Children's Environmental Health - Intergenerational Equity in action

According to the US National Academy of Sciences, *"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 physiologically more susceptible than adults to the hazards associated with exposures to chemicals"*

The 1989 Convention on the Rights of the Child

The 1989 Convention on the Rights of the Child (CRC) enshrines a child's inherent right to life (Article 6), the right of the child to health (Article 24) and refers to the adequate standard of living (Article 27). It is recognised that all of these rights depend on an environment free from pollution. In the 1994, the United Nations Draft Declaration on Human Rights and the Environment Special Rapporteur Ksentini recognised that the rights accorded to children by the CRC all entail environmental concerns.[1]

The European Court of Human Rights in the Lopez Ostra case[2] also interpreted the 1950 Convention on Human Rights in an environmental manner, particularly where the environmental rights of the child were involved. The court recognised that severe environmental pollution even without causing serious damage to health, can result in impacts on an individual's well being and impedes enjoyment of their homes in such a manner as to affect their private and family life adversely.

International programs

Globally, there are now a plethora of policies and programs attempting to address the needs of children's environmental health. The UNICEF Berlin Commitment of 2002[3] outlined goals and focused on the development of children's policies in fifteen European countries over the following decade. It recognised that children are:

- citizens in their own right;
- must be consulted and involved in issues that concern them; and that
- investing in their development is the key to building a peaceful and prosperous world.

The Berlin Commitment focused on the importance to

- adjust national legislation;
- provide necessary funding;
- monitor progress; and
- take other action to realize the rights of children.

The June 2004, the Fourth Ministerial Conference on Environment and Health 'The Future of Our Children' in Budapest considered the European Union (EU) and World Health Organisation's (WHO) policy on children and the environment and health. The Conference aimed for a 'children and the environment' action plan for Europe. They concluded that lead, indoor air pollution and unsafe water are some of the major threats to children.

In the same month, the Final Communication from the European Commission to the European Council, European Parliament and the European Economic and Social Committee stated "that environmental effects on vulnerable groups are of particular concern." Their strategy provides special evidence that children's exposure and susceptibility are greater than those of adults. They identified the challenge to put into practice the commitments regarding children's rights to grow and live in a healthy environment. Similar commitments have been made in the Convention on the Rights of the Child and the World Summit on Sustainable Development.

The European Environment Commissioner, Margot Wallstrom in her forward to the joint WHO/European Environment Agency Review 'Children's Health and Environment'[4] stated that "children's health and environment need to be high on the political agenda. It is not possible to talk about health and quality of life without taking into consideration and paying special attention to the needs of children. We must never forget that a **healthy environment is not a privilege but a basic human right, not least for our children."**

Other international initiatives[5] have been undertaken by United Nations Environment Program, the World Health Organisation, World Bank and the International Society of Doctors for the Environment. The 2003, Bangkok meeting of the Intergovernmental Forum on Chemical Safety (Nov. 2003) focused on protecting children from harmful chemicals and reaffirmed that children are not little adults.

Legislating for Children

The United States has implemented far reaching legislation to protect the environmental health of children. *The Children's Environment Protection Act 1997* aims to protect children from exposure to environmental pollutants. The Act requires that as national policy all USEPA standards be set at levels that protect children and other vulnerable groups including the elderly, pregnant women, people with serious problems and others. Children are defined as 18 years of age and under.

The legislation requires the USEPA to consider all environmental health risks to vulnerable sub populations in all risk assessments, risk characterizations environmental and public health standards and regulatory decisions undertaken by the Agency. The Act also requires the development of a list of USEPA recommended 'safer for children' products and chemicals that minimize potential risks to children.

It also requires the USEPA to conduct research on the health effects of exposure of children to environmental pollutants and to create a family "right to know" information kit that includes practical suggestions on how parents may reduce their children's exposure to environmental pollutants.

The USEPA has set up a Specialist Office of Children's Health Protection in order to set health standards to ensure the protection of children and vulnerable sub populations. The statutory Children's Health Protection Advisory Committee has as one of its objectives the making of annual recommendations to the USEPA on standards that need re-evaluation.

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.

Similarly, regulations under the Residential Lead-Based Paint Hazard Reduction Act of 1992--Title X, require the disclosure of lead-based paint hazards in target housing which is offered for sale or lease and government grants to ensure widespread awareness of the need to employ licensed lead-trained contractors for renovation of these homes. 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."

Children's unique vulnerability to chemicals

The unique vulnerability of children to hazardous chemicals is well recognised by the United Nations and the World Health Organisations.

A number of factors contribute to the unique vulnerability of children to some chemicals.[6] They include a child's individual biology, physiology and behaviour. Most systems of a foetus or child's body are immature and constantly developing. Children have a higher respiration and metabolic rate than adults. They eat and drink more per bodyweight than adults and their skin absorption may be higher. The higher skin absorption rate can result in a proportionally greater chemical dose.

Children live life closer to the ground where contaminants often accumulate and have a lot of hands to mouth activity as part of their normal development. Being unaware of the risks, they are less able to protect themselves from exposures. 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 day (‘pica event’). Australia assumes 100 milligrams per day while the USEPA factors in a much larger soil ingestion of up to 5 grams.

Depending on the state of development and a variety of other factors, a child's detoxification systems and ability to excrete toxins differs from adults. While at times this can offer greater protection it can also increase vulnerability. 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. Some studies suggest that early exposure to carcinogens can increase the risk of developing cancer if exposures begin early in childhood. Exposure to some chemicals and heavy metals in utero and early childhood can produce life long disabilities in neurological function and learning ability.[7] It has long been known that lead can cause delinquency and reduced IQ.[8] New evidence links even low levels of lead (that is, the current "acceptable" level of 10 micrograms per decilitre) with an average loss of 7.4 IQ points by comparison with pre-school children whose lifetime average blood lead concentrations remained at 1 microgram per decilitre.[9] In addition to the known links with hearing loss, poor reading, writing and maths ability, reduced life-time earnings and reduced growth, balance and proprioreception (spatial sense of body) problems etc, childhood lead exposure has also been linked with osteoporosis later in life,[10] and foetal lead exposure is now thought to be a contributing factor of schizophrenia.[11]

Other substances can affect the endocrine system and result in developmental problems. For example, the US Centers for Disease Control and Prevention has reported an increase in the percentage of severe cases of hypospadias.[12] 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 the foetus's development.

In 2002 the WHO, UNICEF and the United Nations Environment Program undertook a review of environmental impacts on children.[13] They identified a growing body of epidemiological research and studies of laboratory animals, which suggest the possible link of long term exposure to children from certain pesticides. These include:

- abnormal growth and development, and failure to acquire normal organ function;
- endocrine/hormone disruption: certain pesticides in very small doses may mimic or block hormones or trigger inappropriate hormone activity, which can cause, for example, sterility, lowered sperm counts and breast cancer;
- impaired development of the nervous system that can result in lowered intelligence and behavioural abnormalities;
- cancers, including leukaemia, sarcoma, lymphoma, Wilm's (malignant tumour of the kidney) and brain cancer in children. Studies have indicated that the risk of developing cancer might be higher if exposure to carcinogens begins in childhood; and
- compromised immune system, which in children further exacerbates the risk of infectious disease and cancer, thus increasing mortality rates. This is of special concern as children are simultaneously exposed to both pesticides and infectious pathogens when their immune systems are already compromised by other factors.

Is there a problem for children?

While, internationally, there is growing evidence of widespread contamination of children with persistent bio-accumulative toxics (PBTs), there is little ongoing biomonitoring involving children. Persistent bioaccumulative toxic substances that make up the body burden of chemicals in the developed world include:

- **Dioxins** – by-products of PVC production, industrial bleaching, and incineration. which is listed in the international Stockholm Convention on Persistent Organic Pollutants as the major industrial source of dioxins and furans

- **Brominated Flame Retardants -** used in a wide range of products including plastics for computer casings, white goods, car interiors, carpets and carpet underlay, polyurethane foams in furniture and bedding.

- **Perfluorochemicals -** perfluorooctanoic acids (PFOA) used in the manufacture of clothing, cosmetics and in the production of fluoropolymers for non-stick coatings for cookware e.g. teflon. PFOAs may also form as degradation products of small polymers called Telomers used in fire fighting foams, and soil, stain and grease resistant coatings on carpets, textiles, paper, and leather.

- **Organochlorine pesticides -** include the persistent organic pollutants DDT, dieldrin, heptachlor, chlordane and mirex.

- **Organophosphate insecticide metabolites-** breakdown products of organophosphate pesticides such as, chlorpyrifos.

- **Phthalates** – used as plasticizers and in personal products, such as perfumes, lotions, babies teething rings and tubing used in hospitals to deliver medications, nutrients etc

- **Metals** - lead, organomercury, organotin

PBTs are stored in the blood and fat; and for women in their breastmilk, while lead is stored in every organ but mainly in bone. The US based Centers for Disease Control and Prevention have been tracking human exposure and recently released their second National Report on Human Exposure to Environmental Chemicals.[14] The report presents exposure data from 1999-2000 for 116 chemicals and concluded that some chemicals in our bodies, for example the phthalates, are now at levels at which you would expect health impacts. There is particular concern for children and women of childbearing age.

In a 2003 study led by Mount Sinai School of Medicine in New York, researchers at two major laboratories tested nine volunteers who did not work with chemicals on the job nor live near an industrial facility.[15] They found an average of 91 industrial compounds, pollutants, and other chemicals in their blood and urine. This contamination is referred to as a person's 'body burden'.

A total of 167 chemicals were found in the group, of which;

- 76 cause cancer in humans or animals,
- 94 are toxic to the brain and nervous system, and
- 79 cause birth defects or abnormal development.

Importantly, the dangers of exposure to these chemicals in combination have never been studied.

In 1990s, in Australia paediatricians concerned with a chronic illness in a group of children tested their blood for levels of persistent organic pollutants (POPs), PBTs and other volatile compounds. A range of chemicals was detected in all samples taken from the children surveyed. They included POPs pesticides, PCBs, HCB, benzene and toluene. [16] In 1998, doctors at Townsville Hospital tested the meconium (first bowel discharge) of 46 newborn babies (first bowel discharge) and found a wide range of hazardous chemicals including POPs and pesticides such as chlorpyrifos.[17]

Persistent Bioaccumulative Toxins of Concern for Children

Brominated Flame Retardants

An emerging issue are the polybrominated diphenylethers (PBDEs) a type of brominated flame retardants (BFRs). PBDEs have been shown to disrupt thyroid hormones, mimic oestrogen, and are linked with cancer and reproductive damage.[18]

The PBDEs are used by the electronics industry and include the penta-BDE, octa-BDE, and deca-BDE. Deca-BDE is the most commonly used in electronic products. 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 that is more bioaccumulative in the environment. [19]

In June 2004 the flame retardant, deca-BDE, previously claimed not to bioaccumulate, was found in Arctic animals, providing further evidence of the persistency of BFRs and their ability to travel far from their original source.[20] PBDEs have now been found in umbilical cord blood, breast milk, breast fat, as well as adult blood and fat.[21] This year, the US government's National Institute of Standards and Technology has found high levels of PBDEs in dust from 27 homes.[22] The levels of the chemical components of deca, the most widely used of the PBDE mixtures, ranged from 160 parts per billion to 8,700 ppb. Levels of penta, the second-most widely used

mixture, ranged from 200 to 25,000 ppb. The source of the contamination is likely to be the off gassing of treated domestic products and furnishings, a process referred to as 'blooming'. The report, Brominated Flame Retardants In Dust on Computers released in June 2004, reports on 'wipe samples' taken from computers which found toxic PBDE residues in every sample.[23]

These studies raise new concerns about potential health risks to young children. Toddlers who crawl on the floor and have a lot of hand to mouth activity, generally have a far greater exposure to household dust than adults. Research indicates that PBDEs can effect a child's development and may also place them at greater risk of cancer.

Our vulnerable children are at risk while still in the womb as exposure to PBDEs can occur through maternal blood and through the amniotic fluid. Most worrying, is a Norwegian study of PBDEs which has found higher levels in 4-year-olds than in adults.[24]

While PBDEs are not manufactured in all countries, they may be imported as chemicals or as part of a mixture and come in on finished products. National regulators have no reliable estimates of quantities of BFRs in their country and all face the ultimate disposal of contaminated articles including the growing quantities of 'technotrash' (computer waste, white goods waste, etc). When electronic wastes are incinerated or combusted they can form the highly toxic brominated dioxins.

The World Health Organization's International Program on Chemical Safety has concluded that (brominated flame retardants are significant sources of polybrominated dioxins and furans. Their report concluded that they should not be used where suitable replacements are available and all future if it should be encouraged to develop further substitutes. [25] Both the European Union and California have now moved to ban at least two BFRs; penta-BDE and octa-BDE.

Perfluorochemicals

Perfluorooctane sulfonate (PFOS) is a fully fluorinated organic compound and a member of a large family of perfluoroalkyl sulfonate based chemicals. In 2000, the 3M Corporation had notified the USEPA of internal scientific studies on PFOS, including one that resulted in the deaths of all the rat offspring within four days.[26] In response to these adverse findings, 3M withdrew its stain repellent Scotchgard from the marketplace and has since stopped manufacturing and marketing all perfluorochemicals. However, there are other manufacturers who continue the production of PFOS.

Other perfluorochemicals such as perfluorooctanoic acids (PFOAs) are used in the manufacture of clothing, cosmetics and in the production of fluoropolymers for non-stick coatings for cookware. PFOAs may also form as degradation products of small polymers called telomers. These polymers are used in a range of commercial products including fire fighting foams, as well as soil, stain and grease resistant coatings on carpets, textiles, paper, and leather. Fluorotelomer alcohols are also found in a wide range of household and consumer products like hair shampoo, rug cleaners, and food paper products. These are volatile and can be carried long distances with air currents.

All perfluorochemicals have the potential to degrade back to PFOS, which does not appear to degrade further. PFOS is highly bioaccumulative[27] and has been shown to cause cancer, liver damage and development and reproductive effects.[28]

Once released into the environment, perfluorochemicals persist and are now widely distributed throughout the global environment, found in food samples and have been detected in almost all

human blood samples tested. The American Red Cross has already expressed concern about contamination of the blood supply with PFOS.[29] In 3M studies released in 2001, PFOAs were found in green beans, bread and ground beef samples.

PFOS was detected in the blood of nearly 600 US children, aged from 2-12 years, reported to the OECD Joint Chemicals Meeting in Paris in November 2002.[30] .

The USEPA has completed a preliminary review of the chemical, C8, a perfluorooctanoic acid (PFOA) used in Teflon manufacture.[31] The review found that C8 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 deem as unacceptable for toxic substances.

In Australia, where there has been a voluntary phase out agreement for PFOS since 2000, the national regulator, the National Industrial Chemical Notification and Assessment Scheme, (NICNAS) recommends[32] that PFOS users exercise caution in selecting PFOA as an alternative, as PFOA may have the same environmental and health concerns as PFOS.

Chlorpyrifos

Chlorpyrifos (0,0-diethyl O-(3,5,6-trichloro-2-pyridyl) phosphorothioate) is used as a broad spectrum insecticide both in agriculture and domestic pest control. Chlorpyrifos is a chlorinated organophosphorus insecticide dating from the mid 1960s and is now the active ingredient in many agricultural and domestic products; for example, 164 products are registered in Australia, with an estimated current annual consumption of 1,000 tonnes.[33]

In 2000, the USEPA entered an agreement with Dow Agro-Sciences to withdraw the domestic use of chlorpyrifos in homes, hospitals and preschools as well as severely restricting the crops on which it may be used in the United States. Other regulatory authorities have decided not to follow the USEPA example citing differences in risk assessment uncertainty/safety factors.

The USEPA review of chlorpyrifos acknowledged that the insecticide and its breakdown products had been found in the urine of 89% of children tested in one US study.[34] In fact, the chemical manufacturer's (Dow AgroSciences) own data showed that the breakdown products of chlorpyrifos (TCP-3,5,6-trichloro-2-pyridinol) had been detected in 100% of a sample of 416 children tested in the USA in 1998, aged from 0-6 years.[35]

Analysis of pesticide-related data collected by the Centers for Disease Control and Prevention (CDC) in a study of chemicals in 9,282 people, found pesticides in 100% of the people who had both blood and urine tested. The CDC data showed that the average 6 to 11 year-old sampled is exposed to chlorpyrifos at four times the level USEPA considers "acceptable" for a long-term exposure. This was despite the restrictions introduced in 2000.

In 1998, a study in regional Australia had shown chlorpyrifos was present in the meconium (first bowel discharge) of new-born babies. Nearly 60% of babies in the study had chlorpyrifos in their bodies at the time of birth.[36]

Phthalates

Phthalates are a group of chemicals used in consumer products as plasticizers or softening agents in vinyl products. These include furnishings, flooring coverings, medical devices (eg catheters, IV- and blood bags), babies feeding bottles, toys, teething rings and food wrap. Phthalates are

also used in 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,[37] immunotoxins,[38] cancer promoters and are reproductive and developmental toxins.[39] DEHP has been classified as a "probable human carcinogen" by the USEPA. Rats and mice fed DEHP and DINP also showed an increase in liver cancers. Phthalates have been detected in the blood and urine of both adults and children.

The presence of pthalates in children's toys, teethers and dust containing phthalates may indicate that children are at greater risk. In the CDC study of phthalates, [40] the breakdown product of diethyl phthalate (DEP) was detected in the highest level in the tested population. The US National Toxicology Program (NTP) have expressed concern over the 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.

Dioxins

Dioxins are the by-products of PVC production, industrial bleaching, and incineration. Incineration, particularly of chlorinated compounds, is acknowledged as a priority source of dioxins, furans and other toxic byproducts, both by the USEPA and by the international community in Annex C of the Stockholm Convention on Persistent Organic Pollutants 2001. Growing concerns over the impact of dioxin contamination on children and future generations are evident in the international and national programs to address dioxin contamination.

The United Nation's Environment Program (UNEP)[41] describes dioxins and furans as posing particular hazards from their toxicity to humans and wildlife, their persistency, and their high lipid solubility; accumulating in the body fat of people, marine mammals, and other wildlife, and then bioaccumulating up the food chain. Dioxins are passed from mother to the fetus in the womb and to the child through breastmilk. They are semivolatile and mobile, traveling great distances on wind and water currents.

The effects of dioxins can include diseases of the immune system, reproductive and developmental disorders, as well as cancers.[42] They have a particular impact on women [43] where exposure has been implicated in endometriosis and increased breast cancer rates, and, through them, future generations. Studies have linked prenatal exposure to PCBs and dioxins with developmental and immune impacts in children. [44]

Heavy Metals

Toxic heavy metals do not break down in the environment and are not destroyed at any temperature. When released into air, toxic metals are deposited and remain in street dusts, inside building voids (eg ceiling spaces), on plants, water, sediments and soil. Metals bioaccumulate, moving from the environment into tissues of living beings where they build up over time. Low levels of lead can cause mental retardation, learning disabilities and stunted growth in children and lead is widely regarded as the most prevalent industrial chemical and the most common paediatric environmental health problem.

Since the bans on leaded petrol, a common source of poisoning of children is lead paint and ceiling dust released during renovations of pre-1970 houses but many children are also poisoned

by take-home dust on parents clothing or by hobby activities at home (eg car body repair, sinker- or bullet-making, ceramics or leadlighting).

Conclusions and Recommendations

There are many other emerging chemical issues that have the potential for significant impacts on children's health. The persistent bioaccumulative toxins currently contaminating the environment and our homes can impact adversely on the health and wellbeing of children. PBTs can affect their development, their immune system and later their reproductive viability. The following recommendations address these concerns as well as already recognised risks.

Recommendations :

- Establishment at a national level of a specialist office for Children's Environmental Health.
- Establishment of national and State legislation for a Child Environment Protection Act;
- Priority review of all uses of persistent bioaccumulative toxins/PBTs including PFOAs, BFRs, phthalates, chlorpyrifos, metals to identify both their intergenerational impacts and their alternatives;
- Establish ongoing biomonitoring of PBTs in children's blood and urine, in breastmilk and in infant meconium and cord blood;
- An immediate ban of penta and octaBDE, with an accompanying phaseout of decaPBDE over 2 years;
- Phase out chlorpyrifos and other organophosphates detected in children's meconium;
- Review fluorotelomer alcohols including their environmental fate
- Introduce legislation to warn home-buyers and potential tenants of asbestos and lead hazards in houses prior to sale or rent.
- Introduce regulations to ensure paint contractors and ceiling dust removalists are trained and then licensed to deal with lead (and other heavy metals) and asbestos in paint and building cavity dust.
- Set a new target for blood lead levels below 10 micrograms per decilitre 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.

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Endnotes

[1] The Right of the Child to a Clean Environment, Edited by Agata Fijalkowski & Malgosia Fitzmaurice, Ashgate Publishing, UK 2000

[2] ibid

[3] UNICEF Press Release 18 May 2002, "The Berlin Commitment, Pledging to create an environment fit for children" Available at <www.unicef.org/specialsession/press/01pr47.htm>

[4] 'Children's health and environment: A review of evidence,' A joint report from the European Environment Agency and the WHO Regional Office for Europe. Edited by: Giorgio Tamburlini, Ondine S. von Ehrenstein, Roberto Bertollini., WHO Regional Office for Europe, Environmental Issue Report No 29

[5] See Children's health and environment. A review of evidence, A joint report from the European Environment Agency and the WHO Regional Office for Europe; IFCS Forum IV 'Protecting Children from Harmful Chemical Exposures, Chemical Safety and Children's Health'; UNEP, UNICEF & WHO, Children in the New Millennium: Environmental Impact on Health (2002); and TOXICS AND POVERTY: The Impact of Toxic Substances On the Poor in Developing Countries, Prepared by Lynn Goldman and Nga Tran for the World Bank.

[6] Landrigan, P J et al. 1998. Children's health and the environment: A new agenda for prevention research. Environmental Health Perspectives 106, Supplement 3:787-794.

[7] See Pluim, H.J., Koppe, J.G., Olie, K., van der Slikke, J.W., Slot, P.C., & van Boxtel, C., 'Clinical laboratory manifestations of exposure to background levels of dioxins in the perinatal period,' (1994) Vol. 83 *Acta Paediatrica* pp 583-587. Also see OIlsen A., Briët J.M., Koppe J.G., Pluim H.J., Oosting J.,'Signs of enhanced neuromotor maturation in children due to perinatal load with background levels of dioxins', (1996) Vol.33, No 7*Chemosfere* pp1317-1326. Also see Weisglas-Kuperus, N., Sas, T.C.J., Koopman-Esseboom, C., Vanderzwan, C.W., Deridder, M.A.J., Beishuizen, A., Hooijkaas, H.& Sauer, P.J.J. 'Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants', (1995) Vol.38 (3) *Pediatric Research* pp 404-410

[8] Needleman, Herbert L; Shell, Alan; Bellinger, David; Leviton, Alan; & Allred, Elizabeth N, 'The Long-Term Effects Of Exposure To Low Doses Of Lead In Childhood – An 11-Year Follow-Up Report', The New England Journal Of Medicine Vol 322 No 2 (1989); H. L. Needleman, J. A. Riess, M. J. Tobin, G. E. Biesecker and J. B. Greenhouse, 'Bone lead levels and delinquent behavior', JAMA [Journal of the American Medical Association] Vol. 275 No. 5, February 7, 1996.

[9] Canfield, Richard L; Henderson, CR; Cory-Slechta, DA; Cox, C; Jusko, Todd A; Lanphear, Bruce P, 'Intellectual Impairment in Children with Blood Lead Concentrations below 5 micrograms per Deciliter', New England Journal of Medicine Volume 348:1527-1536 Number 16, April 17, 2003.

[10] James R. Campbell, Randy N. Rosier, Leonore Novotny and J. Edward Puzas, 'The Association between Environmental Lead Exposure and Bone Density in Children', Environmental Health Perspectives, Volume 112, Number 11, August 2004.

[11] MGA Opler, AS Brown, J Graziano, M Desai, W Zheng, C Schaefer, P Factor-Litvak, & ES Susser, 'Prenatal Lead Exposure, delta-Aminolevulinic Acid, and Schizophrenia', Environmental Health Perspectives, Vol 112 No 5 April 2004.

[12] Press Release Nov. 6, 1997 Centre for Disease Control and Prevention , "Hypospadias Trends in Two US Surveliance Systems"

[13] UNEP, UNICEF & WHO, Children in the New Millennium: Environmental Impact on Health (2002) *Sources include the United Nations Environment Programme, United Nations Children's Fund, World Health Organization, Food and Agricultural Organization, Childhood Pesticide Poisoning: Information for Advocacy and Action (draft), 2000; and Mott, Lawrie et al., Our Children at Risk: The 5 worst environmental threats to their health. Natural Resource Defense Council, New York, 1997.*

[14] Second National Report on Human Exposure to Environmental Chemicals (January 2003), Department of Health and Human Services, Centers for Disease Control and Prevention

[15] Environmental Working Group, Body Burden; the Pollution in People Jan 2003. Available at <http://www.ewg.org/reports/bodyburden/es.php>

[16] Dr L. E. Budd M.B. B.S. FRACP, Children's Health & Chemicals, One Paediatrician's Experience, July 1993 (Unpublished Lecture Notes)

[17] Deuble L, Whitehall JF, Bolisetty S, Patole SK, Ostrea EM* and Whitehall JS.Department of Neonatology, Kirwan Hospital for Women, Townsville. *Deparment of Pediatrics, Wayne State University, Michigan. "Environmental Pollutants In Meconium In Townsville, Australia." 2000

[18] Fire Retardant, Polybrominated diphenyl ethers (PBDEs), UNEP Chemicals, Regional Reports of the Regionally Based Assessment of Persistent Toxic Substances Program (2002) UNEP Chemicals 11-13, chemin des Anemones, CH-1219 Chatelaine, GE Switzerland. Available from <http://www.chem.unep.ch/pts>

[19] Stapleton, H. et al. Debromination of the flame retardant Decabromodiphenyl Ether by Juvenile Carp (Cyprinus carpio) following Dietary Exposure. Env. Sci & Tech 38 (1) pp. 112-119, 2004

[20] **'**Hazardous substance in the Arctic' (3/6/2004)**,** Norwegian Pollution Control Authority (SFT), the Norwegian Polar Institute, National Veterinary Institute of Norway/Norwegian School of Veterinary Science; Available at <http://www.sft.no/english/news/dbafile11556.html> [Accessed 10/6/2004] Also see' Brominated Flame Retardants (BFRs) in the Artic Marine Food Chain' Bjørn Munro Jenssen, Eugen G. Sørmo, Maria P. Salmer, Kine Bæk, and Janneche U. Skaare, Department of Biology, Norwegian University of Science and Technology, NO-7491 Trondheim, Norway; Norwegian Veterinary Institute, PO Box 8146, NO-0033 Oslo, Norway. Presentation to the Third International Workshop on BFRs, Uni. of Toronto, Canada 6-9 June 2004

[21] See Choi JW, Fujimaki TS, Kitamura K, Hashimoto S, Ito H, Suzuki N, Sakai S, Morita M (2003) Polybrominated dibenzo-p-dioxins, dibenzofurans, diphenyl ethers in Japanese human adipose tissue. *Environ Sci Technol;* 37 (5):pp817-21; Guvenius DM, Aronsson A, Ekman-Ordeberg G, Bergman A, Noren K (2003) Human prenatal and postnatal exposure to poly-brominated diphenyl ethers, poly chlorinated biphenyls, polychlorobiphenylols and pentachlorophenol. *Environmental Health Perspective*; 111(9) pp1235-41; Hardell L, Lindstrom G, van Bavel B, Wingfors H, Sundelin E, Liljegren G (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-Hodkin's lymphoma. *Oncol Res;*10(8) pp429-32; Mazdai A, Dodder NG, Abernathy MP, Hites RA, Bigsby RM (2003) Polybrominated diphenyl ethers in maternal and fetal blood samples. *Environmental Health Perspective*;111(9):1249-52; She J, Petreas M, Winkler J, Visita P, Mc Kinney M, Kopec D (2002) PBDEs in San Francisco Bay area: Measurements in harbor seals blubber and human breast adipose tissue. *Chemosphere;* 46(5) pp697-707

[22] Heather M. Stapleton, Michele Schantz and Stephen Wise (2004) Polybrominated Diphenyl Ether Measurements in Household Dust, Institute of Standards and Technology. Presentation to the Third International Workshop on BFRs, Uni. of Toronto, Canada 6-9 June 2004; Also see Sharp R, Lunder S,(2004) In the dust; Toxic Fire Retardants in American Homes, Environmental Working Group Available at <http://www.ewg.org/reports/inthedust/> [Accessed 4/6/2004] The studies 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 8,700 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 home around the country. The average combined levels of deca, penta and octa _ a third commercial mixture _ for nine of the homes was over 4,600 ppb.

[23] Alexandra McPherson, Beverly Thorpe & Anne Blake (2004) Brominated Flame Retardants in Dust on Computers; The Case For Safer Chemicals and Better Computer Design. Available at <www.computertakeback.org>

[24] Thomsen C, Lundanes E, Becher G. 2002. Brominated flame retardants in archived serum samples from Norway: a study on temporal trends and the role of age. Environmental Science and Technology. 6(7):1414-8.[18]

[25] World Health Organization. International Programme on Chemical Safety, Environmental Health Criteria 205: Polybrominated dibenzo-p-dioxins and dibenzofurans.

[26] Organisation for Economic Co-operation & Development (OECD) Environment Directive, 'Preparation of a screening Level Risk Assessment of Perfluorooctanyl Sulfonate (PFOS) and its derivatives' ENV/EHS/RV/jg/2000.177

[27] ibid

[28] PFOS, UNEP Chemicals, Regional Reports of the Regionally Based Assessment of Persistent Toxic Substances Program (2002) UNEP Chemicals 11-13, chemin des Anemones, CH-1219 Chatelaine, GE Switzerland. Available from <http://www.chem.unep.ch/pts>

[29] Olsen, G W et al. 2003. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. Environmental Health Perspectives 111:1892-1901 http://dx.doi.org

[30] REVISED DRAFT HAZARD ASSESSMENT OF PERFLUOROOCTANOIC ACID AND ITS SALTS, U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics Risk Assessment Division, November 4, 2002

[31] PRELIMINARY RISK ASSESSMENT OF THE DEVELOPMENTAL TOXICITY ASSOCIATED WITH EXPOSURE TO PERFLUOROOCTANOIC ACID AND ITS SALTS, U.S. Environmental Protection Agency Office of Pollution Prevention and Toxics Risk Assessment Division, April 10, 2003

[32] NICNAS Alert No. 2, 30 April 2003, Perfluorooctane sulfonate (PFOS), Available at <http://www.nicnas.gov.au/publications/infosheets.asp>

[33] Lloyd-Smith, M., 'Polluter Pays Principle, Myth or Legend', Vol. 8. 2002 *Australasian Journal of Ecotoxicology*

[34] Comments of New York State Attorney General Elliott Spitzer. In re: United States Environmental Protection Agencies Preliminary Risk Assessment for Chlorpyrifos Reregistration Eligibility Decision, Docket Control Number 0PP - 34203, December 27, 1999, amended January 3, 2000

[35] HED DOC. NO. 014077, April 4, 2000, MEMORANDUM, SUBJECT: CHLORPYRIFOS - Re-evaluation Report of the FQPA Safety Factor, Brenda Tarplee, Executive Secretary, FQPA Safety Factor Committee Health Effects Division (7509C)

[36] Environmental pollutants in meconium in Townsville, Australia. Deuble L, Whitehall JF, Bolisetty S, Patole SK, Ostrea EM* and Whitehall, JS.Department of Neonatology, Kirwan Hospital for Women, Townsville. *Deparment of Pediatrics, Wayne State University, Michigan. 1999 (Unpublished)

[37] Lovekamp TN, Davis BJ (2001) Mono-(2-ethylhexyl) phthalate suppresses aromatase transcript levels and estradiol in cultured rat granulose cells. Toxicol Appl Pharmacol 172(3):217-24

[38] Nencioni A, Wesselborg S, Brossart P (2003) Role of peroxisome proliferators-activiated receptor gamma & its ligands in the control of immune responses. *Crit Rev Immunol*; 23(1-2(1-13)

[39] Sharpe, RM and DS Irvine. 2004. How Strong is the Evidence of a Link Between Environmental Chemical and Adverse Effects on Human Reproductive Health? *British Medical Journal. 328(21 Feb):447-451.*

[40] Second National Report on Human Exposure to Environmental Chemicals (January 2003), Department of Health and Human Services, Centers for Disease Control and Prevention

[41] UNEP Chemicals & UNEP's Information Unit For Conventions, *UNEP Information Kit Stockholm 2001*, Conference of Plenipotentiaries for the Stockholm Convention on Persistent Organic Pollutants, Stockholm, 22- 23 May 2001

[42] ibid

[43] See Høyer, AP, T Jørgensen, JW Brock & P Grandjean 'Organochlorine exposure & breast cancer survival', (2000) 53 *Journal of Clinical Epidemiology* pp 323-330. Also Stewart J. Edmunds G., McCarthy R.A. & Ramsdell J.S.,'Permanent & Functional Male-to-Female Sex Reversal in d-rR Strain Medaka (Oryzias latipes) following Egg Microinjection of o,p'-DDT', (2000) Vol.108, No. 3 *Environmental Health Perspectives*

[44] See Pluim, H.J., Koppe, J.G., Olie, K., van der Slikke, J.W., Slot, P.C., & van Boxtel, C., 'Clinical laboratory manifestations of exposure to background levels of dioxins in the perinatal period,' (1994) Vol. 83 *Acta Paediatrica* pp 583-587. Also see OIlsen A., Briët J.M., Koppe J.G., Pluim H.J., Oosting J.,'Signs of enhanced neuromotor maturation in children due to perinatal load with background levels of dioxins', (1996) Vol.33, No 7*Chemosfere* pp1317-1326. Also see Weisglas-Kuperus, N., Sas, T.C.J., Koopman-Esseboom, C., Vanderzwan, C.W., Deridder, M.A.J., Beishuizen, A., Hooijkaas, H.& Sauer, P.J.J. 'Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants', (1995) Vol.38 (3) *Pediatric Research* pp 404-410