Application of a food web model to assess the dynamics of polychlorinated naphthalenes (PCNs) in a Lake Ontario food web

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Introduction

Food web contaminant models are useful as heuristic tools for teasing apart complex and interacting processes and generating testable hypotheses on the mechanisms controlling contaminant bioaccumulation. However, most food web models have only been applied to legacy contaminant, such as PCBs, which are not typically metabolized at a significant rate in aquatic food webs.[1,2,3]

Polychlorinated naphthalenes (PCNs) are a group of 75 compounds that were manufactured as complex technical mixtures for a range of industrial applications.[4] They are also found in PCB formulations and in combustion emissions.[4] PCNs have similar physical properties as PCBs and bioaccumulate in aquatic food webs.[4,5,6] However, unlike PCBs, PCN congeners with two or more vicinal carbon atoms unsubstituted with chlorine can be metabolized by fish.[7] In addition, all PCNs have a planar configuration whereas only non-ortho and mono-ortho PCBs can rotate into the same plane.[8] PCN concentrations, as well as concentrations of non-ortho and mono-ortho PCBs, were recently determined in a Lake Ontario food web.[9] This data set provides an opportunity to study PCN dynamics in this food web using a modeling approach.

The first objective of this study is to evaluate a food web model by applying it to predict the concentrations of well studied and persistent PCB congeners in a Lake Ontario food web. The second objective is to use the model to assess the mechanisms controlling PCN dynamics in this system.

Model Structure and Parameterization

The food web model is based on those developed by Gewurtz et al.[10] and Arnot and Gobas[1]. The model is fugacity based[3], assumes steady-state conditions, and incorporates mechanistic equations to describe biomagnification. We determined dietary interactions from stomach content and stable isotope analysis, as well as data obtained from the literature (e.g. Kiriluk et al.[11]). Since no PCN water measurements were available, we back calculated water concentrations from net plankton data (which contain a mixture of phytoplankton and zooplankton) assuming equilibrium. The model assumes no metabolic degradation. Therefore if metabolic degradation is occurring, the model should over-predict concentrations.

Results and Discussion

We first evaluate the model using two representative PCB congeners, PCB 118 and PCB 105, which are mono-ortho substituted, contain no vicinal carbon atoms unsubstituted with chlorine, and do not typically degrade in either invertebrates or fish.[12] The model predicts biota concentrations of both of these PCB congeners within the error associated with the measured data (Figure 1).

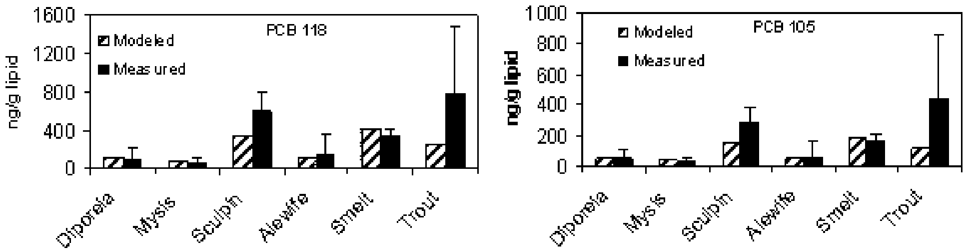

Figure 1. Modeled and measured concentrations of PCB 118 and PCB 105 in a Lake Ontario food web. Error bars represent 95% confidence intervals.

We next use the model to predict concentrations of PCN congeners which contain no vicinal carbon atoms unsubstituted with chlorine and thus do not typically degrade in freshwater organisms.[4] Figure 2 shows results for PCN 42 and PCN 52/60, which represent two examples of such PCN congeners.

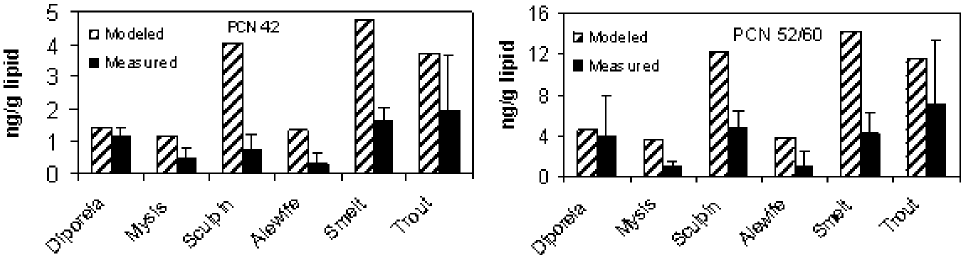

Figure 2. Modeled and measured concentrations of PCN 42 and PCN 52/60 in a Lake Ontario food web. Error bars represent 95% confidence intervals.

The concentrations of these PCN congeners are over-predicted even in benthic invertebrates, which are unlikely to metabolize PCNs. This suggests that a process not accounted for in the model, other than metabolism, is influencing PCN bioaccumulation.

Planar compounds, such as PCNs, show stronger sorption to sediment then would be expected based on equilibrium partitioning.[13] Biota sediment accumulation factors (BSAFs), calculated as the ratio of the lipid normalized concentration in an organism to the organic carbon normalized concentration in sediment, are consistently greater for PCBs than for PCNs, in both diporeia and mysis (Figure 3).

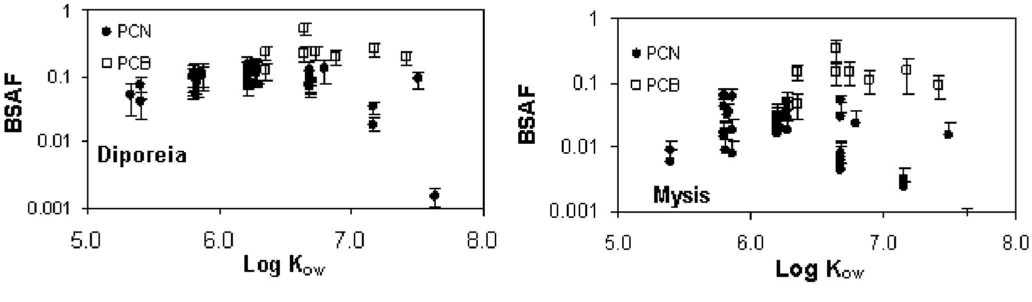

Figure 3. BSAFs for PCN and PCB congeners in both diporeia and mysis. Error bars represent ±1 standard error of the mean.

Similar results were found by Kannan et al.[14]. These data suggest that the bioavailability of contaminants from sediment is greater for PCBs than for PCNs.

The extent of decreased bioavailability from sediment likely varies among invertebrates due to species-specific differences in digestive abilities. In order to account for this, we calibrate the model by maximizing correspondence between measured and modeled concentrations of persistent PCNs in benthic invertebrates by decreasing bioavailability in sediment. Based on this calibration, we decrease the bioavailability of PCNs in sediment for diporeia and mysis 2 and 10 times, respectively, which improves correspondence of model predicted concentrations to measured data in fish (Figure 4).

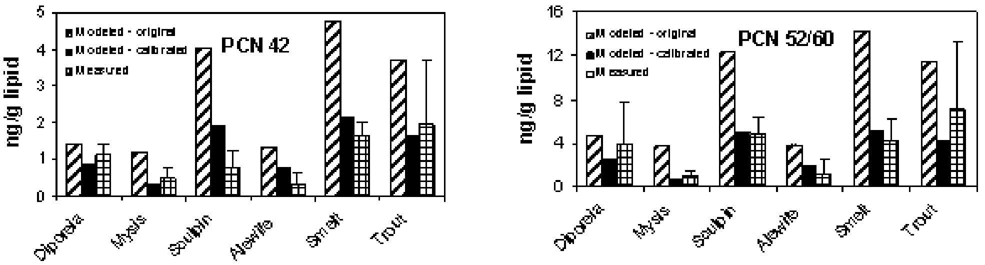

Figure 4. Modeled and measured concentrations of PCN 42 and PCN 52/60 in a Lake Ontario food web. Error bars represent 95% confidence intervals.

Finally we use the model to predict concentrations of PCN congeners which contain vicinal carbon atoms unsubstituted with chlorine. Figure 5 shows results for PCN 38/40 and PCN 27/30/39, which represent two examples of such PCN congeners.

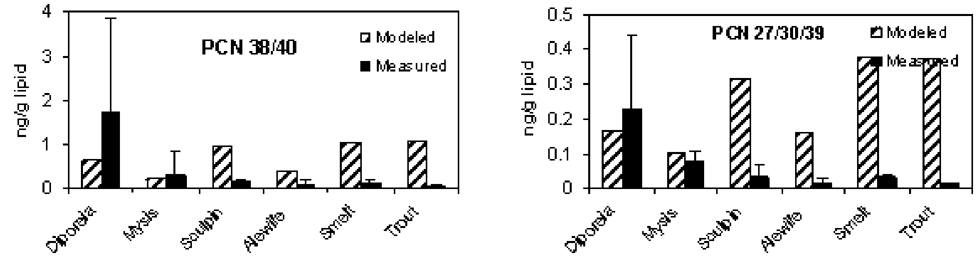

Figure 5. Modeled and measured concentrations of PCN 38/40 and PCN 27/30/39 in a Lake Ontario food web. Error bars represent 95% confidence intervals.

The model over-predicts concentrations of both PCN 38/40 and PCN 27/30/39 in fish but not in invertebrates. This result corresponds with other studies[7,15] and suggests that metabolic degradation of these congeners is occurring in the higher trophic levels of the Lake Ontario food web.

References

1. Arnot J. A. and Gobas F. A. P. C. (2004) *Environ Toxicol Chem*. 23: 2343 2355.
2. Morrison H. A., Gobas F. A. P. C., Lazar R., Whittle D. M. and Haffner G. D. (1997) *Environ SciTechnol*. 31: 3267-3273.
3. Campfens J. and Mackay D. (1997) *Environ SciTechnol*. 31: 577-583.
4. Falandysz J. (1998) *Environ Pollut*. 101: 77-90.
5. Lei Y. D., Wania F. and Shiu W. Y. (1999) *J Chem Eng Data*. 44: 577-582.
6. Opperhuizen A. (1987) *Toxicol Environ Chem*. 15: 249-264.
7. Lundgren K., Tysklind M., Ishaq R., Broman D. and Van Bavel B. (2002) *Environ SciTechnol*. 36: 4004-5013.
8. Metcalfe C. D. and Haffner G. D. (1995) *Environ Rev*. 3: 171-190.
9. Helm P. A., Whittle D. M., Tomy G. T. and Fisk A. T. (2005) In Preparation.
10. Gewurtz S. B., Gandhi N., Christensen G. N., Evenset A., Gregor D. and Diamond M. L. (2005) Submitted to *Environ. Pollut.*
11. Kiriluk R. M., Servos M. R., Whittle D. M., Cabana G. and Rasmussen J. B. (1995) *Can J Fish AquatSci*. 52: 2660-2674.
12. Buckman A. H., Brown S. B., Hoekstra P. F., Solomon K. R. and Fisk A. T. (2004) *Environ Toxicol Chem*. 23: 1725-1736.
13. Jonker M. T. O. and Koelmans A. A. (2002) *Environ SciTechnol*. 36: 3725-3734.
14. Kannan K., Imagawa T., Blankenship A. L. and Giesy J. P. (1998) *Environ SciTechnol*. 32: 2507-2514.

15. Falandysz J. and Rappe C. (1996) *Environ SciTechnol*. 30: 3362-3370.