

MIND, DISRUPTED

How toxic chemicals change how we think and who we are



Bisphenol-A

What Is Bisphenol-A?

Bisphenol-A (BPA) is a high-volume production chemical used to make epoxy resin and polycarbonate plastic products, including certain water bottles, baby bottles, as well as food storage and heating containers.¹ It is also used in the epoxy resin lining of metal food cans and in dental sealants, carbonless paper,² and as an additive to certain plastics used in children's toys.^{1,3} The chemical was first developed as a synthetic estrogen and was later polymerized to produce polycarbonate.^{4,5} Bisphenol-A mimics estrogen activity and is known as an "endocrine disruptor," a chemical that interferes with hormonal systems in animals and humans and contributes to adverse health effects.¹ New scientific evidence is emerging that demonstrates exposure to bisphenol-A may affect multiple organs and systems within the bodies of animals and people.

How Are We Exposed to Bisphenol-A?

Humans are exposed to bisphenol-A on a daily basis through consumption of food and beverages contaminated with bisphenol-A, as well as environmental contamination. Polycarbonate plastic can become unstable over time and with use, allowing bisphenol-A to leach into material in contact with the plastic.⁵ Other consumer items, such as carbonless paper and DVDs, also contain bisphenol-A. Additionally, bisphenol-A is now found nearly everywhere in the environment and commonly found in dust particles, surface water and drinking water, as over 6 billion pounds are produced worldwide each year.⁶ Production of bisphenol-A releases approximately two hundred thousand pounds of the chemical into the atmosphere annually.⁷

Bisphenol-A in Our Bodies

A recent study by the U.S. Centers for Disease Control and Prevention (CDC) found that 92.6% of Americans now carry bisphenol-A in their urine at an average level of 2.6 µg/L (micrograms per liter).⁸

Although the United States Environmental Protection Agency (EPA) considers exposure to 50 µg/kg/day (micrograms per kilogram of body weight per day) of bisphenol-A safe, this standard

was set in 1993 and is based on studies from the 1980s.⁹ Several studies have suggested that humans are exposed to greater concentrations of BPA than 50 µg/kg/day.¹⁰ In a review of scientific literature on BPA conducted in 2005, researchers found numerous studies indicate a wide range of health effects from exposure to bisphenol-A at much lower doses (as low as 0.23 parts per trillion) than considered "safe" by the EPA.¹¹ In August 2007, over 30 scientific experts on bisphenol-A, known as the Chapel Hill panel, published a consensus statement in the peer-reviewed journal *Reproductive Toxicology*, stating significant evidence indicates adverse health effects occur in animals at levels within the range of exposure that is typical for humans living in developed countries.¹²

In 2007, the Canadian Government began several studies researching the effects of bisphenol-A on human health and the environment. They found that newborns and infants up to 18 months of age were exposed to levels of BPA similar to exposure levels associated with health effects in laboratory animals.¹³

More recently, the US National Toxicology Program (NTP) Center for the Evaluation of Risks to Human Reproduction (CERHR) released the *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol-A*, which contributed significantly to the discussion of bisphenol-A's health effects. This report identified evidence from animal studies which raises "some concern" that current exposure levels to bisphenol-A to human infants, fetuses, and children may result in developmental changes to the prostate gland and brain.¹⁴ Due to the concern about the effects of bisphenol-A on children under six, the NTP has teamed up with the CDC to investigate further the effects of bisphenol-A on young children, infants, and neonates.¹⁵ In infants, bisphenol-A remains in the body longer than adults due to the absence of a liver enzyme that is needed to break down bisphenol-A. One study indicated that infants can have levels up to eleven times higher than adults.¹⁶

What Does Exposure to Bisphenol-A Mean for Our Health?

The presence of environmental chemicals in the human body does not necessarily imply that they are causing adverse health

effects; however, environmental chemical exposures can and do affect human health. It is important to note that both the dosage and the timing of exposure have significant effects on any potential health outcome.

The following information is intended to inform the reader about the current state of knowledge on the health effects of Bisphenol-A, including both human and animals studies.

While the majority of research on bisphenol-A has been conducted on animals and cell cultures, there is strong evidence that similar effects occur in humans. The Chapel Hill panel concluded in August 2007, "Based on existing data we are confident ... the similar effects observed in wildlife and laboratory animals exposed to bisphenol-A predict that similar effects are also occurring in humans."¹² Moreover, research on estrogenic compounds all over the world is consistently finding that "animal studies of the effects of estrogenic substances are highly predictive of human impacts."¹⁷

Recent research has linked bisphenol-A to the following health effects:

Altered Brain Development and Behavior

BPA has estrogenic activity and as such alters both sexual development and neurobehavior.¹⁸ For some behaviors tested, results suggest that bisphenol-A exposure was linked to both **demasculinization** of males and **defeminization** of females.

At doses considered safe by the EPA, bisphenol-A blocked the receptors for estradiol in the hippocampus and prefrontal cortex of rodents and non-human primates.¹⁹ This signaling pathway may play a critical role in mood and cognition for humans.¹⁹

Research has shown that BPA inhibited the proliferation of neuronal progenitor cells. In fact, BPA inhibited development of these cells to a greater extent than the natural hormone 17 β -estradiol. Neural progenitor cells develop into neurons, and their growth is partially controlled by estradiol concentrations.²⁰ BPA was found to reduce nerve cell density in certain parts of the mouse brain. Interestingly, a lower dose (3 mg/g in food) of BPA had a stronger effect than the higher dose (8 mg/g in food).²¹ A study of mice found that prenatal exposure to BPA produced memory impairments. Researchers hypothesized that this occurred due to the observed decrease in acetylcholine in the brain, as acetylcholine is an important neurotransmitter associated with normal learning and memory.²²

Exposure to 10 μ g/kg/day of BPA decreased post-natal maternal behavior in female mice. This exposure also altered the behavior and development of the offspring either directly or through alteration of maternal behavior. There is also evidence that developmental exposure to BPA can impair dopaminergic circuits in the brain. Dopamine is one of many neurotransmitters that modify behavior and mood.²³

Primates exposed to BPA prenatally when mothers were given 10 μ g/kg/day of BPA showed significant behavioral changes, specifically regarding sex specific behaviors. Male rhesus

monkeys exhibited feminized behavior after BPA exposure *in utero*.²⁴ Prenatal and neonatal low-dose BPA exposure caused deficits in the ability to control physical behavior in rats.²⁵ BPA has also been found to significantly alter the behavior of female rats exposed to 40 μ g/kg/day during pregnancy and lactation. Female rats were found to exhibit less social behaviors characterized by a reduction of playful interactions.²⁶ Research also shows that prenatal BPA exposure is associated with altered behavior in 2-year old children, especially females.²⁷

Endocrine Disruption

As early as 1936, bisphenol-A was shown to be an environmental estrogen. Compared to natural estrogen, bisphenol-A is a less potent activator of the classic estrogen receptor, but in recent years it has been recognized that "BPA is equipotent with estradiol in its ability to activate responses via recently discovered estrogen receptors associated with the cell membrane," as found in several studies on cell culture and laboratory animals.¹² In addition to binding to estrogen receptors, evidence suggests that bisphenol-A also can change endogenous hormone synthesis, hormone metabolism and hormone concentrations in blood.¹² Exposure to bisphenol-A has been shown to cause changes in tissue enzymes and hormone receptors as well as interacting with other hormone-response systems.¹²

Reproductive Effects

Researchers found that women with a history of recurrent miscarriage had average blood serum levels of bisphenol-A at 2.59 ng/ml, more than three times higher than women with successful pregnancies,²⁸ a finding predicted by previous animal studies.²⁹ There is evidence that maternal exposure to BPA during pregnancy with a female fetus can cause formation of egg cells with an improper number of chromosomes in the fetus.³⁰ The abnormal eggs cells created during development remain during the reproductive lifespan. These abnormally developed eggs may be responsible for miscarriages, as well as genetic disorders such as Down's syndrome.³⁰ Several other studies have shown that BPA exposure disrupts normal egg production.^{31,32}

BPA has also been shown to increase the rate of development in some fish, causing premature hatching. The authors of the study speculate that endocrine disruptors such as BPA may play a role in the rising rates of preterm births in humans.³³ Preterm birth is a known risk factor for various diseases later in life.

Altered Mammary Gland Development:

In a laboratory study, mammary gland development during puberty was significantly altered in mice exposed to 25 ng/kg body weight/day of bisphenol-A perinatally.³⁴ This is 2,000 times lower than the EPA's safety standard for bisphenol-A (50 μ g/kg/day) and comparable to levels found in the environment.³⁵ Scientists suggest that this study's implications for human health include increased susceptibility to breast cancer after perinatal exposure to bisphenol-A.²⁹

Low doses of bisphenol-A have also been shown to decrease the effectiveness of chemotherapeutic agents used for breast cancer.³⁶

Prostate Cancer

Research using cell cultures showed that a concentration of bisphenol-A of 1 nM (nanomolar) made prostate cancer cells less responsive to the hormone treatment used to bring prostatic adenocarcinomas into remission.³⁷ Whether this cell culture impact also occurs in people is uncertain, but the concentration is lower than the average level of bisphenol-A found in Americans, as reported in 2008.⁸

Insulin Resistance and Cardiovascular Diseases

A recent study in adult mice provided evidence of a link between bisphenol-A exposure and increased risk of type II diabetes, hypertension and dyslipidemia.³⁸ In this study, scientists found that chronic exposure to low doses of bisphenol-A leads to insulin resistance in adult mice. Doses used in their experiments were 5 times lower than the dose considered safe by the EPA.⁹

Based on exposure data from 2003-2004, scientists examined links between urinary bisphenol-A levels in the U.S. population and chronic diseases in adults between the ages of 18 and 74.³⁹ They found that high urinary levels of bisphenol-A are associated with cardiovascular diagnoses. Adults in this study who had high urinary levels of BPA were also more likely to have diabetes. These effects were found at typical levels of exposure for the U.S. population.³⁹

Developmental Origins of Adult Health and Disease

In 2006, an expert panel of scientists convened to discuss the potential negative impact of BPA on human health. This panel stated that BPA exposure is within the range of biologically active concentrations in 95% of people.

The panel implicated BPA in the rise of multiple diseases including “prostate and breast cancer, uro-genital abnormalities in male babies, a decline in semen quality in men, early onset of puberty in girls, metabolic disorders including insulin resistant (type 2) diabetes and obesity, and neurobehavioral problems such as attention deficit hyperactivity disorder (ADHD).”

The panel further stated, “These developmental effects are irreversible and can occur due to low dose exposure during brief sensitive periods in development [in utero], even though no BPA may be detected when the damage or disease is expressed.”¹²

Regulations for Bisphenol-A

Federal regulation of toxic chemicals is critical as a measure to protect public health. However, according to the Environmental

Reducing Your Exposure

You can prevent or minimize exposure to bisphenol-A in the following ways:

- Use glass, stainless steel, or polyethylene bottles (PETE, PET, or #1; HDPE or #2; LDPE or #4) instead of polycarbonate (PC or #7) bottles.⁴⁶
- Avoid using polycarbonate higher temperatures⁴⁷ Use glass or ceramic containers instead.
- Cut back on consumption of canned foods to reduce exposure to bisphenol-A contamination from the interior coating of the container. Also, avoid canned foods with higher fat content, which may have higher levels of bisphenol-A.⁴⁷
- Before getting dental sealants, check with your dentist about the ingredients in the products they use, as some formulations may leach bisphenol-A.⁴⁷

Working Group, “The nation’s system of regulations for industrial chemicals like [bisphenol-A] are embodied in the Toxic Substances Control Act, a law passed in 1976, and the only major environmental or public health statute that has never been updated.”⁴⁰ Furthermore, “under this law, companies are not required to test chemicals for safety before they are sold, and are not required to track whether their products end up in people or the environment at unsafe levels.”⁴⁰ To date, the U.S. Food and Drug Administration has not performed a standard toxicology study or determined an Acceptable Daily Intake (ADI) for bisphenol-A.^{40,41}

Policies are currently being considered on a federal level that prohibit the sale of products that contain bisphenol-A.⁴² On March 13, 2009, leaders from the U.S. House of Representatives and the U.S. Senate introduced the BPA-Free Kids Act, legislation which would ban the use of bisphenol-A in all food and beverage containers.⁴³

Several states have also introduced legislation which would limit the sale of bisphenol-A, especially in children’s products. In 2008, Maine enacted the “Toxic Chemicals in Children’s Products Act,” legislation which will phase out the use of chemicals, including BPA, associated with cancer, reproductive toxicity, developmental toxicity, or endocrine disruption.⁴⁴ In 2009, both Minnesota and Connecticut enacted legislation which would phase out certain consumer products intended for children that contain BPA. In March 2009, Canada became the first nation to restrict the use of bisphenol-A in children’s products.⁴⁵

Endnotes

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Fact sheets on toxic chemicals for the *Mind, Disrupted* Biomonitoring Project provided by the Alaska Community Action on Toxics (www.akaction.net) and Commonwealth (www.commonweal.org). For more information, please visit the *Mind, Disrupted* website at www.minddisrupted.org, or contact Pam Miller at pkmiller@akaction.net or Sharyle Patton at spatton@igc.org.