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Dietary Exposures to Food Contaminants across the United States[1]

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Food consumption is an important route of human exposure to pesticides and industrial pollutants. Average dietary exposures to 37 pollutants were calculated for the whole United States population and for children under age 12 years by combining contaminant data with food consumption data and summing across food types. Pollutant exposures were compared to benchmark concentrations, which are based on standard toxicological references, for cancer and noncancer health effects. Average food ingestion exposures for the whole population exceeded benchmark concentrations for arsenic, chlordane, DDT, dieldrin, dioxins, and polychlorinated biphenyls, when nondetects were assumed to be equal to zero. For each of these pollutants, exposure through fish consumption accounts for a large percentage of food exposures. Exposure data for childhood age groups indicated that benchmark concentrations for the six identified pollutants are exceeded by the time age 12 years is reached. The methods used in this analysis could underestimate risks from childhood exposure, as children have a longer time to develop tumors and they may be more susceptible to carcinogens; therefore, there may be several additional contaminants of concern. In addition, several additional pollutants exceeded benchmark levels when nondetects were assumed to be equal to one half the detection limit. Uncertainties in exposure levels may be large, primarily because of numerous samples with contaminant levels below detection limits. © 2000 Academic Press

Key Words: **consumption; contaminant; cumulative; data base; exposure; food.**

INTRODUCTION

Food consumption represents an important pathway for exposure to contaminants from a variety of sources, including pesticide application and contamination of water from industrial sources. Recent studies have indicated that exposures to contaminants in food may pose a public health risk (National Research Council, 1993; MacIntosh *et al*., 1996). For example, MacIntosh *et al*. (1996) found that some portion of the adult population may be exposed to individual contaminants in food at concentrations above thresholds of concern. Reports from the National Research Council of the National Academy of Sciences (NRC) and the Environmental Working Group have also found that pesticide exposures to children could be high enough to cause immediate adverse health outcomes (National Research Council, 1993; Wiles *et al*., 1998).

These reports have focused either on a small group of contaminants and specific types of foods, such as pesticides in fruits and vegetables (National Research Council, 1993; Wiles *et al*., 1998), or on a subset of the United States population, such as adults (MacIntosh *et al*., 1996) or children (National Research Council, 1993; Wiles *et al*., 1998). A more comprehensive assessment of food contaminant exposures, which would include both pesticides and industrial contaminants, as well as both children and adults, will help identify which populations are most at risk from contaminant exposure in foods and which contaminants have the greatest public health significance.

To better assess the national distribution of exposures to a broad array of contaminants in food, data were collected on 37 contaminants in foods and combined with estimates of consumption from dietary profiles for demographic groups across the

[1] The views expressed in this paper are those of the authors and do not necessarily reflect those of the USEPA.

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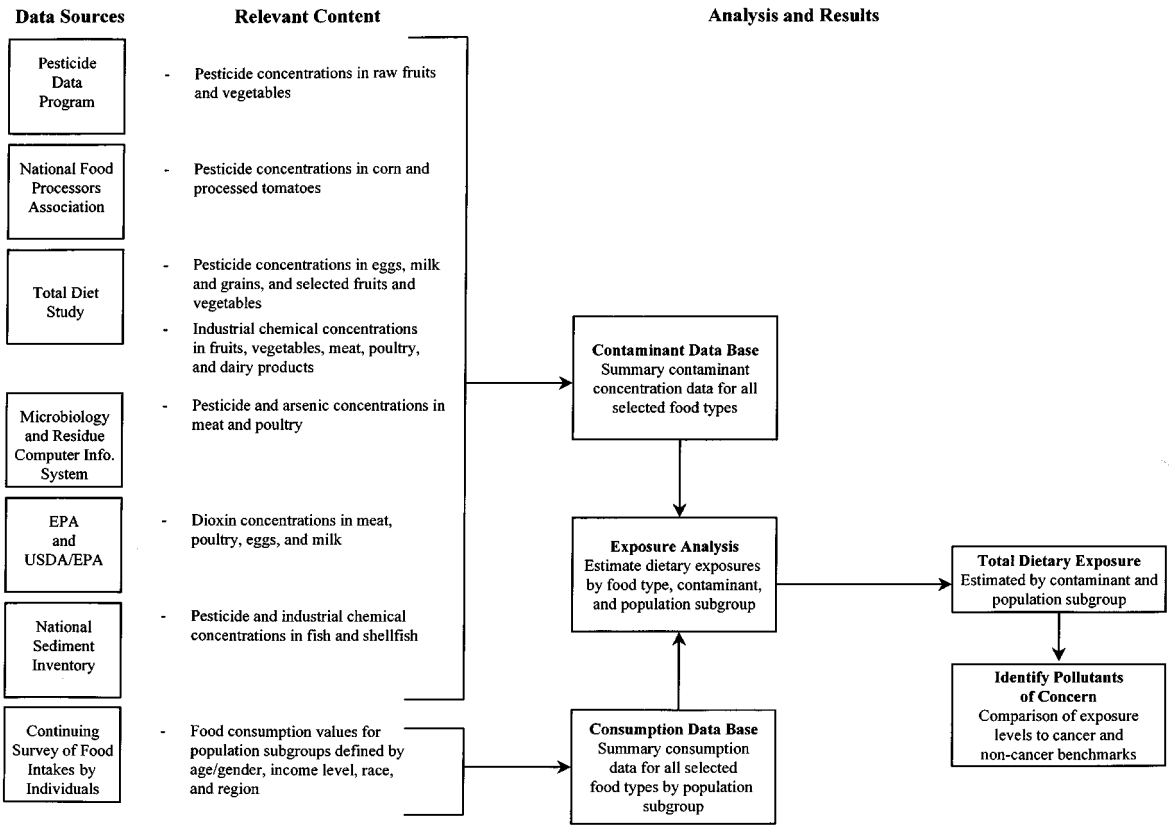

FIG. 1. Overview of analytical methods for food contamination exposures.

United States. The analysis considers exposures to individual contaminants from multiple foods and multiple contaminants in combination. This information provides a screening-level assessment for identifying contaminants of highest concern. This article presents an overview of the analysis of exposures from ingestion of food contaminants, including a description of the contaminants, foods, and population subgroups analyzed, the sources of data used, the analytical approach taken, and estimates of average food ingestion exposures for the whole population and for children under age 12 years.

METHODS

This analysis estimates dietary exposures to certain pesticides and industrial chemicals through selected foods considered representative of the diet of the American population. The analysis included (1) creating a food consumption database that provides information on consumption patterns by demographic characteristics, (2) creating a food contaminant database by obtaining and compiling contaminant data, (3) combining contaminant and consumption information to estimate exposures from individual food types, (4) estimating total dietary exposures by summing across all food types, and (5) comparing exposures to benchmark concentrations to determine potential public health impacts. Figure 1 provides an overview of each of these steps.

Foods selected for the analysis were those that comprised at least 1% of the average diet either for the entire United States population or for certain age groups based on the 1977–1978 Nationwide Food Consumption Survey, posed the greatest cancer risk from contaminants according to the National Research Council's "Regulating Pesticides in Food—The Delaney Paradox" (National Research Council, 1987), or were evaluated by the U.S. Department of Agriculture (USDA)[2] in its Pesticide Data Program

[2] Abbreviations used: CSFII, Continuing Survey of Food Intakes by Individuals; DDT, dichlorodiphenyltrichloroethane; FDA, United States Food and Drug Administration; MARCIS, Microbiology and Residue Computer Information System; NFPA, National Food Processors Association; NSI, National Sediment Inventory; PCB, polychlorinated biphenyls; PDP, Pesticide Data Program; RfD, Reference Dose; TAS, Technical Assessment Systems, Inc.; TDS, Total Diet Study; USDA, United States Department of Agriculture; USEPA, United States Environmental Protection Agency.

(PDP) (U.S. Department of Agriculture, 1994). Due to a lack of contaminant data, sugar beets, cane sugar, coconut oil, soybean oil, and soybeans were dropped from the analysis, even though they met the first criterion listed above. The foods selected for the analysis are listed in Table 1.

The analysis estimates the dietary intake of 37 contaminants, including 30 pesticides and 7 industrial chemicals. These chemicals, listed in Table 2, are considered to be most important for the analysis of contaminant exposure through food ingestion and have available data. Note that some of the contaminants have been completely banned from use in the United States but, because of their persistence, continue to have measurable residues in the food supply. Selection of these contaminants was based upon the following factors and primary sources: (1) toxicity of the contaminant, based on toxicity

TABLE 1
Data Sources Used for the Food Contamination Database

Food	Pesticides	Industrial chemicals	Dioxin
Fruits, vegetables and grains			
Apples, processed	TDS	TDS	
Apples, raw	PDP/TDS	TDS	
Bananas	PDP/TDS	TDS	
Broccoli	PDP	TDS	
Cabbage	TDS	TDS	
Carrots, processed	PDP/TDS	TDS	
Carrots, raw	PDP/TDS	TDS	
Celery, processed	PDP/TDS	TDS	
Celery, raw	PDP/TDS	TDS	
Corn	NFPA/TDS	TDS	
Grapefruit	PDP/TDS	TDS	
Grapes	PDP/TDS	TDS	
Green beans	PDP/TDS	TDS	
Lettuce	PDP/TDS	TDS	
Oats	TDS/NFPA	TDS	
Orange juice	TDS/NFPA	TDS	
Oranges	PDP/NFPA	TDS	
Peaches	PDP/TDS	TDS	
Pears	TDS/NFPA	TDS	
Peas, green	TDS/NFPA	TDS	
Potatoes, white	PDP/TDS	TDS	
Rice	TDS	TDS	
Spinach	TDS	TDS	
Tomatoes, processed	NFPA/TDS	TDS	
Tomatoes, raw	TDS	TDS	
Wheat flour	TDS/NFPA	TDS	
Meat and Poultry			
Beef	MARCIS/TDS	TDS	USEPA/USDA
Chicken	MARCIS/TDS	TDS	USEPA
Pork	MARCIS/TDS	TDS	USEPA
Fish			
Fish, freshwater	NSI	NSI	NSI
Fish, saltwater	NSI	NSI	NSI
Shellfish	NSI	NSI	NSI
Dairy and Egg Products			
Milk	TDS	TDS	USEPA
Eggs	TDS	TDS	USEPA

Note. For pesticides, the sample data were derived primarily from the first database listed. Data were taken from the second database for those pesticides not included in the first. Sources are as follows: TDS, Total Diet Study, 1988–1993; PDP, Pesticide Data Program, 1992–1993; MARCIS, Microbiology and Residue Computer Information System, 1990–1995; NSI, National Sediment Inventory, 1988–1993; NFPA, National Food Processors Association, 1987–1992; USEPA/USDA, USEPA/USDA joint study on dioxins in beef, 1989–1991; USEPA, USEPA Dioxin Report, 1989–1991.

TABLE 2
Contaminants Included in the Food Analysis

Pesticides		Industrial chemicals
2,4-D	*Dieldrin*	Arsenic
Acephate	Endosulfans	Cadmium
Alachlor	Heptachlor[b]	Dioxins
Atrazine	*Hexachlorobenzene*	Manganese
Azinphos-methyl	Imazalil	Mercury
Captan	Iprodione	PCBs
Carbaryl	Lindane	Selenium
Chlordane	Malathion	
Chlorothalonil	Methamidophos	
Chloropropham	Methoxychlor	
Chlorpyrifos	*O*-Phenylphenol	
Cyanazine	Permethrin	
DDT[a]	Simazine	
Diazinon	Thiabendazole	
Diphenylamine	*Toxaphene*	

Note. Contaminants in italics have been banned for all uses.

[a] Exposures to DDT assessed in this study represent the sum of the isomers *o*,*p*′-DDT, *p*,*p*′-DDT, *o*,*p*′-DDE, *p*,*p*′-DDE, *o*,*p*′-DDD and *p*,*p*′-DDD.

[b] Heptachlor is almost completely banned and may be used only to control fire ants inside buried, pad-mounted electrical transformers and in underground cable television and telephone cable boxes.

information in USEPA's Integrated Risk Information System (IRIS) and information on chemical carcinogenicity from USEPA's Office of Prevention, Pesticides and Toxic Substances and a National Research Council study (National Research Council, 1987); (2) frequency of detection in foods as determined by the Food and Drug Administration's (FDA) Total Diet Study (U.S. Food and Drug Administration, 1994), USDA's National Residue Program, and USEPA's National Sediment Inventory (U.S. Environmental Protection Agency, 1992); (3) prevalence in the environment, based on information on the quantity of chemicals applied to crops annually from a National Research Council study (National Research Council, 1987); and (4) availability of contaminant data.

Other sources, including the National Human Exposure Survey (NHEXAS) (Technical Assessment Systems, 1994) and USDA's Pesticide Data Program (U.S. Department of Agriculture, 1995a), were also used in evaluating contaminants for inclusion in the analysis.

Food Consumption Database

Food consumption levels for the foods included in the analysis were compiled from USDA's Continuing Survey of Food Intakes by Individuals (CSFII) (Technical Assessment Systems, 1995). From 1989 to 1991, the CSFII collected food consumption data annually from 12,000 individuals considered representative of the United States population over a nonconsecutive 3-day sampling period. Survey participants recorded the weight of each food eaten at each meal during the sampling period. Consumption was reported in a standardized manner using body weights (grams of food ingested per kilogram of body weight). Average daily consumption values were available for consumers only (individuals that consumed the food at least once during the sampling period) and for all individuals.

Food consumption data were summarized for nine populations, stratified by age and gender: children less than 1 year of age, children 1 to 5 years of age, males and females 6 to 11 years of age, males and females 12 to 19 years of age, males and females 20 to 64 years of age, and adults greater than 64 years of age. Food consumption values were calculated for each of the nine population subgroups and for each of the 34 food types included in the analysis, as long as there were more than three individuals in the subpopulation who consumed the food. Means and Standard deviation for each subpopulation were calculated by averaging 3-day individual averages to represent population averages and variability. This analysis calculated average food ingestion exposures using average daily consumption for all individuals and for children less than 12 years of age.

CSFII data include consumed quantities of prepared foods such as pizza and spaghetti sauce, which were converted into quantities of raw agricultural commodities. Standardized recipes were used to calculate the quantity of the raw agricultural commodity in each prepared food (Technical Assessment Systems, 1995). Total consumption of each raw commodity was estimated by summing the amount in all foods.

Food Contamination Database

Residue data for the selected contaminants were obtained from various food contaminant data sources, including the USDA Pesticide Data Program, National Food Processors Association (NFPA) (Chemical Information Services, 1995), U.S. Food and Drug Administration Total Diet Study (TDS), Microbiology and Residue Computer Information System (MARCIS) (U.S. Department of Agriculture, 1995b), USEPA Dioxin Report (U.S. Environmental Protection Agency, 1994), USEPA/USDA Dioxin in Beef Study (Winters *et al*., 1994), and the National Sediment Inventory (NSI). Of the seven food

contaminant data sources used, the NSI, MARCIS, and USEPA Dioxin Reports used raw samples, whereas the others used prepared and/or cooked samples.

Years selected for each data source were based on availability as well as sample size (i.e., more years of data were used in databases in which the number of samples per year is small), but were generally representative of the early 1990s. As shown in Table 1, residue data for most vegetables were taken from the PDP database. If PDP data were not available for a particular food type, the NFPA data were used. If NFPA data also were not available, TDS data were used. The MARCIS database supplied most of the residue data for beef, chicken, and pork, while the USEPA/USDA Dioxin in Beef Study and the USEPA Dioxin Report provided supplemental dioxin data on beef, pork, chicken, milk, and eggs. TDS data were used for industrial chemicals in all food types except fish. Residue data for fish were obtained from the National Sediment Inventory database.

USDA Pesticide Data Program. The USDA Pesticide Data Program started in 1991 as a monitoring program to collect residue data on selected fruits and vegetables. Fresh fruits and vegetables evaluated by PDP are collected as close to the consumer as possible and prepared as for consumption, i.e., washed and peeled. The commodities are selected to accurately represent national distribution levels and pesticide use for a specific crop by growing region and season. Of the 37 selected industrial chemicals and pesticides, the PDP database contains information on 23.

PDP data from 1992 and 1993 were used in this analysis. In 1992, the USDA collected PDP samples from six states representing 40% of the United States population, and in 1993, the USDA collected samples from nine states representing 50% of the population.

The National Food Processors Association. The NFPA monitors the illegal or unnecessary presence of pesticide residues in processed food. The NFPA database consists of residue data, from both random and targeted sampling, of raw foods purchased specifically for processing and processed foods, such as canned and frozen foods. The database contains residue data on 28 of the 37 contaminants. The database has small sample sizes and less sensitive detection techniques than PDP, resulting in higher detection limits. Data were obtained from the years 1987–1992.

FDA Total Diet Study. The FDA's Total Diet Study collects residue data for approximately 300 pesticides, radionuclides, and industrial chemicals in 261 foods that represent 3500 typically consumed foods. Sampling procedures consist of purchasing the 261 foods in three cities in each of four regions of the country. The samples from the three cities within each region are combined into one "market basket." Samples are collected four times per year. The foods sampled include processed foods (bottled, canned, and frozen), fresh foods including fruits and vegetables, baby foods, dairy products, fresh meats, cereals, peanut butter, and prepared foods such as pizza. The three samples of like foods are combined into one sample, prepared as for consumption, and analyzed for contaminants. Data from the TDS database, which includes 28 of the 37 contaminants used in this analysis, were from 1988 to 1993. An important limitation of the study is its small sample sizes.

The Microbiology and Residue Computer Information System. MARCIS is a USDA-sponsored monitoring program, the purpose of which is to ensure that USDA-inspected products are safe for human consumption. MARCIS contains data from tests on meat, poultry, and egg products for any of 100 compounds in eight classes of animal drugs and pesticides, including nine contaminants in this study: chlordane, chlorpyrifos, DDT, dieldrin, heptachlor, hexachlorobenzene, lindane, methoxychlor, and PCBs. Contaminant testing occurs in raw fat samples. Thus, the residue data do not reflect contaminant levels in cooked or processed foods, as cooking is likely to decrease the percentage of fat and thus decrease concentrations of lipophilic contaminants. Processing and cooking of foods may also break down contaminants and reduce concentrations. Data from the years 1990 to 1995 were used in this analysis.

USEPA Dioxin Report and USEPA/USDA Dioxin in Beef Study. The USEPA Dioxin Report presents average dioxin levels in United States meat and dairy products, including pork, chicken, milk, and eggs, based on data from the years 1989 to 1991. In addition, the 1994 USEPA/USDA Dioxin in Beef Study analyzed the concentration of dioxins in the back fat of United States beef animals using a statistically based sampling survey that accounted for 99.9% of all beef animals slaughtered in the United States. Data for all foods are presented as dioxin toxic equivalents, which were calculated using the international toxic equivalency factors for chlorinated dibenzo-*p*-dioxin (CDDs) and chlorinated dibenzofurans (CDFs). Tests were performed on raw foods, resulting in possible overestimation of total

dioxin exposure. An additional limitation of the USEPA Dioxin Report is the small sample sizes that were used to develop average contaminant levels.

National Sediment Inventory. The National Sediment Inventory was implemented in the early 1980s to monitor 96 compounds, including PCBs, endosulfans, dioxins, and DDT in fish and shellfish. The National Sediment Inventory compiled information on fish tissue contaminants from several data sources, including STORET (Storage and Retrieval for Water Quality Data), EMAP (Environmental Monitoring and Assessment Program), ODES (Ocean Data Evaluation System), and DMTS (Dredged Material Tracking System). Sampling data were collected on a national and a regional scale, in both freshwater and saltwater bodies. Some NSI sampling procedures, such as targeted sampling in areas thought to have high contamination levels and sampling in raw fish fillets, may overestimate contaminant concentrations and therefore exposure levels. Data used were for the years 1988 to 1993.

Estimating Exposure

The analysis estimates exposure to specific contaminants via individual foods by combining data on contaminant concentrations in foods with data on consumption of those foods. For each contaminant and food evaluated, the following equation was used to calculate average exposure levels for a particular population subgroup:

$$\text{Average Daily Exposure } (\mu\text{g/kg body weight} \times \text{day})$$

$$= \text{Consumption (g/kg body weight} \times \text{day})$$

$$\times \text{Contaminant Concentration } (\mu\text{g/g}).$$

Having calculated exposures from each food, the estimates for each contaminant were summed across food types, providing estimates of each population subgroup's total average daily exposure to each contaminant through the food pathway. There were a number of nondetects in the contaminant data. To account for the possibility that there were concentrations below the detection limit, we used two alternative assumptions. We first assumed that actual concentrations for nondetect samples were equal to half of the detection limit. However, this would overestimate the concentrations if there were truly no contamination of the sampled foods. Therefore, we also calculated exposures with the alternate assumption that actual concentrations for all nondetect samples were equal to zero. These two assumptions provide a range of exposure estimates for each contaminant with nondetects. Separate exposure analyses were conducted for children (less than 12 years old) and for the whole population.

Comparison to Benchmark Concentrations

To screen for the potential public health significance of estimated exposures, exposure values were compared to benchmark concentrations for each contaminant. A benchmark concentration represents a daily concentration below which there is a high probability of no adverse health effect. This is different than a benchmark dose, which is a statistically derived value used in setting a Reference Dose for noncancer health effects. The benchmark concentrations for carcinogenic effects were derived using USEPA cancer slope factors and represent exposure concentrations at which lifetime cancer risk is one in one million. This level is defined as a public health protective concentration in the Congressional House Report to the Food Quality Protection Act of 1996 (104th Congress, 1996). In addition, the one in a million cancer risk has been used in other regulatory programs, such as those for air toxics, as a de minimus concentration and thus an appropriate level for screening (1990, Clean Air Act Amendments; Caldwell *et al*., 1998). The benchmark concentrations for noncarcinogenic effects are USEPA Reference Doses. The Reference Dose is an estimate, with an uncertainty spanning perhaps an order of magnitude, of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime (U.S. Environmental Protection Agency, 1990).

Of the 37 contaminants studied, 20 have available cancer benchmark concentrations, 34 have available noncancer benchmarks, and 17 have both cancer and noncancer benchmark concentration values (Table 3).

Exposure levels were compared to benchmarks by calculating hazard ratios. Hazard ratios are calculated by dividing the average daily exposures by the benchmark concentrations. Hazard ratios greater than one indicate that the average exposure level exceeds the benchmark concentration.

Calculation of cancer hazard ratios for children's exposures involved a step in which childhood exposures were converted to equivalent whole-life exposures. A lifetime average daily dose (LADD) from childhood exposure was calculated by summing together the estimated intake levels over the first 12

TABLE 3

National Average Exposures and Benchmark Concentrations for Contaminants Included in the Food Ingestion Analysis[a]

Chemical name	Number of samples (and detects)	Exposure per capita nondetects = 0 (μg/kg × day)	Exposure per capita nondetects = 0.5 DL (μg/kg × day)	Oral RfD (μg/kg × day)	Cancer benchmark concentration[b] (μg/kg × day)
2,4 D	3,965(88)	0.001	0.009	10	—
Acephate	6,366(961)	0.02	0.04	4	0.115
Alachlor	169(0)	0	**0.02**	10	0.0125[c]
Arsenic[e]	1,421(136)	**0.2**	**0.2**	0.3	0.001
Atrazine	169(0)	0	**0.02**	35	0.0045[c]
Azinphos-methyl	9,548(581)	0.02	0.1	1.5[d]	—
Cadmium[f]	1,577(751)	0.2	0.2	1	—
Captan	6,901(573)	0.03	0.06	130	0.29
Carbaryl	8,850(388)	0.02	**0.06**	100	0.044
Chlordane	26,598(1340)	**0.01**	**0.07**	0.06	0.001
Chlorpyrifos	35,591(510)	0.8	0.8	3	—
Chlorothalonil	8,281(1079)	0.007	0.03	15	0.09[c]
Chloropropham	3,864(744)	0.004	0.04	200	—
Cyanazine	76(0)	0	**0.03**	2[c]	0.001[c]
DDT[g]	26,098(1927)	**0.04**	**0.1**	0.5	0.003
Diazinon	11,709(535)	0.003	0.02	0.9[c]	—
Dieldrin	26,608(1155)	**0.003**	**0.03**	0.05	0.0000625
Dioxins[h]	142(93)	**1e-06**	**2e-06**	—	6.7e-09[c]
Diphenylamine	2,962(347)	0.1	0.2	25	—
Endosulfans[i]	12,933(1130)	0.02	0.05	6	—
Heptachlor	18,377(84)	0.0001	**0.03**	0.5	0.00022
Hexachlorobenzene[j]	27,142(104)	0.0001	**0.04**	0.8	0.001
Imazalil	4,317(831)	0.009	0.05	13	—
Iprodione	10,592(1441)	0.09	0.1	40	—
Lindane[j]	27,614(83)	0.001	**0.02**	0.3	0.00077[c]
Malathion	5,986(92)	0.04	0.07	20	—
Manganese[k]	980(886)	20	20	140	—
Mercury[l]	6,057(5047)	0.04	0.08	0.3[c]	—
Methamidophos	8,820(578)	0.01	0.02	0.05	—
Methoxychlor	2,9180(93)	0.005	0.07	5	—
O-phenylphenol	2,095(12)	0.0002	0.01	—	0.515[c]
PCBs[m]	27,626(1523)	0.07	**0.1**	*0.*02	0.00013
Permethrin	6,591(1136)	0.03	**0.07**	50	0.056
Selenium[n]	1,081(473)	1	1	5	—
Simazine	231(1)	0.0002	**0.03**	5	0.008[c]
Thiabendazole	10,938(1749)	0.2	0.3	100[d]	—
Toxaphene	4,009(13)	0.0004	**0.2**	—	0.001

Note. Contaminants in italics have been cancelled for all uses. Source: Oral RfDs and CSFs obtained primarily from USEPA's Integrated Risk Information System (4th Quarter, 1995) except where indicated otherwise.

[a] Numbers in boldface represent whole-population average estimated exposures that exceed the benchmark concentrations for carcinogenic effects or for both carcinogenic and noncarcinogenic effects.

[b] Benchmark concentration for carcinogenic effects equals 10^{-6} divided by the cancer slope factor and represents the exposure concentration at which lifetime cancer risk is one in one million.

[c] Toxicity values obtained from USEPA's Health Effects Assessment Summary Table, 1994.

[d] Toxicity values obtained from USEPA's Office of Pesticide Programs RfD Tracking Report, 4/14/95.

[e] Listed as Arsenic (inorganic).

[f] Listed as Cadmium (food).

[g] Listed as DDT (*p*,*p*′-Dichlorodiphenyltricloroethane).

[h] Listed as Tetrachlorodibenzo-*p*-dioxin, 2,3,7,8 (TCDD).

[i] CAS 115-29-7.

[j] Listed as Hexachlorocyclohexane, gamma.

[k] Listed as Manganese (food).

[l] Listed as Mercury (inorganic).

[m] PCBs (CAS 1336-36-3) used for Slope Factor, Aroclor 1254 (CAS 11097-69-1) used for Reference Dose.

[n] Listed as Selenium (and compounds).

years of life and dividing the result by 70 years. This approach assumes zero exposure from ages 12 to 70 to convert childhood exposures into lifetime terms for direct comparison with the cancer benchmarks that are also based on lifetime exposure.

RESULTS

Analysis of National Average Contaminant Exposures

Table 3 presents national average daily food ingestion exposures to the 37 contaminants included in the analysis. These exposures are estimated on a per capita basis and therefore are derived from consumption values for all individuals surveyed, including those who did not consume particular foods during the survey period. For each contaminant, exposures are presented separately assuming that nondetects in the food contamination database are equal to zero and one half of the detection limit. Values in bold face in Table 3 indicate exposures that exceed either cancer or noncancer benchmark concentrations.

Figures 2 and 3 present hazard ratios for carcinogenic and noncarcinogenic effects, respectively, under two assumptions: (1) nondetects are equal to zero and (2) nondetects are equal to onehalf of the detection limit. Figure 2 indicates that average exposures to six contaminants exceed benchmark concentrations for their carcinogenic effects when nondetects are assumed to be zero. These contaminants are arsenic, chlordane, DDT, dieldrin, dioxins, and PCBs. Figure 3 shows that, assuming nondetects are equal to zero, the only contaminant that exceeds benchmark concentrations for noncarcinogenic effects is PCBs.

Figures 2 and 3 and Table 3 also show that average exposure estimates are highly dependent upon assumptions about the value of nondetects. When nondetects are assigned a value of one half of the detection limit, exposure values are 2 times as high in most cases and in several cases 10 times as high as when they are assigned a value of zero. However, changing the nondetect value from zero to one half of the detection limit does not significantly change the exposure levels for eight of the contaminants—arsenic, cadmium, chlorpyrifos, iprodione, manganese, PCBs, selenium, and thiabendazole.

The value assigned to nondetects also has an effect on the number of contaminants exceeding benchmark concentrations. When nondetects are assigned a value of zero, 6 of the 37 contaminants exceed benchmark concentrations. When nondetects are assigned values of one half of the detection limit, however, the number of contaminants exceeding benchmark concentrations is 16. Five of the additional 10 contaminants exceeding benchmark concentrations have very small samples sizes and/or a small number of samples with detected contamination. It is unlikely that average exposure levels to

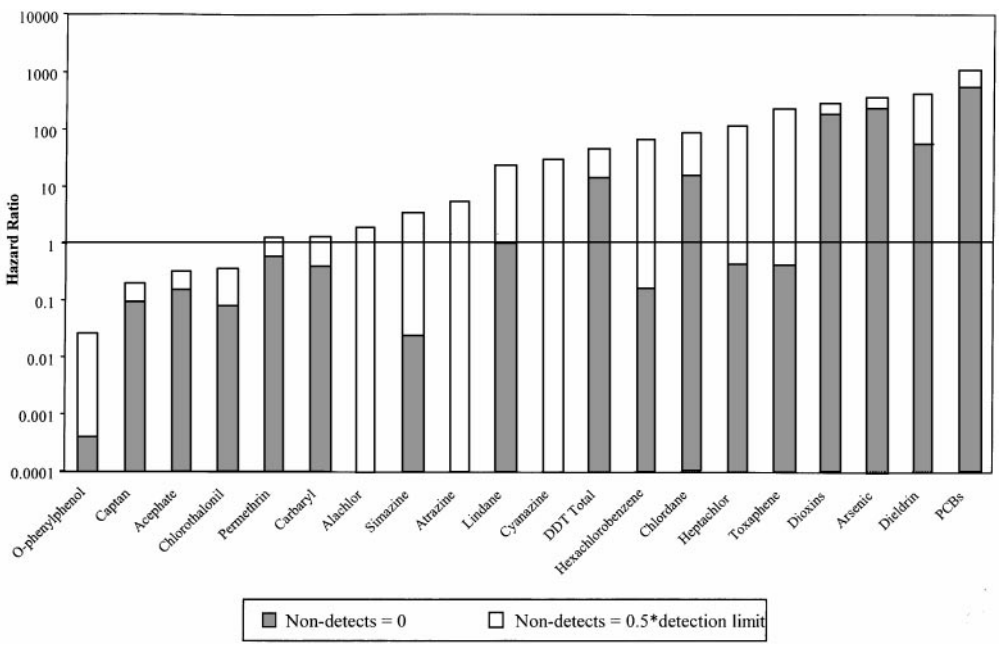

FIG. 2. Cancer hazard ratios for average national exposure (per capita).

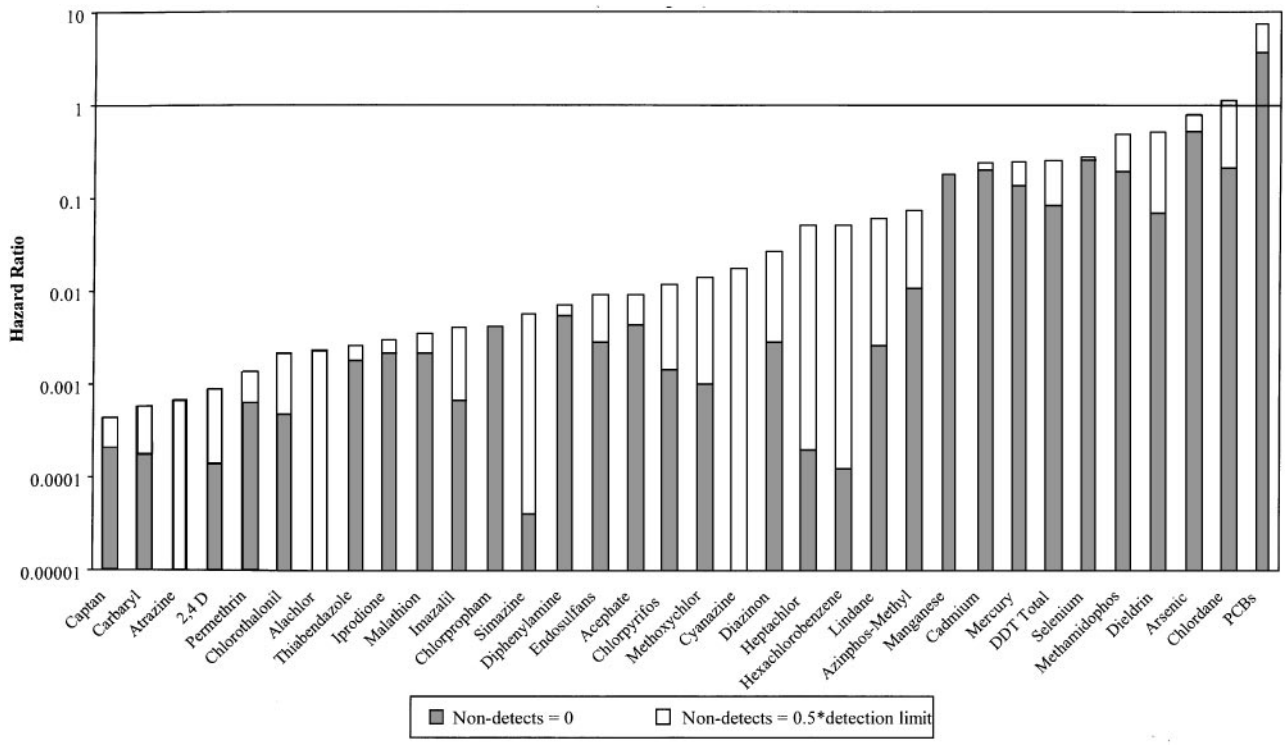

FIG. 3. Noncancer hazard ratios for average national exposure (per capita).

these contaminants—alachlor, atrazine, cyanazine, simazine, and toxaphene—actually exceed benchmark concentrations, since less than 1% of the samples for each of these contaminants are above detection limits.

These results reflect a large number of nondetect samples in the food contamination database. The percentage of detects for different contaminants ranges from 0% for alachlor, atrazine, and cyanazine to over 90% for manganese, though many contaminants are detected in less than half the samples.

Contribution of Individual Foods to National Exposure. Exposures to contaminants in individual foods were analyzed to identify those foods with the largest contributions to average national contaminant exposures. To avoid highlighting exposures that exceed benchmark concentrations solely due to positive values assigned to nondetects, the analysis of the contribution of individual foods focused on the scenario under which nondetects were assumed to equal zero. As indicated by Figs. 2 and 3, average national exposure under this scenario exceeds benchmark concentrations for the following six contaminants: arsenic, chlordane, DDT, dieldrin, dioxins, and PCBs.

Figure 4 presents the contributions of individual foods that contribute more than 5% to total exposure to the six contaminants, assuming that nondetects are equal to zero. As illustrated in Fig. 4, exposure levels from fish account for a large fraction of total exposure for each of the six contaminants analyzed. Exposures from saltwater fish contribute the largest percentage to total exposure of all foods analyzed for three contaminants (chlordane, 67%; dioxins, 64%; PCBs, 60%). Exposures from freshwater fish contribute the largest percentage for two contaminants (DDT, 75% dieldrin, 52%). Exposures from shellfish contribute the largest percentage for one contaminant (arsenic 44%). No other food type comprises more than 5% of national exposures to any of the six contaminants analyzed in detail, with the exceptions of beef, which accounts for between 6 and 9% of total exposure to DDT, dieldrin, and dioxins, and rice, which accounts for nearly 11% of total exposure to arsenic. After fish, the largest contributors to exposure are other meat and animal products, including beef, chicken, pork, and milk. The only nonanimal products contributing more than 1% of exposure to any of the six contaminants are rice, potatoes, wheat flour, and spinach. All other foods analyzed in the study, including oats, eggs, and other fruits and vegetables, contribute negligible

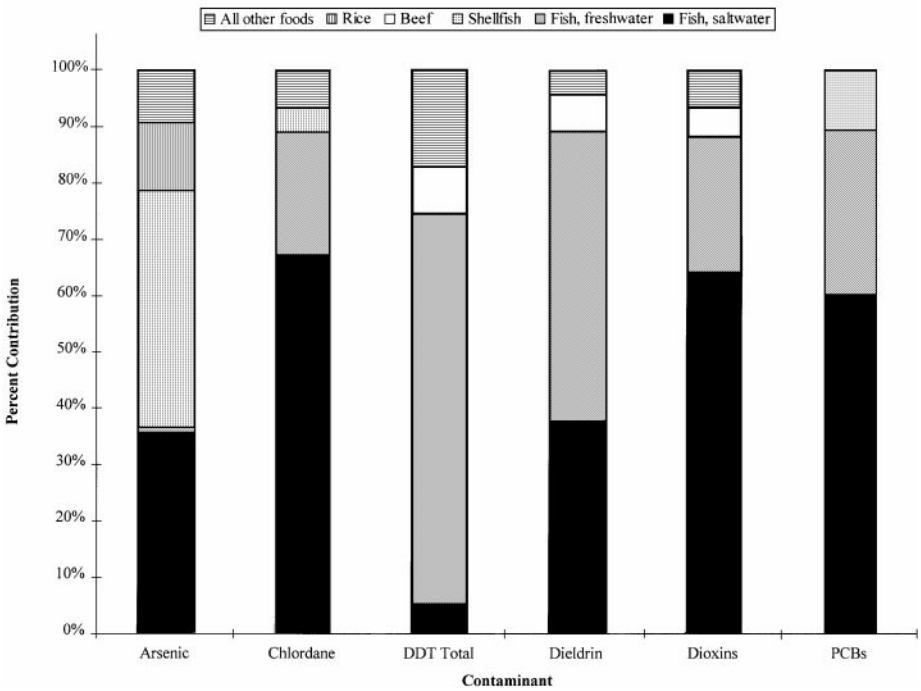

FIG. 4. Contribution of individual foods to total average national exposures.

amounts to national average contaminant exposures for these chemicals.

National Childhood Average Contaminant Exposures

This analysis examines national childhood average food ingestion exposure for three age groups: all children under 1 year of age, all children ages 1 to 5 years, and all children ages 6 to 11 years. For comparison to cancer benchmarks, exposures to children less than 12 years old were converted into lifetime equivalent doses, as described under Methods. Assuming that nondetects are equal to zero, childhood food ingestion exposures exceed the one in one million cancer benchmark concentration for the same six contaminants identified above for the whole population (DDT, chlordane, dioxins, arsenic, dieldrin, and PCBs). Hazard ratios (exposure divided by benchmark concentration) for exposures to these contaminants in the first 12 years of life range from 4 to 127 (see Fig. 5). When nondetects are assumed to equal half of the detection limit, a total of 13 different food contaminants have childhood exposures that exceed the one in one million cancer risk benchmark.

Only one contaminant, PCBs, exceeds noncancer benchmarks for the childhood age groups less than 12 years when nondetects are assigned a value of zero. The number of contaminants exceeding noncancer benchmarks increases when nondetects are set equal to one half of the detection limit. Exposure to chlordane, for example, is greater than the noncancer benchmark for all child age groups above 1 year. For children ages 1 to 5 years, exposures to arsenic, chlordane, dieldrin, and methamidophos are greater than the noncancer benchmarks, and for children ages 6 to 11 years, arsenic is greater than the noncancer benchmark.

To further explore the impact of cumulative exposures, noncancer hazard ratios for each age group were aggregated for all contaminants, since multiple contaminants with exposure levels slightly below the benchmark may be associated with potential health concerns when combined in aggregate. This was done as a screening exercise to ascertain the potential magnitude of cumulative exposures when considering multiple contaminants and accounting for differences in potency and hazard of the various contaminants. Figure 6 shows that children ages 1 to 5 years have the highest aggregate hazard ratios for noncarcinogenic effects. Children ages 6 to 11 years have the second highest aggregate hazard ratios for noncarcinogenic effects, whereas children less than 1 year of age the lowest aggregate hazard ratios and therefore the lowest exposure levels compared with

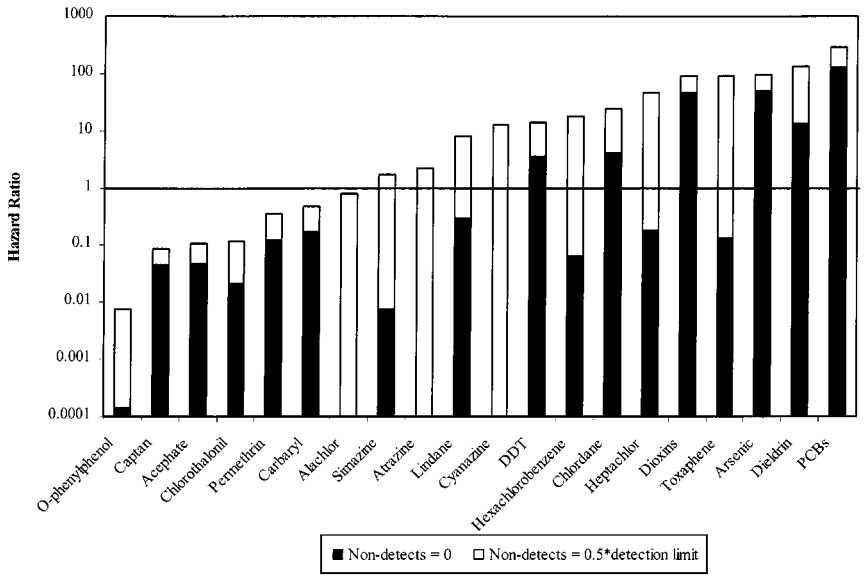

FIG. 5. Cancer hazard ratio for childhood exposures up to age 12 years.

benchmark values. These relationships are the same whether the nondetects are set equal to zero or to one half of the detection limit. Setting nondetects equal to one half the detection limits does, however, more than double the estimate of children's aggregate hazard from pesticide and industrial chemical exposures through food ingestion. Similar results were found for aggregate cancer hazard ratios. However, as with individual contaminant comparisons to cancer benchmarks, the aggregate noncancer hazard ratios for children overestimates risk because the benchmarks assume that children would be exposed at the estimated levels for a lifetime.

Contribution of Individual Foods to Childhood Exposure

Children consume more food per unit body weight and thus more contaminants per unit body weight than adults. Children also consume different foods

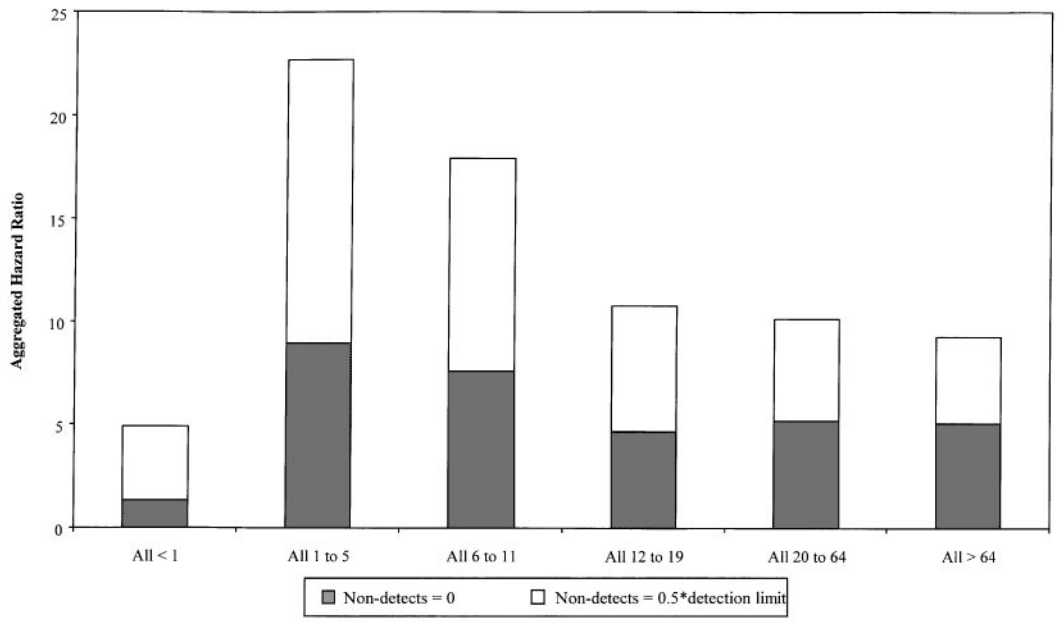

FIG. 6. Aggregated noncancer hazard rations for childhood exposures up to age 12 years.

and different amounts of foods than the general population. Infants, for example, have high arsenic exposure relative to other contaminants, due largely to a high consumption of rice and high arsenic concentrations in rice. Infants, however, have much lower exposures to other contaminants than all other subgroups, because they do not consume freshwater fish or shellfish and only a small amount of saltwater fish per unit body weight.

The contribution of individual foods to total exposures was analyzed for all children ages 1 to 5 years. This analysis was performed for the six contaminants identified in the previous analysis. Results are presented in Fig. 7. Similar to the results for adults, the analysis shows that fish, meat, and other animal products are the largest contributors to exposure for children ages 1 to 5 years. In addition, rice (for arsenic) and milk (for DDT) are larger contributors to children's exposure than to adults' exposure.

DISCUSSION

This analysis represents an important step in assessing and characterizing the potential hazards associated with contamination of food and suggests additional steps to be taken to further refine our understanding of this potential problem. Initial screening of the data found that estimated exposure to a number of contaminants in the average diet of adults and children exceed benchmark concentrations for cancer and noncancer effects, though these results should be interpreted cautiously due to limitations of the available data.

This study provides insights on the magnitude of potential exposures from food contamination. The results of the analysis have identified certain contaminants of particular concern, including arsenic, chlordane, DDT, dieldrin, dioxins, and PCBs. The one in one million benchmark for lifetime cancer risk for each of these contaminants is exceeded by the time age 12 years is reached. Exposures from age 12 to 70 years pose additional risks above those resulting from exposure in the first 12 years of life. The assessment of cancer hazard ratios for childhood exposure applied a standard exposure metric, the lifetime average daily dose. However, this approach may understate cancer risks, as childhood exposures may have greater probability of producing tumors than exposures in adulthood. Risks may be greater for children due to potential enhanced potency of exposures in childhood as well as the increased number of remaining years of life for tumors to form (McConnell, 1992; National Research Council, 1993). Therefore, the risks of several pollutants with childhood exposure hazard ratios somewhat less than one in Fig. 5 may actually exceed the cancer benchmark level of one in one million. In the case in which nondetects are assumed to be equal to zero, these pollutants include toxaphene, heptachlor, lindane, carbaryl, and permethrin. For several of these contaminants, exposures are largely attributable to

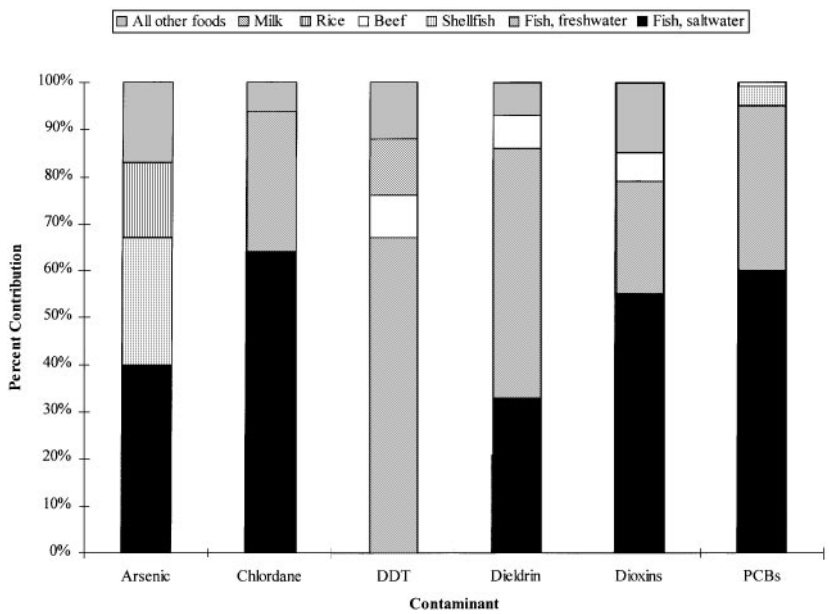

FIG. 7. Contribution of individual foods to total average exposure for children ages 1–5 years.

residues on produce. For example, childhood carbaryl exposure is associated primarily with apples and grapes.

Most of these pollutants were identified because they exceeded the cancer benchmark, which represents a one in a million risk level, rather than the noncancer benchmark. Exposures greater than the one in a million cancer risk screening level can result in large numbers of people potentially at risk. However, the one in a million cancer benchmark tends to be a more conservative screen than the noncancer benchmark. For example, a study by Gaylor (1989) compared the lower 95% confidence level of the dose which results in 1% of test animals exhibiting effects for both teratogenic and carcinogenic effects (this risk metric is often referred to as the LED_{01}). He found that the LED_{01} for teratogenic effects was similar to or lower than the LED_{01} for carcinogenic effects for four of nine chemicals evaluated, indicating that the dose associated with a 1% effect level was similar or lower for teratogenic effects than for carcinogenic effects. This indicates that the benchmark for noncancer effects may not be as conservative a screen as that for the cancer effects.

It is important to note that some of the identified pesticides have been banned for all uses in the United States. DDT, for example, has been banned for over two decades. However, because many of these compounds, such as DDT, are highly persistent, they continue to show up at significant levels in the food supply, indicating the importance of considering persistence when assessing potential risks.

The results indicate that exposures to contaminants identified in the analysis are largely driven by contamination in fish. However, there are some caveats to these results. One is that the fish samples were collected and tested in raw tissues. The total amount of contaminants actually consumed in fish may be less than levels present before cooking because lipids and lipophilic compounds are partially removed during cooking and processing. Previous studies indicate that the cooking and processing of fish decreases contaminant levels of at least five of the six contaminants analyzed in detail—chlordane, DDT, dieldrin, dioxins, and PCBs—with average losses of these contaminants ranging from 20 to 46% (Voiland *et al*., 1991; Sherer and Price, 1993; Zabik and Zabik, 1995; Zabik *et al*., 1995). However, even with a 46% decrease in fish contaminant concentrations, estimated national average exposure levels for the five contaminants would still exceed benchmark concentrations.

In addition, the testing procedures for the fish in the National Sediment Inventory include targeted testing for contaminants expected to be present in fish at the sampling sites or for sites that are expected to have contaminants present. Overall, this potential bias is likely to overestimate contaminant concentrations and resulting exposures. To assess this potential bias, contaminant data in NSI were compared to two other data sources—FDA's Total Diet Study and USEPA's National Study of Chemical Residues in Fish (NSCRF) (U.S. Environmental Protection Agency, 1992). For freshwater fish, it appears that contaminant levels measured in the TDS and NSCRF databases are lower than those measured in NSI, although the comparisons are somewhat uncertain because of the small sample sizes in TDS and NSCRF. For shellfish and saltwater fish the difference in contaminant levels is mixed. For saltwater fish, there are lower contaminant levels of arsenic, cadmium, and hexachlorobenzene in TDS and NSCRF than in NSI, but higher levels of DDT, dieldrin, and PCBs. Similarly for shellfish, there are lower levels of arsenic, cadmium, mercury, and PCBs in TDS and NSCRF than in NSI, but higher levels of DDT.

Evaluating the contribution of different food sources to overall exposure finds that, even if the freshwater fish contaminant levels in NSI are overestimated, other foods still contribute significantly to exposure. Significant decreases in the contribution of freshwater fish to exposure levels would result in somewhere between 20 and 70% reduction in exposure for chlordane, DDT, dieldrin, dioxins, and PCBs. However, the exposure levels for each of these contaminants are at least an order of magnitude greater than the cancer benchmarks and in the case of dioxins and PCBs, 1000 times greater than the cancer benchmarks.

Arsenic is a food contaminant that has been identified through our screening analysis as potentially representing a health risk. It is important to note that the measurements of arsenic in food used in this study are for total arsenic, which is made up of inorganic and organic arsenic, with inorganic arsenic typically comprising less than half the total. Inorganic arsenic is more toxic than organic arsenic; the results of this study could overestimate potential risks because the RfD and cancer benchmark used in the analysis are for inorganic arsenic rather than total arsenic. A recent analysis of arsenic data from Tao and Bolger (1998) by the National Research Council (National Research Council, 1999) assessed the daily intake of inorganic arsenic for various age and gender groups. The assessment was based on

estimates of the percentage of inorganic arsenic in the measured concentrations of total arsenic from the FDA Total Diet Study for 1991–1997. The study estimated a range of 0.066–0.34 μg/kg/day of intake of inorganic arsenic with an average of about 0.14 μg/kg/day. The national average exposure estimate derived in our study for total arsenic, with no adjustment for inorganic arsenic, was 0.2 μg/kg/day, based primarily on measured arsenic concentrations from the National Sediment Inventory. Our study's estimates for total arsenic are similar to the NRC estimates for inorganic arsenic, indicating that our estimates based on total arsenic are unlikely to significantly overestimate exposure to inorganic arsenic.

Our study's exposure estimates represent information from the early 1990s, and levels of contaminants, particularly some of the persistent compounds such as DDT and PCBs, have likely decreased some since then. Most studies evaluating trends in persistent compounds have focused on trends prior to 1990 or through the mid-1990s (Robinson *et al*., 1990; Fensterheim, 1993; Noren, 1993; Papke *et al*., 1994; Becher *et al*., 1995; Bopp *et al*., 1998). A study of PCBs in the human diet found a 2- to 10-fold decline in PCB contamination of shellfish and fish from the early 1970s to the late 1980s and similar decreases in the diet and adipose tissue of the public (Fensterheim, 1993). However, Fensterheim (1993) also notes that the rate of decline will slow, since most of the dramatic declines are associated with major regulatory initiatives in the 1970s and 1980s. Similar results would be expected for other persistent compounds such as DDT and dioxins. A study of sediment in the Hudson River basin, which would contribute to fish concentrations, also found declines from the 1960s to the mid-1980s to mid-1990s in DDT, dioxins, and PCBs due to regulatory measures, though there were a few areas which saw increases in sediment concentrations (Bopp *et al*., 1998). Decreases in dioxins in blood and breast milk are also noted in several European studies (Noren, 1993; Papke *et al*., 1994; Becher *et al*., 1995). Overall, these indicate that there has been a substantial decrease in fish contaminant levels from the 1970s due to regulatory controls, but the rate of decline may be slower and in some cases nonexistent in the 1990s.

An important consideration in this analysis is the large number of nondetects. The percentage of nondetects for the contaminants ranged from 10 to 100%, with an average of 86%. The analysis was performed assuming that nondetects were equal to either zero or one half of the detection limit to assess the impact of different nondetect values on the exposure outcomes. For some contaminants, assuming that nondetects are equal to one half of the detection limit dominates the overall contaminant exposure. In these cases, assuming a value of one half of the detection limit is likely to overstate the contaminant levels. A more refined analysis of nondetects in the case of pesticides currently in use could consider the food source and whether pesticides were applied to that crop to determine whether one half the detection limit is an appropriate assumption. Future work will consider the distribution of the detects in assessing the likely values below the detection limit and other methods for determining appropriate values for the nondetects. Despite the presence of many nondetects, exposures to six contaminants exceed benchmarks even when nondetects are set equal to zero. In addition to the number of nondetects, the sample size is fairly small for some of the contaminants (e.g., dioxins), and thus results for these contaminants may be less reliable than results for other contaminants.

Another source of uncertainty in this analysis arises from the fact that one food type may be comprised of several different forms of a particular food. For example, average freshwater fish contamination levels were calculated by averaging concentrations in different freshwater fish species sampled. However, contamination levels are species dependent. For lipophilic contaminants, species with a higher fat content will have higher contaminant levels. Therefore, if the actual consumption of freshwater fish is composed of fish species that, on average, have a higher fat content than the fish sampled, the preliminary analysis will understate exposures to lipophilic contaminants in freshwater fish. While there are inconsistencies in the composition of other foods in the consumption and contamination databases, it is particularly important for the three fish categories, as fish comprises a large proportion of the average national exposures to certain contaminants.

This analysis estimates average contaminant exposure across both consumers and nonconsumers of the individual food items. Only 4, 12, and 30% of the population are consumers of freshwater fish, shellfish, and saltwater fish, respectively. Thus, the contaminant exposure level from fish for the average consumer is much greater than the average for the whole population. In addition, this analysis examines only the mean level of contaminant exposure and does not assess exposures for those populations that consume higher levels of particular foods or foods with much higher than average levels of contaminants.

There are also limitations associated with the food consumption data due to the short sampling period. USDA collected food consumption data over 3 days, which were scheduled in different seasons to account for the potential for seasonal variation in consumption. Because of the short duration of sampling, however, the consumption data may not accurately represent average daily consumption over a longer period of time. Better characterization of the consumption of foods that drive the analysis (e.g., fish and meats) are needed to reduce the uncertainty associated with the consumption data. This includes better information on both the proportion of individuals that consume each food and the quantity consumed by these consumers.

This analysis has focused on the combined exposures to food contaminants that result from their presence in several different types of foods. Under the Food Quality Protection Act (FQPA) of 1996, the U.S. Environmental Protection Agency is currently developing methods to extend the scope of exposures considered in analysis of pesticides (U.S. Environmental Protection Agency, Office of Pesticide Programs, 2000). FQPA requires that USEPA consider the aggregate exposures to pesticides from multiple routes and pathways—combining food ingestion exposures with drinking water exposures and oral, inhalation, and dermal exposures that may result from residential pesticide application. FQPA also requires that USEPA consider the cumulative risks to human health that may result from exposures to multiple pesticides with common mechanisms of toxicity. When fully implemented, these new approaches will provide a broader perspective on the potential of various pesticides to pose substantial risks to human health.

Despite the limitations associated with the analysis, the results point to potentially high exposures to contaminants in food and represent an important step toward better characterization of these exposures. The analysis has identified research priorities for improving the data needed to better understand food contaminant exposures. The small number of samples for shellfish and saltwater fish combined with the potentially high contribution of these foods to total exposures for some contaminants (arsenic and PCBs in shellfish; arsenic, chlordane, dieldrin, dioxin, and PCBs in saltwater fish) suggest that testing of these contaminants in shellfish and saltwater fish should be a priority for future monitoring activities. In addition, the food contamination database contains a small number of samples of certain contaminants with potentially high exposures, including arsenic, dioxins, carbaryl, permethrin, simazine, and toxaphene. Additional research efforts should focus on collecting data for these contaminants. Results also indicate that detection limits for many samples exceed benchmark concentrations. Lower detection limits are needed to better identify the frequency with which contaminant concentrations are at levels of public health concern.

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