for the physical distribution of chemicals have been developed, which can successfully estimate contaminant concentrations in water and sediments from chemical discharges (IJC 1991). The combination of these fate models with food chain models will provide a capability to assess on an "ecosystem" level the chemical exposure of biota as a result of chemical discharges. Finally, it is possible to formulate models to estimate toxic impacts, possibly following the threshold approach outlined above. This requires that toxic effects are associated with a specific concentration in the organism. Presently, such a threshold level has only been identified for the acute lethality of narcotics. This model is difficult to field test since environmental concentrations are usually too low to reach the threshold levels required to cause the toxic effects. Further research may reveal that other, more subtle toxic effects can be related to a specific threshold concentration, thus providing an ability to interpret chemical loadings in terms of toxic effects to the ecosystem.

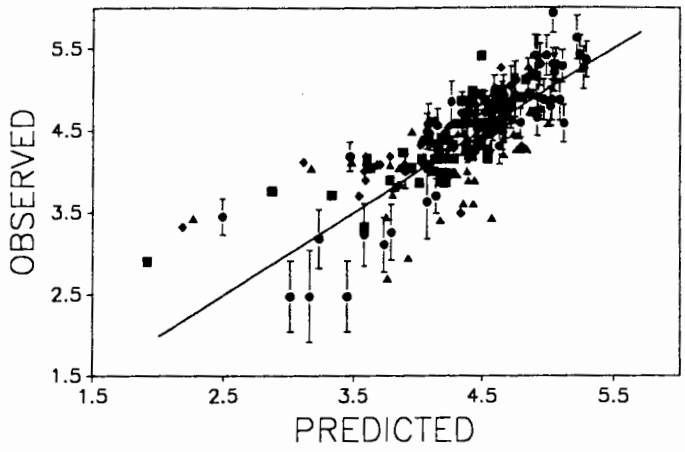

Figure 3. Logarithms of observed concentrations (ng/kg) of various organochlorines in Salmonids (●), smelt (◆), sculpins (▲) and alewife (■) versus model predicted concentrations. The solid line represents the ideal fit.

Table 3. Observed and model predicted concentrations (μg/g) of total PCBs in various organisms of the Lake Ontario food chain [a]

Species	Predicted	Observed
Phytoplankton	0.011	0.05 (±0.012)
Mysids	0.11	0.33 (±0.12)
Photoporeia	0.86	0.79 (±0.48)
Oligochaetes	0.29	0.18 (±0.1)
Sculpins	1.6	1.6
Alewifes	0.99	1.3
Smelt	1.4	1.4
Salmonids	3.5	3.7 (±0.45)

[a] Observed data are from Oliver and Niimi (1988).

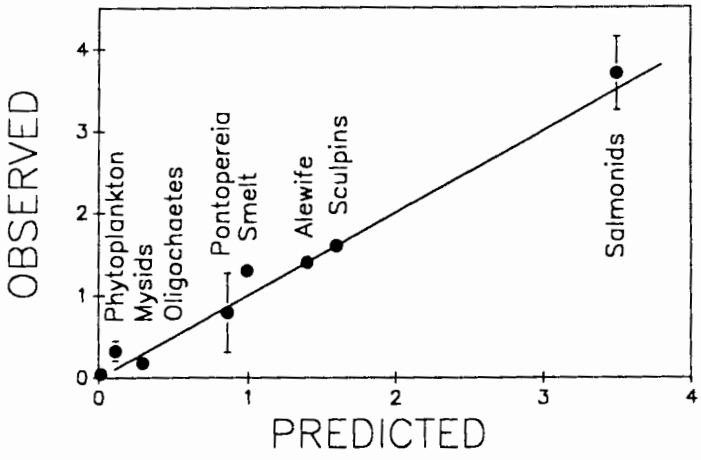

Figure 4. Observed concentrations (ng/kg) of total PCBs in organisms of the lake Ontario food chain versus predicted concentrations. The solid line represents the ideal fit.

REFERENCES

Abernethy, S.G., D. Mackay and L.S. McCarthy. "Volume Fraction Correlation for Narcosis in Aquatic Organisms: The Key Role of Partitioning," *Environ. Toxicol. Chem.* 7:469-481 (1988).

Black, M.C., and J.F. McCarthy. "Dissolved Organic Macromolecules Reduce the Uptake of Hydrophobic Organic Contaminants by the Gills of the Rainbow Trout (*Salmo Gairdneri*)," *Environ. Toxicol. Chem.* 7:593-600 (1988).

Bruggeman, W.A., L.B.J.M. Matron, D. Kooiman and O. Hutzinger. "Accumulation and Elimination Kinetics of Di- , Tri- , and Tetra Chlorobiphenyls by Goldfish after Dietary and Aqueous Exposure," *Chemosphere* 10:811-832 (1981).

Clayton, J.R., S.P. Pavlou and N.F. Breitner. "Polychlorinated Biphenyls in Coastal Marine Zooplankton," *Environ. Sci. Technol.* 18:676-682 (1977).

Clark, K.E., F.A.P.C. Gobas and D. Mackay. "Model of Organic Chemical Uptake and Clearance by Fish from Food and Water," *Environ. Sci. Technol.* 24:1203-1213 (1990).

DiToro, D.M. "A Particle Intercation Model of Reversible Organic Chemical Sorption," *Chemosphere* 15:1503-1538 (1985).

Flint, R.W. "Hypothesized Carbon Flow Through the Deepwater Lake Ontario Food Web," *J. Great Lakes Res.* 12:344-354 (1986).

Fox, M.E., J.H. Carey and B.G. Oliver. "Compartmental Distribution of Organochlorine Contaminants in the Niagara River and the Western Basin of Lake Ontario," *J. Great Lakes Res.* 9:287-294 (1983).

Geyer, H., G. Politzki and D. Freitag. "Prediction of Ecotoxicological Behaviour of Chemicals: Relationship Between n-Ocatnol-Water Partition Coefficient and Bioaccumulation of Organic Chemicals by Alga Chlorella," *Chemosphere.* 13:269-284 (1984).

Gobas, F.A.P.C., A. Opperhuizen and O. Hutzinger. "Bioconcentration of Hydrophobic Chemicals in Fish: Relationship with Membrane Permeation," *Environ. Toxicol. Chem.* 5:637-646 (1986).

Gobas, F.A.P.C., and D. Mackay. "Dynamics of Hydrophobic Organic Chemical Bioconcentration in Fish," *Environ. Toxicol. Chem.* 6:495-504 (1987).

Gobas, F.A.P.C., D.C.G. Muir and D. Mackay. "Dynamics of Dietary Bioaccumulation and Faecal Elimination of Hydrophobic Organic Chemicals in Fish," *Chemosphere* 17:943-962 (1988).

Gobas, F.A.P.C., K.E. Clark, W.Y. Shiu and D. Mackay. "Bioconcentration of Polybrominated Benzenes and Biphenyls and Related Superhydrophobic Chemicals in Fish: Role of Bioavailability and Faecal Elimination," *Environ. Toxicol. Chem.* 8:231-247 (1989a).

Gobas, F.A.P.C., D.C. Bedard, J.J.H. Ciborowski and G.D. Haffner. "Bioaccumulation of Chlorinated Hydrocarbons by the Mayfly *Hexagenia Limbata* in Lake St. Clair," *J. Great Lakes Res.* 15:581-588 (1989b).

Gobas, F.A.P.C., E.J. McNeil, L. Lovett-Doust and G.D. Haffner. "Bioconcentration of Chlorinated Aromatic Hydrocarbons in Aquatic Macrophytes (*Myriophyllum Spicatum*)," *Environ. Sci. Technol.* 25:924-929 (1991).

Gobas, F.A.P.C., E.J. McNeil, L. Lovett-Doust and G.D. Haffner. "A Comparative Study of the Bioconcentration and Toxicity of Chlorinated Hydrocarbons in Aquatic Macrophytes and Fish," *Am. Soc. Test. Mat.* 1115:174-183 (1991).

Gobas, F.A.P.C., J.R. McCorquodale and G.D. Haffner. "Intestinal Absorption and Biomagnification of Organochlorines," *Environ. Toxicol. Chem.* in press (1992).

Hermens, J.L.M. "Quantitative Structure-Activity Relationships of Environmental Pollutants," in *Handbook of Environmental Chemistry, Vol. 2E, Reactions and Processes*, O. Hutzinger, Ed. (Berlin: Springer-Verlag, 1989), pp.111-162.

International Joint Commission. "Proceedings of the Great Lakes Mass Balance Workshop," Niagara on the Lake, in press (1991).

Karickhoff, S.W. "Organic Pollutant Sorption in Aquatic Systems," *J. Hydraul. Eng. ASCE.* 110:707-735 (1984).

Landrum, P.F., M.D. Reinhold, S.R. Nihart and B.J. Eadie. "Predicting the Bioavailability of Organic Xenobiotics to Pontoporeia Hoyi in the Presence of Humic and Fulvic Materials and Natural Dissolved Organic Carbon," *Environ. Toxicol. Chem.* 4:459-467 (1985).

Landrum, P.F., T.D. Fontaine, W.R. Faust, B.F. Eadie and G.A. Lang. "Modelling the Accumulation of Polycyclic Hydrocarbons by the Amphipod Diporeia," This volume (1992).

Mallhot, H. "Prediction of Algal Bioaccumulation and Uptake Rate of Nine Organic Compounds by Ten Physiocochemical Properties," *Environ. Sci. Technol.* 21:1009-1013 (1987).

McCarthy, J.F., and B.D. Jimenez. "Reduction in Bioavailability to Bluegills of Polycyclic Aromatic Hydrocarbons Bound to Dissolved Humic Material," *Environ. Toxicol. Chem.* 4:511-521 (1985).

McCarthy, L.S. "The Relationship Between Aquatic Toxicity QSARs and Bioconcentration for some Organic Chemicals," *Environ. Toxicol. Chem.* 5:1071-1080 (1986).

McKim, J.M., P.K. Schnieder and G. Veith. "Absorption Dynamics of Organic Chemical Transport Across Trout Gills as Related to Octanol-Water Partition Coefficient," *Toxicol. Appl. Pharmacol.* 77:1-10 (1985).

Oliver, B.G., and A.J. Niimi. "Trophodynamic Analysis of Polychlorinated Bipehnyl Congeners and Other Chlorinated Hydrocarbons in the Lake Ontario Ecosystem," *Environ. Sci. Technol.* 22:388-397 (1988).

Shea, D. "Developing National Sediment Quality Criteria," *Environ. Sci. Technol.* 22:1256-1261 (1988).

Thomann, R.V. "Bioaccumulation Model of Organic Chemical Distribution in Aquatic Food Chains," *Environ. Sci. Technol.* 23:699-707 (1989).

Thomann, R.V., and J.P. Connolly. "Model of PCB in the Lake Michigan Lake Trout Food Chain," *Environ. Sci. Technol.* 18:65-71 (1984).

Thomann, R.V., J.P. Connolly and T. Parkerton. "Modelling Accumulation of Organic Chemicals in Aquatic Food-Webs," in Chapter 7 of this book (1992).

Van Hoogen, G., and A. Opperhuizen. "Toxicokinetics of Chlorobenzenes in Fish," *Environ. Toxicol. Chem.* 7:213-219 (1988).

Weininger, D. "Accumulation of PCBs by Lake Trout in Lake Michigan," Ph.D. thesis, University of Wisconsin-Madison (1978).