Serum Persistent Organic Pollutants

Persistent organic pollutants (POPs) are manmade organic chemicals that remain in the environment for years or decades. POPs are of special concern because they often remain toxic for decades or longer after release to the environment. The more persistent a toxic chemical is, the greater the probability for human exposure over time. Because they circulate globally long after being released into the environment, POPs are often detected in locations far from the original source (U.S. EPA, 2009).

One of the major sources of POPs exposure among the general population is food. Food contamination begins with contaminated soil and/or plants, but is of greatest concern to humans as the POPs move up the food chain into animals. Because POPs typically accumulate in fatty tissue and are slow to be metabolized, they bioconcentrate (i.e., increase in concentration) through the food chain. Therefore, foods such as dairy products, eggs, animal fats, and some types of fish are more likely to contain greater concentrations of POPs than fruits, vegetables, and grains. POPs have been linked to adverse health effects such as cancer, nervous system damage, reproductive disorders, and disruption of the immune system in both humans and animals (U.S. EPA, 2009).

This indicator presents data from the Centers for Disease Control and Prevention's (CDC's) National Health and Nutrition Examination Survey (NHANES) for three survey periods: 1999-2000, 2001-2002, and 2003-2004. NHANES is a series of surveys conducted by CDC's National Center for Health Statistics that is designed to collect data on the health and nutritional status of the civilian, non-institutionalized U.S. population using a complex, stratified, multistage, probability-cluster design. CDC's National Center for Environmental Health conducted the laboratory analyses for the biomonitoring samples. Beginning in 1999, NHANES became a continuous and annual national survey; biomonitoring for certain environmental chemicals also was implemented. These data are presented here largely as a baseline, with the intent of reporting trends over larger time periods in the future. Serum levels of POPs or their metabolites were measured in NHANES participants age 12 or older. This indicator includes the following three broad classes of POPs:

- Organochlorine pesticides
- Polychlorinated dibenzo-p-dioxins (dioxins) and polychlorinated dibenzo-p-furans (furans)
- Polychlorinated biphenyls (PCBs)

Organochlorine pesticides were first introduced in the 1940s. Because of their environmental persistence, EPA banned most uses of these chemicals during the 1970s and 1980s. However, many other countries still produce and/or use organochlorines. These fat-soluble chemicals are most commonly absorbed through fatty foods. These pesticides are associated with effects to the central nervous system at acute exposure levels and potential carcinogenic effects with long-term exposure (Reigart and Roberts, 1999). This indicator includes eight organochlorine pesticides that were measured in NHANES 1999-2000, 2001-2002, and 2003-2004; data for three of these pesticides (aldrin, dieldrin, and endrin) first became available with the release of results from NHANES 2001-2002 (CDC, 2013).

- **Aldrin and dieldrin.** These two pesticides were widely used from the 1950s until 1970, when EPA prohibited most agricultural uses. However, they continued to be used to control termites until that use was prohibited in 1987. Aldrin rapidly converts to dieldrin in the environment or after being ingested or absorbed into the body. Dieldrin is more persistent and often accumulates in fatty tissues (CDC, 2009).

- **Chlordane and heptachlor.** EPA banned these pesticides in 1988. Within the body, chlordane is metabolized to oxychlordane and *trans*-nonachlor, and heptachlor is metabolized to heptachlor epoxide (CDC, 2009). Chlordane was commonly used against termites and on some agricultural crops and heptachlor was used primarily against soil insects and termites (Ritter et al., 1995).

- **DDT.** Dichlorodiphenyltrichlorethane, or DDT, was banned in the U.S. in 1973 but is still produced in other countries, where it is used primarily to control mosquitoes. In the body or the environment, DDT breaks down to DDE (dichlorodiphenyldichloroethane), a more persistent chemical. DDT or DDE in the human body may reflect either a relatively recent exposure or cumulative past exposures (CDC, 2009).
- **Endrin.** Endrin is a stereoisomer (i.e., a molecule that is a mirror image of another molecule with the same molecular formula) of dieldrin. Endrin production was discontinued in 1986, primarily because of its persistence in the environment. Unlike many other organochlorine pesticides, endrin does not readily accumulate in body tissues and is metabolized and eliminated from the body relatively quickly (CDC, 2009).
- **Hexachlorobenzene** (HCB) was commonly used as a pesticide until 1965. HCB was also used in the past as a fungicide to protect wheat seeds, and for a variety of industrial purposes, including rubber, aluminum, and dye production and wood preservation. EPA canceled registered use in 1984; however, HCB is still formed as a byproduct during manufacturing of other chemicals and pesticides (CDC, 2012; U.S. EPA, 2011a).
- **Mirex** has not been produced or used in the U.S. since 1978. It was used primarily in the southeastern U.S. to control fire ants. The primary source of exposure is dietary, most often through consumption of fish (U.S. EPA, 2011b).

Dioxins and furans are similar classes of chlorinated aromatic chemicals, usually generated as pollutants or byproducts. In the environment, dioxins and furans occur as a mixture of about 20 compounds (termed "congeners"). This indicator considers seven polychlorinated-p-dioxins and ten polychlorinated dibenzofurans measured as part of NHANES. The half-lives of these congeners range from roughly 3 to 19 years. Human exposure occurs primarily through food; other sources of exposure include industrial accidents, burning of PCBs contaminated with dioxins and furans, burning of plastics such as PVC, and spraying or unintended releases of contaminated herbicides such as Agent Orange. The detection of dioxins and furans in human serum can reflect either recent or past exposures (CDC, 2009).

Researchers continue to study the potential adverse health effects associated with dioxins and furans. Studies of individual congeners have shown immunotoxic, developmental/reproductive, and other systemic effects. The effects of individual congeners in humans are difficult to determine, since exposures are more likely be to mixtures of several congeners. The dioxin congener TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin) is the most toxic form of dioxin and is classified as a known human carcinogen (IARC, 1997). Uncertainties remain, however, about the levels and mechanisms involved in producing harmful effects in humans.

PCBs are chlorinated aromatic hydrocarbons used in a variety of industries as electrical insulating and heat exchange fluids. PCBs are composed of mixtures of up to 209 different chlorinated congeners. U.S. production of PCBs peaked in the early 1970s; PCBs were banned in 1979. The primary source of exposure for the general population is ingesting foods contaminated by PCBs, but other sources include releases from waste sites and fires involving transformers and capacitors (CDC, 2009). PCBs typically accumulate in fatty tissues (ATSDR, 2000).

The detection of PCBs in human serum can reflect either recent or past exposures. PCBs with higher degrees of chlorination persist in the human body from several months to years after exposure. Coplanar and mono-ortho substituted PCBs exhibit health effects similar to dioxins. The human health effects of PCBs include changes in liver function, elevated lipids, and gastrointestinal cancers, as well as low birth weight and developmental effects (CDC, 2009). Given the large number of PCB congeners (dioxin- and non-dioxin like) measured as part of NHANES, this indicator focuses primarily on those PCBs detected with sufficient frequency to calculate a geometric mean across most of the 1999-2004 survey periods.

What the Data Show

Organochlorine Pesticides

Exhibit 1 presents the lipid-adjusted and whole weight geometric means and four percentile values (50th, 75th, 90th, and 95th) for selected organochlorine pesticide metabolites measured in serum. During the most recent survey (2003-2004), the lipid-adjusted geometric mean for *p,p'*-DDE (a metabolite of DDT) was 238 nanograms per gram (ng/g), a decrease from the earlier survey periods. The lipid-adjusted geometric mean for *trans*-nonachlor (a component of technical-grade chlordane) was 14.7 ng/g, also a decrease from the earlier survey periods. The lipid-adjusted geometric mean for HCB was 15.2 ng/g. DDT, aldrin, dieldrin, endrin, heptachlor epoxide (the metabolite for heptachlor), and mirex were not measured with sufficient frequency above the limit of detection to calculate a geometric mean for any survey periods.

With the exception of *p,p'*-DDE, most POPs were not detected with sufficient frequency to enable meaningful comparisons across demographic subpopulations. Geometric mean serum *p,p'*-DDE concentrations were at least two times lower in those age 12-19 years compared to those 20 years and older. The lipid-adjusted geometric mean level in Mexican Americans was 444 ng/g during the most recent survey, nearly two times higher than levels in non-Hispanic whites and non-Hispanic blacks. This trend is consistent across survey periods (CDC, 2013; data not shown).

Dioxins and Furans

In the U.S., quantifiable emissions of dioxin-like compounds from all known sources decreased by an estimated 90 percent between 1987 and 2000 (U.S. EPA, 2006). The generally low lipid-adjusted values reported in NHANES 1999-2004 generally support that estimated decline (CDC, 2013). For example, among the entire NHANES 1999-2000 sample population, TCDD (generally considered the most toxic dioxin) was detected less than 1 percent of the time (CDC, 2003). During the 2003-2004 survey period, TCDD was reported only at the 90th and 95th percentiles in those age 20 years and older (CDC, 2013; data not shown). Exhibit 2 shows those dioxins and furans that were detected frequently enough across the 1999-2004 survey period for geometric means to be calculated. The additional reporting in the most recent survey is primarily due to improvements in the limits of detection for individual congeners.

In general, the more highly chlorinated dioxin and furan congeners were the main contributors to the human body burden. The higher concentrations of these congeners in human samples are a result of their greater persistence in the environment, bioaccumulation in the food chain, resistance to metabolic degradation, and greater solubility in body fat (CDC, 2005).

PCBs

Exhibit 2 displays geometric means and percentile values for two dioxin-like PCBs (PCB 126 and PCB 169) and three non dioxin-like PCBs (PCB 138 & 158; PCB 153; and PCB 180) for which geometric means could be calculated for at least two of the three reported survey periods with one exception: a geometric mean could only be calculated for PCB 169 during the 2001-2002 survey period. As more NHANES data become available, trends across survey periods and subpopulations can be better assessed, especially with the increasing sensitivity of congener-specific measurements.

Limitations

- The relatively small number of samples collected in a two-year cycle (e.g., 1999-2000 or 2001-2002) may, in some cases, result in measures of central tendency that are unstable from one survey period to the next.

- Health-based benchmarks for organochlorine pesticides and dioxin, furan, and PCB congeners in serum have not yet been established.

Data Sources

Data used for this indicator were obtained directly from CDC's Fourth National Report on Human Exposure

to Environmental Chemicals, Updated Tables, September 2013, available at http://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Sep2013.pdf (PDF) (770 pp, 9.5MB) (CDC, 2013).

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Exhibit 1. Serum concentrations of selected organochlorine pesticide metabolites for the U.S. population age 12 years and older, lipid-adjusted and whole weight, 1999–2004

			Geometric mean and selected percentiles for organochlorine pesticide metabolite concentrations (ng/g)				
	Survey years	Sample size	Geometric mean	50th percentile	75th percentile	90th percentile	95th percentile
Aldrin							
Lipid-adjusted	2001–2002	2,275	NC	<LOD	<LOD	<LOD	<LOD
	2003–2004	1,946	NC	<LOD	<LOD	<LOD	<LOD
Whole weight	2001–2002	2,275	NC	<LOD	<LOD	<LOD	<LOD
	2003–2004	1,946	NC	<LOD	<LOD	<LOD	<LOD
Chlordane							
Oxychlordane							
Lipid-adjusted	1999–2000	1,661	NC	<LOD	20.8	34.4	44.8
	2001–2002	2,249	11.4	11.1	21.7	36.4	49.7
	2003–2004	1,978	9.37	10.3	18	29	37.7
Whole weight	1999–2000	1,661	NC	<LOD	0.14	0.26	0.31
	2001–2002	2,249	0.07	0.07	0.14	0.25	0.35
	2003–2004	1,978	0.057	0.063	0.119	0.204	0.269

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Exhibit 2. Serum concentrations of selected polychlorinated dibenzo-p-dioxins (dioxins), polychlorinated dibenzofurans (furans), and polychlorinated biphenyls (PCBs) for the U.S. population age 12 years and older, lipid-adjusted and whole weight, 1999-2004

			Geometric mean and selected percentiles for dioxin, furan, and PCB concentrations				
	Survey years	Sample size	Geometric mean	50th percentile	75th percentile	90th percentile	95th percentile
Dioxins (pg/g)							
1,2,3,4,6,7,8,9-OCDD							
Lipid-adjusted	1999-2000	1,921	NC	<LOD	406	674	913
	2001-2002	1,171	346	333	573	944	1,260
	2003-2004	1,851	NC	<LOD	336	582	767
Whole weight	1999-2000	1,921	NC	<LOD	2.53	4.26	5.95
	2001-2002	1,171	2.23	2.17	3.86	6.46	9.11
	2003-2004	1,851	NC	<LOD	2.18	3.76	5.02
1,2,3,4,6,7,8-HpCDD							
Lipid-adjusted	1999-2000	1,894	NC	<LOD	58.2	86	112
	2001-2002	1,220	39	40.2	68.7	115	147
	2003-2004	1,874	25.3	24.9	42.5	70.4	91.3

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