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Human exposure to dioxins from food, 1999–2002

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Abstract

In response to aggressive attempts to control dioxin emissions over the last 35 years, human exposures to dioxins from the environment have declined significantly. The primary source of human exposure to dioxins at present is food. The sources of dioxins in food are not well understood and are probably varied. Data on the levels of dioxins measured in various foods for samples collected from 2000 to 2002 have recently been released by the US Food and Drug Administration as part of its Total Diet Study. Data on samples collected in 1999, and released in 2002, are also available. Based on those data and on the US Department of Agriculture's most recent food consumption survey (1994–1996 & 1998 Continuing Survey of Food Intakes by Individuals), estimates of dioxin intake for the total US population and for three age groups of children were obtained. Results show that the most recent mean dietary exposures for all groups are below 2 pg TEQ/kg BW/day, the tolerable daily intake established for dioxins by the World Health Organization. Between 1999 and 2002 mean dioxin intakes from food appear to have decreased, but when estimates are adjusted based on a standardized limit of detection and evaluating only those {congener × food} combinations common to all 4 years, no trend is apparent. When dioxin concentrations below the limit of detection are represented by one-half the limit, approximately 5% of the intake estimates for 2-year-olds and 1% of the intake estimates for 6-year-olds exceed the tolerable daily intake by about 10%, although such upper-percentile estimates should not be equated with excess risk. When non-detectable dioxin values are set to zero (i.e., when only dioxin values actually measured are used), only 1% of intake estimates exceed the tolerable daily intake for 2-year-olds. As expected, about 50% of daily dietary dioxin intake by the total US population is attributable to meat and dairy products, based on the same food group classifications used by the National Academy of Sciences' Committee on the Implications of Dioxin in the Food Supply. This information may be useful for targeting future risk management activities.

Keywords: Dioxins; Total diet study; PCDD/PCDFs; Risk

1. Introduction

Potential public health risks from environmental exposures to chlorinated dioxins and related compounds continue to be the subject of much research, regulation, and debate. Available data on emissions, environmental and food levels, and human body burdens of dioxins indicate a several-fold reduction in exposures and body burdens since 1970 (Hays and Aylward, 2003), suggesting that efforts to control dioxin emissions and to reduce exposures are succeeding. For example, the US Environmental Protection Agency

Abbreviations: CSFII, Continuing Survey of Food Intakes in Individuals; EPA, US Environmental Protection Agency (EPA); FAO/WHO, Food and Agriculture Organization of the United Nations/World Health Organization; FARE™, Food And Residue Evaluation; LOD, limit of detection; PCBs, polychlorinated biphenyls; PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; TDI, tolerable daily intake; TEF, toxic equivalency factor; TEQ, toxic equivalence; USDA, US Department of Agriculture; USFDA, US Food and Drug Administration.

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(EPA) reported that emissions of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) from quantified sources decreased from more than 14,000 g TEQ[1]/year in 1987 to about 3300 g TEQ/year in 1995 and are expected to decline an additional 60% by 2005 (USEPA, 2004). Human body burdens of TCDD in the US decreased 10-fold and dioxin TEQ decreased 4–5-fold between 1972 and 1999, which, due to the long elimination half-lives of dioxins, implies a decrease in exposure of more than 95% (Hays and Aylward, 2003). EPA has estimated that more than 90% of the remaining human exposures to dioxins occur through food consumption, primarily from animal fat (USEPA, 2004). Dioxins are not deliberately added to food or created during food processing. Natural sources of dioxins, such as forest fires, contribute to a background level of dioxin exposure from food (USEPA, 2004), as may unregulated anthropogenic sources such as uncontrolled burning of household waste in barrels (Lemieux et al., 2003).

To help evaluate human exposure to contaminants such as dioxins from food, the US Food and Drug Administration (FDA) conducts an ongoing market basket survey of approximately 280 core foods in the US to determine levels of various pesticide residues, contaminants, and nutrients. Data on dioxin concentrations are available for the years 1999–2002. The foods analyzed represent the major components of the diet of the US population, based on results of food consumption surveys conducted by the US Department of Agriculture (USDA). The USDA surveys provide consumption data on more than 3000 individual foods. Using the USDA survey data, FDA derives summary consumption data for representative foods in the US diet. For example, "apple pie" in FDA's summary represents all fruit pies and pastries in the USDA survey. FDA most recently updated its consumption summaries in May 2004.

For this study, we combined FDA's 2004 food consumption summaries with FDA's dioxin TEQ analyses for the years 1999–2002 to obtain annual estimates of average daily dioxin intakes for the total US population and for three age groups of children: 2 years, 6 years, and 14–16 years. We estimated both mean and upper-percentile intakes and evaluated whether there were trends in average daily dioxin intake over time. We compared the results to the tolerable daily intake (TDI) for dioxins, 2 pg TEQ/kg BW/day (EC, 2001; JECFA, 2001), which is considered the amount of dioxin equivalents that can be ingested over a lifetime without appreciable health risk. Finally, we determined which food groups make the largest contributions to dietary exposure.

2. Methods

2.1. Dioxin levels in foods

The Total Diet Study (TDS) is an ongoing market basket survey of approximately 280 core foods in the US food supply to determine levels of various pesticide residues, contaminants, and nutrients in foods. Four market baskets are generally collected each year, one in each of four geographic regions of the US (i.e., West, North Central, South, and Northeast). FDA's Dioxin Monitoring Program analyzed TDS samples from one market basket each year to determine levels of polychlorinated dibenzo *p*-dioxin (PCDD) and polychlorinated dibenzofuran (PCDF) congeners.[2] The limits of detection (LOD) for the various {congener × food} combinations were not constant over the 4 years, however. For this analysis the 2000–2002 TDS dioxin data were obtained online from the FDA website http://www.cfsan.fda.gov/~lrd/dioxdata.html. The 1999 data were obtained from FDA through the Freedom of Information Act.

Because the various congeners have different toxicity levels, a TEQ value was calculated for each food sample by standardizing the individual congener levels detected in each sample by multiplying them with the appropriate Toxic Equivalency Factor (TEF) and summing those standardized values. FDA released three sets of TEQ values for the 2000–2002 data, the first assuming that levels for nondetected congeners are zero, the second assuming they are equal to LOD/2, and the third assuming they are equal to the LOD. A similar approach was applied to the 1999 data. For this analysis, the data that were derived based on the assumption that non-detects are equal to LOD/2 or to zero were used.

2.2. Food consumption levels

FDA derived summary consumption values for the foods in the TDS based on data from USDA's 1987–88 National Food Consumption Survey and updated those summary values in 1990. The consumption summaries were further updated by the FDA in May 2004, based on the 1994–1996 & 1998 Continuing Survey of Food Intakes in Individuals (CSFII), USDA's most

[1] TEQ, toxic equivalence. Because the various congeners have different toxicity levels, a toxicity equivalence (TEQ) value was calculated for each food sample by standardizing the individual congener levels detected in each sample by multiplying them with the appropriate Toxic Equivalency Factor (TEF) and summing these standardized values. The TEFs used were established by the World Health Organization and are calculated relative to 2,3,7,8-TCDD (van den Berg et al., 1998, p. 4).

[2] The specific PCDD and PCDF congeners measured by FDA can be found at http://www.cfsan.fda.gov/~lrd/dioxdata.html [p. 6].

recent food consumption survey. Consumption data for the mean per capita estimates were obtained for this analysis online from the FDA TDS website http://www.cfsan.fda.gov/~comm/tds-toc.html. The upper percentile consumption data were obtained using a proprietary software program developed by Exponent®, FARE™ (Food And Residue Evaluation) version 7.54.

2.3. Dioxin intake estimates

Dietary dioxin intakes were estimated for the total US population by combining the TEQ levels for the 1999–2002 dioxin levels with consumption data from the recently updated 2004 TDS diets using Microsoft® Excel. Intake estimates were based on mean per capita two-day average consumption data for the total US population and for children 2, 6, or 14–16 years of age. The 90th, 95th, and 99th percentile intake estimates were also derived for the total US population and for each age group, using the proprietary software program developed by Exponent®, FARE™ version 7.54. All calculations were performed by Exponent®.

Because fewer foods and {congener × food} combinations were analyzed by FDA in 1999 and 2000 than in 2001 and 2002 (see Table 1), additional analyses were performed to facilitate comparisons of the total dioxin intakes for the purpose of identifying any time trends. For the time-trend analysis, only foods and congeners that were analyzed all 4 years were evaluated. The LODs of the various 9 {congener × food} combinations also varied across the 4 years,[3] so standardized dioxin intake estimates using the same limits of detection for all 4 years of data were derived by using the highest LOD for each {congener × food} combination. The intake estimates obtained in this manner were compared for each year to determine whether any changes over time occurred.

2.4. Contribution analysis

To determine the extent to which different food categories contribute to total dioxin intake, TDS foods were divided into the same categories as defined in Appendix B of the 2003 National Academy of Sciences report, *Dioxins and Dioxin-like Compounds in the Food Supply: Strategies to Decrease Exposure* (NAS/NRC, 2003). That appendix lists all the TDS foods comprising each of the categories: meat, fruits & vegetables, fish, fats/oils, eggs, dairy, poultry, and other. Contribution to total dioxin intake from each food category was calculated as a percentage based on the total intake (summed TEQ × consumption) for each category.

[3] For example, the LOD for 2,3,7,8-TCDD in "whole milk, fluid" was 0.01, 0.008, 0.001, and 0.003 pg/g food in 1999, 2000, 2001, and 2002, respectively [p. 8].

Table 1
Number of foods and {congener × food} combinations analyzed in the Total Diet Study as part of the US Food and Drug Administration's Dioxin Monitoring Program

	1999	2000	2001	2002
Number of foods	200	192	264	210
Number of {congener × food} combinations	3066	3218	4488	3568

A total of 17 congeners were analyzed each year, but not all foods were consistently analyzed for all 17 congeners. The specific congeners tested for by PDA are listed at http://www.cfsan.fda.gov/~lrd/dioxdata.html.

3. Results

Mean dioxin intake estimates during 1999–2002 for the total US population and for each age group evaluated are presented in Fig. 1. As the figure indicates, none of the daily dioxin intakes exceed 2 pg TEQ/kg BW/day, the TDI for dioxins established by the World Health Organization, with the exceptions of those for 2-year-olds in 1999 and 2000 and 6-year-olds in 1999 when non-detects are assumed to equal LOD/2. It is notable that the LODs for 2,3,7,8-TCDD in 1999 and 2000 were about an order of magnitude higher than in 2001 and 2002, so actual exposures were probably lower. When only those dioxin values that were actually measured above the limit of detection are included (i.e., LOD = 0), all of the children's exposure estimates are below the TDI.

Fig. 1 also shows an apparent downward trend in dietary dioxin intakes during 1999–2002. However, as Fig. 2 illustrates, when the same foods are used for all 4 years of intake estimates along with a standardized (highest) limit of detection, no trend over time is observed. A likely explanation for the apparent trend in dioxin intakes over time is the varying numbers of foods evaluated using different limits of detection each year, with higher limits of detection used in the earlier years, and not that a decrease in the levels of dioxins in foods occurred. When analyzed using a standardized approach and assuming non-detects are equal to LOD/2, dioxin intake estimates for 2-year-olds and 6-year-olds appear to exceed the TDI (Fig. 2b and c), indicating that intake estimates are quite sensitive to the LOD. When non-detects are set equal to zero instead of LOD/2, however, intake estimates do not exceed the TDI for those age groups. FDA has stated that assuming non-detects are equal to LOD/2 is likely to overestimate exposure to dietary PCDD/PCDFs and setting non-detects equal to zero (i.e., including only values actually measured) provides more realistic dietary intake estimates (USFDA, 2004).

Table 2 shows the mean, 90th, 95th, and 99th percentile intake estimates during 1999–2002 for the total US population and for each age group evaluated. Estimates

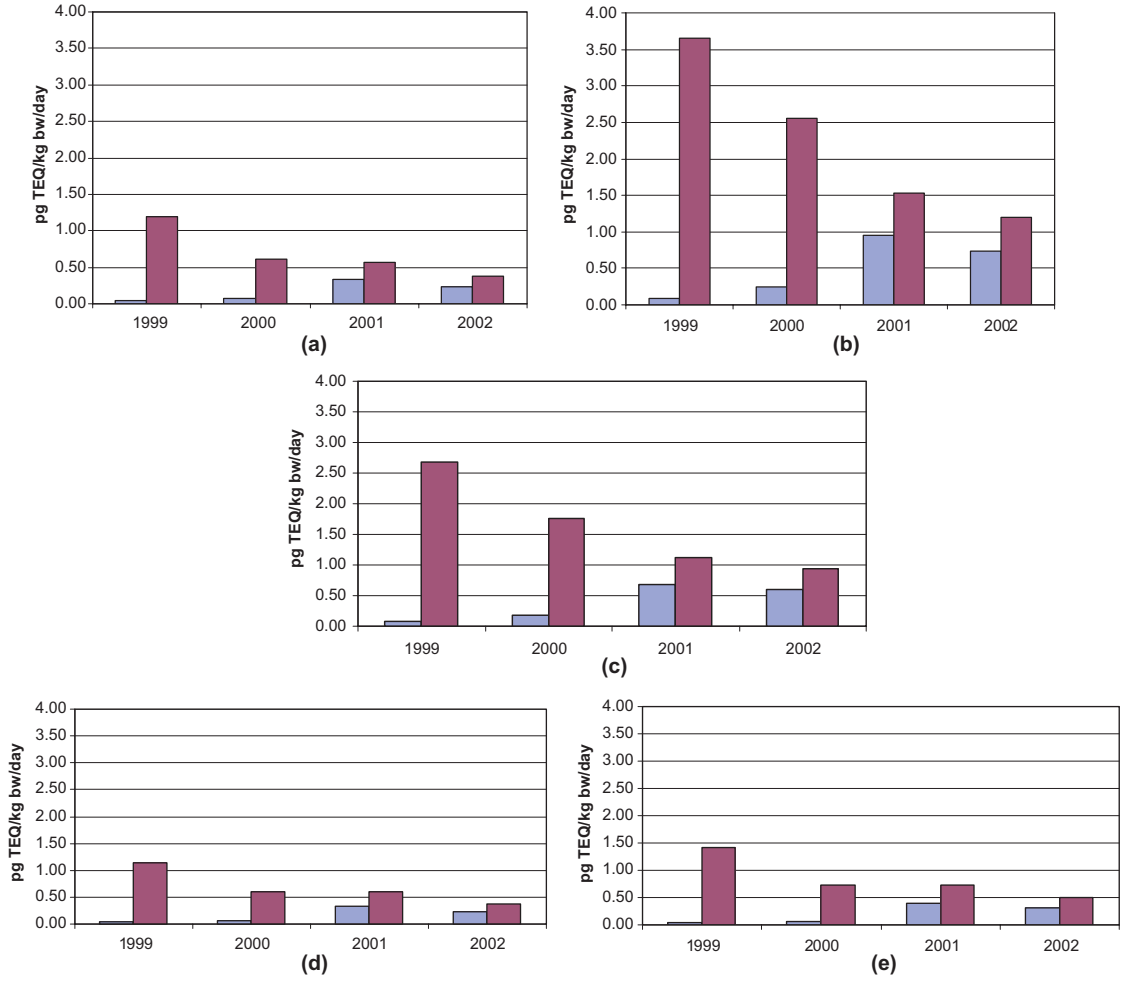

Fig. 1. Mean dioxin intake estimates during 1999–2002 for the total US population (a), 2 year old children (b), 6 year old children (c), 14–16 year old females (d), and 14–16 year old males (e), assuming non-detects are equivalent to either zero (■) or one-half the limit of detection (■).

shown in italics do not meet the minimum sample size criteria for this type of analysis as established by the National Centers for Health Statistics, so should be considered statistically unreliable. Based on the most recent data, approximately 5% of the intake estimates for 2-year-olds and 1% of the intake estimates for 6-year-olds exceed the tolerable daily intake by about 10%. Assuming non-detects are equal to zero, 1% of the intake estimates for 2-year-olds and none of the intake estimates for 6-year-olds exceed the TDI. But because those intake estimates are generated mathematically, they represent only theoretical children and should not be equated with estimates of the number of actual children at excess risk. The upper percentile estimates are based on distributions of two-day averages, which are expected to overestimate the corresponding percentiles of the longterm intake distributions (USEPA, 1996).

Fig. 3 depicts the extent to which different food types contribute to dietary dioxin intake. The largest contributors to total dioxin intakes are meat (32.2%) and dairy products (16.4%). Those contribution estimates are somewhat inaccurate because TDS foods comprise varied ingredients that may not fit strictly into one category. For example, vegetable beef soup is considered "fruits & vegetables" although meat is also present in the soup and the dioxin content is likely attributable to the fat in the beef.

4. Discussion

Since risk management actions aimed at reducing dioxin emissions were initiated in the 1970s, substantial decreases in environmental dioxin levels and in body burdens have been observed. Risk management of dioxin emissions was initiated because of concerns about its potential carcinogenicity, but concern remains due to other potential human health effects, such as developmental toxicity. The US EPA has been debating the appropriate exposure limit for dioxins since 1990; no risk estimate or exposure limit is agreed upon. No acceptable daily intake has been established by the US

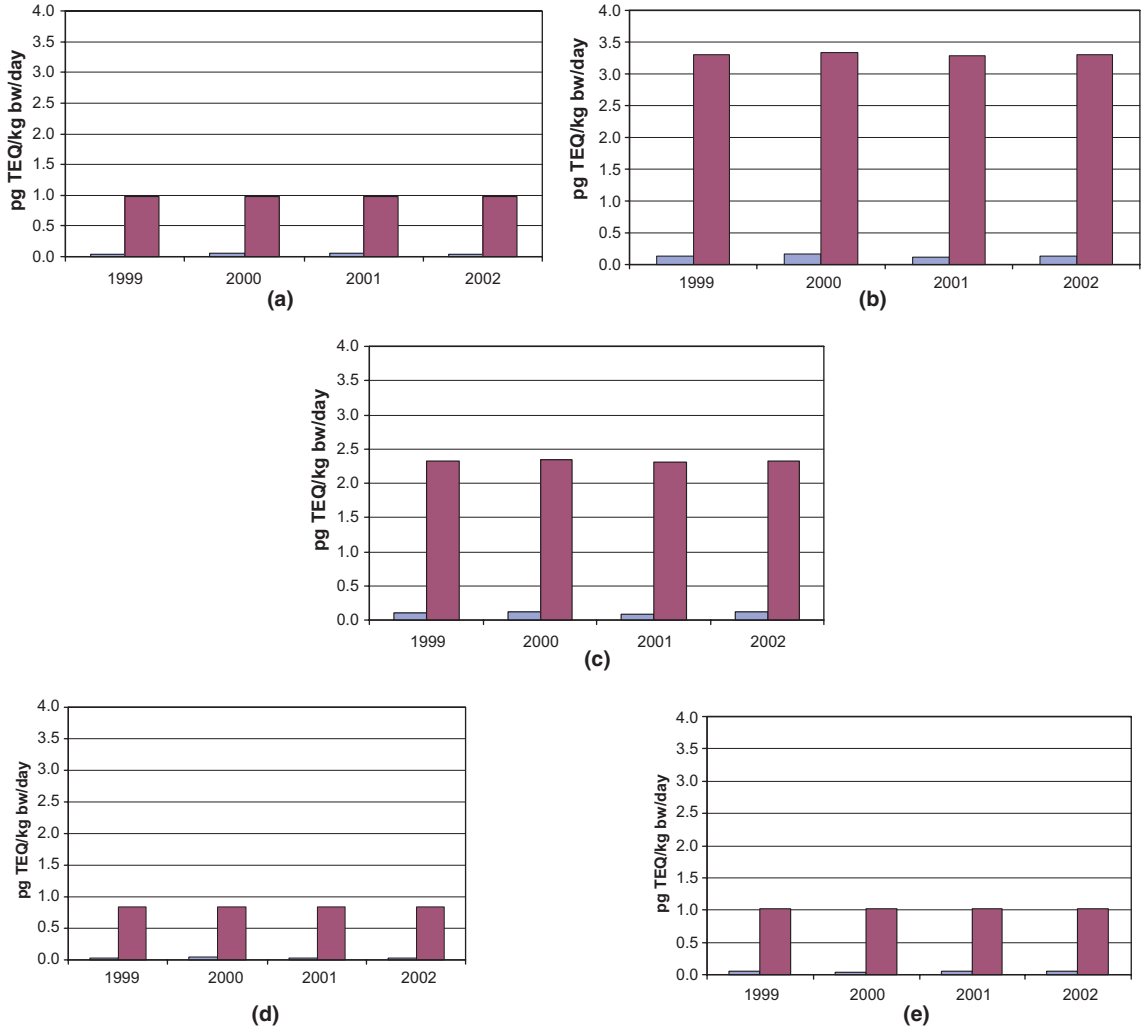

Fig. 2. Mean dioxin intake estimates during 1999–2002 for the total US population (a), 2 year old children (b), 6 year old children (c), 14–16 year old females (d), and 14–16 year old males (e), using a standardized limit of detection for all 4 years, evaluating only those {congener × food} combinations common to all 4 years, and assuming non-detects are equivalent to either zero (■) or one-half the limit of detection (■).

FDA. Meanwhile, the Joint FAO/WHO Expert Committee on Food Additives has proposed a tolerable monthly intake of 70 pg TEQ/kg BW as being protective of human health, based on developmental effects observed in male rats exposed to 2,3,7,8-TCDD during gestation (JECFA, 2001), which translates to a TDI of 2 pg TEQ/kg BW. The UK Food Standards Agency's Committee on Toxicity has recommended a TDI of 2 pg TEQ/kg BW (UKFSA/COT, 2001), as has the European Commission's Scientific Committee on Food (EC, 2001) (although their specific recommendation is 14 pg TEQ/kg BW/week), consistent with the FAO/WHO TDI.

TDI is defined by the World Health Organization as "an estimate of the amount of a substance in food or drinking-water, expressed on a body weight basis… that can be ingested over a lifetime without appreciable health risk" (WHO, 2003). In general, a TDI is established using a no-observed-adverse-effect level identified from the laboratory animal toxicity test that produced the most sensitive effect, which is adjusted downwards by dividing by uncertainty factors intended to produce a TDI protective of the most sensitive people. That is, exposure at or below the TDI is expected to produce no health risk, even in people who may be more susceptible to toxicity than others. Conversely, however, because of the way it is derived, exposure above the TDI does not imply that a health risk is expected. The upper-percentile estimates of dietary dioxin intake obtained by this analysis that exceed the TDI, therefore, do not indicate that, should an individual actually receive such an exposure, that individual would be at risk. This analysis does show that children are exposed to more dietary dioxin on a body-weight basis than teenagers or adults, but greater risk to children cannot be presumed. Because TDIs are established based on chronic lifetime exposure, comparing childhood dioxin exposure to a TDI assumes that diet, and thus dioxin exposure,

Table 2
Mean and upper-percentile dioxin intake estimates for the total US population and for children 2, 6, and 14–16 years of age assuming non-detects are equal to LOD/2 or to zero

	Intake (pg TEQ/kg bw/day)							
	Non-detects = LOD/2				Non-detects = 0			
	1999	2000	2001	2002	1999	2000	2001	2002
Total US population (N = 20607)								
Mean	1.20	0.62	0.56	0.38	0.04	0.07	0.33	0.24
90	2.84	1.72	1.31	0.93	0.12	0.22	0.85	0.61
95	3.80	2.35	1.70	1.21	0.20	0.33	1.17	0.82
99	7.00	4.07	2.64	1.93	0.48	0.67	1.96	1.30
Children 2 years (N = 1056)								
Mean	3.65	2.56	1.54	1.20	0.09	0.25	0.95	0.74
90	5.73	4.30	2.54	1.92	0.21	0.65	1.68	1.29
95	6.52	4.94	2.95	2.24	0.29	0.85	2.13	1.51
99	*8.85*	*6.10*	*4.63*	*3.20*	*0.74*	*7.42*	*3.39*	*2.45*
Children 6 years (N = 570)								
Mean	2.68	1.76	1.13	0.93	0.07	0.17	0.67	0.59
90	4.06	2.90	1.79	1.57	0.19	0.45	1.24	1.07
95	4.71	3.43	2.20	1.79	0.27	0.59	1.60	1.27
99	*5.79*	*4.89*	*3.27*	*2.17*	*0.56*	*0.85*	*2.51*	*1.61*
Males 14–16 years (N = 300)								
Mean	1.42	0.72	0.72	0.50	0.04	0.06	0.41	0.32
90	2.30	1.30	1.23	0.86	0.09	0.18	0.83	0.58
95	*2.91*	*1.59*	*1.53*	*1.04*	*0.13*	*0.26*	*1.15*	*0.74*
99	*4.78*	*2.57*	*2.74*	*1.47*	*0.38*	*0.34*	*1.91*	*1.09*
Females 14–16 years (N = 293)								
Mean	1.13	0.60	0.60	0.38	0.03	0.06	0.33	0.24
90	1.92	1.11	1.07	0.63	0.09	0.16	0.67	0.44
95	*2.22*	*1.31*	*1.25*	*0.71*	*0.11*	*0.25*	*0.85*	*0.57*
99	*2.85*	*2.08*	*1.70*	*0.90*	*0.36*	*0.34*	*1.32*	*0.71*

Mean dioxin intake estimates are based on consumption data from the 2004 Total Diet Study Per Capita Consumption and intake per body weight is based on an average body weight per age group. Upper percentiles of dioxin intake estimates are based on weighted 2-day average per capita consumption data from 1994 to 1998 CSFII. Each individual's intake per kilogram body weight is calculated using each individual's body weight as opposed to an average body weight per age group. Values in italics do not meet minimum sample size criteria for this type of analysis as established by the National Centers for Health Statistics.

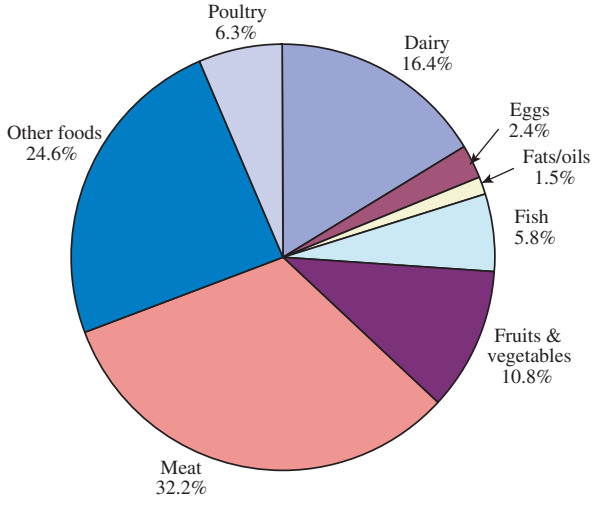

Fig. 3. Percent contribution of different food types to estimated dietary dioxin intakes (pg TEQ/kg body weight/day) for the total US population.

remain constant over a lifetime. That assumption overestimates potential risk, especially in view of decreasing environmental levels and body burdens. In addition, because of children's more efficient fecal clearance of TCDD (Kreuzer et al., 1997), the impact of increased intake rates on a body weight basis are much smaller in children than would be predicted by the ratio of intakes, potentially equating to larger margins of exposure than would be predicted solely on the basis of intake. What these results do indicate is that it is theoretically possible for some individuals to have exposures that exceed the TDI and that any risk management efforts under consideration might be targeted at contributors to those higher exposures.

Sources of uncertainty in this analysis result from extrapolating the foods included in the TDS to represent all foods consumed in the US and from applying a generic recipe for one food to represent all foods in a given food group. For example, recipes that call for added milk assume that everyone in the US uses whole milk, which is unlikely, but will lead to estimates of dioxin

concentrations based on whole milk. Because dioxin is associated with fat, it would be useful in the context of estimating exposure to know which recipe of the TDS food was analyzed by FDA. It may be that the dioxin concentration in a TDS food such as TDS # 137—white potato, baked with skin—is the result of butter or margarine added to the food that FDA analyzed. Thirty of the 272 translated CSFII recipes for this food contain fat as an ingredient. Because dioxin is associated with fat, uncertainty about the extent to which fat is included as an ingredient in a particular recipe contributes to uncertainty about exposure. The actual dioxin concentration of the group of CSFII foods used to derive the consumption values for each TDS food analyzed are thus likely to have been either over- or under-estimated, depending upon whether the TDS analysis included a CSFII food version containing fat and whether everyone uses the same recipe. Some TDS foods may be represented by CSFII food codes that may or may not include fat either as an added ingredient or as a component of a raw agricultural commodity that is an ingredient. A few details regarding food preparation methods relevant to fat content can be found online at http://www.cfsan.fda.gov/~lrd/dioxdata.html.

Another potential source of uncertainty is the accuracy of the TEFs used to estimate the relative potencies of the different congeners. Toyoshiba et al. (2004) have recently suggested that the use of a single relative potency factor (TEF) is not appropriate for comparison of the dose-response behavior of different dioxin-like congeners. In their study, changes in two P450 enzymes resulting from exposure to 2,3,7,8-TCDD, 3,3′,4,4′,5-pentachlorobiphenyl, 2,3,4,7,8-pentachlorodibenzofuran, and a TEF-equivalent mixture of the three agents was reported to produce different congener-specific dose-response curves and results for the mixture that were not dose-additive, suggesting that the use of a single TEF for every response to each dioxin-like congener is inappropriate. However, Gao et al. (2000) evaluated the inhibition of rat ovulation using the same three agents alone and in a similar mixture, producing parallel dose-response curves and effects that were dose-additive when combined. Viluksela et al. (1998) observed similar results in studies of responses to acute and subchronic administration of individual PCDD congeners and mixtures. Rozman et al. (in press) note that TEFs derived from acute toxicity studies are virtually identical to those derived from subchronic and chronic studies and to the results of Gao et al. (2000), concluding that the relative potencies of structurally closely related chemicals are the same for all effects caused by that class of compounds. The apparent difference in response between Toyoshiba et al. and Gao et al. may be the result of different approaches used to establish the TEFs; Toyoshiba et al. used TEFs established by the World Health Organization (WHO), which are based on scientific consensus, while Gao et al. derived relative potencies by determining each dose-response relationship (Rozman et al., in press). In contrast, Walker et al. (2005) found that the tumor responses observed in the NTP bioassays of TCDD, 4PeCDF, PCB-126, and a mixture of the three were consistent with WHO TEF-based additivity. In general, however, dose additivity appears to work best when relative potencies are calculated within the framework of a specific experiment and response evaluated and then used to predict the effect of the mixture, rather than relying on the WHO TEFs. The present analysis relies on FDA data derived using WHO TEFs so, to the extent that those values are incorrect, the results of this analysis are incorrect. However, the TDI to which these results are compared was also derived using WHO TEFs, so the comparison is not invalid. In any case, the TEQ approach generally tends to overestimate rather than underestimate the effects of mixtures compared to pure TCDD (Haws et al., 2004a,b) so, given that the TDI is derived based on the potency of pure TCDD, this uncertainty tends to be conservative (protective).

The results of this study compare favorably with the recent FDA analysis of dietary PCDD/PCDF exposure (South et al., 2004; USFDA, 2004), in which estimates of mean monthly TEQ intakes were obtained for various food groups, for total dietary intake, and for several age-sex categories by combining 2001 and 2002 dioxin data. Converting the mean monthly intake estimates from that study to daily intakes yields estimates very similar to those obtained in this study for the years 2001 and 2002, although FDA did not estimate upper-percentile exposures. The highest mean intakes on a body-weight basis occurred in 2-year-old children followed by 6-year-old children. FDA states that assuming non-detects are equal to LOD/2 overestimates PCDD/PCDF concentrations in foods known to be low in PCDD/PCDFs (e.g., grains, legumes, fruits, vegetables) and, because those foods are consumed in substantial quantities, assuming unrealistically elevated PCDD/PCDF concentrations can result in substantial overestimates of total dietary exposure. FDA concludes that exposure calculations based on LOD = 0 provide the most realistic estimates of actual PCDD/PCDF exposure (USFDA, 2004). In addition, both FDA's dioxin intake estimates and those from this analysis are based on CSFII's 2-day averaging approach to obtaining food consumption data; 30-day food consumption averages obtained using probabilistic methods are likely to yield lower consumption estimates (Tran et al., 2004).

The US EPA's recent exposure and human health reassessment of dioxins includes dietary intake estimates that exceed those determined by FDA and by this study because they are based on old consumption and contaminant level data (USEPA, 2004).

A recent USDA analysis of PCDD/PCDFs and coplanar PCBs in beef, poultry, and pork fat taken from slaughtering facilities across the US reported approximately 50–60% lower TEQs for poultry and pork compared to similar samples taken a decade earlier. Beef TEQs were 30% higher, but the increase was thought to reflect the high number of non-detects in the earlier survey (78%) and unlikely to be a real increase, although more analysis was recommended (Huwe et al., 2004). An earlier study found that pentachlorophenol-treated wood can be a primary source of PCDD/PCDF residues in cattle (Fries et al., 2002). Analysis of historical meat samples found that PCDD/PCDF concentrations began rising in the 1940s, peaked in the late 1960s, and have now returned to pre-1940s levels (Winters et al., 1998).

The absence of a downward trend in estimated dioxin intakes between 1999 and 2002, despite continued decreases in environmental levels of dioxins, is of interest. It is possible that a trend, should it be occurring, would not be observable during the relatively short time interval considered in this analysis. It is also possible that dioxin emissions from quantified, controllable, inventoried sources are no longer the most important contributors to dioxin levels in foods. For example, investigation of the few recent USDA beef samples with higher-than-expected dioxin levels revealed elevated dioxin levels in treated wood used in construction of feeding troughs and improper inclusion of incinerator waste in a feed additive. These cases indicate that dioxin levels in foods may now be most highly impacted by reservoir sources or unusual occurrences rather than the low level of ongoing emissions from controlled sources, which are now small compared to uncontrolled natural (e.g., forest fires) and anthropogenic (e.g., backyard barrel burning of trash) sources, and suggest that further risk management actions to reduce emissions from controlled sources beyond those already in place will have little or no additional impact on food levels.

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