



Sign up to get e-mail alerts
e-mail address

[ISSUES](#) [NEWS](#) [HOW YOU CAN HELP](#) [RESULTS](#) [ABOUT](#)
[US](#) [RESEARCH & POLICY CENTER](#)

ISSUES

OCEANS

Save Our Ocean Legacy
Stop Offshore Drilling

ENERGY

Million Solar Roofs
Fuel Efficient Cars
Clean Energy L.A.
Smart Energy Cities Project

PRESERVATION

Wild Forests

ENVIRONMENTAL HEALTH

Stop Toxic Toys
Detecting Toxic Chemicals
Healthy Day Cares

CLEAN WATER

Safe Drinking Water

CLEAN AIR

GLOBAL WARMING

STATE LEGISLATURE

2006 Legislative Agenda
2005 Legislative Scorecard

[Home](#) » [Issues](#) » [Environmental Health](#) » [Stop Toxic Toys](#) » [Phthalates Overview](#)

Phthalates Overview

Phthalates (pronounced "tha-lates") are a common class of chemicals used in many household products and polyvinyl chloride (PVC) plastic to improve flexibility, and in cosmetics to bind fragrance to the product. Different types of phthalates include diethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), butyl benzyl phthalate (BBP), di-isodecyl phthalate (DIDP), di-isononyl phthalate (DINP), di-n-octyl phthalate (DNOP), and many others. Global phthalate production is estimated at 11 billion pounds per year.[\[1\]](#)

The vinyl molecules making up PVC plastic form an attraction to one another to produce a very brittle plastic. To make the PVC plastic soft or flexible, a plasticizer—one of the phthalates—is added to allow the molecules to slide against each other. Semi-rigid PVC contains about 10% phthalates; flexible PVC, as much as 50% by weight. Because phthalates are not chemically bonded to PVC molecules, phthalates are given off freely by PVC (called "offgassing"). Offgassing increases with mechanical stress (i.e., bending, pressure, chewing) and exposure to solvents such as fats, oils, saliva, and temperatures over 85° F.

In brief, this summary reveals that there is an extensive scientific literature reporting adverse effects of phthalates and substantial evidence that levels of the phthalates of concern are found in humans at levels associated with adverse effects.[\[2\]](#) Population studies show that virtually everyone carries some level of phthalates in their body. For the general population, the oral route of exposure is considered a major route.[\[3\]](#)

• Significant Government Action Taken Against Phthalates

California Action: Four phthalates—DEHP, BBP, DBP, and DnHP—are listed on the Proposition 65 list. California has also noticed its intent to list DIDP.[\[4\]](#) The Office of Environmental Health Hazard Assessment (OEHHA) of California EPA listed DEHP as a carcinogen in 1988 and as a developmental toxin and male reproductive toxin in 2003.[\[5\]](#) In December 2005, OEHHA listed three additional phthalates—BBP as a developmental toxin; DBP as a developmental toxin and female and male reproductive toxin; and DnHP as a female and male reproductive toxin.[\[6\]](#)

In June 2006, San Francisco became the first jurisdiction in the United States to enact a prohibition on the use of DEHP, DBP, and BBP in all toys and child care articles and prohibition on the use of DINP, DIDP, and DNOP in toys and child care articles intended for use by children under three years of age that can be put in the mouth.[\[7\]](#)

Federal Action

In 2000, the National Toxicology Program (NTP) of the U.S. Department of Health & Human Services reviewed the scientific literature on phthalates and called for additional scientific studies to be conducted. For the most part, these reviews are outdated, especially in light of the explosion of scientific studies on the effects of phthalates in the past five years. In particular, all of the human studies relating phthalates to adverse effects in humans were conducted after the NTP reports were issued. Even without the most recent studies, however, the NTP concludes:

DEHP: In October 2005, an expert panel of the NTP reaffirmed its 2000 finding that DEHP poses a risk to human development and fertility.^[8] In its second review of DEHP in five years, the NTP concluded that DEHP causes reproductive and developmental damage in animal studies and these studies are relevant to humans, especially infants, children, and pregnant and lactating women.

BBP: In October 2000, an expert panel of the NTP concluded that “oral exposure to BBP can cause reproductive toxicity in adult rats and developmental toxicity in rats and mice. These data are assumed to be relevant to humans.”^[9]

DBP: In October 2000, an expert panel of the NTP concluded that “DBP is developmentally toxic to both rats and mice by the oral routes; it induces structural malformations.”^[10]

DINP: The October 2000 report of the NTP expert panel found that “[e]xposure of children to DINP through children’s products is a public concern.”^[11] Furthermore, “[c]hildren may be exposed to higher levels of DINP (up to 10–100 fold higher) than adults because infants and small children mouth toys and other articles that contain DINP that can migrate into saliva and be swallowed.”

DNOP: The October 2000 report of the NTP found that “[b]ased on experimental literature, including toxicity studies in rats and mice with DnOP and other structurally-related phthalates, there is a reasonable basis for assuming relevance of these data for judging potential hazard to humans.”^[12]

DIDP: The October 2000 report of the NTP found that “[c]hildren may have higher levels of exposure to DIDP than adults because infants and small children mouth toys and other objects that may contain DIDP which can migrate into saliva and be swallowed.”^[13] The panel concluded that, based on the results of the toxicology studies on rats showing effects on the developing skeletal system following oral exposure to DIDP, oral exposure to children and pregnant women should be examined.

International Action: The European Union and many countries have restricted the use of phthalates in children’s toys. The European Union has banned DEHP, DBP, and BBP in all toys and child care articles and banned DINP, DIDP, and DNOP in toys and child care articles that can be put in the mouth. Prior to the EU’s permanent ban, the following countries also had banned phthalates in children’s toys: Argentina, Austria, Cyprus, Czech Republic, Denmark, Fiji, Finland, Germany, Greece, Italy, Japan, Mexico, Norway, and Sweden. In many other countries, governments have requested voluntary industry action to remove phthalates, in some cases industry has voluntarily removed phthalates, and governments have issued health advisories related to phthalates.

• Children are Most at Risk

Growing children are particularly at risk to chemicals in their environment because they face greater exposure and are physiologically more susceptible to them.^[14] Children's exposures begin at conception, as chemicals, including phthalates, cross the placenta in a pregnant woman's body^[15] and can affect the embryo or fetus during critical periods of development. Even after birth, children's bodies remain immature, with underdeveloped detoxification mechanisms to protect them from phthalates. Their brains and other organ systems are constantly developing, undergoing periods of particular sensitivity to damage or disruption. Especially because growing children are particularly at risk from phthalate exposure, precautionary measures must be taken to protect children from such exposure from products they use everyday.

• Phthalate Levels in Humans Reach Harmful Levels Found in Studies

In 2000, the first exposure report on adults by the U.S. Centers for Disease Control (CDC) revealed high levels of phthalates in all 289 adult Americans tested.^[16] CDC scientists announced that levels of some phthalates in women of childbearing age, including DBP and DEHP, exceeded the government's safe levels set to protect against birth defects.^[17] The scientists concluded that "[f]rom a public health perspective, these data provide evidence that phthalate exposure is both higher and more common than previously suspected." Soon thereafter, scientists published a study looking at levels of several phthalates in children living in the Imperial Valley, California, finding phthalates in all children examined with levels higher on average than had been reported for adults in 2000.^[18] The scientists concluded that "DBP, BBP, and DEHP exposure on a body weight basis may be at least twice as high for these children compared to the adults in [the initial study]."

In 2003, the CDC confirmed widespread contamination with the largest and most extensive U.S. survey of human chemical contamination to date, finding phthalates in virtually every person tested and the highest levels in children and women of reproductive age, demonstrating the potential for developmental effects on the fetus and children.^[19] For example, the biologically active metabolites of DEHP, BBP, and DBP—which are used in children's toys^[20]—were highest among children. According to the CDC's most recent chemical exposure report in 2005, phthalates are found in virtually 100% of the population.^[21] Of the 12 phthalates tested by the CDC, eleven were concentrated more highly in children than in adults. Concentrations were also higher in females than males.

Studies demonstrate reproductive toxicity at levels to which some people are currently exposed.

• Dangers of Phthalates are Confirmed by Weight of the Science

Some of the adverse health effects of phthalates include:

Early puberty in girls: Premature breast development in young girls is associated with exposure to DEHP. A study of Puerto Rican girls documents concentrations of DEHP seven times greater in girls suffering from premature breast development—girls with an average age of 31 months—than the control group.^[22]

Premature delivery: Rates of premature birth have been steadily rising at least over the last two decades.[\[23\]](#) A study published in November 2003 provides a link between exposure to phthalates and pre-term birth. The scientists found phthalates and their breakdown products in the blood of newborn infants, with higher levels leading to a higher incidence of premature delivery.[\[24\]](#) Babies exposed to phthalates entered the world a week earlier on average than babies with less exposure. The scientists concluded that “phthalate exposure is significantly associated with a shorter pregnancy duration.” Premature babies have an elevated risk for a wide range of diseases throughout the remainder of life.

Impaired sperm quality and sperm damage in men: Several studies show an association between phthalate exposure and sperm damage and impaired sperm quality in men. In three studies in particular, the phthalate levels associated with the damage were well within the range experienced by many American men. In one study, Harvard and CDC scientists show men with higher phthalate levels are more likely to have low sperm count and impaired sperm quality.[\[25\]](#) The highest phthalate concentrations were found in men with the lowest sperm counts. Notably, American men often have phthalate levels two to three times higher than those associated with sperm damage found in this study. In another study by the same research group, the scientists discovered that sperm DNA damage, including reduced sperm count, lower sperm motility, and deformed sperm, is more likely in men with elevated phthalate levels—such as DBP, BBP, and DEP.[\[26\]](#) Finally, another study found significantly higher levels of phthalates in infertile men whose sperm was abnormal or had DNA damage.[\[27\]](#)

Genital defects and reduced testosterone production in boys: In a recent study of fetuses exposed to phthalates in the womb, Dr. Shanna Swan and her colleagues found a strong relationship between phthalates and changes in the size and anatomy of the genitalia of male babies and toddlers. Mothers with the highest levels of phthalates, including DBP, DEP, and DIBP, in their urine late in their pregnancies had babies with a shorter anogenital distance (the space between the anus and penis that forms into the scrotum in males), smaller penises, and more instances of incompletely descended testicles.[\[28\]](#) The adverse effects were seen at phthalate levels below those found in one-quarter of women in the U.S., based on CDC data. The pattern of genital changes seen in these baby boys is consistent with the “phthalate syndrome” (i.e., increased frequency of undescended testicles and genital deformations and impaired sperm quality) observed in rodents prenatally exposed to phthalates and suggestive of “testicular dysgenesis syndrome,” a human health condition with the same characteristics and linked to phthalate exposure. An important component of this syndrome is an increased risk of testicular cancer. Prior to Dr. Swan’s human study, animal studies had shown that phthalates could cause such reproductive defects in male rodents. The similarities between the male reproductive defects induced by phthalates in rodents and the features of male birth defects seen in humans are strong.[\[29\]](#) In rodents, interference with testosterone production during male development has effects on a wide array of developmental processes, not just the genital tract. Long term changes in brain development and behavior are well-documented consequences.

Another recent study looking at the impact of phthalate exposure on over 100 three-month-old baby boys in Denmark and Finland showed reduced

testosterone production and other endocrine abnormalities.[30] The data on reproductive hormone profiles and phthalate exposures in newborn boys provides additional evidence that human testicular development and function may be vulnerable to perinatal exposure to some phthalates, including DBP, DEHP, DEP, and DINP. The findings are thus consistent with the above study showing incomplete masculinization of infant boys exposed to phthalates prenatally.

Genital defects and testicular cancer: Several different types of phthalates are implicated in genital abnormalities, [31] one of which is a risk factor for testicular cancer. [32] In 2000, Dr. Earl Gray of U.S. EPA reported that three types of commonly used phthalates—DEHP, BBP, and DINP—disrupt sexual development in male rats, resulting in hypospadias (a birth defect causing the opening of the urinary tract to develop on the underside of the penis), cleft phallus, reduced testes weight, and other reproductive malformations such as cryptorchidism (undescended testicles).[33] DEHP reduces testosterone production in the developing testes, interfering with the signals that direct normal male reproductive development.[34] Pregnant rats fed DEHP after the second week of pregnancy produced male offspring with reduced testosterone levels in the testes to the same level as in female rodents. In 2004, Dr. Gray and others at the EPA followed up on this finding, showing that the phthalates DEHP, BBP, and DINP reduce the levels of insulin-like hormone #3. Reduced activity of this hormone is another known cause of undescended testicles in mice.[35] Other research groups have implicated DBP as a direct cause of hypospadias and cryptorchidism in rodents. When female rats are fed DBP during the third week of pregnancy, 60% of their male offspring suffer cryptorchidism, hypospadias, infertility, and/or other testicular defects.[36]

[1] Anne Platt McGinn, Worldwatch Institute, *Why Poison Ourselves? A Precautionary Approach to Synthetic Chemicals*, Worldwatch Paper 153, ISBN: 1-878071-55-6, November 2000.

[2] Environment California Research & Policy Center would like to thank the authors of *Our Stolen Future*, Dr. Theo Colborn, Dianne Dumanoski, and Dr. Pete Myers, for their updates on the science of endocrine disruption found at www.ourstolenfuture.org from which we obtained much of the valuable information contained herein.

[3] U.S. Centers for Disease Control and Prevention, *Third National Report on Human Exposure to Environmental Chemicals*, July 2005.

[4] California Regulatory Notice Register (Register 2005, No. 9-Z), March 4, 2005.

[5] OEHHA, California EPA. Chemicals Known to the State to Cause Cancer or Reproductive Toxicity as of December 2, 2005, http://www.oehha.ca.gov/prop65/prop65_list/files/P65single120205.pdf (accessed December 2005).

[6] OEHHA, California EPA. Chemicals Listed Effective December 2, 2005 as Known to the State of California to Cause Reproductive Toxicity, December 2, 2005, http://www.oehha.ca.gov/prop65/prop65_list/120205list.html (accessed December 2005).

[7] San Francisco Ordinance no. 120-06 (2006). Effective date is December 1, 2006.

[8] 70 Fed. Reg. 69567 (Nov. 16, 2005).

[9] National Toxicology Program, U.S. Dept. of Health and Human Services, NTP-CERHR Expert Panel Report on Butyl Benzyl Phthalate, October 2000.

[10] National Toxicology Program, U.S. Dept. of Health and Human Services, NTP-CERHR Expert Panel Report on Di-n-butyl Phthalate, October 2000.

[11] National Toxicology Program, U.S. Dept. of Health and Human Services, NTP-CERHR Expert Panel Report on Di-isononyl Phthalate, October 2000.

[12] National Toxicology Program, U.S. Dept. of Health and Human Services, NTP-CERHR Expert Panel Report on Di-n-octyl Phthalate, October 2000.

[13] National Toxicology Program, U.S. Dept. of Health and Human Services, NTP-CERHR Expert Panel Report on Di-isodecyl Phthalate, October 2000.

[14] Philip J. Landrigan et al, Pesticides in the Diets of Infants and Children, National Academy Press (1993).

[15] Dostal LA, Weaver RP, Schwetz BA. Transfer of di(2-ethylhexyl) phthalate through rat milk and effects on milk consumption and the mammary gland. Toxicol Appl Pharmacol. 91:315 –325 (1987);

Parmar D, Srivastava SP, Srivastava SP, Seth PK. Hepatic mixed function oxidases and cytochrome P-450 contents in rat pups exposed to di-(2-ethylhexyl)phthalate through mother's milk. Drug Metab Dispos. 13:368 – 370 (1985);

Srivastava S, Awasthi VK, Srivastava SP, Seth PK. Biochemical alterations in rat fetal liver following in utero exposure to di(2-ethylhexyl)phthalate (DEHP). Indian J Exp Biol. 27:885 –888 (1989).

[16] BC Blount et al, "Levels of Seven Urinary Phthalate Metabolites in a Human Reference Population," Environmental Health Perspectives 108: 979-982 (2000).

[17] J Houlihan and R Wiles, Environmental Working Group, Beauty Secrets: Does A Common Chemical in Nail Polish Pose Risks to Human Health?, November 2000.

[18] Brock, JW, SP Caudill, MJ Silva, LL Needham, and ED Hilborn. 2002. Phthalate Monoester Levels in the Urine of Young Children. Bulletin of Environmental Contamination and Toxicology 68:309–314 (2002).

[19] U.S. Centers for Disease Control and Prevention. 2003. Second National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Environmental Health, Division of Laboratory Sciences (2003);

Manori J Silva et al, "Urinary Levels of Seven Phthalate Metabolites in the

U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999-2000," *Environmental Health Perspectives* 112: 331-338, March 2004.

[20] R Gibson and M Purvis, Environment California Research & Policy Center, *The Right Start: The Need to Eliminate Toxic Chemicals from Baby Products*, October 2005.

[21] U.S. Centers for Disease Control and Prevention. 2005. *Third National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Environmental Health, Division of Laboratory Sciences.

[22] I. Colón, D Caro, CJ Bourdony and O Rosario, "Identification of Phthalate Esters in the Serum of Young Puerto Rican Girls with Premature Breast Development," *Environmental Health Perspectives* 108: 895-900 (2000).

[23] AM Branum and KC Schoendorf, "Changing Patterns of Low Birthweight and Preterm Birth in the United States, 1981-98," *Paediatric and Perinatal Epidemiology*, 16: 8-15, January 2002;

Cande Ananth et al, "Rates of Preterm Delivery among Black Women and White Women in the United States over Two Decades: An Age-Period-Cohort Analysis," *American Journal of Epidemiology* 154: 657-665 (2001).

[24] G Latini et al, "In-Utero Exposure to Di-(2-ethylhexyl)-phthalate and Human Pregnancy Duration," *Environmental Health Perspectives* 111:1783-1785 (2003).

[25] Duty, SM, MJ Silva, DB Barr, JW Brock, L Ryan, Z Chen, RF Herrick, DC Christiani and R Hauser. *Phthalate Exposure and Human Semen Parameters*. *Epidemiology* 14:269 -277 (2003).

[26] Duty, SM, NP Singh, MJ Silva, DB Barr, JW Brock, L Ryan, RF Herrick, DC Christiani and R Hauser 2003. The relationship between environmental exposures to phthalates and DNA damage in human sperm using the neutral comet assay. *Environmental Health Perspectives* 111:1164-1169 (2003).

[27] Rozati, R, PP Reddy, P Reddanna and R Mujtaba. 2002. Role of environmental estrogens in the deterioration of male factor fertility. *Fertility and Sterility* 78:1187-1194 (2002).

[28] Shanna H. Swan, Katharina M. Main, Fan Liu, Sara L. Stewart, Robin L. Kruse, Antonia M. Calafat, Catherine S. Mao, J. Bruce Redmon, Christine L. TERNAND, Shannon Sullivan, J. Lynn Teague, and the Study for Future Families Research Team, *Decrease in Anogenital Distance among Male Infants with Prenatal Phthalate Exposure*. *Environmental Health Perspectives* 113: 1056-1061 (2005).

[29] Jane Fisher, "Environmental Anti-Androgens and Male Reproductive Health: Focus on Phthalates and Testicular Dysgenesis Syndrome," *Reproduction* 127: 305-315 (2004).

[30] Katharina M. Main, Gerda K. Mortensen, Marko M. Kaleva, Kirsten A. Boisen, Ida N. Damgaard, Marla Chellakooty, Ida M. Schmidt, Anne-Maarit Suomi, Helena E. Virtanen, Jørgen H. Petersen, Anna-Maria Andersson, Jorma Toppari, and Niels E. Skakkebaek, *Human Breast Milk Contamination*

with Phthalates and Alterations of Endogenous Reproductive Hormones in Three Months Old Infants, Environmental Health Perspectives doi:10.1289/ehp.8075 available via <http://dx.doi.org> [Online 7 September 2005].

[31] Gray, LE, C Wolf, C Lambright, P Mann, M Price, RL Cooper and J Ostby. 1999. Administration of potentially antiandrogenic pesticides (procymidone, linuron, iprodione, chlozolinate, p,p'-DDE, and ketoconazole) and toxic substances (dibutyl- and diethylhexyl phthalate, PCB 169, and ethane dimethane sulphonate) during sexual differentiation produces diverse profiles of reproductive malformations in the male rat. Toxicology and Industrial Health 15:94-118 (1999).

[32] As cited in, CG Ohlson and L Hardell, "Testicular Cancer and Occupational Exposures with a Focus on Xenoestrogens in Polyvinyl Chloride Plastics," Chemosphere 40: 1277-1282, May-June 2000.

[33] LE Gray et al, "Perinatal Exposure to the Phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, Alters Sexual Differentiation of the Male Rat," Toxicological Science 58: 350-365, December 2000.

[34] Louise Parks et al, U.S. EPA, "The Plasticizer Diethylhexyl Phthalate Induces Malformations by Decreasing Fetal Testosterone Synthesis during Sexual Differentiation in the Male Rat," Toxicological Sciences 58, 339-349 (2000).

[35] Vickie Wilson et al, "Phthalate Ester-Induced Gubernacular Lesions are Associated with Reduced Ins13 Gene Expression in the Fetal Rat Testis," Toxicology Letters 146: 207-215 (2004).

[36] JS Fisher et al, "Human 'Testicular Dysgenesis Syndrome': A Possible Model Using in-utero Exposure of the Rat to Dibutyl Phthalate," Human Reproduction 18: 1383-1394 (2003).

Clean air. Clean water. Open spaces.

[Contact Us](#)

[Privacy Policy](#)

[Jobs](#)

[Site Map](#)

[Search](#)

3435 Wilshire Blvd. #385 • Los Angeles, CA 90010

Phone (213) 251-3688 • Fax (213) 251-3699

E-mail: info@environmentcalifornia.org

Top Photos Courtesy of the U.S. Fish & Wildlife Service and the National Park Service