

PERSISTENT ORGANIC POLLUTANTS IN HUMAN MILK

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Persistent organic pollutants (POPs) in human milk. POPs are chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in the fatty tissue of living organisms, and are toxic to humans and wildlife

This summary is based on data on concentrations of various persistent organic pollutants (POPs) in human milk in a number of European countries. It contains information on the environment and health context and the policy relevance, and an assessment of the situation in the WHO European Region.

KEY MESSAGE

☺ The indicator shows clear differences in POP levels among European countries: for dioxins, the differences were initially as much as three- to five-fold. It also shows a clear decrease in most countries, especially in those with the highest initial levels. This mainly reflects successful abatement at source, such as bans on production and use, as well as improved waste incinerators. Polychlorinated dibenzo-p-dioxins (PCDDs) and dioxin-like compounds, including polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs), are probably the group of POPs with the lowest safety margin. The levels of pesticide POPs in human milk are very low. On the other hand, some newer compounds have emerged in the form of polybrominated and polyfluorinated compounds. Although these are still present at reasonably low levels, they need to be monitored.

RATIONALE

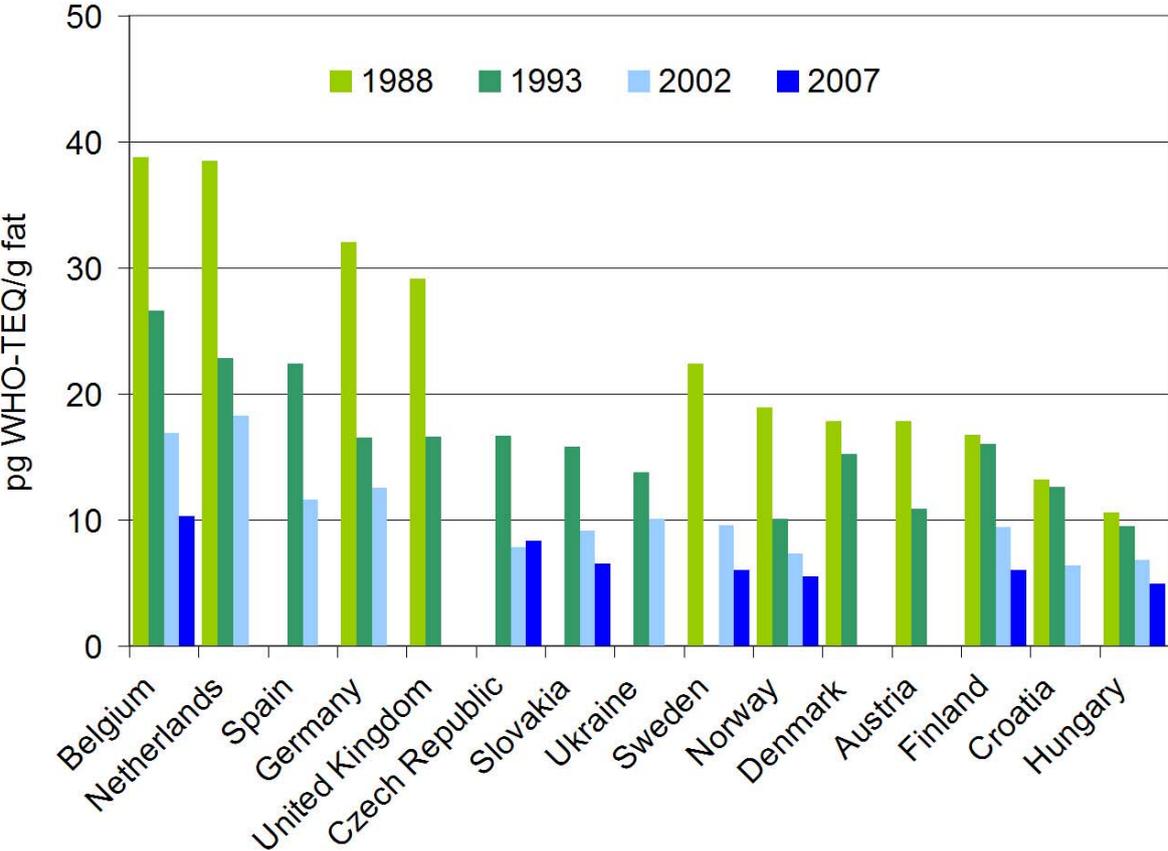
POPs are a group of organochlorine and related chemicals. They are lipophilic and resistant to both physicochemical and biological degradation and thus accumulate in living organisms and subsequently in humans via the food chain. This causes a risk of, for example, developmental effects, which are the most sensitive adverse health endpoints (1–4). Levels in human milk fat are a good indicator of levels in the population as a whole. This measure is also relevant in measuring the developmental exposure of unborn children.

PRESENTATION OF DATA

The most systematic information on POPs in humans is based on four rounds of human milk analysis studies of dioxins and PCBs coordinated by WHO (5). The first round, in 1987/1988, included 12 European countries and indicated major differences between them in lipid-based concentrations expressed in WHO toxic equivalents (WHO-TEQs): for example, about 10 pg/g WHO-TEQs in Hungary to about 40 pg/g WHO-TEQs in the Netherlands (Fig. 1). The decrease in concentrations has been in the order of 5% or more per year, highest in countries with the highest initial concentrations. More countries,

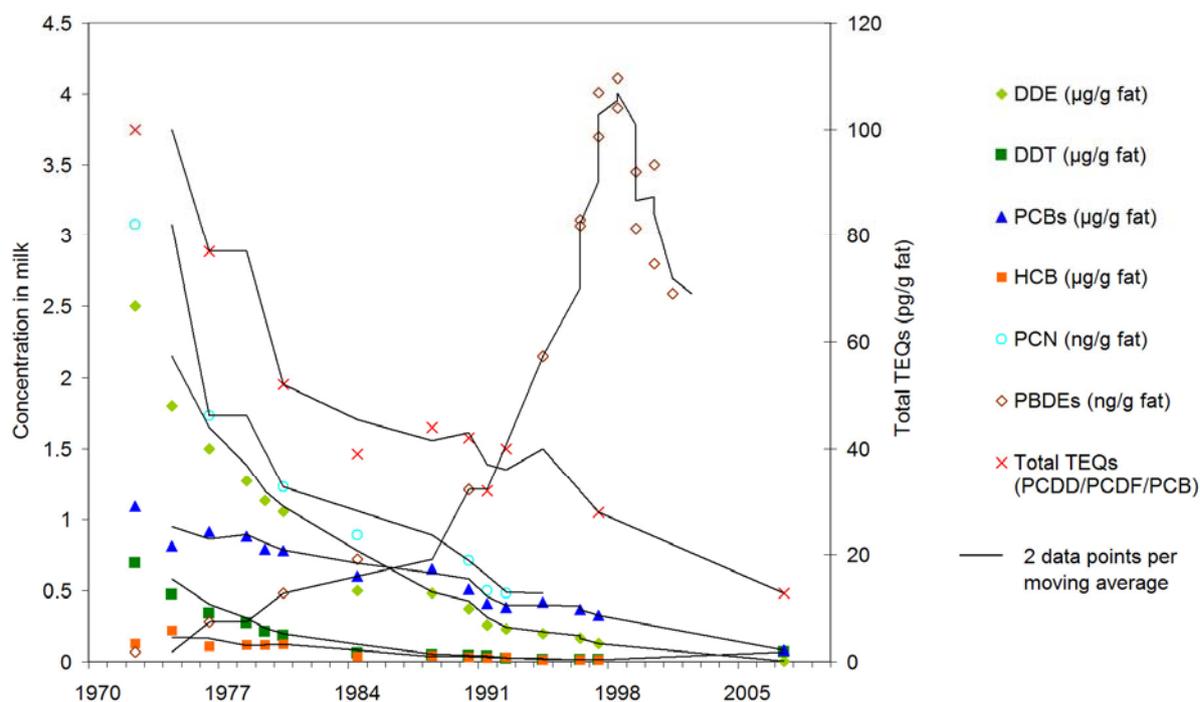
including non-European countries, joined the second and third rounds; only four countries participated in all four rounds. The methodology is controlled by intercalibration and is considered highly reliable. The present concentrations are 4–10 pg/g in participating countries (Fig. 1). There are longer series of measurements from some countries, such as Sweden, showing that the decrease started as early as the late 1970s when concentrations were about ten times higher than the present levels (Fig. 2).

Fig. 1. Dioxin levels in human milk in selected countries, 1988–2007



Source: Van Leeuwen & Malisch (5); Malisch et al., unpublished data.

Fig. 2. POPs levels in human milk, Sweden, 1972–2007



Source: Lunden & Noren (6); Noren & Meironyte (7); Lind et al. (9); Malisch et al., unpublished data.

There are fewer systematic data on other POPs. Norwegian and Swedish long-term analyses of human milk (6–8) indicate a decrease of 90% in DDT and its metabolite *p,p'*-DDE and smaller decreases in total PCBs, hexachlorobenzene (HCB) and polychlorinated naphthalenes (PCNs) (Fig. 2; note different units for different compounds: pesticides and PCBs are in micrograms, PCNs and polybrominated diphenylethers (PBDEs) in nanograms, and dioxin-like compounds in picograms). Because there are no coordinated analyses, data from different countries are difficult to compare. However, all organochlorine pesticide levels in Europe are very low. During the fourth round of coordinated dioxin analyses, PCBs and pesticides were also measured. In none of the samples was aldrin, chlordane, endrin or heptachlor detected, although low concentrations of chlordane and heptachlor metabolites were still detected. There have been recent increases in PBDEs and perfluorinated compounds. PBDEs now seem to be decreasing as a result of the ban on penta- and octa-derivatives taken up by biota and humans. It is noteworthy that the half-lives in humans of some PBDEs, especially decabromodiphenyl ether (BDE-209), are a matter of weeks (10), whereas those of dioxins are measured in years. This means that the PBDEs currently used may not be very cumulative.

HEALTH AND ENVIRONMENT CONTEXT

POPs have long been recognized as a serious concern for human health and the environment. As early as the 1960s and 1970s, some POPs such as DDT and PCBs were banned or phased out in many industrialized countries. Over time, it became clear that this was not sufficient. POPs are very persistent and stay in the environment for long periods. They are also prone to accumulate in higher organisms and to magnify in the food-chain: levels increase by several orders of magnitude from sea plankton through food items such as fish up to humans (11). Owing to their semi-volatility and persistence, some of them are transported over long distances to locations where they have never been used, such as the Arctic. At high concentrations, POPs cause severe environmental effects, such as reproductive and developmental effects in wild and laboratory animals (1,12,13). There is more uncertainty about health effects in humans, especially at the current environmental levels, because the intakes of humans are much lower than those of some animal species.

WHO recognizes the concern about the potential risks of POPs in human milk. Nevertheless, the beneficial effect of breastfeeding as the optimal food source for newborn babies should always be emphasized. In particular, when sharing information with the general public, it should be made clear that the presence of dioxins and PCBs in human milk is not an indication for avoiding breastfeeding (14,15). Body burden is clearly age-dependent and is lowest in the young age groups most at risk during pregnancy or breastfeeding (16). Tolerable daily intake values (TDIs) have been set so as to keep mater-

nal intake low enough to prevent harmful exposure of the breastfed infant (1). TDIs are not intended for mother's milk.

POLICY RELEVANCE AND CONTEXT

The international community has responded to the threat from POPs by negotiating a global treaty, the Stockholm Convention on POPs, with the objective of protecting human health and the environment from POPs (17). The Convention was adopted in May 2001, entered into force in May 2004 and has at present 162 parties (2008). The first 12 POPs included in the Convention are aldrin, chlordane, DDT, dieldrin, endrin, heptachlor, HCB, mirex, toxaphene, PCBs, PCDDs and PCDFs.

The Convention requires that parties phase out or ban the production, use, import and export of intentionally produced POPs, with the exception of DDT, which will be permitted for vector control according to WHO guidelines until feasible alternatives have been found. For unintentionally produced POPs, namely PCDDs and PCDFs, parties should minimize and, where feasible, eliminate emissions from anthropogenic sources. In doing this, parties shall promote the application of measures that may achieve a realistic and meaningful level of reduction of releases or elimination of sources, such as by introducing and using the best available technologies and best environmental practices. A financial mechanism is included to support the implementation of the Convention, particularly in developing countries and countries with economies in transition, for which the major entity at present is the Global Environmental Facility. This Facility has opened a POPs area to provide funding for the development of national implementation and action plans and further implementation activities. By January 2009, 89 parties had submitted their national implementation plans.

The Convention also requires the Conference of Parties to evaluate its effectiveness, starting four years after its entry into force. To facilitate this evaluation, the Conference of Parties should initiate arrangements to provide itself with comparable monitoring data on the presence of POPs and their regional and global environmental transport. The first evaluation will take place in 2009 and should be a baseline for future evaluations. It will focus on core data from appropriate matrices, such as air and human tissues (blood and/or human milk) and rely extensively on existing programmes (18), such as the Arctic Monitoring and Assessment Programme, the Global Air Passive Sampling project and the WHO Global Survey of Human Milk for POPs (19). The aim is to provide core data for the 12 POPs from all 5 United Nations regions. Strategic arrangements and partnerships should be established, involving the health sector.

European Union (EU) Regulation (EC) No. 850/2004 on POPs entered into force on 20 May 2004 (20). This implements the provisions of the Stockholm Convention. Dioxins, PCDFs and PCBs are listed as unintentionally released POPs, the releases of which should be continuously and cost-effectively reduced as quickly as possible (21). The links between the exposure of children to POPs and their health effects are specifically addressed by the EU's SCALE initiative (11) and the Children's Environment and Health Action Plan for Europe indicator RPG IV (22). Policy action should follow from these European strategies that take into account children's sensitivity to these pollutants (23).

ASSESSMENT

Among the POPs, dioxins (PCDDs) and dioxin-like chemicals (including PCDFs and dioxin-like PCBs) seem to have the lowest safety margin and to be the most likely group to cause adverse effects in humans. During the 1970s, at concentrations 5–10 times higher than at present, they were possibly the cause of subtle effects such as effects on tooth development (12,24). The sources of these compounds have been the incineration of municipal waste, chlorine gas bleaching of wood pulp and the metal industries, together with a number of minor sources. Until the 1980s, there were also important impurities in the production of certain chemicals (PCBs, chlorophenols and their derivatives). Advances in abatement have been most remarkable in waste incineration and major industries, including forest industries. This can be seen in environmental samples, such as lake and sea-bed sediment layers, fish, fish-eating birds and seals, and human milk (Fig. 1 and 2). The largest remaining sources are the metal and cement industries, landfill fires and small-scale wood and biomass burning. There may still be considerable variations among countries.

Developmental effects are thought to be those effects that occur at the lowest dose levels (1,12,24). Human milk is an ideal medium for monitoring these: it provides a long-term average of the body burden because most of these compounds have long half-lives, and it is thus relevant both for risk during pregnancy and for measuring the intake of the breastfed baby. Both of these steps are believed to be crucial for assessing the risk to the whole population of developmental effects, which is also the basis of the latest WHO risk assessment of dioxins (1). For older populations with higher body burdens (16), the relative risk of cancer, while real, is not very high even at the highest industrial exposures (1,25). Recent results of studies on families of fishermen indicated that, despite much higher dioxin

and PCB body burdens, mortality (including cancer mortality) was lower than in the general population (26). In Seveso, Italy, after a very high level of accidental exposure, there were reports of developmental effects on teeth, altered sex ratios and a possible increase in some rare types of cancer (4,27,28).

It is more difficult to assess the health risks of compounds other than dioxins, as the data on both exposures and effects are less systematic. Organochlorine pesticides or their metabolites can be still found in human samples in Europe, but the concentrations are low and their health relevance has clearly decreased. This is clearly seen with DDT, for which some countries have long-term exposure data (Fig. 2). The parent compound is present at very low concentrations but some of its long-half-life metabolites, such as p,p'-DDE, can still be found, albeit at reduced concentrations.

Some compounds have more recently come into focus. PBDEs (flame retardants used in plastics and textiles) were found in human milk at the end of 1990s (Fig. 2). Lower brominated diphenylethers, such as tetra- to octa-congeners, are absorbed by different animal species and they bioaccumulate to some extent. They were therefore banned by the European Commission in 2004 and their concentrations in Europe are now decreasing, in contrast to the United States where levels are about an order of magnitude higher and still rising (29). Monitoring is still warranted even in Europe because there is uncertainty about the metabolic fate of BDE-209, which continues to be produced. It is itself very poorly absorbed by biota and fairly rapidly eliminated in humans (10), but it may be broken down to lower brominated species.

Another new group of halogenated compounds is perfluorinated alkyl compounds (PFAs), such as perfluorooctane sulfonate, which were increasingly used as water repellents and for many other uses. These are also highly resistant in the environment and have been shown to accumulate in biota. Some of these compounds have therefore been voluntarily phased out by industry but are worth monitoring because of their persistence.

DATA UNDERLYING THE INDICATOR

Data source

References (5–7,16), including the unpublished results of the fourth round of monitoring of human milk, were the main data sources used in this fact sheet. In addition, the Global Environment Monitoring System – Food Contamination Monitoring and Assessment Programme (GEMS/Food) Contaminants Database, accessed through the WHO Summary Information on Global Health Trends (SIGHT), was also consulted (30). The latter gives information on 20 countries throughout the WHO European Region and the EU.

Description of data

Data from references (5–7) were mostly from pooled representative human milk samples collected and analysed according to the WHO guidelines on the biomonitoring of human milk for POPs (19). Results were generally expressed as ng/g milk fat and WHO-TEQs (pg/g fat).

The data were collected by GEMS/Food participating institutions using standardized methods for sampling and measuring contaminants and for submitting data to the GEMS/Food database. They included levels of dioxins, PCDFs and PCBs in human milk gathered under the SCOOP/EU/RIVM project. The database contains data on many other POPs. Results are expressed as concentrations of POPs in µg/kg milk fat and µg/kg whole milk, but mostly as µg/kg of unspecified matrix (that is, no distinction as to whether it is whole milk or only milk fat).

Method of calculating the indicator

Studies with concentrations of POPs in µg/kg (or an alternative mass/mass unit) in milk fat were used. The concentrations of PCDDs, PCDFs and dioxin-like PCBs were converted to WHO-TEQs relative to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), based on toxic equivalency factors as recommended by WHO (31).

Geographical coverage

Belgium, Croatia, the Czech Republic, Finland, Germany, Hungary, the Netherlands, Norway, Slovakia, Spain, Sweden and Ukraine.

Period of coverage

1971–2008.

Frequency of update

Varies between compounds and countries.

Data quality

Data quality varied between compounds and countries. However, data used in this fact sheet were of good quality, representative of the whole country and sampled and analysed in officially accredited laboratories or in laboratories that had successfully participated in relevant proficiency tests.

REFERENCES

1. WHO Temporary Adviser Group. Consultation on assessment of the health risk of dioxins; re-evaluation of the tolerable daily intake (TDI): executive summary. *Food Additives & Contaminants*, 2000, 17:223–240.
2. Safety evaluation of certain food additives and contaminants. Polychlorinated dibenzodioxins, polychlorinated dibenzofurans and coplanar polychlorinated biphenyls. Geneva, World Health Organization, 2002 (*Food Additives Series No. 48*).
3. Compilation of EU dioxin exposure and health data [web site]. Brussels, European Commission, 2007 (<http://ec.europa.eu/environment/dioxin/download.htm#CompilationofEUDioxinexposureandhealthdata>, accessed 12 August 2009).
4. Alaluusua S et al. Developmental dental aberrations after the dioxin accident in Seveso. *Environmental Health Perspectives*, 2004, 112:1313–1318.
5. Van Leeuwen FXR, Malisch R. Results of the third round of the WHO-coordinated exposure study on the levels of PCBs, PCDDs and PCDFs in human milk. *Organohalogen Compounds*, 2002, 56:311–316.
6. Lunden A, Noren K. Polychlorinated naphthalenes and other organochloride contaminants in Swedish human milk, 1972–1992. *Archives of Environmental Contamination and Toxicology*, 1998, 34:414–423.
7. Noren K, Meironyte D. Certain organochlorine and organobromine contaminants on Swedish human milk in perspective of past 20–30 years. *Chemosphere*, 2000, 40:1111–1123.
8. Polder A et al. Levels and temporal trends of chlorinated pesticides, polychlorinated biphenyls and brominated flame retardants in individual human breast milk samples from Northern and Southern Norway. *Chemosphere*, 2008, 73:14–23.
9. Lind Y et al. Polybrominated diphenyl ethers in breast milk from Uppsala County, Sweden. *Environmental Research*, 2003, 93:186–194.
10. Thuresson K et al. Apparent half-lives of hepta- to decabrominated diphenyl ethers in human serum as determined in occupationally exposed workers. *Environmental Health Perspectives*, 2006, 114:176–181.
11. Technical Working Group on Integrated Monitoring. SCALE. Baseline report on integrated monitoring of dioxins & PCBs in the Baltic Region. Brussels, European Commission, 2004 (<http://www.environmentandhealth.org/>, accessed 12 August 2009).
12. Miettinen H. The effects of TCDD on the development of teeth and cortical bone in rats: Implications for risk assessment [dissertation]. Helsinki, National Public Health Institute, 2006 (http://www.ktl.fi/attachments/suomi/julkaisut/julkaisusarja_a/2006/2006a10.pdf, accessed 12 August 2009).
13. Vos JG et al. Health effects of endocrine-disrupting chemicals on wildlife, with special reference to the European situation. *Critical Reviews in Toxicology*, 2000, 30:71–133.
14. Pronczuk J, Moy G, Vallenias C. Breast milk: an optimal food. *Environmental Health Perspectives*, 2004, 112(13):A722–723.
15. Mead MN. Contaminants in human milk. Weighing the risks against the benefits of breastfeeding. *Environmental Health Perspectives*, 2008, 116(10):A427–434.
16. Kiviranta H. Exposure and human PCDD/F and PCB body burden in Finland [dissertation]. Helsinki, National Public Health Institute, 2005 (http://www.ktl.fi/attachments/suomi/julkaisut/julkaisusarja_a/2005/2005a14.pdf, accessed 12 August 2009).
17. The Stockholm Convention on Persistent Organic Pollutants (POPs) [web site]. Geneva, United Nations Environment Programme, 2004 (<http://www.pops.int>, accessed 12 August 2009).
18. Global monitoring of persistent organic pollutants (POPs) [web site]. Nairobi, United Nations Environment Programme (<http://www.chem.unep.ch/gmn/default.htm>, accessed 12 August 2009).
19. Biomonitoring of human milk for persistent organic pollutants (POPs) [web site]. Geneva, World Health Organization, 2007 (<http://www.who.int/foodsafety/chem/pops/en/index.html>, accessed 12 August 2009).
20. Regulation (EC) No 850/2004 of the European Parliament and of the Council of 29 April 2004 on persistent organic pollutants and amending Directive 79/117/EEC. Official Journal of the European Union, 2004, L158:7–49.
21. Dioxin exposure and health [web site]. Brussels, European Commission, 2007 (<http://ec.europa.eu/environment/dioxin/index.htm>, accessed on 12 August 2009).
22. Children's Environment and Health Action Plan for Europe. Fourth meeting of the CEHAPE Task Force, Cyprus, 16–17 October 2006. Copenhagen, WHO Regional Office for Europe, 2006 (document EUR/06/5067855/8) (http://www.euro.who.int/Document/EEHC/CEHAPE_Cyprus_8.pdf, accessed 12 August 2009).
23. Planning to protect children against hazards. Copenhagen, WHO Regional Office for Europe, 2005 (<http://www.euro.who.int/document/e88395.pdf>, accessed 12 August 2009).
24. Alaluusua S et al. Polychlorinated dibenzo-p-dioxins and dibenzofurans via mother's milk may cause developmental defects in the child's teeth. *Environmental Toxicology and Pharmacology*, 1996, 1:193–197.
25. Tuomisto JT et al. Soft tissue sarcoma and dioxins – a case control study. *International Journal of Cancer*, 2004, 108:893–900.
26. Turunen AW et al. Mortality in a cohort with high fish consumption. *International Journal of Epidemiology*, 2008, 37:1008–1017.
27. Mocarelli P et al. Paternal concentrations of dioxin and sex ratio of offspring. *Lancet*, 2000, 355:1858–1863.
28. Steenland K et al. Dioxin revisited: developments since the 1997 IARC classification of dioxin as a human carcinogen. *Environmental Health Perspectives*, 2004, 112:1265–1268.
29. Birnbaum LS, Hubal EAC. Polybrominated diphenyl ethers: a case study for using biomonitoring data to address risk assessment questions. *Environmental Health Perspectives*, 2006, 114:1770–1775.
30. WHO Summary Information on Global Health Trends (SIGHT) [web site]. Copenhagen, WHO Regional Office for Europe, 2007 (http://www.euro.who.int/foodsafety/Chemical/20040728_1, accessed 12 August 2009).
31. Van den Berg M et al. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives*, 1998, 106:775–792.

FURTHER INFORMATION

Persistent organic pollutants [web site]. Nairobi, United Nations Environment Programme, 2007 (<http://www.chem.unep.ch/pops/>, accessed 12 August 2009).

International Programme on Chemical Safety [web site]. Geneva, World Health Organization, 2007 (<http://www.who.int/ipcs/en/>, accessed 12 August 2009).

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