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A FOOD WEB BIOACCUMULATION MODEL FOR ORGANIC CHEMICALS IN AQUATIC ECOSYSTEMS

JON A. ARNOT and FRANK A.P.C. GOBAS*
School of Resource and Environmental Management, Simon Fraser University, 8888 University Drive, Barnaby, BC V5A 1S6, Canada

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Abstract—The present study examines a new bioaccumulation model for hydrophobic organic chemicals in aquatic food webs. The purpose of the model is to provide site-specific estimates of chemical concentrations and associated bioconcentration factors, bioaccumulation factors, and biota–sediment accumulation factors in organisms of aquatic food webs using a limited number of chemical, organism, and site-specific data inputs. The model is a modification of a previous model and incorporates new insights regarding the mechanism of bioaccumulation derived from laboratory experiments and field studies as well as improvements in model parameterization. The new elements of the model include: A model for the partitioning of chemicals into organisms; kinetic models for predicting chemical concentrations in algae, phytoplankton, and zooplankton; new allometric relationships for predicting gill ventilation rates in a wide range of aquatic species; and a mechanistic model for predicting gastrointestinal magnification of organic chemicals in a range of species. Model performance is evaluated using empirical data from three different freshwater ecosystems involving 1,019 observations for 35 species and 64 chemicals. The effects of each modification on the model's performance are illustrated. The new model is able to provide better estimates of bioaccumulation factors in comparison to the previous food web bioaccumulation model while the model input requirements remain largely unchanged.

Keywords—Bioaccumulation Bioaccumulation factor Biota–sediment accumulation factor Screening level risk assessment Aquatic ecosystem

INTRODUCTION

Bioaccumulation is a fundamental process in environmental toxicology and risk assessment, because it controls the internal dose of potential toxicants [1]. Information regarding chemical accumulation is important in determining environmental-quality guidelines, establishing total maximum daily loadings, categorizing substances that are potential hazards, and quantifying the risk of chemicals on ecosystems and human health [2–5] (http://laws.justice.gc.ca/en/C-15.31). Typical measures for assessing bioaccumulation include the octanol–water partition coefficient (K_{OW}), bioconcentration factor (BCF), bioaccumulation factor (BAF), and biota–sediment accumulation factor (BSAF). These can be obtained from empirical measurements and from mathematical models. Empirical information is often preferable, but models are also quite useful, particularly when empirical measurements do not exist or cannot be made for either technical or economic reasons.

A number of useful bioaccumulation models have been proposed. The earliest dealt with bioaccumulation from the water (or bioconcentration). Based on the studies of Hamelink et al. [6], simple equilibrium partitioning models (i.e., log K_{OW} − log BCF models) were put forward by Neely et al. [7] and Veith et al. [8], followed by many others for species ranging from phytoplankton to fish. Then came simple two-compartment (i.e., organism–water) kinetic models (see, e.g., [9]) that described the exchange of chemicals between organisms and water. The introduction of a fugacity-based bioconcentration model by Mackay [10] established a solid theoretical footing for these bioconcentration models, which further evolved in several directions. Physiological models were developed by Barber et al. [11,12]. Physiologically based pharmacokinetic (PBPK) models for bioconcentration in fish were developed by Nichols et al. [13], Law et al. [14], and others. Kinetic models were pursued by several authors [15–17], and fugacity-based descriptions of the bioconcentration process were refined by Gobas and Mackay [18]. Recognition of the role of dietary uptake and biomagnification in the bioaccumulation of hydrophobic organic contaminants (HOCs) resulted from the studies of Bruggeman et al. [19], Connolly and Pedersen [20], Muir and Yarechewski [21], and several others (see, e.g., [22]) as well as from the models of Norstrom et al. [23]. Several bioaccumulation models for fish that include both dietary and gill uptake were developed (see, e.g., [24,25]), and Mackay and coworkers produced theoretical formulations of these processes [26]. At the same time, the exchange of chemicals between sediments and benthic invertebrates became better understood [27,28]. This work resulted in a number of models, ranging from simple equilibrium partitioning [29–32] to more detailed kinetic models [33,34]. Bioaccumulation of HOCs in phytoplankton was studied by Geyer et al. [35], Swackhamer and Skoglund [36–38], and several other investigators (see, e.g., [39−41]).

The enormous progress made since Hamelink's PhD work has cleared the way for a generation of models with the ability to track the movement of chemicals through aquatic food webs and to estimate BAFs and BSAFs under actual environmental conditions. Several food web models have emerged. Thomann and colleagues [42,43] as well as Gobas [44] have developed kinetic food web models, whereas Campfens and Mackay [45] have developed a fugacity-based food web bioaccumulation model. These food web models combine the work of many investigators. It is to the credit of all those who have studied—and who continue to study—uptake and bioaccumulation that these models are increasingly used by regulators, engineers,

* To whom correspondence may be addressed (gobas@sfu.ca).

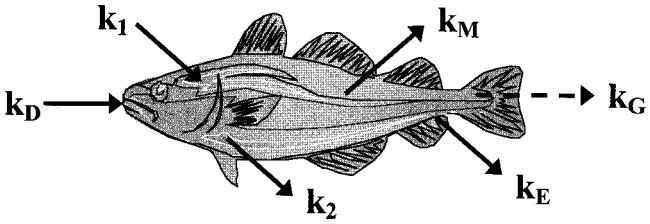

Fig. 1. A conceptual diagram representing the major routes of chemical uptake and elimination in an aquatic organism. k_D = dietary uptake rate constant; k_1 = gill uptake rate constant; k_2 = gill elimination rate constant; k_M = metabolic transformation rate constant; k_E = fecal egestion rate constant; k_G = growth dilution rate constant.

and toxicologists to conduct a range of activities. For example, bioaccumulation models are being used to screen new and existing chemicals for their potential to bioaccumulate [46], to develop water- and sediment-quality criteria [29,32,47], to develop total maximum daily loadings and remediation targets for impacted aquatic ecosystems [48], to assess the exposure of biota affected by pollution sources [49], and to determine the responsiveness to cleanup efforts [50,51].

The purpose of the present study is to examine another step in the evolution of bioaccumulation models. This paper presents new formulations of the bioaccumulation of HOCs in aquatic food webs resulting from insights obtained during recent laboratory experiments [52,53], analyses of field data [37,54], and improvements in data availability for model parameterization [55]. The food web model is presented in rate constant format for assessing the bioaccumulation of HOCs in aquatic ecosystems. The model is limited to species of aquatic macrophytes, algae, phytoplankton, zooplankton, invertebrates, and fish of different trophic levels. The purpose of the model is to provide site-specific estimates of chemical concentrations and associated BCFs, BAFs, and BSAFs. The model is a modified version of the 1993 Gobas food web model [44]. Four fundamental elements that differ from the original model include a new model for the partitioning of chemicals into organisms, a new kinetic model for predicting chemical concentrations in algae and phytoplankton, new allometric relationships for predicting gill ventilation rates in a wide range of aquatic species, and a new mechanistic model for predicting gastrointestinal magnification of organic chemicals. The model performance is evaluated with external empirical data from three different freshwater ecosystems involving 1,019 observations for 35 species and 64 chemicals. The effects of the modifications on the model are illustrated by comparing the performance of the revised model to that of the 1993 food web bioaccumulation model.

THEORY

General model

Figure 1 provides a conceptual overview of major routes of chemical uptake and elimination in aquatic organisms. Our model is based on the presumption that the exchange of nonionic organic chemicals between the organism and its ambient environment can be described by a single equation for a large number of aquatic organisms:

$$dM_B/dt = \left\{ W_B \cdot \left(k_1 \cdot [m_O \cdot \phi \cdot C_{WT,O} + m_P \cdot C_{WD,S}] + k_D \cdot \sum (P_i \cdot C_{D,i}) \right) \right\} - (k_2 + k_E + k_M) \cdot M_B \quad (1)$$

where M_B is the mass (g) of chemical in the organism, dM_B/dt is the net flux of parent chemical being absorbed or depurated by the organism at any point in time t (d), W_B is the weight of the organism (kg) at time t, k_1 is the clearance rate constant (L/kg · d) for chemical uptake via the respiratory area (i.e., gills and skin), m_O is the fraction of the respiratory ventilation that involves overlying water, m_P is the fraction of the respiratory ventilation that involves sediment-associated pore water, ϕ (unitless) is the fraction of the total chemical concentration in the overlying water that is freely dissolved and can be absorbed via membrane diffusion, $C_{WT,O}$ is the total chemical concentration in the water column above the sediments (g/L), $C_{WD,S}$ is the freely dissolved chemical concentration in the sediment associated pore (or interstitial) water (g/L), k_D is the clearance rate constant (kg/kg · d) for chemical uptake via ingestion of food and water, P_i is the fraction of the diet consisting of prey item i, $C_{D,i}$ is the concentration of chemical (g/kg) in prey item i, k_2 is the rate constant (d^{-1}) for chemical elimination via the respiratory area (i.e., gills and skin), k_E is the rate constant (d^{-1}) for chemical elimination via excretion into egested feces, and k_M is the rate constant (d^{-1}) for metabolic transformation of the chemical. For phytoplankton, algae, and macrophytes, k_D is zero, and k_E is considered to be insignificant.

This model is based on several key assumptions. First, it is assumed that the chemical is homogeneously distributed within the organism as long as differences in tissue composition and phase partitioning are taken into account. Considerable evidence, especially for poorly metabolizable substances after long exposure periods, supports this assumption (see, e.g., [56,57]). However, because the model is not designed to estimate concentrations in specific organs, it is best applied in situations when the mass or concentration of the chemical in the whole organism is of interest. Internal PBPK models are more suitable to estimate the differences in concentration between various parts of the organism. Second, it is assumed that the organism can be described as a single compartment in terms of its exchange with its surrounding environment. Many studies can be quoted to support this (see, e.g., [9]). The one-compartment model for an organism is best applied in situations when variations in concentration over time are relatively slow or of secondary concern. A third assumption of the model concerns the chemical elimination via egg deposition or sperm ejection. Studies in fish have shown that lipid-normalized concentrations of many persistent organic chemicals in eggs and adult female fish are equal (see, e.g., [57]). This implies that whereas egg deposition transfers a significant fraction of the chemical body burden from the adult female fish into the eggs, the lipid equivalent concentration within the organism remains the same. The mechanism in the model by which egg deposition can lower the internal concentration in the organism compared to fish that do not produce eggs (see, e.g., for male fish, [58]) is through growth dilution associated with the formation of eggs in the fish. Growing eggs produce extra tissue in which the chemical resides, hence reducing the chemical's concentration. However, Equation 1 illustrates that this growth dilution effect is counteracted by uptake of chemical from both water and the diet and that the balance of these processes controls the ultimate concentration in the organism.

The practical application of Equation 1 to problems of environmental pollution typically is limited by access to time-dependent model input parameter values. Hence, for the model to become useful, it often needs to be further simplified by

applying a steady-state assumption ($dM_B/dt = 0$), resulting in

$$C_B = \left\{k_1 \cdot (m_O \cdot \phi \cdot C_{WT,O} + m_P \cdot C_{WD,S}) + k_D \cdot \sum P_i \cdot C_{D,i}\right\} / (k_2 + k_E + k_G + k_M) \quad (2)$$

where C_B is the chemical concentration in the organism (g/kg ww; i.e., M_B/W_B). The BAF is then $C_B/C_{WT,O}$, and the wet weight–based BSAF ($BSAF_W$) is C_B/C_S, where C_S is the concentration (g/kg dry sediment) in the bottom sediment. The steady-state assumption is reasonable for applications to field situations in which organisms have been exposed to the chemical over a long period of time, often throughout their entire life. It applies best to chemicals that are subject to relatively fast exchange kinetics (e.g., lower-K_{OW} substances, small organisms), because steady-state is achieved rapidly in these situations. It should be used with caution when the exchange kinetics are relatively slow (e.g., slowly metabolizable chemicals of high K_{OW} [i.e., $>10^{7.5}$] in large, lipid-rich organisms), because steady state takes a long time to achieve. When changes in concentrations with the age of the organism are of interest, it is possible to introduce various age classes of the species and then apply the steady-state model to each age class independently. One of the implications of applying a steady-state assumption is that the growth of the organism needs to be expressed as a growth rate constant k_G, which is $dW_B/(W_B \cdot dt)$ and assumes that over the period of time during which the model applies, the growth of the organism can be represented by a constant fraction of the organism's body weight. The main driving forces of the kinetic bioaccumulation model are the chemical partitioning of chemical between water and the organism, represented by k_1/k_2; the dietary digestion of the prey and subsequent partitioning of chemical between the gastrointestinal tract (GIT) and the organism, represented by k_D/k_E; metabolic transformation; and growth dilution.

Phase partitioning

The partitioning of organic chemicals between biological organisms and water is believed to occur into lipid, nonlipid organic matter (NLOM; e.g., proteins and carbohydrates), and water. Each of these media has its own capacity to sorb and store the chemical. Hence, for every organism, we define an organism–water partition coefficient (K_{BW}) on a wet weight basis as

$$K_{BW} = k_1/k_2 = v_{LB} \cdot K_{OW} + v_{NB} \cdot \beta \cdot K_{OW} + v_{WB} \quad (3)$$

where v_{LB} is the lipid fraction (kg lipid/kg organism ww), v_{NB} is the NLOM fraction (kg NLOM/kg organism ww), v_{WB} is the water content (kg water/kg organism ww) of the organism, and β is a proportionality constant expressing the sorption capacity of NLOM to that of octanol. Based on previous work [53], a value of approximately 0.035 is a reasonable first estimate of β; however, further research is required to better characterize β. This suggests that the nonionic organic chemical sorption affinity of NLOM is approximately 3.5% that of octanol. Whereas the sorption affinity of NLOM is low compared to that of lipid, it can play an important role in controlling the partitioning of organic chemicals in organisms with low-lipid content (e.g., phytoplankton, algae, certain invertebrates). Good databases exist (see, e.g., [59]) to parameterize the three-phase partitioning model, especially for fish, crustaceans, and shellfish consumed by humans.

Submodels

To estimate k_1, k_2, k_E, k_M, k_G, and ϕ for different chemicals in aquatic organisms of food webs, we propose the following submodels.

ϕ. Hydrophobic organic contaminants have a high affinity for organic matter, such as particulate organic carbon (POC) and dissolved organic carbon (DOC) in the water column [16,60,61]. If associated with particulate or dissolved organic matter, the chemical is believed to be unavailable for uptake via diffusion into organisms. Therefore, ϕ is the ratio of the freely dissolved water concentration $C_{WD,O}$ (g/L) to the total water concentration $C_{WT,O}$ (g/L) in the overlying water. In the absence of reliable empirical data, ϕ can be estimated for nonionizing HOCs as

$$\phi = C_{WD}/C_{WT} = 1/(1 + \chi_{POC} \cdot D_{POC} \cdot \alpha_{POC} \cdot K_{OW} + \chi_{DOC} \cdot D_{DOC} \cdot \alpha_{DOC} \cdot K_{OW}) \quad (4)$$

where χ_{POC} and χ_{DOC} are the concentrations of POC and DOC in the water (kg/L), respectively; D_{POC} and D_{DOC} are the disequilibrium factors for POC and DOC partitioning, respectively, and represent the degree to which POC–water and DOC–water distribution coefficients vary from POC–water and DOC–water equilibrium partition coefficients; and α_{POC} and α_{DOC} are proportionality constants describing the similarity in phase partitioning of POC and DOC, respectively, in relation to that of octanol. Disequilibrium factors for POC partitioning or D_{DOC} values greater than 1.0 indicate distribution coefficients in excess of equilibrium partition coefficients, whereas values less than 1.0 represent conditions in which equilibrium has not been reached. Disequilibrium factors for POC partitioning and D_{DOC} values equal to 1.0 represent equilibrium partitioning. Disequilibria between organic carbon and water have been observed for a range of organic chemicals (including polychlorinated biphenyls, polycyclic aromatic hydrocarbons, and chlorobenzenes) in several ecosystems (see, e.g., [54]), but their values remain difficult to predict at this point. The proportionality constants α_{POC} and α_{DOC} can vary substantially among different types of organic carbon. Based on a literature survey, Seth et al. [62] suggested that α_{POC} can be estimated as 0.35 (with error bars equivalent to a factor of 2.5) for chemicals that range in log K_{OW} from 1.5 to 7.5. Burkhard [63] has suggested that α_{DOC} can be estimated as 0.08, with a similar magnitude of variability. Differences in composition of DOC and POC (especially when soot carbon is involved; see, e.g., [64,65]) likely play a key role. When reliable measured POC and DOC distribution coefficients are available, it therefore often is preferable to use the empirical data to represent the distribution coefficients $D_{POC} \cdot \alpha_{POC} \cdot K_{OW}$ and $D_{DOC} \cdot \alpha_{DOC} \cdot K_{OW}$ in Equation 4.

k_1 and k_2. The rate at which chemicals are absorbed from the water via the respiratory surface (e.g., gills and skin) is expressed by the aqueous uptake clearance rate constant k_1 (L/kg · d). In fish, invertebrates, and zooplankton, it can be viewed as a function of the ventilation rate G_V (L/d) and the diffusion rate of the chemical across the respiratory surface area [18,44]:

$$k_1 = E_W \cdot G_V/W_B \quad (5)$$

where E_W is the gill chemical uptake efficiency and W_B is the wet weight of the organism (kg). The E_W is a function of the

K_{OW} of the chemical and can be approximated based on observations in fish by [66]:

$$E_W = (1.85 + (155/K_{OW}))^{-1} \quad (6)$$

In many cases, G_V or a related measure (i.e., oxygen consumption rate) can be obtained from literature sources. Good databases exist for fish (see, e.g., [55]). Similar data are less accessible for invertebrates. In the absence of good-quality data, G_V can be approximated from an allometric relationship between wet weight and oxygen consumption. For example, based on 3,573 observations under routine metabolic test conditions from approximately 200 different fish species ranging in weight between 2.0×10^{-5} and 60 kg, oxygen consumption V_{OX} (mg O_2/d) can be estimated [55] ($r^2 = 0.84$) as

$$V_{OX} = 980 \cdot W_B^{0.65} \quad (7)$$

Adding oxygen consumption rate and G_V data for zooplankton and aquatic invertebrate species (SETAC Supplemental Data Archive S.1) produces only a slight modification to this equation, suggesting that a single linear relationship provides a reasonable model for ventilation rates in zooplankton, invertebrate, and fish species:

$$G_V = 1{,}400 \cdot W_B^{0.65}/C_{OX} \quad (8)$$

where C_{OX} is the dissolved oxygen concentration (mg O_2/L) and a function of temperature [67] as

$$C_{OX} = (-0.24 \cdot T + 14.04) \cdot S \quad (9)$$

where T is water temperature (°C) and S is the degree of oxygen saturation of the water column (%). Because the relationship is derived from oxygen consumption directly, it is not necessary to include an oxygen uptake efficiency [55].

For algae, phytoplankton, and aquatic macrophytes, we propose a biphasic relationship for k_1 and k_2 based on a water–organic carbon two-phase resistance model:

$$k_1 = (A + (B/K_{OW}))^{-1} \quad (10)$$

where A and B are constants (with units of time) describing the resistance to chemical uptake through, respectively, the aqueous and organic phases of the algae, phytoplankton, or macrophyte. To obtain reasonable values for A and B for phytoplankton, we evaluated several datasets. Constant B (default value = 5.5) is derived by calibration to empirical k_2 values from various phytoplankton, algae, and cyanobacteria species over a range of K_{OW} values [68–71] (SETAC Supplemental Data Archive S.2). Constant A (default value = 6.0×10^{-5}) is derived from calibration to phytoplankton field-BCF data from the Great Lakes [37,54] such that k_G begins to control the BCF for chemicals with a log K_{OW} larger than 6.0. A mean annual k_G value of 0.08 d^{-1} is selected from the same empirical data in which slow-growth conditions (winter) were 0.03 d^{-1} and active-growth conditions (summer) were 0.13 d^{-1} [37].

The elimination rate constant k_2 (d^{-1}) is closely related to k_1, because both k_1 and k_2 involve the same processes of water ventilation and membrane permeation. The k_2 is determined as k_1/K_{BW}. This also applies to phytoplankton. However, in the calculation of the phytoplankton–water partition coefficient (K_{PW}), NLOM in Equation 3 is replaced by nonlipid organic carbon (kg/kg organism ww) with a proportionality constant of 0.35 (i.e., $K_{PW} = v_{LP} \cdot K_{OW} + v_{NP} \cdot 0.35 \cdot K_{OW} + v_{WP}$). Since the BAF is a function of the ratio of k_1 and k_2, errors in the exact determination of G_V and E_W typically have a minor effect on the BAF, because errors in k_1 will cancel out similar errors in k_2. This makes the model relatively insensitive to parameterization error in G_V and E_W and allows a single equation to represent ventilation rates and uptake efficiencies in a range of species. The partitioning properties of the chemical, represented by K_{BW}, play a more important role. This is reasonable, because the main roles of k_1 and k_2 are to describe how quickly or slowly equilibrium partitioning in the organism will be achieved. The model is most sensitive to k_1 and k_2 for substances that are absorbed from water and food in comparable amounts and/or that are eliminated by gill ventilation at rates comparable to the combined elimination rate of fecal egestion, metabolic transformation, and growth dilution.

m_O *and* m_P. Organisms that are in close contact with the bottom sediments, such as benthic fish and invertebrates, can exchange chemicals with the pore water. Freely dissolved chemical concentrations in pore water can exceed the overlying water concentrations as a result of sediment–water disequilibria, which can be very large under certain conditions (see, e.g., [43]). In many cases, benthic fish and invertebrates do not ventilate a large amount of pore water because of poor oxygen concentrations and low food content. Although pore-water ventilation may be small, it can have a large effect on the BAF for chemicals that are at large sediment–water column disequilibria. In most cases, values for m_P are equal to or less than 5% (see, e.g., [72]). For organisms that have no direct contact with the pore water, m_P is zero. In all cases, m_O equals $1 - m_P$.

$C_{WD,P}$. Freely dissolved chemical concentrations in pore water can be estimated from the chemical concentration in the sediment [73] as

$$C_{WD,P} = C_{S,OC}/K_{OC} \quad (11)$$

where $C_{WD,P}$ is the freely dissolved chemical concentration in the pore water (g/L), $C_{S,OC}$ is the chemical concentration in the sediment normalized for organic carbon content (g/kg organic carbon), and K_{OC} is the organic carbon–water partition coefficient (L/kg organic carbon).

k_D *and* k_E. The rate at which chemicals are absorbed from the diet via the GIT is expressed by the dietary uptake clearance rate constant k_D (kg food/kg organism · d) and is a function of the dietary chemical transfer efficiency E_D, the feeding rate G_D (kg/d), and the weight of the organism W_B (kg) [44]:

$$k_D = E_D \cdot G_D/W_B \quad (12)$$

Empirical E_D observations are highly variable in aquatic invertebrates, ranging between 0 and 100% in amphipods, mollusks, oligochaetes, snails, clams, and bivalves [27,34,74–79] and between 0 and 90% in fish [52,75,80–83]. Explanations have been proposed for the variations in E_D, including differences among the sorption coefficient of chemicals in dietary matrices, composition of dietary matrices (e.g., organic carbon and soot carbon content), digestibility of the dietary matrix, metabolic transformation, steric hindrance in gut membrane permeation, experimental artifacts, differences in gut morphology, and variability in food digestion between different species. Because of the large variability in the empirical data, it is difficult to develop accurate models for the dietary uptake rate. However, some notable trends in the E_D data can provide guidance in model development. First, several authors have observed a reduction in dietary uptake efficiency with increasing K_{OW} for high-K_{OW} chemicals in invertebrates [75,76] and fish [75,80]. Second, the average dietary chemical transfer efficiency (E_D) for chemicals with a log K_{OW} of between four

and six is approximately 50% in aquatic invertebrates and fish that were fed continuously. These trends are consistent with a two-phase resistance model for gut–organism exchange, which is further documented elsewhere [66,80]. The following equation based on the lipid–water two-phase resistance model was selected to represent the relationship between dietary chemical absorption efficiencies and K_{OW} (SETAC Supplemental Data Archive S.3):

$$E_D = (3.0 \times 10^{-7} \cdot K_{OW} + 2.0)^{-1} \quad (13)$$

Empirical data concerning feeding rates of different organisms are often available. The use of such data in the model is preferable, but caution should be exercised to consider the energetic content of the food and select growth rates that are consistent with the energy intake of the organism. In the absence of relevant empirical feeding rates, we suggest a general bioenergetic relationship based on studies in trout [84] for estimating feeding rates in coldwater fish species. In some cases, this equation can also be used to estimate feeding rates in zooplankton and aquatic invertebrate species (as demonstrated in the SETAC Supplemental Data Archive S.1):

$$G_D = 0.022 \cdot W_B^{0.85} \cdot \exp(0.06 \cdot T) \quad (14)$$

Filter-feeding species have a distinct mechanism of dietary uptake that can be represented by a modifying Equation 14 [34] to

$$G_D = G_V \cdot C_{SS} \cdot \sigma \quad (15)$$

where the feeding rate is a product of gill ventilation rate G_V (L/d), the concentration of suspended solids C_{SS} (kg/L), and the scavenging efficiency of particles σ (%) absorbed from the water.

The rate at which chemicals are eliminated by the egestion of fecal matter can be expressed by the fecal elimination rate constant k_E (d^{-1}) [52]:

$$k_E = G_F \cdot E_D \cdot K_{GB}/W_B \quad (16)$$

where G_F (kg feces/kg organism · d) is the fecal egestion rate and K_{GB} is the partition coefficient of the chemical between the GIT and the organism. The G_F is a function of the feeding rate and the digestibility of the diet, which in turn is a function of the composition of the diet according to

$$G_F = \{(1 - \varepsilon_L) \cdot v_{LD}) + (1 - \varepsilon_N) \cdot v_{ND} + (1 - \varepsilon_W) \cdot v_{WD}\} \cdot G_D \quad (17)$$

where ε_L, ε_N, and ε_W are the dietary assimilation efficiencies of lipid, NLOM, and water, respectively, and v_{LD}, v_{ND}, and v_{WD} are the overall lipid, NLOM, and water contents of the diet, respectively. In fish, the assimilation efficiencies of lipid and NLOM are approximately 92% and 60%, respectively [53,83]. Dietary assimilation efficiencies for invertebrates range from 15 to 96% [75,85–87]. In general, these efficiencies are a reflection of the dietary matrix (e.g., organic matter quantity and quality) and of the digestive physiology of the organism (e.g., feeding rates and gut retention time). Species with low assimilation efficiencies (e.g., worms) typically feed on poor-quality substrate (e.g., sediment or detritus) but maintain high feeding rates to obtain required nutrients for energy budgets and survival. A value of 75% is suggested for lipid and NLOM assimilation efficiencies in aquatic invertebrates. In zooplankton, assimilation efficiencies for organic matter range from 55 to 85% [88], whereas carbon and phosphorus assimilation are measured at approximately 85% [89]. A value of 72% is assumed for lipid and NLOM assimilation efficiencies in zooplankton. Water assimilation varies between freshwater and marine organisms as a result of their distinct requirements for osmoregulatory balance. Because water is not a significant contributor to the storage capacity of HOCs, its value has a negligible impact on the mechanism of biomagnification for these chemicals. The water assimilation efficiency for all freshwater species is assumed to be 25%.

K_{GB}. The partition coefficient of the chemical between the contents of the GIT and the organism expresses the change in phase partitioning properties that occurs as a result of digestion of the diet after ingestion. It can be estimated as

$$K_{GB} = (v_{LG} \cdot K_{OW} + v_{NG} \cdot \beta \cdot K_{OW} + v_{WG}) / (v_{LB} \cdot K_{OW} + v_{NB} \cdot \beta \cdot K_{OW} + v_{WB}) \quad (18)$$

where v_{LG}, v_{NG}, and v_{WG} are the lipid (kg lipid/kg digesta ww), NLOM (kg NLOM/kg digesta ww), and water (kg water/kg digesta ww) contents, respectively, in the gut. The sum of these fractions (i.e., total digesta) approach one and are dependent on the assimilation efficiency for each component of the diet as

$$v_{LG} = (1 - \varepsilon_L) \cdot v_{LD} / [(1 - \varepsilon_L) \cdot v_{LD} + (1 - \varepsilon_N) \cdot v_{ND} + (1 - \varepsilon_W) \cdot v_{WD}] \quad (19)$$

$$v_{NG} = (1 - \varepsilon_N) \cdot v_{ND} / [(1 - \varepsilon_L) \cdot v_{LD} + (1 - \varepsilon_N) \cdot v_{ND} + (1 - \varepsilon_W) \cdot v_{WD}] \quad (20)$$

$$v_{WG} = (1 - \varepsilon_W) \cdot v_{WD} / [(1 - \varepsilon_L) \cdot v_{LD} + (1 - \varepsilon_N) \cdot v_{ND} + (1 - \varepsilon_W) \cdot v_{WD}] \quad (21)$$

Because the bioaccumulation model (Eqn. 2) is based on the ratio of k_D to k_E, which is $G_D/(G_F \cdot K_{GB})$, the model parameterization errors for the feeding rate G_D (and, hence, G_F; see Eqn. 17) and the dietary uptake efficiency E_D tend to cancel out to a significant extent. Hence, the model can be expected to provide reasonable estimates of the BAF even if G_D and E_D are not characterized accurately. This is an attractive feature of the model, because the variability and error in G_D and E_D are often large. For higher-K_{OW} chemicals that are predominantly absorbed via the diet, the BAF model is more sensitive to diet digestion and, hence, to the composition of the diet and the absorption efficiencies of lipid and NLOM. Error and natural variability occur in these parameters as well, but the magnitude of the error and variability in these parameters is often substantially smaller than that in G_D and E_D.

k_G. In many cases, reliable data for the growth rate of organisms are available. Growth rates vary considerably among species but also within species as a function of size, temperature, prey availability and quality, and other factors. In the absence of the required data, the following generalized growth equations from Thomann et al. [43] provide a reasonable approximation for the growth rate constant of aquatic organisms k_G (d^{-1}):

$$k_G = 0.0005 \cdot W_B^{-0.2} \text{ for temperatures around } 10°C \quad (22)$$

$$k_G = 0.00251 \cdot W_B^{-0.2} \text{ for temperatures around } 25°C \quad (23)$$

k_M. The rate at which a parent compound can be eliminated via metabolic transformation is represented by the metabolic transformation rate constant k_M (d^{-1}). This process is chemical and species dependent, and empirical data regarding k_M are

Table 1. Summary of site-specific model input parameters, units, and their source[a]

Definition	Parameter	Units	Lake Ontario	Lake Erie	Lake St. Clair
Chemical concentration in water (total)	C_{WT}	g/L	[93]	[94]	[95]
Chemical concentration in water (dissolved)	C_{WD}	g/L	$(C_{WT})/(1 + [0.5 \cdot \alpha_{DOC} \cdot K_{OW} \cdot \chi_{DOC}])$	[95]	
Chemical concentration in sediment	C_S	g/kg	[93]	[94]	[95]
Concentration of suspended solids in water	C_{SS}	g/L	N/A [93]	4.0×10^{-5} [94]	5.0×10^{-5}
Octanol–water partition coefficient	K_{OW}	Unitless	[96,97]	[96,97]	[96,97]
Weight of biota	W_B	kg	Table S.5	Table S.5	Table S.5
Lipid fraction in biota ($_B$) and phytoplankton ($_P$)	v_{LB}, v_{LP}	kg/kg	Table S.5	Table S.5	Table S.5
Nonlipid organic matter fraction in biota ($_B$)	v_{NB}	kg/kg	Table S.5	Table S.5	Table S.5
Nonlipid organic carbon fraction in phytoplankton ($_P$)	v_{NP}	kg/kg	Table S.5	Table S.5	Table S.5
Fraction of overlying and pore water respired by benthic organisms	m_O, m_P	%	Table S.6	Table S.6	Table S.6
Mean annual water temperature	T	°C	8 [42]	13 [94]	13 (Estimated)
Dissolved oxygen saturation	S	%	85 (Estimated)	90 (Estimated)	95 (Estimated)
Concentration of particulate organic carbon	χ_{POC}	kg/L	N/A	N/A	N/A
Concentration of dissolved organic carbon	χ_{DOC}	kg/L	2.0×10^{-6} [93]	2.2×10^{-6} [94]	N/A
Disequilibrium factor POC	D_{POC}	Unitless	N/A	N/A	N/A
Disequilibrium factor DOC	D_{DOC}	Unitless	N/A	N/A	N/A
POC–octanol proportionality constant	α_{POC}	Unitless	0.35 [62]	0.35 [62]	0.35 [62]
DOC–octanol proportionality constant	α_{DOC}	Unitless	0.08 [63]	0.08 [63]	0.08 [63]

[a] All study locations are in North America. DOC = dissolved organic carbon; N/A = not applicable; POC = particulate organic carbon.

often lacking. Methods for estimating these rates are suggested elsewhere [46,90,91]. In the present study, we apply the model to nonmetabolizable substances and can assume k_M to be zero.

A complete list of the site-specific model input parameters, their units, and their definitions is provided in Table 1. Table 2 provides a summary of other model parameters.

Model application

Aquatic food webs include many complex relationships and may involve a very large number of organisms. Therefore, it rarely is possible to include all organisms in the model and to recognize all feeding relationships that exist. To describe contaminant movement in food webs, we propose an approach whereby seven trophic guilds (i.e., algae, phytoplankton, and macrophytes; zooplankton and small pelagic invertebrates; benthic invertebrates; water-column filter feeders; small juvenile, medium-sized, and larger upper-trophic-level fish) are recognized. In the model, each trophic guild is represented by at least one organism (e.g., benthic invertebrates may be represented by the mayfly). When actual diet compositions (from measurements or literature surveys) are translated into model input parameters, prey items of an organism consisting of benthic invertebrates can be represented by mayflies even though an organism may prey on a larger range of benthic invertebrate species. For example, an organism may prey on gammarus and mayflies, but mayflies could represent its diet in the model. Clearly, these generalizations should be done with care and consideration of the feeding behavior of the species in the model. However, when this can be achieved, the model can be kept relatively simple using a minimum of species and minimal input parameters. This approach may also be useful in the development of field studies to support the model. Field sampling of biological organisms is often limited by available methods for species collection as well as by analytical costs. If the model is applied to follow changes in the BAF as a function of age of the fish, several age classes of fish can be introduced as independent species in the model. After a reasonable number of species are included in the model, it is possible to use the equations described above to make calculations of the chemical concentrations in these organisms as well as of the BAFs and BSAFs. If the food web is relatively straightforward, with higher-trophic-level organisms feeding on lower-trophic-level organisms, this can be done by simply conducting the calculations for the phytoplankton first and then for the zooplankton, filter feeders, invertebrates, juvenile/small fish, medium-size fish, and upper-trophic-level fish last. If this is not possible, such as those cases in which scavenging (e.g., lower-trophic-level organisms feeding on a higher-trophic-level organism) and/or cannibalism occur, a matrix solution of the model equations could be used. This method has been described in more detail by Campfens and Mackay [45] and can be carried out in Excel® spreadsheets (Microsoft, Redmond, WA, USA).

An important consideration in any model prediction is the uncertainty or error that can be expected in the model output (i.e., BAF or BSAF). One method to assess error is through the application of Monte Carlo simulations. This method assesses the impact of variations in model parameter values in terms of a variation in the model output. A number of authors have applied these techniques to food web bioaccumulation models (see, e.g., [44,92]). This method is particularly useful in determining the sensitivity of the model output to variability and error in the model input parameters. However, care should be taken not to overinterpret these numbers in terms of error or uncertainty in model predictions, both because error in model structure is not considered in a Monte Carlo simulation and because no comparison of the model predictions to an independent dataset is made. An alternative method is based on the comparison of predicted model outcomes and observed data (e.g., BAF or C_B). With a sufficiently large population of observed BAFs, the degree of similarity between observed and predicted BAFs can be used to characterize the overall error of the model. This error includes model and model parameterization errors as well as errors and natural variability associated with the empirical measurements. If these errors can be established for a number of different food webs, chemical substances, and databases, the error can be used as a measure of the model uncertainty in applications for which no empirical data are available (e.g., when the model is applied to food webs for which no empirical data exist). In the present study, we use this second method to evaluate the performance of the

Table 2. Summary of model parameters, units, and their definitions

Definition	Parameter	Units
Chemical concentration in biota	C_B	g/kg
Chemical concentration in diet	C_D	g/kg
Freely dissolved chemical concentration in pore water	$C_{WD,P}$	g/L
Bioavailable solute fraction	ϕ	Unitless
Gill uptake rate constant	k_1	L/kg · d
Dietary uptake rate constant	k_D	kg/kg · d
Gill elimination, fecal egestion, growth dilution, and metabolic transformation rate constants, respectively	k_2, k_E, k_G, k_M	d^{-1}
Biota–water partition coefficient	K_{BW}	Unitless
Phytoplankton–water partition coefficient	K_{PW}	Unitless
Gut–biota partition coefficient	K_{GB}	Unitless
Gill ventilation rate	G_V	L/d
Feeding and fecal egestion rates, respectively	G_D, G_F	kg/d
Efficiency of chemical transfer via gill and intestinal tract	E_W, E_D	%
Nonlipid organic matter–octanol proportionality constant	β	Unitless
Lipid fraction in diet ($_D$) and gut ($_G$)	v_{LD}, v_{LG}	kg/kg
Nonlipid organic matter fraction in diet ($_D$) and gut ($_G$)	v_{ND}, v_{NG}	kg/kg
Water fraction in biota ($_B$), diet ($_D$), gut ($_G$), and phytoplankton ($_P$)	v_{WB}, v_{WD}, v_{WG}, v_{WP}	kg/kg
Dietary absorption efficiency of lipid	ϵ_L	%
Dietary absorption efficiency of nonlipid organic matter	ϵ_N	%
Dietary absorption efficiency of water	ϵ_W	%
Particle scavenging efficiency	σ	%
Density of organic carbon in sediment (0.9)	δ_{OCS}	kg/L
Organic carbon–water partition coefficient	K_{OC}	L/kg
Dissolved oxygen concentration	C_{OX}	mg O_2/L

model, because the outcome of this analysis is useful in the practical application of the model to contamination problems.

MATERIALS AND METHODS

Evaluation of model performance

Both the revised model described above and the 1993 food web model were programmed in Excel spreadsheets. Both models were then parameterized to make predictions of the BAFs and BSAFs for a range of organochlorines in three freshwater lake ecosystems (i.e., Lake Ontario, Lake Erie, and Lake St. Clair). The model performance was then evaluated by comparing predicted BAFs to independent, observed BAFs in these three lake ecosystems [93–95]. Bioaccumulation factor data were available for 59 chemicals in eight species for Lake Ontario (n = 408), 25 chemicals in 20 species for Lake Erie (n = 483), and six chemicals in 22 species for Lake St. Clair (n = 128). The three combined datasets provide 1,019 observations that were used for model performance evaluation. The observed BAF data were not used to make the model predictions. To express quantitatively the general model's performance combining the results for all n chemicals in a single species j, we used the following measure, which we refer to as the model bias for species j (MB_j) [48]:

$$MB_j = 10^{\left(\frac{\sum_{i=1}^{n}[\log(BAF_{p,i}/BAF_{o,i})]}{n}\right)} \quad (24)$$

The overall model bias, combining the results for all n chemicals in all m species, is given by

$$MB = 10^{\left[\frac{\sum_{j=1}^{m}\left(\frac{\sum_{i=1}^{n}[\log(BAFp,i,j/BAFo,i,j)]}{n}\right)}{m}\right]} \quad (25)$$

where BAF_P is the model-predicted BAF, BAF_O is the observed BAF, and the subscripts i and j refer to the number of chemicals and the number of species, respectively, included in the model performance evaluation. In essence, MB is the geometric mean (assuming a log-normal distribution of the ratio $BAF_{P,i,j}/BAF_{O,i,j}$) of the ratio of predicted and observed BAFs for all chemicals in all species for which empirical data were available. The MB is a measure of the systematic overprediction (MB $>$ 1) or underprediction (MB $<$ 1) of the model. For example, MB = 2 indicates that the model in general overpredicts the empirical data by a factor of two. Conversely, a model bias of 0.5 indicates that the model underpredicts the observed data by a factor of two. The 95% confidence intervals of the geometric mean represent the accuracy of the model.

Because of the log-normal distribution of the ratio of predicted and observed BAFs, variability can be expressed as a factor (rather than as a term) of the geometric mean. One of the key characteristics of MB and its 95% confidence interval is that it represents all sources of error, including model parameterization errors, errors in model structure and philosophy, and also analytical errors in the empirical data (e.g., chemical concentrations in water, sediment, and biota) as well as natural, spatial, and temporal variability in the empirical data used for the model performance. The rationale for using this measure of model performance is that it is relevant for cases in which the model is used to make practical estimations of the BAF for exposure assessment or water-quality criteria development. In those cases, the 95% confidence intervals represent the range of BAFs that includes 95% of the observed BAFs. With caution, the 95% confidence limits can be extrapolated from one system for which empirical BAFs exist to another system for which empirical BAFs may not exist.

To explore the impact of each model modification on the model performance (i.e., MB), we ran the model under six sequential scenarios as outlined in Table 3. Each scenario explored one particular model modification by incorporating all modifications in previous scenarios. In each scenario, the performance of the new model (including the new formulation) was compared to that of the 1993 model (including the old formulation). To enable a direct comparison of the new and old models for a particular model modification, both the new

Table 3. Model bias in phytoplankton (MB_P), zooplankton (MB_Z), invertebrates (MB_I), and fish (MB_F) and the combined model bias for all species (MB_{TOT}) with its 95% confidence interval of the revised model resulting from specific model modifications in relation to the model bias of the 1993 model[a]

			Lake Ontario		Lake Erie		Lake St. Clair	
Scenario	MB	Ecosystem revision	1993	Revised	1993	Revised	1993	Revised
1	MB_P	Kinetic model applied to phytoplankton	0.15	1.20	0.12	0.72	N/A	N/A
2	MB_Z	Bioaccumulation model applied to zooplankton	N/A	N/A	N/A	N/A	0.42	1.17
3	MB_I	Bioaccumulation model applied to invertebrates	1.95	1.04	0.30	1.13	0.37	0.92
4	MB_{F1}	Organism composition model applied to fish	0.45	0.47	0.34	0.45	0.25	0.37
5	MB_{F2}	Allometric gill ventilation rate applied to fish	0.47	0.52	0.45	0.48	0.37	0.40
6	MB_{F3}	Diet digestion model applied to fish	0.52	1.00	0.48	1.05	0.40	0.71
7	MB_{TOT}	Overall model comparisons[b]	0.86	1.04 (0.13–8.08)	0.16	1.05 (0.24–4.64)	0.17	0.78 (0.08–7.89)

[a] All study locations are in North America. N/A = no empirical data available.
[b] The p values of normality testing (Shapiro–Wilk) of log-transformed ratios of predicted and observed bioaccumulation factors are 0.064 (Lake Ontario), 0.439 (Lake Erie), and 0.056 (Lake St. Clair) for the current model and 0.004 (Lake Ontario), 0.47 (Lake Erie), and 0.96 (Lake St. Clair) for the 1993 model ($p > 0.05$ indicates log-normal distributions).

and the 1993 model included the model formulation of the new model tested in the previous scenarios. The MB was calculated using observed data from the relevant compartment of the food web for each respective scenario (e.g., MB_P calculated using observed phytoplankton data). The first scenario included the use of a kinetic model for phytoplankton–water distribution in the new model compared to the equilibrium partitioning model used in the 1993 model. The second scenario included the kinetic bioaccumulation model (i.e., Eqn. 1) for zooplankton in the revised model compared to the equilibrium partitioning model used in the 1993 model. The third scenario used the kinetic bioaccumulation model to describe bioaccumulation in aquatic invertebrates in the revised model compared to the equilibrium partitioning model used to estimate BAFs in the 1993 model. The fourth scenario applied the new three-phase partitioning model (i.e., Eqn. 3) to fish in the new model as well as model modifications made as part of the first three scenarios, whereas the 1993 model only included revisions 1 to 3 (i.e., the 1993 phase partitioning model for fish). The fifth scenario added new allometric relationships for respiratory ventilation in fish in the new model. The sixth scenario added the gastrointestinal magnification model for fish in the new model. In each of these scenarios, the 1993 model included the new model formulations evaluated in the previous scenarios other than the revision for comparison. For example, in scenario 6, the revised model predictions for BAFs in fish included all revisions described in scenarios 1 through 6 (i.e., Eqn. 1 applied for fish), whereas the 1993 model BAFs in fish were simulated by including revisions described in scenarios 1 through 5 only. A final simulation, scenario 7, compared the revised model in its entirety (i.e., revisions 1–6) to the original 1993 model (i.e., no changes) for all species and chemicals for which empirical data were available (MB from Eqn. 25).

Model parameterization

The model requires input parameters to characterize the chemical substance, relevant environmental conditions, biological species-specific characteristics, and food web structure. The only chemical-specific parameter that is required is K_{OW}. The K_{OW} values were obtained from Hawker and Connell [96] for polychlorinated biphenyls and from Mackay and Shiu [97] for the remaining organochlorines and are summarized in Table S.4 [SETAC Supplemental Data Archive, Item ETC-23-10-002; http://etc.allenpress.com]. Ecosystem specific-input parameters are listed in Table 1. Because the empirical studies used for testing model performance report freely dissolved water rather than total concentrations in water, several ecosystem parameters were not needed for the model performance evaluation in the present study. Species-specific parameters are listed in Table S.5 [SETAC Supplemental Data Archive, Item ETC-23-10-002; http://etc.allenpress.com]. In this model performance evaluation, we did not use empirical values for respiratory ventilation, feeding, and growth rates. Instead, these rates were estimated from organism body weights according to the models explained above. Food web–specific parameters are listed in Table S.6 [SETAC Supplemental Data Archive, Item ETC-23-10-002; http://etc.allenpress.com].

RESULTS AND DISCUSSION

Phytoplankton

Figure 2a and Table 3 (scenario 1; MB_P) illustrate that use of a combination of a more detailed phase partitioning model, which recognizes organic carbon in addition to lipids as an important medium in which bioaccumulation can occur, and a kinetic model, which recognizes the growth dynamics of phytoplankton, improves MB_P for the BAFs in phytoplankton compared to the 1993 model. Now, 65% of the model-predicted BAFs ($n = 83$) are within a factor of two of the observed BAFs, and 88% of the model-predicted BAFs are within a factor of 10 of the observed BAFs. In comparison, only 6% and 43% of the 1993 model-predicted BAFs are within a factor of 2 and a factor of 10, respectively, of the observed BAFs. The apparent improvements in the model's predictability are mainly a result of the new model producing greater BAFs than the lipid–water partitioning model for the lower K_{OW} chemicals in the 1993 model (because of partitioning in nonlipid organic carbon) while calculating lower BAFs than the lipid–water partitioning model for the higher-K_{OW} chemicals (because of phytoplankton growth).

When comparing model predictions for phytoplankton to empirical data, it is important to consider the nature of the phytoplankton samples. Typically, phytoplankton samples are collected with plankton nets, which can collect a range of different organisms and materials. In many cases, these phytoplankton samples contain zooplankton and other suspended matter, which can collect chemicals by mechanisms other than those assumed in the model for phytoplankton. Hence, any

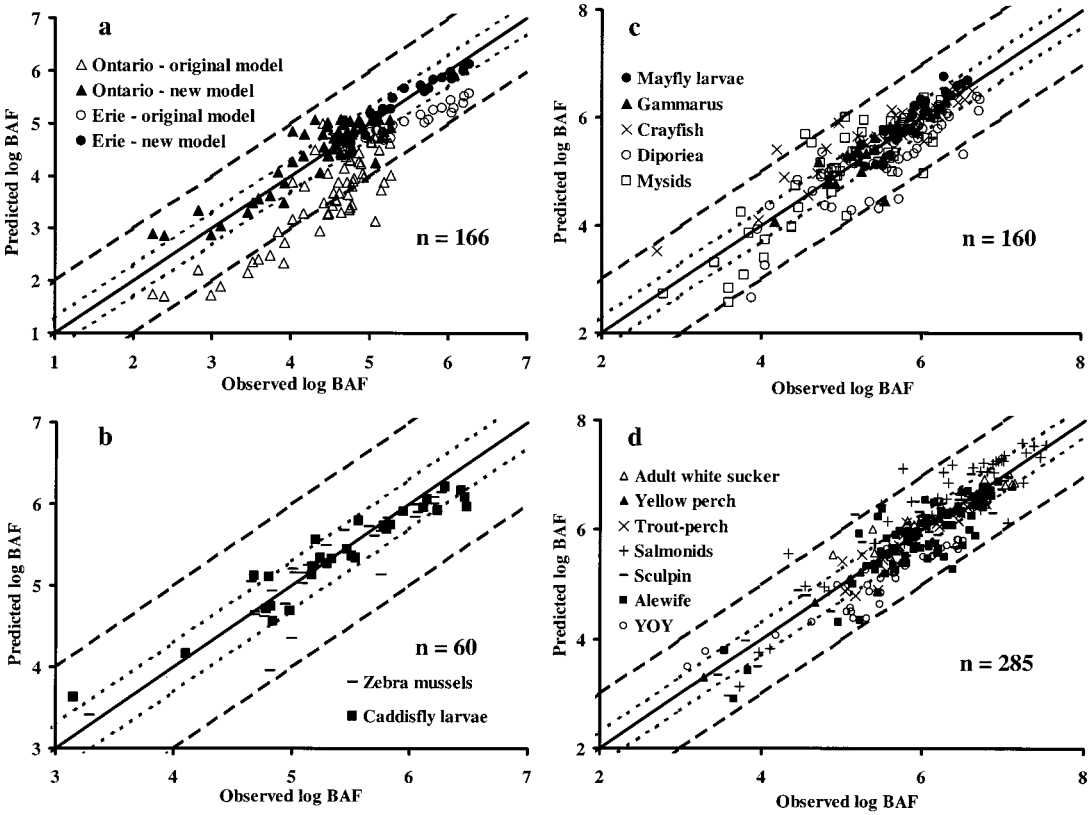

Fig. 2. An illustrative comparison of model-predicted bioaccumulation factors (BAF) and observed BAFs for (**a**) phytoplankton, (**b**) filter feeders, (**c**) detritus feeders, and (**d**) fish species from three freshwater ecosystems (when available). The solid black line represents the ideal fit (model bias = 1), the short dashed lines represent a factor of two of the ideal fit, and the long dashed lines represent a factor of 10 of the ideal fit. YOY = young of the year.

apparent failures of the model to predict BAFs in phytoplankton in comparison to observed data should not necessarily be interpreted entirely as model error. Furthermore, errors in the prediction of actual concentrations in phytoplankton field samples may not necessarily prevent the model from making accurate estimates of concentrations in higher-trophic-level organisms. For example, chemical concentrations in organisms that feed directly or indirectly on benthos are relatively independent of concentrations in phytoplankton.

Zooplankton

The effect of using the kinetic bioaccumulation model to estimate BAFs in zooplankton species could only be assessed for Lake St. Clair (North America), because the required empirical data for model testing were not available for Lake Ontario and Lake Erie. Table 3 (scenario 2; MB_Z) indicates that application of the kinetic bioaccumulation model to zooplankton improves the MB_Z for zooplankton: It changes from 0.42 to 1.17. The improvements are caused by dietary magnification elevating BAFs above those derived from equilibrium partitioning.

Aquatic invertebrates

Table 3 (scenario 3; MB_I) indicates that replacing the equilibrium partitioning model with the kinetic bioaccumulation model results in better model predictions, because the MB_I for invertebrates achieves values closer to 1.0 compared to the 1993 model. Figure 2b and c illustrate that the majority of the kinetic bioaccumulation model predictions for both filter feeders and detritus feeders are within a factor of two of the observed concentrations. For the three ecosystems combined, 60% and 95% of the model-predicted BAFs for nine species and 64 chemicals (n = 324) are within, respectively, a factor of 2 and a factor of 10 of the observed BAFs. In comparison, only 37% and 89% of model-predicted BAFs are within a factor of 2 and a factor of 10, respectively, of the observed data when the equilibrium partitioning model is used.

The revised model more adequately predicts the frequently observed, apparently parabolic shape of the BSAF–K_{OW} relationship for nonmetabolizable chemicals (SETAC Supplemental Data Archive S.7). In the revised model, BSAF increases with increasing hydrophobicity up to a log K_{OW} of approximately 7 as a result of the increasing contribution of dietary uptake (compared to uptake from water) and magnification. The BSAFs fall with further elevation of K_{OW} because of reduced gastrointestinal absorption efficiency, which decreases with increasing K_{OW}. Evidence suggests that the bioavailability of HOCs in sediments can be a function of different matrices (i.e., soil and organic carbon type) and time [73,98,99]. The current model does not capture these factors well. Therefore, the model should be applied with caution in cases when these factors are important (e.g., polycyclic aromatic hydrocarbons associated with soot carbon).

Fish

Figure 2d illustrates by example that when combining data for all chemicals and all fish (n = 606), 60% and 98% of the model-predicted BAFs are within a factor of 2 and a factor of 10, respectively, of the empirical BAFs. In comparison, 19% and 71% of the BAF estimates fish using the entire 1993 food

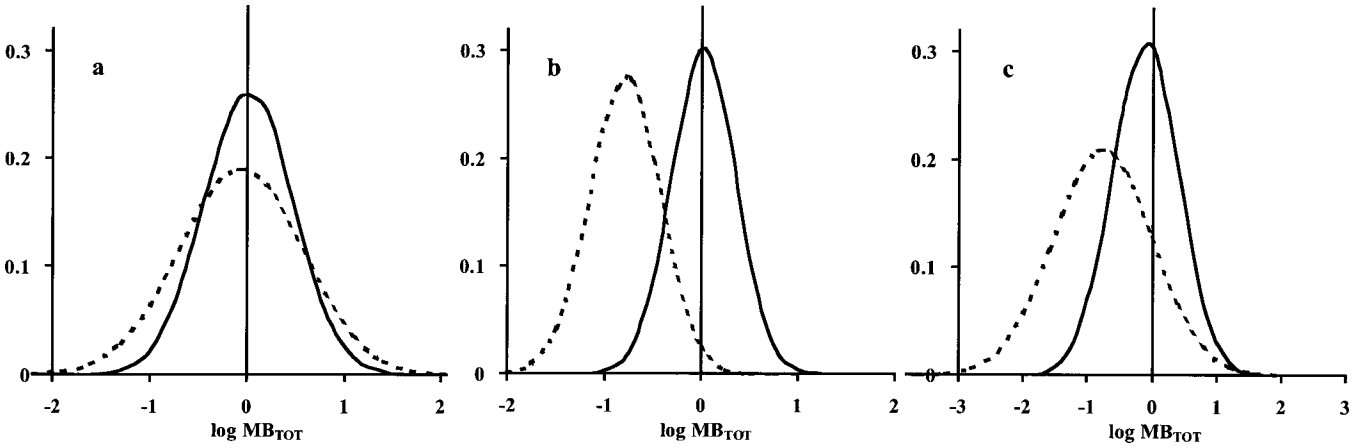

Fig. 3. A comparison of the probability distributions for the combined model bias for all compounds in all species (MB_{TOT}) between the 1993 (dashed curves) and revised (solid curves) food web models for (**a**) Lake Ontario, (**b**) Lake Erie, and (**c**) Lake St. Clair ecosystems (North America). The MB_{TOT} is log transformed such that ideal fit = 0 (vertical line).

web model are within a factor of 2 and a factor of 10, respectively, of the observed data. Table 3 (scenario 4; MB_{F1}) demonstrates that the inclusion of NLOM (v_{NB}) and water content (v_{WB}) organism composition fractions as potential storage sites for organic chemicals in fish results in modest increases in the chemical concentrations in fish and subsequent reductions in model error. This is most apparent for Lake Erie and Lake St. Clair, where MB_{F1} changes from 0.34 to 0.45 and 0.25 to 0.37, respectively. The size and lipid contents for fish in these ecosystems are generally less than in Lake Ontario, causing the effect of including the NLOM as a storage compartment to be more pronounced. A NLOM content of 20% contributes the equivalent of approximately 1% of lipid to the organism. This may be viewed as a small addition to the overall lipid content in a fish with a 10% lipid content, but it is equivalent to doubling the storage capacity in a fish with a 1% lipid content.

The new allometric relationship for gill ventilation rates in fish (scenario 5; MB_{F2}) also results in modest improvements in model performance as the MB_{F2} moves closer to unity in all three ecosystems. The gill ventilation rate is not a particularly sensitive parameter in the model, because it affects both the respitory uptake and elimination rates, leaving the BAF (i.e., the ratio of uptake rate and elimination rates) largely unaffected. The improvements in the model predictions emerge from a better characterization of the relationship between the gill ventilation rate, the dietary uptake rate, and the growth rate.

The inclusion of a mechanistic gastrointestinal magnification model results in the most substantial (i.e., 2- to 3-fold) improvements in predictions of the BAF (scenario 6; MB_{F3}). These results suggest that the species- and prey-specific gastrointestinal magnification model is preferable over the constant gastrointestinal magnification factor used in the 1993 model.

Model performance

Table 3 demonstrates that each revision to the model described in this paper results in incremental reductions in model error compared to the 1993 model for each part of the food web in all three food webs studied. The majority of revised model predictions are within a factor of two of the empirical data for these compartments (SETAC Supplemental Data Archive S.8). Figure 3 illustrates that when data for all chemicals and organisms are combined, the overall model bias, which measures the systematic error in the BAF calculations, is reduced substantially compared to the 1993 model. The revised food web model produces the largest reductions in overall model bias for the Lake Erie and Lake St. Clair ecosystems. The combined model bias for all species (MB_{TOT}) of the new model is much closer to unity in Lake Erie and Lake St. Clair and marginally closer to 1.0 in Lake Ontario. In calculating MB_{TOT}, overpredictions (e.g., for one substance) and underpredictions (e.g., for another substance) have a tendency to cancel out; therefore, it is important to consider the confidence limits of MB_{TOT}, which represent the accuracy of the model in predicting concentrations or BAFs of an individual substance. In Figure 3, the accuracy of the model is represented by the width of the bell plots. Figure 3 illustrates that the revised model produces improvements in model accuracy over the 1993 model. Achieving even better model accuracy will be a challenge, because variability in contaminant concentrations among individuals of sampled populations is high. This variability is a key factor controlling the model accuracy expressed by the 95% confidence limits of the MB. It is important to characterize this source of variability in bioaccumulation models (because it is real) and to recognize it in exposure and risk assessments.

The ability to predict organic chemical transfer and distribution provides a useful tool to assess chemical exposure in organisms of aquatic food webs and in humans who consume fish products. The described model is relatively simple and requires only basic information. The observation that the model performance is comparable among different food webs builds confidence in its ability to be a reliable tool for exposure assessment of nonionizing hydrophobic organic chemicals with a log K_{OW} from 1 to approximately 9. However, we stress that caution should be exercised when the model is applied beyond its bounds.

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REFERENCES

1. Mackay D, Fraser A. 2000. Bioaccumulation of persistent organic chemicals: Mechanisms and models. *Environ Pollut* 110:375–391.
2. Government of Canada. 1999. Canadian Environmental Protection Act, 1999. Canada Gazette Part III. Ottawa, ON.
3. U.S. Environmental Protection Agency. 2000. Methodology for deriving ambient water quality criteria for the protection of human health. EPA-822-B-00-004. Washington, DC.
4. Burkhard LP. 2003. Factors influencing the design of bioaccumulation factor and biota–sediment accumulation factor field studies. *Environ Toxicol Chem* 22:351–360.
5. Organisation for Economic Cooperation and Development. 2001. Harmonized integrated hazard classification system for human health and environmental effects of chemical substances and mixtures. OECD Series on Testing and Assessment. ENV/JM/MONO(2001)6. Paris, France.
6. Hamelink JL, Waybrant RC, Ball RC. 1971. A proposal: Exchange equilibria control the degree chlorinated hydrocarbons are biologically magnified in lentic environments. *Trans Am Fish Soc* 100:207–214.
7. Neely WB, Branson DR, Blau GE. 1974. Partition coefficients to measure bioconcentration potential of organic chemicals in fish. *Environ Sci Technol* 8:1113–5.
8. Veith GD, Defoe DL, Bergstedt BV. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. *J Fish Res Board Can* 36:1040–1048.
9. Branson DR, Blau GE, Alexander HS, Neely WB. 1975. Bioconcentration of 2,2,4,4-tetrachlorobiphenyl in rainbow trout as measured by an accelerated test. *Trans Am Fish Soc* 104:785–792.
10. Mackay D. 1982. Correlation of bioconcentration factors. *Environ Sci Technol* 16:274–278.
11. Barber MC, Suarez LA, Lassiter RR. 1988. Modeling bioconcentration of nonpolar organic pollutants by fish. *Environ Toxicol Chem* 7:545–558.
12. Barber MC, Suarez LA, Lassiter RR. 1991. Modeling bioaccumulation of organic pollutants in fish with an application to PCBs in Lake Ontario salmonids. *Can J Fish Aquat Sci* 48:318–337.
13. Nichols JW, McKim JM, Andersen ME, Gargas ML, Clewell HJ III, Erickson RJ. 1990. A physiology based toxicokinetic model for the uptake and disposition of waterborne organic chemicals in fish. *Toxicol Appl Pharmacol* 106:433–447.
14. Law FCP, Abedini S, Kennedy CJ. 1991. A biologically based toxicokinetic model for pyrene in rainbow trout. *Toxicol Appl Pharmacol* 110:390–402.
15. Gobas FAPC, Opperhuizen A, Hutzinger O. 1986. Bioconcentration of hydrophobic chemicals in fish: Relationship with membrane permeation. *Environ Toxicol Chem* 5:637–646.
16. Gobas FAPC, Clark KE, Shiu WY, Mackay D. 1989. Bioconcentration of polybrominated benzenes and biphenyls and related superhydrophobic chemicals in fish: Role of bioavailability and elimination into the feces. *Environ Toxicol Chem* 8:231–245.
17. Walker CH. 1987. Kinetic models for predicting bioaccumulation of pollutants in ecosystems. *Environ Pollut* 44:227–240.
18. Gobas FAPC, Mackay D. 1987. Dynamics of hydrophobic organic chemical bioconcentration in fish. *Environ Toxicol Chem* 6:495–504.
19. Bruggeman WA, Matron LBJM, Kooiman D, Hutzinger O. 1981. Accumulation and elimination kinetics of di-, tri-, and tetrachlorobiphenyls by goldfish after dietary and aqueous exposure. *Chemosphere* 10:811–832.
20. Connolly JP, Pedersen CJ. 1988. A thermodynamic-based evaluation of organic chemical accumulation in aquatic organisms. *Environ Sci Technol* 22:99–103.
21. Muir DCG, Yarechewski AL. 1988. Dietary accumulation of four chlorinated dioxin congeners by rainbow trout and fathead minnows. *Environ Toxicol Chem* 7:227–236.
22. Niimi AJ, Oliver BG. 1987. Influence of molecular weight and molecular volume on the dietary absorption efficiency of chemicals by fish. *Can J Fish Aquat Sci* 45:222–227.
23. Norstrom RJ, McKinnon AE, de Freitas ASW. 1976. A bioenergetics based model for pollutant accumulation by fish. Simulation of PCB and methyl mercury residue levels in Ottawa River perch (*Perca flavascens*). *J Fish Res Board Can* 33:248–267.
24. Thomann RV, Connolly JP. 1984. Model of PCB in the Lake Michigan Lake trout food chain. *Environ Sci Technol* 18:65–71.
25. Clark KE, Gobas FAPC, Mackay D. 1990. Model of organic chemical uptake and clearance from fish from food and water. *Environ Sci Technol* 24:1203–1213.
26. Gobas FAPC, Muir DCG, Mackay D. 1989. Dynamics of dietary bioaccumulation and fecal elimination of hydrophobic organic chemicals in fish. *Chemosphere* 17:943–962.
27. Landrum PF, Poore R. 1988. Toxicokinetics of selected xenobiotics in *Hexagenia limbata*. *J Gt Lakes Res* 14:427–437.
28. Gobas FAPC, Bedard DC, Ciborowski JJH, Haffner GD. 1989. Bioaccumulation of chlorinated hydrocarbons by the mayfly *Hexagenia limbata* in Lake St. Clair. *J Gt Lakes Res* 15:581–588.
29. Shea D. 1988. Developing national sediment-quality criteria. *Environ Sci Technol* 22:1256–1261.
30. Bierman VJJ. 1990. Equilibrium partitioning and biomagnification of organic chemicals in benthic animals. *Environ Sci Technol* 24:1407–1412.
31. Lake JL, Rubinstein NI, Lee HI, Lake CA, Heltshe J, Pavignano S. 1990. Equilibrium partitioning and bioaccumulation of sediment-associated contaminants by infaunal organisms. *Environ Toxicol Chem* 9:1095–1106.
32. DiToro DM, Zarba CS, Hansen DJ, Berry WJ, Swartz RC, Cowan CE, Pavlou SP, Allen HE, Thomas NA, Paquin PR. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals by using equilibrium partitioning. *Environ Toxicol Chem* 10:1541–1583.
33. Landrum PF, Fontaine TD, Faust WR, Eadie BF, Lang GA. 1992. Modeling the accumulation of polycyclic aromatic hydrocarbons by the amphipod *Diporeia* spp. In Gobas FAPC, McCorquodale JA, eds, *Chemical Dynamics in Fresh Water Ecosystems*. Lewis, Boca Raton, FL, USA, pp 111–128.
34. Morrison HA, Gobas FAPC, Lazar R, Haffner DG. 1996. Development and verification of a bioaccumulation model for organic contaminants in benthic invertebrates. *Environ Sci Technol* 30:3377–3384.
35. Geyer HJ, Politzki G, Freitag D. 1984. Prediction of ecotoxicological behavior of chemicals: relationship between *n*-octanol–water partition coefficient and bioaccumulation of organic chemicals by alga *Chlorella*. *Chemosphere* 13:269–284.
36. Swackhamer DL. 1991. Bioaccumulation of toxic hydrophobic organic compounds at the primary trophic level. *J Environ Sci (China)* 3:15–21.
37. Swackhamer DL, Skoglund RS. 1993. Bioaccumulation of PCBs by algae: Kinetics versus equilibrium. *Environ Toxicol Chem* 12: 831–838.
38. Skoglund RS, Swackhamer DL. 1999. Evidence for the use of organic carbon as the sorbing matrix in the modeling of PCB accumulation in phytoplankton. *Environ Sci Technol* 33:1516–1519.
39. Sodergren A. 1968. Uptake and accumulation of C^{14}-DDT by *Chlorella* sp. (*Chlorophyceae*). *Oikos* 19:126–138.
40. Reinert RE. 1972. Accumulation of dieldrin in algae (*Scenedesmus obliquus*), *Daphnia magna*, and the guppy (*Poecilia reticulata*). *J Fish Res Board Can* 29:1413.
41. Mailhot H. 1987. Prediction of algal bioaccumulation and uptake rate of nine organic compounds by ten physicochemical properties. *Environ Sci Technol* 21:1009–1013.
42. Thomann RV. 1989. Bioaccumulation model of organic chemical distribution in aquatic food chains. *Environ Sci Technol* 23:699–707.
43. Thomann RV, Connolly JP, Parkerton TF. 1992. An equilibrium model of organic chemical accumulation in aquatic food webs with sediment interaction. *Environ Toxicol Chem* 11:615–629.
44. Gobas FAPC. 1993. A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food webs: Application to Lake Ontario. *Ecol Model* 69:1–17.
45. Campfens J, Mackay D. 1997. Fugacity-based model of PCB bioaccumulation in complex food webs. *Environ Sci Technol* 31: 577–583.
46. Arnot JA, Gobas FAPC. 2003. A generic QSAR for assessing the bioaccumulation potential of organic chemicals in aquatic food webs. *QSAR and Combinational Science* 22:337–345.
47. U.S. Environmental Protection Agency. 1995. Great Lakes water-quality initiative technical support document for the procedure to determine bioaccumulation factors. EPA-820-B-95-005. Washington, DC.
48. Gobas FAPC, Pasternak JP, Lien K, Duncan RK. 1998. Development and field validation of a multimedia exposure assessment

model for waste load allocation in aquatic ecosystems: Application to TCDD and TCDF in the Fraser River watershed. *Environ Sci Technol* 32:2442–2449.
49. Mackay D, Diamond M, Sang S, Vlahos P, Gobas FAPC, Dolan D. 1994. A simple rate constant model of chemical dynamics in a lake ecosystem. *J Gt Lakes Res* 20:625–642.
50. Gobas FAPC, Z'Graggen MN. 1995. Time response of the Lake Ontario ecosystem to virtual elimination of PCBs. *Environ Sci Technol* 29:2038–2046.
51. Morrison HA, Gobas FAPC, Lazar R, Whittle DM, Haffner GD. 1998. Projected changes to the trophodynamics of PCBs in the western Lake Erie ecosystem attributed to the presence of zebra mussels (*Dreissena polymorpha*). *Environ Sci Technol* 32:3862–3867.
52. Gobas FAPC, Zhang X, Wells R. 1993. Gastrointestinal magnification: The mechanism of biomagnification and food-chain accumulation of organic chemicals. *Environ Sci Technol* 27:2855–2863.
53. Gobas FAPC, Wilcockson J, Russell RW, Haffner GD. 1999. Mechanism of biomagnification in fish under laboratory and field conditions. *Environ Sci Technol* 33:133–141.
54. Gobas FAPC, Maclean LG. 2003. Sediment–water distribution of organic contaminants in aquatic ecosystems: The role of organic carbon mineralization. *Environ Sci Technol* 37:735–741.
55. Thurston RV, Gehrke PC. 1990. Respiratory oxygen requirements of fishes: Description of OXYREF, a data file based on test results reported in the published literature. In Russo RC, Thurston RV, eds, *Fish Physiology, Toxicology, and Water-Quality Management*. EPA/600/R-93/157. U.S. Environmental Protection Agency. Washington, DC, pp 95–108.
56. Ernst W, Goerke H. 1976. Residues of chlorinated hydrocarbons in marine organisms in relation to size and ecological parameters. I. PCB, DDT, DDE, and DDD in fishes and mollusks from the English Channel. *Bull Environ Contam Toxicol* 15:55–65.
57. Russell RW, Gobas FAPC, Haffner GD. 1999. Maternal transfer and in-ovo exposure of organochlorines in oviparous organisms: A model and field verification. *Environ Sci Technol* 33:416–420.
58. Johnston TA, Fisk AT, Whittle DM, Muir DCG. 2002. Variation in organochlorine bioaccumulation by a predatory fish; gender, geography, and data analysis. *Environ Sci Technol* 36:4238–4244.
59. Payne SA, Johnson BA, Otto RS. 1999. Proximate composition of some northeastern Pacific forage fish species. *Fish Oceanogr* 8:159–177.
60. McCarthy JF. 1983. Role of particulate organic matter in decreasing accumulation of polynuclear aromatic hydrocarbons by *Daphnia magna*. *Arch Environ Contam Toxicol* 12:559–568.
61. McCarthy JF, Jimenez BD. 1985. Reduction in bioavailability to bluegills of polycyclic aromatic hydrocarbons bound to dissolved humic material. *Environ Toxicol Chem* 4:511–521.
62. Seth R, Mackay D, Muncke J. 1999. Estimating the organic carbon partition coefficient and its variability for hydrophobic chemicals. *Environ Sci Technol* 33:2390–2394.
63. Burkhard LP. 2000. Estimating dissolved organic carbon partition coefficients for nonionic organic chemicals. *Environ Sci Technol* 34:4663–4668.
64. Gustafsson O, Haghseta F, Chan C, Macfarlane J, Gschwend PM. 1997. Quantification of the dilute sedimentary soot phase: Implications for PAH speciation and bioavailability. *Environ Sci Technol* 31:203–209.
65. Jonker MTO, Koelmans AA. 2002. Sorption of polycyclic aromatic hydrocarbons and polychlorinated biphenyls to soot and soot-like materials in the aqueous environment: Mechanistic considerations. *Environ Sci Technol* 36:3725–3734.
66. Gobas FAPC. 1988. Bioaccumulation of hydrophobic organic chemicals in fish. PhD thesis. University of Toronto, Toronto, ON, Canada.
67. Benson BB, Krause D. 1980. The concentration of isotopic fractionation of gases dissolved in freshwater in equilibrium with the atmosphere. 1. Oxygen. *Limnol Oceanogr* 25:662–671.
68. Koelmans AA, Jiminez CJ, Lijklema L. 1993. Sorption of chlorobenzenes to mineralizing phytoplankton. *Environ Toxicol Chem* 12:1425–1439.
69. Koelmans AA, Anzion SFM, Lijklema L. 1995. Dynamics of organic micropollutant biosorption to cyanobacteria and detritus. *Environ Sci Technol* 29:933–940.
70. Koelmans AA, van der Woude H, Hattink J, Niesten DJM. 1999. Long-term bioconcentration kinetics of hydrophobic chemicals in *Selensatrum capricornutum* and *Microcystis aeruginosa*. *Environ Toxicol Chem* 18:1164–1172.
71. Wang X, Harada S, Watanabe M, Koshikawa H, Geyer HJ. 1996. Modeling the bioconcentration of hydrophobic organic chemicals in aquatic organisms. *Chemosphere* 32:1783–1793.
72. Winsor MH, Boese BL, Lee H, Randall RC, Specht DT. 1990. Determination of the ventilation rates of interstitial and overlying water by the clam *Macoma nasuta*. *Environ Toxicol Chem* 9: 209–213.
73. Kraaij R, Seinen W, Tolls J, Cornelissen G, Belfroid AC. 2002. Direct evidence of sequestration in sediments affecting the bioavailability of hydrophobic organic chemicals to benthic deposit-feeders. *Environ Sci Technol* 36:3525–3529.
74. Lydy MJ, Landrum PF. 1993. Assimilation efficiency for sediment sorbed benzo[*a*]pyrene by *Diporeia* spp. *Aquat Toxicol* 26:209–224.
75. Parkerton TF. 1993. Estimating toxicokinetic parameters for modeling the bioaccumulation of nonionic organic chemicals in aquatic organisms. PhD thesis. Rutgers University of New Jersey, New Brunswick, NJ, USA.
76. Bruner KA, Fisher SW, Landrum PF. 1994. The role of the zebra mussel, *Dreissena polymorpha*, in contaminant cycling: II. Zebra mussel contaminant accumulation from algae and suspended particles and transfer to the benthic invertebrate, *Gammarus fasciatus*. *J Gt Lakes Res* 20:735–750.
77. Kukkonen J, Landrum PF. 1995. Measuring assimilation efficiencies for sediment-bound PAH and PCB congeners by benthic invertebrates. *Aquat Toxicol* 32:75–92.
78. Wang WX, Fisher NS. 1999. Assimilation efficiencies of chemical contaminants in aquatic invertebrates: A synthesis. *Environ Toxicol Chem* 18:2034–2045.
79. Mayer LM, Weston DP, Bock MJ. 2001. Benzo[*a*]pyrene and zinc solubilization by digestive fluids of benthic invertebrates—A cross-phyletic study. *Environ Toxicol Chem* 20:1890–1900.
80. Gobas FAPC, Muir DCG, Mackay D. 1988. Dynamics of dietary bioaccumulation and fecal elimination of hydrophobic organic chemicals in fish. *Chemosphere* 17:943–962.
81. Gobas FAPC, McCorquodale JR, Haffner GD. 1993. Intestinal absorption and biomagnification of organochlorines. *Environ Toxicol Chem* 12:567–576.
82. Fisk AT, Norstrom RJ, Cymbalisty CD, Muir DCG. 1998. Dietary accumulation and depuration of hydrophobic organochlorines: bioaccumulation parameters and their relationship with the octanol/water partition coefficient. *Environ Toxicol Chem* 17:951–961.
83. Nichols JW, Fitzsimmons PN, Whiteman FW, Kuehl DW, Butterworth BC, Jenson CT. 2001. Dietary uptake kinetics of 2,2′,5,5′-tetrachlorobiphenyl in rainbow trout. *Drug Metab Dispos* 29:1013–1022.
84. Weininger D. 1978. Accumulation of PCBs by lake trout in Lake Michigan. PhD thesis. University of Wisconsin, Madison, WI, USA.
85. Gordon DCJ. 1966. The effects of the deposit feeding polychaete *Pectinaria gouldii* on the intertidal sediments of Barnstable Harbor. *Limnol Oceanogr* 11:327–332.
86. Berg DJ, Fisher SW, Landrum PF. 1996. Clearance and processing of algal particles by Zebra mussels (*Dreissena polymorpha*). *J Gt Lakes Res* 22:779–788.
87. Roditi HA, Fisher NS. 1999. Rates and routes of trace element uptake in zebra mussels. *Limnol Oceanogr* 44:1730–1749.
88. Conover RJ. 1966. Assimilation of organic matter by zooplankton. *Limnol Oceanogr* 11:338–345.
89. Lehman JT. 1993. Efficiencies of ingestion and assimilation by an invertebrate predator using C and P dual isotope labeling. *Limnol Oceanogr* 38:1550–1554.
90. Van der Linde A, Hendriks AJ, Sijm DTHM. 2001. Estimating biotransformation rate constants of organic chemicals from modeled and measured elimination rates. *Chemosphere* 44:423–435.
91. Fisk AT, Tomy GT, Cymbalisty CD, Muir DCG. 2000. Dietary accumulation and quantitative structure–activity relationships for depuration and biotransformation of short (C10), medium (C14), and long (C18) carbon-chain polychlorinated alkanes by juvenile rainbow trout (*Oncorhynchus mykiss*). *Environ Toxicol Chem* 19: 1508–1516.
92. Von Stackelberg K, Burmistrov D, Linkov I, Cura J, Bridges TS.

2002. The use of spatial modeling in an aquatic food web to estimate exposure and risk. *Sci Total Environ* 288:97–110.
93. Oliver BG, Niimi AJ. 1988. Trophodynamic analysis of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in the Lake Ontario Ecosystem. *Environ Sci Technol* 22: 388–397.
94. Morrison HA, Gobas FAPC, Lazar R, Whittle DM, Haffner DG. 1997. Development and verification of a benthic/pelagic food web bioaccumulation model for PCB congeners in western Lake Erie. *Environ Sci Technol* 31:3267–3273.
95. Russell RW. 1996. Bioavailability and biomagnification of organochlorinated chemicals in aquatic ecosystems. PhD thesis. University of Windsor, Windsor, ON, Canada.
96. Hawker DW, Connell DW. 1988. Octanol–water partition coefficients of polychlorinated biphenyl congeners. *Environ Sci Technol* 22:382–387.
97. Mackay D, Shiu WY, Ma KC. 1999. *The Physical–Chemical Properties and Environmental Fate Handbook.* CRC, Boca Raton, FL, USA.
98. Cornelissen G, Hassell KA, van Noort PCM, Kraaij R, van Ekeren PJ, Dijkema C, de Jager PA, Govers HAJ. 2000. Slow desorption of PCBs and chlorobenzenes from soils and sediments: Relations with sorbent and sorbate characteristics. *Environ Pollut* 108:69–80.
99. Mayer LM, Chen Z, Findlay RH, Fang JS, Sampson S, Self RFL, Jumars PA, Quetel C, Donard OFX. 1996. Bioavailability of sedimentary contaminants subject to deposit-feeder digestion. *Environ Sci Technol* 30:2641–2645.