

## **Bisphenol-A**

In order to protect the health of children in Connecticut by further reducing their exposure to the toxic environmental chemical Bisphenol-A (BPA), the Connecticut Public Health Association (CPHA) supports legislation that would ban bisphenol-A from thermal receipt paper.

### **Background**

Humans are routinely exposed to the chemical BPA, an endocrine disruptor, that it is widely used in the manufacturing of polycarbonate plastics and epoxy resins. It is one of the highest volume chemicals produced worldwide and studies have shown that under normal conditions BPA leaches from consumer products such as baby bottles, canned foods and beverages, dental sealants and many other consumer goods [1,2]. The Centers for Disease Control and Prevention (CDC) latest National Health and Nutrition Examination Survey (NHANES) analysis of over 2,500 Americans ages 6 and older found BPA in the urine of most people tested, which indicates widespread exposure to BPA in the U.S. population [3]. Additionally, researchers caution that at the current levels of BPA measured in humans, it is highly likely that BPA has the potential to cause disease or dysfunction [2].

Exposure to BPA has been associated with adverse animal and human health effects. Studies show possible low dose effects that include altered development of the fetal prostate and mammary glands, reduced testosterone production after birth, and changes in development of the neurological system [4,5,6] and linked BPA exposure to rodent mammary cancer, prostate cancer, and other dysfunctions[7,8,9,10]. Adding to these findings, the first major epidemiological study examined the health effects associated with BPA on a representative sample of U.S. adults. The findings suggest a significant relationship between urine concentrations of BPA and an increased prevalence of heart disease, diabetes, and liver enzyme abnormalities [11]. According to the Chapel Hill BPA Expert Panel [12], recent trends in human disease mirror the adverse effects observed in animals exposed to low doses of BPA, such as:

- an increase in prostate and breast cancer
- abnormalities of the urinary and genital systems in male babies
- decline in semen quality in men
- early on-set of puberty in girls
- metabolic disorders including Type 2 diabetes and obesity
- neurobehavioral developmental disorders such as attention deficit disorder (ADD)

Humans are particularly vulnerable to the adverse health effects of endocrine disruptors such as BPA during the various stages of development. There are scientific concerns about the negative effects in fetuses, infants, and children at current human exposure levels to bisphenol-A. A recent study showed exposure to BPA may even play a role in premature births [13]. Another study involving cord blood analyses showed every baby born today has toxic chemicals in his or her blood, including BPA, PCBs, phthalates, flame retardants, pesticides, lead, and mercury [14, 15]. This scientific data suggests embryos and fetuses are exposed to a vast array of toxic chemicals that cross the placental barrier throughout fetal development.

### **Recommendation**

Many harmful effects from toxic chemicals are preventable. CPHA supports public policies that help reduce children's exposure to BPA. The Canadian Government, which banned BPA for use in baby

bottles in 2008, recently added BPA to a national list of toxic substances [16]. Although U.S. regulatory agencies are moving slowly regarding BPA, the Connecticut General Assembly was an environmental leader when it passed HB 6572 in 2009.

- 1) Vandenberg, L., R Hauser, M Marcus, N Olea, and WV Welshons. Human exposure to bisphenol A (BPA). *Reproductive Toxicology*, 2007. 24(2): p. 139-177.
- 2) Vandenberg, L., M Maffini, C Schaeberle, A Ucci, C Sonnenschein, B Rubin, and A Soto. Perinatal exposure to the xenoestrogen bisphenol-A induces mammary intraductal hyperplasias in adult CD-1 mice. *Reproductive Toxicology*, 2008. 26(3): p. 210-219.
- 3) Lakind, JS & D Naiman. Daily intake of bisphenol A and potential sources of exposure: 2005–2006 National Health and Nutrition Examination Survey. *Journal of Exposure Science and Environmental Epidemiology*, March 17, 2010.
- 4) Leranthe, C., K Szigeti-Buck, NJ MacLusky, and T Hajszan. Bisphenol A prevents the synaptogenic response to testosterone in the brain of adult male rats. *Endocrinology*, 2008. 149: p. 988-994.
- 5) National Toxicology Program Center for the Evaluation of Risks to Human Production (NTP-CERHR). NTP-CERHR Panel Report on Reproductive and Developmental Toxicity of Bisphenol A. September, 2008. Available at URL: <http://cerhr.niehs.nih.gov/evals/bisphenol/bisphenol.html>
- 6) Timms, B.G., KL Howdeshell, L Barton, S Bradley, CA Richter, and FS vom Saal. Estrogenic chemicals in plastic and oral contraceptives disrupt development of the fetal mouse prostate and urethra. *Proc Natl Acad Sci USA*, 2005.102(19): p. 7014-7019.
- 7) Hunt P, M Susiarjo, C Rubio, and T Hassold. The Bisphenol A Experience: A Primer for the Analysis of Environmental Effects on Mammalian Reproduction. *Biology of Reproduction*, 2009. 81(5): p. 807-813.
- 8) Ho, S.M., WY Tang, J Belmonte de Frausto , and GS Prins. Developmental exposure to estradiol and bisphenol A increases susceptibility to prostate carcinogenesis and epigenetically regulates phosphodiesterase type 4 variant 4. *Cancer Research*, 2006. 66 (11): p. 5624-5632.
- 9) Jenkins S, N Raghuraman, I Eltoun, M Carpenter, et al. Oral exposure to Bisphenol A increases dimethylbenzanthracene-induced mammary cancer in rats. *Environmental Health Perspectives*, 2009. 117(6): p. 910-915.
- 10) Leranthe, C., T Hajszan, K Szigeti-Buck, J Bober, and NJ MacLusky. Bisphenol A prevents the synaptogenic response to estradiol in hippocampus and prefrontal cortex of ovariectomized nonhuman primates. *Proc Natl Acad Sci USA*, 2008. 105(37): p. 14187-14191.
- 11) Lang IA, T Galloway, and A Scarlett. Association of urinary bisphenol-A concentration with medical disorders and laboratory abnormalities in adults. *Journal of the American Medical Association*, 2008: 300 (11): p. 1303-1310.
- 12) Chapel Hill bisphenol-A expert panel consensus statement: Integration of mechanisms, effects in animals and potential to impact human health at current levels of exposure. *Reproductive Toxicology*, 24(2), August-September 2007, p. 131-138.
- 13) Cantonwine D., Meeker J., Hu H., Sanchez B, et. al. Bisphenol-A exposure in Mexico City and risk of prematurity: A pilot nested case control study. *Environmental Health*. 2010. 9(62).
- 14) Barr D, Bishop A, Needham L. Concentrations of xenobiotic chemicals in the maternal-fetal unit. *Reproductive Toxicology*, 2007. 23: p. 260-266.
- 15) Lee Y, Ryu H, Kim H, Min C, et.al. Maternal and fetal exposure to bisphenol-A in Korea. *Reproductive Toxicology*, 2008. 25: p. 413-419.
- 16) Canada Medical Association Journal, November 23, 2010. 182(17), E757.