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DETERMINISTIC AND STATISTICAL MODELS OF CHEMICAL FATE
IN AQUATIC SYSTEMS

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Deterministic and Statistical Models of Chemical Fate in Aquatic Systems

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This paper has several purposes: (a) to summarize the basic models of the steady state transport and fate of chemicals in aquatic systems including uptake and distribution in the aquatic food chain, (b) to illustrate the deterministic time variable behavior of chemical fate models with several applications to the Great Lakes and (c) to develop some statistical models of chemical variability in aquatic organisms, specifically, the fish.

The ability to analyze and predict the transport of potentially toxic chemicals is one of the central requirements of risk assessment and subsequent risk management. Steady state models can be of specific value in the early stages of chemical screening for generic problem contexts and to elucidate basic principles of chemical fate and uptake into the food chain. Time variable models are particularly useful for predicting recovery times of aquatic systems following some abatement program of chemical control. These steady state and time variable models essentially estimate the average or deterministically varying chemical exposure concentration to aquatic organisms. Risk assessment also requires some evaluation of the stochastic behavior of chemicals both in the water and in fish. The paper is therefore divided into four parts: 1) the basic theory and associated equations; 2) steady state simplifications; 3) deterministic time variable models and 4) analytical and numerical models of statistical behavior of chemicals in fish.

I. THEORY

A. Physical-chemical Fate and Transport Model

The principal components of the physical-chemical fate and transport model framework are reviewed in Thomann and Mueller (1987), Delos et al. (1984), Thomann and Di Toro (1983), and Di Toro et al. (1981), among others.

The development can begin by considering a simple one-dimensional river as shown in Figure 1. The chemical in the water column is transported by the flow Q . Losses of chemical may occur as a result of microbial degradation, volatilization or other pathways. The sediment however in all of the models discussed in this paper is not considered to be moving. There is a transfer of chemical from the sediment to the water column and vice versa via settling and resuspension of particulate chemical forms and sediment diffusion of dissolved chemical.

The one-dimensional mass balance equation for any form of the chemical (dissolved or particulate) is for the water column

$$\frac{\partial c_1}{\partial t} = - \frac{1}{A} \frac{\partial}{\partial x} (Qc_1) + \frac{1}{A} \left[\frac{\partial}{\partial x} EA \frac{\partial c_1}{\partial x} \right] + \text{sources} - \text{sinks} \quad (1)$$

and for the surface sediment

$$\frac{\partial c_2}{\partial t} = \text{Sources} - \text{Sinks} \quad (2)$$

where c_1 and c_2 are the chemical concentrations in the water column and sediment [M_T/L^3 ; M_T = mass of toxicant, L^3 = bulk volume of solids plus water], Q is the river flow [L^3/T], A is the cross-sectional area [L^2], E is the longitudinal dispersion coefficient [L^2/T], x is distance downstream and t is time.

The chemical in the models discussed herein is assumed to be composed to two forms: 1) the dissolved form, c'_d [M_T/L^3 ; L^3 = volume of water], and 2) the particulate form, c_p [M_T/L^3], i.e. the toxicant sorbed onto particulate matter in the water column or sediment. The total chemical concentration is then

$$c_T = c_p + \phi c'_d \quad (3)$$

where ϕ is the porosity [L^3/L^3].

Eq. (3) is

$$c_T = c_p + c_d \quad (4)$$

where

$$c_d = \phi c'_d \quad (5)$$

for c_d [M_T/L^3] as the porosity corrected dissolved concentration.

With the general framework described, the detailed equations for the various forms of the chemical can be presented.

Dissolved chemical. An explicit finite differencing of Equation 1 together with sources and sinks of the dissolved chemical in a temporarily constant control volume (V_1) of the water column is given by

$$\begin{aligned} V_1 \frac{dc_{d1}}{dt} = & [(Qc_{d1}^+ - Q_1c_{d1}) + E'((c_{d1})^+ - c_{d1}) + E'((c_{d1})^- - c_{d1})] \quad (\text{Transport}) \\ & + k_{d1}V_1c_{p1} - k_{u1}m_1V_1c_{d1} \quad (\text{Sorption-desorption}) \\ & + K_{f12}A(c_{d2}^i - c_{d1}^i) \quad (\text{Sediment diffusive exchange}) \\ & - K_{d1}V_1c_{d1} \quad (\text{Decay \& losses}) \\ & - k_{t1}A(c_g/H_e - c_{d1}^i) \quad (\text{Volatilization}) \\ & + W_{d1} \quad (\text{Input}) \quad (6) \end{aligned}$$

The group of terms in brackets represents the transport and dispersion of the dissolved toxicant. Superscript + indicates the upstream direction and superscript - indicates the downstream direction. The net transport flows, Q , are written in an equivalent backward difference approximation to the underlying partial differential equation (Eq. 1). The dispersion or mixing between segments of length Δx is given by the bulk dispersion coefficient which in turn is related to the dispersion coefficient by

$$E' = \frac{EA}{\Delta x} \quad (7)$$

The second line on the left of Eq. (6) is the balance between the desorption of the chemical in the particulate phase ($k_{d1}V_1c_{p1}$) which increases the dissolved form (the desorption rate is k_{d1} [1/T], and the

adsorption from the dissolved phase onto the particulates given by $k_{u1}m_1c_{d1}$. (The sorption rate is $k_{u1}[L^3/M_g-d]$ and the solids concentration is $m_1[M_g/L^3]$. Note that this latter term depends on the mass of solids available for sorption from the dissolved phase.

The third line of Eq. (6) represents the diffusive exchange between the sediment dissolved chemical concentration c'_{d2} in the interstitial water and the dissolved chemical concentration in the water column, c'_{d1} . The sediment-water diffusive transfer coefficient, K_{f12} [L/T] can be considered as an overall interfacial transfer coefficient relating to the diffusion of the toxicant across the sediment-water interface.

Decay and loss mechanisms such as biodegradation, photolysis etc. of the dissolved form are included in the fourth line of the equation. Therefore, K_{d1} [1/T] represents the sum of individual rates, some of which in turn may represent rather complex mechanisms. Note that for this model all the loss rates are assumed to be first order.

Volatilization of the dissolved toxicant is given by the fifth line of Eq. (6) where c_g represents the gas phase of the chemical [M_T/L_g^3 ; L_g^3 = volume of gas] which may or may not be zero, and H_e is the Henry's constant for the chemical [$M_T/L_g^3 + M_T/L_w^3$].

The last line represents all external sources or inputs of dissolved chemical, W_{d1} [M_T/T] from point direct discharge sources as well as non-point and tributary inputs.

An equation similar to Eq. (6) can be written for the dissolved chemical in the sediment layer underneath the typical water column segment 1. This layer is designated with the subscript 2. Thus,

$$\begin{aligned} V_2 \frac{dc_{d2}}{dt} = & k_{d2}V_2c_{p2} - k_{u2}m_2V_2c_{d2} \\ & + K_{f12}A(c_{d1}^i - c_{d2}^i) \\ & - K_{d2}V_2c_{d2} \\ & - V_{d2}Ac_{d2} \\ & + K_{f23}A(c_{d3}^i - c_{d2}^i) \quad (8) \end{aligned}$$

The first three lines of the right side of Eq. (8) have already been discussed relative to the water column. The fourth line of Eq. (8) expresses the "burial" or transfer down into the sediment of the dissolved toxicant due to net sedimentation or build-up of the sediment layer at a net sedimentation rate of $v_{d2}[L/T]$. The last line of Eq. (8) is the diffusive exchange of dissolved toxicant between the first and second sediment layers under the water column. Similar equations can be written for each successive sediment layer. Note that there are no dissolved transport terms for the sediment thereby indicating that the sediment is assumed to be stationary in the horizontal direction. Also, mechanical mixing of sediment layers (due, for example, to bioturbation) is not included, but is readily added with an additional mixing term.

Particulate chemical. The mass balance equation for the chemical sorbed onto the particulates in the water column segment 1 is given by

$$v_1 \frac{dc_{p1}}{dt} = [(Qc_{p1})^+ - Q_1c_{p1} + E'((c_{p1})^+ - c_{p1}) + E'((c_{p1})^- - c_{p1})](\text{Transport})$$

$$- k_{d1}v_1c_{p1} + k_{u1}v_1c_{d1} \quad (\text{Desorption-sorption})$$

$$- v_sAc_{p1} \quad (\text{Particulate settling})$$

$$+ v_uAc_{p2} \quad (\text{Particulate resuspension})$$

$$- K_{p1}v_1c_{p1} \quad (\text{Decay})$$

$$+ W_{p1} \quad (\text{Input}) \quad (9)$$

The first line of this equation is the transport of the particulate chemical due to net advection (Q) and dispersion (E'). The particulate chemical is assumed to be transported in the same manner as the dissolved form. The second line is the sorption-desorption mechanism discussed above and as can be noted for the particulate form, sorption is a source and desorption is a sink of toxicant. The third and fourth lines are respectively the particulate settling of the chemical from the water column and the resuspension of particulate chemical from the sediment into the water column. The settling velocity, $v_s[L/T]$ and the resuspension

velocity $v_u[L/T]$ are functions of particle type (sand, silt, organics) and the hydrodynamics of the water-sediment interface. The fifth line represents any decay mechanisms (e.g. bacterial degradation) of the chemical on/in the particulates at a rate $K_{p1}[1/T]$ and the last line is the external mass input of particulate toxicant, $W_{p1}[M/T]$.

The particulate chemical in the sediment is given by an equation similar to Eq. (9) except that, as noted, the sediment is assumed to be stationary in the horizontal direction. That is, bed load transport or sediment movement horizontally throughout the water body is not considered.

The particulate chemical equation for the sediment segment underlying the water column segment 1 is then given by

$$v_2 \frac{dc_{p2}}{dt} = - k_{d2}v_2c_{p2} + k_{u2}v_2c_{d2}$$

$$+ v_sAc_{p1} - v_uAc_{p2}$$

$$- K_{p2}v_2c_{p2}$$

$$- v_dAc_{p2} \quad (10)$$

The first three lines of this equation parallel the equivalent mechanisms in the water column (sorption-desorption, settling-resuspension and decay, at rate K_{p2}). The fourth line represents the net downward flux of sediment particulate toxicant due to the net sedimentation velocity v_d . Again, mixing of the sediment due to factors such as bioturbation or deep sediment mixing is not included, but can be added as an additional mixing term.

B. Local Equilibrium Equations

Eqs. (6) and (8) for the dissolved component and Eqs. (9) and (10) for the particulate component in the water column segments and sediment segments respectively represent a set of interactive, differential equations, one for each control volume of the finite difference grid. Note that the coupling of the dissolved and particulate components is through the reaction kinetics of sorption and desorption. For some chemicals,

these reaction kinetics tend to be "fast" (i.e. completion times on the order of hours) compared to the kinetics inherent in other mechanisms of the problem. These latter mechanisms include bacterial decay, net loss rates to the sediment and sedimentation rates that have reaction times on the order of days to years.

The "fast" kinetics of sorption-desorption indicate that for time scales of days to years, there will be a virtually continuous equilibration of the dissolved and particulate forms depending on the local solids concentration. This partitioning between the two components permits the specification of the fraction of dissolved and particulate chemical to the total. The dissolved and particulate chemical are therefore assumed to be always in a "local equilibrium" with each other. Assuming that the kinetics are reversible and that the sorption/desorption kinetics are linear, then a partition coefficient $q[M_T/M_S + M_T/L_W]$ can be defined as follows:

$$q = r/c_d' \quad (11)$$

or since $c_d' = c_d/\phi$

$$q' = q/\phi = r/c_d \quad (12)$$

for q' as $[M_T/M_S + M_T/L^3]$ and r as the chemical concentration on a solids basis $[M_T/M_S]$.

The particulate toxicant concentration relative to the bulk volume is given by

$$c_p = r\phi \quad (13)$$

The fraction of the total that is dissolved, f_d , is given by

$$f_d = (1 + q'm)^{-1} \quad (14)$$

and the particulate chemical as a fraction of total chemical (f_p) is given by

$$f_p = \frac{q'm}{1 + q'm} \quad (15)$$

The local equilibrium assumption therefore permits specification at all times and places of the fraction of the total toxicant in the dissolved and particulate form. It should be stressed again here that this local equilibrium assumption assumes complete reversibility between the solid and liquid phases. There is evidence (e.g. Di Toro, et al. 1982a, and Di Toro, 1985) that this is not the case for certain chemicals.

Also in these relationships it is assumed that the partition coefficient does not depend on the concentration of the sorbing solids. There is considerable evidence, however, as given by O'Connor and Connolly (1980) and Di Toro (1985) who indicate that the partition coefficient does apparently depend on the concentration of solids. The development continues here on the assumption of a constant partition coefficient.

With this assumption, attention can then be focused solely on the mass balance equation for the total chemical. The total chemical in the water column or sediment is given by Eq. (3). Adding the water column equations for dissolved chemical (Eq. 6) and particulate chemical (Eq. 9) and using Eqs. 14 and 15 gives

$$\begin{aligned} V_1 \frac{dc_{T1}}{dt} = & [Qc_{T1}^+ - Q_1c_{T1} + E'(c_{T1}^+ - c_{T1}) + E'(c_{T1}^- - c_{T1})] \\ & + K_f A(f_{d2}c_{T2}/\phi_2 - f_{d1}c_{T1}) - (K_1)V_1c_{T1} \\ & + k_{L1}A[(c_g/H_g) - f_{d1}c_{T1}] - v_s A f_{p1}c_{T1} + v_u A f_{p2}c_{T2} \end{aligned} \quad (16)$$

$$\text{where } K_1 = K_{d1} + K_{p1}$$

Note that the kinetics of sorption-desorption do not appear in this equation because it represents a mass balance of the total. The net loss rates and exchanges that are dependent on the form of the toxicant do however, remain.

A total chemical equation for the sediment segment (subscript 2) can be obtained in a similar manner. Thus adding Eqs. (8) and (10) gives:

$$v_2 \frac{dc_{T2}}{dt} = -K_f A(f_{d2} c_{T2}/\phi_2 - f_{d1} c_{T1}) - (K_2) v_2 c_{T2} \\ + v_s A f_{p1} c_{T1} - v_u A f_{p2} c_{T2} - v_d A f_{p2} c_{T2} + K_f A(f_{d3} c_{T3}/\phi_3 - f_{d2} c_{T2}/\phi_2) \quad (17)$$

$$\text{where } K_2 = K_{d2} + K_{p2}$$

Eqs. (16) and (17) are the fundamental equations used in the succeeding analyses. These equations are coupled parametrically to the suspended solids and sediment solids concentrations (see Eqs. 14 and 15). These concentrations can be specified externally as an input or the mechanisms of solids settling, resuspension and deposition can be explicitly modeled. In addition, an independent tracer can be used to calibrate these parameters (see Thomann and Di Toro, 1983 for the use of plutonium-239,240 as a tracer).

C. Food Chain Model

The transfer of a chemical in the aquatic food chain occurs through two principal routes:

- 1) direct uptake from the water
- 2) accumulation due to consumption of contaminated prey

The uptake of a chemical directly from water through transfer across the gills as in fish or through surface sorption and subsequent cellular incorporation as in phytoplankton is an important route for transfer of chemicals. This uptake is often measured by laboratory experiments where test organisms are placed in aquaria with known (and fixed) water concentrations of the chemical. The accumulation of the chemical over time is then measured and the resulting equilibrium concentration in the organism divided by the water concentration is termed the bioconcentration factor (BCF). A simple representation of this mechanism is given by a mass balance equation around a given organism. Thus,

$$\frac{dv'}{dt} = k_u w c_d' - K v' \quad (18)$$

where v' is the whole body burden of the chemical (M_T), k_u is the uptake sorption and/or transfer rate ($L^3/T \cdot M(w)$; $M(w)$ = mass of organism, wet weight), w is the weight of the organism ($M(w)$), c_d' is the dissolved water concentration (M_T/L^3), K is the desorption and excretion rate ($1/T$) and t is time. This equation indicates that the mass input ($\mu g/d$) of toxicant given by $k_u w c_d'$ is offset by the depuration mass loss rate ($\mu g/d$) given by $K v'$. The whole body burden v' is given by

$$v' = v w \quad (19)$$

where v is the concentration of the chemical ($M_T/M(w)$). Substitution of (19) into (18) gives, after simplification

$$\frac{dv}{dt} = k_u c - K' v \quad (20)$$

$$\text{where } K' = K + G \quad (20a)$$

for $G(1/T)$ as the net growth rate of the weight of the organism. At equilibrium or steady state,

$$v = \frac{k_u c}{K'} \quad (21)$$

and the BCF is given by

$$N_w = \frac{v}{c} = \frac{k_u}{K+G} \quad (22)$$

The ratio N_w , the bioconcentration factor, is in units $M_T/M(w) + M_T/L^3$, e.g., $\mu g/kg + \mu g/l (= l/kg)$.

For organic chemicals, the BCF is conveniently defined on a lipid normalized basis, i.e. $M_T/M(lip) + M_T/L^3$, e.g., $\mu g/kg(lipid) + \mu g/l$. The lipid normalization assumes that the lipid compartment of the organism is the principal receptor of the hydrophobic organic chemical.

The octanol-water partition coefficient (K_{ow}) of a chemical is a useful ordering parameter to express the tendency of the chemical to partition into the lipid pool.

At equilibrium then for organic chemical BCF, to first approximation,

$$N_w = K_{ow} \quad (23)$$

for the laboratory case of no organic growth and N_w as the lipid normalized BCF. Thomann (1987) suggests the following expression for the field BCF as a function of K_{ow}

$$N_w = K_{ow} \left[1 + \frac{10^{-6} K_{ow}^{-1}}{E(K_{ow})} \right] \quad (24)$$

where $E(K_{ow})$ is an efficiency of chemical transfer across the gills as a function of K_{ow} and can be approximately expressed as

$$\begin{aligned} \log E &= -1.5 + 0.4 \log K_{ow} \quad \text{for } \log K_{ow} = 2-3 \\ E &= 0.5 \quad \text{for } \log K_{ow} = 3-6 \\ \log E &= 1.2 - 0.25 \log K_{ow} \quad \text{for } \log K_{ow} = 6-10 \end{aligned} \quad (25)$$

Age-Dependent Model

The general age-dependent model utilizes a mass balance of chemical around a defined compartment of the aquatic ecosystem. In the most general case, a compartment is defined as a specified age class of a specified organism or in steady state simplification, a compartment is considered as an "average" age class or range of ages for a given organism. Figure 2 schematically shows the compartments. As indicated in Figure 2(a), each age class of a given trophic level is considered as a compartment and a mass balance equation can be written around each such age class. The zero trophic level is considered to be the phytoplankton-detritus component representing one of the principal sorption mechanisms for incorporating toxicants into the food chain.

Consider then the phytoplankton, detrital organic material, and other organisms, all of size approximately $< 100 \mu m$ as the base of the food chain. An equation for this compartment is given by a simple reversible sorption-desorption linear equation as:

$$\frac{dv_0}{dt} = k_{uo}c - K_o v_0 \quad (26)$$

where all terms have been defined, the subscript zero refers to the base of the food chain, and t is real time.

For a compartment above the phytoplankton/detritus level, the mass input of the toxicant due to ingestion of contaminated food must be included. This mass input will depend on a) toxicant concentration in the food, b) rate of consumption of food and c) the degree to which the ingested toxicant in the food is actually assimilated into the tissues of the organisms.

The general mass balance equation for the whole body burden for a given compartment, i , is then similar to Eq. (18) for water uptake but with the additional mass input due to feeding. Therefore,

$$\begin{aligned} \frac{dv'_i}{dt} &= \frac{d(vw)_i}{dt} = \frac{w_i dv_i}{dt} + \frac{v_i dw_i}{dt} = k_{ui} v_i c - K_i v'_i \\ &\quad + \sum_j p_{ij} a_{ij} C_j v_j \quad i = 1 \dots m \end{aligned} \quad (27)$$

where a_{ij} is the chemical assimilation efficiency (M_T absorbed/ M_T ingested), C_j is the weight-specific consumption of organism j ($M(w)$ predator/ $M(w)$ prey-d), p_{ij} is the food preference of i on j , and t is real time (days). Consider now a simple case of a sequential food chain where predation is only on the next lowest trophic level.

An equation for the individual organism weight is

$$\frac{dw_i}{dt} = (a_{i,i-1} C_{i-1} - r_i) w_i \quad i = 1 \dots m \quad (28)$$

where $a_{i,i-1}$ is the biomass assimilation efficiency ($M(w)$ predator/ $M(w)$ prey) and r_i is the respiratory weight loss ($1/T$) due to routine metabolism, swimming, and other activities. The weight change is therefore

$$G_i = \frac{dw_i}{dt} / w_i = (a_{i,i-1} C_{i-1} - r_i) \quad (29)$$

Equation (27), for a food chain in contrast to a food web, can then be written as

$$\frac{dv_i}{dt} = k_{u1}c + a_{i,i-1}C_{i-1}v_{i-1} - K'_i v_i, \quad i = \dots, m \quad (30)$$

where i is the predator and $i-1$ is the prey.

The interpretation of w_i and v_i in Equations (28) and (30) is further explained in Figure 3. The variation of the weight (and chemical organism concentration) of a given compartment (i.e. a given age class of a given organism) is shown with real time. If as an example, w_5 is a 0-1 year old alewife then it is seen that the weight of this age class may vary from year to year. Similarly, the distribution of the chemical may change from year to year for a given compartment depending on, for example, the variation in the water column toxicant concentration. The specification of the boundaries of a given compartment depends on the life cycle of the organism.

It should be recognized therefore that several of the biological and chemical parameters of the weight change equation (29) and the food chain equation (30) are functions of organism weight within the time interval defined by the compartment. See Thomann and Connolly, 1984, for an application of the age dependent model to PCB accumulation in the lake trout of Lake Michigan.

II. STEADY STATE SIMPLIFICATION

A. River Physio-Chemical Fate

In spite of the complexity of Equations (16) and (17), it can be shown (see e.g. Thomann and Mueller, 1987) that under steady state and constant spatial parameters that for a single source to first approximation, the maximum concentration c_0 is at the outfall. Thus

$$c_0 = (Q_u c_u + Q_e c_e) / Q$$

where Q_u and c_u are the upstream flow and concentration, respectively, Q_e and c_e are the effluent flow and concentration respectively, and Q is the total river flow.

Since the maximum generally occurs at the outfall, there are generally two situations where one would be interested in estimating the downstream fate of a discharged chemical:

a) There is a critical water use point downstream of a single discharge and the concentration at the point of use (e.g., a water supply withdrawal) needs to be estimated.

b) There are several inputs of the same chemical along the length of the river and the total concentration must be estimated.

The downstream fate of the chemical or mixture depends on:

a) the properties of the river such as the depth, velocity and dilution downstream due to groundwater infiltration or tributary inflow,

b) the chemical properties, such as volatilization, biodegradation, or partitioning onto the solids.

Thomann and Mueller (1987) discuss these factors in some detail. A simplified summary (from Thomann and Salas, 1986) is given here. An important point for the computation of the downstream fate of a chemical or chemical mixture is that the calculation is very similar, indeed for preliminary analyses, the computation is identical to classical stream water quality calculations. The basic equation under steady state conditions is given by

$$c = c_0 \exp[-(K_T + q)t] = c_0 \exp[-(\frac{v_T}{HU} + q)x] \quad (31)$$

$$= c_0 \exp[-(K + q')t^*]$$

where c is the concentration as a function of distance downstream, c_0 is the chemical concentration or toxicity in the river after mixing of the outfall, v_T is the net loss of chemical expressed as a velocity [L/T], t^* is the time of travel ($= x/u$), q is the slope of the natural logarithm of river flow with distance, H is river depth, U is river velocity and q' is the river flow slope on a travel time basis.

The calculation of the downstream fate of a toxic substance or toxicity of an effluent or effluents depends then on the estimation of

the dilution of the river and the loss rate of the chemical. The discharge of a conservative substance with no dilution would result in a constant concentration in the downstream direction, i.e., from Eq. (31) with K_T and $q' = 0$, $c = c_0$.

The determination of whether there is an downstream infiltration of groundwater or overland drainage can be made by examining the downstream distribution of a known conservative substance such as chlorides or total dissolved solids or other tracer.

If dilution exists then a conservative substance may exhibit apparent non-conservative behavior. Then for $K_T = 0$, but $q' \neq 0$, Eq. (31) is

$$c = c_0 \exp(-qt^*) \quad (32)$$

which indicates an exponential decline in the conservative substance due to distributed downstream dilution.

Finally, if the toxic substance is non-conservative, i.e., the chemical undergoes biodegradation or volatilization or other losses and dilution is also occurring then the net loss rate of the chemical, K_T must be estimated.

Table 1 provides some guidelines for a preliminary assessment of downstream fate. Toxicity in this Table is whole-effluent toxicity of the chemical mixture to standard organisms and is expressed on a toxic unit basis. A toxic unit is defined

$$Tu = 100/LC50 \text{ or NOEL} \quad (33)$$

where LC50 is the lethal concentration to 50% of the organisms and NOEL is the no observed effect level. As indicated, both the heavy metals and the toxicity measure are assumed to be a conservative variable for first approximations. Thus, only dilution need be considered for these variables. For the organic chemicals a somewhat arbitrary division has been made based on the water solubility of the chemical.

The rationale is that at solubilities less than about 1 $\mu\text{g/l}$, the chemical will partition onto the solids because of a relatively high partition coefficient (about 10^4 - 10^6 l/kg). Also, the general tendency

will be for such chemicals to biodegrade and volatilize to a lesser degree than the more soluble chemicals. For first approximations, however, such low solubility chemicals may be assumed conservative.

For organic chemicals with solubilities greater than about 1 $\mu\text{g/l}$, the loss rate must be estimated. For first approximations, the net loss velocity $v_T (= K_T H)$ can be estimated from

$$v_T = K_T H = (K_d H + k_L) f_d + v_n f_p \quad (34)$$

where $K_d [T^{-1}]$ is the decay rate of the chemical due to processes such as biodegradation or photolysis, k_L is the loss due to volatilization $[L/T]$, v_n is the net loss velocity of the solids in the river $[L/T]$ and f_d and f_p are the dissolved and particulate fractions of the total (see Eqs. 14 and 15).

For rivers, the net loss of solids often, although not always, can be assumed equal to zero. Thus, $v_n = 0$ and the chemical loss rate depends only on the degradation rate, volatilization rate and fraction dissolved.

Table 2 provides some guidelines for estimating the fraction of the chemical that is in the dissolved form. These guidelines employ a more complex interaction of chemical partitioning and solids concentration as given in Di Toro (1985) and discussed in Thomann and Mueller (1987). The f_d can also be approximated with Eq. 14. Fig. 4 shows the range of the volatilization loss rate as a function of the river depth and reaeration characteristics. This Figure is for substances with Henry's constant $> 10^{-4}$ atm-m³/mol for which the volatilization rate is estimated from the oxygen transfer rate K_L . Thus,

$$k_L = \left(\frac{32}{H}\right)^{1/4} K_L \quad (35)$$

$$\text{where } K_L = (D_L \frac{U}{H})^{1/2} \quad (36)$$

for D = oxygen diffusivity (.000181 m²/d).

The loss rate, K_d , is generally site-specific and chemical specific and no general simplification is available.

Table 2 also gives the approximate ratio of the chemical adsorbed to the suspended particulates to the total water concentration. Therefore,

$$R = r/c_T \quad (37)$$

for R in $\mu\text{g/g(d)} + \mu\text{g/l}$, r in $\mu\text{g/g(d)}$ and c_T in $\mu\text{g/l}$ ($\text{g(d)} = \text{grams dry weight}$).

It can also be shown that for $V_2 = V_1$ and $K_{d2} = 0$, the sediment particulate concentration is equal to the water column particulate concentration, i.e.,

$$r_2 = r_1 \quad (38)$$

where r_2 is the sediment particulate concentration and r_1 is the water column particulate concentration both in $\mu\text{g/g(d)}$.

In summary, it is seen that the calculation of the fate of the chemical or mixture is similar to the procedure for conventional water quality variables. For many chemicals including the toxicity measure, the assumption that the chemical is conservative is appropriate for first approximations. If such an assumption cannot be made, then an estimate of the downstream loss rate must be made using the preliminary guidelines discussed here.

III. DETERMINISTIC TIME VARIABLE MODELS

A. Application of Time Variable Model to Benzo(a)Pyrene and Cadmium in the Great Lakes

In this section, the fully time-variable model (Eqs. (16) and (17)) is applied to two chemicals: a) benzo(a)pyrene, a polycyclic aromatic hydrocarbon (PAH), and b) cadmium, a representative metal. The model uses the Great Lakes segmentation of Thomann and Di Toro (1983) shown in Figure 5 and details are in Thomann and Di Toro (1984).

Benz(a)pyrene

The distribution of this chemical, one of the PAH compounds resulting from incomplete combustion of organic materials has been widely studied (e.g., Neff, 1979) because of its potential carcinogenicity. The fate of benzo(a)pyrene (BaP) in the Great Lakes has recently been evaluated in a series of papers by B.J. Eadie (Eadie et al., 1982; Eadie, 1983; Eadie et al., 1983). In that work, data are presented for the range of concentration of BaP in the water column and surficial sediments as well as preliminary data on the BaP concentration in the pore water of the sediment. It is those data (together with estimates of loading) that can be used as an application of the physico-chemical model.

BaP is sparingly soluble in water $0.172 \mu\text{g/L}$ (Neff, 1979) and as such would be expected to have an affinity for solids. The partitioning onto particulates and in addition, the extent of volatilization of the BaP must be estimated. Although BaP is known to undergo photolysis (Neff, 1979) this pathway is not considered in this application.

The estimated atmospheric loading on an areal basis is about $95 \text{ g BaP/km}^2\text{-yr}$ across all of the lakes but as noted by Eadie (1983), all BaP load estimates are based on quite limited data and therefore may vary as additional information becomes available. From the data given in Eadie (1983) for Lake Michigan, and other data, the model for BaP was run for V from 10,000 to 100,000 l/kg , thereby providing a range of one order of magnitude in the solids partition coefficient.

The time variable calculation using a steady loading for 20 years is shown in Table 3 and in Figures 6 and 7. As shown in Table 3, the lake to lake variation in BaP concentrations either in the water column or the sediment differs by less than about a factor of two. The highest concentration of BaP in the surface sediments for $V = 10,000$ is in Saginaw Bay. It can also be noted in Table 3 and Figure 6 that at $V = 10,000$ the calculated surface sediment concentration for Lakes Michigan and Erie is about 45 ng/g(d) or about one order of magnitude lower than the observed data. Figure 7 shows the calculated time history under the two partition coefficients. For $V = 10,000 \text{ l/kg}$, the water column

and sediment are at about 80 and 60% of steady state respectively while for $\tau = 100,000$ t/kg, the water column and sediment are at about 20% of steady state.

Figure 6 and Table 3 indicate that a more favorable (but not totally desirable) comparison to observed data is obtained at the higher BaP partition coefficient of 100,000 t/kg. The results also indicate the need to determine the partition coefficient for BaP, as a representative PAH for Great Lakes solids concentrations. On the basis of this application of the physico-chemical model to BaP in the Great Lakes, it is concluded that:

(1) the estimate of the BaP partition coefficient obtained from published empirical relationships is probably low by about an order of magnitude for the Great Lakes system,

(2) with an increased BaP partition coefficient and assuming loss due to volatilization, the physico-chemical toxic substances model of the Great Lakes approximate observed BaP water column and sediment data only to order of magnitude,

(3) the model confirms that on a lake-wide scale, the principal external source of BaP is the atmosphere,

(4) for the larger lakes such as Michigan, the 50% response time of the lake to external loads is about 6-10 years for the water column-sediment system while for Lake Erie the response time is about two years,

(5) lake to lake variations in BaP water column and sediment concentrations are less than a factor of two.

Cadmium

Time variable model calculations for cadmium were made using the low and high load estimate of Table 4 and two assumptions on the cadmium partition coefficient: a) variable partition with solids concentration as given in HydroQual (1982) by

$$K = (3.52 \cdot 10^6) C_s^{-0.92} \quad (39)$$

and b) a constant partition coefficient of $2 \cdot 10^5$ t/kg. Cadmium was assumed to be conservative. For all calculations, zero initial conditions were assumed and the loads were inputted as constant over time. It became apparent from initial runs that the time to steady state especially for the upper lakes is long so that the computation was carried out for a 100 year period. The computation therefore represents the response of the Great Lakes system to a constant loading and such loading can be viewed as a loading in addition to the background loading and cadmium concentrations shown in Table 5. If increased loading of cadmium is assumed to have begun in approximately the 1920's then the output from the model calculation at $t = 50$ years would be representative of the 1970's, the period during which some reliable data are available.

Figure 8 shows the comparison of calculated and observed surface sediment cadmium data for $t = 50$ years. As shown, the calculation is reasonable to order magnitude. Figure 9 (high load estimate) show the full 100 year calculation for Lake Michigan and central Lake Erie as illustrations. The sensitivity of the calculation to the assumptions in the partition coefficient is shown. As indicated, the effect of the solids dependent partition coefficient is to greatly increase the time to steady state as a result of the diffusive flux of cadmium from the sediment due to the lower partition coefficient. For Lake Michigan, the surface sediment concentration decreases with the variable partition coefficient but for Lake Erie the surface sediment concentration increases. The continual increase in concentration for central Lake Erie reflects the non-equilibrium condition of the upstream Lakes.

If a constant partition coefficient is assumed (as in Muhlbaier and Tissue, 1981), then it is seen that for the water column a steady state is reached in about 25 years for Lake Michigan and about 10 years for Lake Erie. A calculation then for Lake Michigan that attempts to calibrate to a mean concentration of 27 ng/l (Muhlbaier and Tissue (1981)) then is simply a matter of estimating the approximate average load and may not reflect a non-steady state condition as concluded by Muhlbaier and Tissue (1981). However under a solids dependent partition coefficient for cadmium, the Great Lakes are not in equilibrium with

the external load and for all practical purposes never reach a steady state condition. Clearly then, under this model construct, it is important to determine the solids dependence of cadmium for the range of solids encountered in the Great Lakes water column and sediments (i.e. 0.5-240,000 mg/l). If however it is assumed that a solids dependent partition coefficient as given by Eq. (?) is applicable to the Great Lakes, then the system is not in equilibrium with respect to the external load.

It is concluded from this application of the time variable physico-chemical model to cadmium in the Great Lakes that

- 1) The degree of any dependence of the cadmium partition coefficient with solids has a marked effect on time to steady state and interstitial cadmium concentration.
- 2) Under a solids dependent cadmium partition assumption, the Great Lakes, especially the upper Lakes, do not reach a steady state condition after 100 years of constant loading.
- 3) Under a constant partition coefficient for cadmium, the Lakes do reach an equilibrium condition varying from about 25 years for Lake Michigan to years for Lake Erie.
- 4) The concentration of cadmium in the Lakes would be expected to increase by about 60% over the next 50 years if the average cadmium loading for the preceding 50 years continues.
- 5) Based on assumed sediment cadmium concentrations for Lake Erie, it is estimated that the cadmium concentration in the water column is about an order of magnitude higher than the other Lakes.

IV. STATISTICAL VARIATION IN FISH

The statistical behavior of a chemical in fish is of interest for at least three reasons:

a. U.S. Food and Drug Administration action limits for fish have resulted in the closing of commercial fisheries because of excessive concentrations of chemicals in predators such as the striped bass in the Hudson estuary and surrounding waters. The ability to predict not

only the mean value of chemical in a fish but also the variance of the concentration is of importance in control strategies needed to reopen a fishery.

b. The relationships between variable exposure concentrations in the water column and resulting variability in fish and other organisms is also related to the subsequent acute and chronic toxicity effects on the organism, and, as such, a framework for predicting organism chemical variance is of value in elucidating toxic effects on aquatic animals.

c. The integration of the physico-chemical modeling framework (which includes external inputs of the chemical to the water column) and the biological modeling framework in a time variable sense to predict statistical properties in the water and fish is of particular value in a generalized risk assessment determination.

The variability of chemicals in water over time is large. Figure 10 from the Mississippi River is an example. Recognizing this variability in the water chemical concentration of the food chain model of Section I-C, analytical models of expected response in the fish can be developed.

A. Analytical Model of Statistical Variation of Organic Chemical in Fish - Water Uptake Only

Consider the case where the water concentration to which the fish is exposed is represented by a first-order autoregressive process. Thus, the autocorrelation function for the water concentration is assumed as

$$\rho_{ck} = \exp(-ak); k = 0, 1, 2, \dots, m \quad (39)$$

where ρ_{ck} is the normalized autocovariance, k is the lag number, m is the maximum number of lags and a is the exponential decay rate of the autocorrelation.

Now

$$\rho_{ck} = \rho_{c1}^k \quad (40)$$

and then from (39) and (40),

$$\rho_{c1} = \exp(-a) \quad (41)$$

For the synthetic generation of a time series x_t with a first-order autoregressive input, Bras and Iturbe (1985) give

$$(x_t - \mu) = \rho_1(x_{t-1} - \mu) + \sigma_x(1 - \rho_1^2)^{1/2} Z(t) \quad (42)$$

for mean μ , variance σ_x^2 and $Z(t)$ a standard normal deviate (mean = 0, variance = 1).

Thus if $a \gg 0$, i.e. the autocorrelation function drops rapidly to zero at about lag one, $\rho_1 \rightarrow 0$ and Eq. (42) gives

$$x_t - \mu = \sigma_x Z(t)$$

or a normally distributed uncorrelated random variable, the "white noise" case.

For the autocorrelation given by Eq. (39), Bendat and Piersol (1971) show that the spectrum for frequency f is

$$G_c^N(f) = \frac{4a}{a^2 + 4\pi^2 f^2} \quad (43)$$

where $G_c^N(f)$ is a normalized spectrum. Therefore,

$$G_c(f) = \sigma^2 G_c^N(f) \quad (44)$$

The variance of the fish concentration can then be shown to be (Thomann, 1987)

$$\sigma_v^2 = \sigma_c^2 k_u^2 4a \int_0^\infty (K'^2 + 4\pi^2 f^2)^{-1/2} (a^2 + 4\pi^2 f^2)^{-1/2} df \quad (45)$$

Completing the integration, the ratio of the coefficients of variation between the fish and the water is

$$r = \sqrt{\frac{K'}{a + K'}} \quad (46)$$

$$\text{where } r = \frac{v_v}{v_c}$$

for v_v and v_c as the coefficients of variation of the chemical in the fish and in the water, respectively. This remarkably simple result indicates the significance of the depuration rate plus growth rate, K' , as the principal controlling factors in generating relative variability in the fish concentration.

In Eq. 46, r depends primarily on K' because at low excretion rate, pulses in water concentration tend to be retained by the fish over a longer period of time than at high excretion rates. Similarly, as a fish increases in weight, concentration variability will tend to shift into the low frequency end of the variance spectrum.

Figure 11 shows the behavior of r from Eq. 46 and indicates that correlated water concentrations increases the variability of the fish concentration relative to water. This is a consequence of correlated inputs introducing more "low frequency" variations in water concentration which are not dampened by the fish. Thus additional variance propagates through the fish and is reflected in the increase in the coefficient of variation.

Laboratory data for evaluating this analytical development come from two papers of Oliver and Niimi (1983, 1985). In this work, rainbow trout were exposed to chlorinated and brominated organic chemicals for up to about 100 days.

In Oliver and Niimi (1983), the data reported include the individual fish concentrations and associated water concentrations and the accompanying statistics of mean and standard deviation. For Oliver and Niimi (1985) only the BCF are reported. To use the data in this research, the fish concentrations were calculated from the individual BCF values and the mean water concentration. These data are therefore only an approximation to the actual fish concentration.

As noted frequently by the authors, several of the chemicals did not reach equilibrium during the test. The preceding statistical development assumes a dynamic equilibrium has been reached. The computation of the variance would be severely biased by the increase in con-

centration to a dynamic equilibrium. With a few exceptions, those chemicals excluded by the authors have also been excluded herein.

Figure 12 (top) shows the results of compiling these data and calculating the r ratio: v_v/v_c . As a function of $\log K_{ow}$, it is seen that there is a general downward trend to the data from initial values of >1 at low $\log K_{ow}$. The four circled points at the high $\log K_{ow}$ value of >6 are exceptions. The chemicals are pentabromotoluene, pentabromoethylbenzene, hexabromobenzene and octachloronaphthalene. These points were also noted by Oliver and Niimi (1985) in their evaluation of the BCF data. No ready explanation is available for why these chemicals exhibit less relative variability than expected, although there are no data in this $\log K_{ow}$ range where r values are low. Indeed, the data indicate a trend towards decreasing r values to a minimum at about $\log K_{ow}$ of about 5.5 and then an increase to higher r values with increasing $\log K_{ow}$. This is not consistent with the previously developed theory as shown in Figure 12 (bottom).

This Figure shows the calculation of the theoretical r ratio under three different assumptions. For these calculations the growth rate of the rainbow trout was calculated from the reported data at an average level of about 0.01/d. Also, the excretion rate is given Eqs. 22 and 23 and the uptake rate as a function of the efficiency of transfer E . Thus,

$$K = \frac{10^{-3} w^{-0.2} E/p}{K_{ow}} \quad (47)$$

where p is the fraction lipid and E is given by Eqs. 25.

The first calculation assumed the water concentration was statistically "white noise", i.e. uncorrelated in time. As noted, this calculation underestimates r by a significant amount.

The second calculation using Eq. 46 assumed a correlated first order process ($\alpha = 0.9$) for the water concentration. Now, the lower bound of the r ratios is captured although at $\log K_{ow}$'s of >6 the model deviates from the data.

Case 3 is a calculation assuming some metabolism at a high rate of 1.0/d. This is essentially an additive factor to the excretion

rate and as seen, the calculation now approximates the data somewhat better. As noted however, this calculation is simply an hypothesized mechanism for increasing the loss rate of the chemical. In the theoretical development, the only parameter that influences the shape of the r function is the excretion (+ metabolism) rate. To capture the four circled points with the theory would require an increase in the effective excretion + metabolism rate to levels of >1.0 day.

In summary, the laboratory data of Oliver and Niimi show a general downward trend of the r ratio with $\log K_{ow}$, but with several notable exceptions at high $\log K_{ow}$. The theory approximately duplicates the observed data only with a high level of first order correlated water input at to about $\log K_{ow}$ of about 5.5-6.0. At higher $\log K_{ow}$'s, increased metabolism and/or excretion would be necessary to reproduce the observed data.

V. CONCLUSIONS

The physio-chemical and food chain model structures discussed herein provide a basis for understanding and predicting the fate and transport of chemicals in surface water systems. While the fully time variable equations appear formidable, steady state simplifications permit rapid assessment of chemical fate, both in the water column and in the food chain. Such initial screening models have the same structure as "classical" water quality models; notably a linear response to external inputs of chemicals.

The interaction of the sediment and water column plays a significant role and again under some reasonable assumptions, rapid estimates can be made of chemical concentrations in the sediment.

In a similar setting, the age dependent food chain models can be simplified at steady state and rapid screening assessments of chemical bioconcentration can be made. For organic chemicals, the octanol water partition coefficient is a useful ordering parameter. Long term time variable deterministic calculations for the Great Lakes indicate the significance of the sediment as a reservoir of the chemical. Response times to changes in external load are long (e.g. years) and are signifi-

cantly increased when sediment partition coefficients are assumed to be different from water column partition coefficients.

Stochastic variability in chemical concentration is high. Analytical models of the resulting variability of chemical concentration in fish indicates the importance of the excretion rate of the chemical. However, additional research is necessary to more fully describe the observed variance in laboratory experiments especially for chemicals with log octanol water partition coefficients greater than about 6.5.

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TABLE 1

Guideline for Estimating Downstream Loss Rate of Chemicals and Toxicity

Group	Guideline (1)
Heavy Metals	Conservative ($K_T = 0$) and additive
Toxicity	Conservative ($K_T = 0$) and additive
Organic Chemicals Water Solubility > 1 $\mu\text{g/l}$	Conservative ($K_T = 0$) and additive
Organic Chemicals Water Solubility > 1 $\mu\text{g/l}$	Estimate loss rate (Eq. 34)

(1) In all cases, dilution in the downstream direction must be included.

Source: Thomann and Sales (1986)

TABLE 2

Approximate Fraction of Total Chemical in Dissolved Form
and on ParticulatesORGANIC CHEMICALS

Chemical Solubility (µg/l)	f_d - Fraction of Chemical in Dissolved Form (1)		Ratio of particulate conc. to total conc., (2)	
	Range	Approx. Mean	Range	Approx. Mean
> 100	0.5-1.0	0.7	0-50	10
10-100	0.3-0.9	0.5	0.1-70	50
< 10	0.3-0.8	0.4	0.1-70	60

HEAVY METALS

0.6-1.0	0.8	0.4-16	2.5
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1) Approximated from solids dependent partition coefficient relationships of Di Toro (1985), solids range 10 → 1000 mg/l

$$2) \tau/c_T = (1 - f_d)/(.01 \rightarrow 1.0)$$

Source: Thomann and Selas, 1986

TABLE 3

Comparison of Calculated and Observed BaP for Great Lakes
Under Different Solids Partition Assumptions

Calculated Range of BaP Across All Lakes (a) q = 10,000 t/kg q = 100,000 t/kg			Observed Mean of BaP	Ref.
Total Water (conc. (ng/L))	5-6	1-2	12 ± 8 (b)	Eadie et al. (1983)
Surficial Sediment Conc. (ng/g(d))	38-60	46-133	Mich: 480 ± 246(7) (c) Erie: 255 ± 152(3) Sup.: 28(1) Hur.: 294(1) Ont.: 306(1)	Eadie (1983)
Sediment Pore Water Conc. (ng/L)	3-5	0.5-1.3	850 ± 1260	Eadie et al. (1983)
Particulate Conc. in Water Column (ng/g(d))	46-64	46-165	Mich: 200-400	Eadie (1983)
(a) After 20 years of loading (b) Mean ± Std. Dev. (c) () = No. of samples				

(a) After 20 years of loading

(b) Mean ± Std. Dev.

(c) () = No. of samples

TABLE 4
Summary of Contemporary External Cadmium Loads
(not including upstream loads)

Lake/Region	Atmospheric 1) mt/yr	Tributary 3) mt/yr	Mun. + Ind. 4) (mt/yr)	mt/yr	Total g/km ² -yr
Superior	41-108	13-126	0-0.3	54-234	651- 2823
Michigan	12-120	9-92	0.4-4	21-216	363- 3739
Huron	23-57	14-136	0.1-1	37-194	644- 3378
Sag. Bay 2)	3	2	0	5	1184
Erie-West	1-18	2-20	1-8	4-46	1321-15202
- Central	3-94	2-16	0.2-2	5-112	318- 7126
- East	1-38	1-12	0.1-0.5	2-51	320- 8155
Ontario	10-44	7-66	0.6-6	18-116	924- 5953

1) From Allena and Halley (1980)

2) From Dolan and Bierman (1982)

3) Assuming tributary flow at total cadmium conc. of 0.2-1.0 $\mu\text{g/l}$

4) At 0.5-5.0 $\mu\text{g/l}$ for direct municipal point sources

TABLE 5
Estimate of Approximate Background Cadmium Concentration for Great Lakes

Lake/Region	Range in Total Background Water Conc. (ng/l) ¹	Background Conc. Used in Load Estimate (ng/l)	Background Total Load (mt/yr) ²	External Background Load (mt/yr) ³	g/km ² -yr	Ratio: Contemp. Load Background Load
Superior	2.8- 5.6	4.0	6.1	6.1	37	18-76
Michigan	2.8- 5.6	4.0	3.1	3.1	54	7-69
Huron	2.8- 5.6	4.0	5.2	4.8	83	8-41
Sag. Bay	6.5-13.0	10.0	0.6	0.4	105	11
Erie-West	12.5-25.0	20.0	10.3	8.6	283	5-54
-Central	5.0-10.0	8.0	22.0	10.0	635	0.5-11
-East	5.0-10.0	8.0	12.0	4.7	754	0.4-11
Ontario	2.8- 5.6	4.0	4.0	2.6	132	7-45

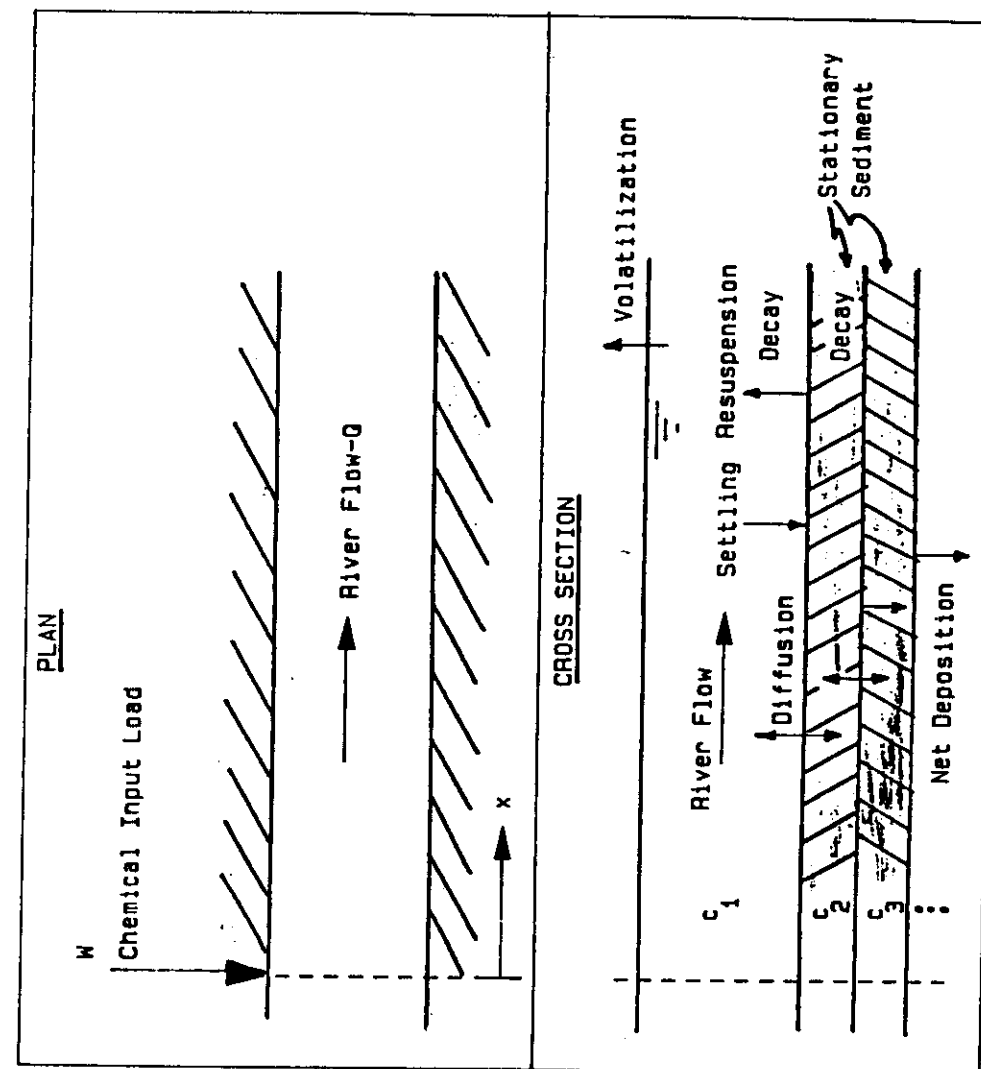
1) At assumed background sediment conc. of 05-1.0 $\mu\text{gCd/g(d)}$; $r_2 = r_1$ and $q = 200,000 \text{ t/kg}$.

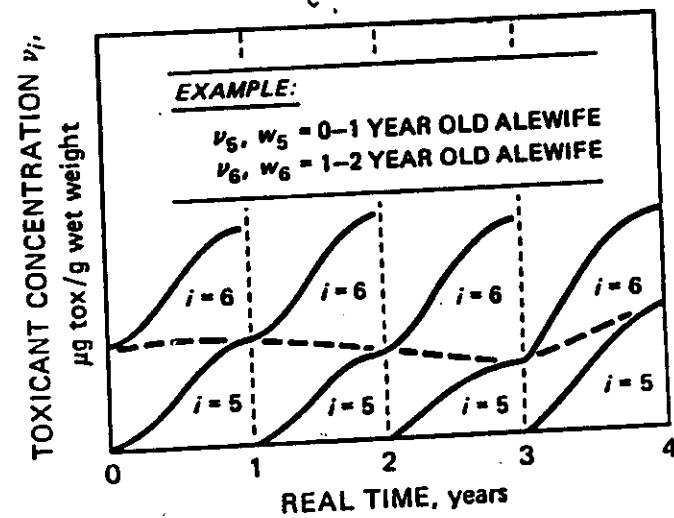
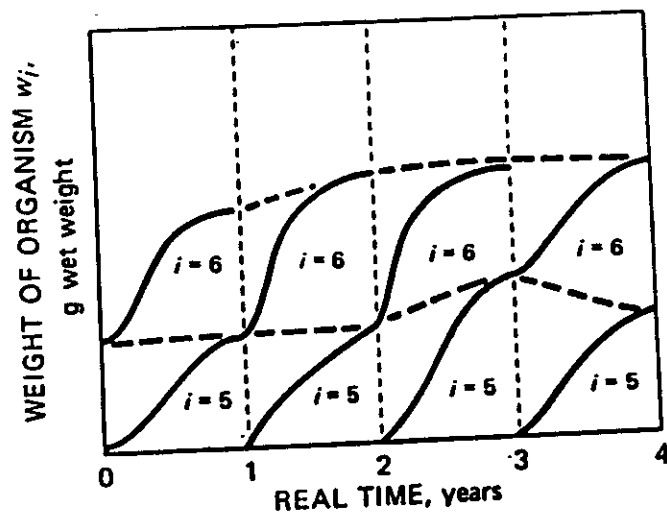
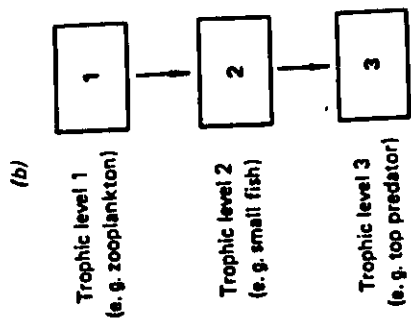
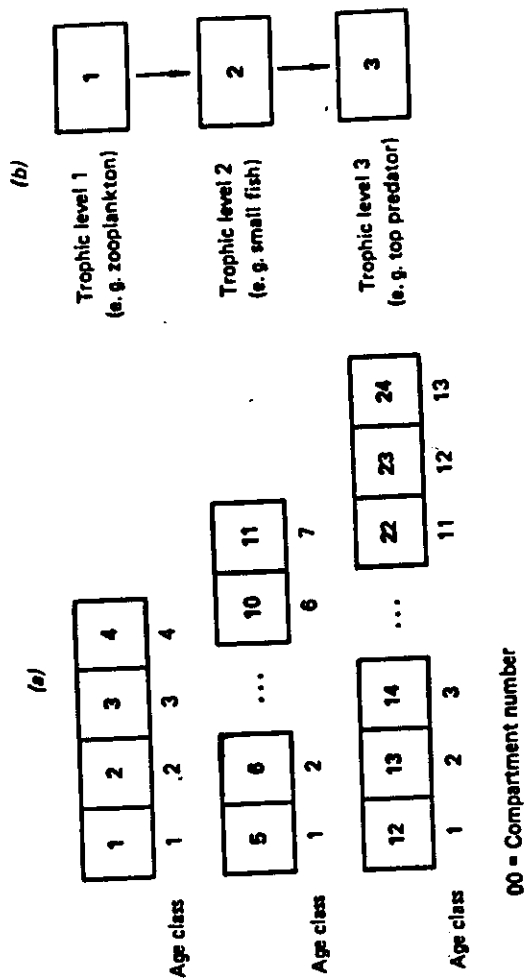
2) Includes any upstream or exchange loads.

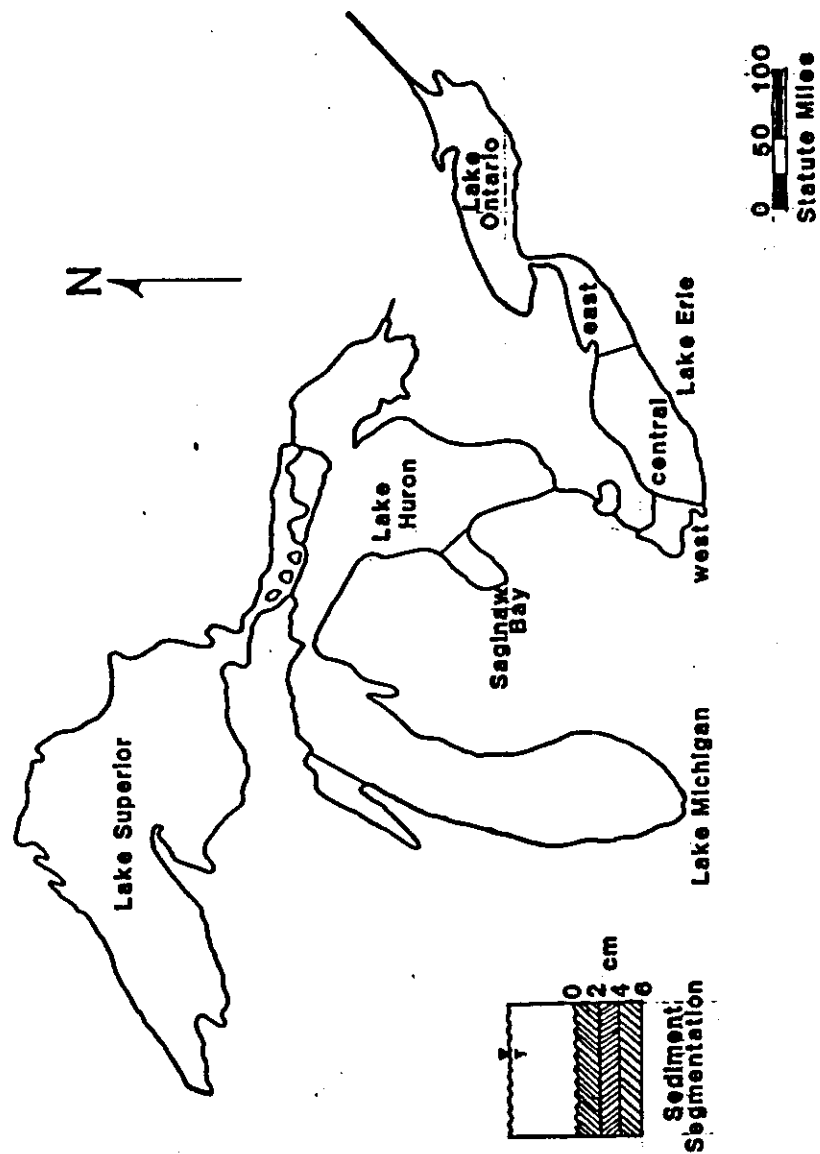
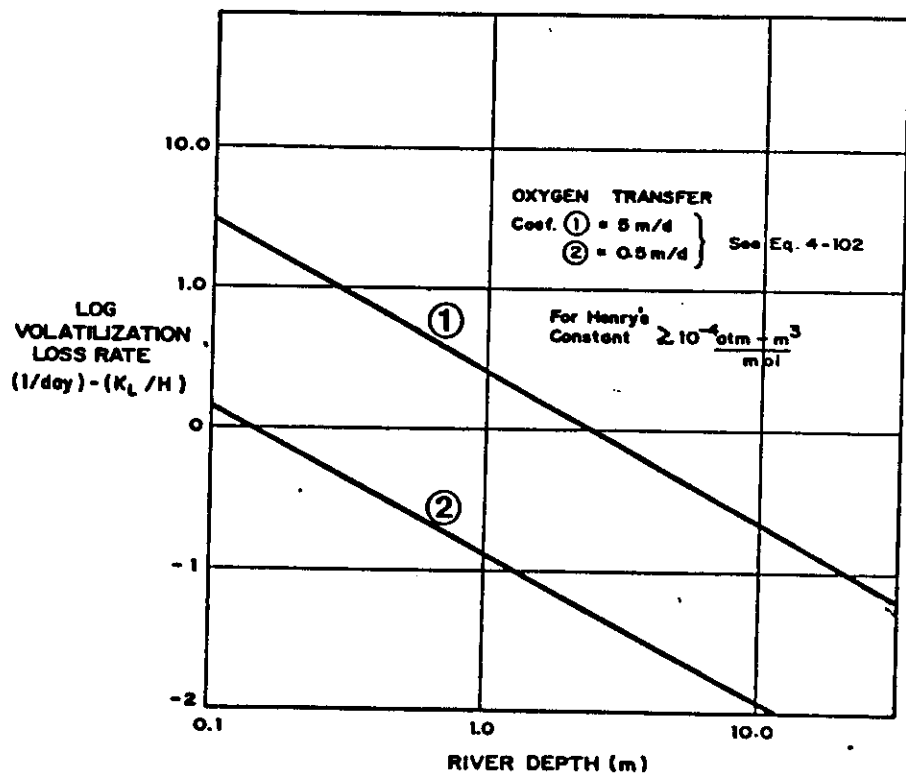
3) Not including any upstream or exchange loads.

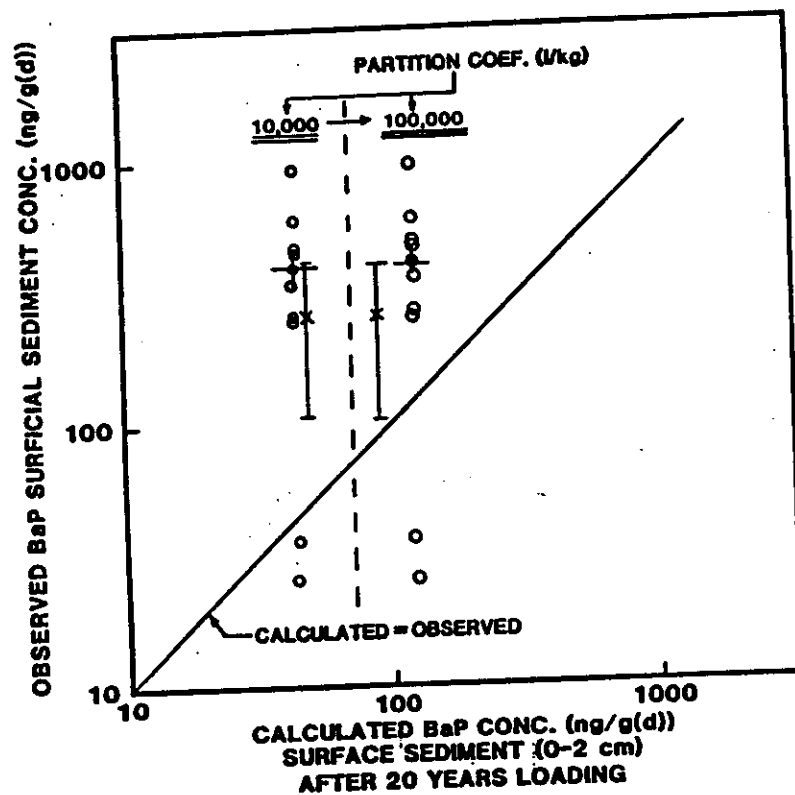
LIST OF FIGURES

- Figure 1. Notation for physio-chemical fate model in streams
- Figure 2. Schematic of compartment definition for a) age dependent model, b) simplified steady state model.
- Figure 3. Illustration of meaning of $w_1(t)$ and $v_1(t)$ showing as an example a 0-1 year old and 1-2 year old alewife.
- Figure 4. Range of volatilization loss rate as a function of river depth for different oxygen transfer rates.
- Figure 5. Great Lakes and Saginaw Bay and sediment segmentation used in model.
- Figure 6. Comparison between calculated surface sediment BaP concentration after 20 years and observed concentration.
- Figure 7. Time variable BaP response in Lake Michigan under two partition coefficients with 20 year constant loading.
- Figure 8. Calculated surface sediment concentration ($\mu\text{g/g(d)}$) at $t = 50$ years with partition coefficient as a function of solids concentration.
- Figure 9. Comparison of calculated cadmium concentration under two assumptions on partition coefficient. High load estimate.
- Figure 10. Organic chemical data from Mississippi River, Jefferson Parish station, Chloroform (top) time series, (bottom) cumulative frequency distribution. (Compiled from data of USEPA, 1986).
- Figure 11. The effect of the first order autocorrelation parameter of the water concentration on the relationship between r and K_{ow} . Growth rate variable, $wt. = 1000$ g.
- Figure 12. (Top) Compiled laboratory r values from experiments of Oliver and Niimi (1983, 1984), (bottom) comparison of calculated and observed r values under assumptions on water correlation structure and metabolic rate.



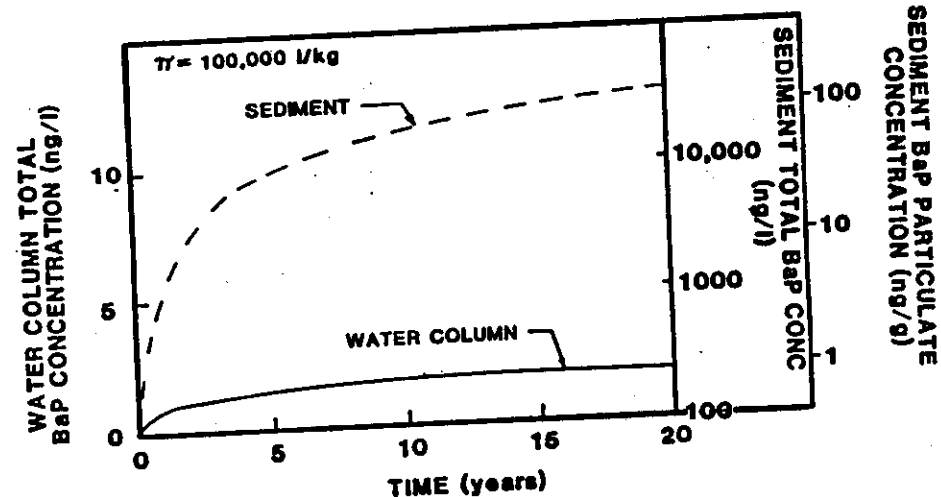
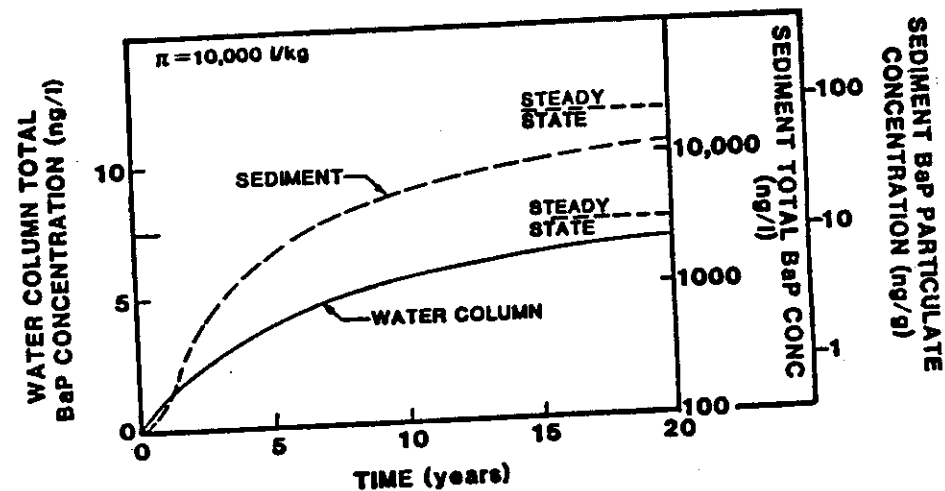


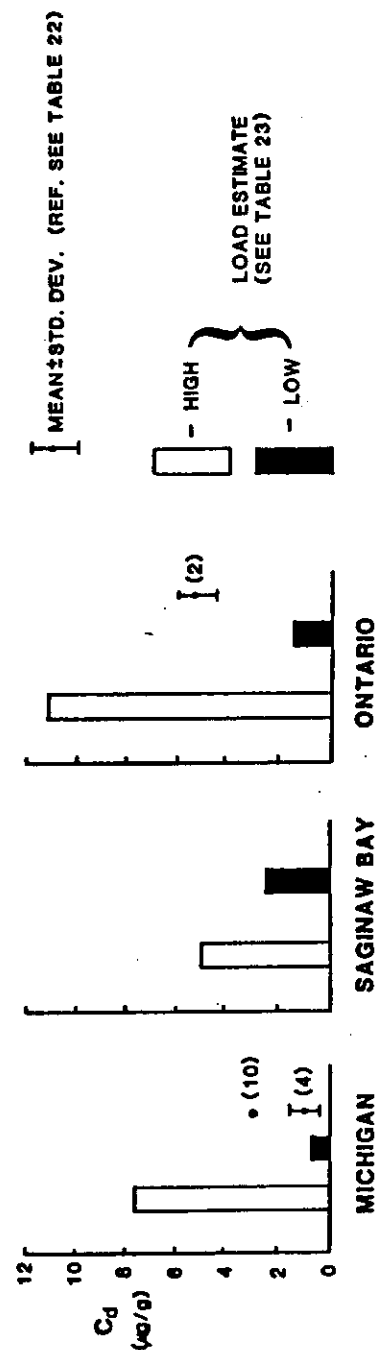
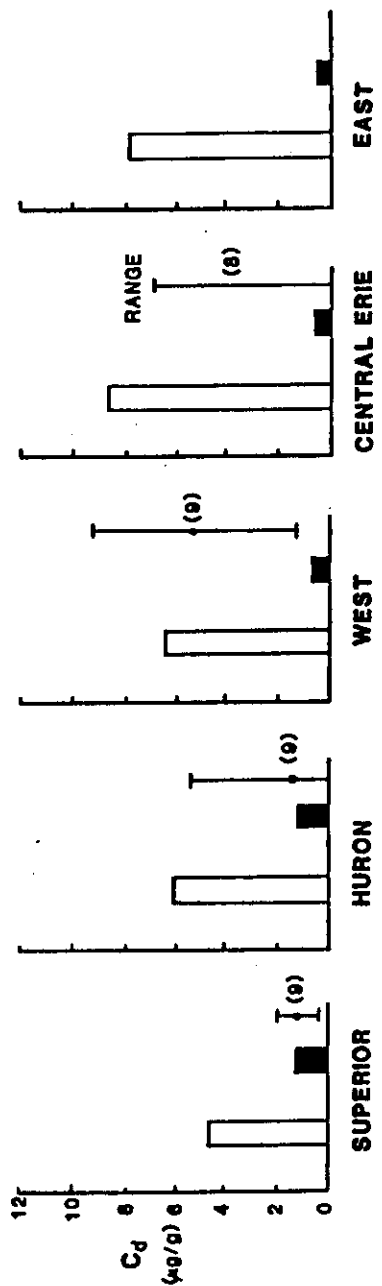




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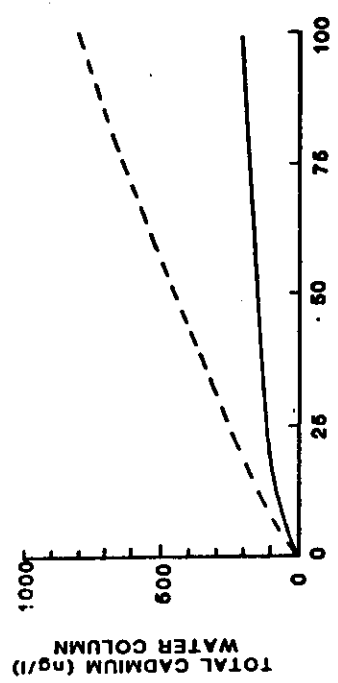
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- + - Mean } Lake Michigan (Eadie, 1983)
- × - Mean ± Std. Dev. } Lake Erie (Eadie, 1983)
- (n=3) } Lake Erie (Eadie, 1983)



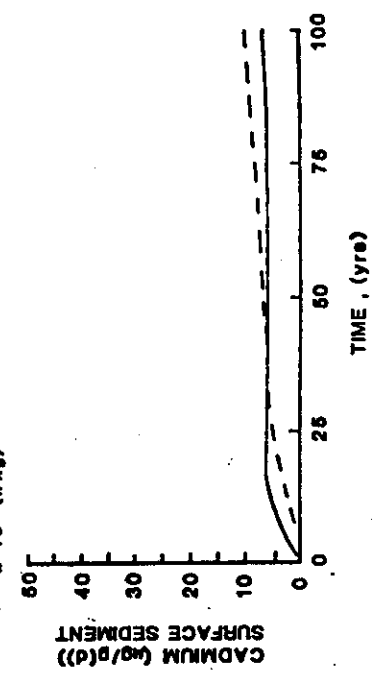
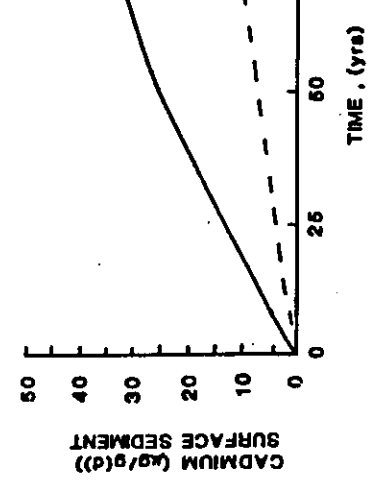
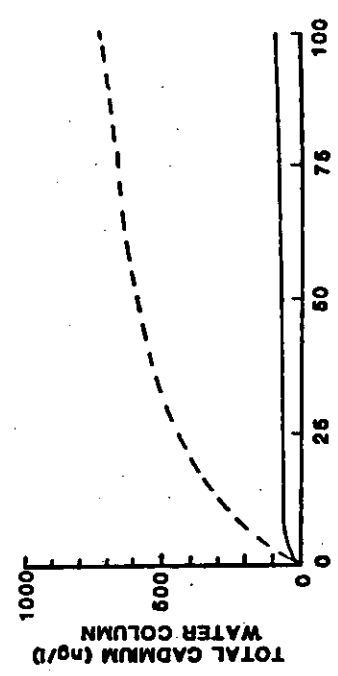


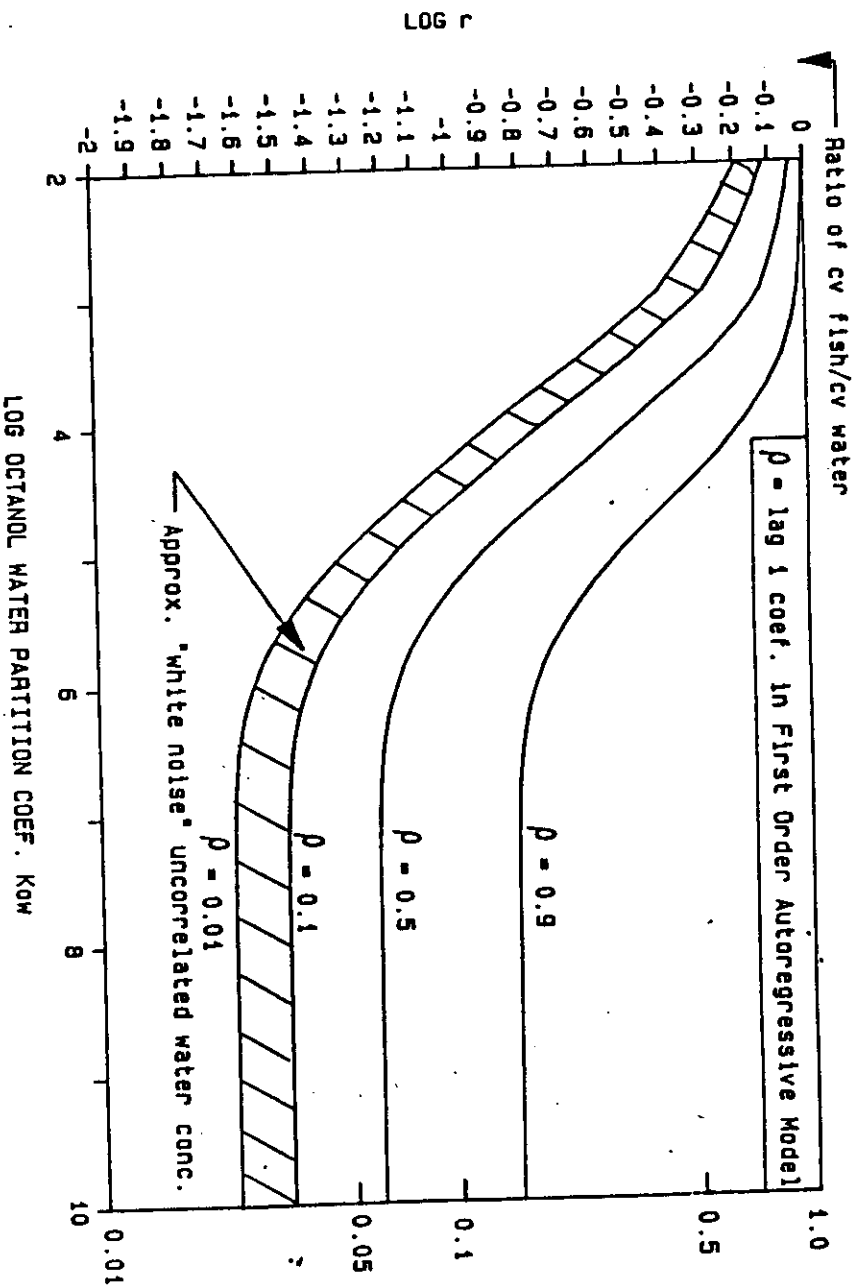
HIGH LOAD ESTIMATE

LAKE MICHIGAN

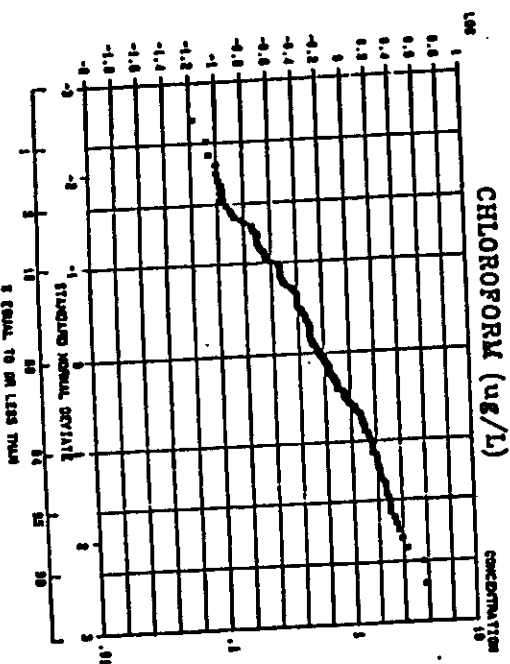
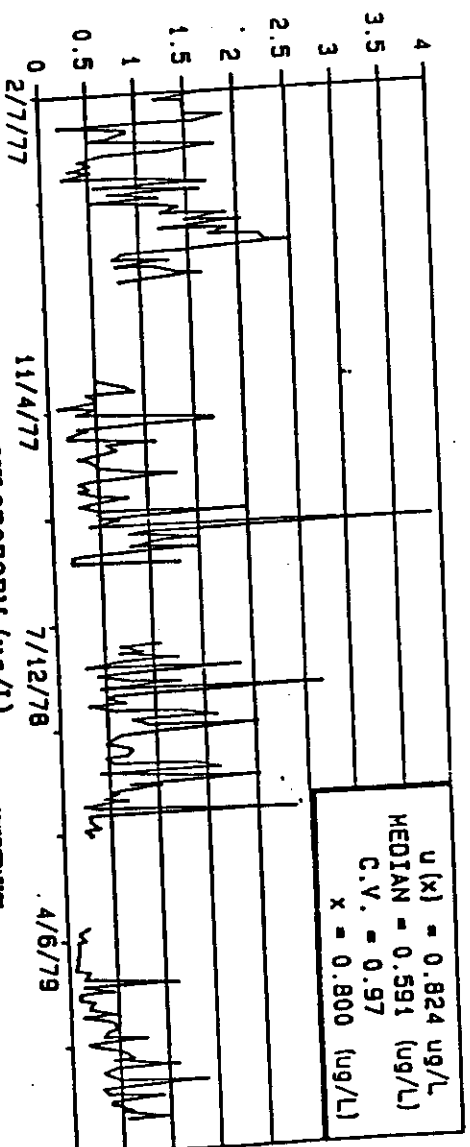


CENTRAL LAKE ERIE



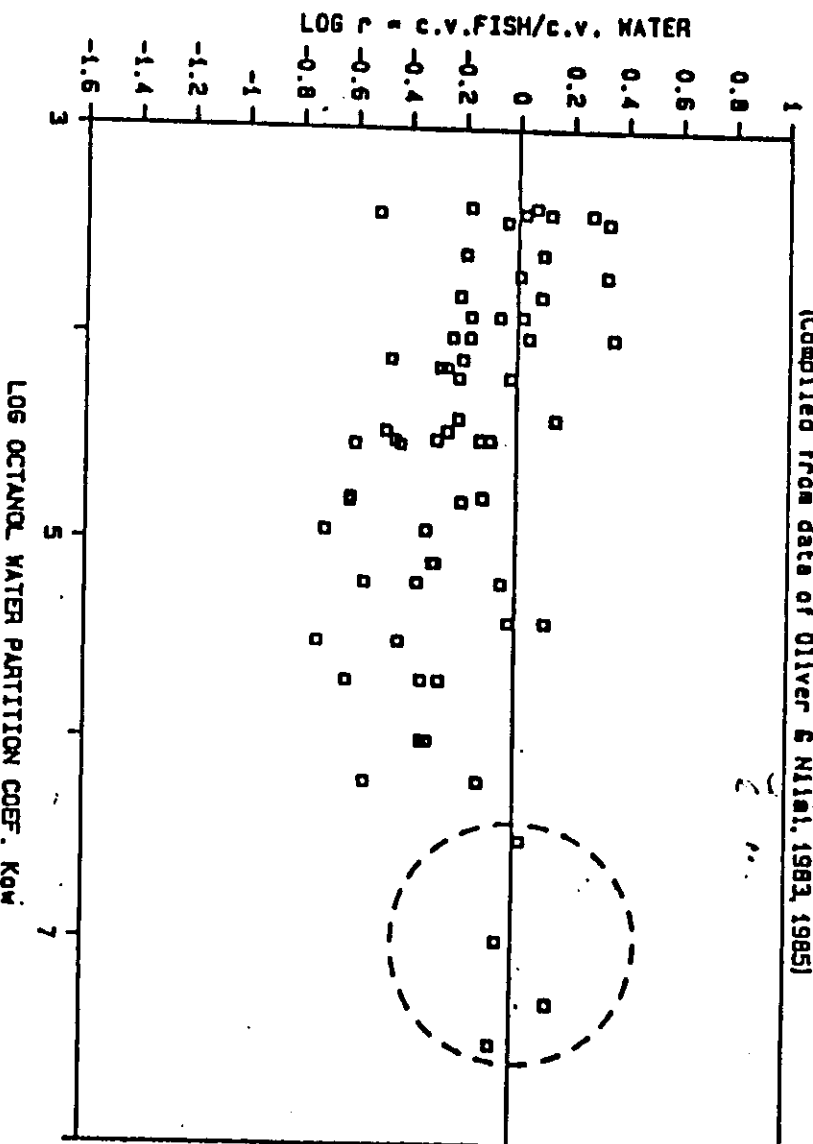


CHLOROFORM (ug/L)



C.V. FISH/c.v. Water. CHLORINATED & BROMINATED CHEMICALS

(Compiled from data of Oliver & Naim, 1983, 1985)



CALCULATED^a VS. OBSERVED^b r^a

