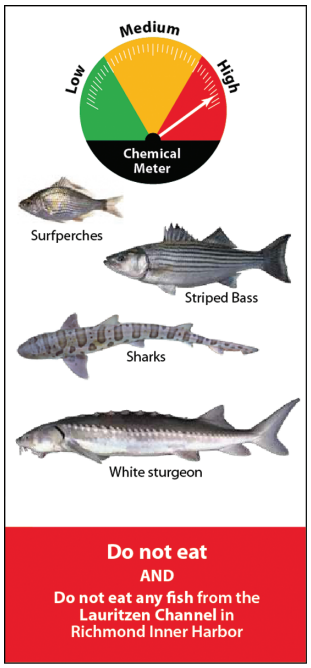
Chapter 16

Equilibrium Partitioning From Water and Air to Biota

Environmental Organic Chemistry, Third Edition. René P. Schwarzenbach, Philip M. Gschwend, and Dieter M. Imboden.
Copyright © 2017 John Wiley & Sons, Inc. Published 2017 by John Wiley & Sons, Inc.
Companion website: www.wiley.com/go/Schwarzenbach/EnvironmentalOrganChem3e

16.1 Introduction



San Francisco Bay fish advisory. Graphic: C.A. OEHHA.

Imagine it's a sunny day outside and you decide to go fishing. At a new fishing site, you discover signs warning you not to eat certain fish as they may be contaminated with organic chemicals like PCBs and DDT, as well as mercury. In 2011, the U.S. EPA reported a total of 4,821 active fish advisories, with over 25% of the affected waterbodies contaminated with "legacy POPs" (see Chapter 3) (U.S. EPA, 2013). These warnings stem from the accumulation of toxic chemicals from the environment in organisms at the base of food webs all the way up to higher organisms, such as humans (Fig. 16.1). As in previous chapters of Parts II and III, to understand how these chemicals transfer to these various organisms ("phases"), we must understand how a chemical's properties affect its behavior. Therefore, the goal of this chapter is to enhance our understanding of the various factors that determine where and to what extent organic chemicals accumulate in living media.

The term *bioaccumulation* denotes a sum of different processes causing the accumulation and therein amplification of contaminants in the tissue of an organism (see MacDonald et al., 2002 and Fig. 16.1). The direct partitioning between water or air and living media is termed *bioconcentration*. *Biomagnification* encompasses a more complicated sequence of transfer processes in that compounds are taken up with food and then transported internally to various parts of the organism. The overall bioaccumulation process then represents a sum of these various concentration amplifications.

We begin our discussion of partitioning to biota by first considering equilibrium partitioning of organic chemicals between water or air and a simple "model organism" made up of defined biological media (Section 16.2). Such equilibrium considerations are very useful for assessing the potential of a given compound to bioaccumulate, a behavior we need to anticipate in order to judge the wisdom of using a particular chemical for purposes that ultimately result in their release to the environment. Such equilibrium considerations are also important for evaluating the chemical gradients driving transfers in real field situations, allowing us to identify environmental compartments such as contaminated sediments most in need of remediation. We then specifically discuss bioaccumulation in both aquatic and terrestrial systems (Sections 16.3 and 16.4). Finally, in Section 16.5, we learn how equilibrium partitioning considerations can be used to assess a compound's effectiveness in inducing non-specific toxic effects, and to identify whether a compound exhibits specific toxic effects in an organism.

16.2 Predicting Biota–Water and Biota–Air Equilibrium Partitioning

The Composition of Living Organisms

To predict the accumulation of organic chemicals in the tissues of organisms, we start by developing an awareness of the "chemical nature" of those living materials. By doing this, we hope to envision the intermolecular interactions that attract organic chemicals into organisms, much like we visualized the interactions that control a

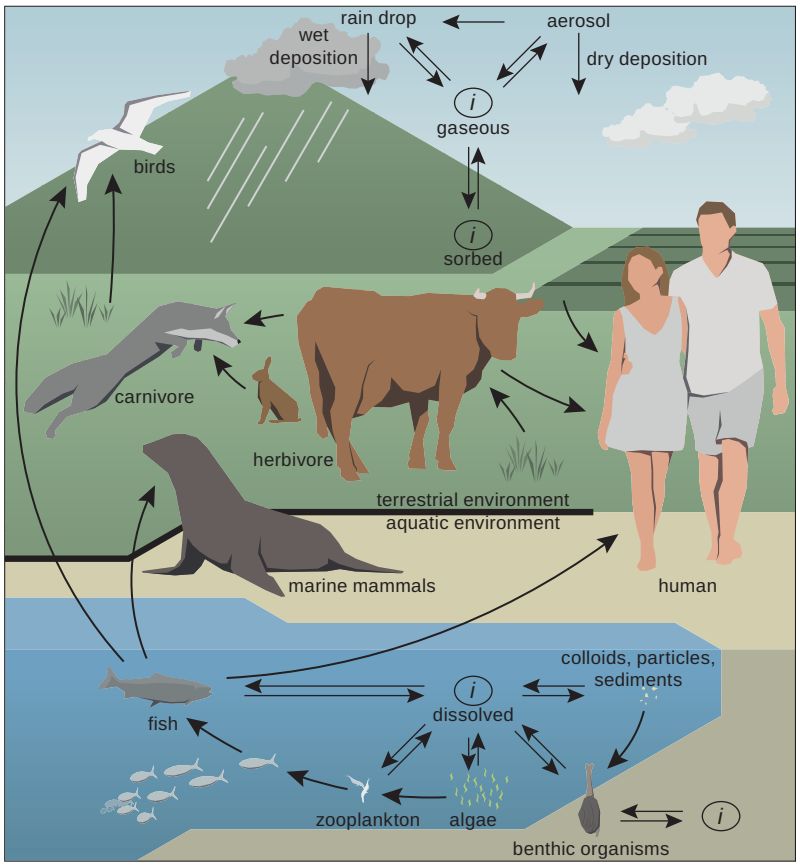


Figure 16.1 Examples of the transfer of a compound *i* from various media within the environment to organisms, including humans, by partitioning from surrounding media (bioconcentration) and by amplification in food webs (biomagnification).

specific compound's affinity for various organic solvents (Chapters 7 and 10). The compounds of interest to us are only about 1 nm or less in size. As such, we focus on organic portions of organisms that are much larger (e.g., proteins or lipids), like we did in discussing partitioning to natural organic matter (Chapter 13). Low-molecular-weight components like acetate or glucose that contribute only a few percent to organism biomass are not addressed.

In addition to water and inorganic solids, such as salts dissolved in cell fluids, shells, and bones, organisms consist of a mix of organic substances. Some of these are macromolecules (e.g., globular proteins and cellulose), while some are combinations of lipids, proteins, carbohydrates, and some specialized polymers like cutin or lignin that form subcellular and tissue "structures" (Fig. 16.2). These diverse organic materials cause organisms to have diverse macromolecular, cellular, and tissue structures that may be apolar, monopolar, or bipolar.

Looking now at the actual composition of different organisms (Table 16.1), in animals, the protein fraction is generally the largest, followed by carbohydrate components, and then a variable lipid content. Since lipids serve both as structural components (e.g., phospholipids in membranes) and as energy reserves (e.g., triacylglycerides), the contributions of total lipids may vary widely from organism to organism and from

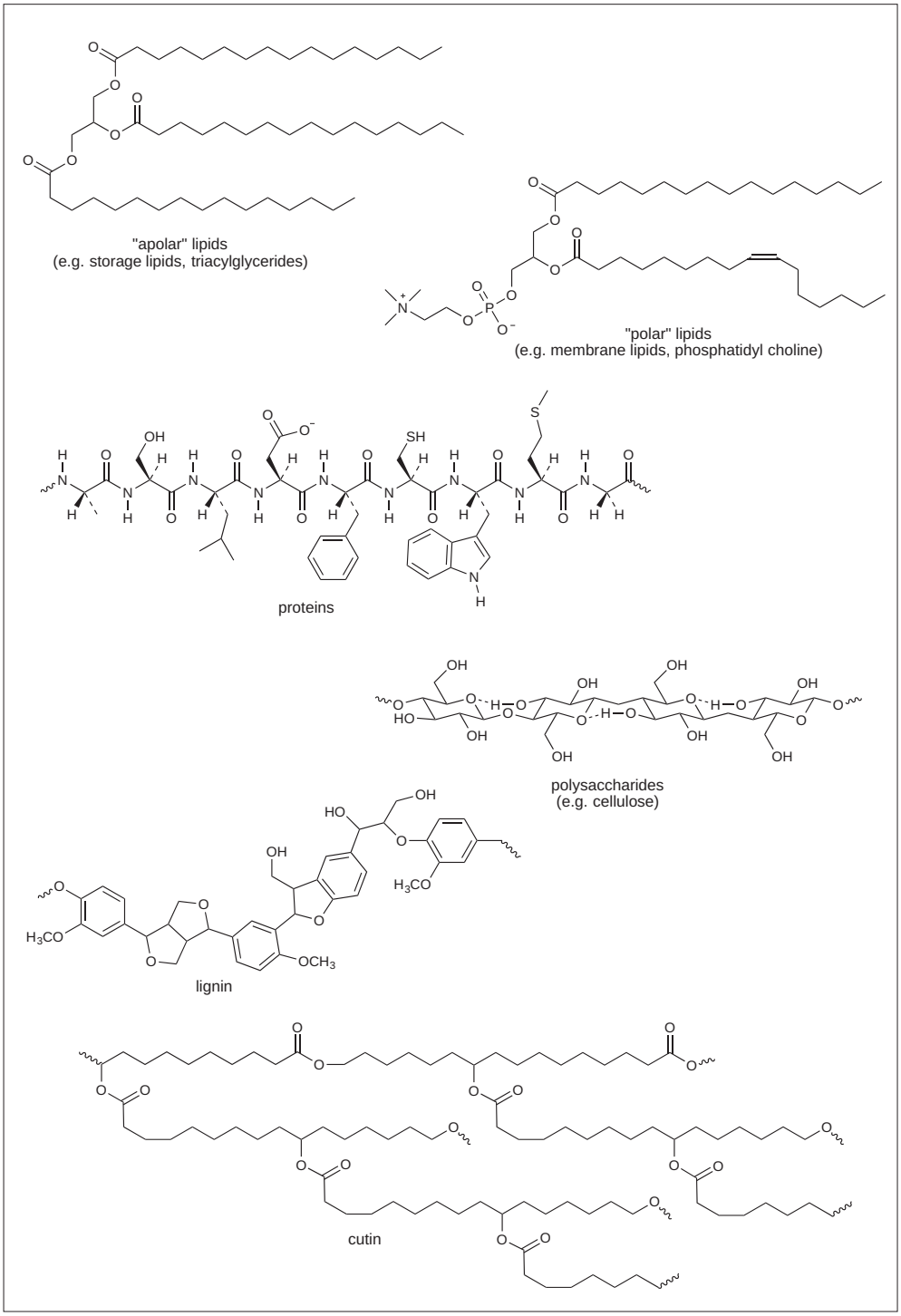


Figure 16.2 Examples of natural organic materials (biological phases) relevant for sorption of organic pollutants in living media. We consider triacylglycerides as primarily apolar although they contain monopolar (ester) groups.

tissue to tissue in the same organism. For example, on a *dry weight basis*, the lipid contents of phytoplankton typically range between about 10 and 30%, but this fraction may go as low as 1% (Shifrin and Chisholm, 1981; Stange and Swackhamer, 1994; Berglund et al., 2000). Similar ranges can be found in fish (Ewald, 1996; Henderson and Tocker, 1987; Berglund et al., 2000), zooplankton (Berglund et al., 2000) and

Table 16.1 Chemical Composition of Some Organisms (dry-weight, ash-free basis)[a]

Organism	% Lipid	% Protein	% Carbohydrate	Other
Bacterium				
Escherichia coli	10	60	5	25% DNA/RNA
Phytoplankton	20 ± 10	50 ± 15	30	
Lichen				
Cladonia spp.	2	3	94	
Vascular land plants				
"grasses"	0.5 - 2	15 - 25		1 - 5% cutin
deciduous leaves (oak, maple)	3	15	42	26% lignin
pine needles	28	8	47	17% lignin
"wood"	4	1	70	25% lignin
apples	5	0	95	
almonds	56	21	22	
spinach	0	50	50	
Aquatic invertebrates				
zooplankton	15 - 35	60 - 70	10	
copepod	10	65	25	
amphipod (*Pontoporeia hoyi*)	9 - 46			
shrimp (*Mysis relicta*)	10 - 41			
oyster	12	55	33	
zebra mussel	8 - 12	50 - 60	30 - 40	
polychaete (*Abarenicola pacifica*)	12			
chironomid larvae	6 - 13	65 - 71	21 - 23	
Terrestrial invertebrates				
earthworm (*Lumbricus rubellus*)	5 -15	60-70	8-20	
Aquatic vertebrates				
fatty fish				
trout filet	30	70	<3	
lake trout	6 - 18			
salmon	11			
lean fish				
cod filet	0.7			
pike filet	0.7			
terrestrial vertebrates				
deer meat	10	90	<1	
beef (roast)	20	80	<3	
caribou muscle	5 - 12			

[a]Data from Lawrence and Millar (1945); Ahti and Hepburn (1967); Parsons and Takahashi (1973); Beattie (1978); Hunt (1979); Shifrin and Chisholm (1981); Thomann and Connolly (1984); Neidhardt et al. (1990); Huston and Pinchak (1991); Napela et al. (1993); Sjöström (1993); Tolls and McLachland (1994); Ewald (1996); Rombauer et al. (1997); Ma et al. (1998); Aber and Martin (1999); Böhme et al. (1999); Cavaletto and Gardner (1999); Weston et al. (2000); Kelly and Gobas (2001); Beaumelle et al. (2014).

benthic invertebrates (Morrison et al., 1996; Cavaletto and Gardner, 1999). In mammals, including humans, individuals exhibit quite different lipid contents and it may vary substantially with time. Within a single organism, the composition can also vary widely, as exhibited by lipids in caribou. Caribou muscle contains only 1 to 2% lipid,

while the liver is comprised of 4 to 13% lipid and the fatty tissues have almost 80% (Kelly and Gobas, 2001).

Plants, such as terrestrial grasses and tree leaves, exhibit a relatively low lipid content of less than 5% (Aber and Martin, 1999; Böhme et al., 1999). Also, the protein content is more variable than in animals. On a dry-weight basis, in phytoplankton, protein contents range between 30% and 60% (Shifrin and Chisholm, 1981), whereas they occur at about 20% in grasses and leaves (Huston and Pinchak, 1991; Aber and Martin, 1999). The amount of protein in fruits and nuts is highly variable (Rombauer et al., 1997) and is virtually insignificant in wood. On the other hand, the wood of vascular land plants consists primarily of lignin (∼1/4) and carbohydrates, in particular, cellulose and hemicellulose (∼ 3/4; Sjöström, 1993).

Accumulation of xenobiotic chemicals in these different types of organic materials, or *biological phases*, may lead to differing local concentrations in an organism, depending on the chemical nature of the biological phase and compound. We need to link these local concentrations together to get an idea of partitioning in the whole organism. Thus, we now introduce a simple model to predict equilibrium partitioning in each of these defined *biological phases* and the organism as a whole.

A Simple Model to Estimate Equilibrium Partition Coefficients Between Whole Organisms and Water ($K_{i\text{orgw}}$) or Air ($K_{i\text{orga}}$)

Accumulation of organic chemicals in organisms is a dynamic process that may involve several uptake and elimination routes acting simultaneously. For many xenobiotic compounds, the physical uptake and depuration processes can occur "passively," that is without an organism's explicit effort to transport these substances. Often, the biological membranes, designed to keep contents and fluids on the inside separate from the outside, prove to be incapable of excluding nonionic organic chemicals. To evaluate the resultant effects of organic pollutants in organisms, it is useful then to try to estimate the concentration that would be established in the organism if that organism achieved partitioning equilibrium with its surroundings (e.g., water, air, soil, sediment).

We acknowledge that predicting the differing internal concentrations of a given organic chemical in organisms is necessary for a sound assessment of the chemical's toxicity, such as at the target tissue or site of toxic action (Sijm and Hermens, 2000; Escher and Hermans, 2004). In most cases, however, this is not possible. Nevertheless, knowing the average concentrations in whole organisms or in major tissues where chemicals tend to accumulate may be sufficient to predict the likelihood of any adverse effects or to estimate the influence of large masses of biota on the overall fluxes of organic compounds in the environment (e.g., forests; Wania and McLachlan, 2001).

To this end, analogous to defining an overall $K_{i\text{d}}$ value to quantify equilibrium sorption to soils or sediments (Chapter 12), we define here an overall organism–water partition

coefficient, $K_{i\text{orgw}}$ (Eq. 16-1) and an organism–air partition coefficient, $K_{i\text{orga}}$ (Eq. 16-2). One can also define overall organ–water and organ–air partitioning coefficients, using the same equations. Recognizing that organisms and organs are heterogenous mixtures of *biological phases* (e.g., lipids, proteins, cutin, lignin, and cellulose), we assume that (1) each of these biological phases acts independently and comes to an equilibrium with all other biological phases present and (2) the total organism's accumulation of the chemical can be estimated as the sum of uptakes into each of these phases:

$$K_{i\text{orgw}} = \frac{C^*_{i\text{org}}}{C_{i\text{w}}} = \sum_{\text{bp}} f_{\text{bp}} K_{i\text{bpw}} \tag{16-1}$$

and

$$K_{i\text{orga}} = \frac{C^*_{i\text{org}}}{C_{i\text{a}}} = \sum_{\text{bp}} f_{\text{bp}} K_{i\text{bpa}} \tag{16-2}$$

where the subscript "bp" denotes a given biological phase, f_{bp} is the fraction of that phase present in the organism considered, and $K_{i\text{bpw}}$ and $K_{i\text{bpa}}$ are the equilibrium partition coefficients between phase bp and water or air, respectively. $C^*_{i\text{org}}$ is the predicted total (average) concentration in the organism. We use an asterisk superscript to indicate that these predicted concentrations are those that would be in equilibrium with the environmental medium concentration, not actual, measured concentrations in the organism (see Sections 16.3 and 16.4). $C^*_{i\text{org}}$ is, therefore, also often referred to as the *Theoretical Bioaccumulation Potential* ($TBP_{i\text{org}}$).

Similar to natural organic matter, concentrations of organic pollutants in a biological phase (bp) are commonly expressed on a per *dry* mass scale (e.g., mol kg^{-1} dry bp):

$$K_{i\text{bpw}}(\text{e.g., in } \text{L}_\text{w}\text{kg}^{-1} \text{ dry bp}) = \frac{C^*_{i\text{bp}}(\text{e.g., in mol kg}^{-1} \text{ dry bp})}{C_{i\text{w}}(\text{e.g., mol L}_\text{w}^{-1})} \tag{16-3}$$

and

$$K_{i\text{bpa}}(\text{e.g., in } \text{L}_\text{a}\text{kg}^{-1} \text{ dry bp}) = \frac{C^*_{i\text{bp}}(\text{e.g., in mol kg}^{-1} \text{ dry bp})}{C_{i\text{a}}(\text{e.g., mol L}_\text{a}^{-1})} \tag{16-4}$$

$K_{i\text{orgw}}$ and $K_{i\text{orga}}$ (Eqs. 16-1 and 16-2) are then expressed in L_w kg^{-1} *dry* organism and L_a kg^{-1} *dry* organism, respectively, and f_{bp} is the mass fraction of bp given in kg bp kg^{-1} *dry* organism (see Table 16.1).

Often in the literature, one finds experimentally reported concentrations on a wet-weight (w.w.) or fresh weight (f.w.) basis. To predict concentrations on a wet-weight

or fresh-weight basis, the model Eqs. 16-1 and 16-2 have to be extended by the fraction of water, f_w, present in the organism considered:

$$K_{i\text{orgw}} = \frac{C^*_{i\text{org}}}{C_{i\text{w}}} = \left[\sum_{\text{bp}} f_{\text{bp}} K_{i\text{bpw}}\right] + f_{\text{w}} K_{i\text{ww}} \; (K_{i\text{ww}} = 1) \tag{16-5}$$

and

$$K_{i\text{orga}} = \frac{C^*_{i\text{org}}}{C_{i\text{a}}} = \left[\sum_{\text{bp}} f_{\text{bp}} K_{i\text{bpa}}\right] + f_{\text{w}} K_{i\text{wa}} \tag{16-6}$$

where f_{bp} is now the mass fraction of bp in kg bp kg^{-1} *wet* organism, $K_{i\text{orgw}}$ and $K_{i\text{orga}}$ are expressed in L_w kg^{-1} *wet* organism and L_a kg^{-1} *wet* organism, respectively, and the water–water and water–air, partition constants, $K_{i\text{ww}}$ and $K_{i\text{wa}}$, are given in $L_w\,kg_w^{-1}$ and $L_a\,kg_w^{-1}$ respectively. Since the density of water is close to 1 kg L^{-1}, the latter constants are virtually the same as when expressed on a volume/volume base. We come back to the application of Eqs. 16-1 and 16-2 or 16-5 and 16-6 after discussing the properties of the various pertinent biological phases with respect to their ability to accumulate organic pollutants.

Prediction of Partition Coefficients Between Defined Biological Phases and Water ($K_{i\text{bpw}}$) or Air ($K_{i\text{bpa}}$)

In order to estimate chemical concentrations in whole organisms, we sum the uptakes into different biological phases present in organisms (Eqs. 16-1 and 16-2). Important biological phases in biota include lipids, proteins, polysaccharides, and lignin. Here, we present current methods to predict the partitioning coefficients of such phases.

Storage and Membrane Lipids. Traditionally, lipids, in particular storage lipids (slip), have been considered the most important biological phases into which organic pollutants partition from both water and air. Considering the rather apolar structure of storage lipids (Fig. 16.2), with the knowledge acquired in Chapter 10, we agree that this assumption is in many cases certainly justified, particularly for accumulation of larger, apolar and weakly polar compounds such as PAHs, PCBs, and PBDEs. The more polar membrane lipids (mlip), which are the main components of biological membranes (Fig. 16.2), are, due to their lower abundances, in many cases not so important when considering a whole organism. In certain organs (e.g., liver, kidneys, brain), they are, however, major lipid components and are thus also important sites of accumulation (Endo et al., 2013). Furthermore, and most importantly, they are the main targets for non-specific toxicity of organic pollutants, which we discuss in Section 16.4.

Similar to how we described other partitioning processes, polyparameter linear free energy relationships (pp-LFERs) also exist for estimation of storage lipid– and membrane lipid–water ($K_{i\text{slipw}}$, $K_{i\text{mlipw}}$) as well as storage lipid– and membrane lipid–air ($K_{i\text{slipa}}$, $K_{i\text{mlipa}}$) partition coefficients (Table 16.2, Eqs 16-7 to 16-22), the latter being derived using the thermodynamic cycle. As demonstrated by Geisler et al. (2012), in the case of *storage lipids*, the specific fatty acid composition of the triglycerides

Table 16.2 pp-LFERs for Some Biological Phase–Water $\log K_{i\mathrm{bpw}}(\mathrm{L_w kg_{bp}^{-1}}) = v_{\mathrm{bpw}}V_i + l_{\mathrm{bpw}}L_i + s_{\mathrm{bpw}}S_i + a_{\mathrm{bpw}}A_i + b_{\mathrm{bpw}}B_i + c$), and Biological Phase–Air ($\log K_{i\mathrm{bpa}}(\mathrm{L_a kg_{bp}^{-1}}) = v_{\mathrm{bpa}}V_i + l_{\mathrm{bpa}}L_i + s_{\mathrm{bpa}}S_i + a_{\mathrm{bpa}}A_i + b_{\mathrm{bpa}}B_i + c$) systems at 37°C[a,b]

Phases		Fitted System Descriptors									
Biological phase–water	Equation	v_{bpw}	l_{bpw}	e_{bpw}	s_{bpw}	a_{bpw}	b_{bpw}	c_{bpw}	r^2	n[c]	S.D.
Storage lipids [d]	16-7	+4.11		+0.70	−1.08	−1.72	−4.14	−0.07	0.98	247	0.29
Membrane lipids [e]	16-8	+3.30		+0.74	−0.72	+0.11	−3.63	+0.29	0.98	131	0.27
Muscle protein [f]	16-9	+3.01		+0.51	−0.51	+0.26	−2.98	−0.80	0.95	46	0.22
BSA [g]	16-10	+2.82		+0.36	−0.26	+0.37	−3.23	+0.14	0.78	82	0.42
Octanol (bp = o)[h]	16-11	+3.74		+0.46	−1.03	−0.13	−3.22	+0.15			
Storage lipids [d]	16-12	+1.99	+0.58		−1.62	−1.93	−4.15	+0.55	0.99	247	0.20
Membrane lipids [e]	16-13	+1.73	+0.49		−0.93	−0.18	−3.75	+0.49	0.98	131	0.28
Muscle protein [f]	16-14	+2.13	+0.33		−0.59	+0.21	−3.17	−1.09	0.95	46	0.23
BSA [g]	16-15	+1.84	+0.28		−0.46	+0.20	−3.18	+0.20	0.79	82	0.41
Octanol (bp = o) [i]	16-16	+2.54	+0.37		−1.37	−0.32	−3.22	+0.35			
Air–water [j]		v_{aw}	l_{aw}		s_{aw}	a_{aw}	b_{aw}	c_{aw}			
	16-17	+2.21	−0.31		−2.26	−3.60	−4.37	+0.63	0.99	374	0.18
Biological phase–air [k]		v_{bpa}	l_{bpa}		s_{bpa}	a_{bpa}	b_{bpa}	c_{bpa}			
Storage lipids	16-18	−0.22	+0.89		+0.64	+1.67	+0.22	−0.08		247	
Membrane lipids	16-19	−0.48	+0.80		+1.33	+3.42	+0.62	−0.14		131	
Muscle protein	16-20	−0.08	+0.64		+1.67	+3.81	+1.20	−1.72		46	
BSA	16-21	−0.37	+0.59		+1.80	+3.80	+1.19	−0.43		82	
Octanol(bp = o)	16-22	−0.33	+0.68		+0.89	+3.28	+1.15	−0.28			

[a]Contains solute descriptors for the size of the compound (V_i), the excess molar refraction (E_i) or the log $K_{i\mathrm{hexadecane–air}}$ (L_i), the H-donor property (A_i), the H-acceptor property (B_i), and a "dipolarity/polarizability" parameter (S_i), plus the complementary fitted system descriptors characterizing the phase involved (lower case letters).
[b]$K_{i\mathrm{bpw}}$ and $K_{i\mathrm{bpa}}$ are given in $\mathrm{L_w\ kg^{-1}}$ bp and $\mathrm{L_a\ kg^{-1}}$ bp respectively.
[c]Number of compounds used for LFER.
[d]Data from Geissler et al. (2012); log $K_{i\mathrm{slipw}}$ range: –1 to 10.
[e]Data from Endo et al. (2011); log $K_{i\mathrm{mlipw}}$ range: –1 to 8.
[f]Data from Endo et al. (2012); log $K_{i\mathrm{improtw}}$ range: 1 to 5.
[g]BSA = Bovin Serum Albumin; Data from Endo and Goss (2011); log $K_{i\mathrm{bsaw}}$ range: 1 to 6.
[h]Data from Endo et al. (2013).
[i]Data from Endo (2014).
[j]Data from Endo et al. (2013).
[k]For the biological phase–air systems, only the equation containing L_i is given because it has been derived by the thermodynamic cycle using the corresponding pp-LFER for air–water partitioning, which uses L_i to take care of entropic contributions when partitioning from or to the gas phase (see Chapter 7): $K_{i\mathrm{bpa}} = K_{i\mathrm{bpw}}/K_{i\mathrm{aw}}$ or $\log K_{i\mathrm{bpa}} = \log K_{i\mathrm{bpw}} - \log K_{i\mathrm{aw}}$.

has no significant influence on the partition constants. Thus, the pp-LFERs given in Table 16.2 for partitioning into storage lipids, which have been derived from a combined experimental data set using various lipid phases including olive oil, milk fat, fish oil, linseed oil, goose fat, and soybean oil (Geisler et al., 2012), can be used

as a general predictive model for $K_{i\text{slipw}}$ and $K_{i\text{slipa}}$. Since *membrane lipids* consist mostly of phospholipids, liposomes made up of phosphatidyl-choline (Fig. 16.2) or of phosphatidyl-choline mixed with other membrane lipids are used as surrogates for membranes. Therefore, the majority of experimental data used by Endo et al. (2011) for the pp-LFERs given in Table 16.2 stem from such surrogates but are referred to as "membrane lipids." The incorporation of other lipids, in particular cholesterol, into the membrane may lead to significantly lower partition coefficients (up to a factor of 10), which has been attributed to a stiffening of the membrane leading to a decrease in fluidity (Kwon et al., 2007; Endo et al., 2011). A decrease in fluidity also occurs below the phase transition temperature (T_c), below which membranes change from a high-fluidity, liquid crystalline phase into a low-fluidity, gel phase, accompanied by a decrease in partition coefficients of up to two orders of magnitude (Endo et al., 2011). In the following discussions, we only consider membranes in the high-fluidity state, which should reflect natural conditions.

Compared to storage lipids, membrane lipids have distinct polar regions, so more polar compounds partition to different domains compared to nonpolar ones, and they accommodate charged species much better. To further compare these types of lipids, let us consider the calculated lipid–water partition coefficients (Eq. 16-12 and 16-13) of our test set of compounds, introduced in Chapter 7 (in the following referred to as "test set"). As is evident in Fig. 16.3, and as we would expect, apolar and weakly polar compounds tend to partition more favorably or similarly to the more apolar storage lipids as compared to polar compounds that generally favor the more polar membrane lipids. A similar trend is, of course, observed for partitioning to air. By subtraction of Eqs. 16-12 and 16-13, we can derive a pp-LFER for calculating the difference for individual compounds expressed by a (hypothetical) storage lipid–membrane lipid partition coefficient, $K_{i\text{slip/mlip}}$ (in kg mlip kg^{-1} slip):

$$\log K_{i\text{slip/mlip}} = 0.26V_i + 0.09L_i - 0.69S_i - 1.75A_i - 0.40B_i + 0.06 \quad (16\text{-}23)$$

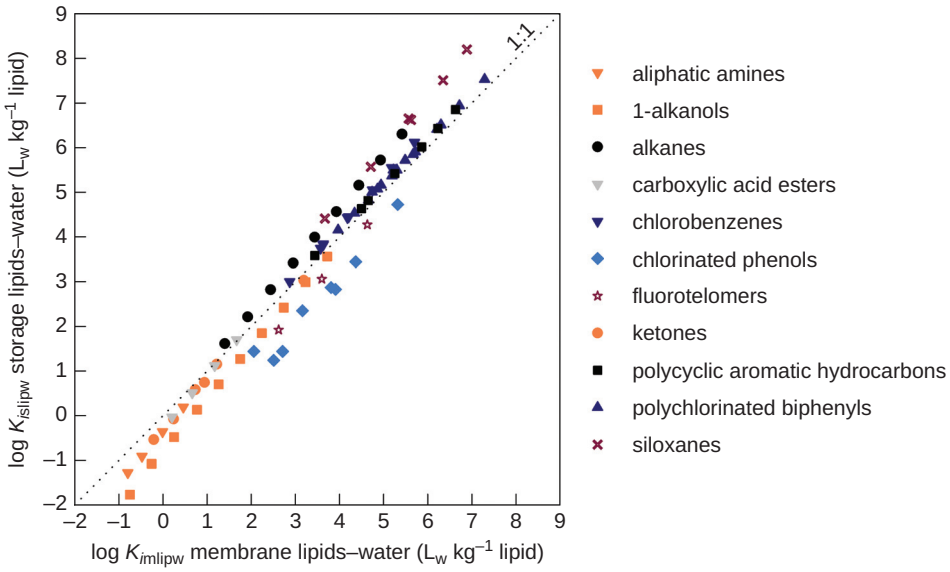


Figure 16.3 Comparison of partitioning to storage lipids and membrane lipids by the calculated log $K_{i\text{slipw}}$ versus log $K_{i\text{mlipw}}$ for our test set of compounds introduced in Chapter 7 at 37°C (Table 16.2, Eqs. 16-12 and 16-13).

Inspection of Eq. 16-23 shows that the cavity and dispersive vdW terms (*V*- and *L*-terms) favor the storage lipids, whereas the polar terms, and in particular, the *A*-term, favor the membrane lipids. For example, for *n*-heptane ($V_i = 1.09$, $L_i = 3.17$, $S_i = A_i = B_i = 0$) we obtain a $K_{i\text{slip/mlip}}$ of about 4, whereas for 1-hexanol ($V_i = 1.01$, $L_i = 3.61$, $S_i = 0.42$, $A_i = 0.37$, $B_i = 0.48$) the ratio is about 0.3, that is, the difference between the two compounds is more than a factor of 10.

Lipid-Like Plant Constituents. Lipid-like plant constituents, which we refer to as "plant lipids" (plip), are important major constituents of the outer barrier in plants (e.g., cuticals present in leaves and fruits, periderm of barks, and underground organs including roots). The major role of these barriers is to control water flux in the plant, but they also control fluxes of chemical species and protect against microbial infections (see Pollard et al., 2008; Heredia-Guerrero et al., 2014; Martin and Rose, 2014). To do so, these hydrophobic barriers contain various lipid-like compounds including *waxes* associated with either *cutin* or *cutan*, constituents of cuticles, or *suberin* or *suberan* which are constituents of, for example, barks or roots (Pollard et al., 2008; Chen and Schnoor, 2009; Shechter et al., 2010).

These plant lipids are somewhat operationally defined. The waxes are monomeric *extractable* mixtures of long-chain aliphatic compounds (C_{25} - C_{35}) exhibiting some polar functions including esters, alcohols, ketones, aldehydes, and carboxylic acids. Cutin and suberin are defined as *saponifiable* (hydrolyzable) long chain polymethylenic biopolymers linked primarily by ester functions, whereas cutan and suberan are referred to as the nonsaponifiable fraction postulated to be linked primarily by ether functions. All four biopolymers are based primarily on C_{16} - C_{18} hydroxy fatty acids or diacids, with subarin and suberan containing somewhat more long-chain components (C_{20} - C_{24}) as well as a substantial proportion of aromatic (phenolic) components (Turner et al., 2013). However, despite some noticeable differences in structure, all these plant lipids can be viewed as primarily aliphatic, and, thus, as mostly apolar organic sorbents exhibiting some H-accepting functional groups, much like the storage lipids (Fig. 16.2). Therefore, from a chemical composition point of view, we would not expect any fundamental differences in the sorbent properties among the plant lipids present in different plants, similarly to what has been found for the storage lipids originating from different organisms. Also, for apolar and monopolar H-acceptor compounds, we would assume quite similar sorbent properties as found for the storage lipids. The rather scarce experimental data available for partitioning of organic pollutants between plant lipids and water seem to support this assumption. We should note, however, that in some cases, nonlinear isotherms and slow sorption kinetics were observed, which can be attributed to the more rigid nature of these lipids as compared to storage or membrane lipids.

To date, the model compound most widely used to study plant lipid–water partitioning of organic pollutants is our companion compound phenanthrene. For a variety of cuticles, shoots, and roots, studies report log $K_{i\text{plipw}}$ values ($K_{i\text{plipw}}$ in L kg^{-1} lip) between 4.7 and 5.1 (e.g., Zhu et al., 2007; Shechter and Chefetz, 2008; Li and Chen, 2009; Shechter et al., 2010). For comparison, the storage lipid–water partition coefficient of phenanthrene (log $K_{i\text{slipw}} = 4.80$; Geisler et al., 2012) is in the same order of magnitude. The few data available for other PAHs show a similar picture (Chen

et al., 2005; Chen et al., 2011). Hence, for monopolar, H-acceptor PAHs, differences between plant lipids stemming from different origins seem to be rather small (within a factor of two to three), and $K_{i\text{slipw}}$ seems to be a reasonable surrogate for $K_{i\text{plipw}}$.

From the scarce experimental data available, no general rules can be derived for the plant lipid-water partitioning of bipolar compounds. Nevertheless, as found for the PAHs, differences in partitioning to different plant lipids seem to be quite small (i.e., within a factor of two to three), as reported for partitioning of phenol, 1-naphthol, atrazine, and chlorinated phenols to various fruit cuticle components (Chen et al., 2005; Shechter and Chefetz, 2008; Li et al., 2012). However, for these bipolar compounds, in contrast to the PAHs, the membrane lipid–water partition coefficient, $K_{i\text{mlipw}}$, tends to be a somewhat better surrogate for $K_{i\text{plipw}}$ than $K_{i\text{slipw}}$.

Proteins. Because of their diverse functions in organisms, proteins (see example in Fig. 16.2) exhibit a much greater structural variability both with respect to their chemical composition as well as with respect to their three-dimensional structure as compared to lipids. Furthermore, proteins exhibit a variety of structural units that may specifically interact with organic pollutants, even by forming covalent bonds (e.g., amino groups with aldehydes or ketones, see example given in Fig. 12.2, Chapter 12). Here, we consider two types of proteins: *structural proteins* that make up a significant portion of many organisms (Table 16.1), and *globular proteins*, in particular, the *serum albumins*, which have important functions in blood.

Structural proteins are bundled fibers or filaments present, for example, in muscles and protective covers of aquatic and terrestrial organisms. Muscle proteins (mprot), which are the most important structural proteins with respect to accumulation of organic pollutants, can be viewed as a mixed solid sorbent. This mixed phase is "solvent-like," in which non-specific binding dominates partitioning. Therefore, muscle proteins from three different sources (fish, chicken, and pig) were found to exhibit quite similar sorption characteristics for a diverse set of organic pollutants (Endo et al., 2012). Also, a single pp-LFER was derived from the combined data set for muscle protein–water partitioning coefficients, $K_{i\text{mprotw}}$, with quite reasonable accuracy (Table 16.2, Eqs. 16-9 and 16-14; see also Endo et al., 2012 and 2013).

In contrast to structural proteins, globular proteins, such as serum albumins, are spherical colloid type entities that exhibit several distinct hydrophilic as well as hydrophobic domains for "binding" organic pollutants. They can not, therefore, be viewed as a "solvent" into which organic pollutants simply partition. In fact, as compared to muscle proteins, much less accurate pp-LFERs (Table 16.2, Eqs. 16-10 and 16-15) were obtained for *bovine serum albumin (BSA*), an often-used protein surrogate. Furthermore, binding has been found to be highest for polar and smaller apolar compounds ($\log K_{\text{ibsaw}} < 5$), and much weaker for large, apolar chemicals (Endo and Goss, 2011). Finally, for many compounds, specific binding to BSA was up to an order of magnitude (or even more) stronger than partitioning to muscle proteins (Endo et al., 2012). An example of compounds that strongly bind to BSA and other serum albumins are the perfluoroalkyl acids (Bischel et al., 2010 and 2011), not too surprising as one of the main functions of serum albumins is to "carry" fatty acids in body fluids (van der Vusse, 2009). Such binding may also explain the substantial bioaccumulation of

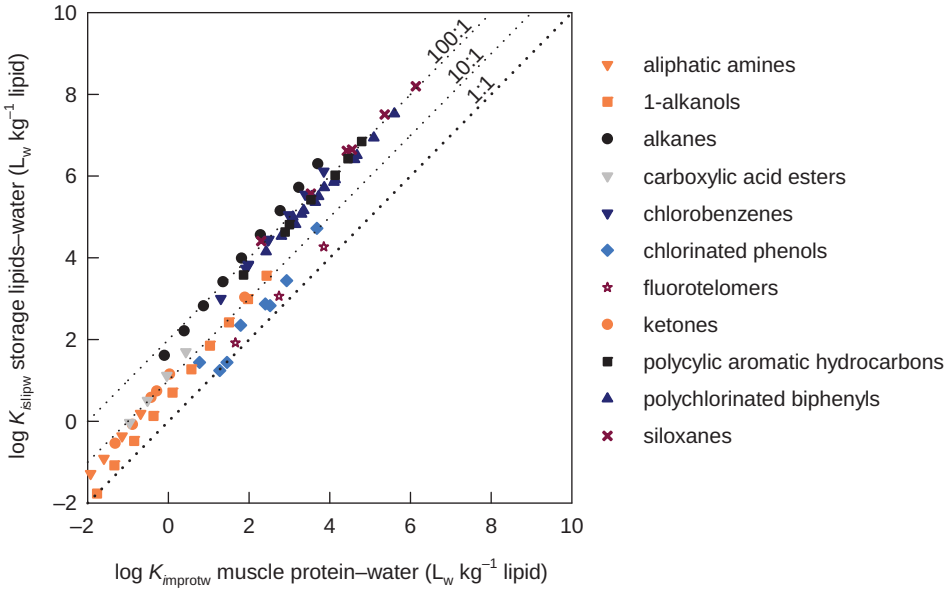


Figure 16.4 Comparison of partitioning to storage lipids and muscle protein by the calculated logK_{islipw} versus log$K_{improtw}$ for our test set at 37°C (Table 16.2, Eqs. 16-12 and 16-14).

perfluoroalkyl acids in the aquatic environment, a phenomenon not predicted by partitioning models using solely storage lipids and muscle proteins as relevant biological phases (see Section 16.2).

Comparison of Partitioning to Storage Lipids and Muscle Proteins. As previously mentioned, traditional models for quantifying bioaccumulation commonly assume lipids to be the major biological phase into which organic pollutants partition. However, in certain organisms, proteins, particularly muscle proteins, may be more than one order of magnitude more abundant than lipids (Table 16.1). Hence, we need to evaluate in which situations partitioning into proteins is significant and should be included in our simple model (Eqs. 16-1 and 16-2). To compare partitioning to storage lipids and muscle proteins, we use the calculated biological phase–water partition coefficients (Table 16.2, Eqs. 16-12 and 16-14) of our test set. As shown in Fig. 16.4, larger apolar and weakly polar compounds particularly tend to partition much more favorably (up to two orders of magnitude) to the more apolar storage lipids as compared to the more polar muscle proteins. For polar compounds, in particular for bipolar compounds, however, partition coefficients are more often in the same order of magnitude. A similar general trend is, of course, observed for partitioning from air.

For further illustration, we can derive a pp-LFER for calculating the difference for individual compounds expressed by a (hypothetical) storage lipid–mussel protein partition coefficient, $K_{islip/mprot}$ (in kg mprot kg^{-1}slip), by subtraction of Eqs. 16-12 and 16-14:

$$\log K_{islip/mprot} = -0.14V_i + 0.26L_i - 1.03S_i - 2.14A_i - 0.98B_i + 1.64 \quad (16\text{-}24)$$

As is evident in Eq. 16-24, the polar terms, in particular the *A*-term, favor the protein phase. Since most monopolar compounds are H-acceptors (*B*-term), the effect is

largest for bipolar compounds. To illustrate, the calculated $K_{i\text{slip/mprot}}$ of chlorobenzene ($V_i = 0.84$, $L_i = 3.66$, $S_i = 0.65$, $A_i = 0$, $B_i = 0.7$) is about 60 kg mprot kg^{-1}slip, whereas the value for 4-chlorophenol ($V_i = 0.90$, $L_i = 4.78$, $S_i = 1.08$, $A_i = 0.67$, $B_i = 0.20$) is 1. For assessing biota–water as well as biota–air partition coefficients (Eqs. 16.1 and 16.2), proteins may, therefore, be neglected for chlorobenzene, but not for 4-chlorophenol.

Polysaccharides. Other abundant biological phases present in many organisms are polysaccharides (psach), which are polymeric carbohydrates such as cellulose (Fig. 16.2), hemicellulose, and starch in plants; glycogen, an animal "starch"; and chitin, present in both plants and animals. Polysaccharides have structural functions (e.g., cellulose, chitin) or serve as energy storage (e.g., starch, glycogen). Compared to lipids and proteins, they are much more polar polymers, which may form crystalline "glassy" phases. Hence, we would expect much higher costs for cavity formation when transferring an organic solute into a polysaccharide, and thus significantly lower partition coefficients, as compared to other biological phases. Hung et al. (2010) determined almost identical partition coefficients, $K_{i\text{psachw}}$, for two polysaccharides (cellulose and starch) for 55 apolar and weakly monopolar compounds, obtaining a sp-LFER from the combined data set at 25°C:

$$\log K_{i\text{psachw}}(L_w kg^{-1}_{\text{sach}}) = 0.74 \log K_{i\text{ow}} - 1.86 \qquad \text{(16-25)}$$

$$\text{(number of chemicals} = 55,\ r^2 = 0.99;\ \log K_{i\text{ow}} \text{ range:1–6.5)}$$

Comparing the corresponding polysaccharide–water and storage lipid–water partition coefficients for apolar and weakly polar compounds shows that $K_{i\text{slipw}}$ is about two (for small apolar molecules) to four (for large molecules) orders of magnitude larger than $K_{i\text{psachw}}$. For example, for our companion compound phenanthrene, a $K_{i\text{psachw}}$ value of 25 L kg^{-1} psach has been determined whereas its $K_{i\text{slipw}}$ is on the order of 100,000 L kg^{-1} slip. Only for small bipolar compounds, such as aniline or phenol, can the differences be expected to be somewhat smaller. Among the very few bipolar compounds investigated, aniline (not included in the regression Eq. 16-25) has a 10 times higher experimental partition coefficient than predicted by Eq. 16-25 (about 1 versus 0.1 L kg^{-1} psach), indicating again the limitations of such simple sp-LFERs. In summary, we may conclude that, in most cases of interest to us, partitioning to polysaccharides is negligible. Also, the thermodynamic cycle is not applicable for estimating partitioning from air since, because of their polar nature, "dry" polysaccharides most likely have quite different sorbent properties as compared to polysaccharides immersed in water.

Lignin. We conclude our discussion of defined biological phases by briefly addressing the biopolymer lignin (lig), which is an important constituent of woody tissues, primarily present in trees and other terrestrial plants. Compared to the lipid-like plant polymers, lignin exhibits a much more aromatic structure and also somewhat more polar groups (Fig. 16.2). It has a "glassy" character, and as a consequence, sorption isotherms tend to be non-linear. From the rather scarce experimental data available for lignin–water partitioning, which has been reported primarily for aromatic hydrocarbons (e.g., alkyl benzenes, Mackay and Gschwend, 2000; and PAHs, Wang et al., 2007 and Wang and Xing, 2007), we may conclude that for these type of compounds,

lignin–water partition constants, $K_{i\text{ligw}}$, are approximately five to ten times smaller as compared to plant lipid–water partition coefficients. For example, for phenanthrene, log $K_{i\text{ligw}}$ values of between 4.0 and 4.2 have been reported (Wang et al., 2007; Wang and Xing, 2007), which is a factor of more than five times smaller as compared to the partition coefficients reported for plant lipids. The same trend holds true for some other PAHs (Wang and Xing, 2007), as well as for toluene and 1,2-xylene (Mackay and Gschwend, 2000). For bipolar compounds, the difference seems to be smaller; for example, for 2,4-dichlorophenol, a difference of a factor of 2 to 3 has been reported (Mackay and Gschwend, 2000; Li et al., 2012). However, no general mechanistic conclusions can be drawn from these data.

Temperature Dependence of K_{ibpw} and K_{ibpa}. Since many $K_{i\text{bpw}}$ and $K_{i\text{bpa}}$ values, particularly for lipids and proteins, are reported for 37°C (body temperature), we need to address the temperature dependence of biological phase–water and, particularly, biological phase–air partitioning. As discussed in Chapter 4 (Eqs. 4-32 to 4-36), for a narrow temperature range (e.g., the ambient temperature range), the temperature dependence of any partition coefficient is given by the familiar van't Hoff relationship. Therefore, for biological phase–water and biological phase–air partitioning, we may write:

$$\ln K_{i\text{bpw}} = -\frac{\Delta_{\text{bpw}}H_i}{R} \cdot \frac{1}{T} + \text{constant} \tag{16-26}$$

and

$$\ln K_{i\text{bpa}} = -\frac{\Delta_{\text{bpa}}H_i}{R} \cdot \frac{1}{T} + \text{constant} \tag{16-27}$$

We also recall from Chapter 4 that, if one of the phases considered is the gas phase and the partition constant is expressed in molar concentrations, due to the temperature dependence of the molar volume of gases, $\Delta_{\text{bpa}}H_i$ has to be replaced by $\Delta_{\text{bpa}}H_i + RT_{\text{av}}$ where T_{av} is the average temperature (in K) of the temperature range considered (Atkinson and Curthoys, 1978). However, because RT_{av} is equal to only about 2.5 kJ mol^{-1}, we will neglect this term, just like we did when discussing air–water partitioning (Eq. 9-25) or organic solvent–air partitioning (Eq. 10-16). Finally, using the thermodynamic cycle, we can relate the enthalpies in Eqs. 16-26 and 16-27 with the enthalpy of water–air exchange, $\Delta_{\text{wa}}H_i$ (see Table 9.2 for some examples; note that the table gives $\Delta_{\text{wa}}H_i = -\Delta_{\text{aw}}H_i$ values) by:

$$\Delta_{\text{bpw}}H_i = \Delta_{\text{bpa}}H_i - \Delta_{\text{wa}}H_i \tag{16-28}$$

Unfortunately, experimental enthalpy data for partitioning of organic pollutants between biological phases and air or water are scarce. From the available data measured primarily for lipid–water partitioning (Endo et al., 2011; Geisler et al., 2012), we may, however, conclude that for liquid biological phase–water partitioning, we expect ΔH_i values that are in the same order of magnitude as found for organic solvent–water partitioning, in particular for the octanol–water system (Endo et al., 2011). We also assume that this holds for liquid biological phase–air partitioning. Thus, if no experimental enthalpy values are available, for a crude approximation (± 10 kJ mol^{-1}) of $\Delta_{\text{bpw}}H_i$ and $\Delta_{\text{bpa}}H_i$, we will use the pp-LFERs introduced for estimating the

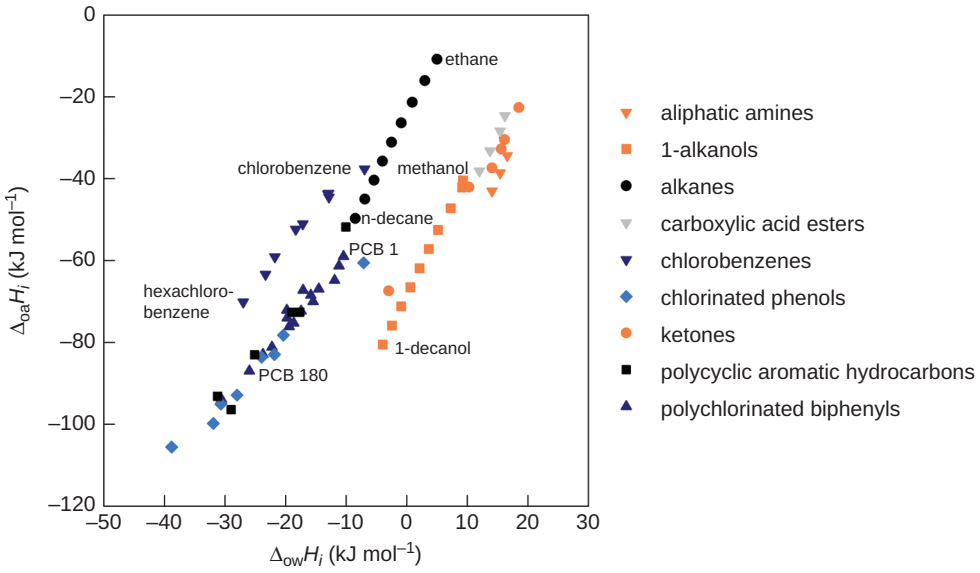


Figure 16.5 Range of calculated enthalpies of organic phase–air ($\Delta_{oa}H_i$; Eq. 10-17) and organic phase–water ($\Delta_{ow}H_i$; Eq. 10-18) partitioning for our test set, using octanol as representative organic phase.

corresponding enthalpies in Chapter 10 for the *octanol–water* (Eq. 10-18) and *octanol–air* (Eq. 10-17) system:

$$\Delta_{ow}H_i(\text{kJ mol}^{-1}) = +18.9V_i - 8.3L_i + 5.3S_i - 20.1A_i + 34.3B_i + 1.7 \quad (10\text{-}18)$$

$$\Delta_{oa}H_i(\text{kJ mol}^{-1}) = +1.6V_i - 9.7L_i + 6.0S_i - 53.7A_i - 9.2B_i - 6.7 \quad (10\text{-}17)$$

As we discussed in detail in Chapter 10 and as is illustrated by Fig. 16.5 for the octanol–water and octanol–air systems, the enthalpies of partitioning of organic pollutants in organic phase–water systems are commonly rather small and are in the range of –40 kJ mol^{-1} for large aromatic compounds to +20 kJ mol^{-1} for small aliphatic polar compounds. In contrast, and similarly to the enthalpies of condensation (see Table 10.3), for partitioning involving the gas phase, ΔH_i values may be significantly larger, ranging from –10 kJ mol^{-1} for small aliphatic compounds up to more than –110 kJ mol^{-1} for large apolar and polar aromatic compounds (Fig. 16.5). We recall that a negative ΔH_i means a decrease of the partition coefficient with increasing temperature. A ΔH_i value of 50 kJ mol^{-1} means a factor of two change in the partition coefficient per 10 degree change in temperature and a factor of 4 if ΔH_i is 100 kJ mol^{-1}.

16.3 Bioaccumulation and Biomagnification in Aquatic Systems

Parameters Used to Describe Experimental Bioaccumulation and Biomagnification Data

In the aquatic environment, organic pollutants from water and sediments can transfer to organisms. Bioaccumulation is thus the sum of differing processes causing the accumulation and therein amplification of these contaminants in an organism. Various parameters, represented in Fig. 16.6, are defined to relate the *actual* concentration determined for a given compound in an organism or biological phase to the

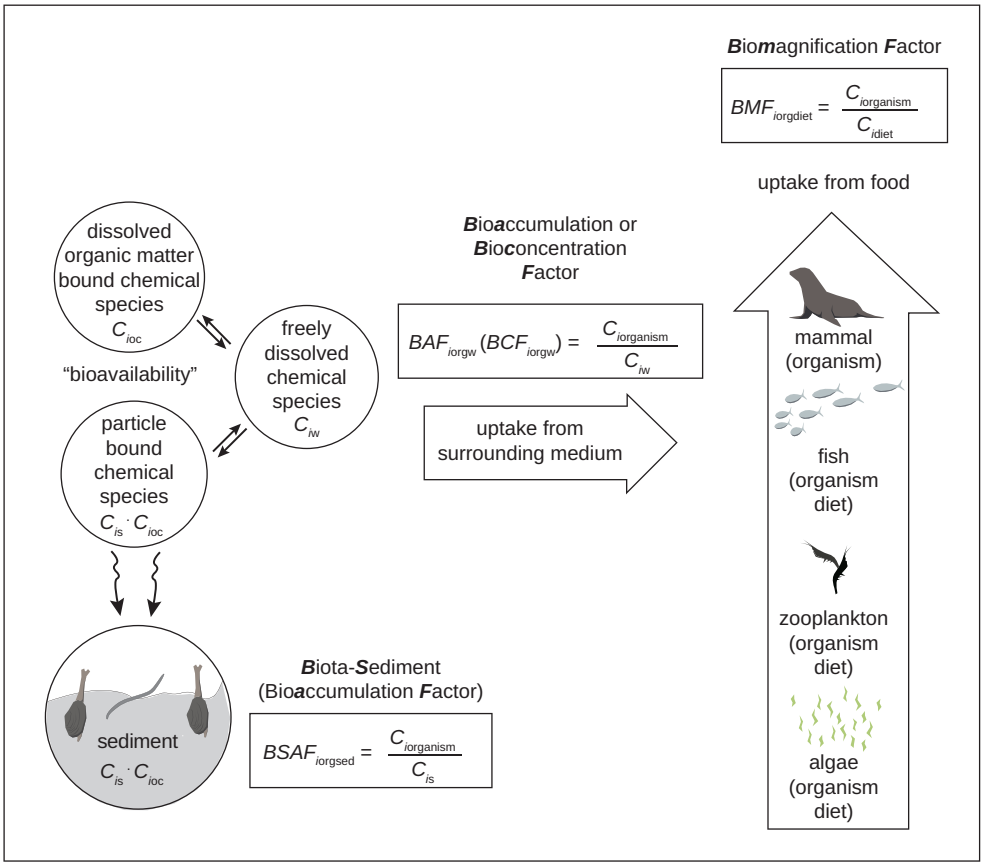


Figure 16.6 Terms and parameters frequently used to describe accumulation of chemicals in organisms in an aquatic environment. The term bioaccumulation describes the total accumulation by all possible routes (e.g., passive uptake, intake by food, and digestion). Similar terms can be derived for a terrestrial system by exchanging the main environmental media, water and sediment, with air and soil.

compound's concentration in the medium (i.e., water, diet, sediment) from which the compound is thought to be primarily transferred. Similar parameters can be defined if the medium in which the organism lives is a different one, such as for air (see Section 16.4).

Of course, a given organic compound can be accumulated in an organism via various simultaneous routes. For example, animals living at the sediment–water interface can experience passive uptake of dissolved compound from water (bioconcentration) as well as uptake of the sorbed molecules on sediment particles (bioaccumulation) or present in their diet (biomagnification). Thus, to describe the net result of all uptake and elimination processes occurring, one simplifies and defines a bioaccumulation factor in the aquatic environment, $BAF_{i\mathrm{orgw}}$, which relates the *actual* concentration in the organism (org) to the *actual* concentration in one medium of the environment in which the organism primarily lives, in this case, water (w):

$$BAF_{i\mathrm{orgw}} = \frac{C_{i\mathrm{org}}}{C_{i\mathrm{w}}} \tag{16-29}$$

In aquatic systems, $C_{i\mathrm{w}}$ denotes the truly dissolved concentration (e.g., not including the fraction sorbed to particles or DOM). Similarly, in the terrestrial environment, this term is the truly gaseous concentration (i.e., $C_{i\mathrm{a}}$, not including any fraction in

aerosols, see Section 16.4). However, total filter-passing concentrations are often used in the literature, values that may also contain compound molecules that are associated with fine particles or colloids (see Chapter 13). This is particularly a problem for very hydrophobic compounds that have a strong tendency to associate with particles, since some of these colloids can pass through filters. Failure to use the right value of $C_{i\mathrm{w}}$, thus, may cause the experimentally determined $BAF_{i\mathrm{orgw}}$ to be erroneously low.

Sometimes, a bioconcentration factor ($BCF_{i\mathrm{orgw}}$) is also defined, which only reflects the passive uptake of a dissolved compound from water:

$$BCF_{i\mathrm{orgw}} = \frac{C_{i\mathrm{org}}(\text{only passive uptake})}{C_{i\mathrm{w}}} \tag{16-30}$$

This factor can only be measured under controlled conditions in which uptake from particles or diet is excluded. For typical $BAF_{i\mathrm{orgw}}$ and $BCF_{i\mathrm{orgw}}$ values for organic pollutants, see Arnot and Gobas (2006).

For organisms living at the sediment–water interface, another parameter, the biota–sediment bioaccumulation factor, $BSAF_{i\mathrm{orgsed}}$, is sometimes used:

$$BSAF_{i\mathrm{orgsed}} = \frac{C_{i\mathrm{org}}}{C_{i\mathrm{sed}}} \tag{16-31}$$

which is, of course, somewhat arbitrary since these organisms live both in the sediment and in the water column, and these two compartments are usually not in equilibrium with each other. For hydrophobic compounds that partition primarily into storage lipids and for which sediment organic matter (SOM) is the dominant sorbent (Chapter 13), we can also define a storage lipid–SOM normalized experimental bioaccumulation factor, $BSAF_{i\mathrm{slipoc}}$:

$$BSAF_{i\mathrm{slipoc}} = \frac{C_{i\mathrm{slip}}}{C_{i\mathrm{oc}}} \tag{16-32}$$

To parameterize the phenomenon of the concentration of a given compound in organisms increasing as one examines successive organisms along a given food chain, one defines the biomagnification factor, BMF_i:

$$BMF_{i\mathrm{orgdiet}} = \frac{C_{i\mathrm{org}}}{C_{i\mathrm{diet}}} \tag{16-33}$$

which is the ratio of the chemical concentration in the organism of a higher trophic level divided by the same chemical's concentration in the organism contributing a major part of the diet. Equation 16-33 can also be written in terms of the observed bioaccumulation factors (Eq. 16-29) of the organism considered and of its diet,

if both have been referenced to the same environmental medium, in this case, water:

$$BMF_{i\text{orgdiet}} = \frac{BAF_{i\text{orgw}}}{BAF_{i\text{dietw}}} \tag{16-34}$$

In the following discussion, we use several of these parameters to evaluate experimental data by comparing them to the corresponding *theoretical accumulation potential*, TBP_i , the predicted concentration in an organism at equilibrium with the surrounding environmental media. By doing so, we are able to assess whether equilibrium has been established between a given organism and its surroundings, and if not, how far from equilibrium we are in a given situation. In this context, we should recall that due to the uncertainties in parameter estimation, any predicted bioaccumulation coefficients, $K_{i\text{orgw}}$ or $K_{i\text{orga}}$ (Eqs. 16-1 and 16.2), should be considered good estimates if they are within a factor of 2 to 3 of the measured bioaccumulation factors.

Bioaccumulation as a Dynamic Process

In the real world, equilibrium partitioning between an organism and its surroundings may not be achieved, which is especially true for compounds that are metabolized by the organism. Metabolism can actually change the *BAF* by orders of magnitude (see Nichols et al., 2013). Also, slow chemical exchanges between the tissues of organisms with the media in which they live can lead to a lack of equilibrium. Therefore, the observed $BAF_{i\text{orgw}}$ value (Eq. 16-29) may differ significantly from the theoretical equilibrium expectation, $K_{i\text{orgw}}$ (Eq. 16-1). In fact, because the accumulation of a given chemical may depend on several different processes occurring at the same time (see Fig. 16.7), $BAF_{i\text{orgw}}$ may change continuously with time for a given organism. Hence, in a given natural system (e.g., a lake), even two individuals of the same species (e.g., two trouts) may exhibit different $BAF_{i\text{orgw}}$ (e.g., $BAF_{i\text{fishw}}$) values.

In order to estimate the $C_{i\text{org}}$ over to time, we now introduce a simple conceptual model that considers simultaneous inputs and outputs of a compound in an organism: the

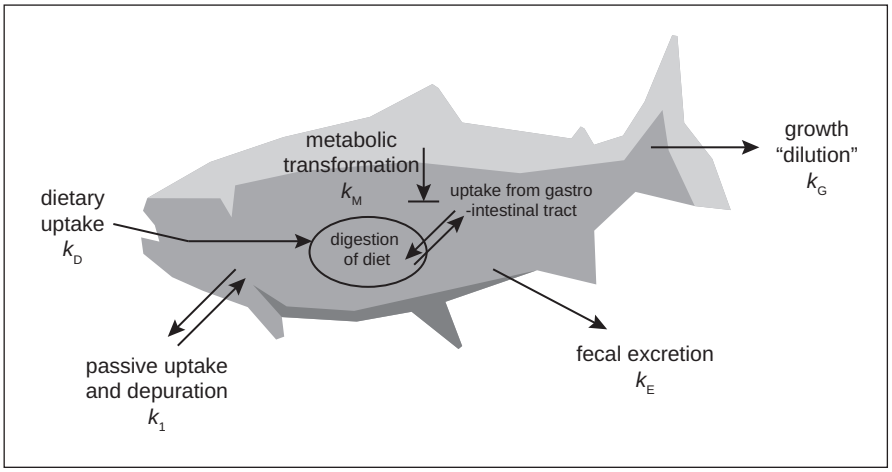


Figure 16.7 Illustration of some processes determining bioaccumulation of a chemical in a fish. The various k values are formulated as first-order rate constants for description of the kinetics dependent on the physiology and behavior of the fish (for a detailed discussion of bioaccumulation models, see e.g., Gobas and Morrison, 2000; Arnot and Gobas, 2004; Xiao et al., 2015).

one-box model approach (see Chapter 6 for a general discussion of one-box models). In this approach, we assume that the organism (i.e., the fish) is a well-mixed reactor (of course, it is not), and we define all processes as first-order reactions. The temporal change in concentration of a given compound *i* in the fish, $C_{i\text{fish}}$ can then be described simply by:

$$\frac{dC_{i\text{fish}}}{dt} = \underbrace{k_1(K_{i\text{fishw}}C_{i\text{w}})}_{\text{gill exchange}} + \underbrace{k_D(K_{i\text{fish-diet}}C_{\text{diet}})}_{\text{dietary uptake}} - \underbrace{k_E(K_{i\text{fish-excreta}}C_{i\text{excreta}})}_{\text{excretion}} - \underbrace{k_{\text{M}}C_{i\text{fish}}}_{\text{metabolism}} - \underbrace{k_{\text{G}}C_{i\text{fish}}}_{\text{growth}} \quad (16\text{-}35)$$

where k_1, k_{D}, k_{E}, k_{M}, and k_{G} are the first-order rate constants (T^{-1}) for the various processes indicated in Fig. 16.7. $K_{i\text{fishw}}$, $K_{i\text{fish-diet}}$, and $K_{i\text{fish-excreta}}$ are the *equilibrium* coefficients (hence we use *K*) for partitioning of chemical *i* between the various combinations of fish, water, diet, and excreta. This equation indicates that the chemical's concentration in the fish can evolve over time as the various mechanisms act simultaneously. The time-evolving solution of this expression is given in Chapter 6, Box 6.2.

For now, we consider one special situation called the *steady-state* case. In this case, the concentration in the organism (i.e., in the fish) does not change with time (i.e., $dC_{i\text{fish}}/dt = 0$). Steady state occurs if the total rate of chemical elimination equals the total rate of its uptake. By setting $dC_{i\text{fish}}/dt$ equal to zero in Eq. 16-35, we can solve for the steady-state concentration in the fish (indicated by the superscript ∞):

$$C^{\infty}_{i\text{fish}} = \frac{k_1K_{i\text{fishw}} + k_{\text{D}}K_{i\text{fish-diet}}C_{i\text{diet}} - k_{\text{E}}K_{i\text{fish-excreta}}C_{i\text{excreta}}}{k_1 + k_{\text{M}} + k_{\text{G}}} \quad (16\text{-}36)$$

or dividing this result by the water concentration, $C_{i\text{w}}$, we have:

$$BAF^{\infty}_{i\text{fishw}} = \frac{C^{\infty}_{i\text{fish}}}{C_{i\text{w}}} = \frac{k_1K_{i\text{fishw}} + k_{\text{D}}K_{i\text{fish-diet}}BAF_{i\text{diet}} - k_{\text{E}}K_{i\text{fish-excreta}}K_{i\text{excretaw}}}{k_1 + k_{\text{M}} + k_{\text{G}}} \quad (16\text{-}37)$$

where $BAF_{i\text{diet}} = C_{i\text{diet}}/C_{i\text{w}}$ and $K_{i\text{excretaw}} = C_{i\text{excreta}}/C_{i\text{w}}$. In this model, we assume a constant aqueous concentration ($C^{\infty}_{i\text{w}} = C_{i\text{w}}$), and, when using Eqs. 16-36 or 16-37, we also assume that all parameters, including $BAF_{i\text{diet}}$, are constant. These assumptions are, however, not generally true. For example, the lipid content of a fish can vary widely throughout its lifetime (e.g., Henderson and Tocher, 1987), thus the value of $K_{i\text{fishw}}$ would vary with time. Likewise, the specific growth rate of organisms does not remain constant over their entire lifespan, so the "dilution" of the chemical does not continuously occur at the same rate.

Depending on the rates of the various exchange and transformation processes, equilibrium conditions may not be achieved under real-world conditions, and this simple

mathematical model can help us to qualitatively understand the reasons for such disequilibrium. For example, if uptake and elimination is dominated by exchange at the gills, the $BAF^{\infty}_{i\text{fishw}}$, which, here, would also be referred to as the $BCF^{\infty}_{i\text{fishw}}$ (Eq. 16-30), would correspond to:

$$BAF^{\infty}_{i\text{fishw}} = \frac{k_1 K_{i\text{fishw}}}{k_1} = K_{i\text{fishw}} = \frac{C^{\infty}_{i\text{fishw}}}{C_{i\text{w}}} \tag{16-38}$$

The result is the equilibrium coefficient, $K_{i\text{fishw}}$, and may correspond to the predicted equilibrium value (Eq. 16-1). In contrast, if uptake is via exchange at the gills but losses occur primarily by metabolism ($k_{\text{M}} > k_1$), the $BAF^{\infty}_{i\text{fishw}}$ would be smaller than $K_{i\text{fishw}}$ by the factor k_1/k_{M}.

Evaluating Bioaccumulation Disequilibrium

Let us think back to that new fishing site where you discovered signs warning you not to eat certain fish as they may contain harmful organic chemicals. At such a polluted site, the concentration in the fish may not be in equilibrium with the surrounding water. Therefore, predicting the extent of bioaccumulation is an important step to evaluate the potential hazard to the fish and to humans.

Example: Disequilibrium in Organisms Living at the Sediment–Water Interface. In many cases, a likely cause of any disequilibrium is the exposure of an organism to more than one environmental medium. For example, organisms living at the sediment–water interface (e.g., clams, polychaetes, amphipods, insect larvae) are exposed to the water column as well as sediment beds, which often contain much higher levels of hydrophobic compound contamination than the overlying water. Hence, organisms may take up contaminants from sediments and simultaneously release them to the water column (Fig. 16.8). In order to eat, such organisms often ingest particles from the sediment bed, incurring high exposures. In order to "breath," these organisms must also pump water containing oxygen in and out, thus coming into intense contact with the less contaminated overlying water. Consequently, we may hypothesize that in such organisms, concentrations of persistent chemicals will be between those concentrations anticipated if the organism was in equilibrium with either the water column or the sediment.

Let us check this hypothesis by inspecting some field data of the accumulation of organic pollutants by benthic organisms. We first assume that the compounds are present primarily in the storage lipid phases of the animals, and we solve for equilibrium lipid-normalized concentrations. Such an assumption is generally made when considering animals with significant lipid levels (>1% by dry weight) and compounds whose affinities for lipids are much greater than their affinities for proteins (see Fig. 16.4). We also assume that in the sediment bed, the pollutants are chiefly absorbed into the natural organic matter of those solids, and we use organic carbon normalized concentrations. Finally, by focusing on persistent compounds, we are probably justified in neglecting any effect of metabolism on the observed animal concentrations.

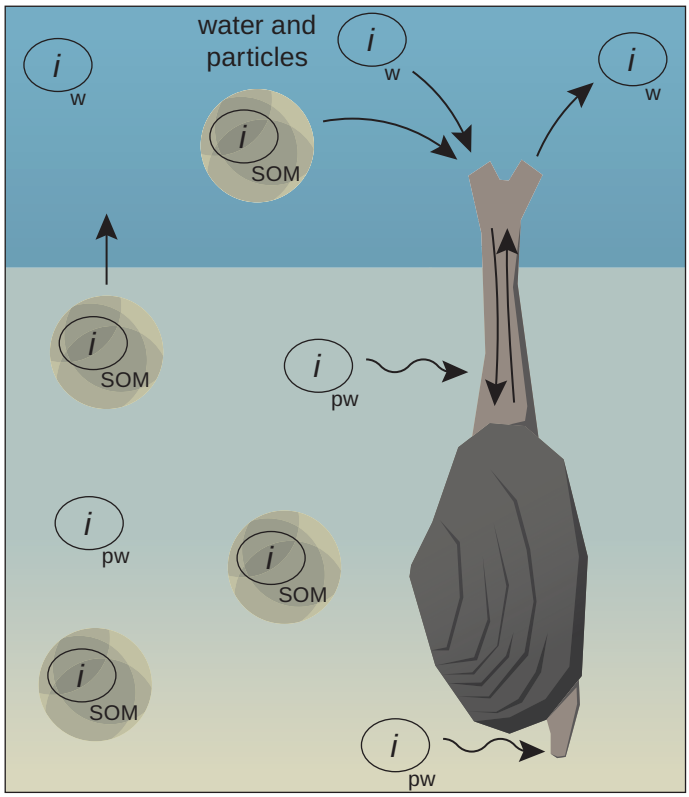


Figure 16.8 Schematic illustration of uptake and depuration of chemicals by a benthic organism, a clam dwelling near the sediment–water interface. i_w is the truly dissolved compound in the water column, i_{SOM} is the compound associated with the sediment organic matter, and i_{pw} is the compound in the sediment porewater.

PCBs are persistent compounds that likely fulfill all these assumptions. As an example, we use our companion compound PCB 153 ($V_i = 2.06$, $L_i = 9.59$, $S_i = 1.74$, $A_i = 0$, $B_i = 0.11$) for which experimental data for four different sediment-dwelling organisms (a caddishfly larvae, an amphipod, a zebra mussel, and a crayfish) are available (Morrison et al., 1996; Table 16.3).

In order to evaluate to what extent equilibrium partitioning was established between the different compartments (organism, sediment, and overlying water column) at this field site, we compare the experimental concentration ratios ($BAF_{i\text{lipw}}$, $BSAF_{i\text{lipoc}}$) with predicted values using the pp-LFERs derived for $K_{i\text{ocw}}$ (Pahokee Peat, Eq. 13-14, Table 13.3 for 25°C) and $K_{i\text{slipw}}$ (Eq. 16-12, Table 16.2 for 37°C), adjusted for temperature. By dividing the two equilibrium coefficients, we can then calculate the equilibrium partition coefficient between storage lipid and SOM, $K_{i\text{slipoc}}$:

$$K_{i\text{slipoc}} = \frac{K_{i\text{slipw}}}{K_{i\text{ocw}}} \tag{16-39}$$

Since the temperature at the field site was most probably <10°C (not indicated by the authors), a temperature of 5°C is assumed. Table 16.3 summarizes the calculated partition coefficients at 5°C as well as the experimental concentration ratios for PCB 153. We note that for PCB 153, a ΔH_i value of –25 kJ mol^{-1} has been assumed

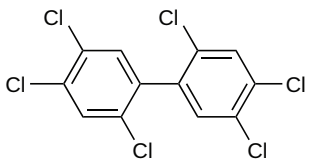
Table 16.3 Measured Average Concentrations and Calculated Partitioning Coefficients of PCB 153 in Four Organisms Living at the Sediment–Water Column Interface

Concentration in the water column, C_{iw} (μg L^{-1})[a]	6×10^{-6}
Concentration in the sediment, C_{ioc} (μg kg^{-1}oc) [a]	78
Estimated K_{iocw} (5°C, L kg^{-1} oc)[b]	7.8×10^{5}
Estimated K_{islipw} (5°C, L kg^{-1} lipid)[c]	2.3×10^{7}
Estimated $K_{islipoc}$ (kg oc kg^{-1} lipid)[d]	29

Organism	Concentration[a] (μg kg^{-1} lipid)	BAF_{ilipw} (L kg^{-1} lipid) BAF_{ilipw}/K_{islipw}	$BSAF_{ilipoc}$ (kg oc kg^{-1} lipid) $BSAF_{ilipoc}/K_{islipoc}$
Caddysfly larvae	612	1.0×10^{8}	7.8
		4.3	*0.27*
Amphipod (gammarus)	405	6.8×10^{7}	5.1
		3.0	*0.18*
Zebra mussel	544	9.0×10^{7}	6.9
		3.9	*0.24*
Crayfish	365	6.1×10^{7}	4.6
		2.7	*0.16*

[a]Concentrations measured in water, sediment, and organisms from Morrison et al. (1996). Samples were collected in Lake Erie in July/August 1993 and 1994.
[b]Calculated values using Eq. 13-14, Table 13.3 for Pahokee Peat (25°C) and adjusted for temperature (5°C) using Eq. 16-26.
[c]Calculated values using Eq. 16-12, Table 16.2 for storage lipids (37°C) and adjusted for temperature (5°C) using Eq. 16-26.
[d]Ratio calculated using Eq. 16-39.

(Eq. 10-17) for the temperature dependence of both K_{iocw} and K_{islipw}, which means that for a 10°C decrease in temperature both partition coefficients increase by a factor 1.4. This also means that $K_{islipoc}$ is assumed temperature independent. From the data in Table 16.3, the following conclusions can be drawn for PCB 153 partitioning at this particular field site: (1) by comparing the experimental sediment–water concentration ratio, C_{ioc}/C_{iw}, with the estimated K_{iocw}, we see the overlying water was by a factor of almost 20 undersaturated with respect to equilibrium with the sediments; (2) similar BAF_{ilipw} and $BSFA_{ilipoc}$ values (within less than a factor of 2) were found for the four benthic organisms; and (3) the BAF_{ilipw} values were 3 to 4 times higher than the estimated K_{islipw} values, and 3 to 5 times smaller then the estimated $K_{islipoc.}$. Therefore, as we hypothesized, PCB 153 in the lipids of the four organisms was neither in equilibrium with the SOM nor with the dissolved PCB 153 in the water column, but at a concentration somewhere in between.



PCB 153

Using Fugacities for Evaluation of Bioaccumulation Disequilibrium. With the example of the sediment–water interface, we evaluated bioaccumulation disequilibrium using a comparison of observed (lipid normalized) bioaccumulation factors (BAF_{ilipw} and $BSAF_{ilipoc}$) with expectations from phase equilibrium considerations (K_{islipw} and $K_{islipoc}$). Using the K_i values, we can also calculate the concentrations of a given compound expected in an organism if it were in equilibrium with either the water or

the sediment, and then compare these concentrations with the actual measured ones. Using this approach, we have, however, assumed an identical temperature in all media considered, which is not necessarily the case.

Another way of evaluating disequilibrium, particularly when considering the partitioning of a given compound between several environmental compartments exhibiting different temperatures, is to compare the *chemical activities* (a_i) or *fugacities* (f_i) of the compound in the various compartments. In Chapter 4, we highlighted that it is the difference in activity or fugacity, and not in concentration, that determines in which direction a net flux of compound will occur. Such transfers will continue until the activities or fugacities are equal in the interacting phases. We now use fugacities to assess the degree of disequilibrium, and thus the direction of fluxes in multicompartment systems. Fugacities and activities are, of course, closely related, that is, $a_i = f_i/f_{\text{ref}}$ (see Chapter 4, Section 4.2).

The fugacity of a given compound in a given molecular environment (e.g., in a condensed phase) is a measure of the fleeing tendency of the compound from that environment (see Fig. 4.2). A quantitative measure of fugacity is the partial pressure, p_i, which the compound would exert in the gas phase, if the gas phase were in equilibrium with the phase under consideration and behaved like an ideal gas. Hence, for calculating the fugacity, $f_{i\text{phase}}$, of a compound in a given condensed phase, we need to know the compound's gas (a)–condensed phase (e.g., water (w), SOM (oc), biological phase (bp)) partition coefficient, $K^p_{i\text{aphase}}$, where we use a superscript "p" to indicate that we express $K^p_{i\text{aphase}}$ as the equilibrium ratio of the *partial pressure* of the compound and its *molar concentration* in the phase considered (recall that for air–water partitioning we used $K_{i\text{H}}$ (Eq. 9-14):

$$K^p_{i\text{aphase}} = \frac{p_i}{C_{i\text{phase}}} = K_{i\text{aphase}}/RT \tag{16-40}$$

where the units of $K^p_{i\text{aphase}}$ are, for example, Pa (mol $L^{-1})^{-1}$ or Pa (mol $kg^{-1})^{-1}$. In the commonly used fugacity models, $K^p_{i\text{aphase}}$ is referred to as the *fugacity capacity* of the compound in the given phase (Mackay, 1979 and 2004), and, for organic phases, $K^p_{i\text{aphase}}$ and $K_{i\text{aphase}}$ are the reciprocal $K^p_{i\text{phasea}}$ and $K_{i\text{phasea}}$ values, respectively, which we usually use. The fugacity of the compound in a given phase at a given temperature can then be expressed by:

$$f_{i\text{phase}} \cong K^p_{i\text{aphase}} C_{i\text{phase}} = (K_{i\text{aphase}}/RT) C_{i\text{phase}} \tag{16-41}$$

When calculating the fugacities of a given compound in a given phase, one faces a few difficulties. First, except for air–water partitioning (Chapter 9), few experimental data are available for partitioning of organic compounds between the gas phase (i.e., air) and important environmental phases including biological media. However, as discussed earlier, applying the thermodynamic cycle, values can often be estimated

from the corresponding condensed phase–water partition coefficient and the air–water partition constant of the compound:

$$K^{p}_{i\text{aphase}} = \frac{K_{i\text{aw}}RT}{K_{i\text{phasew}}} \tag{16-42}$$

Second, we recall that, in contrast to partitioning between water and natural condensed phases, partitioning involving the gas phase is strongly temperature dependent (see Fig. 16.5). Consequently, when using fugacities to assess compound fluxes between environmental compartments, one has to be careful to apply the appropriate $K^{p}_{i\text{aphase}}(T)$ values. Finally, for calculating fugacities of compounds in whole organisms for which several biological phases are relevant (e.g., lipids and proteins), we simply have to substitute "phase" by "org" in Eqs. 16-40 to 16-42 and use Eqs. 16.1 to 16.5 to get the appropriate partition coefficients by assuming the same temperature dependence for the partition coefficients involving different biological phases (e.g., lipid–air and protein–air partition coefficients).

Let us now come back to our example of PCB 153 bioaccumulation in sediment-dwelling organisms (Table 16.3). As an example, we compare the fugacities of PCB 153 in the water column, in the bed sediment organic matter, and in the lipids of the zebra mussel. For simplicity, we assume a temperature of 5°C in all parts of the system. We also assume ΔH_i values of –25 kJ mol^{-1} (Eq. 10-15) and +70 kJ mol^{-1} (Table 9.2) for PCB 153 for assessing the temperature dependence of organic phase–water and air–water partitioning, respectively, resulting in a ΔH_i value of +95 kJ mol^{-1} for air–SOM and air–lipid partitioning.

Table 16.4 gives the calculated fugacities of PCB 153 in the water column, the sediment organic matter, and in the zebra mussel lipids. Of course, these fugacities lead to the same conclusions as already found by comparing the accumulation factors and related partition coefficients given in Table 16.3, but they provide a somewhat more transparent picture of the situation. A possible interpretation of the results

Table 16.4 Calculated Fugacities of PCB 153 in the Water Column, the Surface Sediments (Organic Carbon Normalized), and in Zebra Mussels (Lipid Normalized) at a Sediment–Water Column Interface (see also Table 16.3)

Phase	Concentration[a] $C_{i\text{phase}}$ (mol kg^{-1} phase)	$K^{p}_{i\text{aphase}}$(5°C)[b] (Pa (mol kg^{-1} phase)$^{-1}$)	f(5°C)[c] (nPa)
Water	1.7×10^{-14}	5.1×10^{3}	0.087
Sediment oc	2.2×10^{-7}	7.0×10^{-3}	1.5
Zebra mussel lipids	1.5×10^{-6}	2.2×10^{-4}	0.33

[a]Concentration data from Morrison et al. (1996). 1 µg PCB 153 = 2.77×10^{-9} mol; 1 L water ≈ 1 kg water.
[b]Calculated using Eq. 16-42.
[c]Calculated using Eq. 16-41.

summarized in Tables 16.3 and 16.4 is that the sediment-dwelling organisms acquire some PCB 153 from their diet, which most likely includes resuspended sediments, and they release these contaminants back to the "cleaner" water.

Biomagnification in Aquatic Food Webs

As a consequence of biomagnification, persistent compounds may be present at significantly higher concentrations in certain tissues of higher organisms (e.g., in lipid or protein phases) than one would predict by using a simple partitioning model between this tissue and the media surrounding the organism (e.g., water, air). Biomagnification factors (BMF_i; Eq. 16-33) significantly greater than one may reflect real processes building up the effective concentrations in a given organism's tissues. More accurately, the chemical activity or fugacity of the compound may prove to be greater as one moves up the food chain. Hence we may speak of "real" biomagnification when:

$$f_{i\text{org}} > f_{i\text{diet}} \quad \text{or, equivalently} \quad a_{i\text{orga}} > a_{i\text{diet}} \tag{16-43}$$

Let us now identify some situations where "real" biomagnification may occur. First, in many organisms, the organic mass making up the diet is substantially degraded in the animal gut. For example, herbivorous fish typically excrete about 30 to 40% of their ingested food while carnivorous fish excrete about 20% (Brett and Groves, 1979). Moreover, particular biochemical fractions of the diet may be especially utilized; for example, Gobas et al. (1999) observed that the gut contents of a rock bass contained only about one-third of the lipid content of the predominant prey, a crayfish species. Therefore, biomagnification would be enhanced in the herbivore or carnivore's gastrointestinal tract, since it can be assumed that large parts of the lipids and proteins present in the diet are degraded there, but not a biochemically recalcitrant organic pollutant. This digestion of the sorbent leads, of course, to an increase in fugacity of the compound in the gastrointestinal tract of an organism as compared to the original diet. Because elimination by passive depuration through the gills or lungs is slow in higher organisms, concentration levels well above predicted equilibrium values may thus be established in such organisms for long time periods (i.e., even for the lifetime of the organism). Thus, various compound- and organism-specific factors exist that determine whether "real" biomagnification occurs in a given food chain or food web. Finally, we mention that the term "*bioamplification*" is used for cases in which a higher fugacity of the compound in an organism is due to the loss of those biological phases (e.g., lipids, proteins) in which the compound is primarily present, which also may lead to a redistribution of the chemical within the organism (see Daley et al., 2014). However, it is often not clear which processes are responsible for the resulting disequilibrium situation in a given organism, so we do not distinguish between the different processes and just use the parameters defined in Fig. 16.6.

Values of the defined biomagnification factor (BMF_i, Eq. 16-33) may, however, also be significantly smaller than one. Such is the case for compounds that are more efficiently metabolized in the organism compared to the diet; that is, $BAF_{\text{orgw}} < BAF_{i\text{dietw}}$. In fact, significant metabolism in organisms at a higher trophic level has been suggested for a series of polyhalogenated compounds, including specific PCB (Bodin et al., 2008; Cullon et al., 2012) and PBDE congeners (Stapleton et al., 2004; Shaw et al., 2009;

Tomy et al., 2009; Borga et al., 2012). Furthermore, if the compound concentrations are expressed on a *total organism weight basis*, a $BMF_i \neq 1$ does not necessarily mean that the concentration of the compound in a particularly important location in the organism (e.g., in the membrane lipids where a toxic effect may occur, see Section 16.5) is different in the organism and its diet. A $BMF_i \neq 1$ may simply reflect differences in composition of the organism and its diet. For example, if, compared to its diet, an organism exhibits a much higher fraction of organic phases that are favorable for the compound to partition into (in particular, lipids), a BMF_i value > 1 is likely to be found (see e.g., LeBlanc, 1995; Kucklick and Baker, 1998). On the other hand, a higher lipid content of the diet tends to decrease the whole body weight–normalized BMF_i.

Biomagnification of POPs in Aquatic Food Chains. Let us now examine some classical examples of persistent organic pollutants (POPs), particularly apolar organochlorine compounds, in organisms forming simple aquatic food chains (Fig. 16.9). As discussed earlier, these apolar compounds tend to accumulate predominantly in the lipids of organisms. Hence, we normalize all the observed concentrations by the lipid contents of these organisms, yielding values of $C_{i\text{lip}}$ (mg kg^{-1} lip) for each organism or organism tissue. To the extent that this dominance of partitioning in lipids is correct, at a given temperature, the resultant values are then linearly proportional to the individual compound's fugacities (or activities) as expressed by Eq. 16-41; phase=lip).

As is evident in planktonic food webs (e.g., Fig. 16.9*,* Harding et al., 1997; Paterson et al., 1998), rather persistent hydrophobic compounds such as our companion compound PCB 153 as well as some other PCB congeners do not tend to show large biomagnification. The ranges of $C_{i\text{lip}}$ values observed at one trophic level often overlap the ranges of observations made at higher levels. For example, Fig. 16.9*a* shows plankton samples with lipid-normalized concentrations of PCB 153 ranging from 25 to 170 mg kg^{-1} lip, while the juvenile fish at a higher trophic level exhibited 90 to 500 mg kg^{-1} lip. Using mean values, $C_{i\text{lip}}$ values of the zooplankton are indistinguishable from the phytoplankton they eat, while the fish are only a factor of 3 "magnified." Apparently, in smaller organisms like plankton, depuration and excretion is fast enough that equilibrium may be established, even for highly hydrophobic compounds.

In higher organisms, however, disequilibrium with the environment may occur. Thus, if an organism's diet serves as the main source of a contaminant, and in digesting the diet, the compound's fugacity in the gastrointestinal (GI) tract is increased up to a factor of 10, then the larger animal will exhibit a body fugacity between that of its GI tract and the environmental medium in which it respires. This disequilibrium and thus the $BMF_{i\text{orgdiet}}$ value tend to increase with increasing hydrophobicity of the compound (e.g., Walters et al., 2011; see Fig. 16.9*b* and *c*). This phenomenon is probably mostly due to slower depuration processes. Thus, for example, when considering biomagnification of PCBs, one usually observes a change in the mixture's composition, that is, a shift toward higher chlorinated congeners along a given food chain (e.g., Russel et al., 1995; Feldman and Titus, 2001; Jude et al., 2010). In contrast, faster depuration may explain the diminished biomagnification of the much less hydrophobic HCHs (log $K_{i\text{ow}} \sim 3.8$) as compared to the other more hydrophobic organochlorine compounds. In addition, the $BMF_{i\text{orgdiet}}$ value of 0.3 for ΣHCH observed for water birds (Fig. 16.9*c*)

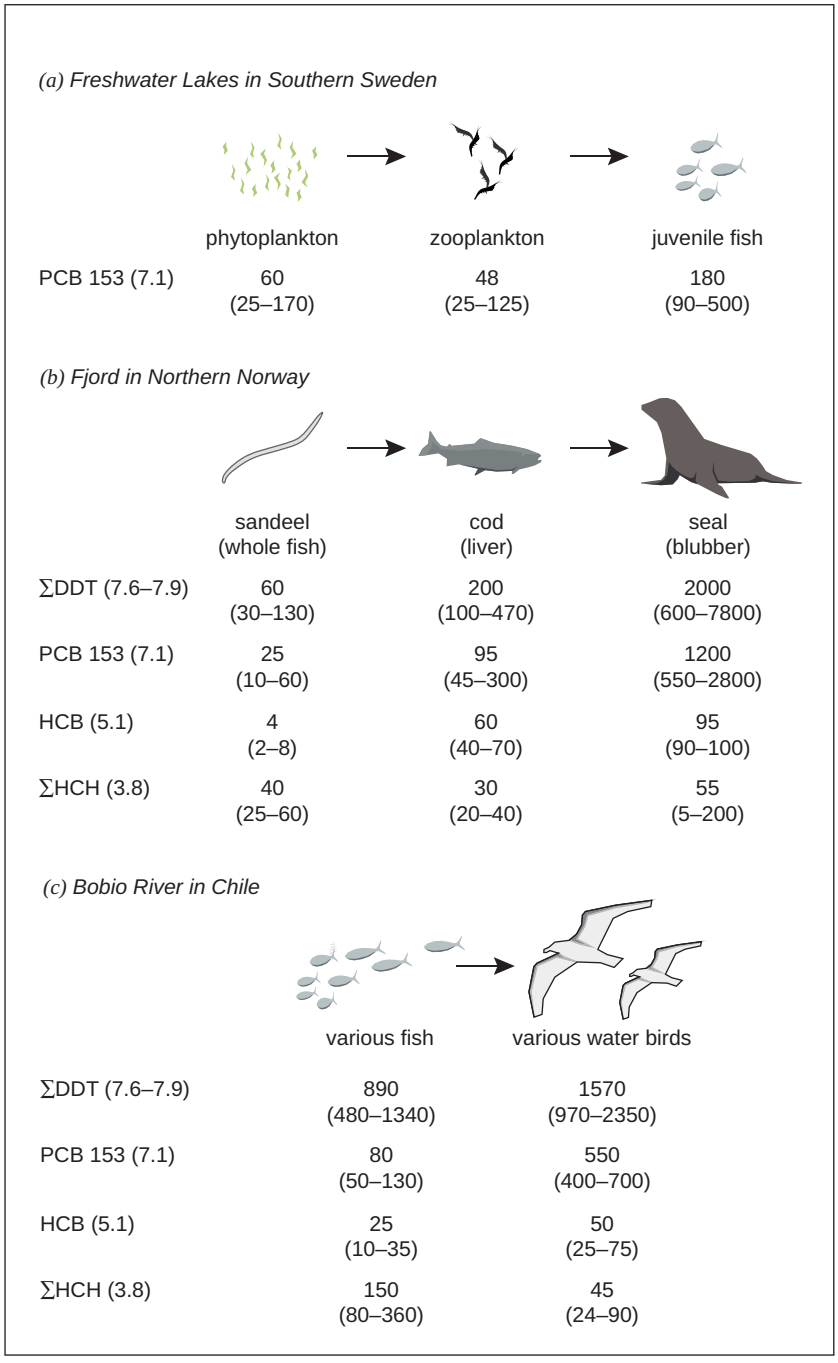


Figure 16.9 Average values of lipid-normalized concentrations (ranges in parentheses) of some organochlorine compounds: PCB153, ΣDDT = o,p-DDT + p,p-DDT + o,p-DDE + p,p-DDE, ΣHCHs = α- + β- + γ-hexachlorocyclohexane, and HCB = hexachlorobenzene (for structures see Figs. 2.7 and 3.1). log K_{iow} values are given in parentheses after the compound names). All concentrations are expressed in μg kg^{-1} lip. (*a*) Planktonic food webs in 19 lakes in Southern Sweden (Berglund et al., 2000). (*b*) Local marine food chain in a fjord in Northern Norway (Ruus et al., 1999). (*c*) Fish and fish-eating water birds from the Santa Barbara location, Bobio River, Chile (Focardi et al., 1996).

can be attributed to an increased metabolic transformation of these compounds in the birds (Focardi et al., 1996).

In addition to assessing biomagnification using BMF_i for individual organisms along a food chain, an approach exists to quantify biomagnification among trophic levels in whole ecosystems. A *trophic magnification factor* (*TMF*) is determined for a given compound by a linear regression analysis of the logarithms of the averaged fugacities

(or normalized concentrations) of all organisms assigned to the same trophic level and their relative trophic position (Gobas et al., 2009). The assignment to a given trophic level is commonly performed by using stable carbon and nitrogen isotope signatures (see Chapter 27). Thus, a $TMF > 1$ indicates increasing fugacity with increasing trophic level. For some illustrative case studies, we refer to the literature (e.g., Bodin et al., 2008; Walters et al., 2011; Borga et al., 2013; Figueiredo et al., 2014). Finally, we should point out that biomagnification in aquatic food webs may have a significant impact on humans, primarily on those for which aquatic organisms are an important food source. For example, in a survey conducted between 1990 and 1995 in Canada (Greizerstein et al., 1999), the lipid-normalized PCB concentrations in human milk of Inuits (who consume a lot of fish) were about one order of magnitude higher (i.e., $\Sigma PCB = 400\ \mu g\ kg^{-1}$ lip) as compared to other populations in the same region.

16.4 Bioaccumulation and Biomagnification in Terrestrial Systems

Bioaccumulation in Plants and Soil Organisms

Plants. Bioaccumulation of organic pollutants in plants is of great interest because plants form the diet of numerous animals and of humans (Lü et al., 2014), and, like phytoplankton in aquatic systems, they are also at the bottom of important food chains (Fig. 16.1). Figure 16.10 summarizes the various pathways by which organic pollutants may be taken up by plants or parts of plants, and how the chemicals may be transported within the plant. Furthermore, many pollutants may be transformed in the root zone of a plant and within the plant, thus making plants attractive for bioremediation purposes in contaminated soils (see White and Newman, 2011; Carvalho et al., 2014; Yavari et al., 2015). Similarly to bioaccumulation in aquatic systems, bioaccumulation in terrestrial plants is a complex, dynamic process commonly involving more than one compartment, in this case, the soil and the atmosphere (Collins et al., 2011;

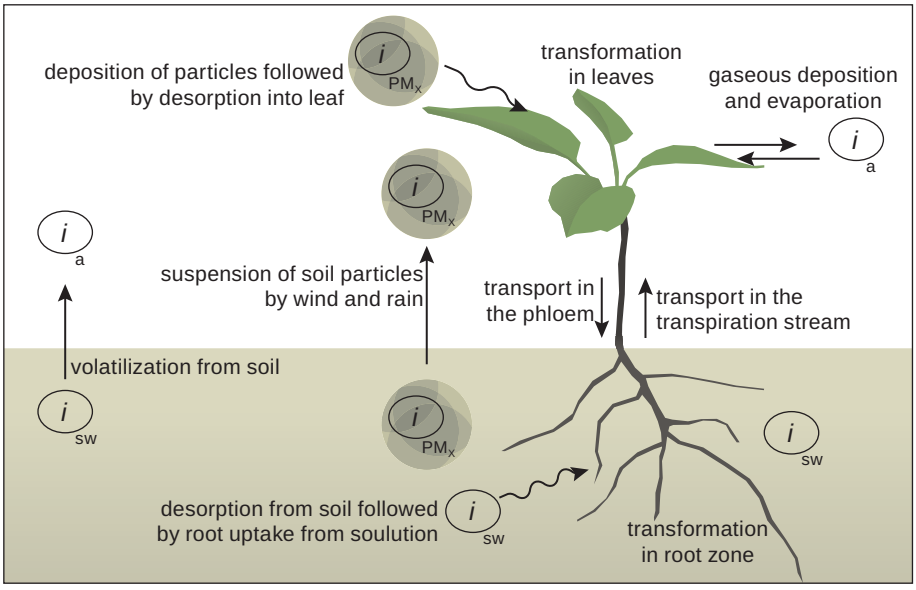


Figure 16.10 Major pathways for plant uptake of organic pollutant *i* from soil water (sw), air (a), and particulate matter (PM_x). The relative importance of the various uptake routes depends on the properties of the compound and on the type of plant. For nonvascular plants (e.g., mosses), deposition from the atmosphere is the main uptake route. Adapted from Collins et al. (2011).

Desalme et al., 2013). Hence, models for assessing bioaccumulation in plants need to address all these processes (see Prosser et al., 2014; Takaki et al., 2014). Furthermore, since transport within a plant may be slow, particularly for highly hydrophobic compounds, different concentrations in different parts of the plant (e.g., roots, shoots, leaves, fruits) may be found.

The simplest cases for quantification of bioaccumulation in plants are the *nonvascular* plants such as mosses and lichens, or parts of plants that are disconnected from the rest of the plant, such as tree bark or conifer needles. In these cases, only uptake from the air (gaseous and particulate) is important. Therefore, such plants or biomaterials have been widely used as "biomonitors" or "passive samplers" for assessing atmospheric pollution of semivolatile persistent organic chemicals including DDT, PCBs, PBDEs, PAHs, and PCDD/PCDFs (Zhao et al., 2008; Augusto et al., 2013; Harmens et al., 2013; Salamova and Hites, 2013; Yang et al., 2013; Peverly et al., 2014; Marc et al., 2015). The relative importance of gaseous uptake versus deposition by particles depends strongly on the speciation of the compound in the atmosphere, that is, to what extent it is sorbed to aerosols (see Chapter 15).

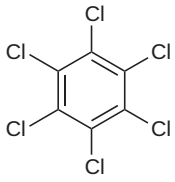
For highly hydrophobic compounds in *vascular* plants, we can assume the dominant uptake is from air, including soil air or air just above the soil into which pollutants may diffuse from contaminated soils or be carried into by resuspended soil particles (Fig. 16.10). For more water-soluble compounds, direct uptake from soil water, e.g., by the root system, may also be important (Barbour et al., 2005; Dettenmaier et al., 2009). Furthermore, from our discussions in Section 16.1, we conclude that for apolar compounds, the lipids present in the plants may be the dominant sorbent, whereas for monopolar and, particularly, bipolar compounds, other phases including proteins, lignin, carbohydrates, or even plant water may also be relevant reservoirs. Finally, we should point out that bioaccumulation from air (gaseous as well as particulate form) is strongly temperature dependent (see Section 16.1), which may lead to significant seasonal and geographical differences in concentration of organic pollutants in plants. For example, Kelly and Gobas (2001) found that concentrations of PCBs in lichens collected from a tundra system in the Arctic region of Canada in the spring exceeded those collected in the summer by a factor of 10 or more. Also, the difference in the concentrations of PCBs in arctic air between winter and summer is primarily attributed to the strong effect of temperature on air–plant partitioning (Hung et al., 2001).

As in our discussion about bioaccumulation in aquatic systems, concentrations of pollutants in plants may not be in equilibrium with the surrounding environmental media. Therefore, we now evaluate to what extent equilibrium has been established in a given terrestrial system, illustrating with the simple example of a series of POPs in air, soil, and grass. McLachlan (1996) reported average concentrations of several POPs, including hexachlorobenzene (HCB; $\log K_{i\mathrm{aw}}$ (18°C) = –1.63; $V_i = 1.45$, $L_i = 7.39$, $S_i = 0.99$, $A_i = 0$, $B_i = 0.0$), in the region of Bayreuth, Germany (Table 16.6). The average air temperature during the sampling period was 18°C. In analogy to our example dealing with the water–sediment interface (Tables 16.3 and 16.4), we may ask whether HCB is in equilibrium with either the atmosphere or the soil. To find the answer, we assume that HCB partitions primarily to SOM and to the lipids present in

Table 16.5 Comparison Between Measured and Predicted Partition Coefficients and Fugacities of HCB in an Air, Soil, and Grass System

Phase	Measured concentrations[a] $C_{i\text{phase}}$	Experimental Bioaccumulation Factors[b]	$K_{i\text{lipphase}}$ 18°C, pred.	$K^p_{i\text{aphase}}$ (18°C)[e]	Fugacity (18°C)[f]
air	1.6×10^{-15} mol L^{-1}	$BAF_{i\text{lipa}}$ $9.4\times10^6 L_a$ kg^{-1} lipid	1.1×10^8 L_a kg^{-1} lipid[c]	2.4×10^6 Pa (mol L^{-1} air)$^{-1}$	4 nPa
soil oc	6.3×10^{-8} mol kg^{-1} oc	$BSAF_{i\text{lipoc}}$ 0.24 kg oc kg^{-1} lipid	31 kg oc kg^{-1} lipid[d]	6.6×10^{-1} Pa (mol kg^{-1} oc)$^{-1}$	40 nPa
grass lipid	1.5×10^{-8} mol kg^{-1} lipid			2.1×10^{-2} Pa (mol kg^{-1} lipid)$^{-1}$	0.3 nPa

[a]Data from McLachlan (1996) for a series of POPs in air, soil, and grass (mostly ryegrass) in Bayreuth, Germany.
[b]Calculated using Eqs. 16-29 and 16-32.
[c]Predicted $K_{i\text{lipw}}$ value (2.5×10^6 L kg^{-1} lip) using Eq. 16-12, Table 16.2 for storage lipids (37°C) and adjusted for temperature (18°C) using Eq. 16-26 and an estimated $\Delta_{\text{lipw}}H_i$ of –25 kJ mol^{-1} (Eq. 10-18). Using the thermodynamic cycle and $K_{i\text{aw}}$ (0.023) yields $K_{i\text{lipa}} = K_{i\text{lipw}}/K_{i\text{aw}}$.
[d]Predicted $K_{i\text{ocw}}$ value (8×10^4 L kg^{-1} oc) using Eq. 13-14, Table 13.3 for Pahokee Peat (25°C) and adjusted for temperature (18°C) using Eq. 16-26 and an estimated $\Delta_{\text{ocw}}H_i$ of –25 kJ mol^{-1} (Eq. 10-18). Using the thermodynamic cycle yields $K_{i\text{lipoc}} = K_{i\text{lipw}}/K_{i\text{ocw}}$.
[e]Calculated using Eq. 16-42. For the gas phase, $K^p_{i\text{aphase}} = RT$.
[f]Calculated using Eq. 16-41.



hexachlorobenzene (HCB)

the grass, and we assume an average soil organic carbon content, $f_{\text{oc}} = 0.02$ kg oc kg^{-1} soil, and an average lipid content of the grasses, $f_{\text{lip}} = 0.02$ kg lip kg^{-1} d.w. Now, we can either compare the experimental $BAF_{i\text{lipa}}$ and $BSAF_{i\text{lipoc}}$ values for accumulation of HCB in the grass at 18°C with the corresponding $K_{i\text{lipa}}$ and $K_{i\text{lipoc}}$ values or just calculate the fugacities of HCB in air, soil, and grass at 18°C. For both we need, of course, to estimate $K_{i\text{lipa}}$ and $K_{i\text{lipoc}}$ at 18°C. The calculated value of $K_{i\text{lipa}}$ is about 10 times higher than the experimental $BAF_{i\text{lipa}}$, and the $K_{i\text{lipoc}}$ is more than 100 times higher as compared to the experimental $BSAF_{i\text{lipoc}}$ (Table 16.5). Therefore, the measured HCB concentrations in the grass are far from equilibrium with either the air or the soil, although they are about an order of magnitude closer to equilibrium with the air. The exact same result is, of course, obtained when just comparing the fugacities of HCB in different phases (Table 16.5).

Earthworms. Due to their high abundance and importance in maintaining the structure and fertility of soils, earthworms play a pivotal role in soil ecosystems (Edwards and Bohlen, 1995; Karaca, 2011). Therefore, accumulation and the effect of organic pollutants in earthworms is of considerable interest, particularly with the use of manure and wastewater derived biosolids in agricultural soils (Kinney et al., 2008 and 2012; Pannu et al., 2012; Gaylor et al., 2013; Carter et al., 2014; Wang et al., 2014). Because the activities of earthworms may also promote degradation of organic pollutants, they are considered useful organisms in enhancing bioremediation of contaminated soils (see Rodriguez-Campos et al., 2014).

When evaluating bioaccumulation of organic pollutants in earthworms, we have an analogous situation to sediment-dwelling organisms discussed in Section 16.2.

Earthworms may take up contaminants directly from the soil water through dermal contact or by ingestion of soil particles and subsequent release of the chemicals in the gut of the worm (Qi and Chen, 2010; Wang et al., 2014). Compounds may also undergo transformation reactions in the gut (Shan et al., 2010; Belden et al., 2011). Therefore, we can evaluate to what extent equilibrium partitioning occurs in earthworms as we did for organisms living at the sediment–water interface.

Biomagnification in Terrestrial Food Webs

As in aquatic systems, we use the term biomagnification to signify the process leading to significantly higher concentrations of persistent compounds in higher organisms. Thus, a $BMF_i > 1$ (Eq. 16-33) signifies an increase of concentration or fugacity moving up a food chain, and a $TMF > 1$ indicates increasing fugacity with increasing trophic level in an ecosytem. The bottom of most studied food webs consists of plants. We can then track the concentrations or fugacity of a given pollutant to model the accumulation along the chain.

Biomagnification of POPs in Terrestrial Food Chains. Let us again examine the biomagnification of some POPs, this time in terrestrial food chains. As previously discussed, McLachlan et al. (1996) reported hexachlorobenzene (HCB) concentrations in air, soil, and grasses (Table 16.5). HCB concentrations in cows' milk (9.0×10^3 ng kg^{-1} lip) and human milk (2.3×10^6 ng kg^{-1} lip) were also measured. Thus, one can convert these concentrations into fugacities (Fig. 16.11) and evaluate biomagnification along the food chain of grass to cow to human. Comparing HCB in cow milk lipids to pasture grasses, only a small biomagnification ($BMF_{i\text{milklipgrasslip}} = 2$) is noted, but the fugacity in human milk lipids is more than an order of magnitude larger than in cow's milk. These findings suggest that humans were not acquiring HCB chiefly from cow's milk or that humans are very efficient in biomagnifying compounds from their food sources. Instead, other sources that contain higher levels of HCB, such as seafood, may be more important food sources in terms of biomagnification. This result was also found in a study of highly brominated PBDEs, such as PBDE 209, which indicated that beef meat rather than dairy product consumption may be an important human exposure route (Kierkegaard et al., 2007).

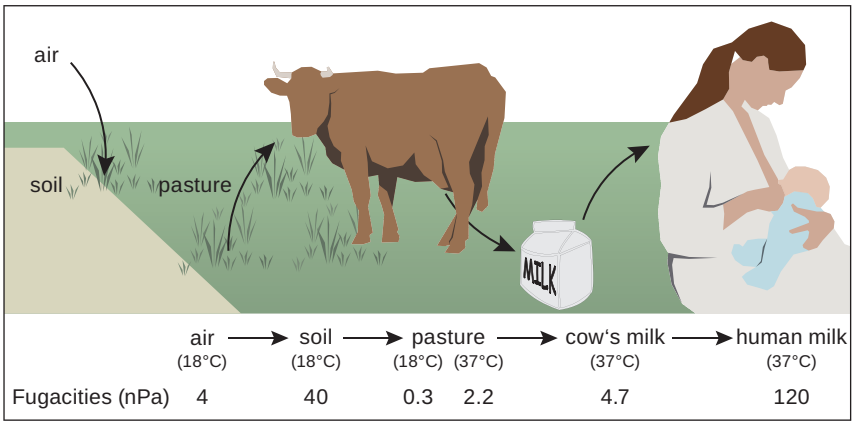
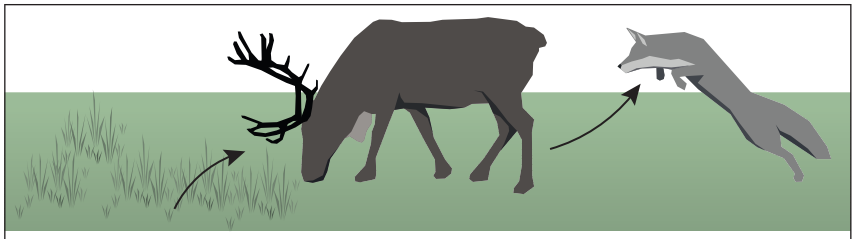


Figure 16.11 Predicted fugacites of HCB in air, soil, pasture grasses, cow milk, and human milk. Fugacities calculated using Eq. 16-41 and 16-42. Average concentrations given in McLachlan (1996) measured in Bayreuth, Germany in 1989/1990. Data for temperature corrections given in Table 16.5.



	lichens/willow leaves (37°C)		caribou lipids (37°C)	wolf lipids (37°C)
fugacities (nPa)	spring	summer	summer/fall	summer/fall
PCB 52:	8	0.4	0.4	0.4
PCB 153:	5	0.05	0.03	0.3
PCB 180:	0.4	0.01	0.003	0.1

Figure 16.12 Calculated average fugacities of PCB 52, PCB 153, and PCB 180 in pasture (i.e., lichens, willow leaves), caribou lipids, and wolf lipids in an arctic food chain in Canada (Bathurst Region). The caribou and wolf data represent averages of male and female animals. Data from Kelly and Gobas (2001).

In our second example, we consider a wildlife food chain involving the transfer of three PCB congeners from lichen and willow leaves to caribou to wolves in an Arctic environment (Kelly and Gobas, 2001). As with HCB biomagnification (Fig. 16.11), we can calculate the fugacities of the PCBs at 37°C using observations of chemical concentrations and assuming lipids are the main phases for bioaccumulation (Fig. 16.12). When considering the very similar seasonal concentration pattern of the compounds in arctic air during these years (Hung et al., 2001), the calculated fugacities should be reasonably representative.

Looking now at Fig. 16.12, we notice the much higher fugacities (i.e., concentrations) of the compounds in the pasture in spring as compared to summer. However, air concentrations are generally higher in summer as compared to winter (Hung et al., 2001). Because the air–plant (i.e., air–lipid) partition coefficients are highly temperature dependent, the large seasonal concentration variations found for PCBs in arctic plant tissues are likely due primarily to the large seasonal temperature fluctuations in such regions. Since the body concentrations of contaminants like PCBs in animals like caribou and wolves represent a time-averaging over months and years, such time-varying exposures make it, of course, difficult to determine any biomagnification.

Another important result is that, very similar to the discussed agricultural food chain (Fig. 16.11), no significant biomagnification occurs from pasture to consumers (in this case, the caribou). In fact, the caribou consistently exhibited fugacities that were much less than their springtime diet. Furthermore, for the higher chlorinated congeners (PCB 153, PCB 180), biomagnification is observed between animal prey (i.e., the caribou) and predator (i.e., the wolf). This result may be explained if the wolf digests much of its caribou diet and thereby substantially increases the PCB fugacities in its GI tract. If excretion and respiration only slowly return the PCBs to the arctic air from where they came, then the wolf may exhibit a continuous fugacity excess relative to its prey. Finally, in the fall, female animals exhibited somewhat lower PCB and other organochlorine compound concentrations than male animals (data not shown). This may be explained in part by lactation throughout the nursing period that occurs during the summer (for more details see Kelly and Gobas, 2001; Kelly et al., 2007).

16.5 Baseline Toxicity (Narcosis)

We conclude this chapter with a brief discussion of a toxicological issue that results from accumulation of chemicals in biological membranes. Organic chemicals can exert a variety of toxic effects on organisms. Depending on both the chemical and the organism, these may include unintended reactions or interactions of the contaminant or its reactive metabolites with critical functional components like enzymes or genetic macromolecules (DNA, RNA). Even apolar compounds can, without forming covalent bonds, physically associate with enzymes or genetic materials and thus disrupt their normal three-dimensional structure, which may harm the functioning of the macromolecule. All these effects require quite specific chemical interactions with components of the organism so they are referred to as specific toxic effects or *specific toxicity* (see e.g., Escher et al., 2011; Sturla et al., 2014).

However, any compound, even if chemically inert, when present at a high enough concentration in biological membranes, can change those membranes' properties and disrupt their functions. Consequently, membrane-associated processes like photosynthesis, energy transduction, transport in and out of the cell, enzyme activities, or transmission of nerve impulses may deteriorate (see van Wezel et al., 1995; Escher and Hermens, 2002 and 2004). Since these effects seem to be primarily dependent on the environment of the membrane that the chemical molecules occupy and not on the chemical structure of the compound itself, this type of toxicity is called *nonspecific toxicity*. As such nonspecific interactions include disrupting nerve functions, they are also often referred to as *narcosis*. Furthermore, because any additional specific toxic effect would increase the overall toxicity of a compound, this mode of toxic action is often described using terms like *minimum toxicity* or *baseline toxicity*. In the following discussion, we will confine our considerations to simple equilibrium conditions. For kinetic aspects (i.e., toxicokinetics), we refer to the literature (e.g., Stadnicka et al., 2012; Kühnert et al., 2013; Nyman et al., 2014; Mackay et al., 2014a). We also relate *external concentrations* to bulk effects within the organism, which is, of course, a very simple way to describe toxicity. What we would ultimately need to know is the concentrations of a given chemical at the target sites within an organism at which effects occur. This is, of course, not a trivial task (see e.g., Escher and Hermens, 2002 and 2004; Escher et al., 2011; Sturla et al., 2014).

Let us now evaluate how we can assess the baseline toxicity of organic chemicals in a quantitative way. We have already mentioned that certain membrane functions may be disrupted if a chemical occupies a certain volume fraction of a membrane. This means that for two compounds of the same size, we would anticipate that when they are present at equal concentrations in the membrane, they would exert the same effect. Furthermore, since the majority of chemicals of interest to us do not differ in size by more than a factor of 3 to 4 (compare molar volumes in Chapter 7, e.g., Fig. 7.2), the membrane concentration required for any compound to cause *a narcotic effect will be in the same order of magnitude*. Therefore, we may expect that the concentration of a compound required in an environmental medium (e.g., water, air) to cause a narcotic effect in a given organism should be inversely proportional to the tendency of the compound to accumulate from that medium into biological membranes. Importantly,

when interpreting experimental toxicity data, we need to know the "freely dissolved" (bioavailable) concentration of a given compound in the experimental system, not just the total (or nominal) concentration calculated from the amount of compound added. In many cases, the media used to conduct the toxicity tests contain dissolved and colloidal organic phases to which the compound may sorb, which would make the compound look less toxic than it actually is (see Escher and Hermens, 2004).

As evident from numerous studies in aquatic media, for chemicals that exhibit only, or primarily, baseline toxicity, the "truly dissolved" concentration in water required to exert a certain effect on (i.e., the effective concentration, $EC_{iw,}$ in mol L^{-1}), or even kill (i.e., the lethal concentration, LC_{iw} in mol L^{-1}), an organism has been found to correlate reasonably with parameters used to describe organic phase–water partitioning. Traditionally, the parameter used for such *quantitative structure-activity relationships (QSARs)* has been the octanol–water partition constant K_{iow} (e.g., Escher and Schwarzenbach, 2002, Box 1; Netzeva et al., 2008; Mackay et al., 2014b):

$$\log \frac{1}{LC_{iw50} \text{ or } EC_{iw50}} = a \log K_{iow} + b \tag{16-44}$$

where, commonly, the concentration required to exert 50% of the maximum effect, e.g., to kill 50% of a population (LC_{iw50}) or to inhibit some function by 50% (e.g., growth, photosynthesis, motility, etc., EC_{iw50}) after a certain exposure time (e.g., 24, 48, 96 hours) is used to express the toxic endpoint. As seen in earlier chapters when discussing partitioning in other organic phase–water or –air systems, in sp-LFERs (or sp-QSARs) such as Eq. 16-44, K_{iow} is not an optimal parameter, particularly when considering compounds exhibiting different polarities. In the case of membrane toxicity, better correlations can be achieved when using actual membrane–water partition coefficients, K_{imlipw} (Escher and Schwarzenbach, 2002):

$$\log \frac{1}{LC_{iw50} \text{ or } EC_{iw50}} = a' \log K_{imlipw} + b' \tag{16-45}$$

If no experimental K_{imlipw} values are available, they can be estimated by the pp-LFER Eq. 16-13 (Table 16.2).

Furthermore, when dealing with ionizable compounds, for example with organic acids, in analogy to sorption from water to natural organic matter (Chapter 13, Eq. 13-16) we need to substitute K_{iow} and K_{imlipw} in Eqs. 16-44 and 16-45 by the overall distribution ratios $\mathcal{D}_{iow}$ (Eq. 16-46) and $\mathcal{D}_{imlipw}$ (Eq. 16-47) respectively.

$$\mathcal{D}_{iow} = \frac{[HA]_{ow} + [A^-]_{ow}}{[HA]_w + [A^-]_w} = \alpha_{ia} K_{iow}^{HA} + (1 - \alpha_{ia}) K_{iow}^{A^-} \tag{16-46}$$

$$\mathcal{D}_{imlipw} = \frac{[HA]_{mlipw} + [A^-]_{mlipw}}{[HA]_w + [A^-]_w} = \alpha_{ia} K_{imlipw}^{HA} + (1 - \alpha_{ia}) K_{imlipw}^{A^-} \tag{16-47}$$

where α_{ia} is the fraction in nondissociated form and is given by $\alpha_{ia} = (1 + 10^{pH-pK_{ia}})^{-1}$(Chapter 4, Eq. 4-59), and $K_{iow}^{A^-}$ and $K_{imlipw}^{A^-}$, respectively, are the partition constants of the anionic species.

Hence, for organic acids we need to express Eqs. 16-44 and 16-45, respectively as:

$$\log \frac{1}{LC_{iw50} \text{ or } EC_{iw50}} = a \log \mathcal{D}_{iow} + b \tag{16-48}$$

$$\log \frac{1}{LC_{iw50} \text{ or } EC_{iw50}} = a' \log \mathcal{D}_{imlipw} + b' \tag{16-49}$$

We should note that $K_{iow}^{A^-}$ and $K_{imlipw}^{A^-}$, and thus $\mathcal{D}_{iow}$ and $\mathcal{D}_{imlipw}$, depend on the ionic strength in the aqueous phase (partitioning of ion pairs into the organic phase), and, even more importantly, that anions generally partition much more favorably into membrane lipids as compared to octanol (for details see Escher and Schwarzenbach, 1996). Therefore, the accumulation of organic acids in membranes is much more realistically described by $\mathcal{D}_{imlipw}$ than by $\mathcal{D}_{iow}$. Thus when evaluating toxicity data, plots of $1/\log LC_{iw}$ or $1/\log EC_{iw}$ versus $\log \mathcal{D}_{imlipw}$ should be used in order to assess whether a chemical exerts only a non-specific effect or whether it is more toxic than predicted by a QSAR such as Eq. 16-49. This enhanced toxicity is commonly referred to as "*excess toxicity*".

As an illustration we consider some acute fish toxicity of some substituted benzenes and phenols. Figures 16.13*a* and *b* show plots of log $1/LC_{iw50}$ for guppies versus $\log \mathcal{D}_{iow}$ and $\log \mathcal{D}_{imlipw}$, respectively, for a series of chlorinated benzenes, some chlorinated phenols including the pesticide and disinfectant pentachlorophenol (PCP), and the nitrophenolic herbicide 2-*sec*-butyl-4,6-dinitrophenol (dinoseb). As can be seen, the toxicity of the neutral chlorinated benzenes is well described by the QSARs Eqs. 16-48 (Fig. 16.13*a*) and 16-49 (Fig. 16.13*b*). In contrast, all chlorinated phenols, and in particular, the nitrophenol, are more toxic than their predicted baseline toxicity,

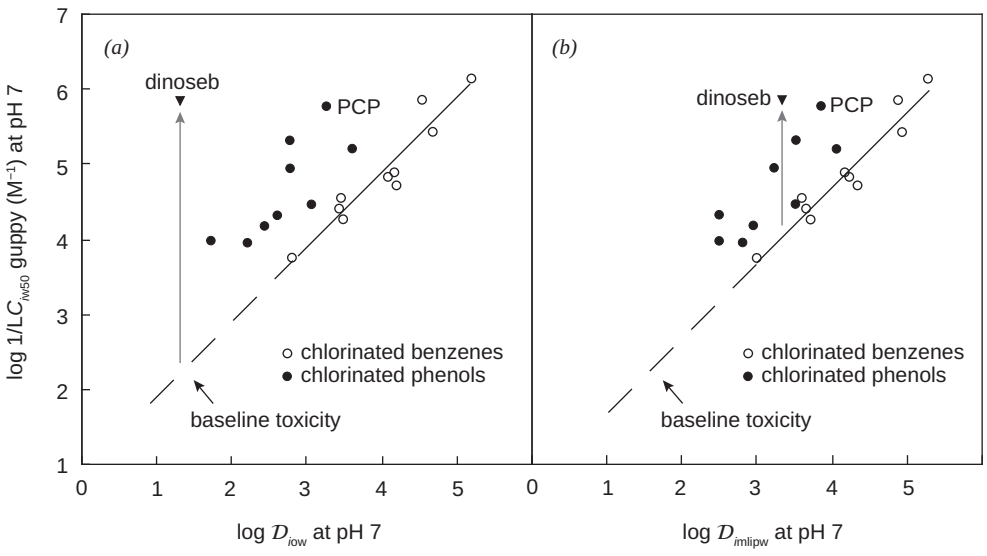
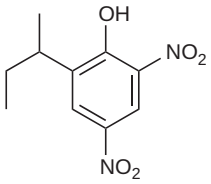


Figure 16.13 Plot of log $1/LC_{iw50}$ for guppies versus (*a*) log octanol–water distribution ratio (log D_{iow}) and (*b*) log liposome–water distribution ratio (log D_{imlipw}) at pH 7 for a series of chlorinated benzenes and chlorinated phenols as well as for the herbicide 2-*sec*-butyl-4,6-dinitrophenol (dinoseb). The lines represent base-line toxicities (Eqs. 16-48 and 16-49) established from the chlorinated benzene data. The liposomes used were L-α-dimyristoyl-phosphatidylcholine (chlorinated benzenes) and L-α-dioleyl-phosphatidylcholine (chlorinated phenols and dinoseb). Data from Gobas et al. (1988); Saarikoski and Viluskela (1992); Escher and Schwarzenbach (1996); and Gunatilleka and Poole (1999).

but there are significant differences in their relative excess toxicities. This enhanced toxicity of the phenols may not be too surprising since these compounds are known to exert specific effects, including interference with the energy transduction of cells by destroying the electrochemical proton gradient (i.e., uncoupling; for more details, e.g., Escher et al., 2002). This excess toxicity of a given compound is often quantified by its so-called *toxic ratio*, TR_i, which is defined by (Verhaar et al., 1992; Maeder et al., 2004):

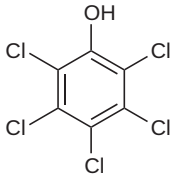


2-*s*-butyl-4,6-dinitrophenol (dinoseb)

$$TR_{i50} = \frac{1/L(E)C_{i50}(\text{actually measured})}{1/L(E)C_{i50}(\text{baseline, predicted by QSAR})}$$

or

$$TR_{i50} = \frac{L(E)C_{i50}(\text{baseline, predicted by QSAR})}{L(E)C_{i50}(\text{actually measured})} \qquad (16\text{-}50)$$



pentachlorophenol (PCP)

We should point out again that using external concentrations for assessing toxicity is not optimal; a more appropriate way would be to use internal concentrations in order to identify whether a chemical exhibits excess toxicity and how significant this excess toxicity is compared to baseline toxicity (e.g., Escher et al., 2011; Su et al., 2014). Commonly, if octanol is used to predict $L(E)C_{i50}$ (Eq. 16-44), a toxic ratio of >10 is considered to be indicative of specific toxicity (e.g., Maeder et al., 2004; Li et al., 2015).

Back to our example. As can be seen from Fig. 16.13*a*, if we use $D_{i\text{ow}}$ for prediction of the baseline toxicity, we get a TR_{i50} value of almost 10^4 for dinoseb. But this result is at least partially due to our significant underestimating of the partitioning of this compound from water to the membranes. A more "correct" TR_i value takes into account the partitioning of the anion to the membrane by using the $D_{i\text{mlipw}}$ value; that is, a more realistic TR_{i50} value for dinoseb is on the order of 10^2 (Fig. 16.13*b*). The same general finding is true, although to a somewhat lesser extent, for the chlorinated phenols. We note that in all these cases, we assume that the anionic species exhibits the same narcotic effect as the neutral species. This example shows again that using octanol to mimic partitioning to membranes, particularly if ionizable compounds are considered, is not optimal, and that, in general, we have to be very careful in choosing appropriate molecular parameters for describing partitioning of organic compounds to biological media.

Finally, we should point out that in natural systems, organisms may be exposed to a variety of chemicals at the same time (e.g., after an oil spill, in PCB-contaminated sediments, etc.). Since the chemical nature of the compound is not so important for narcotic effects, it is easy to imagine that the sum of all compounds accumulating in an organism's membrane will lead to an adverse effect. Thus, for example, an already contaminated organism may exhibit a much lower $EC_{i\text{w}}$ or $LC_{i\text{w}}$ value when exposed to a single chemical as compared to an organism that, prior to exposure, had lived in a clean environment. Therefore, even though the $EC_{i\text{w}}$ or $LC_{i\text{w}}$ values to achieve

narcotic effects may be quite high for a single compound, because of its concentration-additive nature, this mode of action may be of ecotoxicological significance in contaminated environments (see, e.g., Schmidt et al., 2013; Smith et al., 2013).

16.6 Questions and Problems

Special note: Problem solutions are available on the book's website. Solutions to problems marked with an asterisk are available for everyone. Unmarked problems have solutions only available to teachers, practitioners, and others with special permission.

Questions

Q 16.1

In general, why can bioaccumulation not be treated as an equilibrium process?

Q 16.2

What are the most important organic phases present in living media into which organic pollutants may partition? Characterize these phases with respect to their ability to "dissolve" organic solutes via various intermolecular interaction mechanisms. In which cases are which phases important?

Q 16.3

Describe in words, the parameters: (1) bioconcentration factor, (2) bioaccumulation factor, (3) biomagnification factor, and (4) biota–sediment and biota–soil accumulation factor.

Q 16.4

Through what mechanisms can bioaccumulation lead to a "real" biomagnification?

Q 16.5

Why do biomagnification and biota–sediment accumulation factors of persistent chemicals in aquatic systems tend to increase with increasing hydrophobicity of the compounds?

Q 16.6

Why is it useful to compare the activities or fugacities of a given compound in various environmental compartments?

Q 16.7

Explain in words how you calculate the fugacity of a given compound in a given environmental compartment (e.g., water, sediment, lipid components of an organism, etc.). What are the major problems encountered when calculating (or estimating) fugacity values?

Q 16.8

What is meant by the terms "baseline toxicity" or "narcosis"? Why is this type of toxicity relevant in the environment? How is the toxicity ratio of a compound defined? Why is this a useful parameter?

Q 16.9

When is the *n*-octanol–water partition constant an acceptable parameter for the quantification of nonspecific membrane toxicity? In what situations does it fail?

Problems

P 16.1 * *Evaluating the Accumulation of Toluene and 4-Chlorophenol from Water and Air to Lettuce Leaves*

Chang et al. (2013) investigated the uptake from water of some organic pollutants by various vegetables at neutral pH (assumed, not reported, would pH matter at all?) and 25°C. For lettuce leaves they obtained apparent equilibrium bioaccumulation factors, $BAF_{i\text{lettw}}$, of 5.3 and 4.6 L kg^{-1}wet weight (w.w.) for toluene (log $K_{i\text{ow}} = 2.69$; $V_i = 0.86$, $L_i = 3.33$, $S_i = 0.52$, $A_i = 0$, $B_i = 0.14$) and 4-chlorophenol (log $K_{i\text{ow}} = 2.42$; $V_i = 0.90$, $L_i = 4.78$, $S_i = 1.08$, $A_i = 0.67$, $B_i = 0.20$), respectively. On a w.w. basis, the composition of the lettuce was determined to be 0.3% lipids, 5.5% carbohydrates, and 94.2% water.

(a) Using the simple model introduced in Section 16.2, estimate the lettuce–water partition coefficients, $K_{i\text{lettw}}$ (Eq. 16-1), of the two compounds using appropriate predictive tools, and compare these values with the experimental $BAF_{i\text{lettw}}$ previously given. What fraction of the totally accumulated compound would you expect for toluene and 4-chlorophenol in the lipid fraction of the lettuce leaves? What compound fractions in the water present in the leaves?

(b) Assuming that the thermodynamic cycle can be applied, estimate the lettuce–air partition coefficients at 25°C of the two compounds. Relative to water, what air concentrations would be required to lead to similar equilibrium concentrations in the lettuce leaves? Comment on all assumptions that you make.

Comment: For any temperature corrections, use an average ΔH_i of –25 kJ mol^{-1} for both compounds.

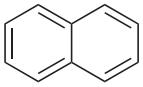
P 16.2 *Evaluating Triolein–Water and Algae–Water Partitioning of Naphthalene and Bisphenol A*

Hung et al. (2014) have determined triolein–water partition constants for 53 apolar and weakly monopolar compounds at 25°C. Triolein (subscript "tr"), which is a triglyceride esterified with three oleic acids, is a common surrogate for storage lipids. From their data they derived the following sp-LFER for triolein–water partitioning at 25°C:

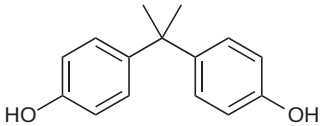
$$\log K_{i\text{trw}} = 1.03 \log K_{i\text{ow}} + 0.11$$
$$(\text{number of chemicals} = 53;\ r^2 = 0.99)$$

For naphthalene they measured a $K_{i\mathrm{trw}}$ of 3630 L_w kg^{-1}triolein. They concluded that for PAHs and all other investigated compounds, the octanol–water partition constant describes storage lipid–water partitioning quite well. But what about more polar compounds?

(a) Estimate the triolein–water partition coefficients of naphthalene (log $K_{i\mathrm{ow}} = 3.38$; $V_i = 1.09, L_i = 5.16, S_i = 0.92, A_i = 0, B_i = 0.20$) and of the bipolar endocrine disruptor bisphenol A (log $K_{i\mathrm{ow}} = 3.40$; $V_i = 1.86, L_i = 9.60, S_i = 1.56, A_i = 0.99, B_i = 0.91$) by using the above sp-LFER and the pp-LFER given for storage lipid–water partitioning in Table 16.2. Compare and comment on the results.



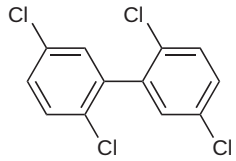
napthalene



bisphenol A

(b) In the same study, Hung et al. (2014) measured algae–water partition coefficients for the 53 compounds. The composition on a *dry* weight (d.w.) basis of the algae used (*Chlorella sorokiniana)* was determined as 12.9% lipids, 60.2% proteins, 15.6% carbohydrates, 7.4% ash, and 3.9% moisture. For naphthalene they obtained an algae–water bioaccumulation factors, $BAF_{i\mathrm{algaew}}$, of 525 L_w kg^{-1} d.w. algae. They concluded that naphthalene partitioned primarily to the lipid phase in the algae. Is this a reasonable assumption? Would this assumption also be reasonable for the accumulation of bisphenol A in this algae? Or would partitioning of this bipolar compound to the proteins and/or to the carbohydrates be equally or even more important?
Comment: For any temperature corrections, use an average ΔH_i of –25 kJ mol^{-1} for both compounds.

P 16.3 *Using Fugacities for Evaluation of Bioaccumulation Disequilibrium of a Sediment-Dwelling Organism for PCB 52*



2,2′,5,5′-tetrachlorobiphenyl (PCB 52)

Morrison et al. (1996) reported average concentrations of PCB 52 (V_i = 1.81, L_i = 8.14, S_i = 1.48, A_i = 0, B_i = 0.15) determined in the water column, in the sediments and in zebra mussels at a field site in Western Lake Erie. Calculate the fugacities in nPa of PCB 52 in the three compartments using the information given below and assuming that this compound is primarily associated with the organic matter in the sediment (SOM) and with the lipid phases in the zebra mussels. Assume a temperature of 5°C for all three compartments. Compare the result with the fugacities of PCB 153 given in Table 16.4 for the same system. Comment on your findings.

Concentration in the water column, $C_{i\mathrm{w}}$ (μg L^{-1}): 2.8×10^{-5}
Concentration in the sediment, $C_{i\mathrm{s}}$ (μg kg^{-1}solid): 4.0
Fraction organic carbon in sediment, f_{oc} (kg oc kg^{-1}solid): 0.074
Concentration in zebra mussels, $C_{i\mathrm{zmussel}}$ (μg kg^{-1}wet weight): 1.5
Lipid fraction in zebra mussels, f_{lip} (kg lipid kg^{-1}wet weight): 0.013

P 16.4 *Comparing Lipid–Sediment Organic Matter (SOM) Partitioning of Cyclic Decamethylpentasiloxane (D5) with PCB 153 and PBDE 99*

Kierkegaard (2011) and Borga et al. (2012) investigated the bioaccumulation and biomagnification of some cyclic volatile methylsiloxanes, as well as of some PCBs and PBDEs, in an estuary and in a lake respectively. From their field data, they conclude that, compared to PCBs or PBDEs, the siloxanes partition much more favorably in lipids than in SOM (Kierkegaard, 2011). Is this assumption reasonable? Calculate the $K_{i\mathrm{lipoc}}$ values (Eq. 16-39) for our companions D5 (V_i = 2.93, L_i = 5.24, S_i = –0.10,

$A_i = 0$, $B_i = 0.50$), PCB 153 ($V_i = 2.06$, $L_i = 9.59$, $S_i = 1.74$, $A_i = 0$, $B_i = 0.11$), and PBDE 99 ($V_i = 2.26$, $L_i = 11.71$, $S_i = 1.51$, $A_i = 0$, $B_i = 0.44$) and compare the results. To this end derive a pp-LFER for estimating log $K_{i\text{slipoc}}$ by combining the pp-LFERs for storage lipid–water (Table 16.2) and SOM (Pahokee Peat)–water (Table 13.3) partitioning. Neglect the effect of temperature (is this reasonable?). Try to rationalize any differences found between the three compounds by discussing the different terms of the derived pp-LFER.

P 16.5 *Evaluating Biomagnification of PBDE Congeners in Harbor Seals*

Shaw et al. (2009) reported average lipid-normalized concentrations of 16 PBDE congeners (among them also for our companion PBDE 99) for 7 different teleost fishes (winter flounder, Atlantic herring, American place, white hake, alewife, Atlantic mackerel, silver hake) and for the blubbers of seven northwest Atlantic adult harbor seal whose major diet are these type of fishes. The fishes exhibited lipid contents between 1 and 10% wet weight, the harbor seal blubbers about 60%. The following *lipid-normalized* concentrations were measured for 4 selected congeners:

PBDE congener	PDBE Nr.	concentration in fish (ng g^{-1} lipid)	average concentration in seal (ng g^{-1} lipid)[a]
2,2′,4,4′-tetrabromodiphenylether	47	26 ± 12	904
2,2′,4,5′-tetrabromodiphenylether	49	4.6 ± 3.9	1.5
2,2′,4,4′,5-pentabromodiphenylether	99	4.5 ± 2.6	134
2,2′,4,4′,5,5′-hexabromodiphenylether	153	0.68 ± 0.45	210

[a]No standard deviations reported

Note: Lower brominated congeners may also be metabolites of higher brominated congeners (e.g., Stapleton et al., 2004)

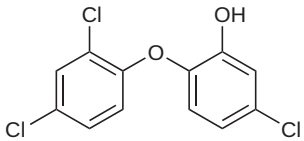
(a) Is it reasonable to use lipid-normalized concentrations to evaluate biomagnification between these two trophic levels? Would not the proteins in the fish also be an important phase for accumulation of the PBDEs?

(b) Calculate the $BMF_{i\text{sealfish}}$ values for the 4 PBDE congeners. Comment on any differences found in these $BMF_{i\text{sealfish}}$ values.

(c) The body temperature of the seals is around 37°C, whereas the fish live in the northwest Atlantic with water temperatures of significantly less than 20°. Has temperature any influence on the calculated $BMF_{i\text{sealfish}}$ values?

P 16.6 *Evaluating Bioaccumulation of the Antimicrobial Triclosan in Agricultural Plants*

The land application of treated wastewater and of biosolids stemming from waste water treatment plants (WWTP) represent a source of pharmaceuticals and personal care products (PPCPs) in agricultural soils. Therefore, there has been a growing



triclosan

interest in the possible accumulation of such chemicals into food crops. As such, quite a number of laboratory, greenhouse and field studies have been conducted to assess the bioaccumulation of PPCPs in vegetables and fruit (see Carter et al., 2014; Prosser et al., 2014 for an overview of the literature). For example, in a greenhouse study, Holling et al. (2012) have investigated the accumulation of a series of PPCPs including our companion triclosan (log $K_{i\mathrm{ow}} = 4.80$, $V_i = 1.81$, $L_i = 8.96$, $S_i = 1.81$, $A_i = 0.92$, $B_i = 0.30$) in cabbage grown in a soil amended with biosolids from a WWTP. The cabbage plants (roots and aerials) were harvested at maturity (71 days) and analyzed together with soil samples for triclosan. The concentration of triclosan, $C_{i\mathrm{s}}$, in the soil (organic carbon content 2.5%) was 0.4 mg kg^{-1} soil, the average concentration in the roots, $C_{i\mathrm{root}}$, 1.5 mg kg^{-1} wet root, and the concentration in the aerials 0.04 mg kg^{-1} wet leaves.

(a) Try to find reasons why the concentration of triclosan in the roots are significantly higher than in the aerials of the cabbage (which is actually good news, or not?). Note that the same findings are reported for accumulation of triclosan into other plants grown in biosolid amended soils (Pannu et al., 2012) or from hydroponic systems into different vegetables (Mathews et al., 2014).

(b) Assess to what extent equilibrium has been established between the roots and the biosolid amended soil. To this end, assume that triclosan sorption is governed by SOM and compare the experimental $BSAF_{i\mathrm{rootoc}}$ in kg oc kg^{-1}dry root with predicted lipid–oc, protein–oc, and carbohydrate–oc partition coefficients using the appropriate pp- and sp-LFERs. Neglect any temperature dependences of the partition coefficients. Assume a water content of the roots of 90%, the composition of the root (% lipid, protein, carbohydrate, etc.) was unfortunately not determined, so make some assumptions. Comment on any assumptions that you make.

(c) For another sort of cabbage grown in a hydroponic system, Mathews et al. (2014) reported a root–water bioaccumulation factor, $BAF_{i\mathrm{rootw}}$, of 730 L growth media kg^{-1} dry root. Compare this value with the one calculated for the soil grown cabbage roots evaluated under (b). Comment the result.

P 16.7 *Evaluating the Bioaccumulation of PAHs by Earthworms in Contaminated Soils*

In a field study, Ma et al. (1998) determined the concentration of PAHs in earthworms (*Lumbricus rubellus*) present in various contaminated soils. The concentrations measured for our companions phenanthrene ($V_i = 1.45$, $L_i = 7.58$, $S_i = 1.25$, $A_i = 0$, $B_i = 0.24$) and benzo[a]pyrene ($V_i = 1.95$, $L_i = 11.47$, $S_i = 1.82$, $A_i = 0$, $B_i = 0.31$) in the worms, $C_{i\mathrm{worm}}$, collected from two of the soils investigated are given as follows, together with the respective soil concentrations of the two compounds. The f_{oc} values of the soils were 0.038 (soil 1) and 0.015 (soil 2) kg oc kg^{-1} solid. The average lipid content of the worms was 0.012 ± 0.06 kg lip kg^{-1} f.w. (f.w. = fresh weight, same as wet weight).

Calculate the lipid– and organic carbon–normalized biota–soil accumulation factors ($BSAF_{ilipoc}$) for the two compounds and soils. Compare these values with estimated equilibrium accumulation factors ($K_{i\mathrm{slipoc}}$). Discuss the results.

	Phenanthrene	Benzo[a]pyrene
Soil 1:		
$C_{i\text{worm}}$ (mg kg^{-1} f.w.)	17	28
C_{is} (mg kg^{-1} solid)	840	990
Soil 2:		
$C_{i\text{worm}}$ (mg kg^{-1} f.w.)	4	6
C_{is} (mg kg^{-1} solid)	22	27

P 16.8 *Evaluating the Effect of Biochar Amendment on the Bioaccumulation of Atrazine from Soil into Two Different Earthworms*

Wang et al. (2014) have investigated the effect of biochar amendment to soils on the accumulation of our companion atrazine ($V_i = 1.62$, $L_i = 7.78$, $S_i = 1.28$, $A_i = -0.10$, $B_i = 0.96$) in two different earthworms, *E. foetida* (average weight 0.4 g, lipid content 7% d.w.) and *M.guillelmi* (average weight 2 g, lipid content 11% d.w.). By assuming that atrazine sorbs primarily to SOM and that it accumulates primarily in the lipids of the worms, they reported the following $BSAF_{i\text{lipoc}}$ values:

Biochar dose (% mass fraction)	*E.foetida* $BSAF_{i\text{lipoc}}$ (kg oc kg^{-1} lip)	*M.guillelmi* $BSAF_{i\text{lipoc}}$ (kg oc kg^{-1} lip)
0	0.08	0.4
0.5	0.05	0.1
2	0.04	0.04

The experiments were conducted at 22°C.

(a) Is the assumption that the lipids are the dominant biological phase for atrazine partitioning correct? What about the role of proteins, assuming that 60% of the worm biomass are proteins (Table 16.1).

(b) The concentration of atrazine in the worms is a result of the rates of uptake, biotransformation, and depuration. To what extent has equilibrium with the soil bound atrazine been established in the case of *M.guillelmi*?

(c) Try to find some explanation for the finding that in nonamended soil $BSAF_{i\text{lipoc}}$ is 5 times higher for *M.guillelmi* as compared to *E.foetida*, but that for soil exhibiting 2% biochar the two values are equal.

P 16.9 *Assessing the Fugacities of Three PCB Congeners in an Agricultural Food Chain*

In his study on the transfer of POPs in an agricultural food chain, McLachlan (1996) measured the concentrations of various PCBs in pasture (grass, cow milk, and human

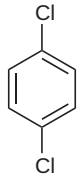
milk in the region of Bayreuth, Germany. For PCB 52 (V_i = 1.81, L_i = 8.14, S_i = 1.48, A_i = 0, B_i = 0.15), PCB 153 (V_i = 2.06, L_i = 9.59, S_i = 1.74, A_i = 0, B_i = 0.11), and PCB 180 (V_i = 2.18, L_i = 10.42, S_i = 1.87, A_i = 0, B_i = 0.09) they reported the following lipid-normalized concentrations (assumption: compounds partition into lipids; lipid content of the grass is 5%):

	PCB 52 (2,2′,5,5′)	PCB 153 (2,2′,4,4′,5,5′)	PCB 180 (2,2′,3,4,4′,5,5′)
$C_{i\text{grass}}$ (ng kg^{-1} lip)	1600	8000	1600
$C_{i\text{cow}}$ (ng kg^{-1} lip)	27	5900	1900
$C_{i\text{human}}$ (ng kg^{-1} lip)	1500	175000	71300

Calculate and compare the fugacities of the three PCB congeners in the grass, cow milk, and human milk at 37°C. Compare the results also with the fugacities calculated for hexachlorobenzene from data reported in the same study (Fig. 16.11).

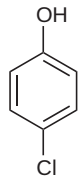
P 16.10 *Evaluating the Toxicities of 1,4-Dichlorobenzene, 4-Chlorophenol, and 4-Chloroaniline to Fish (Guppy)*

Gunatilleka and Poole (1999) have compiled toxicity data for various aquatic organisms for a large number of chemicals that are supposed to be baseline toxicants. For guppy (*Poecilia reticulate*) they report the following $LC_{i\text{w}50}$ (in mol L^{-1} measured at pH 7 at room temperature) for the three compounds:



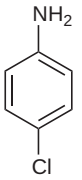
1,4-dichlorobenzene

1,4-dichlorobenzene (DCB): $\log(1/LC_{i\text{w}50}) = 4.56$; $\log K_{i\text{ow}} = 3.45$; $V_i = 0.96$, $L_i = 4.44$, $S_i = 0.75$, $A_i = 0$, $B_i = 0$),



4-chlorophenol

4-chlorophenol (CP, $\log(1/LC_{i\text{w}50}) = 4.18$; $\log K_{i\text{ow}} = 2.42$; $V_i = 0.90$, $L_i = 4.78$, $S_i = 1.08$, $A_i = 0.67$, $B_i = 0.20$), and



4-chloroaniline

4-chloroaniline (CA, $\log(1/LC_{i\text{w}50}) = 3.67$; $\log K_{i\text{ow}} = 1.83$; $V_i = 0.94$, $L_i = 4.89$, $S_i = 1.13$, $A_i = 0.30$, $B_i = 0.31$).

(a) Calculate the toxic ratio, TR_{i50} (Eq. 16-60) for the three compounds using (i) a QSAR with $K_{i\text{ow}}$ or (ii) a QSAR with $K_{i\text{mlipw}}$ as molecular parameter. The corresponding baseline toxicity QSARs for guppy can be found in Escher and Schwarzenbach (2002, Box 1):

with $K_{i\text{ow}}$: $\log 1/LC_{i\text{w}50} = 0.87 \log K_{i\text{ow}} + 1.21$ for nonpolar compounds
$\log 1/LC_{i\text{w}50} = 0.75 \log K_{i\text{ow}} + 1.58$ for polar compounds
with $K_{i\text{mlipw}}$: $\log 1/LC_{i\text{w}50} = 0.83 \log K_{i\text{mlipw}} + 1.52$ for nonpolar and polar compounds

Neglect the effect of temperature on the partition coefficients. Comment on the result.

16.7 Bibliography

Aber, J. D.; Martin, M., Leaf Chemistry, 1992-1993 (Accelerated Canopy Chemistry Program). Data set. Oak Ridge National Laboratory Distributed Active Archive Center, 1999. http://www.daac.ornl.gov.

Ahti, T.; Hepburn, T. L., *Preliminary Studies on Woodland Caribou Range, Especially on Lichen Stands in Ontario*; Ontario Dept. Lands and Forests, Research Branch, Toronto, ON, 1967.

Arnot, J. A.; Gobas, F. A. P. C., A food web bioaccumulation model for organic chemicals in aquatic ecosystems. *Environ. Toxicol. Chem.* **2004**, *23*(10), 2343–2355.

Arnot, J. A.; Gobas, F. A. P. C., A review of bioconcentration factor (BCF) and bioaccumulation factor (BAF) assessments for organic chemicals in aquatic organisms. *Environ. Rev.* **2006**, *14*(4), 257–297.

Aruoja, V.; Moosus, M.; Kahru, A.; Sihtmae, M.; Maran, U., Measurement of baseline toxicity and QSAR analysis of 50 non-polar and 58 polar narcotic chemicals for the alga Pseudokirchneriella subcapitata. *Chemosphere* **2014**, *96*, 23–32.

Atkinson, D.; Curthoys, G., The determination of heats of adsorption by gas-solid chromatography. *J. Chem. Educ.* **1978**, *55*(9), 564–566.

Augusto, S.; Maguas, C.; Branquinho, C., Guidelines for biomonitoring persistent organic pollutants (POPs), using lichens and aquatic mosses - A review. *Environ. Pollut.* **2013**, *180*, 330–338.

Barbour, J. P.; Smith, J. A.; Chiou, C. T., Sorption of aromatic organic pollutants to grasses from water. *Environ. Sci. Technol.* **2005**, *39*(21), 8369–8373.

Beattie, D. M., Life-cycle and changes in carbohydrates, proteins and lipids of *Pentapedilum uncinatum* Goet. (Diptera; Chironomidae). *Freshw. Biol.* **1978**, *8*(2), 109–113.

Beaumelle, L.; Lamy, I.; Cheviron, N.; Hedde, M., Is there a relationship between earthworm energy reserves and metal availability after exposure to field-contaminated soils? *Environ. Pollut.* **2014**, *191*, 182–189.

Belden, J. B.; Lotufo, G. R.; Chambliss, C. K.; Fisher, J. C.; Johnson, D. R.; Boyd, R. E.; Sims, J. G., Accumulation of (14)C-trinitrotoluene and related nonextractable (bound) residues in *Eisenia fetida*. *Environ. Pollut.* **2011**, *159*(5), 1363–1368.

Berglund, O.; Larsson, P.; Ewald, G.; Okla, L., Bioaccumulation and differential partitioning of polychlorinated biphenyls in freshwater, planktonic food webs. *Can. J. Fish. Aquat. Sci.* **2000**, *57*(6), 1160–1168.

Bischel, H. N.; MacManus-Spencer, L. A.; Luthy, R. G., Noncovalent interactions of long-chain perfluoroalkyl acids with serum albumin. *Environ. Sci. Technol.* **2010**, *44*(13), 5263–5269.

Bischel, H. N.; MacManus-Spencer, L. A.; Zhang, C. J.; Luthy, R. G., Strong association of short-chain perfluoroalkyl acids to serum albumin and investigation of binding mechanisms. *Environ. Toxicol. Chem.* **2011**, *30*(11), 2423–2430.

Bodin, N.; Le Loc'h, F.; Caisey, X.; Le Guellec, A. M.; Abarnou, A.; Loizeau, V.; Latrouite, D., Congener-specific accumulation and trophic transfer of polychlorinated biphenyls in spider crab food webs revealed by stable isotope analysis. *Environ. Pollut.* **2008**, *151*(1), 252–261.

Böhme, F.; Welsch-Pausch, K.; McLachlan, M. S., Uptake of airborne semivolatile organic compounds in agricultural plants: Field measurements of interspecies variability. *Environ. Sci. Technol.* **1999**, *33*(11), 1805–1813.

Borga, K.; Fjeld, E.; Kierkegaard, A.; McLachlan, M. S., Food web accumulation of cyclic siloxanes in Lake Mjosa, Norway. *Environ. Sci. Technol.* **2012**, *46*(11), 6347–6354.

Borga, K.; Fjeld, E.; Kierkegaard, A.; McLachlan, M. S., Consistency in trophic magnification factors of cyclic methyl siloxanes in pelagic freshwater food webs leading to brown trout. *Environ. Sci. Technol.* **2013**, *47*(24), 14394–14402.

Brett, J. R.; Groves, T. D. D., Physiological energetics. In *Fish Physiology* Hoar, W. S.; Randall, D. J.; Brett, J. R., Eds. Academic Press: New York, NY, 1979; Vol. 8, pp 279–352.

C.A. OEHHA, *Guide to Eating Fish and Shellfish from San Francisco Bay*; California Office of Environmental Health Hazard Assessment. http://www.oehha.ca.gov/fish/nor_cal/2011SFbay.html.

Carter, L. J.; Garman, C. D.; Ryan, J.; Dowle, A.; Bergstrom, E.; Thomas-Oates, J.; Boxall, A. B. A., Fate and uptake of pharmaceuticals in soil-earthworm systems. *Environ. Sci. Technol.* **2014**, *48*(10), 5955–5963.

Carvalho, P. N.; Basto, M. C. P.; Almeida, C. M. R.; Brix, H., A review of plant-pharmaceutical interactions: From uptake and effects in crop plants to phytoremediation in constructed wetlands. *Environ. Sci. Pollut. Res.* **2014**, *21*(20), 11729–11763.

Cavaletto, J. F.; Gardner, W. S., Seasonal dynamics of lipids in freshwater benthic invertebrates. In *Lipids in Freshwater Ecosystems*, Arts, M. T.; Wainman, B. C., Eds. Springer: New York, 1999; pp 109–131.

Chang, M. L.; Wang, M. J.; Kuo, D. T. F.; Shih, Y. H., Sorption of selected aromatic compounds by vegetables. *Ecol. Eng.* **2013**, *61*, 74-81.

Chen, B. L.; Johnson, E. J.; Chefetz, B.; Zhu, L. Z.; Xing, B. S., Sorption of polar and nonpolar aromatic organic contaminants by plant cuticular materials: Role of polarity and accessibility. *Environ. Sci. Technol.* **2005**, *39*(16), 6138–6146.

Chen, B. L.; Schnoor, J. L., Role of suberin, suberan, and hemicellulose in phenanthrene sorption by root tissue fractions of switchgrass (*Panicum virgatum*) seedlings. *Environ. Sci. Technol.* **2009**, *43*(11), 4130–4136.

Chen, B. L.; Yuan, M. X.; Liu, H., Removal of polycyclic aromatic hydrocarbons from aqueous solution using plant residue materials as a biosorbent. *J. Hazard. Mater.* **2011**, *188*(1-3), 436–442.

Collins, C. D.; Martin, I.; Doucette, W., Plant uptake of xenobiotics. In *Organic Xenobiotics and Plants: From Mode of Action to Ecophysiology*, Schroder, P.; Collins, C. D., Eds. Springer: Dordrecht, **2011**; Vol. 8, pp 3–16.

Cullon, D. L.; Yunker, M. B.; Christensen, J. R.; Macdonald, R. W.; Whiticar, M. J.; Dangerfield, N. J.; Ross, P. S., Biomagnification of polychlorinated biphenyls in a harbor seal (*Phoca vitulina*) food web from the strait of Georgia, British Columbia, Canada. *Environ. Toxicol. Chem.* **2012**, *31*(11), 2445–2455.

Daley, J. M.; Paterson, G.; Drouillard, K. G., Bioamplification as a bioaccumulation mechanism for persistent organic pollutants (POPs) in wildlife. In *Reviews of Environmental Contamination and Toxicology*, Whitacre, D. M., Ed. Springer: New York, **2014**; Vol. 227, pp 107–155.

Desalme, D.; Binet, P.; Chiapusio, G., Challenges in tracing the fate and effects of atmospheric polycyclic aromatic hydrocarbon deposition in vascular plants. *Environ. Sci. Technol.* **2013**, *47*(9), 3967–3981.

Dettenmaier, E. M.; Doucette, W. J.; Bugbee, B., Chemical hydrophobicity and uptake by plant roots. *Environ. Sci. Technol.* **2009**, *43*(2), 324–329.

Edwards, C. A.; Bohlen, P. J., *Biology and Ecology of Earthworms*. 3rd ed.; Chapman & Hall: London, 1995; p 426.

Endo, S., Helmholtz Center for Environmental Research (UFZ), Leipzig, Germany. Personal Communication. November 25, 2014.

Endo, S.; Bauerfeind, J.; Goss, K.-U., Partitioning of neutral organic compounds to structural proteins. *Environ. Sci. Technol.* **2012**, *46*(22), 12697–12703.

Endo, S.; Brown, T. N.; Goss, K. U., General model for estimating partition coefficients to organisms and their tissues using the biological compositions and polyparameter linear free energy relationships. *Environ. Sci. Technol.* **2013**, *47*(12), 6630–6639.

Endo, S.; Escher, B. I.; Goss, K. U., Capacities of membrane lipids to accumulate neutral organic chemicals. *Environ. Sci. Technol.* **2011**, *45*(14), 5912–5921.

Endo, S.; Goss, K. U., Serum albumin binding of structurally diverse neutral organic compounds: Data and models. *Chem. Res. Toxicol.* **2011**, *24*(12), 2293–2301.

Escher, B. I.; Ashauer, R.; Dyer, S.; Hermens, J. L. M.; Lee, J. H.; Leslie, H. A.; Mayer, P.; Meador, J. P.; Warne, M. S. J., Crucial role of mechanisms and modes of toxic action for understanding tissue residue toxicity and internal effect concentrations of organic chemicals. **2011**, *7*(1), 28–49.

Escher, B. I.; Hermens, J. L. M., Modes of action in ecotoxicology: Their role in body burdens, species sensitivity, QSARs, and mixture effects. *Environ. Sci. Technol.* **2002**, *36*(20), 4201–4217.

Escher, B. I.; Hermens, J. L. M., Internal exposure: Linking bioavailability to effects. *Environ. Sci. Technol.* **2004**, *38*(23), 455A–462A.

Escher, B. I.; Schwarzenbach, R. P., Partitioning of substituted phenols in liposome-water, biomembrane-water, and octanol-water systems. *Environ. Sci. Technol.* **1996**, *30*(1), 260–270.

Escher, B. I.; Schwarzenbach, R. P., Mechanistic studies on baseline toxicity and uncoupling of organic compounds as a basis for modeling effective membrane concentrations in aquatic organisms. *Aquat. Sci.* **2002**, *64*(1), 20–35.

Ewald, G., Role of lipids in the fate of organochlorine compounds in aquatic ecosystems. Ph.D. Thesis, Lund University, Lund Sweden, 1996.

Feldman, R. S.; Titus, J. E., Polychlorinated biphenyl accumulation differs among pumpkinseed sunfish during experimental field exposure: The role of invertebrate prey. *Aquat. Toxicol.* **2001**, *51*(4), 389–404.

Figueiredo, K.; Maenpaa, K.; Leppanen, M. T.; Kiljunen, M.; Lyytikainen, M.; Kukkonen, J. V. K.; Koponen, H.; Biasi, C.; Martikainen, P. J., Trophic transfer of polychlorinated biphenyls (PCB) in a boreal lake ecosystem: Testing of bioaccumulation models. *Sci. Total Environ.* **2014**, *466*, 690–698.

Focardi, S.; Fossi, C.; Leonzio, C.; Corsolini, S.; Parra, O., Persistent organochlorine residues in fish and water birds from the Biobio river, Chile. *Environ. Monit. Assess.* **1996**, *43*(1), 73–92.

Gaylor, M. O.; Harvey, E.; Hale, R. C., Polybrominated diphenyl ether (PBDE) accumulation by earthworms (*Eisenia fetida*) exposed to biosolids-, polyurethane foam microparticle-, and penta-BDE-amended soils. *Environ. Sci. Technol.* **2013**, *47*(23), 13831–13839.

Geisler, A.; Endo, S.; Goss, K. U., Partitioning of organic chemicals to storage lipids: Elucidating the dependence on fatty acid composition and temperature. *Environ. Sci. Technol.* **2012**, *46*(17), 9519–9524.

Gobas, F.; Morrison, H. A., Bioconcentration and biomagnification in the aquatic environment. In *Handbook of Property Estimation Methods for Chemicals*, Boethling, R. S.; Mackay, D., Eds. CRC Press: Boca Raton, FL, 2000; pp 189–231.

Gobas, F. A. P. C.; de Wolf, W.; Burkhard, L. P.; Verbruggen, E.; Plotzke, K., Revisiting bioaccumulation criteria for POPs and PBT assessments. *Integr. Enviro. Assess. Manage.* **2009**, *5*(4), 624–637.

Gobas, F. A. P. C.; Lahittete, J. M.; Garofalo, G.; Wan, Y. S.; Mackay, D., A novel method for measuring membrane–water partition coefficients of hydrophobic organic chemicals: Comparison with 1-octanol–water partitioning. *J. Pharm. Sci.* **1988**, *77*(3), 265–272.

Gobas, F. A. P. C.; Wilcockson, J. B.; Russell, R. W.; Haffner, G. D., Mechanism of biomagnification in fish under laboratory and field conditions. *Environ. Sci. Technol.* **1999**, *33*(1), 133–141.

Greizerstein, H. B.; Stinson, C.; Mendola, P.; Buck, G. M.; Kostyniak, P. J.; Vena, J. E., Comparison of PCB congeners and pesticide levels between serum and milk from lactating women. *Environ. Res.* **1999**, *80*(3), 280–286.

Gunatilleka, A. D.; Poole, C. F., Models for estimating the non-specific aquatic toxicity of organic compounds. *Anal. Commun.* **1999**, *36*(6), 235–242.

Harding, G. C.; LeBlanc, R. J.; Vass, W. P.; Addison, R. F.; Hargrave, B. T.; Pearre, S.; Dupuis, A.; Brodie, P. F., Bioaccumulation of polychlorinated biphenyls (PCBs) in the marine pelagic food

web, based on a seasonal study in the southern Gulf of St. Lawrence, 1976-1977. *Mar. Chem.* **1997**, *56*(3-4), 145-179.

Harmens, H.; Foan, L.; Simon, V.; Mills, G., Terrestrial mosses as biomonitors of atmospheric POPs pollution: A review. *Environ. Pollut.* **2013**, *173*, 245–254.

Henderson, R. J.; Tocher, D. R., The lipid composition and biochemistry of freshwater fish. *Prog. Lipid. Res.* **1987**, *26*(4), 281–347.

Heredia-Guerrero, J. A.; Benitez, J. J.; Dominguez, E.; Bayer, I. S.; Cingolani, R.; Athanassiou, A.; Heredia, A., Infrared and Raman spectroscopic features of plant cuticles: A review. *Front. Plant Sci.* **2014**, *5*, 14.

Holling, C. S.; Bailey, J. L.; Heuvel, B. V.; Kinney, C. A., Uptake of human pharmaceuticals and personal care products by cabbage (Brassica campestris) from fortified and biosolids-amended soils. *J. Environ. Monit.* **2012**, *14*(11), 3029–3036.

Hung, H.; Halsall, C. J.; Blanchard, P.; Li, H. H.; Fellin, P.; Stern, G.; Rosenberg, B., Are PCBs in the Canadian Arctic atmosphere declining? Evidence from 5 years of monitoring. *Environ. Sci. Technol.* **2001**, *35*(7), 1303–1311.

Hung, H. W.; Lin, T. F.; Chiou, C. T., Partition coefficients of organic contaminants with carbohydrates. *Environ. Sci. Technol.* **2010**, *44*(14), 5430–5436.

Hung, W. N.; Chiou, C. T.; Lin, T. F., Lipid-water partition coefficients and correlations with uptakes by algae of organic compounds. *J. Hazard. Mater.* **2014**, *279*, 197–202.

Hunt, J. M., *Petroleum Geochemistry and Geology*. W.H. Freeman and Company: San Francisco, 1979.

Huston, J. E.; Pinchak, W. E., Range animal nutrition. In *Grazing Management: An Ecological Perspective*, Heitschmidt, R. K.; Stuth, J. W., Eds. Timber Press: Portland, Oregon, 1991; pp 27–63.

Jude, D. J.; Rediske, R.; O'Keefe, J.; Hensler, S.; Giesy, J. P., PCB concentrations in walleyes and their prey from the Saginaw River, Lake Huron: A comparison between 1990 and 2007. *J. Gt. Lakes Res.* **2010**, *36*(2), 267–276.

Karaca, A., Ed., *Biology of Earthworms*. Springer-Verlag: Berlin, 2011; Vol. 24, p 316.

Kelly, B. C.; Gobas, F. A. P. C., Bioaccumulation of persistent organic pollutants in lichen-caribou-wolf food chains of Canada's Central and Western Arctic. *Environ. Sci. Technol.* **2001**, *35*(2), 325–334.

Kelly, B. C.; Ikonomou, M. G.; Blair, J. D.; Morin, A. E.; Gobas, F., Food web-specific biomagnification of persistent organic pollutants. *Science* **2007**, *317*(5835), 236–239.

Kierkegaard, A.; Asplund, L.; de Wit, C. A.; McLachlan, M. S.; Thomas, G. O.; Sweetman, A. J.; Jones, K. C., Fate of higher brominated PBDEs in lactating cows. *Environ. Sci. Technol.* **2007**, *41*(2), 417–423.

Kierkegaard, A.; van Egmond, R.; McLachlan, M. S., Cyclic volatile methylsiloxane bioaccumulation in flounder and ragworm in the Humber Estuary. *Environ. Sci. Technol.* **2011**, *45*(14), 5936–5942.

Kinney, C. A.; Campbell, B. R.; Thompson, R.; Furlong, E. T.; Kolpin, D. W.; Burkhardt, M. R.; Zaugg, S. D.; Werner, S. L.; Hay, A. G., Earthworm bioassays and seedling emergence for monitoring toxicity, aging and bioaccumulation of anthropogenic waste indicator compounds in biosolids-amended soil. *Sci. Total Environ.* **2012**, *433*, 507–515.

Kinney, C. A.; Furlong, E. T.; Kolpin, D. W.; Burkhardt, M. R.; Zaugg, S. D.; Werner, S. L.; Bossio, J. P.; Benotti, M. J., Bioaccumulation of pharmaceuticals and other anthropogenic waste indicators in earthworms from agricultural soil amended with biosolid or swine manure. *Environ. Sci. Technol.* **2008**, *42*(6), 1863–1870.

Kucklick, J. R.; Baker, J. E., Organochlorines in Lake Superior's food web. *Environ. Sci. Technol.* **1998**, *32*(9), 1192–1198.

Kühnert, A.; Vogs, C.; Altenburger, R.; Kuster, E., The internal concentration of organic substances in fish embryosA toxicokinetic approach. *Environ. Toxicol. Chem.* **2013**, *32*(8), 1819–1827.

Kwon, J. H.; Liljestran, H. M.; Katz, L. E.; Yamamoto, H., Partitioning thermodynamics of selected endocrine disruptors between water and synthetic membrane vesicles: Effects of membrane compositions. *Environ. Sci. Technol.* **2007**, *41*(11), 4011–4018.

Lawrence, R. D.; Millar, H. R., Protein content of earthworms. *Nature* **1945**, *155*(3939), 517-517.

LeBlanc, G. A., Trophic-level differences in the bioconcentration of chemicals: Implications in assessing environmental biomagnification. *Environ. Sci. Technol.* **1995**, *29*(1), 154–160.

Li, J. J.; Tai, H. W.; Yu, Y.; Wen, Y.; Wang, X. H.; Zhao, Y. H., Comparison of toxicity of class-based organic chemicals to algae and fish based on discrimination of excess toxicity from baseline level. *Environ. Toxicol. Pharmacol.* **2015**, *40*(1), 292–299.

Li, Y. G.; Chen, B. L., Phenanthrene sorption by fruit cuticles and potato periderm with different compositional characteristics. *J. Agric. Food Chem.* **2009**, *57*(2), 637–644.

Li, Y. G.; Deng, Y. Q.; Chen, B. L., Sorption of chlorophenols onto fruit cuticles and potato periderm. *J. Environ. Sci.* **2012**, *24*(4), 675-681.

Lü, H. X.; Cai, Q. Y.; Jones, K. C.; Zeng, Q. Y.; Katsoyiannis, A., Levels of organic pollutants in vegetables and human exposure through diet: A review. *Crit. Rev. Environ. Sci. Technol.* **2014**, *44*(1), 1–33.

Ma, W. C.; van Kleunen, A.; Immerzeel, J.; de Maagd, P. G. J., Bioaccumulation of polycyclic aromatic hydrocarbons by earthworms: Assessment of equilibrium partitioning theory in in situ studies and water experiments. *Environ. Toxicol. Chem.* **1998**, *17*(9), 1730–1737.

MacDonald, R.; MacKay, D.; Hickie, B., A contaminant amplification in the environment. *Environ. Sci. Technol.* **2002**, *36*(23), 456A–462A.

Mackay, A. A.; Gschwend, P. M., Sorption of monoaromatic hydrocarbons to wood. *Environ. Sci. Technol.* **2000**, *34*(5), 839–845.

Mackay, D., Finding fugacity feasible. *Environ. Sci. Technol.* **1979**, *13*(10), 1218–1223.

MacKay, D., Finding fugacity feasible, fruitful, and fun. *Environ. Toxicol. Chem.* **2004**, *23*(10), 2282–2289.

Mackay, D.; Arnot, J. A.; Celsie, A.; Orazietti, A.; Parnis, J. M., QSARs for aquatic toxicity: Celebrating, extending and displaying the pioneering contributions of Ferguson, Konemann and Veith. *SAR QSAR Environ. Res.* **2014b**, *25*(5), 343–355.

Mackay, D.; McCarty, L. S.; Arnot, J. A., Relationships between exposure and dose in aquatic toxicity tests for organic chemicals. *Environ. Toxicol. Chem.* **2014a**, *33*(9), 2038–2046.

Maeder, V.; Escher, B. I.; Scheringer, M.; Hungerbuhler, K., Toxic ratio as an indicator of the intrinsic toxicity in the assessment of persistent, bioaccumulative, and toxic chemicals. *Environ. Sci. Technol.* **2004**, *38*(13), 3659–3666.

Marc, M.; Tobiszewski, M.; Zabiegala, B.; de la Guardia, M.; Namiesnik, J., Current air quality analytics and monitoring: A review. *Anal. Chim. Acta* **2015**, *853*, 116–126.

Martin, L. B. B.; Rose, J. K. C., There's more than one way to skin a fruit: Formation and functions of fruit cuticles. *J. Exp. Bot.* **2014**, *65*(16), 4639–4651.

Mathews, S.; Henderson, S.; Reinhold, D., Uptake and accumulation of antimicrobials, triclocarban and triclosan, by food crops in a hydroponic system. *Environ. Sci. Pollut. Res.* **2014**, *21*(9), 6025–6033.

McLachlan, M. S., Bioaccumulation of hydrophobic chemicals in agricultural feed chains. *Environ. Sci. Technol.* **1996**, *30*(1), 252–259.

Morrison, H. A.; Gobas, F. A. P. C.; Lazar, R.; Haffner, G. D., Development and verification of a bioaccumulation model for organic contaminants in benthic invertebrates. *Environ. Sci. Technol.* **1996**, *30*(11), 3377–3384.

Nalepa, T. F.; Cavaletto, J. F.; Ford, M.; Gordon, W. M.; Wimmer, M., Seasonal and annual variation in weight and biochemical content of the zebra mussel, *Dreissena polymorphia,* in Lake St. Clair. *J. Gt. Lakes Res.* **1993**, *19*(3), 541–552.

Neidhardt, F. G.; Ingraham, J. L.; Schaechter, M., Ed., *Physiology of the Bacterial Cell. A Molecular Approach*. Sinauer Associates: Sunderland, M.A., 1990.

Netzeva, T. I.; Pavan, M.; Worth, A. P., Review of (quantitative) structure-activity relationships for acute aquatic toxicity. *QSAR Comb. Sci.* **2008**, *27*(1), 77–90.

Nichols, J. W.; Huggett, D. B.; Arnot, J. A.; Fitzsimmons, P. N.; Cowan-Ellsberry, C. E., Toward improved models for predicting bioconcentration of well-metabolized compounds by rainbow trout using measured rates of in vitro intrinsic clearance. *Environ. Toxicol. Chem.* **2013**, *32*(7), 1611–1622.

Nyman, A. M.; Schirmer, K.; Ashauer, R., Importance of toxicokinetics for interspecies variation in sensitivity to chemicals. *Environ. Sci. Technol.* **2014**, *48*(10), 5946–5954.

Pannu, M. W.; O'Connor, G. A.; Toor, G. S., Toxicity and bioaccumulation of biosolids-borne triclosan in terrestrial organisms. *Environ. Toxicol. Chem.* **2012**, *31*(3), 646–653.

Parsons, T. R.; Takahashi, M., *Biological Oceanographic Processes*. Pergamon Press: Oxford, 1973.

Paterson, M. J.; Muir, D. C. G.; Rosenberg, B.; Fee, E. J.; Anema, C.; Franzin, W., Does lake size affect concentrations of atmospherically derived polychlorinated biphenyls in water, sediment, zooplankton, and fish? *Can. J. Fish. Aquat. Sci.* **1998**, *55*(3), 544–553.

Peverly, A. A.; Salamova, A.; Hites, R. A., Air is still contaminated 40 years after the Michigan Chemical Plant Disaster in St. Louis, Michigan. *Environ. Sci. Technol.* **2014**, *48*(19), 11154–11160.

Pollard, M.; Beisson, F.; Li, Y. H.; Ohlrogge, J. B., Building lipid barriers: Biosynthesis of cutin and suberin. *Trends Plant Sci.* **2008**, *13*(5), 236–246.

Prosser, R. S.; Trapp, S.; Sibley, P. K., Modeling uptake of selected pharmaceuticals and personal care products into food crops from biosolids-amended soil. *Environ. Sci. Technol.* **2014**, *48*(19), 11397–11404.

Qi, Y. C.; Chen, W., Comparison of earthworm bioaccumulation between readily desorbable and desorption-resistant naphthalene: Implications for biouptake routes. *Environ. Sci. Technol.* **2010**, *44*(1), 323–328.

Rodriguez-Campos, J.; Dendooven, L.; Alvarez-Bernal, D.; Contreras-Ramos, S. M., Potential of earthworms to accelerate removal of organic contaminants from soil: A review. *Appl. Soil Ecol.* **2014**, *79*, 10–25.

Rombauer, I. S.; Becker, M. R.; Becker, F., *Joy of Cooking*. Scribner: New York, NY, 1997.

Russell, R. W.; Lazar, R.; Haefner, G. D., Biomagnification of organochlorines in Lake Erie white bass. *Environ. Toxicol. Chem.* **1995**, *14*(4), 719–724.

Ruus, A.; Ugland, K. I.; Espeland, O.; Skaare, J. U., Organochlorine contaminants in a local marine food chain from Jarfjord, Northern Norway. *Mar. Environ. Res.* **1999**, *48*(2), 131–146.

Saarikoski, J.; Viluksela, M., Relation between physicochemical properties of phenolics and their toxicites and accumulation in fish. *Ecotoxicol. Environ. Safety* **1982**, *6*(501–512).

Salamova, A.; Hites, R. A., Brominated and chlorinated flame retardants in tree bark from around the globe. *Environ. Sci. Technol.* **2013**, *47*(1), 349–354.

Schmidt, S. N.; Holmstrup, M.; Smith, K. E. C.; Mayer, P., Passive dosing of polycyclic aromatic hydrocarbon (PAH) mixtures to terrestrial springtails: Linking mixture toxicity to chemical activities, equilibrium lipid concentrations, and toxic units. *Environ. Sci. Technol.* **2013**, *47*(13), 7020–7027.

Shan, J.; Wang, T.; Li, C. L.; Klumpp, E.; Ji, R., Bioaccumulation and bound-residue formation of a branched 4-nonylphenol isomer in the geophagous earthworm *Metaphire guillelmi* in a rice paddy soil. *Environ. Sci. Technol.* **2010**, *44*(12), 4558–4563.

Shaw, S. D.; Berger, M. L.; Brenner, D.; Kannan, K.; Lohmann, N.; Papke, O., Bioaccumulation of polybrominated diphenyl ethers and hexabromocyclododecane in the northwest Atlantic marine food web. *Sci. Total Environ.* **2009**, *407*(10), 3323–3329.

Shechter, M.; Chefetz, B., Insights into the sorption properties of cutin and cutan biopolymers. *Environ. Sci. Technol.* **2008**, *42*(4), 1165–1171.

Shechter, M.; Xing, B. S.; Chefetz, B., Cutin and cutan biopolymers: Their role as natural sorbents. *Soil Sci. Soc. Am. J.* **2010**, *74*(4), 1139–1146.

Shifrin, N. S.; Chisholm, S. W., Phytoplankton lipids: Interspecific differences and effects of nitrate, silicate and light-dark cycles. *J. Phycol.* **1981**, *17*(4), 374–384.

Sijm, D. T. H. M.; Hermens, J. L. M., Internal effect concentration: Link between bioaccumulation and ecotoxicity for organic chemicals. In *The Handbook of Environmental Chemistry*, Beek, B., Ed. Springer: Berlin **2000**; Vol. 2J, pp 167–199.

Sjöström, E., *Wood chemistry: Fundamentals and applications*. 2nd ed.; Academic Press: San Diego, CA, 1993; p 293.

Stadnicka, J.; Schirmer, K.; Ashauer, R., Predicting concentrations of organic chemicals in fish by using toxicokinetic models. *Environ. Sci. Technol.* **2012**, *46*(6), 3273–3280.

Stange, K.; Swackhamer, D. L., Factors affecting phytoplankton species-specific differnences in accumulation of 40 polychlorinated biphenyls (PCBs). *Environ. Toxicol. Chem.* **1994**, *13*(11), 1849–1860.

Stapleton, H. M.; Alaee, M.; Letcher, R. J.; Baker, J. E., Debromination of the flame retardant decabromodiphenyl ether by juvenile carp (*Cyprinus carpio*) following dietary exposure. *Environ. Sci. Technol.* **2004**, *38*(1), 112–119.

Sturla, S. J.; Boobis, A. R.; FitzGerald, R. E.; Hoeng, J.; Kavlock, R. J.; Schirmer, K.; Whelan, M.; Wilks, M. F.; Peitsch, M. C., Systems Toxicology: From Basic Research to Risk Assessment. *Chem. Res. Toxicol.* **2014**, *27*(3), 314–329.

Su, L. M.; Liu, X.; Wang, Y.; Li, J. J.; Wang, X. H.; Sheng, L. X.; Zhao, Y. H., The discrimination of excess toxicity from baseline effect: Effect of bioconcentration. *Sci. Total Environ.* **2014**, *484*, 137–145.

Takaki, K.; Wade, A. J.; Collins, C. D., Assessment of plant uptake models used in exposure assessment tools for soils contaminated with organic pollutants. *Environ. Sci. Technol.* **2014**, *48*(20), 12073–12082.

Thomann, R. V.; Connolly, J. P., Model of PCB in the Lake Michigan lake trout food chain. *Environ. Sci. Technol.* **1984**, *18*(2), 65–71.

Tolls, J.; McLachlan, M. S., Partitioning of semivolatile organic compounds between air and Lolium multiflorum (Welsh ray grass). *Environ. Sci. Technol.* **1994**, *28*(1), 159–166.

Tomy, G. T.; Pleskach, K.; Ferguson, S. H.; Hare, J.; Stern, G.; Macinnis, G.; Marvin, C. H.; Loseto, L., Trophodynamics of some PFCs and BFRs in a Western Canadian Arctic marine food web. *Environ. Sci. Technol.* **2009**, *43*(11), 4076–4081.

Turner, J. W.; Hartman, B. E.; Hatcher, P. G., Structural characterization of suberan isolated from river birch (Betula nigra) bark. *Org. Geochem.* **2013**, *57*, 41–53.

U.S. EPA, *2011 National Listing of Fish Advisories*; EPA-820-F-13-058. 2013. http://www.epa.gov/fish-tech/national-listing-fish-advisories-2011-data.

van der Vusse, G. J., Albumin as fatty acid transporter. *Drug Metab. Pharmacokinet.* **2009**, *24*(4), 300-307.

van Wezel, A.; Punte, S. S.; Opperhuizen, A., Lethal body burdens of polar narcotics: Chlorophenols. *Environ. Toxicol. Chem.* **1995**, *14*(9), 1579–1585.

Verhaar, H. J. M.; Vanleeuwen, C. J.; Hermens, J. L. M., Classifying environmental pollutants. 1: Structure-activity relationships for prediction of aquatic toxicity. *Chemosphere* **1992**, *25*(4), 471–491.

Walters, D. M.; Mills, M. A.; Cade, B. S.; Burkard, L. P., Trophic magnification of PCBs and its relationship to the octanol-water partition coefficient. *Environ. Sci. Technol.* **2011**, *45*(9), 3917–3924.

Wang, F.; Ji, R.; Jiang, Z. W.; Chen, W., Species-dependent effects of biochar amendment on bioaccumulation of atrazine in earthworms. *Environ. Pollut.* **2014**, *186*, 241–247.

Wang, X. L.; Cook, R.; Tao, S.; Xing, B. S., Sorption of organic contaminants by biopolymers: Role of polarity, structure and domain spatial arrangement. *Chemosphere* **2007**, *66*(8), 1476–1484.

Wang, X. L.; Xing, B. S., Importance of structural makeup of biopolymers for organic contaminant sorption. *Environ. Sci. Technol.* **2007**, *41*(10), 3559–3565.

Wania, F.; McLachlan, M. S., Estimating the influence of forests on the overall fate of semivolatile organic compounds using a multimedia fate model. *Environ. Sci. Technol.* **2001**, *35*(3), 582–590.

Weston, D. P.; Penry, D. L.; Gulmann, L. K., The role of ingestion as a route of contaminant bioaccumulation in a deposit-feeding polychaete. *Arch. Environ. Contam. Toxicol.* **2000**, *38*(4), 446–454.

White, J. C.; Newman, L. A., Phytoremediation of soils contaminated with organic pollutants. In *Biophysico-Chemical Processes of Anthropogenic Organic Compounds in Environmental Systems*, Xing, B.; Senesi, N.; Huang, P. M., Eds. John Wiley & Sons Inc: Hoboken, 2011; pp 503–516.

Xiao, R. Y.; Arnot, J. A.; MacLeod, M., Towards an improved understanding of processes controlling absorption efficiency and biomagnification of organic chemicals by fish. *Chemosphere* **2015**, *138*, 89–95.

Yang, R. Q.; Zhang, S. J.; Li, A.; Jiang, G. B.; Jing, C. Y., Altitudinal and spatial signature of persistent organic pollutants in soil, lichen, conifer needles, and bark of the Southeast Tibetan Plateau: Implications for sources and environmental cycling. *Environ. Sci. Technol.* **2013**, *47*(22), 12736–12743.

Yavari, S.; Malakahmad, A.; Sapari, N. B., A review on phytoremediation of crude oil spills. *Water Air Soil Pollut.* **2015**, *226*(8), 18.

Zhao, Y. L.; Yang, L. M.; Wang, Q. Q., Modeling persistent organic pollutant (POP) partitioning between tree bark and air and its application to spatial monitoring of atmospheric POPs in mainland China. *Environ. Sci. Technol.* **2008**, *42*(16), 6046–6051.

Zhu, Y. H.; Zhang, S. Z.; Zhu, Y. G.; Christie, P.; Shan, X. Q., Improved approaches for modeling the sorption of phenanthrene by a range of plant species. *Environ. Sci. Technol.* **2007**, *41*(22), 7818–7823.