

Bisphenol A (BPA) and Breast Cancer

Bisphenol A (BPA) is one of the most pervasive chemicals in modern life. More than 2 million pounds of BPA are produced in the United States each year. As the building block of polycarbonate plastic and a component of epoxy resins, BPA is used in thousands of consumer products, including food packaging.

Breast cancer strikes more women in the world than any other type of cancer except skin cancer. In the United States, a woman's lifetime risk of breast cancer is 1 in 8.¹ Research suggests that BPA exposure may contribute to the epidemic of breast cancer now and in the future. Furthermore, BPA exposure has been shown to interfere with chemotherapy treatment for breast cancer.²

BPA: a synthetic estrogen

BPA was developed in the 1930s as a synthetic estrogen (also called xenoestrogen) so it is not surprising that it acts like an estrogen in humans, increasing the risk of breast cancer.³ Decades of research have shown that extensive exposure to estrogens, both natural and synthetic, increases breast cancer risk.⁴ Reducing exposure to estrogens appears to reduce the risk of breast cancer. For example, experts attribute the recent decline in breast cancer incidence to decreased use of postmenopausal hormone replacement therapy (HRT), following the major study that implicated HRT in increased risk of breast cancer.⁵

Studies of human breast cancer cells in culture show that BPA acts through the same response pathways as natural estrogen (estradiol),⁶ and induces cell growth and proliferation.⁷ In addition, BPA has been shown to mimic natural estrogen (estradiol) in causing direct damage to the DNA of cultured human breast cancer cells.⁸

Principal route of exposure to BPA: food packaging

BPA is found in the lining of metal food cans and in some plastic food containers, including some baby bottles, water bottles, microwave ovenware and eating utensils. Because BPA is an unstable polymer and is lipophilic (fat-seeking), it can leach into infant formula and other food products, especially when heated.⁹ Once in food, BPA can move quickly into people—a real concern for women of childbearing age and for young children.

Exposure to BPA begins in the womb

Exposure to BPA is ubiquitous in the United States¹⁰ and other developed countries, and the exposure begins before birth, when the risk of harm is greatest. BPA has been found in blood samples from developing fetuses as well as in placental tissue and the surrounding amniotic fluid,¹¹ in umbilical cord blood of newborn infants¹² and in human breast milk.¹³ Finding BPA in breast milk confirms the presence of this environmental estrogen in the target organ for breast cancer.

A number of animal studies show that prenatal and early life exposure to extremely low levels of BPA alters development of the mammary gland in ways that predispose the animals to cancer in adult life. Exposure also increases sensitivity to estrogen at puberty.^{14 15 16 17 18} Early exposure to BPA also leads to abnormalities in mammary tissue that can be seen during gestation.¹⁹

Animal studies implicate BPA in childhood obesity, which raises the risk of early puberty, a known risk factor for breast cancer.²⁰ Formula feeding (BPA lined containers and/or baby bottles) rather than breastfeeding is also linked with childhood obesity.²¹

What other countries are doing about BPA

In 2007 Norway banned the import of consumer products containing BPA. Canada has declared BPA a toxic chemical and is moving to ban it in baby bottles, the lining of infant formula containers and children's tableware. Taiwan has proposed listing BPA as a potentially toxic substance.

Banning the use of BPA could reduce the risk of breast cancer in our daughters and in generations to come. It is time to call a halt to this toxic hand-me-down chemical.

Breast cancer facts

- An estimated 182,000 women will be diagnosed with breast cancer in 2008 and more than 40,000 women will die of the disease.²²
- Breast cancer is the second leading cause of death in U.S. women, second only to lung cancer.²³
- Between 1975 and 2000, breast cancer incidence increased in women ages 20-29.²⁴
- Breast cancer incidence for African American women ages 20-45 is twice that for white women the same ages and mortality from breast cancer is higher in African American women.²⁵

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³ Dodds EC, Goldberg L, Lawson W, Robinson R (1938). Oestrogenic activity of certain synthetic compounds. Nature 141:247-248.

⁴ Russo J, Russo I (2004). Ch. 4: The role of estrogen in breast cancer. In *Molecular Basis of Breast Cancer*, Springer-Verlag: Berlin.

⁵ Ravdin PM, Cronin KA, Howlander N, Berg CD, et al (2007). The decrease in breast cancer incidence in 2003 in the United States. New England Journal of Medicine 356:1670-1674.

⁶ Welshons WV, Nagel SC, vom Saal FS (2006). Large effects from small exposures. III. Endocrine mechanisms mediating effects of bisphenol A at levels of human exposure. Endocrinology 147:S56-S69.

⁷ Watson CS, Bulayeva NN, Wozniak AL, Finnerty CC (2005). Xenoestrogens at picomolar to nanomolar concentrations trigger membrane estrogen receptor-alpha-mediated Ca²⁺ fluxes and prolactin release in GH3/B6 pituitary tumor cells. Environmental Health Perspectives 113:431-439.

⁸ Iso T, Watanabe T, Iwamoto T, Shimamoto A, Furuichi Y (2006). DNA damage caused by bisphenol A and estradiol through estrogenic activity. Biology and Pharmaceutical Bulletin 29:206-210.

⁹ Brotons JA, Olea-Serrano MF, Villalobos M, Pedraza V, Olea N (1995). Xenoestrogens released from lacquer coatings in food cans. Environmental Health Perspectives 103:608-612.

¹⁰ Calafat AM, Kuklennyik Z, Reidy JA, Caudill SP, et al (2005). Urinary concentrations of bisphenol A and 4-nonylphenol in a human reference population. Environmental Health Perspectives 113:391-395.

¹¹ Ikezuki Y, Tsutsumi O, Takai Y, Kamei Y, Taketani Y (2002). Determination of bisphenol A concentrations in human biological fluids reveals significant prenatal exposure. Human Reproduction 17:2839-2841.

¹² Schoenfelder G, Wittfoht W., Hopp H, et al (2002). Parent bisphenol A accumulation in the human maternal-fetal-placental unit. Environmental Health Perspectives 110:A703-A707.

¹³ Kuruto-Niwa R, Tateoka Y, Usuki Y, Nozawa R (2006). Measurement of bisphenol A concentrations in human colostrum. Chemosphere 66:1160-1164.

¹⁴ Munoz-de-Toro M, Markey CM, Wadia PR, Luque EH, Rubin BS, Sonnenschein C, Soto AM (2005). Perinatal exposure to bisphenol A alters peripubertal mammary gland development in mice. Endocrinology 146:4138-4147.

¹⁵ Maffini MV, Rubin BS, Sonnenschein C, Soto AM (2006). Endocrine disruptors and reproductive health: The case of bisphenol A. Molecular and Cellular Endocrinology 254-255:179-186.

¹⁶ Markey CM, Luque EH, Munoz-de-Toro M, Sonnenschein C, Soto AM (2001). In utero exposure to bisphenol A alters the development and tissue organization of the mouse mammary gland. Biology of Reproduction 65:1215-1223.

¹⁷ Murray TJ, Maffini MV, Ucci AA, Sonnenschein C, Soto AM (2007). Induction of mammary gland ductal hyperplasias and carcinoma in situ following fetal bisphenol A exposure. Reproductive Toxicology 23:383-390.

¹⁸ Durando M, Kass L, Piva J., Sonnenschein C, Soto A, Luque E, Munoz-de-Toro M (2007). Prenatal bisphenol A exposure induces preneoplastic lesions in the mammary gland in Wistar rats. Environmental Health Perspectives 115:592-598.

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²⁰ vom Saal FS (2007). "Perinatal programming of obesity: Interaction of nutrition and environmental exposures," presentation to the American Association for the Advancement of Science annual meeting, San Francisco, February 16, 2007.

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²² American Cancer Society (2008). Cancer Facts and Figures. www.cancer.org

²³ American Cancer Society (2008). Cancer Facts and Figures. www.cancer.org

²⁴ National Cancer Institute (2008). Cancer epidemiology in older adolescents and young adults 15-29 years of age: Including SEER incidence and survival: 19075-2000.

²⁵ National Cancer Institute (2008). Cancer epidemiology in older adolescents and young adults 15-29 years of age: Including SEER incidence and survival: 19075-2000.