Bull. Environ. Contam. Toxicol. (1997) 58:364-371

Environmental Contamination and Toxicology

Dynamics of Chlordane Under Nonconstant Exposure Conditions: A Numerical Bioaccumulation Model

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Received: 24 August 1995/Accepted: 3 November 1996

Much attention has been paid to the bioaccumulation of toxicants in aquatic organisms during the last several years. In order to accurately predict and assess toxicant fate in natural ecosystems, most efforts have employed mathematical models to determine the relationship between aqueous concentrations and organism accumulation (Barron et al 1990; Landrum et al. 1992; Newman, 1995). In most cases, bioaccumulation models are based on the assumption that the concentration in water remains constant over the exposure time period. Thus, many exposure experiments are designed to achieve constant aqueous concentration of chemicals using dynamic flow-through systems (Bruggeman, 1981).

Constant exposure regimes greatly simplify the estimation of bioconcentration factor (BCF) as well as the model kinetics. In natural systems, however, the aqueous concentration may significantly and continuously change over time through dynamic processes of volatilization, sedimentation, and biochemical degradation, such that the steady-state equilibrium conditions are rarely established (Newman and Jogue, 1996). Even in dynamic exposure experiments, it is a constraint to maintain constant aqueous concentrations of chemicals in these systems (Karara and McFarland, 1992). Bioaccumulation itself can also cause significant changes of aqueous concentrations not accounted for in constant concentration models. In order to describe the bioaccumulation processes in the laboratory as well as in natural systems more accurately, these models should incorporate the changes of concentrations in both the water phase, as well as in aquatic organisms.

This paper presents a bioaccumulation model where the concentrations of toxicants continuously change over time both in organisms and in water while incorporating volatilization, biochemical degradation, or sediment interaction terms. This model was developed based on a mass balance numerical approach and validated against data obtained from the bioaccumulation of *trans*- and *cis*-chlordane into goldfish *(Carassius auratus).*

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MATERIALS AND METHODS

Cis- and *trans*- chlordane solutions (analytical reference standards supplied by Velsicol Chemical Co., Rosemont, IL; minimum purity: >99% by GC) were added to 30 liter deionized water in a glass aquarium, where twenty-four goldfish *(Carassius auratus;* obtained from Carolina Biological Supply Co., Burlington, NC) were added after 48 hours. Aqueous concentrations were not held constant. At the time of fish exposure, initial aqueous concentrations of *trans*- and *cis*-chlordane were 0.12 and 0.30, respectively, which are 100 times lower than 96 hr LC_{50} observed in fish (Erstfeld, 1986). Samples of fish (four) and duplicate 90 mL water were taken for chlordane analysis at time zero, 24, 48, 52, 60, 72, and 96 hours. The aquarium was covered with glass to minimize volatility. The aquarium was aerated by bubblers and was placed in an environmental chamber, set at a temperature of 21 ± 2°C and a relative humidity of 70%. A photoperiod of 12 hours daylight was maintained throughout the experiment.

Fish and water sample were extracted and analyzed for *trans* -chlordane, *cis*-chlordane, and oxychlordane, the metabolite of both isomers, by GC utilizing EC detection. The chemical analyses are fully described in Erstfeld et al. (1989).

The bioaccumulation of toxic chemicals can be viewed as a dynamic process with competing rates of uptake and elimination between aquatic organisms and water. Assuming a two compartment system, where all possible mass changes are defined by linear transfer coefficients as diagramed in Figure 1, two mass balance differential equations are derived as follows:

$$\frac{dX_1}{dt} = -a_{11} X_1 - a_{21} X_1 + a_{12} X_2 \qquad (1)$$

$$\frac{dX_2}{dt} = a_{21} X_1 - a_{12} X_2 - a_{22} X_2 - a_{32} X_2 - a_{42} X_2 \qquad (2)$$

where, X_1 and X_2 are the amount of toxicant in aquatic organisms and water [M], respectively; a_{ij}'s are the linear transfer coefficients or the first order decay coefficient [1/T], and t is time. After substituting the concentration terms ($X_1=WC_1$; $X_2=VC_2$) into equation 1 and 2, respectively, yields the following equations:

$$\frac{dC_1}{dt} = -(a_{11} + a_{21})C_1 + \frac{a_{12}}{W} V C_2 \qquad (3)$$

$$\frac{dC_2}{dt} = a_{21} \frac{W}{V} C_1 - (a_{12} + a_{22} + a_{32} + a_{42}) C_2 \qquad (4)$$

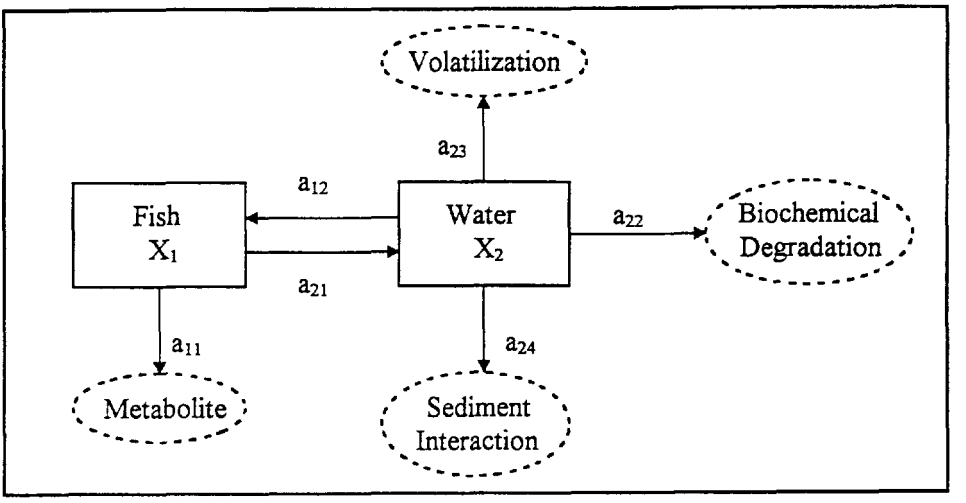

Figure 1, Schematic diagram of linear transport processes in the model

where, C_1 and C_2 are the concentrations in organisms [M/M] and in water [M/L^3]; W and V are weight of organisms [M] and volume of water [L^3]. The linear transfer coefficients remains constant over time and with regard to volume of water as well as weight of organisms. Due to removal of fish during the

experiment, the weight of fish in the system changes over time. Therefore, the linear transfer coefficient changes over time. The coefficient, a_{12} (uptake rate by organisms) is dependent on weight of organisms. Therefore, we considered the linear transfer coefficients constant, with the exception of a_{12}. The uptake rate per unit weight ($\alpha = a_{12} / W$) is kept constant.

The steady-state bioconcentration factor for aquatic organisms under constant aqueous exposure conditions is usually expressed by the equilibrium constants, ($BCF=C_1/C_2$). Since the bioconcentration factor is easier to measure than transport coefficients and well documented in literature, transport coefficients for elimination is replaced by BCF ($a_{21} = \alpha \cdot V/BCF$). After substituting BCF into equations 3 and 4, two coupled differential equations are derived and they can be numerically integrated by the Euler method (Keen and Spain, 1993) as follows:

$$\begin{bmatrix} C_1 \\ C_2 \end{bmatrix}_{t+\Delta t} \leftarrow \begin{bmatrix} C_1 \\ C_2 \end{bmatrix}_t + (\Delta t) \cdot \begin{bmatrix} -(a_{11} + \alpha \cdot \frac{V}{BCF}) & \alpha \cdot V \\ \alpha \cdot \frac{W}{BCF} & -(\alpha \cdot W + a_{22} + a_{32} + a_{42}) \end{bmatrix} \times \begin{bmatrix} C_1 \\ C_2 \end{bmatrix}_t \quad (5)$$

The integration process was programmed in FORTRAN and extensively used in this study. Based on input conditions of initial concentrations, transport and decay coefficients, and weight/volume ratios, this model can generate the time course of non-constant chemical concentrations in both fish and water compartments.

RESULTS AND DISCUSSION

The developed non-constant exposure model was applied to data from the exposure study. Based on material balance and previous air sampling data (Erstfeld, 1986), volatilization from water was accounted for in the test system and included as a model input parameter. The inclusion of varying fish weights and reduction in water volume for each period were accounted for in the model. No metabolism and biochemical degradation were assumed during this experimental period. The values for uptake rate, volatilization rate, and BCF were kept constant throughout the exposure period. Metabolism and biochemical degradation were determined negligible during this experimental period since the metabolite, oxychlordane, was not detected in either fish or water. In accordance to the sampling time schedule, six exposure periods were set up in the model and no incremental decrease in mass and volume in the compartments was assumed within one period. The results from the developed model were compared with the experimental data. System coefficients were appropriately calibrated until reasonable agreement between model results and experimental data was achieved.

The conventional constant exposure model was also applied to the experimental data to examine differences between the two model results. In the constant exposure model, the concentration in fish is described as:

$$C_1 = \frac{k_u}{k_e} C_2 (1 - e^{-k_e t}) \qquad (6)$$

where k_u is the uptake rate constant [L^3/M/T] and k_e is the elimination rate constant [l/T]. The aqueous concentration (C_2) is assumed to remain constant throughout the experiment in this model. The uptake and elimination rate constants were estimated by nonlinear curve fitting to the experimental data.

In the constant exposure model, the input for the aqueous concentration remains constant, however, the experimental data significantly decrease during the testing period due to both fish uptake and volatilization from the test system. In contrast, results from the nonconstant exposure model better predict the decrease in aqueous concentration. Figure 2 illustrates that both bioaccumulation models represent the experimental fish data quite accurately. From a closer examination of these figures, it can be observed that there are some differences between the two models. Results from nonconstant exposure model appear to approach maximum fish concentrations approximately after 30 hours and then decrease at very slow

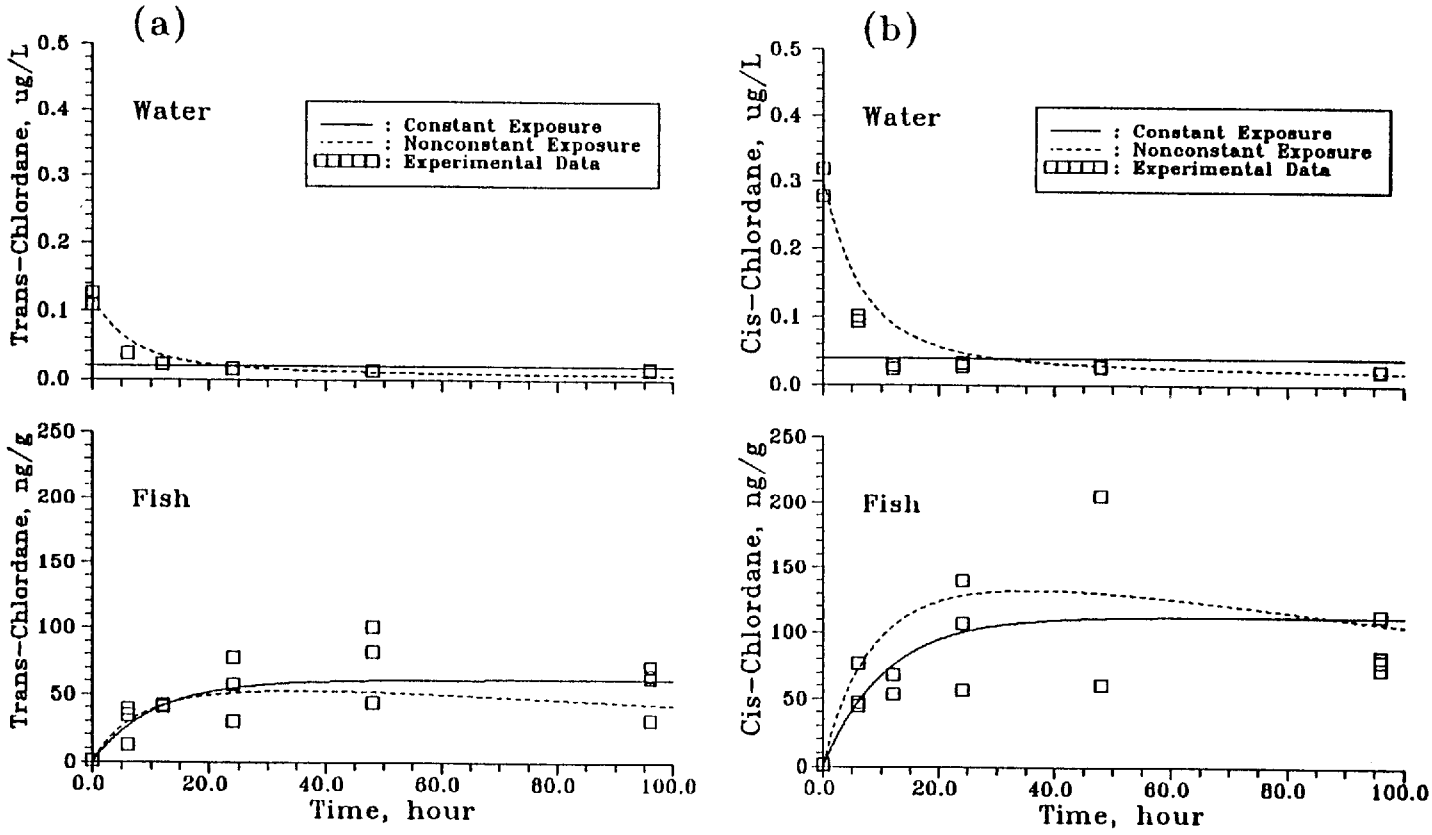

Figure 2. Comparison of model predictions with experimental data

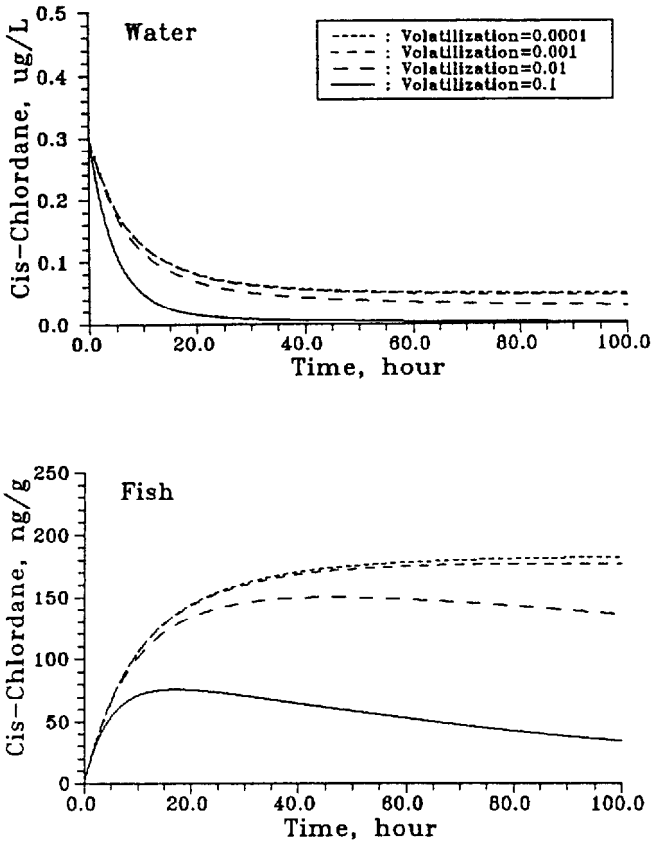

Figure 3. Effect of volatilization rate on the time course of chemical concentration in water and fish.

rate, whereas the bioconcentration remains constant in the constant exposure model, achieving an equilibrium state. Thus, the nonconstant exposure model is a better predictor of bioconcentration since the aqueous concentration continuously decreases in this system.

Using the non-constant exposure model allows us to develop two new experimental parameters: critical time (Tc) and critical bioaccumulation ($Conc_{crit}$). These values would be important indicators to assess the ecological risk of chemicals under the nonconstant exposure conditions. The critical time, time to reach the maximum concentration, and the critical concentration, need to be estimated under the nonconstant exposure conditions. These values are largely

dependent upon volatilization and biochemical degradation. In water and metabolism in fish, as well as the uptake and elimination rates. The developed nonconstant exposure model was programmed to predict these values as well as the time course of chemicals in both aqueous and organism compartments.

Overall transfer coefficients for both models are summarized in Table 1. As seen in this table, there are significant discrepancies in both the uptake rate and calculated bioconcentration factor between the two models. Differences are due to the assumption of constant exposure in the experimental system where volatility and fish uptake are not accounted for. It is recognized that the description of bioaccumulation processes should be based on actual exposure conditions. Therefore, the use of the nonconstant exposure model is more appropriate under these experimental conditions.

In order to examine the effects of volatilization rates on critical time and critical concentration, the model was executed using the validated input parameters for *cis* -chlordane and varying the volatilization rate from 0.1 to 0.0001 [l/hour]. The results presented in Figure 3 indicate that T_c varies from 17.1 to 149.8 hours and $Conc_{crit}$ decreases from 183.1 to 76.2 ng/g. It is important to note that the maximum concentration in fish changes significantly depending on the rate of volatilization from water.

Based on this investigation, the use of the non-constant exposure model may more accurately reflect the bioaccumulation in natural systems, where the aqueous concentration of organic chemicals may significantly change over time.

Table 1. Comparison of model parameters

Model	Chlordane	Uptake [mL/g/hour]	Elimination [1/hour]	BCF	Volatilization [1/hour]
Constant	*Trans-*	285.02	0.093	3065	0.0
Exposure	*Cis-*	261.53	0.093	2815	0.0
Nonconstant	*Trans-*	57.71*	0.015**	3826	0.02
Exposure	*Cis-*	57.71*	0.015**	3826	0.02

*$\alpha \times V$, ** $(\alpha \times V) / BCF$

Acknowledgments: The study was partially supported by funding from the Environmental and Occupational Health Science Institute, Piscataway New Jersey. The authors would like to thank Dr. Dona Schneider for encouragement and technical assistance.

REFERENCES

Barron MG, Stehly GR, Hayton WL (1990) Pharmacokintetic modeling in aquatic animals I. models and concepts. Aquat Toxicol 18:61-81.

Bruggeman WA, Martron LBJM, Kooiman D (1981) Accumulation and elimination kinetics of di-, tri- and tetrachlorobiphenyls by goldfish after dietary and aqueous exposure. Chemosphere 10:811-832.

Erstfeld KM (1986) Bioavailability of *trans*- and *cis* -chlordane as a function of their sorption and desorption in sediments. Ph.D. Dissertation University of Michigan Ann Arbor, Michigan 205pp.

Erstfeld KM, Simmons MS, Atallah YH (1989) Analysis of chlordane in laboratory-generated environmental fate samples. Int J Analyt Chem, 37:199-211.

Karara AH, McFarland VA (1992) A pharmacokinetic analysis of the uptake of polychlorinated biphenyls (PCBs) by golden shiners. Environ Toxicol Chem 11: 315-320.

Keen, RE, and Spain JD (1993) Computer simulation in biology. Wiley-Liss, New York, NY 498pp.

Landrum PF, Lee H, Lydy MJ (1992) Toxicokinetics in aquatic systems: model comparisons and use in hazard assessment. Environ Toxicol Chem 11:1709-1725.

Newman, MC (1995) Quantitative methods in aquatic ecotoxicology. Lewis Publishers, Boca Raton, FL 426pp.

Newman MC, Jogoe RH (1996) Bioaccumulation models with time lags: dynamics and stability criteria. Ecological Modelling 84:281-286.