Model of Organic Chemical Uptake and Clearance by Fish from Food and Water

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■ A comprehensive model is presented that describes the bioaccumulation of organic chemicals by fish from food and water, using size- and species-dependent parameters describing transport and transformation "resistances" and parameters for metabolic conversion and bioavailability. Uptake of a nonmetabolizing chemical from water tends to result in the chemical adopting a fugacity in the fish approaching that in the water, as expressed by a bioconcentration factor. Uptake from food may result in a fish fugacity that is higher than the food or water fugacity, corresponding to biomagnification. This is postulated to be due to food digestion causing a fugacity increase in the gastrointestinal tract. This biomagnification phenomenon is most significant for very hydrophobic, slowly clearing, nonmetabolizing chemicals. The model also describes food chain biomagnification, the dependence of fish concentration on rates of metabolism and growth, and the effect of reduced bioavailability.

Introduction

One of the most important environmental transport and partitioning processes is bioaccumulation. This process may result in concentrations of toxic chemicals in fish that are large multiples of those of the water in which the fish dwell. The phenomenon of bioconcentration is generally considered to be an equilibrium partitioning or "thermodynamic" process in which the fish/water concentration ratio is a reflection of the different affinities of the chemical for water and for the lipids of the fish and is usually well correlated with the chemical's octanol–water partition coefficient K_{OW} (*1–5*). The fish and water thus approach a state in which the chemical's fugacities or chemical potentials in fish and water are equal. This phenomenon is readily investigated in the laboratory in uptake–depuration experiments. When loss of correlation occurs, it may be due to the slow kinetics of uptake, fish growth, reduced bioavailability of the chemical due to sorption in the water, unusual resistances to transfer, or metabolism of the chemical (*5–13*).

In real situations the fish receives chemical both from water by gill transfer and from food by ingestion. The latter process may lead to biomagnification, or a fish/water concentration ratio that exceeds the bioconcentration ratio. Connolly and Pedersen (*14*) and Oliver and Niimi (*15*)

demonstrated the existence of a biomagnification phenomenon by comparing concentrations and fugacities of fish of various trophic levels with those of the water. Apparently the fish achieves a fugacity that may be a multiple (e.g., 3) of the food or water fugacity as a result of food uptake. If the food and water are at similar fugacities, the fish must thus be in a state in which the chemical is being taken up against a fugacity gradient, i.e., transport is from low to high fugacity. In principle there must be simultaneous loss by depuration, but the rate is presumably too slow to allow equilibrium to be reestablished.

Successful "kinetic" models have been developed to describe entire food chains (*16–19*) in which the concentration achieved in the fish is a balance between input and output rates expressed in terms of rate constants. In such models it is not necessary to consider the thermodynamic issue. Notable is the recent model by Thomann (*19*), which involved analysis of a large data base and elucidates the dependence of food chain biomagnification on K_{OW}.

In this paper we review and suggest mechanisms for these processes and assemble a comprehensive model describing uptake of chemical from water and food and losses to water and feces and by metabolism. The model brings together several existing models that have been individually validated for components of the overall process. The model is presented as a hypothesis in the hope that it may be tested and improved as a result of future experimental studies. The approach is to develop the model in fugacity format, suggest equations containing chemical-specific and fish-specific parameters that may be used to correlate or predict process rates, and test the model with available data. Many of these equations have been suggested in previous studies (*7, 8, 20*). It is shown that the "kinetic" and "thermodynamic" models of bioaccumulation, which are occasionally viewed as being inconsistent or competitive, are in reality merely different methods of expressing the same phenomena. Finally, the model is used to explore how differences in chemical properties affect the relative importance of food and water as sources of chemical, and how metabolism and bioavailability influence bioaccumulation.

Model Development in Fugacity Format

Figure 1A shows the fish uptake and loss processes in fugacity terms, in which each process is expressed as a product of a fugacity (f, in Pa) and a rate parameter or D value with units of (mol/Pa·h), thus Df has units of mol/h. Equilibrium is characterized by Z values (mol/m^3·Pa) that relate concentration C (mol/m^3) to fugacity (f) by equating C to Zf. Z values are estimated for the phases of water, lipid, octanol, and whole fish (denoted with subscripts W, L, O, and F, respectively). A dimensionless partition coefficient is merely the ratio of Z values. The calculation of Z and D values has been described in detail elsewhere (*21*), but briefly, three types of D values can apply.

For reactions (such as metabolism) D is the product $V_F Zk$, where k is the reaction rate constant, V_F is fish volume, and Z is for the fish. For bulk flow of chemical D is GZ, where G is the phase flow rate (m^3/h), and Z is for the flowing fluid. This is applied to water flow into and out of the gills, ingestion and egestion. Diffusive processes, for example transfer across membranes, can be expressed as $D(f_1 - f_2)$ where $(f_1 - f_2)$ is the fugacity difference or driving force. The D value can be viewed as a product KAZ where K is a mass-transfer coefficient (m/h), A is area (m^2), and Z is that of the chemical in the medium in which diffusion is occurring. K may be further viewed as a diffusivity divided by a diffusion path length. The

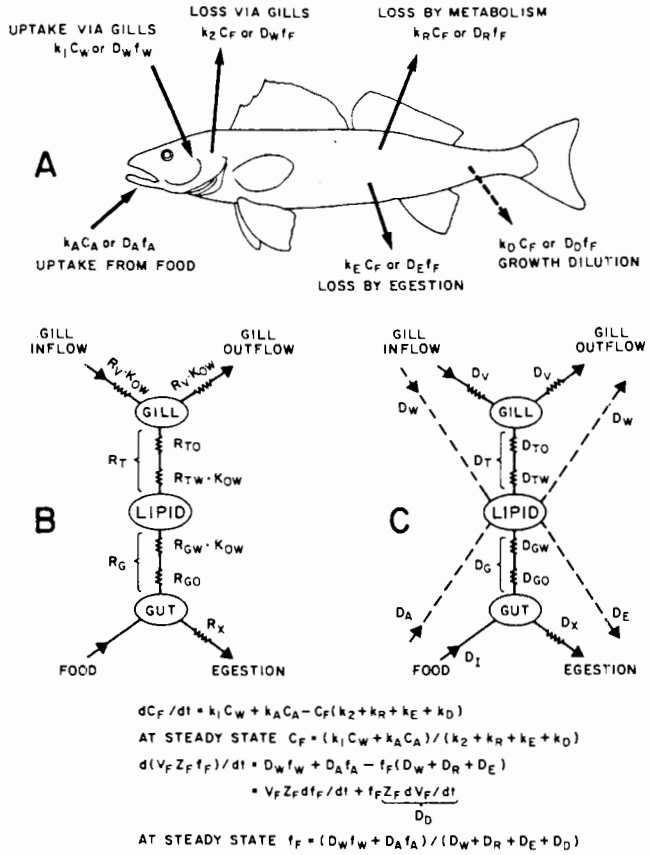

Figure 1. Schematic diagram of chemical transport in a fish (A), with flows expressed in resistance terms (B) and fugacity terms (C).

diffusion may be in a fluid boundary layer or in a stationary membrane.

As shown in Figure 1A, the rates of chemical transfer (in mol/h) may be expressed either in the form of products of rate constants and concentrations or as fugacities and D values. The corresponding differential equations are also given in Figure 1. Included is a term for growth dilution describing the change in concentration attributable to fish growth. The rates are defined as applying to transport into the fish body, i.e., through the epithelium, thus chemical that is in the gill cavity or the gastrointestinal (GI) tract is not included in the fish concentration, C_F. The terms k_1 or D_W, thus include an efficiency of uptake from the gill, and k_A or D_A a similar gut absorption efficiency. The differential equation can be integrated and various boundary conditions applied to give equations expressing C_F as a function of time, which can be fitted to experimental data. At constant exposure conditions dC_F/dt eventually becomes zero, steady state is reached, and C_F can be expressed as shown. The ratio C_F/C_W, the bioaccumulation factor K_B, then becomes dependent on k_A and k_E or D_A and D_E and, if metabolism occurs, on k_R or D_R. Steady state is thus not necessarily a true equilibrium (equifugacity) condition. When these terms are significant, it is expected that purely thermodynamically based correlations between K_B and K_{OW} will break down.

Inherent in this model is the use of the concept of resistance to transfer. These resistances add when they are in series. As has been discussed by Flynn and Yalkowsk (*22*) and Mackay and Hughes (*7*), when resistances in two different phases (e.g., organic and water) are in series, it is necessary to include the phase partition coefficient, usually the octanol–water partition coefficient. Large resistances tend to occur in phases (usually water) in which concentrations are low. Diffusive flux is proportional to

concentration, thus low concentrations constrain fluxes and result in high resistances. Since a D value is a conductivity, its reciprocal, $1/D$, is the resistance.

Equilibrium. At thermodynamic equilibrium K_B or C_F/C_W becomes Z_F/Z_W. If L_F is the lipid or octanol-equivalent volume fraction of the fish, Z_F is $L_F Z_O$, where Z_O is the fugacity capacity of octanol and Z_O/Z_W is K_{OW}; thus K_B is $L_F K_{OW}$. As discussed by Mackay (*5*), L_F is typically approximately 0.05.

Gill Transfer. The upper part of Figure 1C shows a model of gill-transfer processes, which is essentially that of Gobas and Mackay (*8*). It is assumed that the gill acts as a continuous-stirred tank reactor or well-mixed compartment into and out of which water flows, with chemical (and oxygen) being transferred to the fish by diffusion. The fish, enclosed within epithelial tissue, is treated as being at pseudo-steady-state composition and fugacity f_F. D_T contains terms for blood flow resistance and blood-to-lipid transfer as well as gill membrane resistances. The ventilation flow rate G_V (m^3/h) can be used to estimate D_V as $G_V Z_W$. A steady-state mass balance over the gill cavity gives

$$D_V f_W = D_V f_V + D_T(f_V - f_F) \quad (1)$$

thus

$$f_V = (D_V f_W + D_T f_F)/(D_V + D_T) \quad (2)$$

where f_V is the fugacity of the chemical in the water in the gill.

The gill uptake efficiency, E_T, is then given by the ratio of uptake to input:

$$E_T = D_T(f_V - f_F)/D_V f_W = D_T(f_W - f_F)/[(D_V + D_T)f_W] \quad (3)$$

This implies that gill uptake efficiency depends on the state of the fish, having a maximum value E_{TM} of $D_T/(D_V + D_T)$ when the fish is uncontaminated; falling to zero when the fish is in equilibrium with the water and $(f_W - f_F)$ is zero. Measurement of E_T is a convenient method of estimating D_T, because D_V can be measured (with difficulty) from the water flow rate past the gills. For example, a 50% efficiency implies that D_V and D_T are equal.

Following Gobas and Mackay (*8*) and Mackay and Hughes (*7*), it is suggested that D_T is a conductivity or reciprocal resistance made up of resistances in series, which are either "organic" (characterized by octanol) or water phase in nature, thus

$$1/D_T = 1/D_{TO} + 1/D_{TW} \quad (4)$$

Further, D_{TO} and D_{TW}, the organic and water D values, can be expressed as GZ products where G is a fictitious flow rate of organic matter or water, i.e.

$$D_{TO} = G_{TO} Z_O \quad (5)$$

and

$$D_{TW} = G_{TW} Z_W \quad (6)$$

These G values were designated Q_O and Q_W by Gobas and Mackay (*8*).

The relationship between the overall parameter, D_W, and D_T and D_V can now be established by equating the net uptake rates through the gill membrane, using eq 2 as

$$D_W(f_W - f_F) = D_T(f_V - f_F) = D_T D_V(f_W - f_F)/(D_V + D_T) = E_T D_V f_W \quad (7)$$

It follows that

$$D_W = D_T D_V/(D_V + D_T) \quad (8)$$

or

$$1/D_W = 1/D_V + 1/D_T = 1/D_V + 1/D_{TW} + 1/D_{TO} = 1/(G_V Z_W) + 1/(G_{TW} Z_W) + 1/G_{TO} Z_O = (1/G_V + 1/G_{TW})/Z_W + 1/G_{TO} Z_O \quad (9)$$

The overall resistance ($1/D_W$) is thus the sum of three resistances in series, two of which contain Z_W and the third Z_O.

It is noteworthy, as discussed by Gobas and Mackay (*8*), that when K_{OW} or Z_O/Z_W is relatively small, e.g., 10^2, the term $1/D_{TO}$ dominates and transfer is lipid phase controlled. When K_{OW} is larger, e.g., 10^6, the term $1/D_{TO}$ is negligible and transfer is controlled by water-phase flow and diffusion. The reason for this is the constraint introduced by the low concentration in the water phase relative to that in the organic phase.

Gastrointestinal Tract. A similar approach is taken for the GI tract as shown in the lower part of Figure 1C, but in this case it is not feasible to assign the same D value to food ingestion and egestion because digestion causes the food to lose mass and change composition. We follow Gobas et al. (*20*) and Amidon et al. (*23*) by suggesting that absorption be described by assuming that the gut is a well-mixed reactor. A steady-state mass balance gives

$$D_I f_A = D_X f_G + D_G(f_G - f_F) \quad (10)$$

and

$$f_G = (D_I f_A + D_G f_F)/(D_G + D_X) \quad (11)$$

where f_G is the fugacity of the chemical in the gut contents, D_I is the food intake D value, D_X is the egestion value, and D_G expresses resistances to transfer through the gut wall and blood to the lipid tissues, which are the ultimate destination of the chemical. D_I is not equivalent to D_A, nor is D_X equivalent to D_E, because the mass balance is now over the gut contents, not the fish.

The food uptake efficiency is given by the ratio of uptake to input:

$$E_A = D_G(f_G - f_F)/D_I f_A = D_G(f_A - D_X f_F/D_I)/[(D_G + D_X)f_A] \quad (12)$$

Again it follows that E_A depends on the condition of the fish. When the fish is uncontaminated and f_F is zero, the uptake efficiency is a maximum E_{AM} of $D_G/(D_G + D_X)$. As with gill absorption, E_A may be used to estimate D_G if the feeding rate is known. Note that if D_I and D_X were equal, the algebraic form would be identical with that for the gills, the common D value in that case being D_V. In practice it is suspected that D_X is considerably smaller than D_I due to food absorption and especially to fat hydrolysis and absorption.

Following Gobas et al. (*20*), it is suggested that D_G may be treated similarly to D_T, as being comprised of organic and water resistances, i.e.

$$1/D_G = 1/D_{GO} + 1/D_{GW} = 1/G_{GO} Z_O + 1/G_{GW} Z_W \quad (13)$$

The relationship between D_A and D_E, and D_I, D_X, and D_G can now be established by equating the net food uptake rate as

$$D_A f_A - D_E f_F = D_G(f_G - f_F) = [D_G D_I/(D_G + D_X)]f_A - [D_X D_G/(D_G + D_X)]f_F \quad (14)$$

thus

$$D_A = D_G D_I/(D_G + D_X) = E_{AM} D_I \quad (15)$$

$$D_E = D_G D_X/(D_G + D_X) = E_{AM} D_X \quad (16)$$

or

$$1/D_E = 1/D_G + 1/D_X = 1/D_{GO} + 1/D_{GW} + 1/D_X \quad (17)$$

If it is assumed that egestion of chemical is in association with an organic medium which can be expressed in terms of an equivalent octanol flow G_{XO}, then D_X is $G_{XO}Z_O$ and $1/D_E$ can be expressed in water and organic terms as

$$1/D_E = (1/G_{GO} + 1/G_{XO})/Z_O + 1/G_{GW}Z_W \quad (18)$$

The initial or maximum food uptake efficiency is given by

$$E_{AM} = D_G/(D_G + D_X) = (1/D_X)/(1/D_G + 1/D_X) \quad (19)$$

These $1/D$ terms reflect the resistance to egestion $1/D_X$, and gut absorption resistance $1/D_G$. Absorption is thus viewed as a competitive process between absorption and egestion with absorption efficiency being high when $1/D_X$ is large compared to $1/D_G$. Substances of very large K_{OW}, e.g., exceeding 10^7, may be absorbed inefficiently as discussed by Gobas et al. (*20*).

A steady state may be reached at which there is no net gut transfer because f_G equals f_F. Thus, from eq 10, $D_I f_A$ equals $D_X f_G$ and $D_X f_F$, and the ratio of fish to food fugacity becomes D_I/D_X, which we call the "digestion coefficient" and designate as Q. Combining eq 15 and 16

$$D_A/D_E = D_I/D_X = Q = f_F/f_A \text{ (at steady state)} \quad (20)$$

The digestion coefficient, Q, represents the maximum biomagnification factor, or ratio of fish to food fugacity, and has an expected magnitude of 3–10 as discussed by Connolly and Pedersen (*14*).

Metabolism. The D value for metabolism, D_R, can be expressed as $V_F Z_F k_R$.

Overall Mass Balance. The gill, gut, and metabolism D values may be combined to give the overall equation given in Figure 1. If growth is included, it may be treated as a pseudo-D value (D_D), as shown. Clearly, it is possible for the fish fugacity to achieve values ranging up to Q times the food fugacity depending on the relative magnitudes of the D values.

Equivalence of Fugacity and Rate Constant Models

Examination of the fugacity and rate constant equations shows that they are algebraically equivalent as follows:

$$k_1 = D_W/V_F Z_W = k_2 L_F K_{OW} \quad (21)$$

$$k_2 = D_W/V_F Z_F \quad (22)$$

$$k_R = D_R/V_F Z_F \quad (23)$$

$$k_D = D_D/V_F Z_F = (dV_F/dt)/V_F \quad (24)$$

$$k_A = D_A/V_F Z_A = G_I E_A/V_F \quad (25)$$

$$k_E = D_E/V_F Z_F \quad (26)$$

where G_I is the feeding rate and Z_A is the fugacity capacity of the food.

Resistance Parameters

If a set of D values or rate constants is available, the steady- and unsteady-state concentrations in the fish can be determined for various exposure regimes. There is an incentive to devise correlations, and thus a predictive ability, by expressing D values and rate constants in terms of parameters that are separately specific to the fish and to the chemical. This is conveniently done by using resistances, denoted R, which have units of time (h). They can be viewed as the time required to achieve a certain degree of chemical transfer. Thus, a long time implies a high resistance and slow transfer. A schematic diagram of a fish in terms of resistances is shown in Figure 1B. The resistances are related to D values and rate constants as follows.

gill water flow resistance:

$$R_V = V_L/G_V = V_L Z_W/D_V \quad (27)$$

gill membrane water resistance:

$$R_{TW} = V_L/G_{TW} = V_L Z_W/D_{TW} \quad (28)$$

gill membrane organic resistance:

$$R_{TO} = V_L/G_{TO} = V_L Z_O/D_{TO} \quad (29)$$

gut membrane water resistance:

$$R_{GW} = V_L/G_{GW} = V_L Z_W/D_{GW} \quad (30)$$

gut membrane organic resistance:

$$R_{GO} = V_L/G_{GO} = V_L Z_O/D_{GO} \quad (31)$$

egestion resistance:

$$R_X = V_L/G_{XO} = V_L Z_O/D_X \quad (32)$$

The water and organic flow rates G_{TW}, G_{TO}, G_{GW}, G_{GO}, and G_{XO} are fictitious and represent the product of an area and a mass-transfer coefficient. Substituting these expressions into the rate constant equations (eq 21–26) gives

$$1/k_2 = (R_V + R_{TW})K_{OW} + R_{TO} \quad (33)$$

$$1/k_E = R_X + R_{GO} + R_{GW}K_{OW} = R_X + R_G \quad (34)$$

$$E_{TM} = R_V K_{OW}/[(R_V + R_{TW})K_{OW} + R_{TO}] \text{ (maximum value)} \quad (35)$$

$$E_{AM} = R_X/(R_X + R_{GO} + R_{GW}K_{OW}) = R_X/(R_X + R_G) = 1/(1 + R_G/R_X) \text{ (maximum)} \quad (36)$$

or

$$1/E_{AM} = 1 + R_{GO}/R_X + (R_{GW}/R_X)K_{OW}$$

These reciprocal rate constants are thus the sum of resistances in series and the efficiencies are ratios of resistances. The six resistances are specific to the fish, but are believed to vary systematically with fish size and apply to all chemicals that transfer by passive diffusion. The chemical specificity is contained entirely in K_{OW}, at least to a first approximation, and in k_R if metabolism occurs. The parameters required for the model are summarized in Table I.

An analogous resistance term, R_I, could be defined for food uptake as V_L/G_{IO} where G_{IO} is the octanol-equivalent inflow of food such that D_I is $G_{IO}Z_O$. It then follows that Q, which is the ratio D_I/D_X and D_A/D_E, is also R_X/R_I and G_{IO}/G_{XO}, i.e., it is the ratio of the egestion resistance to the feeding resistance. A high value of Q implies that the fish experiences difficulty in egesting the chemical, thus having a tendency to retain and biomagnify it.

Bioavailability in the Water Column

McCarthy and Jimenez (*24*) and Landrum et al. (*25*) have convincingly demonstrated that the presence of sorbing material such as humic acids in the water column reduces bioavailability and hence uptake of chemicals by aquatic organisms. The effect is particularly important for substances of high K_{OW}. To quantify this we assume that the chemical is partly in sorbed state in the water with a dimensionless partition coefficient K_{PW}, and the con-

Table I. Model Parameters

	Fundamental Parameters
K_{OW}	octanol–water partition coefficient (chemical specific)
L_F	fish lipid or octanol-equivalent volume fraction
L_A	food lipid or octanol-equivalent volume fraction
R_V	gill ventilation flow rate resistance (h)
R_{TW}	gill membrane water-phase resistance (h)
R_{TO}	gill membrane organic-phase resistance (h)
k_R	metabolism rate constant (h^{-1})
G_I/V_F	feeding rate (m^3 food/h)/(m^3 fish volume) or (h^{-1})
R_{GW}	gut absorption water resistance (h)
R_{GO}	gut absorption organic resistance (h)
Q	digestion coefficient (ratio of ingestion to egestion D values)
k_D	growth dilution rate constant (h^{-1})
	Secondary or Derived Parameters
K_B	equilibrium bioconcentration factor
E_{TM}	gill uptake efficiency (eq 35)
E_{AM}	food uptake efficiency (eq 36)
R_T	gill membrane total resistance
R_G	gut membrane total resistance
R_X	egestion resistance (eq 32)
k_1	gill uptake rate constant (eq 21)
k_2	gill depuration rate constant (eq 22)
k_A	food uptake rate constant (eq 25)
k_E	excretion rate constant (eq 26)

centration of sorbent is a volume fraction X (typically 10^{-5}). A mass balance gives

$$C_W = C_T/(1 + K_{PW}X) \tag{37}$$

where C_T and C_W are, respectively, the total and dissolved concentrations. Conventionally X is expressed in units of kg/L and K_{PW} in units of L/kg. K_{PW} is usually related to organic carbon content and K_{OW}.

DiToro (*26*) suggested that the organic carbon be treated as equivalent to octanol, i.e., K_{PW} is yK_{OW} where y is the organic carbon content (g/g) of the suspended matter. There is some controversy about the dependence of K_{PW} on sorbent concentrations (*27, 28*), but there is no doubt that the presence of appreciable quantities of sorbent reduces bioavailability, i.e., C_W is less than C_T, or equivalently, the fugacity of the chemical in the water is reduced.

This issue is peripheral to the central task of describing the kinetics of chemicals in fish, but it can become an important determinant of bioaccumulation. We suggest the simple expedient of employing an "octanol-equivalent" sorbent concentration Y (volume fraction) in the water such that the sorptive capacity $K_{PW}X$ is equal to $K_{OW}Y$. Following DiToro, Y is then approximately the concentration (kg/L) of suspended organic carbon in the water. We thus sidestep the issue of determining K_{PW}, or relating X to Y, but we quantify the effect by introducing Y, a typical range of environmental values being 10^{-6}–10^{-5} kg/L. The water fugacity is thus controlled by the dissolved concentration, C_W. We assume that only the dissolved chemical is available for transfer through the gill surface, the sorbed chemical passing through the gill cavity unchanged.

Fish Size Dependence

It is suggested that the resistances are dependent on fish size. As discussed by Gobas and Mackay (*8*), it appears that diffusion, volumetric ventilation, and circulation rates increase as fish size increases but at a rate less than proportional to volume. Since the resistances can be regarded as ratios of fish volume to a flow rate, resistance is expected to increase with fish volume. It is thus postulated that the terms R_V, R_{TW}, R_{TO}, R_{GO}, and R_{GW} increase in proportion to fish volume raised to a low power, n, possibly in the range 0.2–0.4. The feeding rate (G_I/V_F) may also fall with increasing fish volume (V_F). For each resistance, an appropriate correlation is

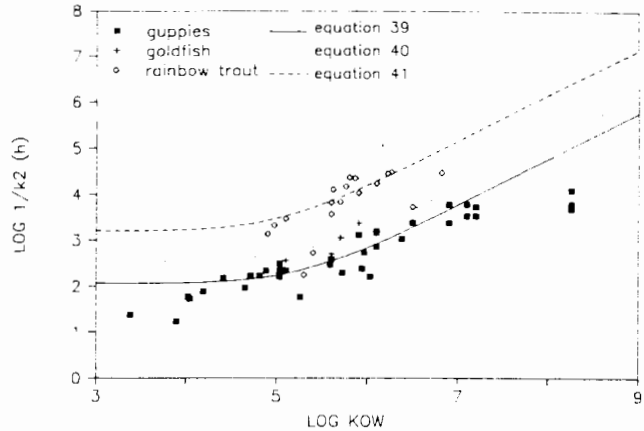

Figure 2. Plot of the log of the inverse elimination rate constant ($1/k_2$) as a function of log K_{OW}, for guppies, goldfish, and rainbow trout. The lines refer to eqs 39–41 in the text.

$$R_i = A_i V_i^n \tag{38}$$

where A_i is a constant.

Fitting Parameters

The following procedure may be used to fit the model to experimental data.

From fish uptake-clearance experiments k_1, k_2, and K_B can be estimated by conventional procedures. If data are available for a series of chemicals, with a range of K_{OW}, the parameters R_{TO} and ($R_V + R_{TW}$) can be determined as the intercept and slope, respectively, of a plot of $1/k_2$ (the inverse elimination rate constant) versus K_{OW} (eq 33). Care must be taken in this regression to assign correct weights to the data points, because $1/k_2$ may vary by many orders of magnitude. We prefer to fit initially by inspection, i.e., "by eye", but a nonlinear regression technique may be used. If gill uptake efficiency data into clean fish are available, R_V and R_{TW} may be estimated by using eq 35, otherwise subsequent calculations can be performed using the sum of these terms.

Food uptake efficiency data into clean fish, for chemicals with a range of K_{OW}, may be used to estimate the ratios R_{GO}/R_X and R_{GW}/R_X (eq 36). Estimating the absolute values of R_{GO}, R_{GW}, and R_X requires a determination of Q. There are two options. If the flows and properties of the food and the feces are known, Q may be estimated as the ratio of octanol-equivalent flows G_{IO}/G_{XO}, but this may not be possible because of the complex nature of these phases. Gobas et al. (*20*) measured the fecal rate to be 37% of the volumetric food consumption rate for guppies fed Tetramin at 2% of fish volume per day. Thus, if the sorptive characteristics of food and feces are similar, a Q of approximately 3 is expected. The second method uses eq 20 and is to measure the fish and food fugacities after prolonged exposure to a conservative chemical, as was essentially done by Connolly and Pedersen (*14*). This yields a value of 3–10. For the purposes of this study we select a value of 3, recognizing that it may be in error.

The metabolic transformation rate is best estimated by comparison of bioconcentration data for the metabolizing chemical with that of a chemical of similar K_{OW}, which is known to be conservative.

Table II presents experimentally measured inverse elimination rate constants ($1/k_2$) and food absorption efficiencies (E_A) for guppies, goldfish, and rainbow trout, for

Table II. Experimentally Measured Inverse Elimination Rate Constants ($1/k_2$) and Food Absorption Efficiencies for Guppies, Goldfish, and Rainbow Trout[a]

chemical	log K_{OW}	$1/k_2$, day	E_A	chemical	log K_{OW}	$1/k_2$, day	E_A
				Guppies			
1,4-dibromobenzene	3.89 (*31*)	0.708 (*29*)		2,2′,3,3′,4,4′,5,5′-octachlorobiphenyl	7.1 (*32*)	250 (*12*)	
1,3,5-tribromobenzene	5.26 (*31*)	2.40 (*29*)		decachlorobiphenyl	8.26 (*32*)	500 (*12*)	
4,4′-dibromobiphenyl	5.72 (*31*)	8.13 (*29*)		2-monochloronaphthalene	4.19 (*9*)	3.24 (*9*)	
2,4,6-tribromobiphenyl	6.03 (*31*)	6.76 (*29*)		1,4-dichloronaphthalene	4.88 (*9*)	9.12 (*9*)	
2,2′,5,5′-tetrachlorobiphenyl	6.1 (*32*)	61.7 (*29*)		1,8-dichloronaphthalene	4.41 (*9*)	6.31 (*9*)	
2,2′,5,5′-tetrabromobiphenyl	6.5 (*31*)	102 (*29*)		2,3-dichloronaphthalene	4.71 (*9*)	7.08 (*9*)	
2,2′,4,4′,6,6′-hexabromobiphenyl	7.2 (*31*)	141 (*29*)		2,7-dichloronaphthalene	4.81 (*9*)	7.08 (*9*)	
				1,3,7-trichloronaphthalene	5.59 (*9*)	12.0 (*9*)	
2,4,5-trichlorobiphenyl	5.6 (*32*)	15.8 (*29*)		1,2,3,4-tetrachloronaphthalene	5.94 (*9*)	10.0 (*9*)	
decachlorobiphenyl	8.26 (*32*)	200 (*29*)		1,3,5,7-tetrachloronaphthalene	6.38 (*9*)	45.7 (*9*)	
mirex	6.9 (*33*)	219 (*29*)		1,3,5,8-tetrachloronaphthalene	5.96 (*9*)	22.4 (*9*)	
pentachlorobenzene	5.03 (*34*)	6.61 (*12*)		pentachlorobenzene	5.03 (*34*)	12.9 (*9*)	
2,5-dichlorobiphenyl	5.1 (*32*)	9.12 (*12*)		2,3′,4′,5-tetrachlorobiphenyl	5.9 (*32*)	55.0 (*9*)	
2,2′,5,5′-tetrachlorobiphenyl	6.1 (*32*)	66.1 (*12*)	0.51 (*12*)	1,4-dichlorobenzene	3.38 (*34*)	1.00 (*35*)	
2,2′,4,4′,5,5′-hexachlorobiphenyl	6.9 (*32*)	251 (*12*)	0.51 (*12*)	1,2,3-trichlorobenzene	4.04 (*34*)	2.24 (*35*)	
				1,3,5-trichlorobenzene	4.02 (*34*)	2.51 (*35*)	
2,2′,3,3′,4,4′,5,5′-octachlorobiphenyl	7.1 (*32*)	141 (*12*)	0.31 (*12*)	1,2,3,5-tetrachlorobenzene	4.65 (*34*)	3.89 (*35*)	
				pentachlorobenzene	5.03 (*34*)	9.12 (*35*)	
decachlorobiphenyl	8.26 (*32*)	251 (*12*)	0.19 (*12*)				
2,2′,5,5′-tetrachlorobiphenyl	6.1 (*32*)	30.3 (*12*)					
2,2′,4,4′,5,5′-hexachlorobiphenyl	6.9 (*32*)	100 (*12*)					
				Goldfish			
2,5-dichlorobiphenyl	5.1 (*32*)	15.2 (*11*)	0.56 (*11*)	2,2′,5,5′-tetrachlorobiphenyl	6.1 (*32*)	66.7 (*11*)	0.53 (*11*)
2,2′,5-trichlorobiphenyl	5.6 (*32*)	20.8 (*11*)	0.49 (*11*)	2,3′,4′,5-tetrachlorobiphenyl	5.9 (*32*)	99.9 (*11*)	0.48 (*11*)
2,4′,5-trichlorobiphenyl	5.7 (*32*)	47.6 (*11*)	0.6 (*11*)				
				Rainbow Trout			
3,3′-dichlorobiphenyl	5.3 (*32*)	7.22 (*36*)	0.62 (*36*)	2,4,6,2′,5′-pentachlorobiphenyl	6.22 (*37*)	1184 (*36*)	0.75 (*36*)
3,5-dichlorobiphenyl	5.4 (*32*)	21.7 (*36*)	0.80 (*36*)	2,3,4,2′,5′-pentachlorobiphenyl	6.5 (*32*)	224 (*36*)	0.75 (*36*)
2,2′-dichlorobiphenyl	4.9 (*32*)	57.7 (*36*)	0.79 (*36*)	2,4,6,3′,4′-pentachlorobiphenyl	6.58 (*37*)	>1443 (*36*)	0.80 (*36*)
2,3-dichlorobiphenyl	4.97 (*37*)	88.0 (*36*)	0.77 (*36*)	2,4,6,2′,6′-pentachlorobiphenyl	5.81 (*37*)	>1443 (*36*)	0.73 (*36*)
2,5-dichlorobiphenyl	5.1 (*32*)	123 (*36*)	0.73 (*36*)	2,3,4,5,6-pentachlorobiphenyl	6.3 (*32*)	>1443 (*36*)	0.85 (*36*)
2,5,2′-trichlorobiphenyl	5.6 (*32*)	274 (*36*)	0.77 (*36*)	2,4,5,2′,5′-pentachlorobiphenyl	6.4 (*32*)	>1443 (*36*)	0.78 (*36*)
2,5,4′-trichlorobiphenyl	5.7 (*32*)	283 (*36*)	0.78 (*36*)	2,3,4,5,2′,5′-hexachlorobiphenyl	6.82 (*37*)	1227 (*36*)	0.84 (*36*)
3,4,3′,4′-tetrachlorobiphenyl	6.1 (*32*)	63.5 (*36*)	0.68 (*36*)	2,3,4,6,2′,4′-hexachlorobiphenyl	6.67 (*37*)	>1443 (*36*)	0.77 (*36*)
2,3,2′,3′-tetrachlorobiphenyl	5.6 (*32*)	154 (*36*)	0.73 (*36*)	2,3,4,2′,3′,4′-hexachlorobiphenyl	6.74 (*37*)	>1443 (*36*)	0.78 (*36*)
2,3,4,5-tetrachlorobiphenyl	5.9 (*32*)	450 (*36*)	0.78 (*36*)	2,4,6,2′,4′,6′-hexachlorobiphenyl	7.0 (*32*)	>1443 (*36*)	0.64 (*36*)
2,5,2′,6′-tetrachlorobiphenyl	5.62 (*37*)	527 (*36*)	0.75 (*36*)	2,4,5,2′,4′,5′-hexachlorobiphenyl	6.9 (*32*)	>1443 (*36*)	0.75 (*36*)
2,3,2′,4′-tetrachlorobiphenyl	5.76 (*37*)	620 (*36*)	0.66 (*36*)	2,3,4,5,3′,4′-hexachlorobiphenyl	7.18 (*37*)	>1443 (*36*)	0.76 (*36*)
2,5,2′,5′-tetrachlorobiphenyl	6.1 (*32*)	722 (*36*)	0.74 (*36*)	2,3,4,5,2′,3′,4′,5′-octachlorobiphenyl	7.1 (*32*)	>1443 (*36*)	0.78 (*36*)
2,3,5,6-tetrachlorobiphenyl	5.86 (*37*)	938 (*36*)	0.77 (*36*)	2,3,4,5,6,2′,3′,4′,5′-nonachlorobiphenyl	7.2 (*32*)	>1443 (*36*)	0.80 (*36*)
2,4,3′,4′-tetrachlorobiphenyl	5.8 (*32*)	967 (*36*)	0.83 (*36*)				
2,5,3′,5′-tetrachlorobiphenyl	6.26 (*37*)	1285 (*36*)	0.76 (*36*)	decachlorobiphenyl	8.26 (*32*)	>1443 (*36*)	0.63 (*36*)

[a] References shown in parentheses.

chemicals of log K_{OW} 3.4–8.3. Figure 2 is a plot of the logarithm of the inverse elimination rate constants as a function of log K_{OW} for each organism. Regression to estimate k_2 is best done for chemicals of relatively low log K_{OW} (i.e., <6.5), since as Gobas et al. (*29*) have shown, fecal elimination (k_E) may exceed k_2 for very hydrophobic chemicals. The lines on Figure 2 represent the results of the following linear regressions (95% confidence intervals in parentheses).

guppies:

$$1/k_2 = 5.80\ (\pm 1.03) \times 10^{-4} K_{OW} + 115\ (\pm 310)\ \text{h} \quad (39)$$

goldfish:

$$1/k_2 = 1.39\ (\pm 0.77) \times 10^{-3} K_{OW} + 343\ (\pm 668)\ \text{h} \quad (40)$$

rainbow trout:

$$1/k_2 = 1.42\ (\pm 0.33) \times 10^{-2} K_{OW} + 1585\ (\pm 7012)\ \text{h} \quad (41)$$

Figure 3 presents a plot of the inverse of the experimental food absorption efficiencies as a function of log K_{OW} for the data set in Table II. The lines in the figure represent the following linear regressions (95% confidence intervals in parentheses).

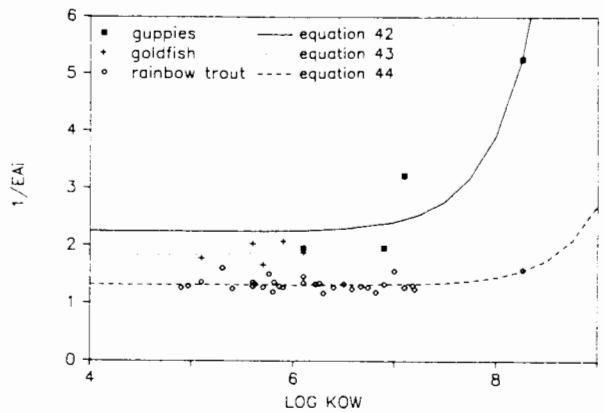

Figure 3. Plot of the inverse of the food absorption efficiency as a function of log K_{OW}, for guppies, goldfish, and rainbow trout. The lines refer to eqs 42–44 in the text.

guppies:

$$1/E_A = 1.67\ (\pm 0.43) \times 10^{-8} K_{OW} + 2.25\ (\pm 0.66) \quad (42)$$

goldfish:

$$1/E_A = 1.02\ (\pm 2.25) \times 10^{-7} K_{OW} + 1.83\ (\pm 0.19) \quad (43)$$

rainbow trout:

$$1/E_A = 1.39\ (\pm 0.57) \times 10^{-9} K_{OW} + 1.32\ (\pm 0.10) \quad (44)$$

These correlations are regarded as only tentative in view of the paucity of data, especially for high-K_{OW} chemicals. The term containing K_{OW} only becomes significant when log K_{OW} exceeds 6.5. The other term almost certainly has a value in the range 1.3–2.5, corresponding to absorption efficiencies ranging from 40 to 77%.

The resistances calculated for guppies, goldfish, and rainbow trout in eq 39–44 are summarized in Table III. The values of R_X were calculated by assuming that the digestion coefficient Q was 3 for each organism.

Resistances may then be related by fish size with eq 38. Figure 4 shows a plot of log resistance as a function of log fish volume. A linear regression for each resistance (95% confidence interval in parentheses) yielded (for R in h, V in m^3)

aqueous gill resistance

$$\log R_W = \log (R_V + R_{TW}) = 0.36\ (\pm 0.05) \log V - 0.81\ (\pm 0.15) \quad (45)$$

or

$$R_W = 0.15 V^{0.36}$$

organic gill resistance

$$\log R_{TO} = 0.29\ (\pm 0.003) \log V + 4.1\ (\pm 0.01) \quad (46)$$

or

$$R_{TO} = 1.26 \times 10^4 V^{0.29}$$

The slopes in Figure 4, and equivalently the power on V, must be regarded as of questionable accuracy or even significance. The values should be regarded as only illustrative, but they are consistent with the analysis by Gobas and Mackay (*8*), who suggested that the gill resistances are related to the organism volume raised to a power between 0.2 and 0.4.

The regressed slopes of the R_{GO} and R_{GW} points are 0.12 and −0.04, respectively, which are believed to differ insignificantly from zero. Thus, in the absence of more data, the simplest expedient is to assume that R_{GO} and R_{GW} are independent of organism size, R_{GO} has a value of 2000 h, and R_{GW} is 3×10^{-5} h.

Additional data are needed to test the assumption for the digestion coefficient, Q. It is emphasized that our present aim is not to fit the model rigorously to all available data, or even to test it thoroughly. Our primary objective is to obtain reasonable parameter values and then to examine and disscuss the model's ability to reproduce observed phenomena.

Discussion

We now discuss several features of the model and its ability to reproduce observed bioaccumulation phenomena.

Limitations. The model treats neutral organic chemicals and is not suitable for substances such as chlorinated phenols that ionize or display unusual phase-partitioning behavior. It is likely that the resistances are dependent to some extent on the diffusivity of the chemical in water and organic media. This dependence is not included but could be, given sufficient data. Processes of renal, biliary, or reproductive loss or dermal exchange are not included.

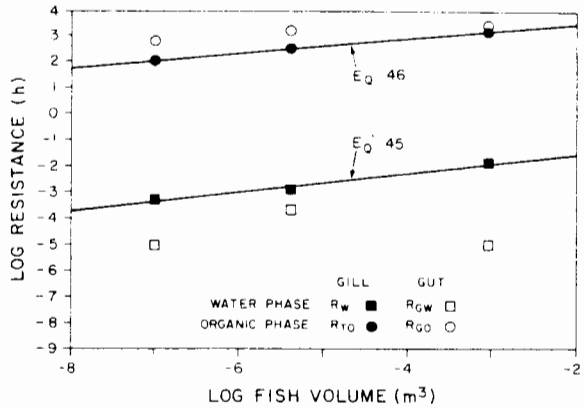

Figure 4. Plot of the log of each resistance as a function of the log of the organism volume, showing eqs 45 and 46. The symbols refer to the following: R_{GO}, organic gut resistance; R_{TO}, organic gill resistance; R_{GW}, aqueous gut resistance; R_W, aqueous gill resistance.

The equations reflect a situation of regular food intake and may be invalid if food consumption is highly intermittent.

The following discussion treats the "steady-state" condition. No true steady state can be achieved if growth occurs, but a "pseudo steady state" can be postulated by setting the differential equation to zero as shown in Figure 1. A problem then arises because the resistance parameters are size dependent and the condition of a growing fish depends not only on the current resistances but also on the (lower) resistances that occurred when the fish was smaller. This is not regarded as a serious limitation, except when the fish is growing rapidly. Accurate simulation is then best done by solving the differential equation numerically.

Simple Bioconcentration. After prolonged exposure, the fish concentration settles at a value

$$C_F = (k_1 C_W + k_A C_A)/(k_2 + k_R + k_E + k_D) \quad (47)$$

and equivalently, the fugacity at

$$f_F = (D_W f_W + D_A f_A)/(D_W + D_R + D_E + D_D)$$
$$= (D_W f_W + E_{AM} D_I f_A)/(D_W + D_R + E_{AM} D_I/Q + D_D)$$

If water is the only source of chemical (C_A and f_A are zero), the chemical is not metabolically transformed, and there is no growth, then

$$C_F/C_W = k_1/(k_2 + k_E) \quad (48)$$

or

$$f_F/f_W = D_W/(D_W + D_E)$$

This suggests that C_F/C_W should approach a limit that is smaller than $L_F K_{OW}$ by an amount controlled by the relative magnitude of k_E and k_2 or D_E and D_W. The success of K_B–K_{OW} correlations suggests that in many cases k_E is small compared to k_2, or D_E is small compared to D_W, i.e., the resistance to exchange through the gills is small compared to that by excretion.

Biomagnification. Recently, Connolly and Pedersen (*14*) and Oliver and Niimi (*15*) suggested that C_F/C_W values in lakes may exceed $L_F K_{OW}$, or equivalently, that fish fugacities exceed that of water, the increase being largest at high trophic levels, and for high-K_{OW} chemicals. The model accounts for this effect, which can be attributed to the term $k_A C_A$. Ignoring growth dilution and metabolism the steady-state BCF will be

$$C_F/C_W = [k_1 + k_A(C_A/C_W)]/(k_2 + k_E) \quad (49)$$

This ratio can exceed k_1/k_2, especially if C_A/C_W becomes large. The amount of excess depends on how k_A, k_2, and

Table III. Resistances (h) and Volumes (cm^3) for Three Fish Species

organism	V_F	L_F	L_A	G_I[a]	$R_V + R_{TW}$	R_{TO}	R_{GW}	R_X	R_{GO}	Q
guppies	0.10	0.02	0.10	2.4	0.00058	115	1.0×10^{-5}	600	750	3
goldfish	4.0	0.029	0.10	1	0.0014	343	2.1×10^{-4}	2088	1733	3
rainbow trout	900	0.097	1	0.1	0.0142	1586	1.0×10^{-5}	7200	2326	3

[a] In % V_F/day.

k_E decrease relative to each other as K_{OW} increases. Rearranging the fugacity differential equation and ignoring metabolism and growth gives

$$f_F/f_W = [D_W + D_A(f_A/f_W)]/(D_W + D_E) \quad (50)$$

Now if the food fugacity is equal to the water fugacity, as is likely for small food organisms, then

$$f_F/f_W = (D_W + D_A)/(D_W + D_E) \quad (51)$$

Clearly, if D_A exceeds D_E, a f_F/f_W ratio greater than unity is expected. Further, when a steady state is reached then $(f_G - f_F)$ in eq 14 will become zero and

$$D_A f_A = D_E f_F \quad (52)$$

and

$$f_F/f_A = D_A/D_E = Q$$

Now, it is likely that D_E is considerably smaller than D_A because the volume of food egested is less than that ingested, and the lipid content is also reduced. If, for example, D_E was 20% of D_A, f_F would approach a value Q of 5 times f_A. Q is thus also the maximum fugacity ratio for the system of food and fish. This high fugacity will drive chemical across the gut wall and induce a high fish fugacity and thus biomagnification. We suggest that biomagnification may be caused by the increase in chemical fugacity in the gut, caused by food digestion. This effect is present, but is not observed, at low K_{OW} because of rapid gill exchange. Only when the gill resistance becomes large, and transfer is slow, is biomagnification apparent. It may be possible to define Q values and hence characterize biomagnification for each fish–food combination.

Detailed Kinetics. We now examine the detailed chemical flows using a hypothetical example of a fish with properties listed in Table IV, exposed to a slowly metabolizing chemical (half-life of 3 years) of log K_{OW} 6, as illustrated in Figure 5. The total inflow of chemical in the gill water is 2.10 nmol/h, of which 1.05 is in dissolved form (available for transfer) and 1.05 is in sorbed form (i.e., equipartitioning). The water fugacity is 0.250 mPa and the total water concentration is 0.001 g/m^3. At steady state (infinite time) the fish concentration and fugacity reach 0.199 mol/m^3 (39.7 g/m^3) and 0.397 mPa, respectively. The fish/water concentration ratio is then 39 700. Since the fish achieves a higher fugacity than the water there is a tendency for the fish to lose chemical by diffusion through the gills. The gill water settles at an intermediate fugacity of 0.312 mPa, resulting in diffusion across the membrane (in units of nmol/h) of 0.442 in, 0.703 out, and 0.261 net diffusion from fish to gill water.

Similarly in the GI tract, when the fugacity of the food is equal to the fugacity of the water, the food input is 1.04 nmol/h. Digestive processes cause the fugacity of the chemical in the gut to increase, by a factor of 2, to 0.525 mPa, a value sufficient to achieve rates of transport to the fish and feces that equal the input rate. These transfer rates (in nmol/h) are 0.729 to the feces, 0.666 from the GI tract to the fish, and 0.353 from the fish to the GI tract, resulting in a net absorption of 0.313 nmol/h.

Table IV. Properties of Hypothetical Fish used in Figure 5

fish properties: $V_F = 10\ cm^3 = 1 \times 10^{-5}\ m^3$
$L_F = 0.05$
$L_A = 0.05$
$R_V + R_{TW} = 2.38 \times 10^{-3}$ h, assume that R_V and R_{TW} are equal (i.e., 1.19×10^{-3} h each)
$R_{TO} = 447$ h
$R_{GW} = 3.0 \times 10^{-5}$ h
$R_{GO} = 2000$ h
$Q = 3$
$G_I/V_F = 2\%$/day
$T_R = 3$ years
$k_D = 0\ h^{-1}$
molecular weight of chemical = 200
$H = 100$ Pa·m^3/mol
[water] = 1 mg/m^3
[sorbent] = 1 ppm

Metabolism results in a loss of 0.052 nmol/h, finalizing the mass balance.

The overall picture is thus of a fish having net input of chemical only from food and output through the gills and feces, and by metabolism. There is "negative" gill uptake.

The situation when the fish has an initial zero chemical concentration is quite different. The inflows of chemical are the same as above, but there is absorption from the gills of 0.442 nmol/h and absorption through the gut of 0.666 nmol/h. The uptake routes and efficiencies thus change with the changing contamination status of the fish. Initially uptake is through the gills; then this reverses as the fish becomes contaminated. There is thus an inherent difficulty in using data from laboratory experiments involving exposure of "clean" fish to assess the toxicokinetics of "field" fish, which are in a near-steady-state contamination condition.

The bioconcentration literature frequently questions and discusses the relative roles of water and food as sources of exposure of chemical to the fish. This analysis suggests that the roles change with level of contamination. It is important to define "exposure" because there is always exposure through the gills, but that exposure may not lead to uptake. It is also necessary to define "the fish", i.e., whether or not the gill cavity and GI tract are included in the fish. Different definitions lead to quite different answers; thus the question must be posed carefully.

The contributions of food and water can be explored by running the model with uptake from food and water, food only, and water only. Because the equations are linear, the concentrations and fluxes obtained from combined exposure equal the sum of the individual contributions, i.e., "linear additivity" applies as has been discussed by Stiver and Mackay (38). In this case the steady-state fish concentrations and fugacities are

	concn, g/m^3	fugacity, mPa
food exposure only	23.9	0.239
water exposure only	15.8	0.158
food and water exposure	39.7	0.397

Since the water fugacity is 0.250 mPa, it is clear that exposure to water results in the fish fugacity approaching

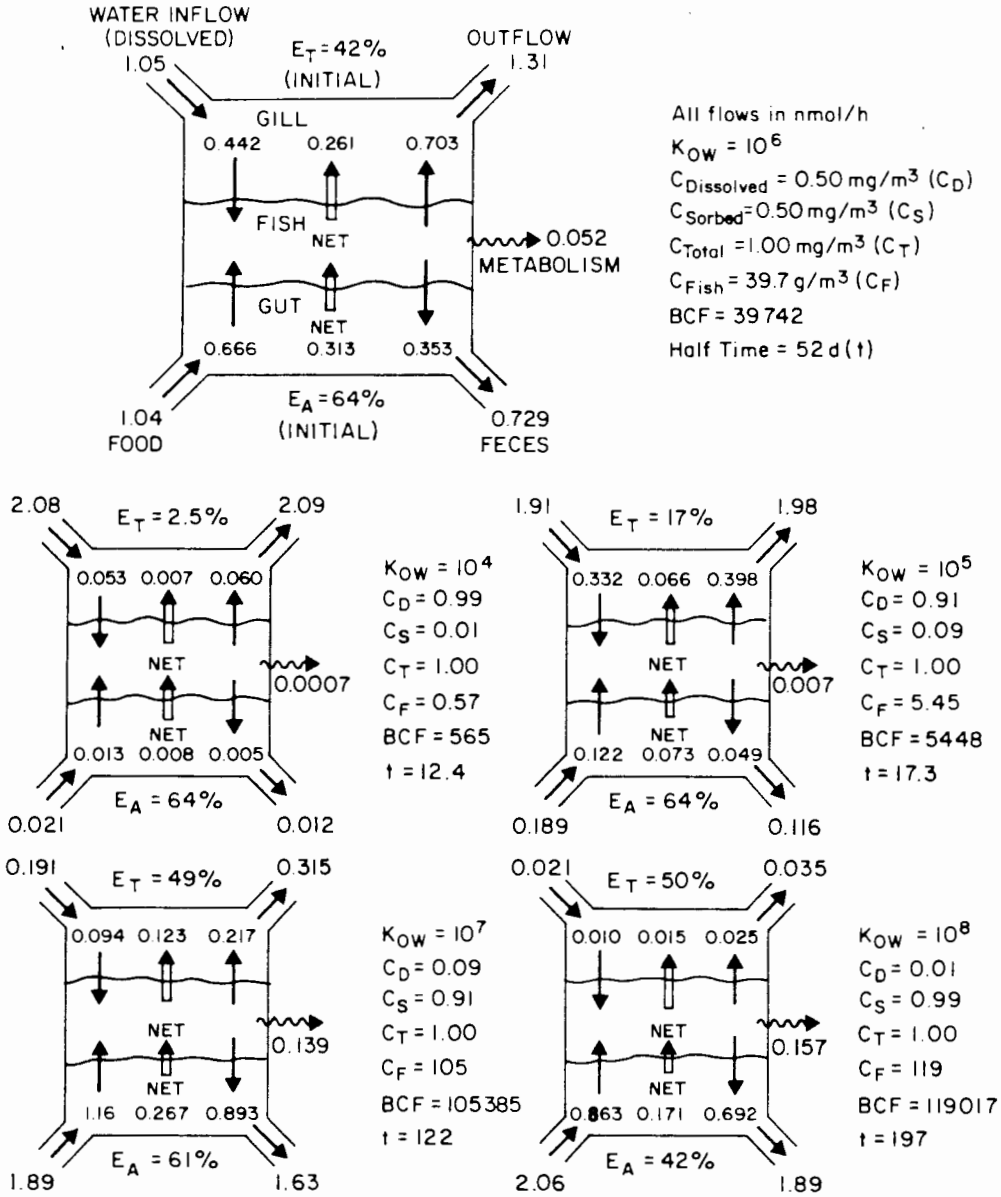

Figure 5. Detailed chemical flows in a fish for a range of K_{OW} values.

the water fugacity, but it is prevented from reaching the water fugacity as a result of loss by egestion. Food ingestion causes the fugacity to increase and exceed that of the water.

The overall half-time to achieve steady state is 52 days. This figure is controlled by the overall rate constant for loss of 5.58×10^{-4} h^{-1}, which comprises 3.54×10^{-4} loss from gills, 1.78×10^{-4} loss by egestion, and 0.26×10^{-4} loss by metabolism. Gill exchange and egestion thus control the uptake and clearance times.

Effect of Hydrophobicity. We now explore the effects of changing the chemical's K_{OW} on the contaminant dynamics in the fish. This is shown as a series of mass balances in Figure 5. The total water concentration is constant at 0.001 g/m^3, but the extent of sorption increases with increasing K_{OW}, as shown in the figure. Thus, the bioavailable fraction of the chemical is reduced, although the total gill water inflows are constant. The fugacity of the chemical in the food is equal to the fugacity in the water in all cases and, thus, as K_{OW} increases the concentration in the food increases, but not in proportion to K_{OW} because of the bioavailability or sorption effect. Therefore, although the quantity of food ingested is constant, the intake of chemical from the food increases only slightly with increasing K_{OW}. Indeed, the principal effect of reduced bioavailability is to control food concentration and hence intake rather than control gill exchange.

When K_{OW} is low (10^4–10^5), virtually all the chemical is in dissolved form in the water; at log K_{OW} of 6, half is sorbed and half is dissolved; and at log K_{OW} 8, nearly all the chemical is in sorbed form. This difference in bioavailable fraction is reflected in the flow of bioavailable chemical in the gill water. At a log K_{OW} of 4, the flow of bioavailable chemical in and out of the gills is 2.08 and 2.09 nmol/h, respectively. These flows decline to 0.021 and 0.035 nmol/h for a chemical of log K_{OW} of 8, although the total water concentration remains constant at 1.00 mg/m^3, as does the total chemical flow.

The ratio of the fish to water concentration increases from 565 at a log K_{OW} of 4 to 105 385 at a log K_{OW} of 7 and remains approximately constant (119 017 at a log K_{OW} of 8). The declining bioavailable fraction of the chemical is the principal reason for the loss of the linear relationship between bioconcentration factor and K_{OW}. Reduction in gut uptake efficiency also plays a role. In all steady-state cases the flow of chemical out of the gills exceeds the flow in, so that the net movement of chemical is from the gills to the water. The ratio of the fugacity of the fish to that

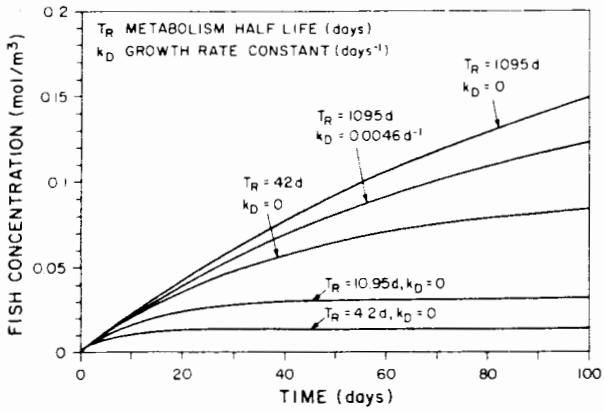

Figure 6. Effect of metabolism on fish concentration, T_R being the chemical half-life (days) and of growth rate, expressed as a growth rate constant (day^{-1}), (k_D = 0.0046 day^{-1} representing a doubling of volume in 150 days).

of the water increases from nearly unity (1.14 at a log K_{OW} of 4) to 2.40 at a K_{OW} of 8.

The fugacity of the food was set equal to that of the water. As K_{OW} increases the concentration of the chemical in the food increases, so that the flow of chemical from food to the GI tract increases from 0.021 nmol/h at a log K_{OW} of 4 to 2.06 nmol/h at a log K_{OW} of 8. This 100-fold increase is the net result of an increase in food–water partitioning, and a decrease in chemical bioavailability. The net flow of chemical from the GI tract increases from 0.008 nmol/h at log K_{OW} of 4 to 0.267 nmol/h at a log K_{OW} of 7. At a higher K_{OW} (10^8), uptake of chemical from the GI tract to the fish declines to 0.171 nmol/h due to increased gut membrane resistance. This increased resistance is also reflected in the initial uptake efficiencies for the gut, which are steady at ~64% for log K_{OW} up to 7 and then decline to 42% at log K_{OW} of 8.

The overall half-time to steady state, representing the time delay in uptake of chemical, increases with K_{OW} from 12.4 days at a log K_{OW} of 4, to 197 days at a log K_{OW} of 8. The metabolism half-time for the hypothetical chemicals is constant at 1095 days (3 years). As the half-time to steady state increases and approaches the metabolism half-time, metabolism becomes an increasingly important route of chemical elimination. At a log K_{OW} of 4, only 0.0007 nmol/h of chemical is metabolized, while at a log K_{OW} of 8, 0.157 nmol/h is metabolized. Thus, at very high K_{OW}'s more chemical is removed by metabolism than through the gills.

Effect of Metabolic Half-Time. Figure 6 illustrates the effect of altering the metabolism half-time on the fish concentration. The hypothetical chemical has a log K_{OW} of 6. The fish properties are listed in Table IV. The upper line in Figure 6 represents a near-conservative chemical with a metabolic half-time of 1095 days (3 years), which is essentially an infinite half-time. The overall half-time for chemical uptake in the fish is much smaller (52 days), and metabolism is thus not an important route of chemical elimination. When the metabolic half-time is decreased to 42 days, the fish concentration at steady state is reduced. Also, less time is required to reach steady state. When the rate of metabolism is rapid (half-time of 4.2 days), the fish concentration and the time required to reach steady state are greatly reduced.

Effect of Growth Dilution. In the other examples, the fish is assumed to have a constant volume. If a growth dilution term is included corresponding to a doubling in volume in 150 days, i.e., a rate constant k_D of 0.0046 day^{-1}, the effect is as shown in Figure 6. The concentrations (or fugacities) are lower in the growing fish. Growth dilution plays a role similar to metabolism and is most important for high K_{OW} chemicals, which exchange slowly with water.

Table V. Application of the Model to a Hypothetical Food Chain[a]

large fish	V_F = 10000 cm^3	f_4 = 13.8 × 10^{-4} Pa
↑		
medium fish	V_F = 100 cm^3	f_3 = 5.93 × 10^{-4} Pa
↑		
small fish	V_F = 1 cm^3	f_2 = 3.30 × 10^{-4} Pa
↑		
plankton		$f_1 = f_W$
↑		
water		f_W = 2.50 × 10^{-4} Pa

[a] Resistances for fish were calculated by using eqs 45 and 46. Feeding rate for fish is 5% of V_F per day and Q = 3. Lipid contents for all levels, 5%. Each level is food for the one above, with f_1 set equal to f_W.

Food Chain Biomagnification. Connolly and Pedersen (*14*) observed that the chemical fugacity in fish, in the field, was higher than the fugacity in the water in which the fish lived. The fugacity also increased with each level of the food chain. To test if the model reflects these observations, a trophic level simulation may be set up as shown in Table V by running the model sequentially using fish from one level as food for the next, and a chemical of log K_{OW} of 6. The first-level food (plankton) is assumed to be in equilibrium with the water (fugacity 2.50 × 10^{-4} Pa). A small fish (1 cm^3) consumes the plankton at a rate of 5% of the fish's volume per day and reaches a steady-state fugacity of 3.30 × 10^{-4} Pa. A medium fish (100 cm^3) then consumes the small fish, also at a rate of 5% of fish volume per day. The medium fish has a fugacity of 5.9 × 10^{-4} Pa at steady state. The largest fish (10 000 cm^3) consumes the medium fish at a rate of 5% per day and reaches a fugacity of 13.8 × 10^{-4} Pa. It is therefore apparent that the model accounts for the observation, described by Connolly and Pedersen (*14*), that fugacity increases with trophic level. At each stage of the food chain the fish fugacity becomes an increasing multiple of the water fugacity. As has been discussed by Clark et al. (*30*), a further increase in fugacity occurs for fish-eating birds, which achieve a fugacity considerably higher than the fish they consume.

Conclusions

A comprehensive model has been developed describing the processes of exchange of organic chemicals between water, food, and fish. The model can be used in the form of rate constants that are dependent on resistances. These resistances have been shown to have physiological significance using a fugacity model and expressions for size dependence have been suggested. The model has been shown to give a satisfactory description of the phenomena of bioconcentration, biomagnification, metabolism, bioavailability, and food chain magnification for a range of chemical hydrophobicities. It demonstrates that the detailed flows of contaminant to and from the fish, through the gills and gut, vary considerably in relative magnitude and direction as the fish becomes contaminated. It is hoped that the model will be tested and, if necessary, modified by fitting to new experimental data.

Registry No. 1,3,5-Tribromobenzene, 626-39-1; 4,4′-dibromodiphenyl, 92-86-4; 2,4,6-tribromobiphenyl, 59080-33-0; 2,2′,5,5′-tetrachlorobiphenyl, 35693-99-3; 2,2′,5,5′-tetrabromobiphenyl, 59080-37-4; 2,2′,4,4′,6,6′-hexabromobiphenyl, 59261-08-4; 2,4,5-trichlorobiphenyl, 15862-07-4; decachlorobiphenyl, 2051-24-3; mirex, 2385-85-5; 2,5-dichlorobiphenyl, 34883-39-1;

2,2′,4,4′,5,5′-hexachlorobiphenyl, 35065-27-1; 2,2′,3,3′,4,4′,5,5′-octachlorobiphenyl, 35694-08-7; 1,4-dichloronaphthalene, 1825-31-6; 1,8-dichloronaphthalene, 2050-74-0; 2,3-dichloronaphthalene, 2050-75-1; 2,7-dichloronaphthalene, 2198-77-8; 1,3,7-trichloronaphthalene, 55720-37-1; 1,2,3,4-tetrachloronaphthalene, 20020-02-4; 1,3,5,7-tetrachloronaphthalene, 53555-64-9; 1,3,5,8-tetrachloronaphthalene, 31604-28-1; pentachlorobenzene, 608-93-5; 2,3′,4′,5-tetrachlorobiphenyl, 32598-11-1; 1,2,3-trichlorobenzene, 87-61-6; 1,3,5-trichlorobenzene, 108-70-3; 1,2,3,5-tetrachlorobenzene, 634-90-2; 2,2′,5-trichlorobiphenyl, 37680-65-2; 2,4′,5-trichlorobiphenyl, 16606-02-3; 3,3′-dichlorobiphenyl, 2050-67-1; 3,5-dichlorobiphenyl, 34883-41-5; 2,2′-dichlorobiphenyl, 13029-08-8; 2,3-dichlorobiphenyl, 16605-91-7; 3,4,3′,4′-tetrachlorobiphenyl, 32598-13-3; 2,3,2′,3′-tetrachlorobiphenyl, 38444-93-8; 2,3,4,5-tetrachlorobiphenyl, 33284-53-6; 2,5,2′,6′-tetrachlorobiphenyl, 41464-41-9; 2,3,2′,4′-tetrachlorobiphenyl, 36559-22-5; 2,3,5,6-tetrachlorobiphenyl, 33284-54-7; 2,4,3′,4′-tetrachlorobiphenyl, 32598-10-0; 2,5,3′,5′-tetrachlorobiphenyl, 41464-42-0; 2,4,6,2′,5′-pentachlorobiphenyl, 60145-21-3; 2,3,4,2′,5′-pentachlorobiphenyl, 38380-02-8; 2,4,6,3′,4′-pentachlorobiphenyl, 56558-17-9; 2,4,6,2′,6′-pentachlorobiphenyl, 56558-16-8; 2,3,4,5,6-pentachlorobiphenyl, 18259-05-7; 2,4,5,2′,5′-pentachlorobiphenyl, 37680-73-2; 2,3,4,5,2′,5′-hexachlorobiphenyl, 52712-04-6; 2,3,4,6,2′,4′-hexachlorobiphenyl, 56030-56-9; 2,3,4,2′,3′,4′-hexachlorobiphenyl, 38380-07-3; 2,4,6,2′,4′,6′-hexachlorobiphenyl, 33979-03-2; 2,3,4,5,3′,4′-hexachlorobiphenyl, 38380-08-4; 2,3,4,5,6,2′,3′,4′,5′-nonachlorobiphenyl, 40186-72-9; water, 7732-18-5.

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