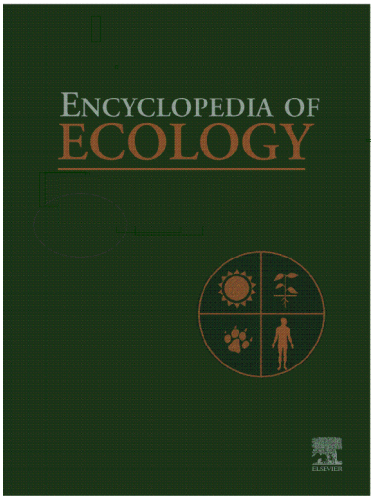

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Food-Web Bioaccumulation Models

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Introduction

Practitioners of ecotoxicology are confronted with some unique problems. For example, when assessing the impacts of chemical contamination (e.g., as result of a spill, a point source emission, or historic sediment contamination) on wildlife species, chemical concentrations at the source (e.g., effluent, water, sediments) may be known but the resulting exposure concentrations and associated risks in affected wildlife species are not. In other cases, the chemical concentration in fish or wildlife are known through monitoring programs and considered of concern, but it is unknown how the organisms acquired their chemical body burden, what the chemical concentrations at the source are, and what source concentrations should be to eliminate the concern. In both cases, it is difficult to justify remediation actions to reduce ecotoxicological risks and improve environmental health of the system as the scientific basis for the cause–effect relationship is either absent or weak. The missing link in both of these examples is knowledge of the relationship between the chemical concentrations in environmental media such as water, air, sediments, and soil, and those in the wildlife species of interest. This relationship is complex. For example, a predatory fish will take up chemical from the water via its gills, ingest contaminated sediments, and feed on a variety of prey items, each of which acquired their contaminant burden via similar mechanisms as the predator. Food-web bioaccumulation models are useful tools to handle this complexity and have been increasingly used to better characterize and understand the relationship between contaminant concentrations in abiotic environmental media and those in wildlife and humans. They have been used in risk assessments, the derivation of total maximum daily loadings (TMDLs) for impacted water bodies, the derivation of criteria for water and sediment quality criteria as well as wildlife criteria, the development of target levels for water and sediment clean-up levels and the derivation of bioconcentration factors (BCFs), bioaccumulation factors (BAFs) and biota-sediment bioaccumulation factors (BSAFs) for chemicals to support the evaluation of large numbers of commercial chemicals for bioaccumulation potential.

Several food-web models exist. The most well-used models are the ones developed by Thomann and co-workers, Mackay and co-workers, Czub and McLachlan, and Gobas and co-workers. Some of the models are readily available and can be downloaded from the Web, while others can be requested from the authors. When applying these tools to address various goals, it is important to be familiar with the main principles included in the model. It is further important to be aware of the key assumptions of the modeling approach and recognize the limitations of the model's application. It is the purpose of this article to summarize the key model principles and discuss the most important aspects of the application of the model. It is expected that this will guide the reader in further studies.

Food-Web Model Philosophy

One of the key challenges in ecotoxicology is to understand the interaction between the chemistry of the contaminant and the biology (including the physiology and ecology) of the organism. Food-web bioaccumulation models are among the important tools to document our knowledge of this interaction and to apply our knowledge to practical problems. These models incorporate a number of fundamental chemical and biological principles. In addition, to apply the model in an acceptable fashion, the model calculations have to be conducted according to a set of modeling and management principles. These fundamental principles need to be understood to recognize the application domain of the models and the strengths and limitations of the model outcomes. This section discusses these principles briefly. Current food-web bioaccumulation models show good agreement on the main principles included in the models. To avoid duplication, this section refers to the Gobas models for specific examples.

Chemical Equilibrium

One of the most important chemical principles embedded in food-web bioaccumulation is the natural tendency of chemicals to involve in net transport with the goal to

achieve a chemical equilibrium. Equilibrium is thermodynamically defined as a situation where the chemical potential μ or the fugacity f(Pa) of the chemical in two or more media (e.g., water and fish) is the same, that is,

$$\mu_B = \mu_W \quad \text{or} \quad f_B = f_W \qquad [1]$$

where μ_B and μ_W and f_B and f_W are the chemical potentials and fugacities in the organism or biota and water, respectively. The fugacity of a chemical, f (in units of pascal), in a given phase is related to the molar concentration C (in mol m^{-3}) by the fugacity capacity Z (in mol m^{-3} Pa^{-1}) of the phase in which the chemical is solubilized:

$$f = C/Z \qquad [2]$$

The fugacity capacity Z is compound and phase specific, and it represents the capacity of that phase to sorb and retain a given chemical within its matrix. Net passive chemical transport occurs from the medium of high fugacity (e.g., water) to the medium of low fugacity (e.g., an organism) until the fugacities in both media are equal (**Figure 1**), at which the chemical concentrations in both media (e.g., in the organism, C_B, and the water, C_W (mol m^{-3})) are related by the chemical's partition coefficient K_{BW} (unitless), that is,

$$C_B/C_W = Z_B/Z_W = K_{BW} \qquad [3]$$

The application of this principle was the foundation of some of the first bioaccumulation models for organic contaminants, in which the BCF (typically expressed in units of l kg^{-1}) of the chemical in fish was found to be related to the octanol–water partition coefficient (K_{OW}); for example, one such model estimates the BCF in fish as $0.048K_{OW}$, where 0.048 is an estimate of the average lipid content of fish.

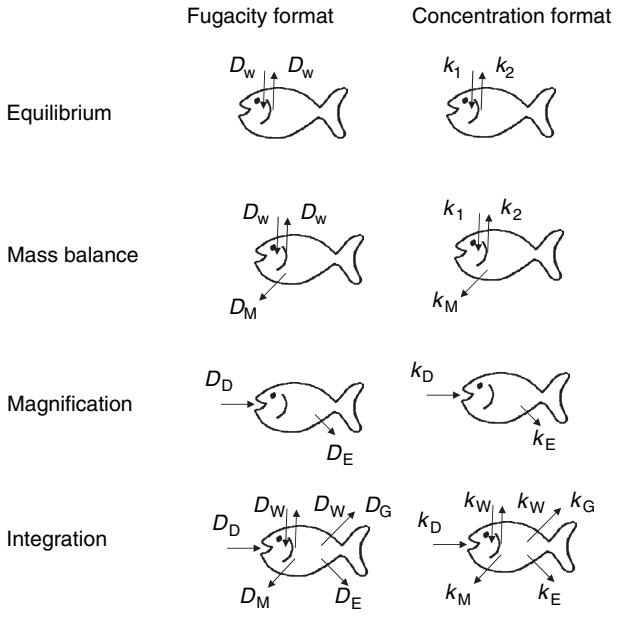

Figure 1 Conceptual diagrams illustrating key chemical principles (equilibrium, mass balance, magnification, and integration of processes) at the organism level.

Mass Balance

The second principle that is embedded in the food-web bioaccumulation models is that physical transport processes (e.g., gill ventilation flows, ingestion rates) and reaction processes (e.g., metabolic transformation) may interfere with the chemical's natural tendency to achieve a chemical equilibrium. This is captured by the mass balance equation where the net flux of chemical N_B (in units of mol day^{-1}) into an organism is the sum of the chemical fluxes into and out of the organism. When expressed in fugacity format, the flux is the product of the transport parameter, D, and the fugacity of the chemical in the medium, f, in which the transport occurs. The transport parameter can represent transport by molecular diffusion, in which case D is the product of the mass transfer coefficient S (in m day^{-1}), the area of diffusion A (m^2), and the Z in which the diffusion occurs, that is, D equals $S{\cdot}A{\cdot}Z$. D can also represent an advective transport process (e.g., gill ventilation rate or ingestion) described by a flow rate G (m^3 day^{-1}) in which case D equals $G{\cdot}Z$. D can further describe a transformation process (e.g., metabolic transformation), often described by a transformation rate constant (day^{-1}), in which case D equals $k{\cdot}V{\cdot}Z$, where V is the volume of the medium in which transformation occurs. One of the attractive features of the fugacity approach is that it separates biological variables such as the flow rate (e.g., gill flow rate, ingestion rate, urine excretion rate) and chemical variables such as Z which control the physical–chemical partitioning of the chemical.

For example, for a chemical that is absorbed by a fish with a volume V_B (in m^3) from the water (via the gills) and eliminated to the water (via the gills) as well as being metabolized in the organisms, the mass balance equation is

$$N_B = V_B{\cdot}Z_B{\cdot}df_B/dt = D_W{\cdot}f_W - D_W{\cdot}f_B - D_M{\cdot}f_B \qquad [4]$$

where t is time (day). When expressed in terms of concentrations and kinetic rate constants, the same balance is given by

$$N = V_B{\cdot}dC_B/dt = k_1{\cdot}V_B{\cdot}C_W - k_2{\cdot}V_B{\cdot}C_B - k_M{\cdot}V_B{\cdot}C_B \qquad [5]$$

where k_1, k_2, and k_M are rate constants (in units of day^{-1}) for chemical uptake from water, elimination to the water, and metabolic transformation, respectively (**Figure 1**). The units of the rate constant can vary depending on the units selected for the concentration in the media involved. The rate constants can also be expressed in terms of

fugacity-based transport parameters, for example, k_1 equals $D_W/(V_B \cdot Z_W)$, which allows one to use both concepts and benefit from the fugacity-based approach to distinguish between biological and chemical variables in the model.

Equations [4] and [5] illustrate that after a long-term exposure, when a steady state is reached (i.e., $N = 0$), the chemical fugacities in water and organism are no longer equal, that is, $f_B/f_W = D_W/(D_W + D_M)$, with the fugacity in the organism being smaller than that in the water. In concentration format, this equates to $C_B/C_W = k_1/(k_2 + k_M)$.

Biomagnification

One of the key principles in a food-web bioaccumulation model is the biomagnification effect, which causes the fugacity and concentration of the chemical to increase with increasing trophic level. This process can lead to food-web magnification of the chemical when this process occurs at each predator–prey interaction in the food web. Food-web biomagnification alone can produce a 10 000–100 000-fold increase in lipid-normalized concentration of a bioaccumulative substance. Biomagnification is of ecotoxicological significance because it can cause organisms at higher trophic levels to be exposed to high concentrations, which can produce toxicological effects or high risk levels. In our models and those of Mackay and co-authors, this occurs as a result of food absorption and digestion and can be described by the following mass balance equation:

$$N_B = V_B \cdot Z_B \cdot df_B/dt = D_D \cdot f_D - D_F \cdot f_B \quad [6]$$

where f_D is the chemical fugacity in the diet, D_D is the dietary ingestion rate, which is the product of the dietary ingestion rate G_D ($m^3\,day^{-1}$) and the fugacity capacity of the diet Z_D (i.e., $G_D \cdot Z_D$), and D_F is the fecal egestion rate of the chemical (**Figure 1**). Hence, at steady state ($N_B = 0$), it follows that

$$f_B/f_D = D_D/D_F = (G_D/G_F) \cdot (Z_D/Z_F) \quad [7]$$

illustrating that the fugacity in the organism (f_B) exceeds that in its diet (f_D) as a result of dietary uptake because the feeding rate G_D exceeds the fecal excretion rate G_F due to food absorption and Z_D exceeds Z_F because of food digestion which leaves the feces depleted of lipids, proteins, and other food constituents that give the food its high fugacity capacity. This magnification effect is approximately 8 times in fish, but much higher in mammals, birds, and humans with a more efficient digestive system. A graphical presentation of the magnification effect is presented in **Figure 2**. However, for the gastrointestinal magnification effect to cause biomagnification, it is key that the combined rate of chemical elimination due to metabolic transformation and excretion in the organisms is slow. However, if the combined rate of chemical elimination is high, then a high chemical concentration in the organism cannot be maintained and the chemical will not biomagnify. Chemicals which are predominantly absorbed via the diet and subject to a high rate of chemical elimination due to metabolic transformation and other excretion rates, will exhibit concentrations in the organisms of a food chain that decline with increasing trophic level. This phenomenon is sometimes referred to as trophic dilution, which is the opposite of biomagnification.

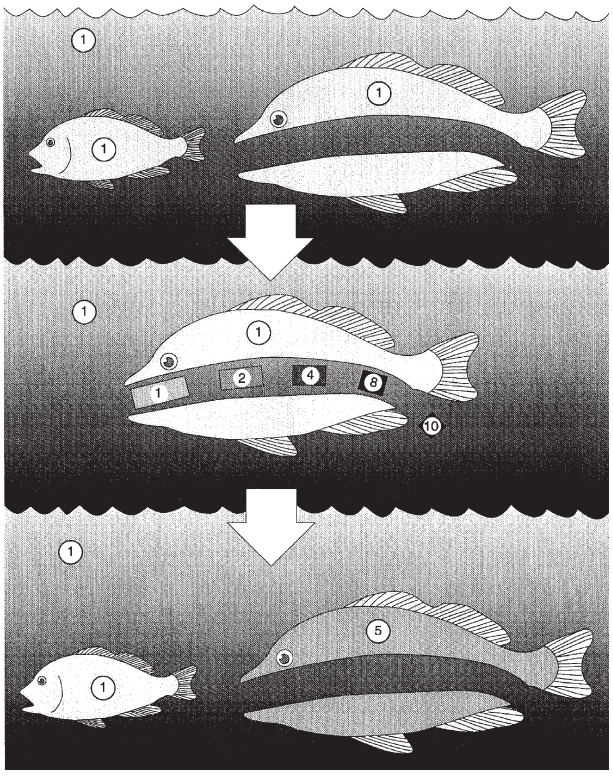

Figure 2 Conceptual diagram depicting the mechanism of biomagnification. Top panel: Predator with a chemical fugacity of 1 Pa in water with a chemical fugacity of 1 Pa consuming prey with a chemical fugacity of 1 Pa. Middle panel: Because predator and prey are at the same fugacity (i.e., no net passive uptake of chemical), food absorption (which reduces the volume of food in the gastrointestinal tract) and food digestion (which reduces the fugacity capacity of the intestinal contents) produce an increase in chemical fugacity in the gastrointestinal tract which leads to net uptake of chemical in the predator Bottom panel: Net chemical uptake will cause a fugacity in the predator that exceeds the fugacity in its prey (i.e., biomagnification) as long as the combined chemical elimination rate by metabolic transformation, elimination to water, and growth dilution are slow.

Integration

It is important to recognize that body burdens of contaminants in animals are the combined result of a number of chemical uptake and elimination processes acting together. In water-respiring organisms (e.g., fish), the

most important processes are respiratory uptake via the gills and body surface area, dietary uptake and elimination via the respiratory surface, fecal egestion, and metabolic transformation. Growth of the animal is also often viewed as an elimination process and referred to as 'growth dilution' although no chemical is actually excreted or transformed. An increase in body mass has a 'diluting' effect on the chemical mass in the organisms. Hence, growth is often treated as an elimination process in bioaccumulation models. A model for bioaccumulation in fish can therefore be formulated as:

$$\begin{aligned} N_B &= V_B \cdot dC_B/dt \\ &= k_1 \cdot V_B \cdot C_W + k_{D,i} \cdot V_B \cdot \sum (P_i \cdot C_{D,i}) \\ &\quad -(k_2 + k_E + k_M + k_G) \cdot V_B \cdot C_B \end{aligned} \quad [8]$$

where k_1, k_D, k_2, k_E, and k_G are the rate constants (in units of day^{-1} if concentrations are in $mol\,m^{-3}$) for chemical uptake via the respiratory area (k_1), uptake via food ingestion (k_D) and elimination via the respiratory area (k_2), excretion into egested feces (k_E), metabolic transformation (k_M) and growth dilution (k_G); P_i is the fraction of the diet consisting of prey item i, $C_{D,i}$ is the concentration of chemical ($g\,kg^{-1}$) in prey item i, k_2 is the rate constant (day^{-1}) for chemical elimination via the respiratory area (i.e., gills and skin), k_E is the rate constant (day^{-1}) for chemical elimination via excretion into egested feces, and k_M is the rate constant (day^{-1}) for metabolic transformation of the chemical (**Figure 1**). A similar approach can be followed to develop models for other species. For example, for mammals (**Figure 3**), we have used

$$\begin{aligned} N_B &= V_B \cdot dC_B/dt \\ &= k_A \cdot V_B \cdot C_A + k_{D,i} \cdot V_B \cdot \sum (P_i \cdot C_{D,i}) \\ &\quad -(k_O + k_E + k_U + k_G + k_P + k_L + k_M) \cdot V_B \cdot C_B \end{aligned} \quad [9]$$

where C_A is the gaseous chemical concentration in the air and k_A, k_O, k_U, k_P, and k_L are the rate constants (in units of day^{-1} if concentrations are in $mol\,m^{-3}$) for chemical uptake via the respiratory area (k_1), elimination via the respiratory area (k_O), excretion into urine (k_U), production in female animals (k_P), and lactation in female animals (k_L).

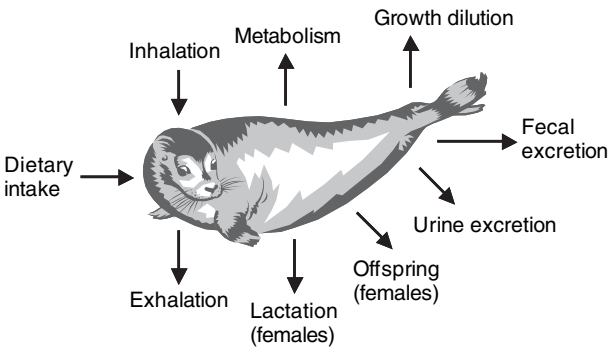

Figure 3 Chemical uptake and elimination processes included in the bioaccumulation model for organic contaminants in mammals.

This modeling approach 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. There is considerable evidence, especially for poorly metabolizable substances after long exposure periods, that supports this assumption. However, since the model is not designed to estimate concentrations in specific organs, the model is best applied in situations where the mass or concentration of the chemical in the whole organism is of interest. Internal physiological based pharmacokinetic (PBPK) models are more suitable to estimate concentration differences between various parts of the organism. Second, it is assumed that the organism can be described as a single compartment in its exchange with its surrounding environment. The one-compartment model for an organism is best applied in situations where variations in concentration over time are relatively slow or of secondary concern. A third assumption of the model concerns chemical elimination associated with sexual reproduction and offspring production. Examples are egg deposition or sperm ejection in fish and parturition in mammals. Studies in fish have shown that lipid-normalized concentrations of many persistent organic chemicals in eggs and adult female fish are often approximately equal. This implies that while 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 (e.g., male fish) is through growth dilution associated with the formation of eggs in the fish. Formation of eggs produces extra tissue in which the chemical resides, hence reducing the chemical's concentration. Offspring production in female mammals and birds follow a similar mechanism. Equations [8] and [9] illustrate that this growth dilution effect is counteracted by uptake of chemical from water and the diet and that the balance of these processes controls the ultimate concentration in the organism.

The practical application of eqns [8] and [9] to environmental pollution problems is often limited by access to time-dependent model input parameter values. Hence, for the model to become useful, it is often further simplified by applying a steady-state assumption ($N_B = 0$). The steady-state assumption transforms eqn [9] into

$$C_B = \left(k_1 \cdot C_W + k_{D,i} \cdot \sum (P_i \cdot C_{D,i})\right) / (k_2 + k_E + k_M + k_G) \quad [10]$$

The steady-state assumption is reasonable for applications to field situations where 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), as steady state is achieved rapidly in these situations. It should be used with caution in situations where the exchange kinetics are very slow (e.g., slowly metabolizable chemicals of high K_{OW} (i.e., larger than $10^{7.5}$) in large, lipid-rich organisms), because steady state takes a long time to achieve. In cases where changes in concentrations with the age of the organism are of interest, it is possible to introduce various age classes of the species and 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 that the model applies, the growth of the organism can be represented by a constant fraction of the organism's body weight W_B. The main driving forces of the kinetic bioaccumulation model are: (1) the chemical partitioning of chemical between water and the organism, represented by k_1/k_2; (2) the dietary magnification of chemical, represented by k_D/k_E; and (3) the combined rate of chemical elimination via metabolic transformation, growth dilution. It is noteworthy that any error in the estimation of the respiration rate, which affects both k_1 and k_2, has a tendency to cancel out, causing the ratio of k_1 and k_2 not to be affected. The same argument applies to the ingestion rate, which is related to the egestion rate. This gives the model some remarkable robustness. However, errors in respiration and ingestion rates do affect the relative contribution of the various uptake and elimination pathways and hence the model outcome.

Trophic Interactions

Describing the transfer of contaminants between organisms due to feeding interactions is challenging as food webs are typically complex and vary as a function of time and space. The role of food-web models is to simplify this process to a level that is understandable and can provide useful information. Since the scope of the model is typically limited to several key species, it is often sufficient to include only the most relevant trophic interactions relating to these species of interest (**Figure 4**). Also, because chemical concentrations in organism lipid tissues tend to increase significantly between trophic positions but considerably less between organisms that occupy a similar trophic level, it is often possible to 'lump' species of comparable trophic guilds. The latter should be done with caution as some species exhibit specific feeding behaviors that cannot be generalized to other organisms.

In the development of a food-web structure for modeling the bioaccumulation of contaminants, some basic rules of thumb can be suggested:

1. Include species of primary management interest.
2. Include species that can be considered residents of the area of interest unless migrant species are of importance.
3. Include species representing trophic guilds that are of key relevance to the food-web transfer and accumulation of polychlorinated biphenyls (PCBs) in the species of interest. For example, phytoplankton and algae, zooplankton, filter-feeding invertebrates, benthic detritovores, juvenile and adult fish, male and female fish-eating birds, and male, female, and juvenile marine mammals.
4. Minimize the number of species included in the model by representing key trophic guilds by one or two species. This is done to simplify the model and make the calculations more transparent.
5. Include species for which empirical concentration data are available. This provides the opportunity to test and ground-truth the model's calculations.

The structure of the food web represented in food-web models is typically subject to uncertainty. As a test to check whether the trophic structure of the model is adequate for chemical bioaccumulation modeling, it is often useful to explore the relationship between the trophic level of the species of the model as assigned by the feeding relationships used in the model, and stable nitrogen isotope ratios measured in samples of the species included in the model (**Figure 5**). Stable isotope ratios provide an empirical measure of the trophic status of the organism, with stable isotope ratios increasing with increasing trophic level. A good correlation between trophic level and stable nitrogen isotope ratios provides confidence in the structure of the food web for the purpose of food-web bioaccumulation modeling of contaminants.

Energy Balance

While the mass balance principle ensures the conservation of mass in the model, it does not necessarily ensure that an energy balance is maintained. The latter is important to avoid implausible scenarios such as an animal growing faster than it eats. It is therefore important to consider the relationships among the growth rate, the fecal egestion rate, and the feeding rate G_D as well as the sorptive capacity of fecal matter Z_F and the diet Z_D. This can be done by applying a general energy budget, that is,

$$I - L = R + P \qquad [11]$$

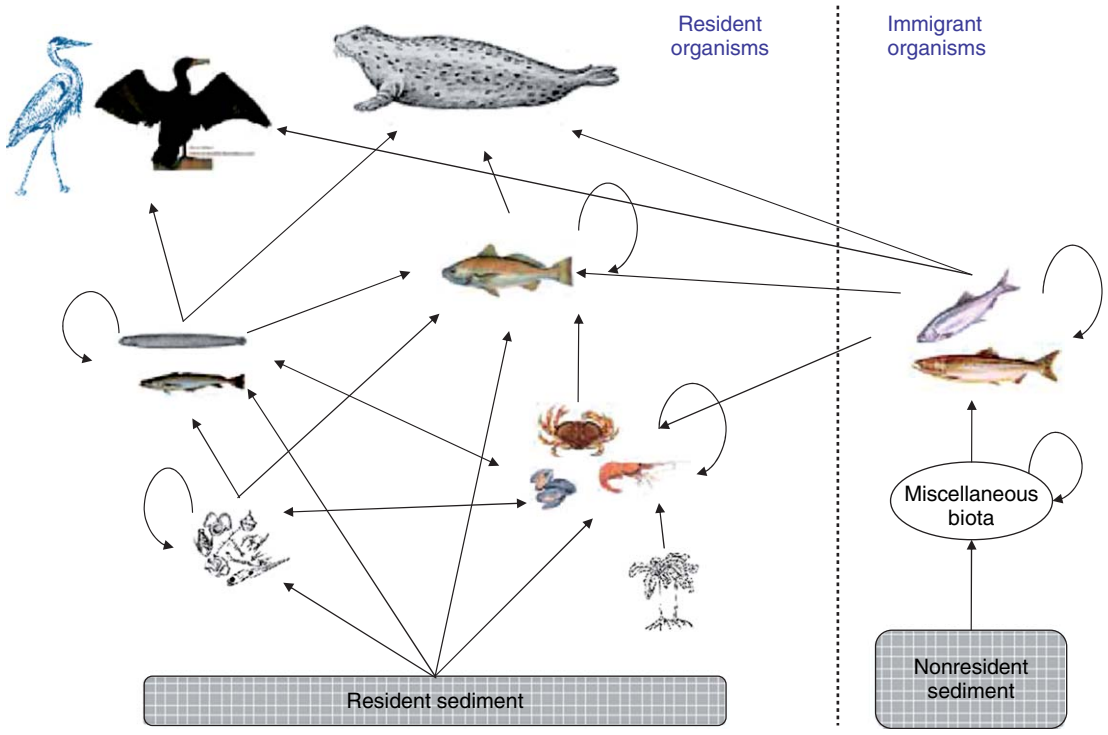

Figure 4 Illustrative example of a food-web structure used to describe the dynamics of contaminants in a West Coast marine food web that includes resident and migrant species.

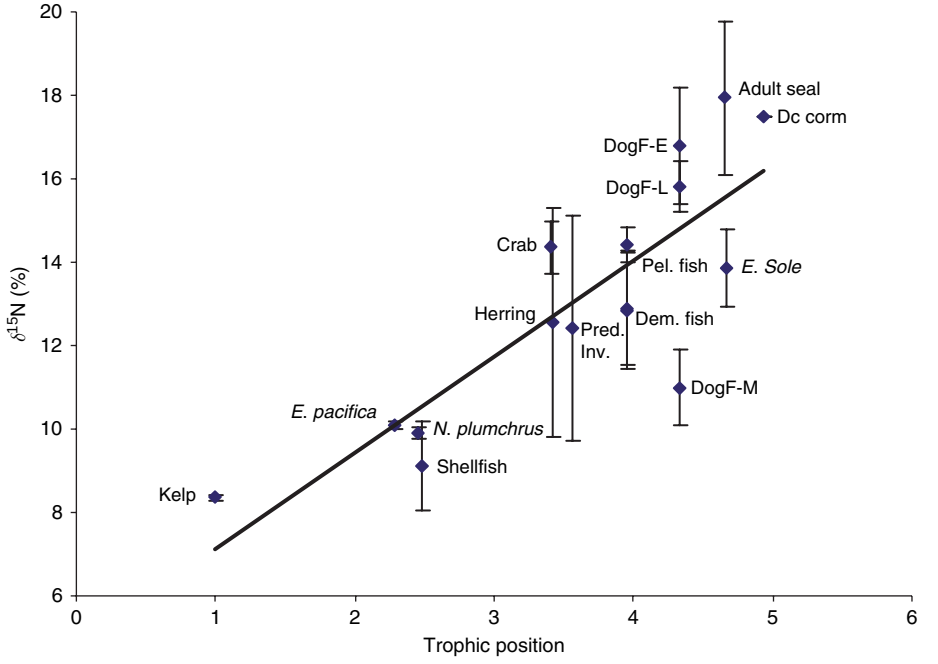

Figure 5 Illustrative example of the application of the relationship between trophic position, derived from gut content studies and stable $^{15}N/^{14}N$ isotope ratios ($\delta^{15}N$) measured in organism tissues, to test the applicability of the trophic structure of the model's food web. A high degree of correlation is indicative of an appropriate food-web structure.

where I is energy ingestion, L is the sum of fecal and urinary losses, P is production, and R is respiration, all expressed in units of energy flux ($kJ\,day^{-1}$). These can be converted to mass fluxes ($g\,day^{-1}$) by energy–biomass interconversion ratios. The application of the energy mass balance in the food-web bioaccumulation model makes it possible to include bioenergetic efficiencies in the food-web model. This provides the

opportunity to apply the model to a large variety of species for which bioenergetic efficiencies are known. This is an interesting application of the model for ecotoxicological evaluations as it makes it possible to assess which species are most likely to receive highest body burdens of particular contaminants and are potentially at the highest risk.

Model Parametrization

Upon verification of the model's logic and correctness, the model's key state variables are parametrized, by compiling data on chemical properties, environmental conditions, and biological variables. An important consideration is to use data that represent the system of interest as best as possible. This ensures that the concentrations of contaminants calculated by the model can be compared to observed concentrations.

Sensitivity analysis

The purpose of the sensitivity analysis is to provide insight into the relative importance of the various state variables to the outcome of the model. This is useful in the analysis of the internal mechanics of the model. It can be used to characterize potential errors in the model and to develop a better understanding of the interaction of the processes that control the behavior of the contaminant in the food web. In multiparameter models like the food-web model, it is important to keep in mind that the sensitivity of each model variable is a function of other state variables of the model. There are various methods that can be used to conduct sensitivity analysis. The simplest technique involves the variation of a particular parameter and recording its effect on the model output; for example, the contaminant concentration in a target organism. Other techniques use stochastic procedures such as Monte Carlo simulation (MCS) to express the relative importance of various state variables and parameters of the model.

Calibration and hypothesis testing

Model calibration is a technique in which the model parametrization and/or the model structure are altered to produce a better agreement between observations and model predictions. Calibration methods include 'fine-tuning' model parameters and the application of concentration observations in the basic structure of the model. While the application of model calibration depends on the objective of the model, the model strategy, and the state of knowledge and information about the contamination problem at hand, we found that for estimating hydrophobic organic chemical concentrations in organisms of food webs, there is rarely a need for model calibration as long as the model is used within its application domain. The food-web modeling approach described in this article is a mechanistic model and can be used without including empirical chemical concentration data. In many cases though, there is a need to check whether the model calculations are consistent with available empirical data such that confidence in the model is gained. This can be done in a 'hypothesis-testing' approach where the model outcomes are compared with independent data (data not used in the construction of the model). For example, if the preferred model outcome is the chemical concentration in a particular species (e.g., fish), this involves the comparison of observed and predicted concentrations in that fish species. Various measures can be used to express the degree to which the model outcomes match the observations. In the past, we have used the mean model bias $\overline{MB}$ to express the central tendency of the model:

$$\overline{MB} = 10^{\left(\sum_{i=1}^{n}\left[\log\left(C_{P,i}/C_{O,i}\right)\right]/n\right)} \quad [12]$$

where $C_{P,i}$ and $C_{O,i}$ are the concentrations of the chemical in a particular species that are respectively predicted by the model and observed in the field and i can refer to the number of observations (i.e., for a particular chemical in a particular species), number of chemicals (in a particular species), or a number of species (for a particular chemical substance). In essence, $\overline{MB}$ is the geometric mean (assuming a log-normal distribution) of the ratio of predicted and observed concentrations. $\overline{MB}$ is a measure of the systematic over- ($MB > 1$) or underprediction ($MB < 1$) of the model. It should be stressed that in the calculation of MB, over- and underestimations have a tendency to cancel out. Hence, it describes the central tendency of the model outcome. Variability in the over- and underestimation of measured values can be represented by the 95% confidence interval of $\overline{MB}$. Due to the lognormal distribution of the ratio of predicted and observed BSAFs, this variability can be expressed as a factor (rather than a term) of the geometric mean. For example, if the 95% confidence interval of the $\overline{MB}$ is 3, it means that 95% of the predicted/observed concentration ratios are found between $\overline{MB}/3$ and $\overline{MB} \times 3$.

Uncertainty analysis

The role of the 'uncertainty analysis' is to assess the error in the model calculations. The uncertainty analysis is important because the magnitude of the model needs to be considered when interpreting the results of the model calculations for management purposes. One of the most established techniques for conducting uncertainty analysis of models is MCS, which calculates the effect of inherent error in the model state variables and parameters on the model outcome. This methodology is based on the representation of the model state variables by statistical distributions rather

than point estimates. The distribution represents the uncertainty in the value of the model state variable used in the model. The distribution expresses how the state variables may vary due to geographical location, time of the year, differences in behavior among individuals of a species, and other factors. In MCS, these distributions are repeatedly sampled and the sampled values are used in the model to produce a distribution of model outcomes (e.g., chemical concentrations in fish). This distribution of model results represents the variability in the model outcome due to variability and error in the model's state variables (temperature, organic carbon content, lipid contents, etc.). It represents the model uncertainty. The uncertainties in all state variables contribute to the magnitude of the range of model outcomes; however, the contributions are not necessarily additive. The uncertainty calculated through MCS has a strong theoretical foundation. However, it is subject to difficulties associated with the characterization of errors in model parameters and it cannot include errors in model architecture.

One of the key requirements for a meaningful MCS is that the model state variables included in the MCS are independent. In a food-web bioaccumulation, this requirement can pose difficulties. For example, for a predator with three prey items, uncertainty in the proportion of prey item 1 consumed by the predator has direct consequence for the proportion of the other two diet items in the predator's prey. Hence, diet item proportions or feeding preferences are typically not independent. Other examples of related state variables are animal size, growth rate, lipid content, and feeding rate. Also, regression coefficients in regression equations introduce co-dependency. Before attempting to apply MCS to food-web bioaccumulation models, it is important to ensure that in the model structure chosen the model state variables are indeed independent since lack of dependence creates biologically implausible model outcomes that should not be considered. A second requirement for MCS is a realistic characterization of the uncertainty in each state variable. The latter is not always available for all model state variables and parameters. For example, feeding preferences derived from gut content studies contain considerable uncertainty that is often not characterized.

An alternative method for determining model uncertainty is to use calculated differences between observed and predicted chemical concentrations. This method applies the mean model bias and its 95% confidence interval to the model outcome (e.g., concentration) to predict a distribution of concentrations that includes 95% of the observed data. This method requires that observed concentration data are available. If this is not the case, it is sometimes possible to use uncertainty calculations from application of the model to other systems as a measure of model uncertainty. The application of available site-specific concentration data to characterize model uncertainty has the advantage that estimates of model uncertainty are grounded in empirical observations. However, this method is subject to the limitations of the sampling programs used to obtain the contaminant concentrations. For example, in larger systems, monitoring programs may only have collected samples from a subpopulation of the larger population of a particular species. In that case, it is possible that the distribution of the concentrations in the sampled organisms does not accurately represent the actual distribution of chemical concentrations in the population of the system. In such cases, the model uncertainty may be underestimated.

Food-Web Model Application

Food-web bioaccumulation models have been applied in a number of different ways. However, in terms of assessing ecotoxicological risks, two main methods of application should be emphasized. The first method, referred to as the 'forward calculation', uses observed distributions in measured chemical concentrations in the water and sediments as the starting point of the model (i.e., the external variable or forcing function) to calculate the anticipated corresponding concentration in the wildlife species of interest. The resulting distribution can then be compared to tissue residue guidelines or toxicological threshold values to derive the fraction of the affected wildlife population that contains chemical concentrations greater than or below the reference value of interest. This is illustrated in **Figure 6**, which illustrates the application of the model to the calculation of PCBs concentrations in harbor seal pups as a result of exposure to PCB concentration distributions in water and sediments. In this particular example, the distribution of PCB concentrations in water and sediments of the affected water body can be expected to produce PCB concentrations that vary substantially as described by the distribution with a large percentage of the concentrations exceeding the threshold effects concentration.

The second method, referred to as the 'backward calculation', applies the model to back-calculate what distribution of PCB concentrations in water and sediments can be expected to produce a particular distribution of concentrations in the target species of ecotoxicological concern or interest. The application of the model is illustrated in **Figure 7** and refers to a management goal to ensure that 95% of the population of a particular target species (e.g., seal pups) contains chemical (e.g., PCBs) concentrations less than a threshold effects concentration (e.g., 5000 ng/g ww). The food-web

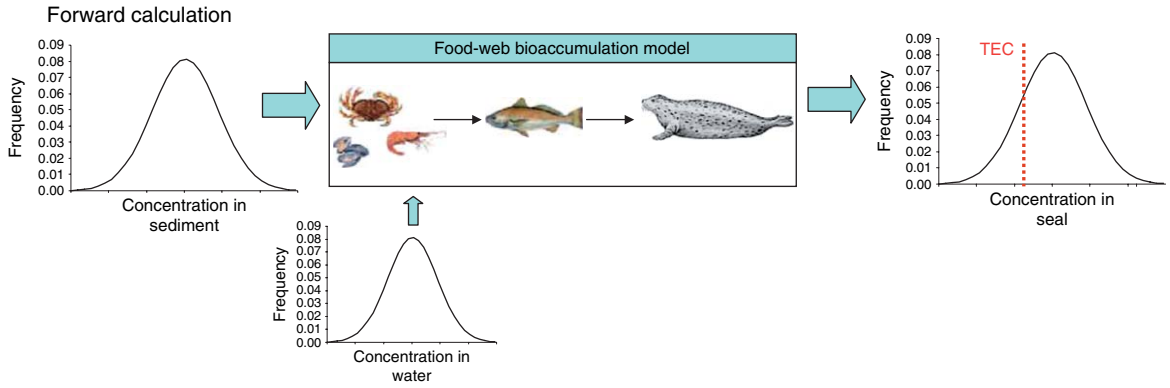

Figure 6 Illustration of the application of the food-web model to assess the ecotoxicological risk of a contaminant in a higher-trophic-level organism (seal). Observed chemical concentrations in sediment and water (presented as statistical distributions) are entered in the model to derive the chemical concentration distribution in a resident seal population and the incidence of concentrations greater than the toxicological threshold effect concentration (TEC).

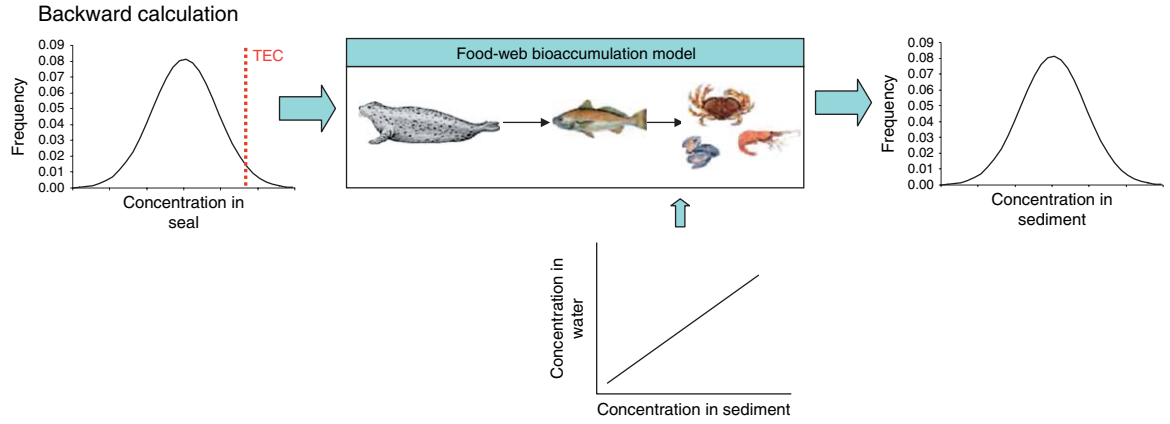

Figure 7 Illustration of the application of the food-web model to derive the system-wide sediment concentration distribution that can be expected to meet an acceptable risk level, set at 5% of the target population of seals exceeding the toxicological threshold effect concentration (TEC).

bioaccumulation model is then used to calculate what distribution in PCB concentrations in the sediments of the system can be expected to produce this distribution. The relationship between PCB concentrations in water and sediments, determined from monitoring programs or estimated using models, needs to be known for this purpose. The calculated distribution can then serve as a remediation or pollution control objective or a sediment quality criterion for the protection of wildlife species.

Examples of the application of the food-web bioaccumulation for ecotoxicological risk assessment, including the use of forward and backward calculations, include the San Francisco Bay food-web bioaccumulation model. The model is documented in a Gobas and Arnot report listed on the website of the San Francisco Bay Clean Estuary Partnership (CEP), and can be downloaded in the form of a Microsoft EXCEL® workbook from http://www.rem.sfu.ca. The purpose of this model is to estimate concentrations of PCBs in a set of key species that reside in the Bay, including double-crested cormorants, the Forster's tern, the harbor seal, and three fish species that are frequently caught by fishermen in the bay, as a result of PCB concentrations in sediments and water in the bay. The model can be used to determine what concentrations of PCBs in the water and sediments of the bay need to be reached to achieve an adequate margin of safety in wildlife and humans exposed to PCBs in the bay area. This information can be used as part of a TMDL characterization to formulate remedial actions to achieve desired water quality goals. The management module includes a simple worksheet to conduct two types of calculations, viz. 'forward' calculations to estimate the concentrations of PCBs in biota of the bay from PCB concentrations in the sediments of the bay and 'backward' calculations to calculate the PCB concentrations in the sediments of

the bay that are required to meet PCB concentration based criteria in fish and wildlife for the bay. The backward calculation is designed to determine target PCB concentrations in sediments that meet ecological and/or human health criteria.

Other applications of the food-web bioaccumulation include the estimation of the BAF and BCF for fish species in lower, middle, and upper trophic levels of aquatic food webs. The model predictions can include the effect of metabolic transformation and trophic dilution on the BAF if a reliable estimate of the chemical's metabolic transformation rate in fish is available. The model is named BAF-QSAR v1.1 and is coded in a Microsoft EXCEL® workbook, is freely available for download, and can be run for a large number of chemicals.

Food-web bioaccumulation models have also been used for the derivation of water quality guidelines. For example, the Gobas 1993 model, which was originally published for application to the Lake Ontario ecosystem and has since been applied to many other ecosystems by several authors, has been reviewed and adopted by the US Environmental Protection Agency for developing water quality criteria and waste load allocations in the US under the Great Lakes Water Quality Initiative (EPA-822-R-94-002). This model has been updated and is now referred to as AQUAWEB v1.1. It provides site-specific estimates of chemical concentrations in organisms of aquatic food webs from chemical concentrations in the water and the sediment. Key revisions included new equations for the partitioning of chemicals into organisms, new 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 novel mechanistic model for predicting gastrointestinal magnification of organic chemicals in a range of species. The model has been evaluated using empirical data from three different fresh water ecosystems involving 1019 observations for 35 species and 64 chemicals. Both models are coded in one Microsoft EXCEL® workbook and can be downloaded from http://www.rem.sfu.catoxicology.

See also: Bioaccumulation; Biomagnification; Food Chains and Food Webs.

Further Reading

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Relevant Website

http://www.rem.sfu.ca – The Environmental Toxicology Research Group, School of Resource and Environmental Management (REM).