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Towards an improved understanding of processes controlling absorption efficiency and biomagnification of organic chemicals by fish

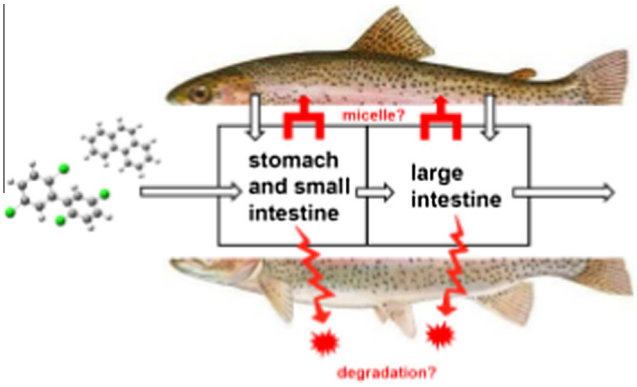
Ruiyang Xiao [a,b,*], Jon A. Arnot [c,d], Matthew MacLeod [a]

[a] Department of Environmental Science and Analytical Chemistry, Stockholm University, Svante Arrhenius väg 8, SE-11418 Stockholm, Sweden
[b] Institute of Environmental Engineering, School of Metallurgy and Environment, Central South University, Changsha 410083, China
[c] ARC Arnot Research & Consulting, 36 Sproat Avenue, Toronto, Ontario M4M 1W4, Canada
[d] Department of Physical and Environmental Sciences, University of Toronto Scarborough, 1265 Military Trail, Toronto, Ontario M1C 1A4, Canada

HIGHLIGHTS

- We developed a fish bioaccumulation model to describe dietary uptake of chemicals.
- Including micelle-mediated diffusion in the model does not improve its performance.
- The model indicates that first-pass biotransformation reduces bioavailability of some PAHs.

GRAPHICAL ABSTRACT



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ABSTRACT

Dietary exposure is considered the dominant pathway for fish exposed to persistent, hydrophobic chemicals in the environment. Here we present a dynamic, fugacity-based three-compartment bioaccumulation model that describes the fish body as one compartment and the gastrointestinal tract (GIT) as two compartments. The model simulates uptake from the GIT by passive diffusion and micelle-mediated diffusion, and chemical degradation in the fish and the GIT compartments. We applied the model to a consistent measured dietary uptake and depuration dataset for rainbow trout ($n = 215$) that is comprised of chlorinated benzenes, biphenyls, dioxins, diphenyl ethers, and polycyclic aromatic hydrocarbons (PAHs). Model performance relative to the measured data is statistically similar regardless of whether micelle-mediated diffusion is included; however, there are considerable uncertainties in modeling this process. When degradation in the GIT is assumed to be negligible, modeled chemical elimination rates are similar to measured rates; however, predicted concentrations of the PAHs are consistently higher than measurements by up to a factor of 20. Introducing a kinetic limit on chemical transport from the fish compartment to the GIT and increasing the rate constant for degradation of PAHs in tissues of the liver and/or GIT are required to achieve good agreement between the modelled and measured concentrations for PAHs. Our results indicate that the apparent low absorption efficiency of PAHs relative to the chemicals with similar hydrophobicity is attributable to biotransformation in the liver and/or the GIT. Our results provide process-level insights about controls on the extent of bioaccumulation of chemicals.

* Corresponding author at: Institute of Environmental Engineering, School of Metallurgy and Environment, Central South University, Changsha 410083, China.
E-mail address: xiao.53@csu.edu.cn (R. Xiao).

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1. Introduction

Bioaccumulation is the net result of competing rates of chemical uptake and elimination in an organism and bioaccumulation includes all possible routes of chemical exposure, such as transport across the respiratory surface, dermal absorption, and dietary absorption (Gobas and Morrison, 2000). Dietary absorption from the gastrointestinal tract (GIT) is recognized to be the dominant uptake pathway for persistent, hydrophobic chemicals by fish in the environment (Gobas and Morrison, 2000). The dietary absorption efficiency (E_D) quantifies the fraction of the ingested chemical that is transferred from the lumen of the GIT into the fish body. Niimi and colleagues used generally consistent methods to measure gross E_D, body burdens and depuration in a series of laboratory studies using rainbow trout (*Oncorhynchus mykiss*) for various organic chemicals (Niimi, 1986; Niimi and Oliver, 1986, 1988; Niimi and Palazzo, 1986). We use the term gross E_D to refer to the fraction of ingested chemical that is transferred from the GIT into the fish body. This definition of gross E_D is used in many process-based models (Xiao et al., 2013). In contrast, net E_D is the apparent absorption efficiency when chemical flux between the GIT and the fish body reaches steady state. Notably, the gross E_D reported by Niimi et al. is low for certain polycyclic aromatic hydrocarbons (PAHs) (often less than 1%), and markedly lower than for other chemicals with comparable hydrophobicity (Niimi, 1986; Niimi and Oliver, 1986, 1988; Niimi and Palazzo, 1986).

Many mass balance models have been developed and applied to quantify chemical flux in (by dietary and gill uptake) and out (by gill elimination, growth dilution, biotransformation, and fecal egestion) of fish (Gobas et al., 1988, 1993a; Barber et al., 1991; Arnot and Gobas, 2004; Kelly et al., 2004; Nichols et al., 2004; Bhavsar et al., 2008). For example, Clark et al. developed a one compartment, fugacity-based steady-state fish bioaccumulation model for chemical exposures from food using transport and transformation resistances (Clark et al., 1990). Barber et al. developed a model to describe uptake of organic chemicals from water and contaminated food, and they applied it to describe the bioaccumulation of polychlorinated biphenyls (PCBs) for different fish species (Barber et al., 1991). Gobas developed a one-compartment steady-state fish bioaccumulation model in rate constant format that agreed well with chemical concentrations measured in the field (Gobas, 1993). Nichols et al. developed a dynamic multi-compartment physiologically-based pharmacokinetic (PBPK) model for fish (Nichols et al., 2004). Some fish bioaccumulation models explicitly model micelle-mediated chemical transport in the GIT (Drouillard and Norstrom, 2000; Drouillard et al., 2012) and some do not (Gobas et al., 1993a,b; Arnot and Gobas, 2004).

Here, we apply a fugacity-based multi-compartment fish bioaccumulation model to explore hypotheses about the roles of micelle-mediated uptake and chemical degradation processes in fish, especially for PAHs. The model is comprised of a single compartment for the fish body and two compartments for the GIT. We have parameterized the model to evaluate the role of micelle-mediated uptake and biotransformation of chemicals in fish by analyzing the measurements of chemical uptake and clearance from laboratory studies using rainbow trout (*O. mykiss*) that were conducted by Niimi and colleagues (Niimi, 1986; Niimi and Oliver, 1986, 1988; Niimi and Palazzo, 1986). Their datasets include chlorinated benzenes and biphenyls, dioxins, chlorinated diphenyl ethers, and PAHs. The Niimi et al. studies of chemical uptake by rainbow trout provide a consistent set of measurements and cover a wide range of structural diversity, hydrophobicity, and potential for degradability in fish (Niimi, 1986; Niimi and Oliver, 1986, 1988; Niimi and Palazzo, 1986). Our goal in this study is to use a mechanistic model to formalize two quantifiable hypotheses about processes that control dietary absorption of chemicals by fish (MacLeod et al., 2010). The two hypotheses are (1) micelle-mediated diffusion does not play a significant role in dietary uptake of certain organic chemicals in trout, and (2) chemical degradation in the liver and/or the GIT reduces dietary uptake of PAHs. We aim to evaluate the hypotheses against the experimental data produced by Niimi and colleagues.

2. Methods

2.1. Model description

The model describes chemical uptake and elimination from three compartments: fish (F), upper part of the GIT (GIT1), and lower part of the GIT (GIT2) (Fig. 1). The model is based on the Arnot and Gobas mass balance fish bioaccumulation model, which treats the fish as one compartment (Arnot and Gobas, 2004). Here, the Arnot and Gobas model is re-formulated using fugacity-based process equations rather than rate constants, the GIT is added as two compartments as described below, and a numerical solution to the dynamic (non-steady-state) mass balance equations is applied.

Fugacity (f, in Pa) reflects an equilibrium criterion and thus provides clear insight into the thermodynamic gradients driving chemical processes, especially passive diffusion. Chemicals diffuse from compartments with high fugacity to compartments with low fugacity. The fugacity capacity (Z, in mol/Pa m^3) of a compartment is a measure of storage capacity for a chemical, and is particularly useful for describing multimedia phases with variable compositions (e.g., digesta in the digestive tract). The Z value depends on the nature of the chemical and the properties of the multimedia phase that contains it.

The division of the GIT and the parameterization used in our model is based on the elegant experiments conducted by Gobas and co-workers using PCBs that showed the fugacity capacity of digesta in the upper part of the GIT of fish is approximately four times higher than that in the lower part of the GIT (Gobas et al., 1999). The physicochemical properties of a chemical remain unchanged through the GIT. However, lipids are mainly absorbed in the upper part of the GIT. Lipids are a major component of the fugacity capacity of the digesta, therefore digesta in the lower part of the GIT has lower fugacity capacity, which in turn causes a stronger thermodynamic gradient for chemical transport from the GIT into the fish (Clark et al., 1990; Gobas et al., 1999). The transport parameter (D, in mol/Pa h) is the fugacity analogue to a first-order rate constant. Slow processes have small D-values, and vice versa (Mackay, 2001). Studies suggest micelle-mediated diffusion plays a role in the mass transport of chemical from the GIT into organisms, and this process can be characterized as a D-value (Drouillard and Norstrom, 2000; Kelly et al., 2004; Drouillard et al., 2012).

Fluxes of chemical uptake and elimination in fish (N, in mol/h) following dietary exposure are expressed as a product Df in our model (Fig. 1). The volume, temperature, and composition of each compartment in the model are assumed to be constant over time, so that for the fish (F), upper GIT (GIT1), and lower GIT (GIT2) compartments, the differential mass balance equations are:change of fugacity in F:

$$V_F \times Z_F \frac{df_F}{dt} = D_{G1F} \times f_{GIT1} + D_{G2F} \times f_{GIT2} - (D_V + D_M + D_X + D_{FG1} + D_{FG2}) \times f_F \quad (1)$$

change of fugacity in GIT1: