



**CHEM (CHEMICALS, HEALTH AND ENVIRONMENT MONITORING)
TRUST'S RESPONSE TO THE UK HEALTH PROTECTION AGENCY'S
CONSULTATION ON "A CHILDREN'S ENVIRONMENT AND HEALTH
STRATEGY FOR THE UNITED KINGDOM."**

28th May 2008

Introduction to CHEM Trust

CHEM Trust has a **mission** to prevent man-made chemicals from causing long term damage to wildlife or humans by ensuring that chemicals which cause such harm are substituted with safer alternatives.

CHEM Trust is a relatively new charity, set up in April 2007, with initial start-up funding from WWF-UK, in recognition of the threats chemicals pose not only to wildlife, but also to human health. In addition, CHEM Trust is funded to a lesser extent by Greenpeace Environmental Trust. CHEM Trust is also funded by, and works with, the Health and Environment Alliance (HEAL) based in Brussels, and thereby ensures its work is translated into many languages for outreach across the EU and more widely. To date, CHEM Trust briefings have been published in Russian, Polish, Czech, Italian, Spanish, French, German, Slovenian, and English. They include the following topics:

- i) "What could new EU chemicals legislation deliver for public health?" looking at the health benefits that the new EU Regulation (REACH¹) could provide.
- ii) "Chemicals compromising our children" which discusses the potential damage chemicals may cause to the developing brain.
- iii) "Factors influencing the risk of breast cancer – established and emerging" a briefing for the public on the potential role of chemicals in breast cancer
- iv) "Breast cancer and exposure to hormonally active chemicals: An appraisal of the scientific evidence" a briefing for medical professionals and scientists by Professor Andreas Kortenkamp of

¹ REACH is the Regulation concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals
<http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:32006R1907:EN:NOT>

the London School of Pharmacy. This briefing is in English only and was launched in the European Parliament in April 2008.

All these publications are available free of charge on CHEM Trust's web site at www.chemtrust.org.uk

CHEM Trust's Vision is a world where humans and wildlife co-exist with a sustainable chemical industry and where chemicals play no part in causing impaired reproduction, deformities, disease, or deficits in neurological function.

CHEM Trust's work programme is currently focussed on securing better controls over chemicals that cause long term harm, including persistent and bioaccumulating chemicals (which build up in our bodies or in wildlife and are passed from mother to baby in utero or via breast milk) and chemicals which can disrupt hormones (endocrine disrupting chemicals).

Contact details:
Gwynne Lyons
Director CHEM Trust
c/o 17 The Avenues
Norwich NR2 3PH

Email: Gwynne.lyons@chemtrust.org.uk
Tel: 01603 507363

I am not under 18 years of age.
CHEM Trust does not have a membership.
This response is not confidential.

**RESPONSE TO THE UK HEALTH PROTECTION AGENCY'S
CONSULTATION ON "A CHILDREN'S ENVIRONMENT AND HEALTH
STRATEGY FOR THE UNITED KINGDOM."**

Overview of CHEM Trust's Response

CHEM Trust welcomes the strategy, but is disappointed that the UK appears content not to take a leading role in taking forward new science to improve health protection policies. We feel that the UK should show leadership in protecting children to a high degree and not just cover the internationally agreed priority areas to a limited extent.

CHEM Trust considers that the proposed Children's Environment and Health Strategy for the UK omits tackling the role chemical exposures play in ill health. The proposal by the Health Protection Agency, commissioned on behalf of the Interdepartmental Steering Group on Environment and Health, is not a forward looking strategy, and does not adequately take into account recent science linking chemical exposures to obesity, birth defects, and adverse effects on brain function and behaviour.

CHEM Trust's detailed response to questions in the consultation

CHEM Trust's response relates only to its area of expertise which include the following issues: obesity, pollution and the effects of chemicals.

In general, CHEM Trust considers that the report does not adequately address the potential for effects to be caused due to exposure to chemicals, particularly effects due to exposure to many chemicals with similar mechanisms of action, or mechanisms of action that converge. Neither does the report adequately address the potential for in-utero exposures to cause effects which only manifest later in life.

Obesity

Q6: There are additional areas concerning obesity that certainly need to be addressed in the UK, and which CHEM Trust is disappointed to see omitted from the strategy. These relate to the role of chemical exposures in obesity. There is now good evidence that exposure to oestrogen mimicking chemicals in utero, particularly at low exposure levels, can cause obesity in offspring.

Newbold et al (2007) have shown that low doses of DES (diethylstilboestrol) either prenatally or neonatally cause an increase in body weight in mice that is significant by 6 weeks of age. Similarly, perinatal exposure to the naturally occurring phyto-oestrogen, genistein, can cause weight gain, and Rubin et al (2001) has also reported that the oestrogen mimicking chemical, bisphenol A, results in body weight gain that continues into adulthood. Furthermore, other hormone disrupting chemicals are also implicated in playing a role in the development of obesity (Heindel et al, 2003). Tributyltin, for example, increases the differentiation of adipocytes in vitro, and alters regulation of adipogenesis (Grun et al, 2006; Grun and Blumberg 2007). Moreover, some PCBs are associated with an increase in body mass index (Goncharov et al, 2007). Therefore, there are significant data in animal models, and limited epidemiological data, supporting a role for environmental exposures in obesity.

CHEM Trust would like to see a summary of the evidence for chemicals playing a role in obesity in the UK children's environment and health strategy. Furthermore, we would like to see outlined in the strategy a framework of measures to reduce exposures to such chemicals implicated in obesity. For example, CHEM Trust would like to see the UK acknowledge the need for more research, while at the same time putting in place exposure reduction measures for hormone disrupting chemicals. Such exposure reduction would have potential benefits for other diseases, including breast cancer and male reproductive health. (See the Prague Declaration which has now been signed by over 200 eminent scientists, many of whom are working at the cutting edge of research into endocrine disrupting chemicals (<http://www.ehponline.org/docs/2007/10517/suppl.pdf>).

CHEM Trust would therefore like the UK Health Protection Agency and the UK Government to support concrete examples of how exposure reduction

could be achieved. For example, (i) CHEM Trust considers that the UK Competent Authority should draft, or support, an Annex XV dossier to put Bisphenol A on the candidate list under the EU REACH Regulation, because it is a substance with endocrine disrupting properties. (ii) To reduce exposure to pesticides with endocrine disrupting properties, CHEM Trust would like the Pesticides Safety Directorate and the UK Government to support the proposed text of the revised EU Plant Protection Products legislation (91/414) which would require pesticides with hormone disrupting chemicals to be banned from use unless human exposure was negligible.

References

Goncharov A, Haase RF, Santiago-Rivera A, Morse G; Akwesasne Task Force on the Environment, McCaffrey RJ, Rej R, Carpenter DO (2008). High serum PCBs are associated with elevation of serum lipids and cardiovascular disease in a Native American population. *Environ Res*, 106(2):226-39.

Grün F, Watanabe H, Samarian Z et al (2006). Endocrine disrupting organotin compounds are potent inducers of adipogenesis in vertebrates. *Mol Endocrinol*, 20: 2144-2155.

Grün F, Blumberg B (2007). Perturbed nuclear receptor signaling by environmental obesogens as emerging factors in the obesity crisis. *Rev Endocr Metab Disord*, 8(2):161-71.

Heindel JJ (2003). Endocrine disruptors and the obesity epidemic, *Toxicol Sci* 76, 247-249

Iguchi T, Watanabe H, Ohta Y, Blumberg B (2008). Developmental effects: oestrogen-induced vaginal changes and organotin-induced adipogenesis. *Int J Androl*. 31(2):263-268.

Newbold RR, Padilla-Banks E, Snyder RJ, Philips TM, Jefferson WN (2007). Developmental exposure to endocrine disruptors and the obesity epidemic. *Reprod Toxicol*, 23: 290-296

Newbold RR, Padilla-Banks E, Jefferson WN, Heindel JJ (2008). Effects of endocrine disruptors on obesity *Int J Androl*. (2):201-208.

Developmental Endocrinology and Endocrine Disruptor Section, Laboratory

Porta, M, (2006). Persistent Organic Pollutants and the burden of diabetes, *The Lancet*, 368: 558

Rubin BS, Murray MK, Damassa DA, King JC, Soto AM (2001). Perinatal exposures to low doses of bisphenol A affects body weight, patterns of estrus cyclicity, and plasma LH levels. *Environ Health Perspect* 2001,109: 675-680.

Indoor and outdoor air pollution

Q10: The UK strategy focuses on carbon monoxide poisoning and passive smoking. These are important, but nevertheless substantive improvements in children's health may be found by addressing other pollutants, including for example, phthalates. Phthalate exposure may arise from kitchen and bathroom floorings, and other consumer articles made of flexible plastics. Some phthalates have been implicated in birth defects of baby boys (Lottrup et al, 2006), precocious breast growth in girls (Colon et al, 2000) and allergy and asthma in children (Bornehag et al, 2005). In general, there is a need to note in the strategy that inhalatory exposures add to the "cup-full" of exposures from other sources, and that total exposures, from all sources, to any particular chemical need to be carefully considered. Moreover, exposure to many volatile chemicals, and chemicals found in house dust, means that children will be exposed to a plethora of chemicals at any one time, such that "mixture" or cocktail effects might be predicted.

Similarly, some outdoor air pollutants, including some poly aromatic hydrocarbons (PAHs) are reported to be endocrine disruptors. CHEM Trust is therefore disappointed that the strategy does not address many of the endocrine disrupting chemicals which are of growing concern with respect to air pollution and this needs to be remedied in the final strategy.

Apart from endocrine disruptors, other chemicals including some found as indoor air pollutants (Mendel, 2007), and some resulting from traffic emissions, may act as allergens in children and this is an issue which deserves more attention.

References

Bornehag CG, Sundell J, Weschler CJ, Sigsgaard T, Lundgren B, Hasselgren M, Hägerhed-Engman L. (2005) The association between asthma and allergic symptoms in children and phthalates in house dust: a nested case-control study. *Environ Health Perspect.* 112(14):1393-1397.

Colón I, Caro D, Bourdony CJ, Rosario O (2000). Identification of phthalate esters in the serum of young Puerto Rican girls with premature breast development. *Environ Health Perspect.* 108(9):895-900.

Lottrup G, Andersson AM, Leffers H, Mortensen GK, Toppari J, Skakkebaek NE, Main KM (2006). Possible impact of phthalates on infant reproductive health. *Int J Androl.* 2006 Feb;29(1):172-180;

Mendell MJ (2007) Indoor residential chemical emissions as risk factors for respiratory and allergic effects in children: a review. *Indoor Air.* 7(4):259-277

Non Ionising Radiation

Q13: CHEM Trust accepts the need to protect children from ultra-violet radiation. However, particularly given some sun screens contain endocrine disrupting chemicals, such as benzophenone and 4-MBC (methylbenzylidene camphor) (Schlumpf et al, 2004; Schlecht et al, 2006; Weisbrod et al, 2007), CHEM Trust would like to see an emphasis on providing shade and using light clothes to cover up the skin in sunlight. Alternatively, there is a need to ensure endocrine disrupting chemicals are removed from sun care products so that public confidence in sun screens is not undermined.

References

Schlumpf M, Schmid P, Durrer S, Conscience M, Maerkel K, Henseler M, Gruetter M, Herzog I, Reolon S, Ceccatelli R, Faass O, Stutz E, Jarry H, Wuttke W, Lichtensteiger W (2004). Endocrine activity and developmental toxicity of cosmetic UV filters--an update. *Toxicology*. 205(1-2):113-22.

Schlumpf M, Durrer S, Faass O, Ehnes C, Fuetsch M, Gaille C, Henseler M, Hofkamp L, Maerkel K, Reolon S, Timms B, Tresguerres JA, Lichtensteiger W (2008). Developmental toxicity of UV filters and environmental exposure: a review. *Int J Androl*. 31(2):144-151.

Schlecht C, Klammer H, Wuttke W, Jarry H (2006). A dose-response study on the estrogenic activity of benzophenone-2 on various endpoints in the serum, pituitary and uterus of female rats. *Arch Toxicol*. 80(10):656-61.

Weisbrod CJ, Kunz PY, Zenker AK, Fent K (2007). Effects of the UV filter benzophenone-2 on reproduction in fish. *Toxicol Appl Pharmacol*. 225(3):255-66.

Additional areas concerning chemicals and children's health which need to be addressed in the UK

Q14: CHEM Trust agrees that techniques such as human biomonitoring are needed to gain better data on exposure to chemicals. We would like to see a UK on-going study of breast milk contaminants (used as a proxy for in-utero exposures) including a comprehensive range of contaminants as determined by a stakeholder and Government Department expert committee.

However, enough is known now about certain exposures, particularly concerning some endocrine disrupting chemicals, to initiate exposure reduction measures in order to protect children. For example, Canada has announced its intention to ban the importation, sale or advertising of baby bottles containing bisphenol A (see http://www.hc-sc.gc.ca/ahc-asc/media/nr-cp/2008/2008_59_e.html), whilst this chemical still has ubiquitous use, including in baby bottles in the UK.

CHEM Trust is therefore disappointed that the strategy does not address better the issue of exposures to endocrine disrupting chemicals, and particularly the potential harm caused by in-utero exposure to such compounds. Furthermore, Bisphenol A, and other endocrine disruptors, have

been linked with altered blood glucose homeostasis, such that some scientists consider that adult exposure may enhance the risk of developing type II diabetes (Roper et al, 2008).

In addition, CHEM Trust considers that the strategy does not pay enough attention to preventing adverse effects on children's brain development and function. In particular, issues such as reducing illegal drug usage and reducing exposure to certain man-made chemicals in consumer products should be tackled.

For example, given that lead (Lanphear et al, 2005; Wigle & Lanphear 2005) and PCBs are believed to have affected the intelligence of many children throughout the world (Patandin et al, 1999; Walkowiak et al, 2001; Lundqvist et al, 2006) more consideration in the strategy should be given to preventing adverse effects of chemicals on the brain. Several chemicals, for example, some brominated flame retardants (Darnerud, 2008) including deca-brominated diphenyl ether (deca-BDE) which is still used as a flame retardant in the EU (Cressey et al, 2006; Viberg et al, 2007), bisphenol A (see EU human health risk assessment of Bisphenol A), mercury (Debes et al, 2006), and others (Grandjean et al, 2006) have been reported to have developmental neurotoxic properties. CHEM Trust considers that it would be prudent to implement strategies to eliminate exposure to such chemicals with developmental neurotoxic properties wherever possible.

The role of chemicals in causing immune system dysfunction should also be noted. The immune system undergoes crucial developmental maturation both before and after birth. New evidence suggests that a number of environmental pollutants may affect the future function of the immune system (Dietert & Piepenbrink, 2006). With regard to potential immune system effects, CHEM Trust is particularly concerned about exposures to PFOA (perfluorooctanoic acid) which has been widely used on carpets (Fairley et al, 2007).

In addition, the study of epigenetics merits mention and future research. The potential for chemicals to cause effects which may last several generations is causing some concern (Anway and Skinner, 2008). Epigenetic changes may lead to altered gene expression, and may in future lead to a fundamental shift in how we think about inheritance and the safety of chemicals.

References

Cressey MA, Reeve EA, Rice DC, Markowski V (2006). Behavioral impairments produced by developmental exposure to the flame retardant decaBDE. *Neurotoxicology and Teratology*. 28(6): 707-708.

Darnerud PO (2008). Brominated flame retardants as possible endocrine disrupters. *Int J Androl*. 31(2):152-60.

Debes F, Budtz-Jorgensen E, Weihe P, White RF, Grandjean P (2006). Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. *Neurotoxicol Teratol*. 28(5):536-47.

Dietert RR, Piepenbrink MS (2008) The managed immune system: protecting the womb to delay the tomb. *Hum Exp Toxicol.* 27(2):129-34.

Fairley KJ, Purdy R, Kearns S, Anderson SE, Meade BJ (2007) Exposure to the immunosuppressant, perfluorooctanoic acid, enhances the murine IgE and airway hyperreactivity response to ovalbumin, *Toxicol Sci.* 97(2):375-83.

Grandjean P, and Landrigan PJ (2006). Developmental neurotoxicity of industrial chemicals. *The Lancet* 16;368(9553):2167-78.

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J, Roberts R (2005). Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect*, 113(7):894-899.

Lundqvist C, Zuurbier M, Leijds M, Johansson C, Ceccatelli S, Saunders M, Schoeters G, ten Tusscher G, Koppe JG (2006). The effects of PCBs and dioxins on child health. *Acta Paediatr Suppl.* (453):55-64.

Patandin S, Lanting C I, Mulder P G H, Boersma ER, Sauer P J J, Weisglas-Kuperus N (1999). Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. *J Pediatr*, 134: 33-41.

Ropero AB, Alonso-Magdalena P, García-García E, Ripoll C, Fuentes E, Nadal A (2008). Bisphenol-A disruption of the endocrine pancreas and blood glucose homeostasis. *Int J Androl.* 31(2):194-200.

Viberg H, Fredriksson A, Eriksson P (2007). Changes in spontaneous behaviour and altered response to nicotine in the adult rat, after neonatal exposure to the brominated flame retardant, decabrominated diphenyl ether (PBDE 209), *Neurotoxicology*, 28(1):136-42.

Walkowiak J, Wiener JA, Fastabend A, Heinzow B, Kramer U, Schmidt E, Steingruber HJ, Wundram S, Winneke G. (2001). Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood. *Lancet.* Nov 10;358(9293):1602-7.

White LD, Cory-Slechta DA, Gilbert ME, Tiffany-Castiglioni E, Zawia NH, Virgolini M, Rossi-George A, Lasley SM, Qian YC, Basha MR (2007). New and evolving concepts in the neurotoxicology of lead. *Toxicol Appl Pharmacol*, 225(1):1-27

Wigle DT and Lanphear BP (2005). Human health risks from low-level environmental exposures: No apparent safety thresholds, *PLoS Med* 2(12) e350 doi:10.1371/journal.pmed.0020350

<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1255761>

Overarching issues and priorities

Q17: CHEM Trust considers that chemicals in general are not covered well in the strategy and action plan. Also, with respect to chemical exposures there is a need to consider total exposures from all sources, and sensitive windows of exposure, particularly in-utero and peri-pubertal.

Moreover, there is a need to address that particularly for endocrine disrupting chemicals there may be additive effects due to exposure to several chemicals which act through the same biochemical mechanism or mechanisms of action which converge. Additivity has now been conclusively shown for endocrine disrupting chemicals including oestrogen mimicking chemicals (Rajapakse et al, 2002; Kortenkamp 2007), anti-androgenic chemicals (Hotchkiss et al, 2004, Christiansen et al 2008), and thyroid disruptors (Crofton et al 2005).

References

Christiansen S, Scholze M, Axelstad M, Boberg J, Kortenkamp A, Hass U (2008) . Combined exposure to anti-androgens causes markedly increased frequencies of hypospadias in the rat, *Int J Androl.* 31(2):241-248.

Crofton KM, Craft ES, Hedge JM, Gennings C, Simmons JE, Carchman RA, Carter WH Jr, DeVito MJ (2005). Thyroid-hormone-disrupting chemicals: evidence for dose-dependent additivity or synergism. *Environ Health Perspect.* 113(11):1549-54.

Hotchkiss AK, Parks-Saldutti LG, Ostby JS, Lambright C, Furr J, Vandenberg JG, Gray LE Jr. *Biol Reprod.* (2004) A mixture of the "antiandrogens" linuron and butyl benzyl phthalate alters sexual differentiation of the male rat in a cumulative fashion. *Biol Reprod.* Dec;71(6):1852-61..

Kortenkamp A (2007). Ten years of mixing cocktails: a review of combination effects of endocrine-disrupting chemicals. *Environ Health Perspect.* 115 Suppl 1:98-105.

Rajapakse N, Silva E, Kortenkamp A. (2002) Combining xenoestrogens at levels below individual no-observed-effect concentrations dramatically enhances steroid hormone action. *Environ Health Perspect.* Sep;110(9):917-21.