

A Survey of Bisphenol A in U.S. Canned Foods

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Summary. Independent laboratory tests found a toxic food-can lining ingredient associated with birth defects of the male and female reproductive systems in over half of 97 cans of name-brand fruit, vegetables, soda, and other commonly eaten canned goods. The study was spearheaded by the Environmental Working Group (EWG) and targeted the chemical bisphenol A (BPA), a plastic and resin ingredient used to line metal food and drink cans. There are no government safety standards limiting the amount of BPA in canned food.

EWG's tests found:

- Of all foods tested, chicken soup, infant formula, and ravioli had BPA levels of highest concern. Just one to three servings of foods with these concentrations could expose a woman or child to BPA at levels that caused serious adverse effects in animal tests.
- For 1 in 10 cans of all food tested, and 1 in 3 cans of infant formula, a single serving contained enough BPA to expose a woman or infant to BPA levels more than 200 times the government's traditional safe level of exposure for industrial chemicals. The government typically mandates a 1,000- to 3,000-fold margin of safety between human exposures and levels found to harm lab animals, but these servings contained levels of BPA less than 5 times lower than doses that harmed lab animals.

BPA testing in canned food. We contracted with a national analytical laboratory to test 97 cans of food we purchased in March 2006 in three major, chain supermarkets in Atlanta, Georgia; Oakland, California; and Clinton, Connecticut. The lab tested 30 brands of food altogether, 27 national brands and 3 store brands. Among the foods we tested are 20 of the 40 canned foods most commonly consumed by women of childbearing age (CDC 2002), including soda, canned tuna, peaches, pineapples, green beans, corn, and tomato and chicken noodle soups. We also tested canned infant formula. The lab detected BPA in fifty-seven percent of all cans.

BPA is a heavily produced industrial compound that has been detected in more than 2,000 people worldwide, including more than 95 percent of 400 people in the United States. More than 100 peer-reviewed studies have found BPA to be toxic at low doses, some similar to those found in people, yet not a single regulatory agency has updated safety standards to reflect this low-dose toxicity. FDA estimates that 17% of the U.S. diet comprises canned food; they last examined BPA exposures from food in 1996 but failed to set a safety standard.

Recommendations

BPA is associated with a number of health problems and diseases that are on the rise in the U.S. population, including breast and prostate cancer and infertility. Given widespread human exposure to BPA and hundreds of studies showing its adverse effects, the FDA and EPA must act quickly to revise safe levels for BPA exposure based on the latest science on the low-dose

toxicity of the chemical.

BPA is at unsafe levels in one of every 10 servings of canned foods (11%) and one of every 3 cans of infant formula (33%)

Source: Chemical analyses of 97 canned foods by Southern Testing and Research Division of Microbac Laboratories, Inc., North Carolina.

EWG calculated people's BPA exposures from canned food using the following assumptions: Calculations reflect a single adult serving, using label serving size and body weight of 60 kg (132 lbs); exposures for concentrated infant formula is calculated for exclusively formula-fed infant using average 3-month-old body weight (6 kg/13 lbs) and average daily formula ingestion (840 g/30 oz); formula is assumed diluted with water free of BPA. Estimated single-serving exposures are compared against BPA dose of 2 ug/kg/d linked in lab studies to permanent damage of reproductive system from in utero exposures and referenced as "toxic dose" in figure above (see Section 3 of this report).

Summary of findings

Widespread exposures, no safety standards. In studies conducted over the past 20 years, scientists have detected BPA in breast milk, serum, saliva, urine, amniotic fluid, and cord blood from at least 2,200 people in Europe, North America, and Asia (CERHR 2006). Researchers at the Centers for Disease Control and Prevention recently detected BPA in 95% of nearly 400 U.S. adults (Calafat et al. 2005). EWG-led biomonitoring studies have detected BPA in people from four states and the District of Columbia (EWG 2007). BPA ranks in the top two percent of high production volume chemicals in the U.S., with annual production exceeding a billion pounds (EPA 2006), and is so common in products and industrial waste that it pollutes not only people but also rivers, estuaries, sediment, house dust, and even air nearly everywhere it is tested. Yet despite its ubiquity and toxicity, BPA remains entirely without safety standards. It is allowed in unlimited amounts in consumer products, drinking water, and food, the top exposure source for most people. The lack of enforceable limits has resulted in widespread contamination of canned foods at levels that pose potential risks. For instance, analysis of our tests reveals that for one of every five cans tested, and for one-third of all vegetables and pastas (ravioli and noodles with tomato sauce), a single serving would expose a pregnant woman to BPA at levels that fall within a factor of 5 of doses linked to birth defects — permanent damage of developing male reproductive organs (Figure 1).

EWG test results — BPA is common contaminant in name-brand canned foods heavily consumed by women and infants

Canned Foods	Number of brands tested	Number of cans tested	Foods tested	BPA % detect	Average BPA level* and range (ppb)
All foods	30	97	57%	7.9	(ND - 385)

Beans 3 6 baked beans 83% 9.7 (ND - 38)
 Fruit 6 17 mixed fruit, cranberry sauce, peaches, pears, pineapple 35% 2.3 (ND - 27)
 Infant formula 2 6 concentrated infant soy and milk-based formula 33% 2.4 (ND - 17)
 Meal replacement 2 5 liquid meal replacements 40% 4.2 (ND - 66)
 Milk products 3 evaporated milk 66% 3.5 (ND - 9)
 Pasta 2 6 ravioli, spaghetti 100% 63.5 (16 - 247)
 Soda 2 12 cola, diet cola 42% 1.7 (ND - 8)
 Soup 5 19 beef stew, chicken noodle, chicken rice, chicken vegetable, tomato, vegetable 89%
 57.6 (ND - 385)
 Tuna 2 6 chunk lite, solid white 50% 9.6 (ND - 108)
 Vegetable 8 17 corn, green beans, mixed vegetables, peas, tomatoes 41% 7.8 (ND - 330)

BPA concentrations are expressed in parts per billion (ppb) by weight (micrograms of BPA per kilogram of food).

* Average is the geometric mean. Non-detects considered to be 1/2 the detection limit (1 ppb) for purposes of this calculation.

Government assessments fail to consider BPA low-dose toxicity. As of December 2004, 94 of 115 peer-reviewed studies had confirmed BPA's toxicity at low levels of exposure. At some of the very lowest doses the chemical causes permanent alterations of breast and prostate cells the precede cancer, insulin resistance (a hallmark trait of Type II diabetes), and chromosomal damage linked to recurrent miscarriage and a wide range of birth defects including Down's syndrome (vom Saal 2005). Few chemicals have been found to consistently display such a diverse range of harm at such low doses.

Yet all of the most recent government reviews of bisphenol A have failed to set safety standards consistent with the chemical's low-dose toxicity. Each one either preceded the development of the low-dose literature, or heavily weighted industry-sponsored studies that are now known to have fundamental design flaws rendering them incapable of detecting BPA toxicity. U.S. safety reviews are described below:

- The U.S. EPA established its generic safety standard for BPA (the reference dose, or RfD) in 1987, a decade before the BPA low-dose literature was established (EPA 1987). The vast majority of studies finding BPA toxic at low doses have been published since 1997, the year that pivotal studies showed BPA's ability to harm the prostate at levels far below what was thought safe (Nagel 1997; vom Saal et al. 1997). EPA's safety standard is 25 times the dose now known to cause birth defects in lab studies (50 ug/kg/d vs. 2 ug/kg/d), and has not been updated for 20 years.
- The U.S. National Toxicology Program's 2001 assessment, which found BPA safe at low doses, relied heavily on industry-sponsored studies showing no low-dose BPA effects (NTP 2001). These studies are now known to have used animals resistant to the effects of estrogen-like chemicals such as BPA (vom Saal 2005). The NTP assessment considered studies published in

2000 or earlier. The six years following this review have seen the publication of dozens of low-dose BPA studies that substantially bolster the now near irrefutable evidence for low-dose effects.

- FDA published estimates of infant and adult BPA exposures 10 years ago. Even though the Agency did not then and has not since assessed the low-dose toxicity of BPA, in 2005 an FDA official asserted, in response to questions from a California legislator considering a state BPA phase-out bill, that "...FDA sees no reason to change [its] long-held position that current [BPA] uses with food are safe" (FDA 2005). FDA makes this assertion even though the Agency has not yet established an Acceptable Daily Intake (ADI) for BPA, and has not even conducted the Agency's standard, basic toxicology study to determine a safe dose for humans (FDA 2007). BPA's low dose toxicity. Companies began using BPA in metal can linings in the 1950s and 1960s (Schaefer 2004), fully twenty years after the chemical was first understood to be toxic (Dodds and Lawson, 1936 and 1938). These early warnings of toxicity were ignored or forgotten while companies steadily increased their reliance on BPA until it reached an annual U.S. production exceeding one billion pounds around 1990. In 1993 the chemical's signature toxic property, its ability to mimic estrogen, was accidentally discovered in a failed lab experiment (Krishnan et al. 1993), and the intervening years have witnessed the development of a body of low-dose science that has transformed our understanding of chemical toxicity.

Bisphenol A demonstrates the fallacy of nearly every long-standing tenet of government-style safety standards and traditional high-dose toxicology:

- Low doses and toxicity. Where traditional toxicology asserts that higher doses confer greater harm, bisphenol A tests show that low doses can be the most toxic of all, below the radar screen of the body's compensatory detoxifying mechanisms, or below overtly toxic doses that destroy the tissues under study. In one investigation a low dose of BPA produced a 70% higher growth rate of prostate cancer cells in lab animals than did higher doses (Wetherill et al. 2002). In another study lower doses of BPA resulted in higher rates of breast cell growth that can precede cancer (Markey 2001). ("Low doses" are typically defined as those that produce tissue concentrations at or below those in the typical range of human exposures.)
- Timing of the dose. While traditional methods set safety standards to control risks defined in adulthood, bisphenol A studies reveal that exposures at other times can confer far higher risks, especially in the womb and during early childhood. For example, recent studies show that prenatal exposure to BPA causes breast cancer in adult rats (Murray 2006), and causes genetic changes resulting in greater risk of prostate cancer in later life (Ho et al. 2006). In another study adult rats which had been dosed in the womb developed breast cancer in adulthood (Munoz-de-Toro 2005); these exposure levels during adulthood would not have caused cancer.
- Genetic susceptibility. Traditional toxicology holds that a chemical's potency and risks are constant, regardless of who is exposed. Bisphenol A suggests a different truth: A person's genetics plays an important role in defining risks and health outcomes from exposures to toxic chemicals. For instance, studies suggest that for some but not all babies, BPA accumulates in amniotic fluid, suggesting differing innate capacities for excretion that would be defined by

genetics (Yamada et al. 2002). A recent study of mammary gland development showed that animals exposed to BPA in utero are more likely to develop mammary tumors when they are exposed to carcinogenic chemicals later in life, compared to animals not previously exposed to BPA (Durando 2007). This study is one of many suggesting that early-life exposures to BPA may alter the expression or strength of genes to dramatically alter disease risk later in life.

Over the past year an average of four new BPA toxicity studies have been published in the peer-reviewed literature every month. New discoveries on BPA surface so routinely that the CERHR review document (CERHR 2006) describes fully 465 studies conducted primarily over the past 14 years. Among recent works:

- A study showing that BPA exposures lead to an error in cell division called aneuploidy that causes spontaneous miscarriages, cancer, and birth defects in people, including Down Syndrome (Hunt 2003).
- An investigation demonstrating that low doses of BPA spur both the formation and growth of fat cells, the two factors that drive obesity in humans (Masumo et al. 2002).
- A study linking low doses of BPA to insulin resistance, a risk factor for Type II diabetes (Alonso-Magdalena 2006).
- A preliminary investigation linking BPA exposures to recurrent miscarriage in a small group of Japanese women, made potentially pivotal by its concordance with lab studies of BPA-induced chromosome damage that could well cause miscarriage (Sugiura-Ogasawara 2005).

The unusually broad toxicity of BPA is explained by a prominent scientist as stemming from the fact that BPA can alter the behavior of over 200 genes — more than one percent of all human genes (Myers 2006). These genes control the growth and repair of nearly every organ and tissue in the body. Taken in its totality, the range of toxic effects linked to BPA is startlingly similar to the litany of human health problems on the rise or common across the population, including breast and prostate cancer, diabetes, obesity, infertility, and polycystic ovarian syndrome (Myers 2006).

Studies show that BPA is toxic to lab animals at doses overlapping with or very near to human exposures, and that the chemical causes toxic effects that are on the rise or very common in people. These disturbing facts raise questions about the extent to which current, widespread exposures to BPA are contributing to the burden of human disease.

Were the federal government to develop safety standards reflecting any of the more than 200 low-dose studies of BPA toxicity, the chemical would become the first widespread industrial compound with a government-recognized, harmful dose at such remarkably low levels that in some cases appear to overlap with human exposures. The science would fully justify a strict safety standard and would force industry to change food packaging to dramatically decrease the widespread BPA exposures to which they are currently subjecting the public.

FDA fails to protect the public. FDA is responsible for ensuring that food packaging chemicals like BPA are safe. In the case of BPA, the Agency has deemed the chemical safe even though its own exposure estimates for infants exceed doses shown to permanently harm the developing male reproductive system.

FDA does not restrict BPA levels in food. In the wake of a 1993 experiment proving that BPA disrupts estrogen levels, FDA tested 14 cans of infant formula and a few foods that adults eat, calculated exposures from these tests, and found them to be within safe levels (CERHR 2006). To make this determination the Agency compared the estimated exposures to "safe" doses far higher than those now known to cause permanent harm to lab animals.

Dr. George Pauli, at the time FDA's associate director for science and policy, offered this rationale: "FDA sees no reason at this time to ban or otherwise restrict the uses now in practice" (FDA 2005). Never mind that the Agency's estimated exposures for infants, at 15 to 24 ug/kg/d, exceed by a factor of up to 10 the dose shown to permanently alter prostate gland growth.

Bisphenol A is just one of hundreds of chemicals that pollute people - proof of critical need to reform our system of public health protections. Studies by European scientists show that BPA is just one of many chemicals that leach out of food can linings. Tests of just three can coatings found at least 23 different BPA-related chemicals leaching into food, all without legal limits (Schaefer 2004). Research shows these contaminants occur at levels that can dwarf better-known environmental pollutants that accumulate in food, like PCBs and DDT. One scientist writes that "Concentrations of [migrant chemicals like BPA] commonly exceed...pesticides by orders of magnitude; most of the migrating compounds are not even identified; and only a few have been tested for toxicity..." (Grob 1999).

FDA has tallied more than 1,000 indirect food additive chemicals in packaging and food processing, but food is just one of the many ways humans are exposed to industrial chemicals. EWG research reveals more than 200 pollutants in tap water supplies across the country; thousands of chemicals in cosmetics and personal care products; 470 industrial chemicals and pesticides in human tissues; and an average of 200 pollutants in each of 10 babies tested at the moment of birth. Nothing is known about the safety of the complex mixtures of low doses of a myriad of industrial chemicals in the human body.

The nation's system of public health protections from industrial chemicals like BPA are embodied in the Toxic Substances Control Act, a law passed in 1976 that is the only major environmental or public health statute that has never been updated. 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 at unsafe levels. As a result of this broken system, BPA is now one of the most widely used industrial chemicals, is found at unsafe levels in people, is allowed in unlimited quantities in a broad range of consumer products, and is entirely without safety standards. BPA gives irrefutable proof that our system of public health protections must be strengthened to protect children and others most vulnerable to chemical harm.