HUMAN EXPOSURE TO DIOXINS FROM CLAY: A CASE REPORT

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Abstract
As part of the University of Michigan Dioxin Exposure Study (UMDES), we measured the 29 congeners of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like polychlorinated biphenyls (PCBs) that have consensus toxic equivalency factors (TEFs) in serum of 946 subjects who were a representative sample of the general population in five Michigan counties. A 77 year-old woman was found to have the highest total toxic equivalency (TEQ) value in the population (211 parts per trillion (ppt) TEQ). She had no apparent opportunity for exposure to dioxins, except that she had lived on property with soil contaminated with dioxins for almost 30 years, and had been a ceramics hobbyist for over 30 years. The congener patterns in serum, soil and clay suggest strongly that the dioxin contamination in the clay, and not the dioxin contamination in soil on her property, was the dominant source of dioxin contamination in this subject's serum. It would appear that clay can be a significant non-industrial source of human exposure to dioxins among ceramics hobbyists.

Introduction
For the general population the dominant source of exposure to dioxin-like compounds is food (>90%), primarily via consumption of dairy, meat, and fish products.[1] Additional circumstances of exposure that can be significant in selected subpopulations include: occupational exposures; consumption of fish or game from contaminated regions; and persons who live in the vicinity of waste incinerators.[1]

The UMDES was designed to determine whether PCDDs, PCDFs, and dioxin-like PCBs (hereinafter collectively referred to as 'dioxins') in soil and/or house dust are related to or explain serum levels of these contaminants, with adjustment for other known risk factors (i.e., diet, occupation, age, body mass index, etc.). Analyzable serum samples were obtained from 946 participants. Eligible subjects also had the same congener analyses performed on soil samples from around their homes (n=766) and on house dust sampled from inside homes (n=764). Full details on study methods and protocols are described elsewhere (http://www.sph.umich.edu/dioxin/protocol.html)

As part of a follow-up investigation, it was found that most of the subjects with the highest serum TEQs reported frequent and prolonged consumption of wild game and/or sport-caught fish; high outlier serum levels did not appear to be related to contamination of soil or house dust, occupation, activities in the contaminated areas of the region, or proximity to incinerators.[2] However, the subject who had the highest serum TEQ in the entire study did not fit these patterns. We report results of further investigations into why this subject had elevated levels of dioxins in her serum.

Materials and Methods
The index case (Case 1) is female, and was 77 years old at the time her blood was sampled. She had lived along the Tittabawassee River for almost 30 years, downstream from the Dow plant located in Midland, Michigan. Her serum TEQ was 211 ppt (using 2005 TEFs).[3] She denied any occupational history that might suggest potential opportunity for exposure to dioxins. She denied consumption of wild game since she was a child. Her consumption of sport-caught fish was modest, and ended approximately 13 years before the study. She denied ever eating fish from the

Tittabawassee River or the Saginaw River. She never prepared or ate store-bought fish at home, but in the 1960's and 1970's she would eat about one fish meal per month at local restaurants. She has never resided in the vicinity of industrial incinerators. She is a lifelong non-smoker, and she denied recent weight change. She did not garden on the property, and she never ate vegetables grown on the property.

Along with a group of friends, she reported that she had been very involved in ceramics as a hobby from the early 1960's up to about the mid 1990's. She purchased clay in liquid form ("slip"), and poured this into molds to harden; excess liquid was then poured out of the mold for later use. She never added anything to the liquid clay except distilled water. When the wet clay had hardened sufficiently, she removed the piece ("green pottery") from the mold and let it dry further. The molds were made of plaster, and she denied ever using organic solvents to clean molds. Rough edges of the green pottery were smoothed with a wet sponge or sometimes she sanded the edges. She estimated that she performed ceramics work on average about 3 afternoons or evenings per week for about 3 decades. She never used gloves or any respiratory protection. She fired the pottery in one of three un-vented electric kilns in the basement of her house. The peak kiln temperature normally attained was approximately 1800^{o} F (cone number 6). After the first firing, she painted the pieces with various glazes, and then re-fired them at the same temperature.

Results of chemical analyses of her serum, house dust, and representative samples of soil collected from her property are shown in Table 1, along with one randomly selected sample each of the subject's fired clay (unglazed), unfired clay (unglazed), and liquid clay (note: because of space limitations results of analyses for PCBs are not shown). All chemical analyses were performed in the same laboratory that performed analyses for the UMDES (Vista Analytical Laboratory, El Dorado Hills, California). Table 1 also displays published data on dioxins in ball clay.[4]

As noted above, the index case performed ceramics with an informal group of friends. Two of these friends were still alive and both agreed to be interviewed and to provide blood samples for analyses of dioxins (Cases #2 and #3). No soil or dust samples were collected in relation to these two cases. Case 2 was 85 years old, and Case 3 was 83 years old at the time of interview. Like the index case, they had no opportunity for occupational exposures to dioxins. They did not live adjacent to the Tittabawassee River, or near any industrial incinerators. They denied fishing, or regular consumption of fish from the Tittabawassee River, the Saginaw River and Saginaw Bay, and they also denied regular consumption of sport caught fish from elsewhere. They denied consumption of wild game from the Tittabawassee River, the Saginaw River and Saginaw Bay, and they denied regular consumption of game from elsewhere. They are non-smokers, and they denied any recent weight change. Their time frame, frequency and duration of ceramics work was approximately the same as for the index case. The manner in which they made pottery (i.e., handling the slip, pouring the clay, trimming the greenware, glazing, etc.) was the same. A distinction was that while Case 1 had 3 kilns in her basement, the other two cases each had only one kiln, these were located in garages, not in the basement or elsewhere inside their homes, and they were used less frequently. Results of chemical analyses of serum for Cases 2 and 3 are also shown in Table 1.

Results and Discussion

Most of the TEQ for Case 1 is attributable to PCDDs, not PCDFs or PCBs. The liquid clay and unfired clay used by Case 1 is contaminated with PCDDs, with only low levels of PCDFs, while the soil, particularly from the flood plain, is dominated by furans. The overall pattern of results shown in Table 1 suggests that the dioxin contamination in the clay, and not the dioxin contamination in soil from the index case's property, was the dominant source of dioxin contamination in this subject's serum.

There are a number of possible exposure pathways by which the dioxins in the clay may have gotten into the body of Case 1: 1) direct absorption of dioxins through her skin while handing liquid clay or unfired ceramics; 2) inhalation of dioxins volatilized when ceramic pieces were fired in the un-vented kilns in her basement; 3) ingestion of clay or

clay particles that landed on food items in her house or during food handing; 4) inhalation of clay dust from handling and sanding unfired ceramic items; 5) inhalation of clay dust that became mixed with house dust. Based on multivariate models from the UMDES study, we do not believe that the last pathway is significant: dioxins in house dust are not a major source of dioxins in serum of household residents (similar models also demonstrate that soil contamination around the home is not a major source of dioxins in serum) (http://www.sph.umich.edu/dioxin/presentations.html). Fired ceramics have very little dioxin, and do not appear to be a source of exposure. Cases 2 and 3 are approximately the same age as Case 1, and handled clay in a manner that was similar to Case 1, but their TEQ and specific congener levels in serum were dramatically lower compared to the index case. The major distinction appears to be that Cases 2 and 3 each had only one kiln, and these were located in garages, not in the basement or elsewhere inside the living space of their homes. Though the number of subjects is small, these results suggest that inhalation of dioxins volatilized when ceramic pieces were fired in the un-vented kilns in the basement of the home was the dominant route of exposure for Case 1. The fact that Cases 2 and 3 had above-average TEQ and specific congener levels in their serum (e.g., 2,3,7,8-TCDD after adjustment for age), could be due to their more limited exposure to kilns, and/or a limited role for exposure from direct handing of clay materials.

Elevated levels of PCDDs have been found in ball clay from various regions in the United States and Europe.[4, 5, 6] Evidence suggests that these PCDDs were formed naturally via an abiotic and non-pyrogenic process and are not the result of anthropogenic activities.[6] Our subjects reported that they purchased clay from regional retail sales outlets, but the precise geological source of the clay used by our subjects is not known. It is uncertain whether their clay was composed of ball clay known to be contaminated, or whether it came from other sources not previously shown to be contaminated with dioxins.

This report documents that clay, in particular firing clay with un-vented kilns, can be a significant non-industrial source of human exposure to dioxins among ceramics hobbyists. Further investigations are warranted to better determine routes of exposure, in particular to confirm whether volatilization of dioxins during firing is the most important route of exposure, and also to determine the extent of dioxin contamination of clay.

Acknowledgments
Financial support for this study comes from the Dow Chemical Company through an unrestricted grant to the University of Michigan. The authors acknowledge Ms. Sharyn Vantine for her continued assistance and Drs. Linda Birnbaum, Ron Hites, Paolo Boffetta and Marie Haring Sweeney for their guidance as members of our Scientific Advisory Board.

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Table 1: Concentrations of PCDDs and PCDFs in Serum, House Dust, Soil, and Clay, and Published Concentrations for Ball Clay (parts per trillion)

Compound	Serum Case 1	Serum Case 2	Serum Case 3	House Dust	House Perimeter	Flood Plain	Liquid Clay	Unfired Clay	Fired Clay	Clay Mixture*	Unfired Clay*	Fired Clay*
2,3,7,8-TCDD	65.4	9	22.1	2.49	2.67	65	31	5.34	0.05	191	212	0.1
1,2,3,7,8-PeCDD	59.8	17	18.4	2.85	2.52	10.6	85	46.1	0.15	155	157	0.4
1,2,3,4,7,8-HxCDD	30.8	12.1	17.5	5.98	2.42	8.7	86.5	44.7	0.14	32	30	0.4
1,2,3,6,7,8-HxCDD	189	83.6	82.3	84.7	6.36	58.6	142	63.5	0.28	103	93	0.4
1,2,3,7,8,9-HxCDD	32.4	10.7	14.1	31	4.66	12.9	454	388	0.28	395	363	0.4
1,2,3,4,6,7,8-HpCDD	149	74.7	57.1	4620	110	652	2430	1280	1.92	1130	1080	0.4
OCDD	541	914	615	20900	851	5800	48500	18400	7.26	29700	23000	1.4
2,3,7,8-TCDF	1.09	0.264	0.716	9.96	20	836	0.07	11	0.09	ND	ND	ND
1,2,3,7,8-PeCDF	0.4	0.141	0.533	6.85	12	543	0.08	17.5	0.21	ND	ND	ND
2,3,4,7,8-PeCDF	50	12.4	13.7	7.97	13.7	442	0.07	7.88	0.13	ND	ND	ND
1,2,3,4,7,8-HxCDF	27	8.46	10	10.4	12.2	375	0.07	4.73	0.08	ND	ND	ND
1,2,3,6,7,8-HxCDF	24.7	8.56	7.96	7.73	5.36	126	0.50	5.2	0.16	ND	ND	ND
1,2,3,7,8,9-HxCDF	1.06	0	0	2.11	3.06	80.4	0.15	1.67	0.07	ND	ND	ND
2,3,4,6,7,8-HxCDF	4.23	1.63	1.33	6.79	5.85	48.7	0.1	1.7	0.13	ND	ND	ND
1,2,3,4,6,7,8-HpCDF	9.45	5.24	6.73	289	53.5	771	0.16	3.29	0.62	ND	ND	ND
1,2,3,4,7,8,9-HpCDF	0.68	0	1	9.4	3.41	65	0.07	1.94	0.08	ND	ND	ND
OCDF	2.1	1.04	1.06	636	92.6	1740	4.57	5.27	0.34	ND	ND	ND
Total TEQ (ppt)**:	211	61	82	85	18	397	223	126	0.5	419	435	<1

NR = not reported; ND = not detected (below limit of detection); All UMDES concentrations below the Limit of Detection (LOD) were substituted with LOD/√2. *Ferrario (2002); **TEQs based on 29 congeners using 2005 TEFs[3]; serum results are reported on a lipid adjusted basis, all other results are reported on a dry weight basis