

Serum Polychlorinated Dibenzo-*p*-dioxins and Polychlorinated Dibenzofurans among People Eating Contaminated Home-Produced Eggs and Beef

Lynn R. Goldman,¹ Martha Harnly,² Jennifer Flattery,³ Donald G. Patterson, Jr.,⁴ and Larry L. Needham⁴

¹School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Maryland, USA; ²Environmental Health Investigations Branch; ³California Occupational Health Branch; California Department of Health Services, Oakland, California, USA; ⁴U.S. Centers for Disease Control and Prevention, Atlanta, Georgia, USA

We compared serum polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) among residents of two homes to levels among age- and sex-matched comparison subjects. The residents of the two homes consumed contaminated eggs and beef from animals raised at the homes. The animals had greater soil contact than those raised with conventional commercial husbandry practices. The comparison subjects were from a similar rural area, but did not consume home-produced beef and eggs. Serum levels of 2,3,7,8-substituted tetra-, penta-, and hexaCDDs and penta-, hexa-, and heptaCDFs were increased between 2- and 6-fold in residents from one home; contaminated eggs and beef were consumed by residents for 2–15 years. Elevations were less for those in the other index home, where only home-produced eggs were consumed for 2 years; a 3-fold elevation of 1,2,3,7,8,9-hexaCDD as compared to controls was most apparent. Very strong bivariate correlations among all of the 2,3,7,8 penta- and hexaCDDs/CDFs were observed. The elevations observed verify that PCDD/PCDF-contaminated food contributed to the body burden of these compounds. The blood levels among the highest exposed participants are generally higher than those observed in other studies of U.S. contaminated-fish consumers and higher than average adipose tissue levels observed in U.S. urban populations. There are sufficient animal toxicologic and human epidemiologic data to recommend that exposures be reduced. In the study area, pentachlorophenol and pentachlorophenol incineration sources have been identified, and the animal contamination and blood elevations probably reflect these sources. Soil reference values and site-specific risk assessments should include estimates of exposures to contamination in home-produced animal products. Such estimates can be verified with limited PCDD/PCDF sampling of animals and humans. *Key words:* beef, chicken eggs, dietary intake, food contamination, human blood levels, polychlorinated dibenzofurans, polychlorinated dibenzo-*p*-dioxins. *Environ Health Perspect* 108:13–19 (2000). [Online 24 November 1999] <http://ehpnet1.niehs.nih.gov/docs/2000/108p13-19goldman/abstract.html>

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are lipophilic, persistent, and bioaccumulative chemicals (1). Animal evidence of toxicity is unequivocal (2). For effects clearly associated with human exposures, humans and animals are equally sensitive (1,2). The food chain contributes the majority of human exposures. Lifetime upper bound cancer risk estimates from the intake of background commercial U.S. high-fat foods and fish are significant, i.e., above 10^{-6} (3). Europeans (4,5) and North Americans (6,7) consuming PCDD/PCDF-contaminated fish and seafood have elevated blood PCDD/PCDF levels, which reflect body fat and hence body burden (2). Such studies verify that food products contribute to body burden.

We analyzed serum from residents of two index households. The residents consumed home-produced PCDD/PCDF-contaminated chicken eggs and beef. The samples were collected and analyzed in 1988. At that time, the U.S. Centers for Disease Control and Prevention (CDC; Atlanta, GA) used a method for measuring 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in serum (8) and had completed a correlation study (9) which

showed that TCDD is evenly partitioned between serum and adipose tissue when both matrices are adjusted for lipid content. CDC investigators also validated the method for PCDD/PCDF analysis in serum (10) and completed several serum matrix validation studies (11–16). Preliminary results indicated elevations of PCDDs among the index home residents as compared to rural comparison subjects (17).

We report 2,3,7,8-substituted PCDD and PCDF levels and results of multivariate modeling of the PCDD/PCDF levels to account for age and sex differences. We also compare intake and serum levels to recent U.S. population estimates and discuss the health significance of the observed levels, comment on whether patterns of PCDD/PCDF in serum are consistent with pentachlorophenol (PCP), and present recommendations to reduce exposures.

Site History

In April 1987, a fire at a wood preservative treatment plant in the area south of Oroville, California, led to a California Department of Health Services (CDHS) investigation of public health impacts of the fire (18). An

estimated 6,000 lb (2,720 kg) PCP burned. PCDDs and PCDFs are contaminants of PCP (19,20) and may also be formed when PCP is burned (18). PCDD/PCDF contamination was detected in products from animals that grazed in the area near the fire (17,21). Initially, California toxic equivalency concentrations (CTEQs) were calculated. CTEQs are a weighted summarization of the 2,3,7,8-substituted PCDD/PCDF concentrations using weights based on the cancer potency of each PCDD/PCDF relative to that of 2,3,7,8-TCDD (22). A similar summarization, international toxicity equivalent (ITEQ) concentrations (23), are now universally used. In this summarization, toxicologic weights based many health end points are used. However, the ITEQ concentrations are generally approximately 30–50% lower than the CTEQ concentrations. ITEQ concentrations found in the chicken products from the two homes initially tested (the index homes) are 5.6–18.3 pg ITEQ/g egg (Table 1) and 177–228 pg ITEQ/g chicken fat. At the second home, cows were also raised; the beef fat contained 27.2 pg ITEQ/g. Similar samples from commercial stores in the area contained 10- to 100-fold lower levels (24) that were equivalent to levels reported in commercial U.S. food by others (3,25).

At the index homes, the chickens were allowed to forage but were also fed table scraps and commercial grain. The cattle were allowed to graze year-round on 2 acres. Animal products on the U.S. commercial market generally come from animals raised in ways that limit soil contact and provide a different diet. Conventionally, chickens are

Address correspondence to M. Harnly, Environmental Health Investigations Branch, California Department of Health Services, 1515 Clay Street, Suite 1700, Oakland, CA 94612. Telephone: (510) 622-4484. Fax: (510) 622-4505. E-mail: mharnly@dhs.ca.gov

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raised in cement shelters with no access to soil and are fed commercial feed. In the beef industry, cattle are generally rotated to different grazing areas and are given harvested feed in feedlots. At the index homes, PCDD/PCDF levels in grain, vegetation, bedding, and water were close to or less than detection limits, which were significantly less than levels in soil (21). Soil from where the chickens grazed contained 34–40 pg ITEQ/g and the PCDD/PCDF contamination profile (of higher chlorinated dioxins dominating over all other PCDDs and PCDFs) matched that in eggs (Figure 1). This pattern is generally seen in PCP (19,26) and PCP incineration (18,27). The pattern in beef fat was generally the same, with somewhat lower levels of the higher chlorinated dioxins. However, in beef liver, preferential accumulation of the higher chlorinated dioxins occurred (21), which was also seen in another study (28). Because of extensive foraging activity, soil was the primary suspected exposure medium to the chickens and cattle. Laboratory and backyard feeding studies substantiate that chickens accumulate PCDDs/PCDFs from

contaminated soil (29,30). Further soil and egg testing in Oroville and elsewhere is in progress (31).

In backyards in the greater Oroville area, chicken husbandry was more prevalent than cattle husbandry. Eggs were also collected from chickens raised with access to soil at 23 additional homes in the greater Oroville area (21). We assessed the upper bound of additional excess cancer risk to humans using assumptions of linearity at low doses, a lifetime of egg consumption, and a cancer potency (q^*) of 130 [($\mu\text{g}/\text{kg}/\text{day}$)] (17,22). Because PCDDs/PCDFs were present at a significant background level (3), the CDHS chose a 10^{-4} lifetime excess cancer risk as a level that clearly distinguished contaminated eggs from background eggs, and that warranted the issuance of public health recommendations to reduce exposures. For a 60-kg adult, consuming half an egg (30 g)/day contaminated at 1–5 pg CTEQ/g egg (or 30–150 pg CTEQ/day) equaled this risk, rounding the risk to an order of magnitude. Residents where testing had occurred and with this level of contamination or higher were advised

in 1988–1989 to restrict consumption. Recently, the Food and Drug Administration used a nearly identical level of 1 pg ITEQ/g egg as a level at which commercial egg producers must consider eggs adulterated (32). Cancer risk estimates from a single prospective study of PCDD/PCDF-exposed workers, published subsequent to this event, indicates higher risks, 10^{-3} – 10^{-2} , for similar intakes of 60–70 pg TCDD/day (33).

Of the eggs from the 25 Oroville homes, 72% contained levels > 1 ppt ITEQ (Table 1). Although the eggs at the index homes were among the highest in concentration, these results indicated contamination in the greater Oroville area. Eggs with levels > 1 ppt came from homes up to 11 km from the wood treatment facility. Eggs from chickens that foraged on soil in a similar rural area with no known industrial sources, Nevada County (24) (80 km from Oroville), had much lower contamination levels, approximately equal to levels in commercial eggs. Notably, in all of our initial analyses, TCDD was not measured and detection limits were not always quantified for PCDD/PCDF congeners. In these instances, zero was entered into the ITEQ calculations. Analysis of more recent Oroville egg samples ($n = 10$) have quantified TCDD and detection limits. In these recent samples, TCDD averages 0.4 ppt, with 40% below the detection limit of 0.1–0.2 ppt; the overall percentage of PCDD/PCDF concentrations below detection limits (20%) is similar to initial sampling; and ITEQ values, calculated using half of the detection limit for concentrations below the detection limit, are minimally different (on average 0.2 ppt ITEQ) than calculations using zero for congener concentrations below detection limits (31). In 1989 in Oroville, the CDHS issued an advisory to residents within 11 km of the facility urging caution in consumption of products from animals that have contact with soil (34). This advisory followed the initial product sampling (Table 1) and the preliminary PCDD serum results.

That the contamination may not be solely attributable to the 1987 fire was suggested by two samples of liver from cows raised at the same index house and slaughtered in 1985 and in 1988; the samples had nearly identical levels and patterns (17). However, in 1963 there was a larger PCP fire at the wood treatment facility; that fire burned for 1 week. Recent estimates indicate a 25–100-year half-life for PCDDs/PCDFs in soil (35). Other potential industrial sources were located near the wood treatment facility; most notably, four teepee burners were within 2 km. Teepee burners were used as incinerators prior to 1980 to burn waste wood, including the remains in PCP wood-treatment

Table 1. Dioxin ITEQ concentrations in chicken eggs from exposure (Oroville) and comparison (Nevada County) areas.

Area sampled	Number of sites/samples	ITEQ concentration (pg ITEQ/g egg or ppt) ^a				Number > 1 ppt
		Average	Geometric mean	Minimum	Maximum	
Home-produced eggs						
Oroville: index homes	2/4 ^b	10.03	9.01	5.6	18.26	4 (100%)
Greater Oroville area (11-km radius)	23/24 ^{c,d}	3.40	1.72	0.08	13.16	15 (65%)
Nevada County	5/6 ^d	0.15	0.04	0.01	0.63	0
Commercial eggs						
Oroville	5/6 ^d	0.03	0.03	0.01	0.48	0

^aWhere PCDD and PCDF values were below detection limits, zero was entered into the ITEQ calculation. ^bSamples were collected at two different times at each home. ^cCorresponding statistics do not include index homes. ^dCorresponding statistics are based on one sample from each collection site.

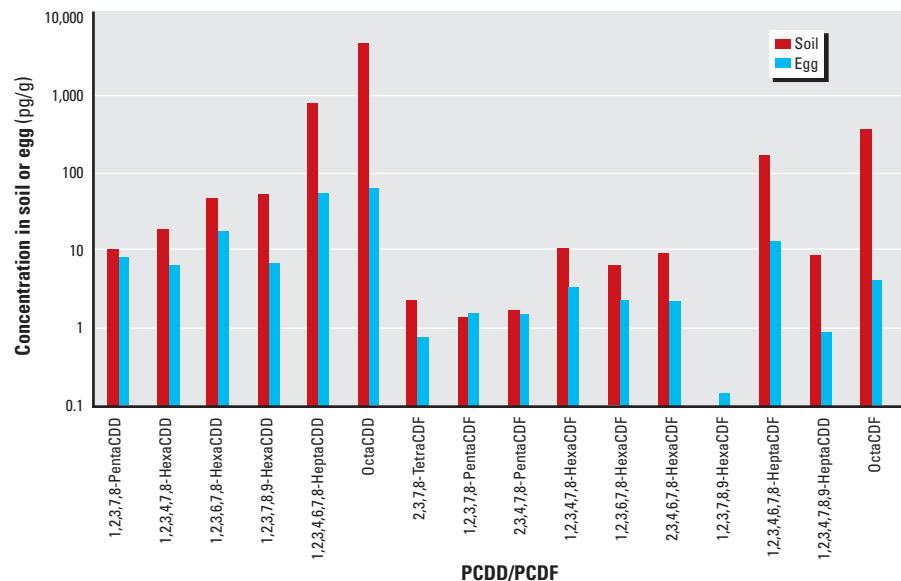


Figure 1. Average PCDD/PCDF concentrations in soil ($n = 2$) and eggs ($n = 4$) from Oroville index homes.

cylinders (36). Burning PCP-treated wood in homes is also a possible source of contamination, but larger burners are present at industrial facilities and residential emissions are unlikely to be as great as emissions from industrial sources.

Methods

All nine individuals over 12 years of age with known consumption of dioxin-contaminated eggs from the two index households were invited to participate in blood sampling; all elected to do so (17). The average age of the nine participants was 28 years. There was an approximately equal sex distribution in the two households, with one more male than female. None had previous occupational exposures to PCDDs/PCDFs. A rural Northern California community in Nevada County, 80 km southeast of the study area, served as a comparison area because it had no major industrial sources of dioxins and furans, including wood treatment facilities, and because it was geographically comparable to the study area. Comparison area subjects were recruited with the assistance of the Nevada County Health Department (Grass Valley, CA), which advertised for volunteers for the study. Between three and seven potential age- and sex-matched comparison subjects volunteered for each South Oroville subject. Each study subject and potential volunteer completed a questionnaire about the consumption of eggs, beef, and chicken. Similarity of food consumption patterns with those of index home participants was used as the basis for final selection of comparison participants. Subjects were matched on consumption of commercial eggs, beef, and chicken to the consumption of home-produced chicken, eggs, and beef of the index home participants. None of the comparison subjects ate home-produced meat and only one ate backyard-produced eggs, which had contamination similar to commercial eggs (Table 1). All participants were informed verbally of the purpose of the study at the time of blood drawing and agreed to participate by a written informed consent.

Collection and analysis procedures for the 2,3,7,8-substituted PCDDs/PCDFs have been described by Goldman et al. (17). In brief, we collected blood in April and May 1988 using standard blood collection supplies. Needles and tubing were provided by the CDC. Blood collection bags were provided by the Red Cross (Oroville, CA) and were screened for dioxins and furans by the CDC. Specimens were blinded prior to shipment to the CDC in May 1988. The laboratory method involves spiking the serum with a mixture of ^{13}C -labeled PCDDs/PCDFs; extraction with saturated aqueous ammonium sulfate, ethanol, hexane, and sulfuric

acid; column cleanup using a method developed by Smith and Stalling (37) and modified by the CDC laboratory; and calculation of total lipid content by a summation procedure (8,11). Each sample was spiked with the same mixture of ^{13}C -labeled standards and the samples were worked up in runs of five samples (one blank, three unknowns, and one quality control). The samples were analyzed by high-resolution gas chromatography (SP2331; Hewlett-Packard, Palo Alto, CA) and high-resolution mass spectrometry on the same instrument (VG 70S; Fisons Instruments, Manchester, England) by the same operator. 2,3,7,8-Substituted isomers were calculated using standard curves developed for each congener by the isotope-dilution mass spectrometry technique.

For PCDDs/PCDFs with sufficient samples above detection limits, descriptive statistics and analysis of variance (ANOVA) were calculated. Blood levels were summarized using the ITEQ method (23). Although these calculations do not provide a risk characterization, ITEQ values ease comparison to levels found in other serum studies. Congener and ITEQ values were apparently log-normally distributed and were log-transformed prior to statistical analysis. However, arithmetic means were also calculated and were used when comparing results to other studies. ANOVA and *t*-tests were used to examine the separate effects of membership in the three exposure groups (comparison subjects and the two index homes). Multiple linear regression was used to test for an association with exposure group, controlling for age and sex. Bivariate linear correlations were computed to examine the relationship between individual isomer levels. All statistical analysis was conducted with SAS software (38).

Results

We reviewed questionnaires to rank subjects by potential exposure to dioxin-contaminated animal products. Index home residents reported consuming home-produced chicken eggs and beef. Participants from one index home lived at the home for 2 years. The adults ate 14–35 eggs/week and the children (12–19 years of age) ate 3–6 eggs/week. They ate no homegrown beef or chicken meat. Subjects from the other index home had lived there for 15 years. All of those subjects ate 3–12 eggs/week and homegrown beef at least 5 times/week. Chicken meat was consumed once, several years prior to testing. Residence in these homes was used as a surrogate index of exposure, both for years of exposure and for quantity of contaminated food consumed per week (eggs alone vs. eggs and beef).

Geometric mean serum levels of 2,3,7,8-substituted PCDD and PCDF and 95%

confidence limits by exposure group are shown in Table 2. PCDD/PCDF distributions were log-normally distributed (*p*-value for *W* statistic prior to transformation < 0.05 for all congeners except heptaCDD and octaCDD, and after transformation, for all congeners *p* > 0.2). However, PCDD/PCDF geometric means were only slightly different (on average, 8% greater) than arithmetic means (not reported). Five PCDD/PCDF congeners were not available for statistical analysis. Specifically, octaCDF levels were not determined by the CDC because of background laboratory contamination; 1,2,3,7,8-pentaCDF and 1,2,3,4,7,8,9-heptaCDF were not detected in any samples, with detection levels between 1 and 18 ppt. 2,3,4,6,7,8-HexaCDF and 1,2,3,7,8,9-hexaCDF levels were available for only a few people. Additionally, all of the determinations for one matched comparison subject were not reportable because one or more quality assurance or control parameters were out of range; there was insufficient sample to repeat the analyses. Other occasional values were not reported or were below detection limits (Table 2).

ANOVA results (Table 2) indicate significant differences between the mean congener levels for the three groups. Specifically, membership in an exposure group predicted an individual's concentration of 2,3,7,8-TCDD, 1,2,3,7,8-pentaCDD, 1,2,3,4,7,8-hexaCDD, 1,2,3,6,7,8-hexaCDD, 1,2,3,7,8,9-hexaCDD, 2,3,4,7,8-pentaCDF, 1,2,3,4,7,8-hexaCDF, 1,2,3,6,7,8-hexaCDF, 1,2,3,4,6,7,8-heptaCDF, and ITEQ. Eating both home-produced eggs and beef conferred the greatest risk for elevated levels of these PCDDs and PCDFs; 95% confidence limits did not overlap with those for comparison subjects for all of the elevated congeners except 2,3,7,8-TCDD and 1,2,3,4,6,7,8-heptaCDF. Index home residents who ate only eggs had lower levels of all PCDDs and PCDFs. The difference between levels of the two index home groups was statistically significant for TCDD, 1,2,3,7,8-pentaCDD, 1,2,3,4,7,8-hexaCDD, 1,2,3,6,7,8-hexaCDD, 2,3,4,7,8-pentaCDF, 1,2,3,4,7,8-hexaCDF, 1,2,3,6,7,8-hexaCDF, and ITEQ values. Nevertheless, although most of the PCDD/PCDF levels among the index home residents who ate eggs only were higher than controls, these differences were statistically significant for only the penta- and hexaCDDs and ITEQ values. This difference was the most pronounced for 1,2,3,7,8,9-hexaCDD.

An increase in serum level was associated with males for most PCDDs/PCDFs, ranging from 15 to 55%. Among females the mean ITEQ concentration was 29 pg/g fat; among males it was 39 pg/g fat. Increasing age was also associated with an increase in serum level

for most PCDDs/PCDFs, ranging from 4 to 19% for each 10 years of life. The mean ITEQ concentration was 31 pg/g fat for the youngest group (10–19 years of age, *n* = 4) and 65 pg/g fat for the oldest (50–59 years of age, *n* = 2). Adjusting for age and sex did not change the correlation between exposure group and PCDD/PCDF concentrations. Multivariate regression models with age, sex, and exposure group as independent variables to predict each of the individual congeners and ITEQ concentrations generated regression coefficients and *p*-values only minimally different from that reported for the one-way ANOVA analyses (Table 2). In these models, of all of the age and sex coefficients, only those for age-associated increases with 1,2,3,7,8-pentaCDD, 1,2,3,4,7,8-hexaCDD, 1,2,3,6,7,8-hexaCDD, 2,3,4,7,8-pentaCDF,

1,2,3,4,6,7,8-heptaCDF, and ITEQ values were statistically significant (*p* < 0.05).

Table 3 shows the bivariate correlation matrix for the individual congeners and TEQ concentrations. Very strong correlations (*p* < 0.001) were found between all of the 2,3,7,8-penta- and hexaCDDs/CDFs. The higher chlorinated congeners showed weaker relationships with others. OctaCDD did not correlate well with most other measurements.

Discussion

These analyses confirm preliminary findings (17) of a dose–response relationship between the consumption of contaminated home-produced eggs and meat and serum dioxin and furan levels. Specifically, people that ate both home-produced eggs and meat for 2–15 years had statistically significant 2- to 6-fold

increases in blood levels of 2,3,7,8-substituted TCDD, pentaCDD, hexaCDDs, tetraCDF, pentaCDF, hexaCDFs, heptaCDF, and ITEQ concentrations. People who ate only home-produced eggs for 2 years had lower elevations, with a 0.5- to 3-fold elevation between those eating eggs alone as compared to controls for the pentaCDD, hexaCDDs, and ITEQ values. The household residents who ate both eggs and meat had higher levels of pentaCDD and hexaCDDs than have been observed among all previously studied contaminated-fish and -seafood consumers (4–7) and higher hexaCDFs, heptaCDFs, and PCDD/PCDF ITEQ concentrations than levels observed among North American fish consumers (6,7) (Figure 2). These data document that contaminated animal product intake may significantly elevate human serum PCDD/PCDF levels.

In our study, age and sex trends are apparent for most congeners and ITEQ concentrations, although only some of the age-associated trends are statistically significant. In one study of U.S. urban populations, associations between age and PCDD/PCDF adipose tissue levels were demonstrated but sex differences were not (39). Recent studies from Seveso, Italy, demonstrated the reverse male/female relationship, with females having markedly higher levels of TCDD than males after 20 years of exposure (40). Fish and seafood consumption studies of PCDDs/PCDFs have generally not been designed to examine sex differences (4–7). Two fish consumption studies demonstrated age relationships; one study showed that males have higher levels of furans than females do (4,16). Nevertheless, these effects may be due to older participants having more years of fish consumption and males eating more fish than females (16). Our study was too small to study the independent effects of years of consumption or consumption differences between men and women. Similarly, other confounders, such as breast-feeding, which may decrease body burden among women, could not be studied.

Table 2. Geometric means, CI, and ANOVA results for PCDDs and PCDFs by exposure group.

Congener	Geometric mean concentration (CI); number not reported/number below DL ^a			ANOVA (<i>p</i> -value)
	Egg consumers (<i>n</i> = 4) ^b	Egg and beef consumers (<i>n</i> = 5) ^{b,c}	Comparison subjects (<i>n</i> = 8)	
2,3,7,8-TCDD	3.2 (2.5–4.2)	5.5 (2.7–11.0)**	2.5 (1.6–3.9) 0/1, < 4.2	< 0.02
1,2,3,7,8-PentaCDD	15.1 (7.6–30.4)**	40.9 (26.0–64.2)###	6.2 (4.2–9.2)	< 0.0001
1,2,3,4,7,8-HexaCDD	10.7 (5.4–20.9)	27.5 (16.1–47.2)###	4.5 (1.9–10.6) 0/1, < 1.2	< 0.001
1,2,3,6,7,8-HexaCDD	66.4 (51.7–85.2)*	150 (91.7–234)###	43.7 (32.9–57.4)	< 0.0001
1,2,3,7,8,9-HexaCDD	20.0 (11.0–40.1) [#]	30.2 (18.3–48.9) [#]	6.7 (4.7–9.5)	< 0.0001
1,2,3,4,6,7,8-HeptaCDD	130 (55.4–306)	140 (53.2–369)	107 (58.7–194)	NS
OctaCDD	659 (320–1358)	685 (515–911)	588 (460–766)	NS
2,3,7,8-TetraCDF	NR ^d	1/0 1.7 (0.7–4.2)	1/0 3.7 (2.2–6.2)	NR ^d
2,3,4,7,8-PentaCDF	4.4 (3.3–5.8)	19.6 (12.2–31.5)###	4.8 (2.9–8.0)	< 0.0001
1,2,3,4,7,8-HexaCDF	6.1 (2.6–14.8) 0/1, < 7.3	28.4 (19.0–42.7)###	5.1 (3.3–8.1)	< 0.0001
1,2,3,6,7,8-HexaCDF	5.6 (3.1–9.8) 0/1, < 7.5	17.3 (11.6–25.8)###	5.4 (3.2–9.0)	< 0.001
1,2,3,4,6,7,8-HeptaCDF	21.1 (11.2–40.0)	27.9 (16.7–46.4)** 1/0	14.5 (10.7–20.0) 2/0	= 0.01
ITEQ	26.7 (19.4–36.9)*	63.7 (40.5–100.2)###	17.0 (12.2–23.8)	< 0.0001

Abbreviations: CI, 95% confidence interval; DL, detection limit; NR, not reported; NS, not significant (*p*-value > 0.05). ^aBlank cell indicates that all values are reported above DLs. When compounds were not detected, half of the detection level was used to compute statistics and ITEQ values. When compounds were not reported, values were considered missing in statistical computations and zero was used to compute ITEQ values. ^bStatistically significantly higher than comparison subjects. ^cStatistically significantly higher than egg consumers. ^dInsufficient number reported to compute means and/or ANOVA. **p* < 0.05. ***p* < 0.01. [#]*p* < 0.001.

Table 3. Pearson correlation coefficients for PCDDs/PCDFs (log-transformed) among exposure and comparison participants.

Congener	2,3,7,8-TCDD	1,2,3,7,8-PentaCDD	1,2,3,4,7,8-HexaCDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	1,2,3,4,6,7,8-HeptaCDD	OctaCDD	2,3,4,7,8-PentaCDF	1,2,3,4,7,8-HexaCDF	1,2,3,6,7,8-HexaCDF	1,2,3,4,6,7,8-HeptaCDF
2,3,7,8-TCDD	...										
1,2,3,7,8-PentaCDD	0.78 [#]	...									
1,2,3,4,7,8-HexaCDD	0.87 ^{##}	0.89 ^{##}	...								
1,2,3,6,7,8-HexaCDD	0.89 ^{##}	0.97 ^{##}	0.84 ^{##}	...							
1,2,3,7,8,9-HexaCDD	0.78 [#]	0.91 ^{##}	0.91 ^{##}	0.87 ^{##}	...						
1,2,3,4,6,7,8-HeptaCDD	0.49*	0.38	0.61 ^{**}	0.33	0.56*	...					
OctaCDD	0.40	0.45	0.59*	0.37	0.31	0.63*	...				
2,3,4,7,8-PentaCDF	0.79 [#]	0.82 ^{##}	0.81 ^{##}	0.86 ^{##}	0.76 [#]	0.48*	0.42	...			
1,2,3,4,7,8-HexaCDF	0.79 [#]	0.85 ^{##}	0.85 ^{##}	0.86 ^{##}	0.82 ^{##}	0.52*	0.39	0.96 ^{##}	...		
1,2,3,6,7,8-HexaCDF	0.82 ^{##}	0.79 ^{##}	0.85 ^{##}	0.77 ^{##}	0.82 ^{##}	0.61*	0.44	0.92 ^{##}	0.95 ^{##}	...	
1,2,3,4,6,7,8-HeptaCDF	0.48	0.65 ^{**}	0.67 ^{**}	0.63*	0.52*	0.48	0.21	0.48	0.68 ^{**}	0.58*	...
ITEQ concentration	0.83 ^{##}	0.99 ^{##}	0.81 ^{##}	0.94 ^{##}	0.81 ^{##}	0.5*	0.29	0.89 ^{##}	0.89 ^{##}	0.84 ^{##}	0.60*

p* < 0.05. *p* < 0.01. [#]*p* < 0.001. ^{##}*p* < 0.0001

Levels among people residing in rural areas of the United States, with age and sex distribution information, have not been reported. Of the U.S. urban populations that have been studied (2,39), adipose tissue concentrations in 48 composites of U.S. regional, age, and sex groups (excluding occupationally exposed) (39) represent a group similar to ours, including younger participants and women and collected during a similar time, 1986–1987. Congener levels among our high exposure participants, those eating both home-produced eggs and beef, are generally higher than the observed levels among this urban population (Figure 2). Among our high consumers the average ITEQ concentration was 64 pg/g, whereas among the urban population the average was 26 pg/g. Our exposed participants, for a two home-produced egg/day diet, would have consumed an estimated 1,200 pg ITEQ/day and, for an average size serving of home-produced beef (150 g), 313 pg ITEQ/day. The estimate of average U.S. intake of PCDDs/PCDFs for commercial dairy, beef, and fish combined is 0.3–192 pg ITEQ/day (3). Although the estimated average intake for the U.S. population

is limited (e.g., small number of samples and eggs were not included), intake among our exposed participants is probably greater than that for average North American consumers and elevations among our participants as compared to average North Americans are not unexpected.

Among our rural comparison group, congener and ITEQ levels were generally lower than the levels in urban U.S. populations (Figure 2). Whether lower levels among rural participants are due to lower levels in rural commercial food products as compared to urban commercial food products or whether these differences suggest that urban residents have greater exposures via other pathways (e.g., air) was not determined.

PCDD/PCDF levels among our exposed participants are well below levels found in highly exposed populations such as chemical workers and levels resulting from poisoning episodes (41). Acute disease is not expected. Observation of human health risks at the serum levels described here are estimated from animal studies but have not, for the most part, been described (2). One study suggested neuropsychologic differences

between groups whose serum levels were comparable to those reported here (42). Two European occupational studies suggested elevated overall cancer rates among those exposed to generally much higher levels of dioxin-like compounds (43,44). However, in one of these studies, the difference observed between background and the lowest exposed group (40 pg ITEQ/g), among whom there were significantly elevated cancer rates (43), is approximately equal to the difference (48 pg ITEQ/g) between the means of our high exposed and comparison groups. Although there were limitations to that study, the combination of laboratory toxicologic data and suggestive human data is sufficient to direct public policy. Exposures such as those identified here should be reduced.

If strides in public health protection or exposure reduction are to be made, the sources of contamination contributing to body burdens require identification. It would be extremely useful to identify sources from blood levels in a population. Blood levels may reflect local industrial sources, as illustrated in Norwegian crab consumers (5). In Oroville, identified industrial sources are primarily PCP or PCP incineration.

The PCDD/PCDF concentration pattern typical of PCP is illustrated by a study of 20 German PCP workers which documents that the higher chlorinated dioxins dominate: mean blood levels of 2,514 and 184,224 pg/g fat for 1,2,3,4,6,7,8-heptaCDD and octaCDD, respectively, and levels between 1 and 240 pg/g fat for all other PCDDs/PCDFs (45). Although heptaCDD and octaCDD levels were the highest of any of the congeners, the mean levels among exposed and comparison subjects are not significantly different. There are several possible explanations. First, there may be unidentified non-PCP sources of dioxins in the study area that contribute to the lower chlorinated dioxin and furan burden. Second, blood from the gastrointestinal tract passes to the liver before systemic absorption. Workers are primarily exposed dermally and through inhalation, and compounds are directly absorbed into the general circulation. Chickens (29), cattle (28), and horses (20) have shown preferential depositing of the higher chlorinated dioxins in the liver as compared to adipose tissue following ingestion. In the blood of Norwegian crab eaters, octaCDD was clearly less dominant than the other congeners as compared to the crabs (5). A third explanation is that other differences between PCDDs/PCDFs in bioavailability and retention alters the profile of contaminants. Soil and egg PCDD/PCDF levels at the index homes (Figure 1) and in a chicken-feeding study (29) indicate relatively

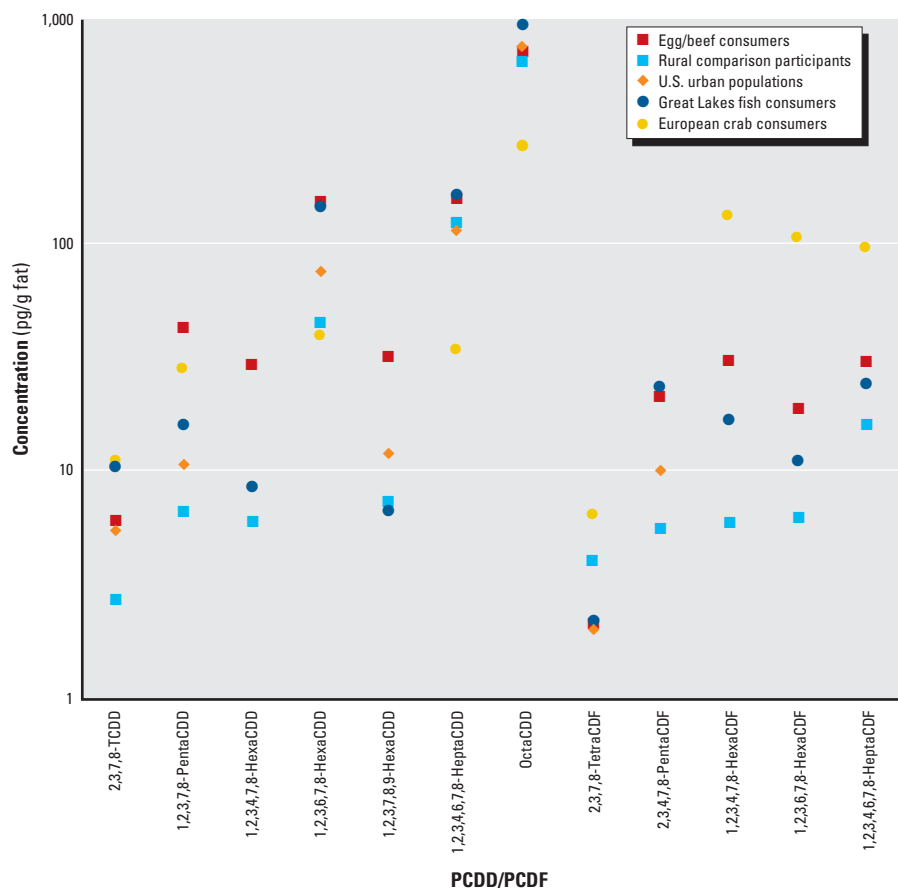


Figure 2. Average PCDD/PCDF serum concentrations of egg and meat consumers, rural comparison participants, high contaminated fish and crab consumers, and in adipose tissue of U.S. urban populations. Congeners with unplotted values were not reported. Data on U.S. urban populations from Orban et al. (39). Data on Great Lakes fish consumers from Anderson et al. (7). Data on European crab consumers from Johansen et al. (5).

higher soil-to-egg concentration ratios for the higher chlorinated congeners. The feeding study further indicated that total absorbed dose of higher chlorinated dioxins, measured by concentrations in many tissues, including liver, is less as compared to the other PCDDs/PCDFs. Thus, evidence supports the latter two explanations of less uptake of the higher chlorinated PCDD congeners by ingestion as compared to workplace exposures. In the food chain, this effect will be manifested both after animals graze and after humans eat the animal products. Among those nonoccupationally exposed to PCDDs/PCDFs in food, a PCP pattern may shift toward the lower chlorinated PCDDs/PCDFs.

The congener profile displayed in PCP and among all participants here is also seen in other background U.S. populations (39). PCP was widely used in the United States on wood, including telephone poles. PCDD/PCDF contamination has been found in wood structures of the commercial cattle industry; the subsequent beef contamination was equivalent to that reported in beef from the index homes (46). However, the major source of dioxin emissions in the United States is considered incineration (47). During long-range environmental fate and transport from incineration sources to direct human exposure, the higher chlorinated dioxins may be enhanced (48). Although congener profiles in human blood fat may reflect local sources (5) (particularly if the source profile is dominated by the lower chlorinated congeners) in background U.S. populations, environmental and biologic transformations may obscure PCP and incineration source profiles.

In PCP-exposed populations, the advantages of analyzing for all dioxin and furan congeners in serum may be limited. Our correlation analysis indicated that measurement of any one of the nine elevated PCDDs/PCDFs could act as a surrogate for any of the other nine PCDDs/PCDFs or ITEQ concentrations. For example, 1,2,3,7,8-pentaCDD, which showed the highest fold elevation, could serve as a marker for exposure.

This study demonstrates that food chain bioaccumulation can occur at small subsistence farms in an area with PCDD/PCDF sources. Contamination may be potentially widespread: soil levels in our study are only slightly above estimates of background soil concentrations of < 1 ppt in rural areas and 11 ppt in urban/suburban areas with no known industrial sources (49). Nevertheless, although many risk assessments have been conducted for beef consumption, involving estimates of over 40 different input parameters (50), there has been little measurement of animal products from areas near PCDD/PCDF industrial sources in the United

States. Chicken egg contamination has been detected in eggs from soil-foraging chickens in Europe (51). In California, contamination has been detected in eggs from chickens raised across the street from a metal recovery incinerator: the PCDD/PCDF pattern in the eggs followed the pattern found in flyash from the incinerator (24). Extensive sampling of milk from cows raised near incinerators in Europe has shown that this milk contains elevated levels of PCDDs/PCDFs (52).

Farming practices on small farms differ considerably from commercial operations and the extent of these practices is difficult to determine. Eggs from range-fed chickens have become increasingly displayed in the commercial market in California. As the family farm has disappeared in the United States, many people who formerly farmed continue to live in rural areas and enjoy raising animals for eggs, meat, and other products. Small farm practices may be in conflict with land use and industrial activities associated with PCDDs/PCDFs. Current guidelines for 2,3,7,8-TCDD in residential soil in the United States use 1 ppb as an action level to protect children while playing in soil (53). Estimates of soil concentrations at the index homes suggest that low parts-per-trillion levels are of concern for dietary exposure pathways. These estimates have been confirmed by laboratory studies using soil with a high organic content that are therefore representative of soil with the probable lowest bioavailability (30) and analysis of further egg and soil samples from Oroville and elsewhere (31). To protect against animal product contamination and subsequent indirect dioxin exposure, it may be necessary to substantially reduce this soil cleanup level.

Environmental and biologic half-lives, each of which may be measured in decades (33,35), strongly indicate that indirect dioxin exposure is a long-term problem. Future research should be directed at environmental fate and biologic uptake and elimination. Source identification and emissions testing not only aid in source attribution but also allow recommendations for source reduction. Certainly enough is now known, however, that site-specific risk assessments include these exposure routes. Notably, poultry products have not been included in facility specific risk assessments (54).

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