COHORT STUDY OF WOMEN IN WEST VIRGINIA: SERUM LEVELS OF DIOXIN AND DIOXIN-LIKE COMPOUNDS

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Introduction

The Kanawha River Valley of West Virginia has had an extensive history of industrial activities, including a number of petroleum and chemical manufacturing facilities. As a result, impacts to multiple environmental media from a wide variety of chemical contaminants have been reported.[1] Dioxins are frequently reported among these contaminants and often represent a substantial fraction of the overall potential risk to human and ecological receptors exposed to contaminated media. The bulk of the dioxin found in this area appears to be derived from the production of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and the disposal of associated wastes that occurred between 1948 and 1969. More than thirty years after production of 2,4,5-T was stopped, dioxins are still present in the soils, groundwater, and river water in portions of the Kanawha Valley. In addition, high levels of dioxins have been detected in the sediments of the Kanawha River and at outfall locations on the river. Dioxin has been also identified in other contiguous water bodies, including both tributaries, as well as hundreds of miles of the Ohio River, which receives the Kanawha at Point Pleasant, WV.

Because the women who participated in the present study[2] reside in the Kanawha/Ohio River Valley area of West Virginia, they are drawn from a population of individuals who have been exposed to dioxins (dioxin and dioxin-like chemicals) at environmental background levels higher than those seen in other areas of the United States. Thus, the study evaluates the impact of this exposure on dioxin body burdens, as measured in blood serum.

Objective

This study addresses human dioxin exposure in a potentially highly exposed population in West Virginia.

Methods

This cohort of women was derived from a larger study investigating the relationship between dioxin body burden and endometriosis in women living in West Virginia.[2] The study was performed at Marshall University (Huntington, WV). Physicians in the Department of Obstetrics and Gynecology at Marshall University Medical Center were the primary care providers for these patients, and were responsible for recruiting patients, obtaining informed consent, and performing surgical and diagnostic procedures. Physicians from the Department of Environmental and Community Health assisted in obtaining blood samples, administered environmental questionnaires, collated and analyzed data. All relevant procedures were performed under Marshall University IRB approval, in accordance with 45 CFR 46.110(A)(8)(a). A detailed medical history, an environmental exposure questionnaire, and a blood (serum) sample were obtained from each of the study participants. The age of the participants ranged from 25 to 46.

Serum was analyzed by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry (HRGC/ID-HRMS) for 7 polychlorinated dibenzo-*p*-dioxins (PCDDs), 10 dibenzofurans (PCDFs), 4 non-ortho-substituted (coplanar) polychlorinated biphenyls (cPCBs; dioxin-like PCBs), 6 mono-ortho-substituted PCBs (mPCBs; dioxin-like PCBs), and 36 ortho-substituted PCBs. Analytical results were reported on a whole-weight and lipid-adjusted basis.[3-7] Total serum lipids were calculated using an enzymatic "summation" method.[8] International toxicity equivalents (WHO_{2005} TEQs) were reported for PCDDs, PCDFs, and "dioxin-like" PCBs, based on the WHO-TEQ system.[9] Detection limits, on a whole-weight and lipid-adjusted basis, were reported for each sample, corrected for sample weight and analyte recovery. If there was a valid non-zero result for a non-detect that number was used. If the non-detect result was zero, then lipid adjusted

concentration was imputed as the lipid adjusted analyte detection limit divided by square root of 2 and noted in the output file.

Results

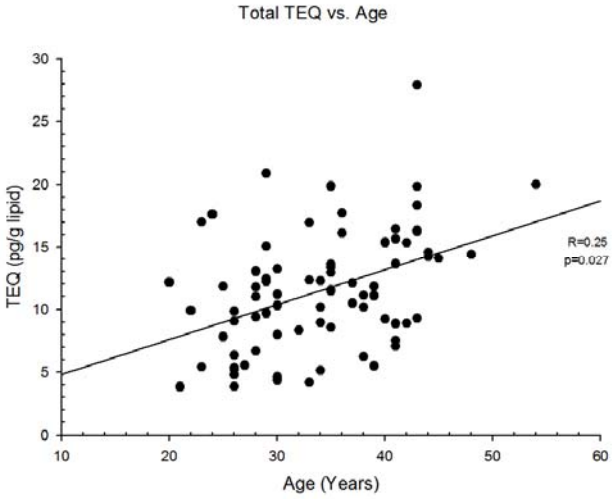

Figure 1.

Total TEQ vs. Age. Preliminary results of the study demonstrate a linear increase of total TEQ (pg/g lipid) vs. age in 91women aged 25 to 46, with a mean ± std of 11.51±4.74, maximum TEQ of 27.9, and a minimum TEQ of 3.8.

Table 1.
Serum Concentrations of PCDDs, PCDFs, and PCBs

Congener	N	Min	Max	Median	Mean	Std	90th %tile	95th %tile
PCDDs (pg/g lipid)								
PCDDs TOTAL	84	119	939	258	296	147	473	550
2378-TCDD	83	0.18	6.7	1	1.11	0.97	2.12	2.64
12378-PeCDD	83	0.22	9	3.5	3.22	1.90	5.3	5.94
123478-HxCDD	83	0.3	8.5	2.7	2.60	1.43	4.12	4.55
123678-HxCDD	83	5.2	46	22	21.5	8.19	32	35
123789-HxCDD	83	0.36	8.4	3.7	3.72	1.74	6.2	6.8
1234678-HpCDD	83	7	110	25	29	17	52	62
OCDD	83	86	850	200	235	128	392	456
PCDFs (pg/g lipid)								
PCDFs TOTAL	82	10.4	60.4	22.4	24.85	9.37	35.6	42.8
2378-TCDF	83	0.15	1.9	0.35	0.412	0.263	0.6	0.97
12378-PeCDF	83	0.15	1.9	0.35	0.461	0.339	0.82	1.3
23478-PeCDF	83	0.25	10	3.4	3.653	1.575	5.48	6.54
123478-HxCDF	83	1.3	9.3	3.2	3.541	1.486	5.02	5.85
123678-HxCDF	83	0.43	8	3	3.301	1.409	4.94	5.81
123789-HxCDF	83	0.16	1.6	0.37	0.416	0.217	0.682	0.727
234678-HxCDF	83	0.19	3.9	0.9	1.02	0.76	1.92	2.47
1234678-HpCDF	83	2.8	17	7	7.87	2.77	12	13.35
1234789-HpCDF	83	0.17	1.5	0.4	0.463	0.238	0.768	0.893
OCDF	82	0.02	16	3.25	3.75	3.78	9.14	11.4

Table 1 (cont'd).								
Congener	**N**	**Min**	**Max**	**Median**	**Mean**	**Std**	**90th %tile**	**95th %tile**
Co-Planar PCBs (pg/g lipid)								
CP PCBs TOTAL	83	21.8	168	69.5	74.6	32.49	116.4	137
PCB-77	83	12	130	47	50.2	28.5	90.6	113.5
PCB-81	83	0.8	8.2	3	3.43	1.7	5.26	7.08
PCB-126	83	3.8	67	12	14.5	10.31	23.4	35.35
PCB-169	83	1.3	17	5.7	6.449	3.69	12	14
Mono-Ortho PCBs (ng/g lipid)								
MO PCBs TOTAL	87	3.2	80.3	9.5	13.75	13.79	26.36	38.6
PCB-118	89	1.2	51	4.7	6.9	8.215	10	20.5
PCB-105	89	0.1	13	1	1.502	1.938	2.66	4.56
PCB-167	89	0.1	4.4	0.7	8.35	0.712	1.6	2.215
PCB-156	89	0.3	22	1.9	3.2	3.67	6.18	7.675
PCB-157	89	0.1	4.8	0.6	0.874	0.867	1.56	2.82
PCB-189	87	0.02	2.8	0.3	0.433	0.486	0.8	1.23

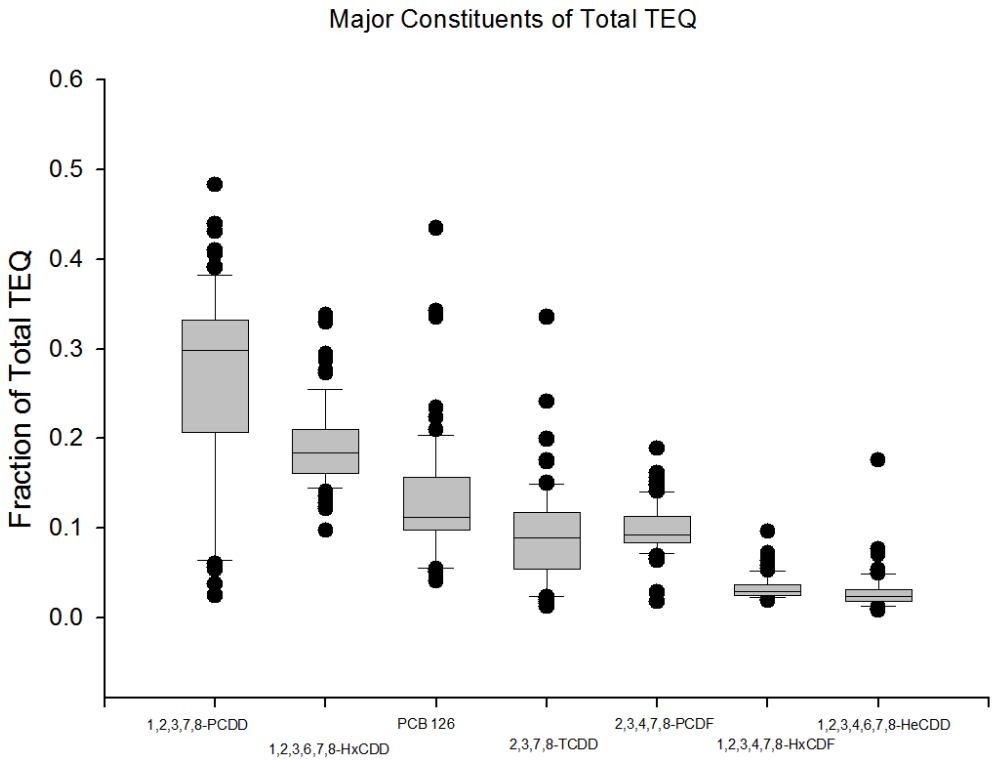

Figure 2.

Box Plot of the major constituents of the total serum TEQ (ppt lipid).

The graph shows the box representing the 25th, 50th, and 75th percentiles, with bars for the 90th and 10th percentiles, and with points above and below the 90th and 10th percentiles.

Conclusions

The present study demonstrates that serum levels of dioxin and dioxin-like compounds in this population are similar to levels previously reported in the general US population.[10] As indicated in the CDC NHANES 2001-2002 Report[5], 25th, 50th ,and 75th percentiles of TEQs for age group 20-39 were 6.6, 10, and 13.7, respectively; while the equivalent values from the present study were 6.61, 10.47, and 12.36. Similarly, as reported in the national study for age group 40-59, the 25th, 50th, and 75th percentiles were 15.8, 19.3, and 23.5, respectively, compared to 9.28, 14.57, and 16.4 for the 40-46 age group of the West Virginia study. In light of these similarities, we conclude that residence in areas associated with elevated environmental levels of dioxins could not be correlated to elevated levels of serum concentrations of dioxins.

Serum concentrations measured in the present study also exhibited a linear relationship between total TEQ (pg/g lipid) and age in 91women aged 25 to 46. The TEQs ranged between 3.8 and 27.9 and the mean ± SD were 11.51±4.74.

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This abstract does not reflect USEPA policy.

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