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Maternal Transfer and in Ovo Exposure of Organochlorines in Oviparous Organisms: A Model and Field Verification

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Because early life stages of many species exhibit a greater toxicological sensitivity to contaminants than the adult life stages, knowledge of the exposure of contaminants during embryonic development is a crucial prerequisite for toxicological risk assessment. This study presents a chemical equilibrium model for estimating the maternal transfer and resulting exposure of developing embryos in eggs of several classes of oviparous organisms to hydrophobic organic chemicals. The model is tested against (i) the results of a field study, including the analysis of 44 chemicals in eggs and maternal tissues of 6 fish species and snapping turtles, and (ii) literature data for 8 additional fish and 3 bird species. Lipid normalization of egg and maternal concentrations reduces the variability in observed egg-to-maternal tissue concentration ratios between species by approximately 20-fold. The majority of observed lipid normalized egg/maternal tissue concentration ratios for individual chemicals and fish were not significantly different from 1.0, and the combined data set shows a logarithmic distribution with a mean of 1.22, and 95% probability intervals of 0.56 and 2.51. This indicates that during fish development, the embryos can be expected to be exposed to the same effective internal concentration as the maternal organisms from which the eggs originated.

Introduction

Considerable scientific evidence demonstrates that early life stages of oviparous organisms, including fish and reptiles, often exhibit a greater toxicological sensitivity to chemical contaminants than adult life stages. For example, field and laboratory studies show that early life stages (eggs-to-fry) of lake trout (*Salvelinus namaycush*) exhibit a 50% mortality when internal concentrations reach 0.065 μg/kg of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in water (*1*), while the adult lake trout do not exhibit similar health effects at similar concentrations (*2*). Similar observations have been reported in anthracene-exposed fathead minnows (*3*), and in snapping turtles (*Chelydra serpentina*) (*4*). These examples stress the importance of including toxic effects at early life stages in ecological risk assessments.

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Since ecologically relevant risk assessments require a method to assess exposure of early life stages to chemical contaminants, we present and investigate a simple chemical equilibrium model for assessing the chemical exposure of developing embryos in eggs of several classes of oviparous organisms. This model is tested in a field study, including 44 chemicals in maternal tissues and eggs of 6 fish species and snapping turtles (*Chelydra serpentina*). The model is further tested against literature data for other animal groups. The model results, including probabilistic assessment of the model's uncertainty, are reported and discussed in terms of their application in ecological risk assessments.

Theory

The premise of a chemical equilibrium model for deposition of hydrophobic organic chemicals in eggs during ovogenesis is that chemical transport from the maternal tissues to the eggs follows a set of passive (non-energy consuming) transport processes resulting in a chemical equilibrium between the chemical's concentrations in maternal tissues and eggs. The inherent assumptions of the model are that (i) the internal distribution of hydrophobic organic chemicals within organisms is relatively rapid (*5*, *6*), resulting in a chemical distribution among maternal tissues that is relatively homogeneous when expressed on an appropriate basis (fugacity or lipid based concentration), (ii) ovogenesis largely involves the transfer of lipoproteins from maternal tissues to eggs (*7*−*9*), which may result in chemical levels in eggs that reflect those in maternal lipoproteins, and (iii) metabolic transformation of contaminants in eggs is expected to be low as critical enzyme systems are poorly developed in embryonic tissues.

A chemical equilibrium of the contaminant between maternal tissues and eggs can be defined in terms of chemical fugacities (Pa) in maternal tissues (f_M) and eggs (f_E) being equal, ($f_E = f_M$). Fugacities can be related to chemical concentrations as f equals the ratio of the chemical concentration C (in mol/m^3) and the fugacity capacity Z (mol/m^3·Pa), i.e., f equals C/Z (*10*). At a chemical equilibrium (when $f_E = f_M$), the egg-to-muscle tissue concentration ratio (EMR), i.e., the ratio of the chemical concentrations in eggs (C_E) and maternal tissues (C_M), will reflect the ratio of the fugacity capacities of the eggs (Z_E) and maternal muscle tissues (Z_M):

$$\mathrm{EMR} = C_E/C_M = Z_E/Z_M \quad (1)$$

Since a large number of studies have demonstrated that hydrophobic organic chemicals in organisms largely reside in lipids (*11*) and that the lipid solubility of contaminants does not differ substantially between different types of lipids (*12*), it is reasonable to assume that Z_E and Z_L are approximately equal to $L_E Z_L$ and $L_M Z_L$, respectively, where L_E and L_M are respectively the lipid content of the eggs and the maternal tissues and Z_L is the fugacity capacity of both maternal and egg lipids. Substituting these relationships in eq 1 gives:

$$C_E/C_M = L_E/L_M \quad (2)$$

Equation 2 states that at a chemical equilibrium, the relationship between the chemical concentrations in eggs and maternal tissues reflects the difference in lipid contents between eggs and maternal tissues. If concentrations are expressed on a lipid weight basis as C_{EL} (i.e., C_E/L_E) and C_{ML} (i.e., C_M/L_M), the relationship between chemical concentrations in eggs and maternal muscle tissues can be

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expressed by

$$EMR_L = C_{EL}/C_{ML} = 1.0 \quad (3)$$

where EMR_L is the lipid normalized egg-to-maternal tissue concentration ratio. If this model applies, the concentration of chemical contaminants in eggs can be assessed by the concentration of the chemical in maternal tissues, and also the concentration of chemical contaminants in maternal tissues can be assessed by the concentration of the chemical in eggs. It should be stressed that eqs 2 and 3 can only be used to represent a chemical equilibrium if tissue components other than the lipids do not contribute significantly to the total fugacity capacity of the eggs or the maternal tissues. For example, if the lipid content in the eggs and/or maternal muscle tissue is very low, the EMR_L can be expected to differ from 1.0 even if a chemical equilibrium between eggs and maternal tissues exists because tissues other than the lipids may have a considerable effect on the egg's or maternal tissue's fugacity capacity for the chemical. The error associated with the assumption that lipids are the sole component providing the fugacity capacity of the egg and muscle tissue for chemicals may be one of the reasons why EMR_Ls may vary somewhat from the model predicted value of 1.0.

Materials and Methods

Fish were collected by gill net and impoundment net from the Detroit River at Peche Island (42°29′ N, 82°91′ W) and Turkey Island (42°11′ N, 83°07′ W), and from Lake Erie at Middle Sister Island (41°51′ N, 83°00′ W). Samples were collected to coincide with the spawning periods of individual species. Black crappie (*Pomoxis nigromaculatus*, $N = 6$) and quillback carpsucker (*Carpoides cyprinus*, $N = 5$) were collected in May, carp (*Cyprinus carpio*, $N = 8$), and gizzard shad (*Dorosoma cepedianum*, $N = 7$) were collected in June; freshwater drum (*Aplodinotus grunniens*, $N = 5$) were collected in early July; and whitefish (*Coregonus clupeaformis*, $N = 9$) were collected in late November. Snapping turtles (*Chelydra serpentina*, $N = 3$) were collected by impoundment net at Peche and Turkey Islands in June. Eggs and 5 g of dorsal muscle were removed from fish, and eggs and 5 g of leg muscle were removed from turtles. Muscle and egg samples were wrapped separately in solvent-rinsed foil and stored at −20 °C until preparation for gas chromatography. Only mature eggs were analyzed. Egg and tissue samples were given unique labels to enable comparisons of chemical concentrations in eggs to individual females following analysis.

Sample preparation is described in ref *13*. A sample (5 g) was prepared for solid−liquid extraction by grinding the sample in 20 g anhydrous sodium sulfate (J.T. Baker Chemical Co.). It was then added to a 0.025×0.60 m^2 glass column containing 10 g of anhydrous sodium sulfate and 70 mL of 1:1 dichloromethane/hexane (BDH Ltd.). The column was eluted with 250 mL of 1:1 dichloromethane/hexane solution after 1 h. The extract was concentrated to 2 mL by rotary evaporation and then added to a 0.01×0.55 m^2 glass column containing 40 g of activated Florisil (60/100 mm mesh, Supelco Canada Ltd.) and 3 g of anhydrous sodium sulfate for cleanup. The column was eluted with 50 mL of hexane and the extract concentrated to 10 mL for gas chromatography. Chemical recoveries were greater than 90%. Two mL of extract were removed for gravimetric lipid determination at the beginning of the cleanup step.

Gas chromatographic analysis was performed on a Hewlett-Packard 5890/ECD equipped with an HP-3396 integrator and an HP-7673 autosampler. The analytical column was a DB-5 (J&W Scientific), dimensions 30 m × 0.25 mm. Injection was 1 μL splitless at 250 °C. Carrier gas was ultrahigh purity He at a 30 cm/s flow rate and makeup gas was Ar/CH_4 (95%/5%) at a 40 mL/min flow rate. The oven was temperature programmed from 100 °C to 270 °C at 3 °C/min. Limits of quantification (LOQ) are reported in ref *13*. Chemicals included in the analysis are listed in Table 1. Only contaminant concentrations above the LOQs were used in subsequent calculations.

TABLE 1. Chemicals and Their Octanol−Water Partition Coefficient That Were Measured in Eggs and Muscle Tissues of Fish and Snapping Turtles

chemical	log K_{ow}	chemical	log K_{ow}
1,2,4,5-tetrachlorobenzene	4.5	PCB 110	6.48
1,2,3,4-tetrachlorobenzene	4.5	PCB 151	6.64
pentachlorobenzene	5	PCB 149	6.67
hexachlorobenzene	5.5	PCB 118	6.74
octachlorostyrene	6.29	PCB 146	6.89
trans-nonachlor	6.1	PCB 153	6.92
p,*p*′-DDE	5.69	PCB 105	6.65
mirex	6.89	PCB 141	6.82
PCB 31	5.67	PCB 138	6.83
PCB 28	5.67	PCB 129	6.73
PCB 52	5.84	PCB 183	7.20
PCB 49	5.85	PCB 185	7.11
PCB 44	5.75	PCB 174	7.11
PCB 42	5.76	PCB 171	7.11
PCB 64	5.95	PCB 200	7.27
PCB 74	6.20	PCB 172	7.33
PCB 70	6.20	PCB 180	7.36
PCB 60	6.11	PCB 201	7.62
PCB 101	6.38	PCB 203	7.65
PCB 99	6.39	PCB 195	7.56
PCB 97	6.29	PCB 194	7.80
PCB 87	6.29	PCB 206	8.09

Egg-to-muscle tissue ratios (EMR) were calculated for individual chemicals and biological species by dividing wet weight based chemical concentrations measured in eggs by wet weight based chemical concentrations in the corresponding muscle tissues. The EMR_L were calculated by using lipid normalized chemical concentrations. The logarithm of EMR_Ls calculated for each individual and each chemical from field data and mean EMR_Ls for literature data were compared to model predictions ($\log EMR_L = 0$) by student's *t*-test. The relationship between EMR_L and $\log K_{ow}$ was investigated by linear regressions and the slopes of the linear regressions were tested for significant differences from 0 using analysis of variance (ANOVA).

Results

Fish. Wet weight based concentrations and lipid contents of the chemicals observed in eggs and muscle tissues in fish and snapping turtles are presented in Table 2, which is included in the Supporting Information of this paper. Figure 1a shows 180 mean EMRs for 44 chemicals in 6 fish species. EMRs ranged between a minimum of 0.33 for mirex in carp to a maximum of 106 for PCB 99 in freshwater drum. The distribution of EMR was bimodal, with black crappie, freshwater drum, and gizzard shad comprising the upper mode and carp, quillback, and whitefish occupying the lower mode. The combined mean EMR for all fish was 11.8. Carp consistently had the lowest EMR and freshwater drum the highest EMR.

Figure 1b shows the mean EMR_L. The combined data set showed an approximate log−normal distribution with a mean of 1.22. The 2.5 percentile of this distribution was 0.56, and the 97.5 percentile was 2.51, indicating that 95% of all EMR_Ls for fish fell within approximately a factor of 2 of the mean. In comparison, the 95% probability intervals for the EMR in fish ranged from 0.46 to 54. Lipid normalization of EMRs resulted in an approximate 20-fold decrease in interspecies variability among egg-to-maternal muscle concentration

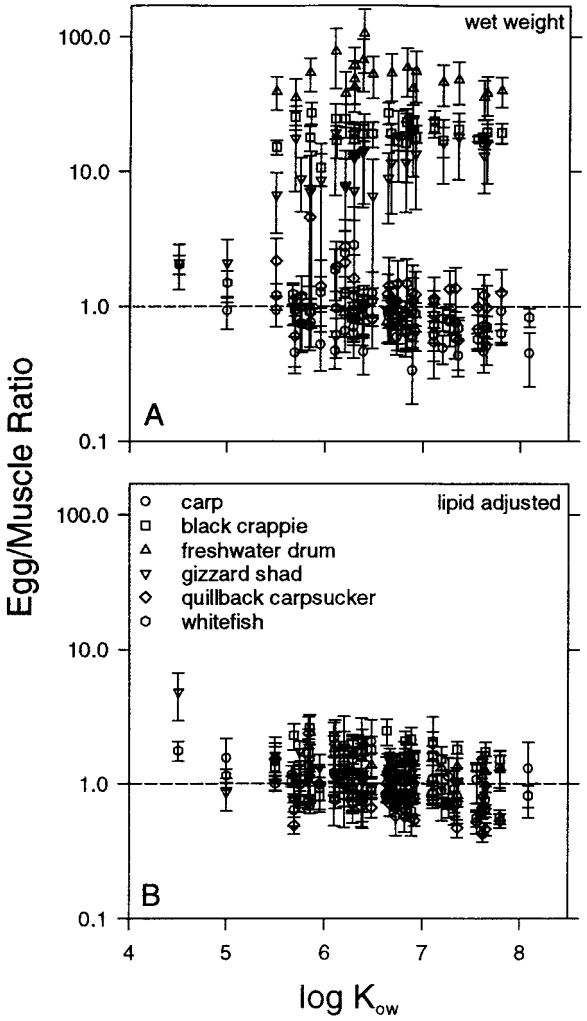

FIGURE 1. Observed mean wet weight based EMR (A) and lipid normalized egg-to-muscle concentration ratios EMR_L (B) for several organochlorines in fish species collected from Lake Erie and the Detroit River. Error bars represent ±1 standard error. The dashed line represents an EMR or EMR_L of 1.0.

TABLE 3. Results of Linear Regressions (i.e., Slope, Intercept, Squared Correlation Coefficient R^2, F Statistic, Degrees of Freedom df, and the Probability Level P) of EMR_L versus log K_{ow} for Each of the Species Included in the Field Study

species	slope	intercept	R^2	F	df	P
carp	−0.112	1.9	0.004	1.2	1, 196	ns
black crappie	−0.095	2.4	0.003	0.6	1, 168	ns
freshwater drum	0.185	0.2	0.01	1.2	1, 114	ns
gizzard shad	0.166	0.08	0.343	112.7	1, 216	<0.001
quillback	−0.361	3.2	0.178	25.6	1, 119	<0.001
whitefish	−0.199	2.2	0.084	32.5	1, 353	<0.001
snapping turtle	0.115	−0.426	0.018	1.2	1, 67	ns

ratios in fish. The bimodal nature of EMR (Figure 1a) was not observed for EMR_L This indicates that the bimodal distribution of EMR reflects interspecies differences in egg-to-muscle tissue lipid content. Results from a linear regression of EMR_L vs log K_{ow}, which is reported in Table 3, showed statistically significant decreases in EMR_L with increasing log K_{ow} in whitefish, gizzard shad, and quillback. In carp and crappie and freshwater drum, no statistically significant decrease in EMR_L with increasing K_{ow} was observed. Table 4, which is included in the Supporting Information, shows that of 40 observed EMR_Ls for carp, 3 EMR_Ls (p,p'-DDE, PCB 87, and PCB201) were significantly different ($P < 0.05$) from the

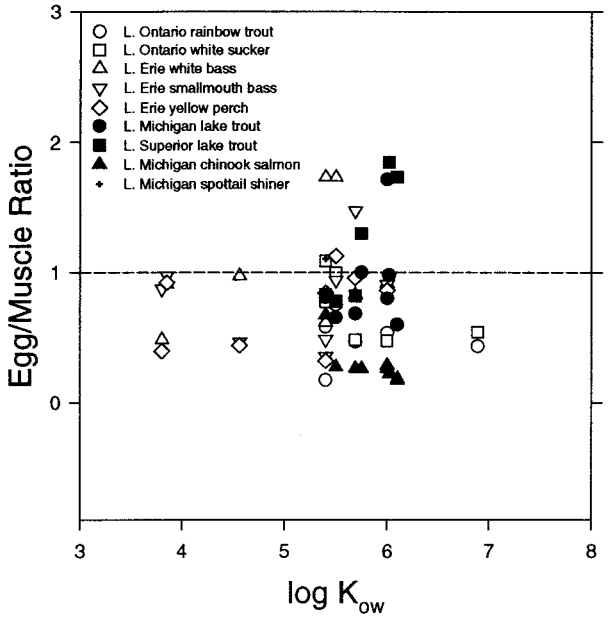

FIGURE 2. Reported mean lipid-normalized egg-to-tissue concentration ratios (EMR_L) for several organochlorines in various fish species. Open symbols are from ref *14*, grayed symbols are from ref *15*, and closed symbols are from ref *16*.

predicted model value as determined by student's t-tests. In freshwater drum, only the EMR_L of PCB 99, and in gizzard shad, only the EMR_Ls of PCB 121 and PCB 28 were significantly different from the predicted model value. In black crappie, 10 EMR_Ls of 32 and in quillback, 9 of 29 observed EMR_Ls were significantly different from 1.0. For whitefish, 23 of 44 observed EMR_Ls were statistically different from 1.0.

Previously reported data on the maternal transfer of organic contaminants from fish to their eggs (*14*−*16*) are presented in Figure 2. Figure 2 shows that EMR_Ls are clustered around 1.0 and that there appears to be no relationship between log K_{ow} and EMR_L. Student's t-tests, comparing the reported mean EMR_L for each species to the model predicted value of 1.0 indicates that EMR_Ls in these fish, were significantly different from 1.0 only for Lake Michigan chinook salmon ($t = -15.79$, df = 9, $p < 0.001$) (*16*) and Lake Ontario rainbow trout ($t = -6.48$, df = 5, $p = 0.001$) (*14*). All other t-tests indicated that EMR_Ls for Lake Michigan lake trout (*16*), Lake Superior lake trout (*16*), Lake Michigan spottail shiner (*15*), Lake Ontario white sucker (*14*), Lake Erie white bass (*14*), Lake Erie smallmouth bass (*14*), and Lake Erie Yellow Perch (*14*) were not significantly different from 1.0.

Reptiles. Figure 3 shows mean EMR and EMR_L for snapping turtles collected in the Detroit River. Due to the small sample size available for snapping turtles, the data are considered preliminary. The combined mean EMR for snapping turtles was 8.4. The combined mean EMR_L for snapping turtles was 0.4. The EMR_L showed no statistically significant increase with increasing log K_{ow} (Table 3). A comparison of the observed EMR_L with the model predicted value of 1.0 by t-test showed that the majority of chemicals exhibited EMR_L values that are statistically different from 1.0 (Table 4).

Birds. Lipid normalized egg-to-whole body chemical ratios for 28 PCB congeners, 12 pesticides, and 9 polychlorinated dibenzo-p-dioxins and furans in Lake Ontario herring gull (*Larus argentatus*, $N = 10$) (*17*) as well as for DDT, DDE, DDD, and dieldrin in Alaska peregrine falcons (*Falco peregrinus*, $N = 4$) (*18*) and total PCB and DDE in Adelie penguins (*Pygoscelis adeliae*, $N = 1$) (*19*) are presented in Figure 4. The EMR_L for herring gulls has a mean of 0.6, a standard deviation of 0.11. A linear regression showed no significant relationship

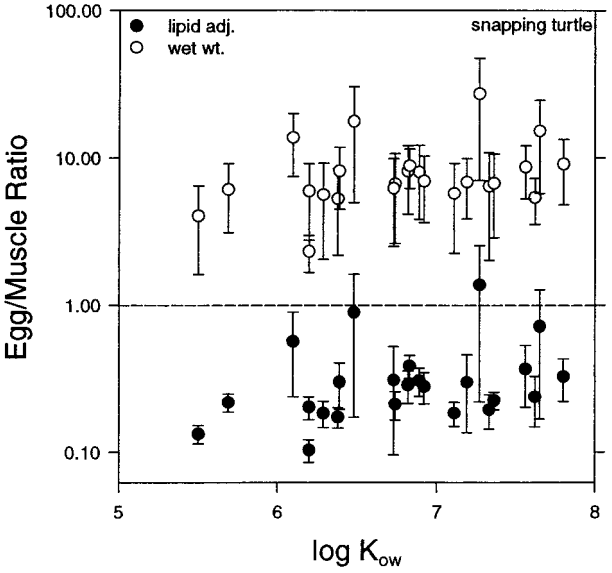

FIGURE 3. Observed mean wet weight based (EMR, open symbols) and lipid normalized egg-to-muscle concentration ratios (EMR_L, closed symbols) for several organochlorines in Detroit River snapping turtles. Error bars represent ±1 standard error.

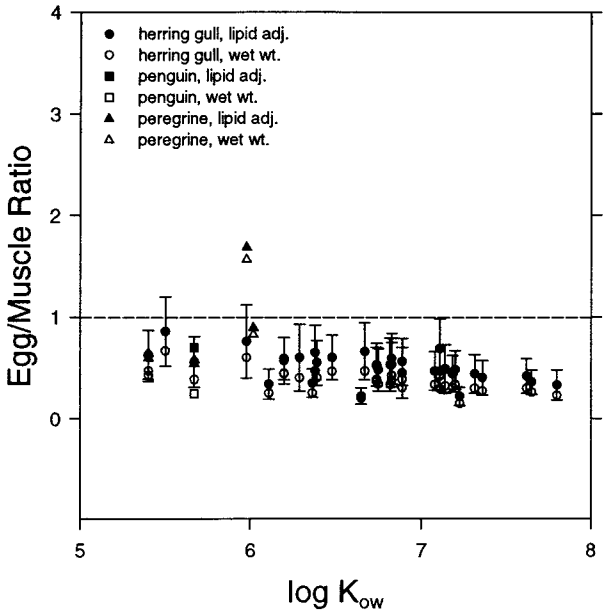

FIGURE 4. Reported lipid normalized egg-to-tissue concentration ratios (EMR_L, closed symbols) for several organochlorines in herring gulls (round symbols) (*17*), peregrine falcons (triangles) (*18*), and Adelie penguins (squares) (*19*). Error bars represent ±1 standard deviation.

of EMR_L with K_{ow} (slope = 0.086, $R^2 = 0.04$, $F_{[1,42]} = 1.7$, p = ns). Student's t-tests showed that the EMR_L in herring gulls for all chemicals combined (i.e., not individual chemicals) were significantly less than 1.0 ($t = -9.625$, df = 46, $P < 0.001$).

Discussion

Both field and literature observations in fish indicate that the equilibrium-based model $C_{EL}/C_{ML} = 1$ describes maternal transfer of organochlorines to eggs well. 95% of 266 observations of maternal tissue and egg concentrations in fish are within a factor of approximately 2 from the model predicted value of 1.0. This illustrates that reasonably accurate predictions can be made of initial in ovo exposure of fish embryos by assuming that the lipid based concentrations in maternal tissues and eggs are the same.

It is recognized that lipid dynamics in oviparous fish play a crucial role in maternal transfer of lipid to eggs. Semelparous fish (salmon in this study) transfer the majority of their somatic lipids into egg development, whereas iteroparous fish species (all other fish species in this study) invest a lower proportion of their lipid stores into reproduction (*20, 21*). The source of maternal lipid also varies among species. For example, catfish (*Clarias batrachus*) utilize abdominal lipid (*8*), whereas Atlantic salmon use muscle lipid (*7*) for egg formation. In contrast, dietary lipid is of primary importance in egg production in northern pike (*22, 23*) and in gilthead seabream (*24*). Despite the fact that this study included fish species with different life histories and lipid mobilization strategies, there appeared to be few differences in EMR_L between fish species and the EMR_L for the majority of chemicals in all fish species were not significantly different from 1.0. This indicates that lipid mobilization has little effect on the relationship between maternal tissue and egg concentrations of contaminants in fish. A possible reason for this is that hydrophobic organic chemicals tend to homogeneously distribute among the lipid tissues of fish. Hence, the type of somatic tissue used for egg development has little influence on the chemical concentration in eggs since all lipid tissues contain approximately the same lipid-based concentration. These results further suggest that in iteroparous fish, which allocate a large portion of dietary lipids in egg production, the absorbed dietary lipids contain approximately the same chemical concentration as maternal somatic lipids at the time of egg deposition. The latter is consistent with observations in the field and laboratory (*25, 26*), which indicate that lipids and hydrophobic organic chemicals are absorbed independently rather than together as presumed by a theory of lipid coassimilation (*27*). Hence, absorbed dietary lipids will adopt the same lipid-based concentration of hydrophobic organic chemicals as somatic lipids instead of retaining the dietary concentration, and the concentration in the eggs will adopt the concentration of the maternal tissues rather than that of the prey. Insufficient time for all lipid compartments (dietary, somatic, egg) to reach an internal steady state, as may occur during periods of fluctuations in lipid reserves, may be one of the main factors causing deviation from equilibrium and contribute to the variability observed in maternal tissue to egg concentration ratios. The latter is consistent with a suggestion in ref *16*, who attributed variations in organochlorine concentrations in both chinook salmon eggs and maternal tissues to limited foraging. The lack of feeding during the fall spawning of whitefish may therefore be responsible for the large number of observed EMR_Ls that were significantly different from 1.0.

Observed EMR_Ls in some bird (herring gull) (*17*) and reptile (snapping turtle) species do not agree with the equilibrium partitioning model to the same degree as EMR_Ls in fish. Hence, generalizing the EMR_Ls among different bird and reptile species may be associated with a somewhat larger error than that for fish. Possible reasons for this phenomenon have been proposed (*9*) but conclusive evidence with regard to the reasons for the deviations of EMR_Ls from 1.0 does not exist.

All available observations for maternal transfer of organic chemicals in oviparous organisms illustrate that lipid-adjusted concentrations in eggs and maternal tissues are fairly close, and typically within a factor of 2 of perfect agreement. This indicates that the initial in ovo exposure of developing embryos in various classes of oviparous organisms to persistent hydrophobic organic pollutants is approximately similar to the exposure of the adults who deposit the eggs. Developing embryos of oviparous organisms at the top of

the food-chain are therefore exposed to the same contaminant levels as the female adults from which they originate. If developing embryos are more susceptible to chemical contaminants than the adult organisms, toxic effects are more likely to occur in developing embryos than in the adult organisms. This observation should be taken in account when conducting ecological risk assessments of chemical substances and when developing environmental quality guidelines.

Acknowledgments

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