HISTORICAL TRENDS OF DIOXINS AND FURANS IN SEDIMENTS AND FISH FROM THE GREAT LAKES

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Abstract
A survey of the literature has been performed in order to determine plausible changes in concentrations of dioxins in fish of the Great Lakes basins over the last century. Data from measurements in carp, walleye, trout, and trout eggs (1976-2002) have been combined with sediment cores from Lake Michigan, Lake Ontario, and Lake Superior (1906-1992), using the 1978 value as a reference. Two different models have been fitted on the data, the first one with all parameters being free, and the second that contains a baseline forced to tend towards a null value. Both scenarios placed the peak in 1968 and had similar trends except for early century levels (<1940's) and recent years (>1990's). An analysis based on available congener specific data for trout in Lake Ontario and human blood concentrations has been carried out and concluded to the existence of trends in the relative contributions of main congeners of the 2005-WHOTEQ between 1977 and 1991.

Introduction
Various studies showed that body burdens of long-lived dioxins and dioxin-like chemicals (further referred to as dioxins) are dependent upon historical exposures. A significant fraction of this historical burden is thought to remain in individuals as a consequence of long half-lives of certain dioxin congeners in the body and past exposure levels in the 1960s and 70s that were exceeding current ones by several orders of magnitudes. The food exposure reconstruction effort was first initiated by Winters et al.[2], who used preserved meat from different decades of the 20th century to measure past concentrations of dioxins in beef, pork and poultry. Pharmacokinetic modeling has also been used determine historical exposure to 2,3,7,8-TCDD and other congeners, mostly based on blood concentration trends observed in cross-sectional studies[4,5]. These studies suggest an increase in PCDD/F concentrations beginning around 1940, peaking in approximately 1970, followed by a sharp decrease to pre-1940 concentrations or lower.

The current work supplements these observations by providing a trend specifically calibrated for fish from the Great Lakes. In particular, we derive a mathematical function describing the trend of the concentrations of 2,3,7,8-TCDD in these water basins. Until potential future availability of congener, region, and species-specific concentration data, we suggest application of the provided historical functions calibrated for a particular species concentration at a certain time, as a basis for calculating historical trends of dioxins and furans in fish species from the Great Lakes.

Materials and Methods
Trends in fish and trout eggs (1976-2002)
Concentrations of 2,3,7,8-TCDD in Trout[1], Carp[3], Walleye[3] and Trout eggs[6], from Lake Ontario and the Tittabawassee river over various periods of time have been assembled to reconstitute a plausible historical trend of dioxins in aquatic species of the Great Lakes region. TCDD wet weight concentrations were fitted assuming an exponential function, and rescaled choosing 1978 as a reference value. This adjustment was made by dividing the concentration data for each type of fish by its value predicted by the model for 1978. The year 1978 corresponds to the earliest data available for various aquatic species, and is located at a point in time where biota and sediment data are overlapping. Moreover, high concentration averages at this date have the practical advantage to contain limited samples under the limit of detection.

Trend in sediments (1906-1992)
Trends of total PCDDs and PCDFs in sediments from Lake Michigan, Lake Huron, and Lake Ontario were retrieved from previously published data[7]. Sediment data from the most elevated point to the last concentration measured were fitted separately for each lake using an exponential model. Sediment data from the beginning of

the 20th century up to 1970 were rescaled using the value predicted by the model in 1978 as a reference and kept for the final regression.

Parameterization of the trend for 2,3,7,8-TCDD

Under the assumption that fish 2,3,7,8-TCDD upward trend closely followed the PCDDs/PCDFs in sediments, the various normalized datasets were gathered and a least square regression for data between 1935 and 2002 was performed using an altered version of the equation previously suggested by Pinsky and Lorber[5]:

$$F(t,\theta_i) = a_i + (b - a_i) * e^{-c*(t_{peak}-t)^d} \quad t < t_{peak}$$
$$F(t,\theta_j) = a_j + (b - a_j) * e^{-c*(t-t_{peak})^d} \quad t \geq t_{peak}$$

where t is the date (in years), $\theta_i = (a_i, b, c, d)$ and $\theta_j = (a_j, b, c, d)$ are two sets of parameters with a_i and a_j the background values relative to the 1978 value in fish before and after the peak, b is the peak value, and t_{peak} is the time of the peak (in years) fitted with the constraint $1960 \leq t_{peak} \leq 1975$. Except for t_{peak}, all parameters were fitted without constraints (model 1) or with a trend forced toward 0 (a = 0, model 2). The dataset does not support different values of c and d in the upward and downward curves, therefore resulting in a symmetrical peak function.

Historical congener specific contribution to the WHO2005-TEQ

In order to derive congener specific historical trends, concentration data on trout from the Lake Ontario[1] were used to study the dynamics of the contribution of selected congeners to the WHO2005-TEQ and compared to a compilation of blood measurements from several human cohorts[8]. Congeners with data available between 1977 and 1991 (i.e. all dioxins and furans except for 1,2,3,4,7,8,-HxCDD, 1,2,3,7,8,9-HxCDF, and 1,2,3,4,7,8,9-HpCDF) were converted into relative contributions to the 2005-WHOTEQ and modeled assuming a linear relationship between their contributions to the calculated TEQ and time.

Results and Discussion

Models and parameters

The best fits for both models lead to similar peak dates and analogous overall shapes. However, the importance of the structure of the model is illustrated by the discrepancy at both recent times and the before 1940's, where model 1 (without constraint) tends towards a residual value of about one fifth of the height of the peak. The present data does not enable to discriminate between one of the two models and additional recent fish data would be needed to gain further insights for the 2000-2005 period. The second model has the advantage of not being influenced excessively by the low measured concentration in Walleye in 2002 (see fig. 1) that drives the end of

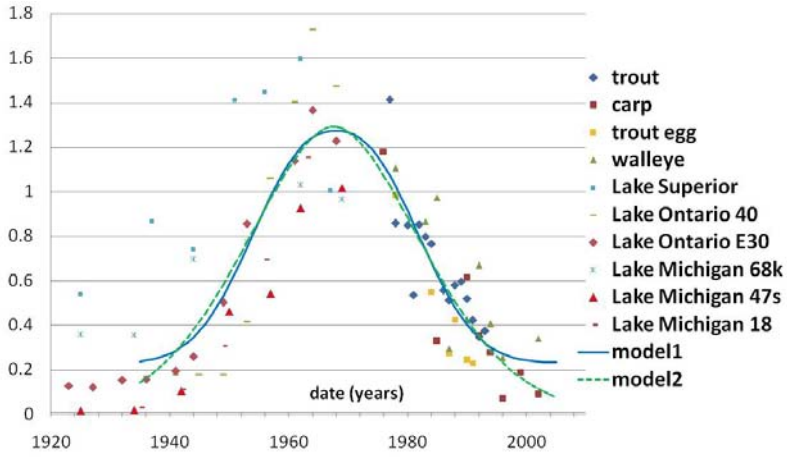

Figure 1. $F(t,\theta)$ for models with all parameters fitted without constraints (model 1) and forced to tend towards 0 (model 2).

Table 1 Fitted Parameters

	model 1	model 2
t_{peak}	1967.6	1967.9
a_i	0.229	0*
a_j	0.231	0*
b	1.27	1.29
c	0.00103	0.00401
d	2.41	1.81
R^2	0.74	0.74

* forced values

the curve of the first model and being more representative of the higher concentrations observed in carps (fig.2). The fitted trend function represented in figure 1 is consistent with reports from the literature[8]. An increase of

PCDDs/PCDFs was observed since the beginning of the 20th century with levels at least one order of magnitude higher at the time of the peak compared to the pre-1935 period. After this initial increase, a decrease or stabilization was observed. Cleverly et al.[9] showed that in lakes mostly affected by atmospheric deposition, CDD/CDF profiles were comparables across all period. The fact that congener patterns were similar suggests that the relative congener-specific inputs from the atmosphere have remained consistent over time even if absolute concentrations were undergoing important variations[9]. This constant congener contribution is an important assumption of the approach used here when connecting CDD/CDF profiles to 2,3,7,8-TCDD profiles, but has to be carefully further evaluated for fish species from the Lake Ontario, since profile for this lake differed from other lakes due to possible non-atmospheric sources[7,10]. An important caveat to the overall estimation is that the decrease of dioxins after the peak occurs more slowly in sediments than in aquatic species analyzed here. Given this different behaviors (i.e. a steeper decrease in fish compared to sediments), the current study probably underestimates the magnitude of the peak due to the combination and normalization with sediment data. Therefore, the magnitude of the peak of the proposed function has to be considered as a minimum magnitude and sensitivity studies should be carried out when using it for modeling purposes.

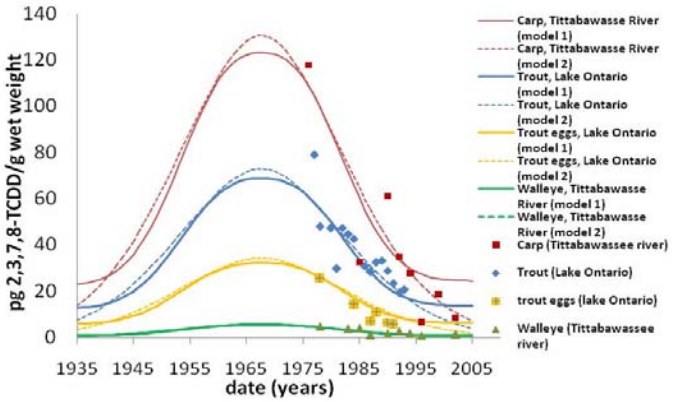

Figure 2. Predicted wet weight fish and trout eggs historical concentrations in several Lake Ontario and Tittabawassee river using $\frac{F(t,\theta)}{F(1978,\theta)} \; x \; conc_i(1978)$ and original data from Huestis et al.[1] and Woodburn et al.[3]

An example of the application of the proposed approach is presented in figure 2, which shows a historical reconstruction of fish species and trout eggs in different lakes calibrated using a single measurement per specie (the interpolated concentration in 1978 has been used).

Historical trends of individual congeners

Table 2. Congener specific parameters estimates and p values for the % change in WHO2005-TEQ contribution per year between 1977 and 1991. The last row provides the estimates for the total 2005-WHOTEQ variation/year during the period.

	Lake Ontario Trout[a]		U.S. Pop. blood[b]	
Congener	$\beta_{\%}$ ($CI_{95\%}$)	p value	$\beta_{\%}$ ($CI_{95\%}$)	p value
2,3,7,8-TCDD	-8.5 (-11.6,-5.4) x 10^{-3}	<0.001	-4.4 (-8.0,-0.8) x 10^{-3}	0.022
1,2,3,7,8-PeCDD	3.2 (1.8,4.6) x 10^{-3}	<0.001	6.9 (-0.2,14.0) x 10^{-3}	0.056
1,2,3,4,7,8-HxCDD		n/a		
1,2,3,6,7,8-HxCDD	1.8 (0.8,2.9) x 10^{-4}	0.002	-1.2 (-6.4,4.1) x 10^{-3}	0.634
1,2,3,7,8,9-HxCDD	8.5 (3.4,13.5) x 10^{-5}	0.003	1.3 (0.2,2.4) x 10^{-3}	0.024
1,2,3,4,6,7,8-HpCDD	9.0 (-1.3,19.3) x 10^{-6}	0.081	-2.5 (-15.7,10.7) x 10^{-4}	0.686
OCDD	-7.8 (-35.8,20.1) x 10^{-7}	0.556	2.1 (-0.2,4.5) x 10^{-4}	0.070
2,3,7,8-TCDF	2.4 (0.6,4.1) x 10^{-3}	0.013	4.4 (3.0,5.8) x 10^{-4}	<0.001
1,2,3,7,8-PeCDF	1.6 (0.9,2.4) x 10^{-4}	<0.001	1.2 (0.7,1.6) x 10^{-4}	0.001
2,3,4,7,8-PeCDF	2.1 (0.6,3.5) x 10^{-3}	0.010	-2.9 (-6,0.1) x 10^{-3}	0.057
1,2,3,4,7,8-HxCDF	-1.0 (-6.9,4.9) x 10^{-4}	0.715	-5.7 (-,-) x 10^{-7}	0.999
1,2,3,6,7,8-HxCDF	3.5 (2.2,4.7) x 10^{-4}	<0.001	4.8 (-1.6,11.2) x 10^{-4}	0.127
1,2,3,7,8,9-HxCDF		n/a		
2,3,4,6,7,8-HxCDF	1.3 (0.7,1.9) x 10^{-4}	0.001	3 (1.0,5.1) x 10^{-4}	0.010
1,2,3,4,6,7,8-HpCDF	1.1 (0.1,2.1) x 10^{-5}	0.040	1.1 (-1.5,3.6) x 10^{-4}	0.374
1,2,3,4,7,8,9-HpCDF		n/a		
OCDF	9 (-3.7,21.7) x 10^{-7}	0.150	4.2 (-0.3,8.7) x 10^{-6}	0.061
2005-WHOTEQ	-3.1 (-4.5,-1.7)	<0.001	-2.5 (-3.6,-1.4)	<0.001

[a]Huestis et al.
[b]Dioxin reassessment report

The percentage of changes in congener contribution to the total 2005-WHOTEQ in fish from the Lake Ontario and in human blood is presented in table 2. Interestingly, most of the congeners with a statistically significant trend exhibit the same tendency in trout and human blood. For example, 2,3,7,8-TCDD which was the third contributor to the TEQ in 1977 (19% of the TEQ contribution), is shown to be significantly reduced in both sample types at a non statistically distinguishable rate. Similarly, the first contributor to the TEQ in blood (1,2,3,7,8-PeCDD, ~30%) was increasing in a comparable manner in fish and human blood in spite of a statistically non significant slope at the α=0.05 level (congener specific blood values were lacking more often than concentrations for fish and therefore lead to more frequent non significant p values). An important caveat of these preliminary analyses is that congener patterns observed in Lake Ontario might be different than patterns

observed in other of the Great Lakes due to suspected important non-atmospheric sources of dioxins in this Lake. However, it suggests that the contribution of some of the most important congeners for the TEQ of fish were varying over time. This observation has sound implication in historical doses reconstruction, and has the potential to explain the trends observed in human cohorts. For example, 2,3,7,8-TCDD exhibits a linear reduction of its contribution to the blood TEQ, falling from ~24% of the TEQ in 1972 (calculated without taking 1,2,3,4,7,8-HxCDD into account) to only ~13% in 1998 according to the most extensive set of human data currently available[8], and thus indicating that 2,3,7,8-TCDD in blood is decreasing faster than the TEQ (table 2).

An approach has been developed to determine historical concentrations of dioxin and dioxin-like congeners. Although certitudes about historical trends are not achievable with current available data, the present work present facts that are coherent with previously stated theories: it agrees on a peak of dioxin and dioxin-like chemicals in fish taking place ca 1968, consistently with most of the studies for sediment, wildlife, and exposure in North America. A mathematical function describing the shape of historical concentration in the Great Lakes and in the Tittabawassee River is provided with the following limitations: the increase of the CDD/CDF concentration measured before the peak is assumed to behave similarly to the increase of TCDD in fish. This could cause the height of the peak to be underestimated by the proposed method. Finally, the approach applies to fish from the Great Lakes region, and cannot be directly applied to distinct phila or species (e.g. algae, herring gulls), for which the historical trends could be substantially different.

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