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A FOOD CHAIN MODEL TO PREDICT THE LEVELS OF LIPOPHILIC ORGANIC CONTAMINANTS IN HUMANS

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Abstract—A fugacity-based, nonsteady state, mechanistic model called ACC-HUMAN was developed to describe bioaccumulation of lipophilic organic pollutants from air, water, and soil to humans. The physical environment was linked via a marine and an agricultural food chain model to a human bioaccumulation model. Contaminant uptake via the primary dietary sources of persistent lipophilic contaminants in industrialized countries was addressed, namely fish, dairy products, and beef. In addition, uptake from air and water was considered, allowing the model also to treat less lipophilic compounds. To evaluate the model, the food chain characteristics were parameterized for southern Sweden and historical scenarios of polychlorinated biphenyl (PCB) concentrations in air, water, and soil in this region were constructed from published data. The resulting model predictions of PCB concentrations in fish, milk, beef, and human tissue agreed well with measured concentrations from Swedish monitoring programs. This suggests that ACC-HUMAN is a useful tool for predicting human exposure to bioaccumulative organic compounds. It can be linked easily to existing multimedia fate and transport models.

Keywords—Food chain model  Bioaccumulation  Polychlorinated biphenyls  Human exposure  Persistent organic pollutants

INTRODUCTION

Persistent lipophilic organic pollutants (PLOPs) have a strong tendency to bioaccumulate in marine and terrestrial food webs. Humans, being at the top of the food web, particularly are exposed to PLOPs. Predicting the external and internal human exposure that will result from a given level of environmental contamination is a central component of contaminant risk assessment.

This task requires an understanding of the transfer of chemicals through the food web to humans. Substantial progress has been made in the development of predictive models, and Don Mackay has played a prominent role in this effort. His central contributions include insisting on a linear relationship between the fish/water bioconcentration factor and the octanol/water partition coefficient [1], which he then incorporated into simple useful models of fish bioaccumulation [2], with Frank Gobas developing a process-based and mathematically underpinned explanation for dietary uptake and biomagnification in fish [3]; pursuing fugacity as a measure of biomagnification [4]; writing one of the first models of organic chemical uptake in plants [5], one of the first nonsteady state models for mammals [6], and the first pharmacokinetic model targeted at describing organic contaminant fate in humans [7]; and creating the fugacity modeling framework, which allows all of these elements to be linked together in an elegant and insightful fashion [8]. This fruitful legacy forms the foundation for the work presented in this paper.

The first model to predict organic chemical exposure to humans via the environment that we are aware of was GEO-TOX, published by Tom McKone in 1986 [9]. This pioneering work flowed into a successor model, CALTOX, and influenced the second major model to address human exposure, USES, and the European Union System for the Evaluation of Substances (EUSES), which is used in the regulation of chemicals in the European Union [10]. For many processes, these models had to rely on simple empirical correlations with no mechanistic basis and, as studies with EUSES have shown, this significantly limited their range of application and predictive capability. Furthermore, the degree of validation of these models is low, in part because of the paucity of high quality data sets giving contaminant levels in the full spectrum of environmental matrices in a given area as well as in the tissue of the local population [11,12].

In this paper we use recent advances in scientific understanding of bioaccumulation in agricultural and aquatic food chains, as well as in humans, to formulate a mechanistically based model called ACC-HUMAN that predicts human tissue levels of organic contaminants starting from concentrations in air, water, and soil. The predictive ability of the model is then evaluated using polychlorinated biphenyls in the Swedish environment as a case study.

MODEL DESCRIPTION

The ACC-HUMAN model structure is shown in Figure 1. The model is subdivided into an agricultural and a marine system, each represented by one food chain. The top predator linking both systems is the human being. This structure was chosen to reflect the primary pathways of human exposure to persistent bioaccumulating chemicals in northern Europe, but it also can be adapted to other regions.

In northern Europe, shellfish generally are a minor vector of human exposure compared to fish. Furthermore, the major fish species harvested in the Baltic Sea (herring, cod) feed little on benthic organisms. Therefore, a simple pelagic food
Food chain model to predict contaminant levels in humans

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The compartment (m$^3$), viewed as the partial pressure of the chemical in the phase, or more abiotic compartments in the environment (air, water, soil) and with the compartments of the next link below it in the food chain. A mass balance of the form is written for each compartment, whereby the fugacity capacity of a given phase to store a given chemical.

In northern Europe, beef and dairy products are the dominant vectors of persistent bioaccumulating organic compounds from the terrestrial environment to humans [13–15]. The importance of beef and dairy products compared to fruit and vegetables can be attributed to the large quantity of herbage that must be fed to cattle to produce a given quantity of milk or meat. Exposure from the terrestrial environment, therefore, is modeled with an air/soil–grass–cow/steer food chain.

The ACC-HUMAN is based on the fugacity approach pioneered by Mackay [16]. The concentration $C$ (mol m$^{-3}$) is expressed as the product of a fugacity $f$ (Pa), which can be viewed as the partial pressure of the chemical in the phase, and a fugacity capacity $Z$ (mol m$^{-3}$ Pa$^{-1}$), a measure of the capacity of a given phase to store a given chemical.

In the model, each link in the food chain is treated as one or several homogenous compartments. Each compartment generally is a mixture of several phases (e.g., water and lipid for mammals) that are assumed to be at equilibrium with each other. The fugacity capacity of the compartment is calculated as the volume weighted sum of the fugacity capacity of the phases.

$$Z = \sum_i (v_i \cdot Z_i)$$  (1)

where $v$ represents the volume fraction of the phase $i$ in the compartment (m$^3$ m$^{-3}$). The fugacity capacities of the phases air, water, soil, and lipid are calculated according to Mackay [16] [Table 1, SETAC Supplemental Data Archive, Item ETC-23-10-003; http://etc.allenpress.com]. The fugacity capacity of lipids in fish and mammals is assumed equal to the fugacity capacity of 1-octanol. The fugacity capacity generally is very sensitive to temperature; it is calculated at the temperature of the compartment (37°C for mammals, ambient temperature for plants and poikilothermal animals) using the appropriate heats of phase change.

Each food chain compartment is interconnected with one or more abiotic compartments in the environment (air, water, soil) and with the compartments of the next link below it in the food chain. A mass balance of the form

$$\frac{d(VZF)}{dt} = \sum_i (D_{ij} f_i)$$  (2)

is written for each compartment, whereby $V$ is the volume of the compartment (m$^3$), $f$ the fugacity (Pa), and $D$ is a transport parameter (mol h$^{-1}$ Pa$^{-1}$), the first index of the suffix identifying the compartment from which the chemical is coming from, the second its destination. The $D$-value is the product of a volume flow rate $G$ with units of m$^3$ h$^{-1}$ and a fugacity capacity. The resulting sets of first order differential equations are solved in a step-wise fashion based on a set of initial conditions and boundary conditions defined by (varying) environmental parameters and chemical fugacities in the abiotic compartments.

**The marine food chain**

*Plankton.* Although to some extent still controversial, there is a growing body of experimental evidence indicating that a partitioning equilibrium rapidly is established between phytoplankton and the water column, even for lipophilic contaminants [17–20]. Furthermore, field studies indicate that no biomagnification occurs between phytoplankton and zooplankton [21–23]. Unfortunately there is little information available on the equilibration kinetics in zooplankton, an issue that is complicated by the great variability in the physiological properties and life cycles of different zooplankton species. In this model we have adopted the approach of Sharpe and Mackay [24] and assumed that the zooplankton is in equilibrium with the water column.

$$f_{zo} = f_w$$  (3)

Note that this assumption renders superfluous a description of phytoplankton uptake in the model.

*Fish.* The bioaccumulation in fish is described using a non-steady state model according to Gobas et al. [3]. The contaminant mass balance is given by the equation

$$\frac{d(VZF_{Z_f})}{dt} = D_{venF} f_w + E_{OF} \sum_i (D_{UF_i} f_{UF_i})$$

$$- \left( D_{vent} + D_{MF} + \frac{1}{Q_f} E_{OF} \sum D_{UF_i} \right) \cdot f_f$$  (4)

where the subscripts $F$, $W$, $venF$, $MF$, and $UF$ stand for fish, water, ventilation, metabolism (in fish), and food, respectively, and $i$ refers to the different prey in the food of the fish. The $E_{OF}$ is the gut absorption efficiency (fraction of the ingested chemical that is absorbed, dimensionless) and $Q_f$ is the ratio of the $D$-values for ingestion and egestion. In the discrete integration of this model, growth of the fish is accounted for by varying $V_f$ and $Z_f$ (see below, this section).

The model has ten boxes for the piscivorous as well as for the planktivorous fish. Each box represents one age class. On March 1st the fish enter the next age class (i.e., box) and a new generation is created, initially as eggs. The fugacity of fish egg (i.e., the initial fugacity $f_{fe}$ of the fish) is assumed to be equal to the fugacity of the mother fish (the mean fugacity of the mature fish weighted according to the age distribution of fish in the study area). For model initiation (i.e., when there are no mother fish) the fugacity of the fish egg is set equal to the fugacity of water.

The $D$-values are defined in Table 2; the fugacity capacity and the transport parameters on which the $D$-values are based are defined in Tables 1 and 3 (SETAC Supplemental Data Archive), respectively. The $D$-value for chemical uptake from food, $D_{up}$, is dependent on the food consumption rate $G_{up}$ (m$^3$ h$^{-1}$) and the fugacity capacity of the prey $Z_{UF}$ (mol m$^{-3}$ Pa$^{-1}$). The $G_{up}$ is allocated to the different prey according to the prey preferences of the fish using...
where $v_{c_i}$ is the volume of prey $i$ consumed divided by the total volume of prey consumed. The food consumption rate $G_{vi}$ is dependent on the fish species, age, and season. In all cases, $Z_{c_i}$ is set equal to the fugacity capacity of the organism as calculated elsewhere in the model. The prey fugacity $F_{c_i}$ is equal to the fugacity calculated for the respective organism in the previous time step of the model.

The terrestrial food chain

Grass. Grass is the main pathway of background exposure of cattle to PLOPs [25]. Contaminants are transferred to grass from the atmosphere and from soil; for hydrophobic compounds, the former generally dominates and for hydrophilic compounds, the latter. The grass model is written to describe uptake in a 1-m² plot of pasture. The atmospheric deposition of gaseous- and particle-bound contaminants to grass are treated separately. The mass balance for the grass is defined as

$$\frac{d(V_o Z_v G_o)}{dt} = D_{G_o}(f_A - f_o) + D_{BG} f_A + D_{PG} f_A - D_{BG} f_o$$

where the subscripts $G$, $A$, and $S$ refer to grass, air, and soil, respectively. The $D_{G_o}$ is the $D$-value (mol Pa⁻¹ h⁻¹) describing exchange of gaseous- and particle-bound contaminants to grass and the atmosphere. $D_{PG}$ is for deposition of aerosol-associated chemical on the grass, $D_{BG}$ accounts for reaction of the chemical on/in the grass, and $D_{BG}$ describes contaminant uptake via transpiration/-root uptake.

The equations for the $D$-values are given in Table 2 (SETAC Supplemental Data Archive). Root uptake is treated as an in-flow of water equal to the grass’s transpiration rate, which is corrected for by the Transpiration Stream Concentration Factor defined in [26] [Table 3, SETAC Supplemental Data Archive, Item ETC-23-10-003; http://etc.allenpress.com]. Gaseous exchange is defined using a two resistance model as in [27]. Deposition of aerosol-associated chemical is calculated using an average net deposition velocity.

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The grass can be harvested up to five times during the growing season. The length of the growing season, the time points for the harvest, and the yield of each harvest can be specified by the user. Linear growth is assumed. The yield-weighted mean contaminant concentration in the harvested grass is determined after the last harvest. This is used as the concentration in cattle feed for the following 12-month period.

Milk cows. The milk cow model is based on [28]. The cow is treated as two compartments: The digestive tract and the cow itself. In addition to the contaminant transport processes described in [28] (dietary ingestion, dietary egestion, transformation, and lactation), this model also includes inhalation, exhalation, and urination. It is assumed that the cow is at steady state. It has been shown that this is a reasonable assumption even for lipophilic contaminants due to the comparatively large rate of lipid excretion via lactation compared to the quantity of lipid in the cow [29]. With these assumptions the mass balance equations for the two compartments can be reduced to the following equation:

$$E_{OC} \sum (D_{UCC} f_{UC}) + D_{UC} f_A = [(1 - E_{OC}) \cdot D_{UC} + D_{UC} \\
+ D_{LC} + D_{MC} + D_{UC}) \cdot f_c$$

where $E_{OC}$ is the dietary absorption efficiency, and the subscripts $UC$, ibIC, reC, LC, MC, and urC refer to dietary uptake, intestinal tract–cow exchange, respiration, lactation, metabolism, and urination, respectively.

The $D$-values are defined in Table 2 [SETAC Supplemental Data Archive, Item ETC-23-10-003; http://etc.allenpress.com]. The transfer across the wall of the intestinal tract is described using a two-film model as described in [28]. Air–lung exchange is treated in accordance with [6]; 70% of the inhaled air is assumed to come into intimate contact with the alveoli and equilibrate with the cow at 37°C. Urination and lactation are treated as advective processes; the fugacity capacities are set equal to those of water and a water/lipid mixture, respectively.

The dietary intake includes contributions from feed, soil, and water. The feed uptake is equal to the grass ration multiplied by the grass concentration (see above, this section). A further 25% is added to this sum to account for the contribution of other feeds to the contaminant load [30,31]. Cows also ingest considerable quantities of soil, either on the pasture or as a result of soil in the harvested feed [32,33]. The water and soil fugacities are external variables. The composition of the dietary intake is assumed to be constant during the year.

Beef cattle. For nonlactating cattle, steady state behavior cannot be assumed for persistent contaminants [34]. In analogy to Equation 7, the mass balance equation is given by

$$\frac{d(V_o Z_v G_o)}{dt} = E_{EB} \sum (D_{UB} f_{UB}) + D_{EB} f_A - [(1 - E_{EB}) \cdot D_{EB} + D_{reB} + D_{ASB} + D_{urB}) \cdot f_B$$

where the subscript B refers to beef.

The steer is born on the day of the last grass harvest of the year with a fugacity equal to the fugacity of cow’s milk $f_c$. The steer is then fed for 28 months until slaughter. The fugacity in meat is assigned the fugacity $f_B$ at the point of slaughter. The volume of the steer $V_o$ is assumed to increase linearly with time. The $U_o$, $D_{ASB}$, $D_{urB}$, and $D_{ASB}$ are defined similarly as for cows, the differences being that the quantity of feed eaten $G_{EB}$ and the respiration rate $G_{reB}$ increase linearly with age of the animal and that the volume in $D_{ASB}$ is not constant but equal to $V_o$.

Humans. The aim of ACC-HUMAN is to describe long-term human exposure to bioaccumulating organic compounds. Therefore it was decided not to address the kinetic aspects of contaminant distribution in the body. A two compartment model similar to that of the milk cow was used, which presumes equilibrium distribution of the chemical in different body tissues [35]. This approach is supported by experimental studies indicating equilibrium partitioning of PLOPs between blood and slowly perfused tissue (e.g., [36]) and between blood and human milk ([37]), as well as by more detailed pharmacokinetic models of PLOPs in mammals indicating near equilibrium distribution among different tissues [6]. The human tissue compartment was modeled as a mixture of water and lipids; binding of contaminants to proteins was not treated.

The model considers contaminant uptake via diet and inhalation, and contaminant elimination via metabolism, percutaneous excretion, digestive tract excretion, exhalation, and, in the case of women, childbirth and nursing. It is based on the digestive tract absorption model in [38], which was ex-
tended to include the other processes listed above. The mass balance equation is

$$\frac{d(V_i z_i f_i)}{dt} = E_{Oli} \sum (D_{Cbi}, f_{Ubi}) + D_{Mhi} f_i$$

$$- (E_{Oli} D_{EH} + D_{reh} + D_{preh} + D_{Mih} + D_{Lh})$$

$$+ D_{nih} + D_{mah}) \cdot f_i$$

(9)

where $D_{Cbi}$ is the $D$-value describing uptake of the chemical from food type $i$, $f_{Ubi}$ is the fugacity of the chemical in the food $i$, $E_{Oli}$ is the efficiency of gastrointestinal absorption of the chemical (Table 4, SETAC Supplemental Data Archive), and the subscripts EH, reH, perH, MH, LH, chH, and urH refer to chemical transfer via egestion, respiration, percutaneous excretion, metabolism, nursing, childbirth, and urine excretion.

The $D$-values are defined in Table 2 (SETAC Supplemental Data Archive). The consumption rate of each food group $G_{Ubi}$ is specified by the user for fish (fresh vol), dairy products (lipid vol), and beef (lipid vol). The $Z$-value for each food is determined from the $Z$-values of lipids and water using Equation 1. Based on the work of Moser and McLachlan [38], dietary egestion is calculated as the product of the feces/blood lipid partition coefficient of the chemical $K_{FB}$ ($\text{m}^3 \text{blood lipid}$/[g dry feces]), the feces dry weight excretion rate $G'_{fb}$ ($\text{g} \cdot \text{h}^{-1}$), and the fugacity capacity of the blood lipids ($= Z_m$) at $37^\circ\text{C}$. The excretion of water via the feces also was accounted for. The dry mass excretion of feces is assumed to be 10% of the dry weight ingestion of food $G'_{fb}$ [38]. Respiration, nursing, and urination are treated as in the cow. Percutaneous excretion is modeled as an excretion of lipid at a rate of $3.7 \cdot 10^{-8} \text{m}^3 \text{h}^{-1}$ based on [39]. Birth is treated as a single loss of $6.0 \cdot 10^{-4} \text{m}^3$ of lipid and $2.6 \cdot 10^{-3} \text{m}^3$ of water.

A human is born every 10 years in the model on December 31. He/she is breastfed for the first six months; the baby’s initial fugacity and the fugacity of the mother’s milk are assumed equal to the fugacity of a 20-year-old woman in the model (or the fugacity of cows’ milk during the first two decades of the simulation). Men and women are treated separately in the model. Women can have children at intervals specified by the user, and the additional elimination pathways of childbirth and nursing are considered. During pregnancy both body weight and lipid volume increase. These parameters decrease gradually after childbirth and reach their prebirth values after 6.5 months. The concentrations in each person are simulated for up to 80 years.

**MODEL PARAMETERIZATION**

*Physical-chemical properties*

Due to their high bioaccumulation potential, PLOPs were the primary target of this modeling exercise, as is reflected in the process descriptions. Consequently polychlorinated biphenyls (PCBs) were selected for the initial model evaluation. Li et al. [40] recently have evaluated the available data on physical-chemical properties of PCBs and selected best estimates based on the internal consistency of all data. These values were used in the evaluation. Some of the relationships in the model are empirical correlations between model parameters and physical-chemical properties that were determined using PCBs. In these cases ($E_{0}$ from fish, cattle, and humans and the fugacity capacity of grass), the correlations were revised using the presumably more accurate physical-chemical properties of Li et al. (see Table 4, SETAC Supplemental Data Archive).

**Target environment**

Due to the availability of data, the Baltic Sea drainage basin, in particular southern Sweden, was chosen for the model evaluation. The ACC-HUMAN was parameterized using environmental characteristics applicable to this region.

**Marine food chain: Zooplankton**

The two most important fish for the Baltic Sea fishery are the planktivorous herring (*Clupea harengus*) and the piscivorous cod (*Gadus morhua*). In the past they made up a considerable portion of the fish consumed by the local population, and they continue to be an important component of the diet today. Their food web is well represented by the model structure.

In the laboratory or in the field there are few measurements of zooplankton/water partition coefficients. For the lack of convincing data to support an alternative approach, the fugacity capacity of zooplankton was assumed equal to the fugacity capacity of lipid (i.e., 1-octanol) multiplied by the lipid volume fraction in zooplankton. The lipid content of zooplankton show seasonal variations [41]. However, to our knowledge there are no representative data available describing this season variation either in the bulk zooplankton of the Baltic Sea or in those zooplankton species that are the primary food sources for Baltic herring and cod. Therefore the lipid mass fraction is assumed to be constant at a level of 4%, which represents a typical value for bulk zooplankton in the Baltic Sea [42–44].

**Marine food chain: Fish**

*Fish volume $V_F$.* The mean weight of Baltic herring in the years 1974 to 2000 and Baltic cod in the years 1966 to 2000 as a function of age [45] were used to generate a fourth order polynomial to predict fish weight $W_F$ as a function of age. This was converted via the fish density to the fish volume $V_F$.

*Lipid fraction $\nu_{lip}$.* Insufficient data were found to establish whether the lipid fractions of Baltic herring and cod are age dependent and to what extent they vary seasonally. Therefore, the lipid fraction of Baltic herring was assumed to be constant at a level of 3.5%, based on [46,47] and the Swedish Environmental Research Institute (IVL) database (http://www.ivl.se/miljo/db/intro.asp). The most detailed data on the lipid content of cod were found for Atlantic cod (*Gadus morhua* L.) [48], giving a mean value of 4.4%. This value refers to the whole fish; the muscle of cod has only a mean lipid fraction of about 0.5% [49] and the fraction in liver is about 57% [47] (http://www.ivl.se/miljo/db/intro.asp).

*Gill ventilation.* Gill ventilation is described as a (first order) diffusive process. The $D$-value is determined by the uptake rate constant $k_i$ and the fugacity capacity of water $Z_w$ (Table 2, SETAC Supplemental Data Archive) [50]. The uptake rate constant was defined as a function of the fish weight according to [51], and a correction for the chemical’s lipophilicity from [50] was applied (Table 3, SETAC Supplemental Data Archive). The latter is only of relevance for compounds with a log octanol/water partition coefficient (log $K_{ow}$) < 4.

*Metabolism.* The time constant for metabolism $k_{met}$ was set to 0 h$^{-1}$ for all PCB congeners.

*Food consumption rate $G_{fe}$.* Data on the overall food consumption of North Sea herring and cod as a function of fish age and season were used [52].

*Food composition.* Baltic herring feed on bulk zooplankton...
during the whole year [53–55]. Consequently, zooplankton was
defined as the only prey for herring. Baltic cod mainly feed
on fish and zooplankton, whereas benthic organisms are of
minor importance [56–58]. The food composition of cod was
defined as a function of the fish age and season in accordance
to [45,56], where herring and cod of young age classes
were used to represent the bulk planktivorous and piscivorous
fish in the prey, respectively.

Egestion factor $Q_E$. The egestion factor describes the quo-
tient of the $D$-value for ingestion and egestion, or the reduction
in the chemical carrying capacity of the ingested food as a
result of digestion. This reduction is due to both a decrease
in the volume of the food and a reduction in its fugacity ca-
pacity. Measurements for PCBs in rainbow trout and rock bass
indicated the combined effect of these processes to be about
a factor of 8 [59], and this value was adopted for $Q_E$ for both
species.

Terrestrial food chain: Grass

Grass volume $V_g$. The growing period was set to start on
March 1st; harvests were on May 15th, July 1st, and October
1st. The total annually harvested biomass was 0.006 m$^3$
(≈4,500 g fresh wt) per m$^2$ of pasture, where the yield of the
first, second, and third harvests contributed 45%, 40%, and
15% to the total crop, respectively.

Fugacity capacity $Z_P$. For the parameterization of the bio-
concentration properties, ryegrass (Lolium multiflorum) was
chosen, as it is the most important pasture grass species in
temperate Europe and has been particularly well investigated
with regard to its bioconcentration properties [60–63].

The fugacity capacity consisted of a contribution from the
water, air, and organic phase [27] (Table 1, SETAC Supple-
mental Data Archive). The fugacity capacity of the organic
phase was calculated from the equilibrium plant/air partition
coefficients $K_{PA}$ of hydrophobic chemicals. Kömp and Mc-
Lachlan [62,64] measured $K_{PA}$ for PCBs and derived a pre-
dictive equation for $K_{PA}$ at 25°C based on the octanol/air par-
tition coefficient $K_{OA}$. However, the temperature dependence
of $K_{PA}$ was very strong, and the heats of phase change between
grazing and air differed from the heats of phase change between
1-octanol and air. To minimize the errors in extrapolating $K_{PA}$
to different temperatures using the heats of phase change be-
tween octanol and air, the $K_{PA}$ to $K_{OA}$ regression was recal-
culated using the $K_{OA}$ data measured at the temperature closest
to the temperatures encountered in southern Sweden during
the growing period (9.8°C) and the $K_{OA}$ values of [40] inter-
polated to the same temperature, yielding

$$ K_{PA} = 8.3811 \cdot 10^{-5} \cdot K_{OA}^{0.999} \quad (10) $$

Atmospheric deposition. The mass transfer coefficient $k'_{P}$
describing the net particle-bound deposition, and the mass
transfer coefficient $k'_{GA}$, describing gaseous transport from the
atmosphere to the plant surface are defined based on mea-
surements of deposition to pasture grass in Germany [65].
The plant side mass transfer coefficient $k'_{GG}$, which describes trans-
port of gaseous chemical deposition from the plant surface
into the plant tissue, was parameterized based on laboratory
studies of contaminant transport in rye grass [60,61] (Table 3,
SETAC Supplemental Data Archive).

The plane through which mass transfer is described must
be consistent for a given pair of $k'$- and $A$-values. As the mass
transfer coefficients $k'_{P}$ and $k'_{GA}$ are defined for the plane of
the canopy, $A_{GP}$ and $A_{GA}$ were assigned a value of 1 m$^2$. The

$k'_{GG}$ is defined for the plane of the leaf surface, so $A_{GG}$ was
calculated by multiplying the specific surface area of the grass
(5,000 m$^2$ m$^{-3}$) by the grass volume in 1 m$^2$ of canopy ($V_{g}$).

Transformation. The time constant for metabolism $k_{GR}$ was
set to 0 h$^{-1}$ for all PCB congeners.

Terrestrial food chain: Milk cows

The parameters for the milk cow model were defined in
accordance [28,34] and are denoted in Tables 1, 3, and
5 (SETAC Supplemental Data Archive). The lipid volume of the
cow was set to 0.1 m$^3$ [28]. The water content in cattle
was assumed to be similar to that of humans (see Human
section) and was set to 70% of the lipid-free body weight,
yielding a water volume of 0.36 m$^3$.

Terrestrial food chain: Beef cattle

Volume $V_{p}$. The lipid volume of the steer $V_{LB}$ was assumed
to increase linearly from 0.01 m$^3$ at birth up to 0.15 m$^3$ at
slaughter. The water volume $V_{WB}$ was deemed to increase pro-
portionally to the lipid volume and was calculated in the same
way as for the milk cow.

Respiration. The respiration rate for the steer was the same
as for the milk cow (150 m$^3$ d$^{-1}$) [66]. For the newborn calf
a value of 40 m$^3$ d$^{-1}$ was assumed.

Food consumption. The feed (grass) uptake increased from
3.5 kg dry weight (dry wt) per day (0.0007 m$^3$ h$^{-1}$) at birth
to up to 13 kg dry wt per day (0.003 m$^3$ h$^{-1}$) at slaughter. The
soil content in feed was the same as for the milk cow (0.023
g dry wt$^{-1}$). The water consumption increased linearly from
6 L d$^{-1}$ for the calf up to 50 L d$^{-1}$ for the full-grown steer.

Urination. The urination rate $G_{UB}$ was set equal to the drinking
rate (i.e., it increased linearly from 6 L d$^{-1}$ at birth up to
50 L d$^{-1}$ at slaughter).

Metabolism. Metabolism was defined with a chemical specif-
ic time constant $k_{MB}$ (Table 5, SETAC Supplemental Data
Archive).

Terrestrial food chain: Humans

Lipid volume in the human $V_{p,h}$. The human lipid volume
was calculated using the approach in [38]. The body mass as
a function of age was estimated using the equation given in
[67]. Body mass was converted into lipid volume using the
estimates of body lipid content given in [68] for children and
adults, and a lipid density of 800 g L$^{-1}$. In accordance with
[38], heights of 1.80 m for men and 1.70 m for women were
assumed; a correction was added for the calculation of the
lipid weight of infants.

Water content. The water mass was set to 71% of the lip-
ide-free body mass (Heseker H, Weiß M. Trinken und Leistungs-
de/studien/Studie4/Studie4.htm]).

Respiration. The respiration rate for 25-year-old men and
women was set to 15 m$^3$ d$^{-1}$ [66]. An age correction was
performed similar to the food consumption rates (see Dietary
habits section).

Dietary habits. Based on data for the food consumption of
the Swedish population [69], time trend scenarios for the con-
sumption rates of dairy products, beef, and fish were estimated
for 25-year-old individuals. The ingestion rates for women
were assumed to be 20% lower than those of men. The con-
sumption of all food groups was assumed to vary with the
human’s age in the same way as the total dry weight ingestion
rate (see Egestion rate section). Drinking was defined anal-
ogously, assuming a water uptake rate for 25-year-old men and women of 0.003 m³ d⁻¹, which also accounts for the water content of ingested food. The age composition of the fish in the diet was deduced from data on the average landings of herring and cod of different age groups [45]. As humans primarily consume fish filet, the lipid fraction of the consumed cod was set to 0.5% in accordance with the lipid content of the muscle (see Fish section).

Egestion rate. The D-value for egestion is calculated from the feces dry weight egestion rate, which was assumed to be 10% of the dry weight ingestion rate $G^{Gestion}_{SW}$ [38]. The $G^{Gestion}_{SW}$ for men was calculated as a function of age using the method adopted in [38]. For women, $G^{Gestion}_{SW}$ was set equal to the ingestion rate of men until an age of seven years. From the age of 12 years women were assumed to ingest 20% less than men.

Urination. The urination rate $G^{Urination}_{SW}$ was set equal to the water uptake rate (see above).

Metabolism. Metabolism was defined with a chemical-specific time constant $k_{Metabolism}$ (Table 5, SETAC Supplemental Data Archive).

Pregnancy. In accordance with the available data on contaminant concentrations in mothers’ milk, the woman had her first child at an age of 29 years. In a second model run, the mother had her first child at 25 and a second child at 29. Nursing was assumed to last six months.

ENVIRONMENTAL CONTAMINATION SCENARIO

The full history of exposure must be known in order to predict human tissue concentrations. Therefore, air, water, and soil fugacity scenarios going back at least several decades are needed for the model input. The time trends of PCB fugacities were estimated from concentrations in guillemot eggs collected in the central Baltic Proper beginning in 1968 [70]. Prior to 1968, the trends were reconstructed using emission estimates for the Baltic watershed [71]. The resulting normalized scenario was then fitted to current concentrations in air (Swedish Environmental Research Institute database http://www.ivl.se/miljo/db/intro.asp) and water [72] (see Fig. 2a). Seasonal variability was included for the air concentrations, but no consistent seasonal variation has been observed for the water concentrations. The air and surface water concentrations were converted into fugacities that served as input to the model. Partitioning equilibrium was assumed between air and the soil and groundwater compartments (i.e., the fugacities in soil and groundwater were set equal to the fugacity in air).

RESULTS AND DISCUSSION

Using the environmental contamination scenario, the model predicted the PCB concentrations as a function of time in each of the trophic levels. The model predictions were compared with measurements in biota from the study area: Herring and cod from the Baltic Proper (IVL database, http://www.ivl.se/miljo/db/intro.asp), dairy products from Sweden and Denmark [73], Swedish beef [74], and Swedish human milk [75]. The results are illustrated in Figures 2 and 3 and discussed for each trophic level in the following sections.

The marine food chain

The predicted PCB concentrations in fish were compared with Swedish monitoring data from Baltic herring and cod (IVL database, http://www.ivl.se/miljo/db/intro.asp). The fish were caught in the western Baltic Proper (Landsort) in autumn. The results for PCB 153 in four-year-old herring and cod are shown in Figure 2c and 2d. Between the predicted and the reported lipid-normalized concentrations for herring there is good qualitative agreement. However, a quantitative evaluation is difficult because the herring data are characterized by high variability, even though the fish were caught at the same location at the same time of year and were sorted according to age. The variability is considerably lower for cod, and here the predicted concentration generally lay within the error bars ($\pm \sigma$) of the measured values.

Figure 3 shows the quotient of the predicted and the average measured lipid-normalized concentrations of different PCB congeners in 1999. In general the model tends to somewhat underpredict the fish concentrations for this year. The PCB 138 is an exception, showing an overprediction by a factor of 1.5 to 2. We suspect that this is related to the $K_{OW}$ values used in the model, because the predicted levels in fish are very sensitive to this parameter. Though Li et al. proposed a $K_{OW}$ value for PCB 138 that is about two times higher than for PCB 153 [40], the field data suggest that the bioaccumulation behavior of these two congeners is similar.

The results for the other years and for the other age classes were similar, with one exception: A tendency to overpredict the concentrations in three- to four-year-old herring was observed. We suspect that this is related to the lipid levels in the fish. As noted above, a constant lipid content was assumed for all herring age classes. However, the lipid content can be expected to vary seasonally due to changes in food availability and the reproductive cycle. Furthermore, there are reports of long-term variations in the lipid content in herring muscle tissue in the Baltic Proper, with a decrease in the 1980s followed by an increase in recent years [47]. A more detailed knowledge of herring lipid dynamics would facilitate improvement of the bioaccumulation model and might lead to a better understanding of the large variability in the monitoring data.

The agricultural food chain

We were unable to find data on PCB concentrations in grass for southern Sweden or Denmark. Consequently, the agricultural food chain model was evaluated on the basis of the reported levels in beef cattle and dairy products.

Good agreement was observed between model predictions of PCB lipid-normalized concentrations in milk and beef and measurements of these concentrations from southern Sweden and Denmark (see Figs. 2 and 3). The strongest deviation (a factor of 2) was observed for PCB 52, which together with PCB 101 was one of the labile congeners simulated. Given the simple system for selecting degradation rate constants based on just three classifications (labile, semipersistent, and persistent; see Table 5, SETAC Supplemental Data Archive), the agreement between the predicted and measured concentrations of these labile substances is good. The differences in the model performance between PCBs 138 and 153 (see Fig. 3) are related to the $K_{OW}$ values, as they were in fish. These compounds have $K_{OW}$ values in the range where the dietary absorption in the cow is nearly inversely proportional to $K_{OW}$. Although the data suggest that the bioaccumulation behavior of these substances is similar, the higher $K_{OW}$ value of PCB 138 ($\sim 2$ times) results in a lower calculated dietary absorption and a tendency to underpredict levels in beef and dairy products.

Similar concentrations in beef and dairy products were predicted for the lower chlorinated congeners. The ratio of the D-values for metabolism to lactation was about 24 for these.
compounds, though elimination of the native compound via exhalation and urination was predicted to be negligible in accordance with observations from mass balance studies [29,30]. This indicates that their elimination was dominated by the same process in both beef cattle and milk cows. Because the dietary uptake of the beef cattle at slaughter was similar to that of the milk cow, this explains the similar concentrations.

The predicted concentrations of the higher chlorinated and persistent congeners were about three times higher in beef than in dairy products. For these substances, lactation dominates elimination in milk cows, whereby elimination in beef cattle is minimal. In this case the ratio of the contaminant levels is related to the ratio of the quantity of contaminant consumed to produce a unit volume of milk or beef fat. This ratio is dependent on the feeding strategy employed. In the simulation here, beef cattle had a large fraction of grass in their feed ration. This led to a high PCB intake per unit beef fat produced. However, if a ration was used with less grass and more feeds with low PCB levels such as grains, the PCB intake per unit beef fat produced would be lower, resulting in lower PCB levels in beef and a smaller difference between levels in beef and dairy products.

**The human model**

The most important endpoint of the bioaccumulation model is human tissue levels. This endpoint was evaluated using data for Swedish mothers’ milk collected between 1972 and 1997 [75]. The women were 27 to 31 years old with a mean age of
concentrations, the model predicts a 17% decrease in the tissue and nursing. For a women exposed to constant environmental main vectors of PLOPs to humans, and these are two of the questionable. However, fish and dairy products constitute the and trade means that this assumption is becoming increasingly living. The increasing internationalization of food production of the organic contaminants taken up with the diet originate metabolism of PCBs in humans.

It should be noted that the model assumes that the majority of the organic contaminants taken up with the diet originate from foods produced in the region in which the individual is living. The increasing internationalization of food production and trade means that this assumption is becoming increasingly questionable. However, fish and dairy products constitute the main vectors of PLOPs to humans, and these are two of the food groups that still are produced and consumed largely in the Baltic watershed.

The human model can be used to simulate the development of contaminant levels in women during pregnancy, childbirth, and nursing. For a women exposed to constant environmental concentrations, the model predicts a 17% decrease in the tissue concentration of PCB 153 between conception and full-term due to the increase in the mother’s lipid mass (results not shown). An estimated 3% of the PCB 153 body burden is lost during delivery, but the tissue concentrations are not affected. During the following six-month nursing period, the elimination of the contaminant via the milk to a large extent is balanced by the decrease in the mother’s lipid mass as she approaches her pre-pregnancy weight; hence the mother’s tissue concentrations decrease only slowly. At the end of the six months she is back to her original lipid mass and the lipid-normalized concentration (and body burden) have decreased to 71% of their pre-pregnancy values. Seen from the baby’s perspective, 11% of the PCB 153 load received from the mother is delivered at birth, while the remaining 89% comes via the mother’s milk.

Responses to changes of environmental levels

A time lag exists between a change in the environmental level of a contaminant and the resulting change in the levels in the organism. The magnitude of the lag, among other things, depends on the rates of metabolism and excretion [4], the organism’s life expectancy, and the kind of food eaten. The time lag for a persistent lipophilic contaminant can be seen in Figure 2. The peak in the environmental concentrations was 1970. The time lag until the peak level of contamination was reached in the various organisms was as follows: Dairy cow, one year; beef cattle, herring, and cod, two years; 29-year-old humans, approximately 10 years. As a result, the peak level of exposure in infants also occurs approximately 10 years after the peak in environmental levels. This is a further argument for a conservative approach to managing these contaminants in the environment. Even if one were able to immediately detect adverse effects of PLOPs and immediately reduce environmental levels, the exposure to infants would continue to increase for about a decade thereafter.

Due to more rapid elimination, shorter time lags were observed for PCB 52 (results not shown). The time lag for herring and cod was just one year, which can be attributed to the more rapid gill elimination of this congener compared to PCB 153. For the dairy cow there was no time lag, and for beef cattle it was one year. In this case the rapid elimination via metabolism accounts for the more rapid response. The same is true for humans; the time lag for 29-year-olds dropped from approximately 10 years to one to four years. The food chain responds rapidly to changes in the environmental levels of labile compounds.

Fugacity/biomagnification

The model enables one to study the change in chemical fugacity beginning in the environment through the food chains to humans. Changes in fugacity are the best measure of whether biomagnification/biodilution are occurring and thereby further our understanding of the bioaccumulation process.

The fugacities of PCB 153 in October 1989 are shown in Figure 4. In the marine food chain, the fugacity in water and zooplankton are the same (equilibrium partitioning was assumed), but a fivefold increase in fugacity is seen from zooplankton to herring, and a further doubling occurs from herring to cod. This is a clear indication that biomagnification is occurring for this chemical (i.e., the dietary ingestion of PCB 153 is more rapid than its elimination via the gills and feces and its dilution due to growth). The degree of biomagnification nevertheless is less than the maximum theoretical value of 8 given by the egestion factor \( Q_e \) [3].
In the agricultural food chain, the fugacity of PCB 153 decreases from air to grass. Due to growth dilution, a partitioning equilibrium between the vegetation and the atmosphere is not approached. A 10-fold increase in fugacity occurs between grass and the milk cow, though for beef cattle the increase is a factor of 30. This very pronounced biomagnification is caused to a large degree by the approximately 25°C increase in grass temperature going from the pasture into the cow’s digestive tract [25]. This results in a 20- to 30-fold increase in the fugacity of PCB 153, leading to a correspondingly higher fugacity in the animal.

Turning to humans, the fugacity increases 70-fold from beef and fish. This pronounced biomagnification also to a large degree is attributable to the increase in fish temperature between the marine environment and the human. In addition, the human digestive tract is very efficient at absorbing lipophilic substances, and there are no effective mechanisms for eliminating PCB 153 once it is absorbed.

It is interesting to note that the fugacities in herring/cod and beef cattle are similar, suggesting that PCB 153 bioaccumulates to a similar degree in each of them. A comparison of the lipid-normalized concentration of PCB 153 shows this to be 40 to 80 times lower in the cattle than in the fish. Also, whereas the fugacity of PCB 153 in humans is 40 times higher than in cod, the lipid-normalized concentration is higher in cod than in humans. Clearly, lipid-normalized concentrations cannot be used as an indicator of the degree of bioaccumulation of a chemical when the organisms live at different temperatures. This also illustrates that poikilothermal organisms living in cold environments can attain particularly high concentrations of PLOPs and pose a high risk to warm-blooded organisms that prey on them.

In a recent paper it was suggested that contaminant amplification in the environment can be explained by two processes termed solvent switching and solvent depletion [76]. The results of this study and previous work [25] demonstrate that changes in temperature also play a key role in organic contaminant amplification in food chains. This temperature effect cannot be subsumed under the fugacity modifying solvent depletion heading; it represents a third important mechanism of contaminant amplification [77].

CONCLUSION

The agreement between the predicted and the measured PCB concentrations on the whole was very good, especially considering the uncertainties in the emissions scenario. This indicates that ACC-HUMAN captures the major features of the bioaccumulation of PLOPs to humans. Reduced to the main vectors of human exposure, it provides a simply structured and useful tool for predicting human exposure to bioaccumulative organic compounds.

An executable version of the ACC-HUMAN model and further model documentation can be downloaded from http://www.itm.su.se/.

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