DIOXINS AND DIOXIN-LIKE COMPOUNDS IN DOMESTIC MEATS

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Abstract
Persistent environmental pollutants including polychlorinated dibenzo-p-dioxins, dibenzofurans (PCDD/Fs), coplanar polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) were analyzed in 65 meat samples collected from supermarkets across the U.S. in 2001. The samples included hamburger, sirloin steaks, pork chops, bacon, and whole chickens from nine different cities. The average dioxin toxic equivalency (TEQ) for all the samples was 0.55 pg/g lipid, with pork having the lowest levels. The TEQ levels were similar to recent values reported in the U.S. and appear to be declining. The sums of PBDEs averaged 1.71 ng/g lipid and represent the first extensive sampling of domestic meats. The PBDE levels in pork and chicken were 4 and 10 times higher, respectively, than levels reported in Europe for these foods. The presence of a few outliers raised the average PBDE sums and indicated that isolated sources of contamination may exist that, if identified, could be removed from the U.S. animal production chain.

Introduction
Polychlorinated dibenzo-p-dioxins, dibenzofurans (PCDD/Fs), and biphenyls (PCBs) are ubiquitous, persistent, lipophilic pollutants. Polybrominated diphenyl ethers (PBDEs) are commonly-used additive flame retardants that are also highly lipophilic and structurally similar to the PCDD/Fs and PCBs. While PCDD/F levels are decreasing in the environment and PCBs are no longer produced, PBDEs continue to be produced and their levels appear to be increasing in the environment and in humans over the past 10-20 years (Darnerud et al., 2001; deWit, 2002). The acute and chronic toxicity of dioxin-like compounds is well documented (Schecter, 1994), and, although PBDEs do not appear to have any acute toxicity, some studies have shown developmental and neurological effects due to PBDE exposure (Darnerud et al., 2001). In order to better assess the risk from PBDEs, routes and magnitudes of exposure need to be investigated and characterized. Because human exposure to PCDD/Fs is almost entirely through the diet, it seems plausible that exposure to PBDEs may occur by this same route. In this study, we have analyzed typical domestic meats to update data on the current levels of PCDD/Fs and PCBs and to begin to define the levels of PBDEs found in these products.

Materials and Methods
Meat and poultry samples were purchased at large supermarkets in nine cities across the U.S. and shipped frozen to the USDA-ARS laboratory in Fargo, ND, for analysis. All samples were collected in 2001 and included bacon, hamburger, whole chickens, sirloin steaks, and pork chops from each location. The bacon and ground beef were analyzed whole; for the chickens, steaks, and chops, fat was trimmed and analyzed for 17 toxic PCDD/Fs and 3 coplanar PCBs, or 7 marker PBDEs in separate analyses. Homogenized bacon or fat trimmings (5 g) were spiked with 15 13C-labeled PCDD/Fs and 3 13C-labeled co-planar PCBs (#71, 126, and 169) or 7 13C-labeled PBDEs (#28, 47, 99, 153, 154, 183, and 209) (Wellington, Laboratories, Guelph, ON) and dissolved in methylene chloride. The sample was exchanged into hexane and applied to a Power Prep instrument (Fluid Management Systems, Waltham, MA) for automated dioxin cleanup on a jumbo and regular triphasic silica cartridge, a basic alumina cartridge, and a carbon cartridge. The carbon cartridge was omitted for PBDE cleanup. The hamburger (5g) was spiked with recovery surrogates, mixed with Celite, and extracted in an Automated Solvent Extractor ( Dionex, Sunnyvale, CA) using isopropanol:hexane:methylene chloride (35:30:35) at 125ºC and 1500 psi. The extract was purified on the Power Prep system as described above. The percent lipid in each sample was determined gravimetrically before application to the Power Prep system. The final fractions containing desired analytes were concentrated into 20uL of dodecane containing 13C-labeled internal standards and analyzed by isotope dilution methods on a high resolution GC/ high resolution MS instrument as previously described (EPA, 1994; Huwe et al., 2002).

A method blank or a method spike containing 17 PCDD/Fs and 3 PCBs or 42 PBDEs were run with each set of four samples. Because method blanks contained detectable levels of some analytes, the sample data were blank-subtracted. Limits of detection were calculated as 3x standard deviations of the blanks or low-level spikes. Values below the detection limit were treated as non-
detects and either set equal to zero or half the limit of detection. Dioxin toxic equivalencies (TEQs) were calculated from the 1998 World Health Organization toxicity factors (Van den Berg et al., 1998).

**Results/Discussion** Table 1 shows the results from the analysis of 65 meat samples for PCDD/Fs, co-planar PCBs, and PBDEs on a lipid-weight basis. Of the four types of meat included in the study, pork (including bacon) had the lowest average TEQ levels (0.22-0.28 ppt) followed by chicken (0.37 ppt), beef (0.68 ppt) and hamburger (1.4 ppt). This is similar to results of a recent USDA dioxin survey in U.S. meat and poultry which showed that beef had the highest TEQ levels compared to chickens and hogs (Huwe et al., 2004).

Unlike the PCDD/Fs and PCBs, beef appeared to have the lowest amounts of PBDEs, while chicken and pork had the highest on average (Table 1). Two explanations for this difference are 1) that the source of PBDE exposure for livestock is different than the source of dioxin exposure, which is generally thought to be from environmental fallout onto forages and feed, or 2) that ruminants, such as cattle, absorb less or metabolize and excrete more PBDEs than other livestock species resulting in lower body burdens. Little or no data exist on how PBDEs enter the food supply or how they are distributed or excreted by animals. Hites et al. (2004a) have shown that fish feed can be contaminated with PBDEs and may be related to elevated concentrations in farm-raised fish.

Inspection of the individual samples, the mean, and the median values for the PBDE data, showed that a few high samples were driving the average upward. In fact, the median value for pork fat was 60% lower than the mean. One pork and two chicken samples were found to have PBDE sums > 15 ng/g lipid. The two chickens were collected at the same time and location and were the same commercial brand. The pork sample was collected at a different location. The presence of these outliers suggests that isolated sources of PBDEs may exist and, if tracked down, may be removed from the food production chain.

Comparison of these market basket data with an earlier market basket study in the U.S. (Fiedler et al., 1997) showed that PCDD/F levels may have declined by 50% or more in retail pork and chicken from the mid 1990s to 2001. This downward trend was also observed in the recent USDA survey of beef, pork, and poultry (Huwe et al., 2004). Compared to European Union regulatory levels for dioxin TEQs (1 pg/g lipid for pork, 2 pg/g lipid for chicken, and 3 pg/g lipid for beef) all samples would be in compliance.

This market basket study represents the largest collection of data on PBDE levels in U.S. meat and poultry to date. Compared with data from Europe, beef samples were similar to meat products analyzed in Sweden (360 pg/g lipid) (Darnerud et al., 2001) and beef levels reported from Spain (290 pg/g lipid) (Bocio et al., 2003). The average PBDE sums in pork and bacon were up to 4-times higher than pork products from Spain (600 pg/g lipid) (Bocio et al., 2003); however, the median values were similar or lower than the Spanish average. Total PBDEs in U.S. chickens averaged 10-times lower than pork samples.
The contribution of individual PBDEs to the total for each meat type is shown in Figure 1, along with a typical pattern from a penta-BDE commercial formulation (DE-71). For all meat samples, BDE-47 and 99 were the largest contributors to the PBDE sum, and the ratio of BDE-47/99 was generally less than unity (average=0.78, range 0.3-3.0). This pattern strongly resembles the penta-BDE formulation shown in Figure 1 which has a BDE-47/99 ratio of 0.4. A similar congener pattern has been seen in other animal products from Canada (Ryan and Patry, 2001), Japan (Ohta et al., 2002), and the U.S. (Huwe et al., 2002; Schecter et al., 2004). In contrast, the congener patterns in fish and seafood (Ohta et al., 2002; Schecter et al., 2004; Hites et al., 2004a) and human samples (Ohta et al., 2002; Ryan and Patry, 2001; Hites, 2004b) generally show a ratio of BDE-47/99 greater than 2.

For populations who are not largely fish eaters, this implies that sources other than meat and poultry may be contributing to their PBDE body burdens or that PBDE congener pattern changes occur in humans via selective uptake from foods or via metabolism.

Conclusions Dioxin TEQs were found to be low in all meat types analyzed, especially in pork, and appear to be on the decline. The average PBDE concentrations present in certain U.S. meats were high compared to European data but were strongly influenced by a few outlying values. The high PBDE levels may reflect the larger amount of penta-BDE being used in North America (www.bsef.com). How PBDE contaminants enter the food chain is unknown, but may include animal feeds (e.g. fish meal), environmental inputs (housing or litter), or even food packaging materials. More data are needed to investigate the inputs of PBDEs to the food supply.

References


EPA Method 1613A, 1994. Tetra- through octa-chlorinated dioxins and furans by isotope dilution HRGC/HRMS.


