### Children's Environmental Health: Intergenerational Equity in Action—A Civil Society Perspective

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Since World War II, approximately 80,000 new commercial synthetic chemicals have been released into the environment, with approximately 1500 new chemicals released annually. Most of these have not been adequately tested for their impacts on human health or their particular impacts on children and the developing fetus. Yet, children are exposed to hazardous chemicals through residues in their food, indoor and outdoor air pollution, and through household products and contaminated house dust. Many of these synthetic chemicals are persistent and bio-accumulative, remaining in the human body long after exposure. Developing fetuses acquire toxic chemicals that have bioaccumulated in the mother's body and readily cross the placental barrier. Babies are now born with many man-made chemicals in their small bodies. Newborns take in more through breast milk or formula. There are no tests to assess the combined impacts of the "chemical soup" to which children are exposed. WHO, UNICEF, and UNEP have reported a growing number of children's health impacts caused by exposure to hazardous chemicals, including asthma, birth defects, hypospadias, behavioral disorders, learning disabilities, autism, cancer, dysfunctional immune systems, neurological impairments, and reproductive disorders. WHO states that approximately 3 million children under the age of five die every year due to environmental hazards, and this is not limited to developing countries. All children, both in the developing and developed world are affected by exposure to hazardous chemicals. In 2004, the European Union's Ministerial Conference on Children's Environmental Health identified air pollution, unsafe water conditions, and lead exposure as the main culprits in the death and disabling of children in Europe. The conference found that by reducing exposure to hazardous chemicals, the lives of many children could be saved. The key issues in children's environmental health and potential policy and management remedies are examined from both national (Australian) and international perspectives.

Key words: children's environmental health; civil society; intergenerational equity

Children are not just little adults. . . they are more vulnerable than adults. They eat more food, drink more water and breathe more air as a percentage of their body weight than adults and as a consequence they are more exposed to the chemicals present in food water and air. Children are growing and developing and may therefore be physiologi-

cally more susceptible than adults to the hazards associated with exposures to chemicals.

—US National Academy of Sciences<sup>1</sup>

Children are not little adults: they have special vulnerabilities to the toxic effects of chemicals. Children's exposure to chemicals at critical stages in their physical and cognitive development may have severe long-term consequences for health. Priority concerns include exposure to air pollutants, pesticides and persistent organic pollutants (POPs),

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lead, mercury, arsenic, mycotoxins and hazardous chemicals in the workplace.

—World Health Organisation (WHO), International Labor Office (ILO), United Nations Environment Program (UNEP)<sup>2</sup>

#### The Problem

Since World War II, approximately 80,000 new synthetic chemicals have been manufactured and released into the environment, with approximately 1500 new chemicals being introduced each year. The vast majority of these have not been adequately tested for their impacts on human health or their particular impacts on children and the developing fetus. Yet, children are exposed to hazardous chemicals through residues in their food, indoor and outdoor air pollution, and through household products and contaminated house dust. Many of these synthetic chemicals are persistent and bio-accumulative, remaining in the human body long after exposure. The developing fetus takes in toxic chemicals that have bio-accumulated in the mother's body and that readily cross over the placental barrier. Babies are now born with many synthetic chemicals already present in their small bodies.<sup>a</sup> Newborns take in more in through breast milk and formula. There are no tests to assess the combined impacts of the chemical soup to which children are exposed.

WHO, the United Nations Children's Fund (UNICEF), and United Nations Environment Project (UNEP) have identified a growing number of children's health impacts from exposure to hazardous chemicals. These include asthma, birth defects, hypospadias, behavioral disorders, learning disabilities, autism, cancer, dysfunctional immune systems, neurological impairments, and reproductive disorders.

WHO has stated that approximately 3 million children under the age of five die every year due to environmental hazards, and this is not limited to developing countries. All children, both in the developing and developed world, are affected by exposure to hazardous chemicals. In 2004, the European Union's Ministerial Conference on Children's Environmental Health identified air pollution, unsafe water conditions, and lead exposure as the main culprits in the death and disabling of children in Europe. The conference found that reducing exposure to hazardous chemicals could save the lives of many children.

## A Child's Unique Vulnerability to Chemicals

The unique vulnerability of children to hazardous chemicals is well recognized by WHO, UNICEF, and UNEP.<sup>3,4</sup> Recent University of California research has shown that newborn children can be much more vulnerable than adults to the commonly used organophosphate pesticides chlorpyrifos (up to 164 times more) and diazinon (up to 65 times more).<sup>5</sup>

Children are not simply little adults. Their bodies are still developing and their detoxification systems are immature. They react to hazardous chemicals differently from adults. They are also more at risk because they have higher respiration and metabolic rates than adults, they eat and drink more per bodyweight, and they live life closer to the ground, crawling, digging in dirt, and putting objects in their mouths. In the debate about the level of risk to children, there is a strong focus on the regulatory decisions about how much dirt a child eats per

<sup>&</sup>lt;sup>a</sup>For example see Environmental Working Group, Body Burden—The Pollution in Newborns; A benchmark investigation of industrial chemicals, pollutants and pesticides in umbilical cord blood, July 14, 2005 Available at http://www.ewg.org/reports/bodyburden2/execsumm.php. In this study spearheaded by the Environmental Working Group (EWG) in collaboration with Commonweal, researchers at two major laboratories found an average of 200 industrial chemicals and pollutants in umbilical cord blood from 10 babies born in August and September of 2004 in U.S. hospitals. Tests revealed a total of 287 chemicals in the group. Also see 'A Present for Life; hazardous chemicals in umbilical cord blood.', a Report compiled for Greenpeace Nederland, Greenpeace International & WWF-UK September 2005(ISBN: 90-73361-87-7.). This study confirms that known or suspected hazardous chemicals from eight chemical groups are commonly present in umbilical cord blood. Available at http://www.panda.org/about\_wwf/what\_we\_do/toxics/publications/ index.cfm

day (pica event). Australia assumes 100 mg of soil ingestion per day, while the U.S. Environmental Protection Agency (USEPA) factors in up to 5 g of soil ingestion. Being unaware of chemical risks, children are less able to protect themselves from exposures, and higher skin absorption rates may also result in a proportionally greater exposure.

Children's detoxification systems and ability to excrete toxins also differs from those of adults. While at times this can offer greater protection, it can also increase vulnerability, for example, where a metabolite is more toxic than the original contaminant. Should the enzyme systems responsible for detoxification be damaged early in life, the result can be a lifetime of disabling chronic illness. The timing of chemical exposures is also significant. Recent research has shown that babies and children experience particular "windows of susceptibility" in their development. If exposures occur during critical times, it may contribute to health problems much later in life; for example, exposure to dioxin in utero can produce disabilities in neurological function and learning ability well into childhood.8-10

Similarly, it has long been known that lead can cause delinquency and reduced IQ.11,12 New evidence links even low levels of lead [that is, the current "acceptable" level of 10 micrograms per deciliter (µg/dL)] with an average loss of 7.4 IQ points by comparison with preschool children whose lifetime average blood-lead concentrations remained at 1 μg/dL.<sup>13</sup> In addition to the known links with hearing loss, poor reading, writing and mathematical ability, reduced lifetime earnings, and reduced growth, balance, and proprioreception (spatial sense of body) problems, and so on forth, childhood lead exposure has also been linked with osteoporosis later in life, and fetal lead exposure is now thought to be a contributing factor of schizophrenia. 14,15

Early exposure to other endocrine disruptors can affect an individual's immune function or ability to reproduce. The U.S. Centers for Disease Control and Prevention (CDC) have reported an increase in the percentage of severe cases of hypospadias. <sup>16</sup> One causal factor being investigated is hormone disruption (in the form of reduced testosterone), caused by synthetic endocrine-disrupting chemicals, at a critical time in fetal development. Studies also suggest that early exposure to carcinogens can increase the risk of developing cancer later in life. <sup>17</sup>

The European Union has launched a new 5-year 15 million euro research project to investigate the connection between childhood cancer and immune disorders and exposure to chemicals in food and the environment. <sup>18</sup> The study will examine maternal exposure during pregnancy to carcinogenic and immunotoxic chemicals and their subsequent effect on young children.

## Is There a Problem for Australian Children?

While there has been a very limited assessment of chemical exposure of Australian children, there is clear evidence of widespread contamination of children in the European Union, United Kingdom, and United States. Childhood cancers are increasing in the developed world, including Australia, where the incidence of asthma is also escalating. Studies in Europe and the United Sstates have identified a wide range of chemicals in umbilical-cord blood as well as in children. 19,20 These include artificial musks, alkylphenols, bisphenol-A, brominated flame retardants, perfluorinated compounds, phthalates, and triclosan. All are found in common products used every day in the home and school; such as cleansers, computers, toys, lotions and perfumes, cookware, clothing, and carpets. Some like the perfluorochemical, perfluorooctanesulfonate (PFOS) and pentabrominated diphenylethers (Penta-BDE) are currently being assessed for inclusion in the Stockholm Convention on Persistent Organic Pollutants (POPs) 2001.

The CDC have been tracking human exposure and released their second National Report

on Human Exposure to Environmental Chemicals in 2003.<sup>21</sup> The report presents exposure data from 1999 to 2000 for 116 chemicals and concluded that some chemicals like the phthalates, are now at levels in the human population at which health impacts would be expected. There is particular concern for babies, children, and women of childbearing age.

While little routine monitoring or testing of baby or children's blood has occurred in Australia, in 1998, doctors at Townsville Hospital tested the meconium (first bowel discharge) of 46 newborn babies and found a wide range of hazardous chemicals, including POPs and pesticides, such as chlorpyrifos. <sup>22</sup> Earlier in the 1990s, an Australian pediatrician concerned with a chronic illness in a group of children tested their blood for levels of POPs, persistent bio-accumulative toxins (PBTs) and other volatile compounds. A range of chemicals was detected in all the children's samples, including POPs pesticies, PCBs, hexachlorobenzene (HCB), benzene, and toluene. <sup>23</sup>

Estimates based on the Human Health Risk Assessment of Dioxins for the Australian National Dioxin Program indicated that breastfed Australian infants are consuming many times the tolerable monthly intake (TMI) for dioxins and furans. In 2002, Australia recommended a TMI for Australians of 70 pg of dioxin toxic equivalent (TEQ) per kilogram of body weight per month.<sup>24</sup> At a crucial time in their development, 3-month old breastfed babies are consuming at least 16 times the TMI of total dioxins.

# Persistent Bio-Accumulative Toxins of Concern

The following PBTs need, as a priority, to be surveyed in Australian umbilical-cord blood, meconium, and breast milk.

• *Dioxins*. By-products of PVC, industrial bleaching, and incineration, can cause cancer and are toxic to the hormone

- system. PCBs once used in industrial insulators, accumulate up the food chain and cause cancer and nervous system problems. Dioxins and PCBs are listed in the international *Stockholm Convention* for reduction and eventual elimination. Australia has released its National Dioxin Plan, which we consider to be very weak on action.
- Brominated Flame Retardants/Polybrominated Diphenyl Ethers (PBDEs). PBDEs are used by the electronics industry and in a wide range of products, including computers, white goods, car interiors, carpets and carpet underlay, and polyurethane foams in furniture and bedding. Some PBDEs have been shown to disrupt thyroid hormones, mimic estrogen, and are linked with cancer and reproductive damage.<sup>25</sup> Deca-BDE has recently been shown to have the potential to break down in the environment and in animals to the smaller, more toxic penta-BDE, which is more bio-accumulative.<sup>26</sup> PBDEs have been found in umbilical-cord blood, breast milk, and breast fat, as well as adult blood and fat.<sup>27–31</sup> A study of PBDEs in Australian adult blood found concentrations higher than those reported from Europe, the United Kingdom, and Japan. 32 Similar high levels of PBDEs were found in Australian breast milk.<sup>33</sup> A Norwegian PBDE study found higher levels in 4-year-olds than in adults.<sup>34</sup> PBDEs have also been detected in house dust from 27 homes at up to 25 parts per million (ppm) due to off-gassing of treated products and furnishings. 35, b Wipe samples from computers universally

bSee also Environmental Working Group (EWG) Report "In the Dust: Toxic Fire Retardants in American Homes" available at http://www.ewg.org/reports/inthedust/. The study found high levels of PBDEs in dust samples taken from houses in the Washington metropolitan area. The levels of the chemical components of deca, the most widely used of the PBDE mixtures, ranged from 160 parts per billion to 8700 ppb. Levels of penta, the second-most widely used mixture, ranged from 200 to 25,000 ppb. The EWG study also found high PBDE levels in dust samples from 10 homes around the country. The average combined levels of deca, penta and octa for nine of the homes was over 4600 ppb.

- contained PBDEs.<sup>36</sup> Penta-BDE has been nominated as a new POP and is currently being assessed by the *Stockholm Convention*'s Persistent Organic Pollutants Review Committee.
- Perfluorochemicals—PFOS/Perfluorooctanoic Acid (PFOA) and their Precursors. PFOS was the active ingredient in Scotchguard Stain Protection and is now used in coatings in photography and microelectronics, and in some specialized fire-fighting foams. PFOA is used in the production of fluoropolymers for nonstick cookware coatings and in the manufacture and treatment of textiles. PFOAs may also form as degradation products of small polymers called telomers used in soil-, stain-, and greaseresistant coatings on carpets, textiles, paper, and leather. PFOS is toxic to the reproductive system, and PFOA is a likely carcinogen. Both persist in the environment forever (terminal product), accumulating in humans and other animals. Many other perfluorochemicals break down to either PFOS or PFOA. The Organisation for Economic Co-operation and Development (OECD) Joint Chemicals Meeting in 2002 reported detection of PFOS in the blood of nearly 600 U.S. children, aged from 2 to 12 years.<sup>37</sup> Recent testing has shown that PFOS and PFOA concentration in the blood of adult Australians is high compared to the findings of studies in other countries.<sup>38</sup> The USEPA review of PFOA used in Teflon manufacture<sup>39</sup> found that it accumulates in the blood system and poses a risk for childbearing women. According to their preliminary risk assessment, the estimated exposure range for humans, based on rat studies, has already overlapped with what the USEPA deems unacceptable for toxic substances. While PFOS is restricted in Australia to essential uses in the firefighting industry, PFOA is subject to only voluntary action. PFOS has been nominated as a new POP, and is currently
- being assessed by the *Stockholm Convention's* Persistent Organic Pollutants Review Committee.
- Metabolites of Organophosphate Pesticides (e.g., chlorpyrifos). Organophosphates are severe neurotoxins and damage the central nervous system. The USEPA review of chlorpyrifos acknowledged that the insecticide and its metabolites had been found in the urine of 89% of children tested in one U.S. study. 40 Dow AgroSciences' 1998 data showed the chlorpyrifos metabolite, TCP-3,5,6-trichloro-2-pyridinol, in 100% of a sample of 416 U.S. children (0–6 years).<sup>41</sup> A 1998 study in regional Australia showed chlorpyrifos was present in the meconium (first bowel discharge) of 59% of newborn babies. 42 Chlorpyrifos is widely used in agriculture and for termite and insect control.
- Phthalates. Phthalates are used as plasticizers or softening agents in vinyl products, including furnishings, floor coverings, medical devices (e.g., catheters, IV, and blood bags), baby feeding bottles, toys, teething rings, food wrap, cosmetics, perfumes, soaps, lotions, and shampoos, and are also added to insecticides and adhesives. Diethylhexyl phthalate (DEHP) has been shown to migrate into food from certain food wraps during storage. Some phthalates are hormone disruptors, immunotoxins, cancer promoters, and are reproductive and developmental toxins. 43–45 DEHP has been classified as a probable human carcinogen by the USEPA. Phthalates have been detected in the blood and urine of children in the United States and the European Union. The presence of phthalates in children's toys, teethers, and in dust may indicate that children are at particular risk. The U.S. National Toxicology Program (NTP) has expressed concern over the potential adverse development of babies born to pregnant women who are exposed to DEHP, the most widely

- used phthalate plasticizer, at the normal levels estimated for an adult.<sup>21</sup> In 2006, Australia's National Industrial Chemicals Notification and Assessment Scheme (NICNAS) declared 11 phthalates as priority existing chemicals.<sup>46</sup>
- Artificial Musks. Nitromusks (musk xylene, musk ketone) and polycyclic musks [tonalide (AHTN), and galaxolide (HHCB)] are used to replace natural aromas in products like washing agents, soap, and cosmetics They are found in breast milk, blood, and fat, and can induce enzymes and disrupt hormones. They are linked to hormonal and gynecological problems in women. Musk ambrette, banned in EU cosmetics since 1995, has recently been found in EU maternal blood and cord-blood samples.<sup>47</sup>
- Alkylphenols (APs). Nonylphenols (NPs), octylphenols (OPs), and nonylphenol ethoxylates (NPEs) are used in plastics, industrial detergents and emulsifiers, and textile and carpet cleaning. Most can degrade back to alkylphenols, which are persistent and bio-accumulative, and have been found in umbilical-cord blood and breast milk. NPs have also been found in foods, rainwater, and house dust. Alkylphenols can mimic estrogen hormones and in test animals, and can alter sexual development in fish and sperm quality in mice.<sup>47</sup>
- Triclosan (5-chloro-2-(2,4-dichlorophenoxy)phenol). Triclosan is used in toothpastes, acne creams, deodorants, lotions, and hand soaps, and is incorporated into a wide range of consumer goods, including kitchen tiles, children's toys, cutting boards, toothbrush handles, hot tubs, and athletic clothing. Triclosan is linked to skin irritation (photoallergic contact dermatitis), allergy susceptibility, and effects on thyroid hormone metabolism. Triclosan is weakly androgenic, causing changes in sex ratios in fish. It bio-accumulates in fatty tissue and has been found in Swedish sam-

- ples of human breast milk. New research shows triclosan can react with chlorinated water to produce carcinogenic chloroform and dioxins. <sup>48</sup> Triclosan was found in 50% of umbilical-cord blood samples. In Australia NICNAS declared triclosan a priority existing chemical in 2003 and in 2006 initiated a breast-milk study of Australian mothers. <sup>49,50</sup>
- Organochlorine Pesticides (OCPs). OCPs include the POPs: DDT, dieldrin, aldrin, endrin, heptachlor, chlordane, and mirex (which until recently was still used in the Northern Territory of Australia). OCPs accumulate up food chains, and can cause cancer and reproductive effects. All are now banned in Australia.
- Volatile and Semivolatile Organic Chemicals (VOCs). VOCs are industrial solvents found in petrol, paints, and household products. Many affect the central nervous system and cause skin and respiratory irritation; some, like benzene, are carcinogenic.
- Metals. Metals, such as lead, organomercury, organotin from industrial emissions, food residues, lead in paint, and leaded petrol, can cause mental retardation and learning disabilities

### **International Programs**

WHO has been working on a children's environmental health since 1999 when it established the Taskforce for the Protection of Children's Environmental Health. In 2002, WHO launched its Healthy Environments for Children Alliance (HECA) at the Johannesburg World Summit for Sustainable Development. HECA has developed the HECA Framework for Action for global action to protect children's environmental health.

UNICEF is a member of HECA, and through its program on water, environment, and sanitation, attempts to protect the environment for children's health.

UNEP has also been active in children's environmental health issues, working closely with UNICEF and WHO. UNEP is a core member of HECA and also serves as the Secretariat for many chemical conventions. In this role, it has been able to work with governments to help ensure that children's environmental health issues are addressed in the development of international agreements on chemical management. In the Stockholm Convention on Persistent Organic *Pollutants 2001*, children and their specific needs are referenced; for example, in Article 7 on implementation, parties are obliged to consult their national stakeholders, including women's groups and groups involved in the health of children.

Protecting children was also a focus of the recently developed Strategic Approach to International Chemical Management (SAICM). The High Level Declaration adopted at the International Conference on Chemical Management in February 2006 states that the signatories "are determined to protect children and the unborn child from chemical exposures that impair their future lives." The Over-Arching Policy Strategy's Statement of Need acknowledged that "risk reduction measures need to be improved to prevent the adverse effects of chemicals on the health of children, pregnant women, fertile populations, the elderly, the poor, workers and other vulnerable groups and susceptible environments." Unfortunately, some of the activities associated with the SAICM Global Plan of Action relating to the Section on Children and Chemical Safety were blocked by the United States and a small number of other countries. Of particular concern were the activities regarding the chemical composition of children's products and toys. International actions to stop manufacturers "marketing products containing substances that have or may have adverse effects on children's health," for example, phthalates of concern and certain fragrances, were blocked.

Since 2000, several international reports on children's environmental health have been prepared, including a review of children's health and environment, undertaken by the WHO, the European Environment Agency, UNEP, UNICEF, and the Intergovernmental Forum on Chemical Safety (IFCS).<sup>3,51,52</sup>

In 2003, the fourth session of the IFCS held in Thailand and attended by 126 governments, agreed on a range of actions and recommendations to protect children from chemical exposure.<sup>53</sup> These included:

- The assessment of chemical exposures during preconception, throughout gestation, infancy, childhood, and adolescence;
- Government-initiated multistakeholder consultation in national assessments of children's environmental health and chemical safety to identify priority concerns and provide a basis for developing action plans to address risks. Governments should provide a progress report to Forum V in 2006:
- Governments, WHO, and UNICEF promotion of education and training on children's chemical safety, and where risks are identified, governments and stakeholders should commit to taking action to prevent or reduce exposure;
- Harmonize data collection, research, legislation and regulations, and consideration of indicators of children's environmental health, and report back to Forum V.

Most importantly, governments should take into consideration the potential enhanced exposures and/or vulnerabilities of children when setting acceptable levels or criteria related to chemicals.

Many nongovernment organizations (NGOs) also have children's environmental health initiatives. The International Network for Children's Health, Environment and Safety (INCHES) is an international forum focused on children's environmental health. It aims to increase understanding of how environmental factors influence child health, to facilitate information exchange on best practices and policies in children's environmental health, to stimulate new research, and to advocate

for children's environmental health in the intergovernmental arena. INCHES won a USEPA 2006 Children's Environmental Health Recognition Award for its development of training material on children's environmental health for different target groups, including public health and pediatrics professionals.

### **Legislating for Children**

Few countries have legislated to protect children's environmental health, although a number like Canada and the European Union are investigating new options. However, the United States has enacted legislation, The Children's Environment Protection Act 1997, that aims to protect children from exposure to environmental pollutants. The act requires USEPA standards to be set at levels that protect children and other vulnerable groups, including the elderly, pregnant women, and people with serious problems. Children are defined as 18 years of age and under.

The legislation requires the USEPA to consider all environmental health risks to vulnerable subpopulations in risk assessments, environmental and public health standards, and regulatory decisions. The act also requires the development of a list of USEPA-recommended "safer for children" products and chemicals that minimize potential risks to children. The agency has set up a Specialist Office of Children's Health Protection to set health standards to ensure the protection of children and vulnerable subpopulations. The statutory Children's Health Protection Advisory Committee has the making of annual recommendations to the USEPA on standards that need reevaluation as one of its objectives.

A number of state acts support these moves; for example, the Washington State Children's Pesticide Right-to-Know Act (SSB 5533). This act requires that school districts post notices warning students and staff whenever pesticides are used in and around schools, and provide advance notification to interested parents.

Regulations under the U.S. Residential Lead-Based Paint Hazard Reduction Act of 1992—Title X, require the disclosure of lead-based paint hazards in target housing that is offered for sale or lease. The preamble to the act states that "low-level lead poisoning is widespread among American children, afflicting as many as 3,000,000 children under age 6" and "the health and development of children living in as many as 3,800,000 American homes is endangered by chipping or peeling lead paint, or excessive amounts of lead-contaminated dust in their homes."

## Next Steps in Protecting Children's Environmental Health in Australia

PBTs represent a real and urgent threat to children's environmental health and our obligations to intergenerational equity. In the face of the current chemical body load of children in the developed world, urgent and precautionary responses are required from government, industry, and the community. The National Toxics Network has recommended the following actions for Australia.

#### Recommendations for Action

- Establishment of a national specialist office for children's environmental health;
- Establishment of national and state child environment protection acts;
- Priority review of all uses of PBTs, including perfluorochemicals, brominated flame retardants, phthalates, and metals, to identify both their intergenerational impacts and appropriate regulatory responses;
- An immediate ban of penta- and octa-BDE, with an accompanying phase out of deca-BDE over 2 years;
- An immediate ban on PFOS, PFOAs, and their precursors;
- Establish ongoing biomonitoring of PBTs in children's blood and urine, in breast

- milk, and in infant meconium and cord blood;
- Phase out chlorpyrifos and other organophosphates detected in children's meconium;
- Introduce legislation to warn home buyers and potential tenants of asbestos and lead hazards in houses prior to sale or rent;
- Set a new target for blood-lead levels below 10 µg/dL before 2010, and carry out an initial national blood-lead survey (all ages) by 2005 to determine the baseline and set priorities for achievement of the new target.

#### **Conflicts of Interest**

The authors declare no conflicts of interest.

#### References

- U.S. National Academy of Sciences. 2004. Putting Research to the Test: Improvements Needed in Clinical Studies Involving Children. National Academies InFocus, Vol. 4 No. 2. Available at http://infocusmagazine.org/4.2/soc\_children.html.
- 2. WHO, ILO & UNEP. 2006. Helping to Protect Children from the Harmful Effects of Chemicals. International Program on Chemical Safety. Available at http://www.who.int/ipcs/en/.
- UNEP, UNICEF & WHO. 2002. Children in the New Millennium: Environmental Impact on Health. Available at www.unep.org, www.unicef.org and www.who.int.
- IFCS Children and Chemical Safety Working Group. 2005. Chemical Safety and Children's Health: Protecting the World's Children from Harmful Chemical Exposures—A Global Guide to Resources, October.
- Furlong, C.E. et al. 2006. PON1 status of farmworker mothers and children as a predictor of organophosphate sensitivity. Pharmacogenet. Genomics 16: 183–190.
- Landrigan, P.J. et al. 1998. Children's health and the environment: a new agenda for prevention research. Environ. Health Perspect. 106(Suppl. 3): 787–794.
- Olin, S.R. & B.R. Sonawane. 2003. Workshop to develop a framework for assessing risks to children from exposure to environmental agents, September 2003. Environ. Health Perspect. 111/12: 1524–1526.

- Pluim, H.J. et al. 1994. Clinical laboratory manifestations of exposure to background levels of dioxins in the perinatal period. Acta Paediatr. 83: 583–587.
- Ilsen A. et al. 1996. Signs of enhanced neuromotor maturation in children due to perinatal load with background levels of dioxins. Chemosphere 33: 1317– 1326.
- Weisglas-Kuperus, N. et al. 1995. Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. Pediatr. Res. 38: 404

  410.
- Needleman, H.L. et al. 1989. The long-term effects of exposure to low doses of lead in childhood—An 11-year follow-up report. N. Engl. J. Med. 322.
- Needleman, H.L. et al. 1996. Bone lead levels and delinquent behavior. JAMA 275.
- Canfield, R.L. et al. 2003. Intellectual impairment in children with blood lead concentrations below 5 micrograms per deciliter. N. Engl. J. Med. 348: 1527– 1536.
- Campbell, J.R., R.N. Rosier, L. Novotny & J.E. Puzas. 2004. The association between environmental lead exposure and bone density in children. *Environ. Health Perspect.* 112.
- Opler, M.G.A. et al. 2004. Prenatal lead exposure, delta-aminolevulinic acid, and schizophrenia. Environ. Health Perspect. 112: 548–552.
- Centers for Disease Control and Prevention. 1997.
   Hypospadias Trends in Two US Survelliance Systems. Press Release, November 6.
- Barton, H.A. et al. 2005. Assessing susceptibility from early-life exposure to carcinogens. Environ. Health Perspect. 13: 1125–1133.
- Euractiv. 2006. Press Release, EU Research to Look into Chemical Exposure of Babies. Press Release February 24. 2006. Available at http:// www.euractiv.com/Article?tcmuri=tcm:29-152907-16&type=New.
- Environmental Working Group, Body Burden. 2005.
   The Pollution in Newborns; A Benchmark Investigation of Industrial Chemicals, Pollutants and Pesticides in Umbilical Cord Blood. July 14. Available at http://www.ewg.org/reports/bodyburden2/execsumm.php.
- Greenpeace Nederland, Greenpeace International & WWF-UK. 2005. A Present for Life; Hazardous Chemicals in Umbilical Cord Blood. Available at http://www.panda.org/about\_wwf/what\_we\_do/toxics/publications/index.cf.
- Department of Health and Human Services, Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals.
- 22. Deuble, L. *et al.* 2000. Environmental pollutants in meconium in Townsville. Australia. Unpublished.

- Budd, L.E. 1993. Children's Health & Chemicals, One Paediatrician's Experience. Unpublished Lecture Notes.
- 24. National Health and Medical Research Council & Therapeutic Goods Administration. 2002. Dioxins: Recommendation for a Tolerable Monthly Intake for Australians.
- UNEP Chemicals. 2002. Fire Retardant, Polybrominated diphenyl ethers (PBDEs): Regional Reports of the Regionally-Based Assessment of Persistent Toxic Substances Program. Available from http://www.chem.unep.ch/pts.
- Stapleton, H. et al. 2004. Debromination of the flame retardant decabromodiphenyl ether by juvenile carp (Cyprinus carpio) following dietary exposure. Environ. Sci. Technol. 38: 112–119.
- Choi, J.W. et al. 2003. Polybrominated dibenzo-pdioxins, dibenzofurans, diphenyl ethers in Japanese human adipose tissue. Environ. Sci. Technol. 37: 817– 821.
- 28. Guvenius, D.M. *et al.* 2003. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols and pentachlorophenol. *Environ. Health Perspect.* **111:** 1235–1241.
- Hardell, L. et al. 1998. Concentrations of the flame retardant 2,2'4,4-tetrabrominated diphenyl ether in human adipose tissue in Swedish persons and the risk for non-Hodgkin's lymphoma. Oncol. Res. 10: 429–432.
- Mazdai, A. et al. 2003. Polybrominated diphenyl ethers in maternal and fetal blood samples. Environ. Health Perspect. 111: 1249-1252.
- She, J. et al. 2002. PBDEs in San Francisco Bay area: measurements in harbor seals blubber and human breast adipose tissue. Chemosphere 46: 697–707.
- Harden, F., L. Toms, J. J. Ryan & J. Müller. Determination of the Levels of Polybrominated Diphenylethers (PBDEs) in Pooled Blood Sera Obtained from Australians Aged 31–45 Years. National Research Centre for Environmental Toxicology, University of Queensland. Brisbane, Australia.
- 33. Harden, F., J. Muller & L. Toms. 2005. Organochlorine Pesticides (OCPs) and Polybrominated Diphenyl Ethers (PBDEs) in the Australian Population: Levels in Human Milk. Report for Environment Protection and Heritage Council. Available at www. ephc.gov.au/ephc/ocp\_pbde\_human\_milk.html.
- 34. Thomsen, C., E. Lundanes & G. Becher. 2002. Brominated flame retardants in archived serum samples from Norway: a study on temporal trends and the role of age. *Environ. Sci. Technol.* **6:** 1414–1418.
- 35. Stapleton, H.M., M. Schantz & S. Wise. 2004. Polybrominated Diphenyl Ether Measurements in

- Household Dust. Presentation to the Third International Workshop on BFRs, Univ. of Toronto, Canada, June 6–9.
- 36. McPherson, A., B. Thorpe & A. Blake. 2004. Brominated Flame Retardants in Dust on Computers; The Case For Safer Chemicals and Better Computer Design. Available at www.computertakeback.org.
- USEPA, Office of Pollution Prevention and Toxics Risk Assessment Division. 2002. Revised Draft Hazard Assessment of Perfluorooctanoic Acid and its Salts. November 4.
- Kärrman, A. et al. 2005. Perfluorinated compounds in serum from Australian urban and rural regions. EMG—Fluorinated Compounds, pp. 780–783.
- 39. USEPA, Office of Pollution Prevention and Toxics Risk Assessment Division. 2003. Preliminary Risk Assessment of the Developmental Toxicity Associated with Exposure to Perfluorooctanoic Acid and its Salts. April 10.
- Spitzer, E. 1999. Comments of New York State Attorney in re: United States Environmental Protection Agency's Preliminary Risk Assessment for Chlorpyrifos Reregistration Eligibility Decision. Docket Control Number 0PP-34203. December 27, amended January 3, 2000.
- 41. Tarplee, B., Executive Secretary, Food Quality Protection Act Safety Factor Committee Health Effects Division. 2000. Memorandum, Subject: Chlorpyrifos—Re-evaluation Report of the FQPA Safety Factor, HED DOC. NO. 014077. April 4.
- 42. Deuble, L. *et al.* 1999. Environmental pollutants in meconium in Townsville. Unpublished.
- 43. Lovekamp, T.N. & B. J. Davis. 2001. Mono-(2-ethylhexyl) phthalate suppresses aromatase transcript levels and estradiol in cultured rat granulose cells. *Toxicol. Appl. Pharmacol.* 172: 217–224.
- Nencioni, A., S. Wesselborg & P. Brossart. 2003.
   Role of peroxisome proliferators-activiated receptor gamma & its ligands in the control of immune responses. Crit. Rev. Immunol. 23: 1–13.
- 45. Sharpe, R.M. & D.S. Irvine. 2004. How strong is the evidence of a link between environmental chemical and adverse effects on human reproductive health? *Br. Med. J.* **328**: 447–451.
- NICNAS. 2006. Applicants for PEC Assessment of Phthalates. Available at http://www.nicnas.gov.au/Industry/Existing\_Chemicals/PEC\_Declarations/Phthalates\_List\_of\_Applicants\_PDF.pdf.
- Greenpeace Nederland, Greenpeace International
   WWF-UK. 2005. A Present for Life: Hazardous
   Chemicals in Umbilical Cord Blood. Available

- at http://www.panda.org/about\_wwf/what\_we\_do/toxics/publications/index.cfm.
- Rule, K.L., V.R. Ebbett & P.J. Vikesland. 2005. Formation of chloroform and chlorinated organics by free-chlorine-mediated oxidation of triclosan. *Environ. Sci. Technol.* 39: 3176– 3185.
- NICNAS. 2003. Declaration of Triclosan as a Priority Existing Chemical. Available at http://www.nicnas.gov.au/Publications/Chemical\_ Gazette/pdf/2003may\_whole.pdf#page=43.
- NICNAS. 2006. Update on the Assessment of Priority Existing Chemical Triclosan. Available at http://

- www.nicnas.gov.au/Industry/Existing\_Chemicals/ PEC\_Declarations/updatetriclosan072006.pdf.
- European Environment Agency & WHO Regional Office for Europe. 2002. Children's Health and Environment. A Review of Evidence. Environmental Issue Report No. 29.
- World Bank. 2002. Toxics and Poverty: The Impact of Toxic Substances on the Poor in Developing Countries.
- Intergovernmental Forum on Chemical Safety. 2003.
   Forum IV Final Report (IFCS/FORUM-IV/16w)
   Executive Summary: Children and Chemical Safety.