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Maternal Transfer of Bioactive Polychlorinated Aromatic Hydrocarbons in Spawning Chinook Salmon (*Oncorhynchus tschawytscha*)

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ABSTRACT

*The biological potency (relative to 2,3,7,8,-tetrachlorodibenzo-p-dioxin, TCDD) of planar polychlorinated aromatic hydrocarbons (PCHs) in extracts of eggs and flesh from spawning female chinook salmon (*Oncorhynchus tschawytscha*) from Lake Michigan was determined by measuring the induction of 7-ethoxyresorufin O-deethylase activity in H-4-II-E rat hepatoma cells. TCDD-equivalents in flesh and egg samples ranged from 0 to 115·8 pg/g, and were approximately 5-fold greater in eggs than in flesh. These results suggest that the maternal transfer of PCHs may play a role in determining the reproductive success of Lake Michigan chinook salmon.*

Numerous studies have established the presence of significant concentrations of polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs) and polychlorinated dibenzodioxins (PCDDs) in fishes from the Laurentian Great Lakes.[1] The most toxic of these polychlorinated aromatic hydrocarbons (PCHs) are PCDFs and PCDDs laterally substituted with four to five chlorines, and PCB congeners (e.g. 3,3',4,4'-tetrachlorobiphenyl) with planar, or nearly planar conformations.[2,3] Although laterally substituted PCDFs and PCDDs and planar PCB congeners can differ markedly in their biological potency, they all produce similar and characteristic patterns of toxicity in mammals, and appear to act via the Ah receptor.[2,3]

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It has been shown that the *in vivo* toxicity of individual, as well as mixtures of, PCH congeners to mammals can be predicted through their ability to induce arylhydrocarbon hydroxylase (AHH) or 7-ethoxyresorufin *O*-deethylase (EROD) activity in the H-4-II-E rat hepatoma cell line.[4] Induction of AHH or EROD activity in the H-4-II-E cells by planar PCHs of unknown potency can then be standardized by expressing the response in terms relative to induction observed using the most toxic PCH known, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD).

In recent years, Lake Michigan chinook salmon (*Oncorhynchus tschawytscha*) have experienced significant reproductive difficulty, and evidence suggests that this lack of reproductive success may be due to planar PCHs.[5] However, due to analytical difficulties in measuring certain planar PCHs, as well as uncertainties concerning the biological potency of complex mixtures of PCHs, it has been difficult to ascertain the potential toxicity of this class of compounds to Great Lakes fishes. The objective of the present study was to use the H-4-II-E bioassay to determine the presence and potency of toxic planar PCHs in the flesh and eggs of spawning female chinook salmon from Lake Michigan.

Details concerning the collection of fish used in the present study are given elsewhere.[6] PCHs in skinless fillets and eggs from 10 female chinook salmon were extracted using standard techniques.[7] Cultures of the H-4-II-E cells were maintained as described elsewhere.[8] Cell cultures were dosed in triplicate with four to six serial dilutions of the flesh or egg extracts dissolved in isooctane. Another series of plates were simultaneously dosed with several concentrations of TCDD (in isooctane), to generate a TCDD standard curve. The cells were harvested 72 h after dosing, and EROD activity was determined using the method of Pohl & Fouts.[9] TCDD-equivalents (TCDC-EQ) in eggs and flesh of the fish were calculated, over the linear portion of the dose–response curve, using the slope ratio method described by Finney.[10]

Extracts from both flesh and eggs of the female chinook salmon induced EROD activity in a dose-dependent manner in the H-4-II-E cells. Figure 1 shows a typical dose–response curve for EROD induction in cells treated with flesh and egg extracts from one of the female salmon.

Only one sample extract (a flesh sample) failed to produce a statistically significant dose-dependent induction of EROD activity in the H-4-II-E cells. TCDD-EQ in flesh samples from the 10 chinook salmon ranged from 0 to 42·9 pg/g, and had a mean value (±SD) of 17·9 (±11·6) pg/g. TCDD-EQ in extracts of the egg samples were considerably higher than in the flesh samples. The range of TCDD-EQ in egg extracts from the 10 chinook salmon was 64·2–115·8 pg/g, with a mean value (±SD) of 91·6 (±18·4) pg/g.

Thus, on a weight basis, TCDD-EQ in the egg extracts were approximately 5-fold greater than in the flesh of the female salmon.

If TCDD-EQ, as determined using the H-4-II-E bioassay, are an accurate measure of the toxicity of planar PCHs to chinook salmon *in vivo*, the observation of an extensive maternal transfer of these compounds may be quite significant. However, relatively little is known concerning the chronic toxicity of TCDD (and related planar PCHs) to fishes, so at present it is not possible to quantitatively assess the potential for reproductive effects due to these compounds. A series of long-term studies concerning the reproductive toxicity of sublethal concentrations of TCDD in salmonids currently are

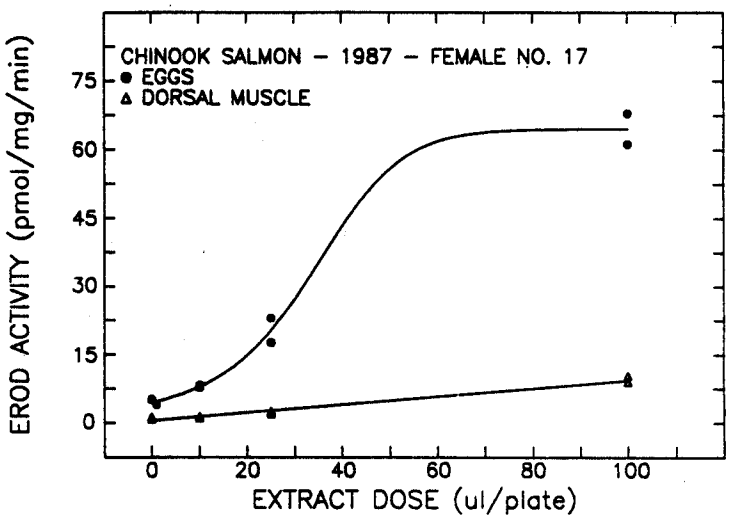

Fig. 1. Dose–response curves for induction of EROD activity in H-4-II-E cells by PCH-containing extracts of flesh and eggs from a female chinook salmon from Lake Michigan.

underway in our laboratory; the results of these experiments should enable us to better assess the potential toxicological consequences of concentrations of TCDD-EQ similar to those found in the present study.

In summary, results obtained with the H-4-II-E bioassay indicated that both flesh and eggs from female Lake Michigan chinook salmon contained measurable, and possibly biologically significant, concentrations of planar PCHs. Moreover, there was a significant maternal transfer of this class of toxic contaminants. Overall, the present study clearly demonstrates the utility of the H-4-II-E bioassay for monitoring studies aimed at determining the presence and biological potency of complex mixtures of PCHs in environmental samples.

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