Order Code RL34572

CRS Report for Congress

Phthalates in Plastics and Possible Human Health Effects

Updated July 29, 2008

Linda-Jo Schierow
Specialist in Environmental Policy
Resources, Science, and Industry Division

Margaret Mikyung Lee
Legislative Attorney
American Law Division

Congressional Research Service

Prepared for Members and Committees of Congress

Phthalates in Plastics and Possible Human Health Effects

Summary

Roughly a dozen chemicals known as phthalates are used to make the plastics found in thousands of consumer products, ranging from medical tubing to automotive dashboards to bath toys. These phthalates are not tightly held by the plastics and are released into the environment over time. Congress is concerned about possible human health effects from exposure to six of these chemicals: di-(2-ethylhexyl) phthalate (DEHP), dibutyl phthalate (DBP), benzyl butyl phthalate (BBP), diisononyl phthalate (DINP), diisodecyl phthalate (DIDP), and di-n-octyl phthalate (DnOP). DEHP, DBP, BBP, and (to less extent) DINP are known to be toxic to the reproductive systems of rodents. Recent experiments demonstrate that pre-natal exposure at a sufficient level to these same phthalates disrupts the normal action of hormones and can cause malformations of the reproductive organs of offspring (especially males).

Disruption of hormonal functions in humans is known to result in abnormal reproductive development. Many scientists believe that the phthalates toxic to rodents might be able to cause similar malformations in humans. However, human health effects of phthalate exposure have not been conclusively demonstrated. Very few studies have looked at possible effects in humans, but their results have been consistent with the results of rodent experiments. More research would be needed to test this hypothesis. Recent surveys have found almost universal exposure to phthalates. Individuals may be exposed to high enough levels of phthalates to cause reproductive abnormalities. Scientists at the National Toxicology Program have expressed "serious concern" about human male infants undergoing intensive medical procedures, and "concern" about development of human males less than a year old who are exposed to DEHP. In light of these concerns, the National Academy of Sciences is evaluating the risk of aggregate human exposure to multiple phthalates.

Federal agencies have taken several actions, some as early as the mid 1980s, to evaluate and regulate phthalates, but no product to date has been banned outright. The agency responsible for regulating toys and most other child-care products is the Consumer Product Safety Commission (CPSC). In March 2008, the Senate approved an amendment to H.R. 4040, the Consumer Product Safety Commission Reform Act, that would restrict the use of six phthalates in toys and child-care products. The House version had no phthalate amendment. On July 29, 2008, the conferees announced approval of an amended version of the Senate provision.

The scientific basis for concerns about human health risks appears to be strong in the case of some phthalates (such as DEHP), adequate with respect to others (perhaps DINP), and weak for the remaining chemicals (for example, DIDP and DnOP). The strongest evidence with respect to developmental effects has been produced since about the year 2000. The Senate amendment would codify the voluntary agreements reached by CPSC with product manufacturers and reduce exposure to one particular phthalate. New formulations for toys and child-care products may pose greater or fewer risks than current formulations.

Contents

Phthalates in Plastics and Possible Human Health Effects

Introduction

"Phthalates"[1] refers to a group of chemical compounds that are heavily produced and widely used to make the plastics found in thousands of consumer products. The most common use of phthalates is to increase the flexibility of polyvinyl chloride (PVC) and polyvinylidene chloride (PVDC) polymers. Phthalates are released from those products over time, and are dispersed to the air, water, soil, and living things. Some (but not all) of these phthalates are known to cause reproductive damage in rodents. Recent interest by governmental bodies, including Congress, in the potential adverse human health effects that might be related to phthalate exposure has focused on six phthalates that are produced and used in very large quantities.[2] The six phthalates are di-(2-ethylhexyl) phthalate (DEHP), dibutyl phthalate (DBP), benzyl butyl phthalate (BBP), diisononyl phthalate (DINP), diisodecyl phthalate (DIDP), and di-n-octyl phthalate (DnOP). H.R. 4040, the Consumer Product Safety Commission Reform Act, as amended and approved by conferees, would prohibit the sale of children's toys and child care articles that contain more than 0.1% of DEHP, DBP, or BBP. A similar ban would apply for DINP, DIDP, and DnOP until the Consumer Product Safety Commission issues a rule either establishing or eliminating the ban on a permanent basis.

Background

Health Effects

Compared to some other chemicals in commerce, phthalates are not extremely toxic. That is, they do not cause acute illness after a short period of low-level exposure. However, controlled experiments with rodents have demonstrated that some phthalates[3] at high doses damage reproduction and development.[4] Moreover,

---

[1] The ph is silent.

[2] A high-production-volume chemical is defined by the U.S. Environmental Protection Agency as a chemical produced in amounts exceeding 1 million pounds annually. Hundreds of millions of pounds of phthalates are produced annually in the United States.

[3] Di-(2-ethylhexyl) phthalate, dibutyl phthalate, and benzyl butyl phthalate.

[4] A high dose is relative and varies depending on the chemical. For DEHP, for example, a high dose might be considered to be roughly more than 100 milligrams of phthalate (mg) per

(continued...)

if administered at sufficient levels[5] and at the appropriate time to pregnant females, some phthalates can cause malformations of the reproductive organs of offspring, especially males.[6] In rats, exposure during gestation to some phthalates can cause testicular cancer in mature offspring.[7] The higher the phthalate exposure, the more frequent and severe are the effects on the reproductive system. Rat fetuses are most susceptible, but older rats can also be affected at somewhat higher levels of exposure.[8]

Disruption of hormonal functions in humans is known to result in abnormal reproductive development. Many scientists believe that the phthalates toxic to rats and mice might be able to cause similar malformations in humans, because the male hormones affected by phthalates are important to the normal development of the male reproductive tract in *all* species of mammals. However, human health effects of phthalate exposure have not been conclusively demonstrated. Very few studies have looked at possible effects in humans,[9] but their results have been consistent with the

---

[4] (...continued)
kilogram of the exposed animal's body weight (kg) per day.

[5] For example, 14-23 milligrams of DEHP per kilogram of body weight per day.

[6] Gray, Jr., L. Earl, Joseph Ostby, Johnathan Furr, et al. 2000. Perinatal exposure to the phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, alters sexual differentiation of the male rat. *Toxicological Sciences*, v. 58, n. 2, p. 350-365.

Mylchreest, E., R.C. Cattley, and P. M. Foster. 1998. Male reproductive tract malformations in rats following gestational and lactational exposure to Di(n-butyl) phthalate: an antiandrogenic mechanism? *Toxicological Sciences*, v. 43, n. 1, p. 47-60.

Parks, Louise G., Joe S. Ostby, Christy R. Lambright, et al. 2000. The plasticizer diethylhexyl phthalate induces malformations by decreasing fetal testosterone synthesis during sexual differentiation in the male rat. *Toxicological Sciences*, v. 58, p. 339-349.

Wilson, Vickie S., Christy Lambright, Johnathan Furr, et al. 2004. Phthalate ester-induced gubernacular lesions are associated with reduced ins13 gene expression in the fetal rat testis. *Toxicology Letters*, v. 146, p. 207-215.

[7] Fisher, Jane S., S. Macpherson, N. Marchetti, et al. 2003. Human 'testicular dysgenesis syndrome': a possible model using inutero exposure of the rat to dibutyl phthalate. *Human Reproduction*, v. 18, n. 7, p. 1383-1394.

[8] Ibid.

U.S. Congress. House of Representatives. Committee on Energy and Commerce, Subcommittee on Commerce, trade, and consumer Protection. Hearing on Safety of Phthalates and Bisphenol-A in Everyday Consumer Products. June 10, 2008. Written testimony of Leon Earl Gray, Jr., Senior Reproductive Biologist and Toxicologist, U.S. EPA.

[9] Main, Katharina M., Gerda K. Mortensen, Marko M. Kaleva, et al. 2006. Human breast milk contamination with phthalates and alterations of endogenous reproductive hormones in infants three months of age. *Environmental Health Perspectives*, v. 114, p. 270-276.

Swan, Shanna H., Katharina M. Main, Fan Liu, et al. 2005. Decrease in anogenital distance
(continued...)

results of rodent experiments. A study published in 2005 provided the first evidence of subtle developmental effects, similar to those seen in animal studies, in human male infants exposed prenatally to breakdown products of phthalates.[10] More research would be needed to determine with certainty the effects of phthalates in humans. Additional information about health effects is provided below in the section "The Six Phthalates."

Human Exposure

Results of the National Health and Nutrition Examination Survey (NHANES) indicated almost universal American exposure to low levels of the most common phthalates, usually multiple phthalates.[11] Women tend to have greater exposure than men, but children appear to be the group most exposed to DEHP, DBP, and BBP. Children also are more exposed to DnOP, but these exposures appear to be lower than those for DEHP, di-n-butyl phthalate, and BBP. Levels of a breakdown product of DINP were not detectable in children surveyed (but see section below on DINP) and DIDP was not measured in 2001-2002.[12] Studies of amniotic fluid have also documented exposure to multiple phthalates for human fetuses.[13] More generally, babies may be the most heavily exposed group.[14]

Phthalates are dispersed throughout the air, water, soil, and living things in the developed world. According to the Department of Health and Human Services (DHHS), food probably is the major source of exposure to some phthalates for the general population.[15] [16] However, air also appears to be important.[17] For the human fetus, maternal exposure leads to prenatal exposure through the placenta. Personal

---

[9] (...continued)
among male infants with prenatal phthalate exposure. *Environmental Health Perspectives*, v. 113, n. 8, p. 1056-1061.

[10] Swan et al. 2005.

[11] DHHS. 2005. Third National Report on Human Exposure to Environmental Chemicals. p. 253. [http://www.cdc.gov/exposurereport/pdf/thirdreport.pdf].

[12] Ibid., p. 282.

[13] Silva, M.J., J.A. Reidy, A.R. Herbert, et al. 2004. Detection of phthalate metabolites in human amniotic fluid. *Bulletin of Environmental Contamination and Toxicology*, v. 72, p. 1226-1231.

[14] Wormuth, Matthias, Martin Scheringer, Meret Vollenweider, et al. 2006. What are the sources of exposure to eight frequently used phthalic acid esters in Europeans? *Risk Analysis*, v. 26, n. 3, p. 803-824.

[15] DHHS, ibid., p. 253.

[16] Fromme, H., L. Gruber, M. Schlummer, et al. 2007. Intake of phthalates and di-(2-ethhylhexyl) adipate: results of the integrated exposure assessment survey based on duplicate diet samples and biomonitoring data. *Environment International*, v. 33, n. 8, p. 1012-1020.

[17] Adibi, Jennifer J., Robin M. Whyatt, Paige L. Williams, et al. 2008. Characterization of phthalate exposure among pregnant women assessed by repeat air and urine samples. *Environmental Health Perspectives*, v. 116, n. 4, p. 467-473.

care items, including baby lotion and powder, may be significant sources of exposure for infants.[18] For some individuals, certain medications also may be important sources.[19] Finally, medical devices may dominate exposure sources for critically ill patients. Individuals, such as newborns in intensive care, may be exposed to levels of DEHP much closer to, but still less than 1% of, levels that cause reproductive harm in rats.[20]

Phthalates do not bioaccumulate in the body or the environment; rather they break down rapidly. However, exposure to phthalates is continuous and substantial in the modern world.[21]

The Six Phthalates

The Senate bill would require regulation of six phthalates, following the example of the European Union and the state of California. They differ from one another in structure, uses, and toxicities. The extent to which they have been studied varies widely. Compared to the other commercially produced phthalates, these six, arguably, are more studied, more toxic, or more prevalent in consumer products and the environment. All six have been evaluated by the National Toxicology Program (NTP), an interagency program administered through the National Institute of Environmental Health Sciences/National Institutes of Health (NIEHS/NIH).[22]

Di-(2-ethylhexyl) phthalate (DEHP). DEHP is the most abundantly produced and the most studied phthalate.[23] It is used primarily to improve the

---

[18] Sathyanarayana, S. C.J. Karr, P. Lozano, et al. 2008. Baby care products: possible sources of infant phthalate exposure. *Pediatrics*, v. 121, n. 2, p. e260-268.

[19] Hauser, Russ, Susan Duty, Linda Godfrey-Bailey, et al. 2004. Medications as a source of human exposure to phthalates. *Environmental Health Perspectives*, v. 112, n. 6, p. 751-753.

[20] Weuve, Jennifer, Brisa N. Sanchez, Antonia M. Calafat, et al. 2006. Exposure to phthalates in neonatal intensive care unit infants: urinary concentrations of monoesters and oxidative metabolites. *Environmental Health perspectives*, v. 114, n. 9., p. 1424-1431.

Calafat, Antonia M., Larry L. Needham, Manori J. Silva, et al. 2004. Exposure to di-(2-ethylhexyl) phthalate among premature neonates in a neonatal intensive care unit. *Pediatrics*, v. 113, n. 5., p. e429-e434.

[21] Silva, Manori J., E. Samandar, J.L. Preau, Jr., et al. 2007. Quantification of 22 phthalate metabolites in human urine. *Journal of Chromatography. B, Analytical Technologies in the Biomedical and Life Sciences*, v. 860, n. 1, p. 106-112.

[22] Center for the Evaluation of Risks to Human Reproduction (CERHR), National Toxicology Program, DHHS. "CERHR Chemicals." [http://cerhr.niehs.nih.gov/chemicals/index.html].

[23] For example, some important scientific studies include Akingbemi, Benson T., Renshan Ge, Gary R. Klinefelter, et al., 2004, Phthalate-induced Leydig cell hyperplasia is associated with multiple endocrine disturbances, *Proceedings of the National Academy of Sciences*, v. 101, n. 3, p. 775-780; Gray, Jr., L. Earl, Joseph Ostby, Johnathan Furr, et al.,

(continued...)

flexibility of "vinyl" (that is, polyvinyl chloride (PVC) plastic). DEHP is found in medical devices, such as plastic tubing used for catheters and intravenous drug and fluid delivery, and many home and garden products. The NTP has expressed "serious concern[24] that certain intensive medical treatments of male infants may result in DEHP exposure levels that adversely affect development of the male reproductive tract."[25] In addition, the NTP expressed "concern for effects of DEHP exposure on development of the male reproductive tract for infants less than one year old," "some concern for effects of DEHP exposure on development of the reproductive tract of male children older than one year," and also some concern for developmental effects for the offspring of pregnant women.[26] After this DEHP monograph was issued, several studies began to explore associations between DEHP and other health effects, such as effects on thyroid hormone levels, asthma, and obesity.[27] Additional research is warranted in these areas and it is too soon to draw any conclusions regarding the potential role of DEHP in causing such problems.

---

[23] (...continued)
2000, Perinatal exposure to the phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, alters sexual differentiation of the male rat, *Toxicological Sciences*, v. 58, n. 2, p. 350-365; Lin, H., Renshan S. Ge, Gary R. Chen, et al., 2008, Involvement of testicular growth factors in fetal Leydig cell aggregation after exposure to phthalate in utero, *Proceedings of the National Academy of Sciences of the United States of America*, v. 105, n. 290, p.7218-7222; Parks, Louise G., Joe S. Ostby, Christy R. Lambright, et al., 2000, The plasticizer diethylhexyl phthalate induces malformations by decreasing fetal testosterone synthesis during sexual differentiation in the male rat, *Toxicological Sciences*, v. 58, p. 339-349; Swan, Shanna H., Katharina M. Main, Fan Liu, et al., 2005, Decrease in anogenital distance among male infants with prenatal phthalate exposure, *Environmental Health Perspectives*, v. 113, n. 8, p. 1056-1061; and Wilson, Vickie S., Christy Lambright, Johnathan Furr, et al., 2004, Phthalate ester-induced gubernacular lesions are associated with reduced ins13 gene expression in the fetal rat testis, *Toxicology Letters*, v. 146, p. 207-215.

[24] The expression of "serious concern" is the greatest level of concern on a qualitative scale used by NTP. In order, from greatest to least, the levels of concern are: serious concern, concern, some concern, minimal concern, and negligible concern.

[25] Center for the Evaluation of Risks to Human Reproduction, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di(2-Ethylhexyl) Phthalate (DEHP).* NIH Publication No. 06-4476. November 2006. p. vii-viii. [http://cerhr.niehs.nih.gov/chemicals/dehp/DEHP-Monograph.pdf].

[26] Ibid.

[27] For example, see Meeker, John D., Antonia M. Calafat, and Russ Hauser, 2007, Di(2-ethylhexyl) phthalate metabolites may alter thyroid hormone levels in men, *Environmental Health Perspectives*, v. 115, n. 7, p. 1029-1034; Stahlhut, R.W., E. van Wijngaarden, T.D. Dye, et al., 2007, Concentrations of urinary phthalate metabolites are associated with increased waist circumference and insulin resistance in adult U.S. males, *Environmental Health Perspectives*, v. 115, n. 6, p. 876-882; Jaakkola, Jouni J.K., and Trudy L. Knight, 2008, The role of exposure to phthalates from polyvinyl chloride products in the development of asthma and allergies: a systematic review and meta-analysis, *Environmental Health Perspectives*, v. 116, n. 7, p. 845-853.

Dibutyl phthalates (DBP). There are two DBPs: di-n-butyl and di-isobutyl phthalate. The former is more studied, especially in Europe. DBPs are used in latex adhesives, nail polish, cosmetics, some inks and dyes, insecticides, and pharmaceutical coatings. The most recent NTP monograph on DBP (di-n-butyl phthalate) found "clear evidence of adverse effects" on the developing male reproductive tract in rodents.[28] Furthermore, the NTP concluded, "Based on recent data ... the NTP believes it is reasonable and prudent to conclude that the results reported in laboratory animals indicate a potential for similar or other adverse effects in humans."[29] As a result, NTP has "some concern for DBP causing adverse effects to human development, particularly development of the male reproductive system."[30] In the seven years since this NTP monograph, numerous studies have bolstered these findings.[31] Of particular note is the study by Lehmann et al. (2004).[32] It established the relationship between exposure to DBP and effects on synthesis of testosterone in fetal male rats. A 2006 study found that di-isobutyl phthalate had testicular and developmental effects similar to di-n-butyl phthalate and DEHP.[33]

Benzyl butyl phthalate (BBP or sometimes BzBP). BBP is used in vinyl flooring, automotive trim, food conveyor belts, and artificial leather. The latest NTP monograph on BBP was released in 2003, but was based on papers published before 2001. NTP determined that the evidence from animal studies was clear that adverse

---

[28] Ibid. p.2.

[29] Center for the Evaluation of Risks to Human Reproduction, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-n-Butyl Phthalate (DBP).* 2001. p. 4. [http://cerhr.niehs.nih.gov/chemicals/phthalates/dbp/DBP_Monograph_Final.pdf].

[30] Ibid. p. 2.

[31] For example, see Barlow, N.J., B.S. McIntyre, and P.M. Foster, 2004, Male reproductive tract lesions at 6, 12, and 18 months of age following in utero exposure to di(n-butyl) phthalate, *Toxicologic Pathology*, v. 32, n. 1, p. 79-90; Fisher, Jane S., S. Macpherson, N. Marchetti, et al., 2003, Human 'testicular dysgenesis syndrome': a possible model using in-utero exposure of the rat to dibutyl phthalate, *Human Reproduction*, v. 18, n. 7, p. 1383-1394; Mahood, I. Kim, Nina Hallmark, Chris McKinnell, et al., 2005, Abnormal Leydig cell aggregation in the fetal testis of rats exposed to di(n-butyl) phthalate and its possible role in testicular dysgenesis, *Endocrinology*, v. 146, p. 613-623; Thompson, Christopher J., Susan M. Ross, and Kevin W. Gaido, 2004, Di(n-butyl) phthalate impairs cholesterol transport and steroidogenesis in the fetal rat testis through a rapid and reversible mechanism, *Endocrinology*, v. 145, p. 1227-1237; Wilson, Vickie S., Christy Lambright, Johnathan Furr, et al., 2004, Phthalate ester-induced gubernacular lesions are associated with reduced ins13 gene expression in the fetal rat testis, *Toxicology Letters*, v. 146, p. 207-215; and Zhang,Y., X. Jiang, and B. Chen, 2004, Reproductive and developmental toxicity in F1 Sprague-Dawley male rats exposed to di-n-butyl phthalate in utero and during lactation and determination of its NOAEL, *Reproductive Toxicology*, v. 18, n. 5, p. 669-676.

[32] Lehmann, Kim P., Suzanne Phillips, Madhabananda Sar, et al. 2004. Dose-dependent alterations in gene expression and testosterone synthesis in the fetal testes of male rats exposed to di-(n-butyl) phthalate. *Toxicological Sciences*, v. 81, p. 60-68.

[33] Borch, J., M. Axelstad, A.M. Vinggaard, et al. 2006. Diisobutyl phthalate has comparable anti-androgenic effects to di-n-butyl phthalate in fetal rat testis. *Toxicology letters*, v. 163, n. 3, p. 183-190.

developmental effects could result from exposure to BBP.[34] However, effects were seen only at high levels of BBP exposure and estimated human exposure was much lower, although detailed exposure data were lacking. NTP concluded that it had minimal concern for fetal and infant developmental effects due to estimated BBP exposure.[35] Papers published after 2001 confirm the developmental toxicity of BBP.[36] However, the testimony of Leon Earl Gray, Jr., Senior Reproductive biologist and Toxicologist with the U.S. Environmental Protection Agency, indicated that DEHP, DBP (both forms), and BBP were equivalent in toxicity, based on four studies.[37]

Diisononyl phthalate (DINP). This phthalate is used primarily to improve the flexibility of plastics in such products as gloves, drinking straws, garden hoses, and toys. It has been used to replace DEHP in toys and other applications. DINP is the most commonly used phthalate for toys, according to the Phthalate Esters Panel of the American Chemistry Council, a trade group representing chemical manufacturers.[38] The NTP monograph on DINP was published in 2003, but like the other monographs that have not been updated, it primarily considers research

---

[34] Center for the Evaluation of Risks to Human Reproduction, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Butyl Benzyl Phthalate (BBP).* NIH Publication No. 03-4487. March 2003. p. 2. [http://cerhr.niehs.nih.gov/chemicals/phthalates/bb-phthalate/BBP_Monograph_Final.pdf].

[35] Ibid. p. 4.

[36] Wilson, Vickie S., Christy Lambright, Johnathan Furr, et al. 2004. Phthalate ester-induced gubernacular lesions are associated with reduced ins13 gene expression in the fetal rat testis. *Toxicology Letters*, v. 146, p. 207-215.

Aso, S. H. Ehara, K. Miyata, et al. 2005. A two-generation reproductive toxicity study of butyl benzyl phthalate in rats. *Journal of Toxicological Sciences*, Dec., v. 30, p. 39-58.

[37] U.S. Congress. House of Representatives. Committee on Energy and Commerce, Subcommittee on Commerce, trade, and consumer Protection. Hearing on Safety of Phthalates and Bisphenol-A in Everyday Consumer Products. June 10, 2008. Written testimony of Leon Earl Gray, Jr., Senior Reproductive Biologist and Toxicologist, U.S. EPA.

The studies referenced by Dr. Gray were: Gray, Jr., L. Earl, Joseph Ostby, Johnathan Furr, et al., 2000, Perinatal exposure to the phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, alters sexual differentiation of the male rat, *Toxicological Sciences*, v. 58, n. 2, p. 350-365; Hotchkiss, A.K., L.G. Parks-Saldutti, J.S. Ostby, et al. 2004, A mixture of the 'antiandrogens' linuron and butyl benzyl phthalate alters sexual differentiation of the male rat in a cumulative fashion, *Biology of Reproduction*, v. 71, n. 6, p. 1852-1861; Howdeshell, Kembra L., Vickie S. Wilson, Johnathan Furr, et al., 2008, A mixture of five phthalate esters inhibits fetal testicular testosterone production in the Sprague Dawley rat in a cumulative, dose additive manner, *Toxicological Sciences Advance Access* (reprint received from the author); and Tyl,R.W., C.B, Myers, M.C. Marr, et al., 2004, Reproductive toxicity evaluation of dietary butyl benzyl phthalate (BBP) in rats, *Reproductive Toxicology*, v. 18, n. 2, p. 241-264.

[38] American Chemistry Council, Phthalates Panel. Phthalates and children's toys. [http://www.phthalates.org/yourhealth/childrens_toys.asp].

published before 2001. The scientific evidence for developmental effects from DINP exposure of rodents is adequate "to conclude that DINP might adversely affect development of the human fetus if the levels of exposure are sufficiently high," according to NTP.[39] But the evidence for effects was not as strong as for DEHP and DBP, and in one study that compared the effects of exposure to various phthalates, DINP was found to be less potent than DEHP or DBP by an order of magnitude.[40] In his recent testimony before a House subcommittee, Dr. Leon Earl Gray rated the relative potency for producing developmental harm of various phthalates. He gave DINP a rating of 0.15 relative to DEHP, DBP (both forms), and BBP which he rated 1.0.[41] A number of studies of the potential effects on rodents of DINP exposure were published after 2001, but they appear consistent with the earlier work with respect to developmental toxicity.[42] In addition, there is some evidence of enhanced allergic responses due to DINP exposure.[43]

A review by the European Commission (EC) in 2006 concluded:

> "[i]n light of the divergent scientific views ... and the conclusions of the assessment of the risk for consumers under this Regulation, and taking into account the uncertainties in the evaluation of exposure to DINP from toys and childcare articles, precautionary considerations support the consideration at Community level of proportionate restrictions ... for the use of DINP in toys and childcare articles. Such measures should be reviewed after 3-4 years, in light of further scientific developments.[44]

---

[39] CERHR, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-isononyl Phthalate (DINP).* NIH Publication No. 03-4484. March 2003. p. 2.

[40] "Less by an order of magnitude" is roughly equivalent to one-tenth. Gray, Jr., L. Earl, Joseph Ostby, Johnathan Furr, et al. 2000. Perinatal exposure to the phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, alters sexual differentiation of the male rat. *Toxicological Sciences*, v. 58, n. 2, p. 350-365.

[41] U.S. Congress. House of Representatives. Committee on Energy and Commerce, Subcommittee on Commerce, trade, and consumer Protection. Hearing on Safety of Phthalates and Bisphenol-A in Everyday Consumer Products. June 10, 2008. Written testimony of Leon Earl Gray, Jr., Senior Reproductive Biologist and Toxicologist, U.S. EPA.

[42] For example, Borch, J., O. Ladefoged, U. Hass, et al., 2004, Steroidogenesis in fetal male rats is reduced by DEHP and DINP, but endocrine effects of DEHP are not modulated by DEHA in fetal, prepubertal and adult male rats, *Reproductive Toxicology*, v. 18, n. 1, p. 53-61; and Wenzel, A., C. Franz, E. Breous, et al., 2005, Modulation of iodide uptake by dialkyl phthalate plasticisers in FRTL-5 rat thyroid follicular cells, *Molecular and Cellular Endocrinology*, v. 244, n. 1-2, p.63-71.

[43] Lee, M.H., J. Park, S.W. Chung, et al. 2004. Enhancement of interleukin-4 production in activated CD4+ T cells by diphthalate plasticizers via increased NF-AT binding activity. *International Archives of Allergy and Immunology*, v. 134, n. 3, p. 213-222.

[44] European Commission. Commission Communication on the results of the risk evaluation and the risk reduction strategies for the substances: Dibutylphthalate; e,4-Dichloroaniline; Di-isodecyl phthalate; 1,2-Benzenedicarboxylic acid, di-c9-11-branched alkyl esters, c9-

(continued...)

Several studies have been conducted to estimate the level of exposure of children to DINP in toys due to mouthing. One study by the Consumer Product Safety Commission (CPSC) estimated DINP exposure through measurement of the time children spent in mouthing behavior and an analysis of DINP movement out of various toys.[45] NTP used that data to conclude that its concern was "minimal" for developmental effects in children.[46] NTP also had minimal concern for DINP causing adverse effects to human reproduction or fetal development.[47] However, this conclusion was controversial.[48] A Japanese study also looked at DINP release from toys and and time spent mouthing and found considerably higher exposures than the CPSC.[49]

As mentioned above, it appeared from the NHANES that exposure to DINP was negligible in children surveyed by the Centers for Disease Control and Prevention (CDC) in 2001-2002.[50] However, this result was based on measurement of the metabolite monoisononyl phthalate (MINP), the traditional measure of exposure to DINP. A recent study of how the human body processes DINP found that MINP is a minor metabolic product, while mono(carboxyisooctyl) phthalate (MCIOP), mono(oxoisononyl) phthalate (MOINP), and mono(hydroxyisononyl) phthalate (MHINP) are the major metabolites in DINP-dosed rats.[51] The authors of this study concluded that estimates of exposure to DINP might be underestimates if based on MINP levels.

Diisodecyl phthalate (DIDP). DIDP is another plasticizer used primarily in electrical cords, leather for car interiors, and PVC flooring. As for the other phthalates addressed by the NTP, the expert panel found "no *direct* evidence that exposure of *people* to DIDP adversely affects reproduction or development" [emphasis added], but "studies with rats have shown that exposure to DIDP can cause

---

[44] (...continued)
rich; Ethylenediaminetetraacetate; Methyl acetate; Monochloroacetic acid; n-Pentane; Tetrasodium ethylenediaminetetraacetate. Official Journal of the European Union. April 13, 2006. Available through Eur-Lex at [http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:C:2006:090:0004:0028:EN:PDF].

[45] Babich, M.A., S.B. Chen, M.A. Greene, et al. 2004. Risk assessment of oral exposure to diisononyl phthalate from children's products. *Regulatory Toxicology and Pharmacology*, v. 40, n. 2, p. 151-167.

[46] CERHR, p. 3.

[47] Ibid.

[48] Shea, K.M. 2003. Pediatric exposure and potential toxicity of phthalate plasticizers. *Pediatrics*, v. 111, n. 6, Pt 1, p. 1467-1474.

[49] Sugita, T., Y. Kawamura, M. Tanimura, et al. 2003. Estimation of daily oral exposure to phthalates derived from soft polyvinyl chloride baby toys. *Shokuhin eiseigaku zasshi. Journal of the Food Hygienic Society of Japan*, v. 44, n. 2, p. 96-102.

[50] DHHS, ibid., p. 282.

[51] Silva, M.J., J.A. Reidy, J.L. Preau, Jr., et al. 2006. Oxidative metabolites of diisononyl phthalate as biomarkers for human exposure assessment. *Environmental Health Perspectives* v. 114, n. 8, p. 1158-61.

adverse developmental effects."[52] NTP concluded that exposures to DIDP were probably not high enough to cause concern, and that scientists had minimal concern for developmental effects in fetuses and children.[53] NTP also found that DIDP would not adversely affect human reproduction.[54] Studies published since 2001 have not conflicted with the NTP conclusions. However, several recent studies have found endocrine-disrupting effects following rodent exposure to DIDP.[55]

Di-n-octyl phthalate (DnOP). DnOP is used primarily to improve the flexibility of plastics. DnOP is found in mixtures of phthalates that are used to make flooring, tarps, pool liners, bottle cap liners, conveyor belts, and garden hoses. There are few studies on which to evaluate the potential toxicity or exposure to DnOP. NTP found limited evidence that DnOP might cause developmental effects in highly exposed rodents.[56] No evidence of reproductive effects was found in the available studies.[57] NTP concluded that it had negligible concern for effects on adult reproductive systems, but it was unable to form an opinion on an appropriate level of concern with respect to developmental risks, due to the lack of available exposure data and lack of toxicity data for exposure levels that might have relevance for human exposure.[58] An online search by CRS of publications using Medline revealed no new studies after 2001 that might better inform a risk evaluation.

---

[52] Center for the Evaluation of Risks to Human Reproduction, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-isodecyl Phthalate (DIDP).* NIH Publication No. 03-4485. April 2003. p. 1.

[53] Ibid. p. 3.

[54] Ibid.

[55] Wenzel, A., C. Franz, E. Breous, et al. 2005. Modulation of iodide uptake by dialkyl phthalate plasticisers in FRTL-5 rat thyroid follicular cells. *Molecular and Cellular Endocrinology*, v. 244, n. 1-2, p.63-71.

Turan, N., R.H. Waring, and D.B. Ramsden. 2005. The effect of plasticisers on "sulphate supply" enzymes. *Molecular and Cellular Endocrinology*, v. 244, n. 1-2, p. 15-19.

Harris, R., N. Turan, C. Kirk, et al. 2007. Effects of endocrine disruptors on dehydroepiandrosterone sulfotransferase and enzymes involved in PAPS synthesis: genomic and nongenomic pathways. *Environmental Health Perspectives*, v. 115, Supp. 1, p. 51-54.

[56] Center for the Evaluation of Risks to Human Reproduction, National Toxicology Program, U.S. Department of Health and Human Services. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Di-n-octyl Phthalate (DnOP).* NIH Publication No. 03-4488. May 2003. p. 1.

[57] Ibid.

[58] Ibid. p. 3.

Federal Evaluation and Regulation of Phthalates[59]

Depending on use, phthalates are potentially regulated by various regulatory agencies, including the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), and the Consumer Product Safety Commission (CPSC). EPA regulates various phthalates released to the environment under most of its statutes. For example, DEHP is regulated as a hazardous air pollutant, a drinking water contaminant, a water pollutant, and a hazardous waste. OSHA regulates worker exposure to phthalates.

The current focus of congressional concern, however, is federal regulation of consumer products from which phthalates might be released. Federal agencies have taken several actions, some as early as the mid 1980s, to evaluate and regulate phthalates. For example, EPA has required manufacturers of phthalates to conduct certain tests to better inform federal regulators. These test orders were withdrawn in 1996, when EPA determined that it had received the necessary information (which focused at the time on carcinogenic potential). To date, however, no phthalate-containing product has been banned outright.

Food and Drug Administration (FDA). FDA-regulated products that may contain phthalates include (1) medical devices; (2) food contact substances, such as plastic wrap;[60] and (3) cosmetics. FDA reported in June 2008 that, in tandem with its review of the safety of bisphenol A (BPA) in the products it regulates,[61] it is also conducting a comprehensive inventory of regulated products that contain phthalates.[62]

FDA regulates a wide variety of medical devices in commerce. Many of these products are made of, or contain PVC. These include intravenous fluid bags and lines, tubing used for procedures such as cardiac bypass and dialysis, and indwelling medical devices, such as peripherally inserted central catheters, or "PICC lines." According to reports, phthalates in tubing can leach into the fluids they contain and pass into the body, and can leach directly from indwelling devices. In 2001, FDA completed a safety assessment of DEHP, which was the softener most commonly used in PVC-containing medical devices.[63] The assessment underpinned a public

---

[59] This section was written by Sarah A. Lister, Specialist in Public Health and Epidemiology, Domestic Social Policy Division.

[60] Manufacturers maintain that phthalates are not used in food wrap in the United States. See the trade group website [http://www.phthalates.org/pdfs/Phthalates_mvf.pdf].

[61] See CRS Report RS22869, *Bisphenol A (BPA) in Plastics and Possible Human Health Effects*, by Linda-Jo Schierow and Sarah A. Lister.

[62] Statement of Norris Alderson, Ph.D., FDA Associate Commissioner for Science, before the Subcommittee on Commerce, Trade and Consumer Protection, House Committee on Energy and Commerce, hearing on "Safety of Phthalates and Bisphenol A in Everyday Consumer Products," June 10 , 2008, 110th Cong., 2nd sess., Washington, DC, hereafter referred to as FDA testimony.

[63] FDA, "Safety Assessment of Di(2-ethylhexyl)phthalate (DEHP) Released from PVC (continued...)

health notification in 2002 in which FDA identified a number of medical procedures that posed the highest risk of exposure to DEHP, and recommended the use of alternatives to DEHP-containing medical devices if these procedures were to be performed on high-risk individuals.[64] Depending on the procedure, these individuals include infants, boys, pregnant or nursing women, and, for some procedures, healthy adults. FDA recommended, however, that needed medical procedures not be deferred solely because of concerns about DEHP exposure.

Phthalates are not added to foods directly, but are regulated by FDA as *food contact substances* or *indirect food additives*, where they are components of packaging that may leach into foods or beverages. FDA permits the use of a variety of phthalates for these purposes.[65] In recent congressional testimony, FDA reported that:

> [FDA] has recently established a Phthalate Task Group (PTG) to review all available use and toxicology information associated with phthalate exposure from food contact use and to better characterize any potential risk from these uses. The primary focus of the PTG will be to determine the most realistic exposure estimation and risk associated with phthalate use in food packaging. The PTG will review and address past studies on phthalates and any new information available. If our review indicates that existing data no longer supports the continued safe use of these materials in food contact material, FDA will take appropriate regulatory action to remove these materials from the marketplace.[66]

Under the authority of the Federal Food, Drug, and Cosmetic Act (FFDCA), FDA regulates the safety of cosmetics and personal care products, such as nail polish, shampoo and lotions,[67] many of which contain phthalates. Cosmetic products and ingredients are not subject to FDA premarket approval, but FDA can prohibit the marketing of a cosmetic product if it is adulterated, meaning, among other things,

---

[63] (...continued)
Medical Devices," undated, at [http://www.fda.gov/cdrh/ost/dehp-pvc.pdf].

[64] FDA, "FDA Public Health Notification: PVC Devices Containing the Plasticizer DEHP," July 12, 2002, at [http://www.fda.gov/cdrh/safety/dehp.html].

[65] For example, a search for "phthalate" in FDA's regulations at [http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm] yields almost two dozen approved phthalates, including DEHP, in adhesives used for food packaging.

[66] FDA testimony. FDA's definition of safety with respect to food additives and food contact substances is that "there is a reasonable certainty in the minds of competent scientists that the substance is not harmful under the intended conditions of use." 21 CFR § 170.3(i).

[67] "Cosmetic" is defined as " (1) articles intended to be rubbed, poured, sprinkled, or sprayed on, introduced into, or otherwise applied to the human body or any part thereof for cleansing, beautifying, promoting attractiveness, or altering the appearance, and (2) articles intended for use as a component of any such articles; except that such term shall not include soap." Federal Food, Drug, and Cosmetic Act (FFDCA) section 201(i); [21 U.S.C. 321(i)]. For more information, see FDA, "Cosmetics: FDA Authority and Policy," at [http://www.cfsan.fda.gov/~dms/cos-pol.html].

that it contains "any poisonous or deleterious substance which may render it injurious to users" under labeled or customary conditions of use.[68] According to FDA, the principal phthalates used in cosmetic products are DBP, dimethylphthalate (DMP), and diethylphthalate (DEP). FDA reports that at the present time, it "does not have compelling evidence that phthalates, as used in cosmetics, pose a safety risk. ...," but that it is conducting a survey of phthalate levels in certain cosmetic products to more accurately assess infant exposure.[69] Under the authority of the Fair Packaging and Labeling Act (Public Law 89-755), FDA requires that retail cosmetic products carry ingredient labels, which would include phthalates. However, the listing of individual fragrance ingredients, which may contain phthalates, is not required.[70]

Consumer Product Safety Commission. The Consumer Product Safety Commission (CPSC) may regulate phthalates in consumer products, including toys and other children's products, under either the Federal Hazardous Substances Act (FHSA, 15 U.S.C. §§1261 et seq.) or the Consumer Product Safety Act (CPSA, 15 U.S.C. §§2051 et seq.), two of the statutes that it administers and enforces. Consumer products as defined in the CPSA does not include food, drugs, medical devices, and cosmetics, which fall under the jurisdiction of the FDA. The FDA has jurisdiction over food containers with regard to substances that may leach into food from the container. CPSC has jurisdiction with regard to other defects, such as shattering or choking hazards. It also has jurisdiction over toys, children's furniture such as cribs, car seats, pacifiers and teething rings, and other children's consumer products.

Although the CPSC may regulate phthalates under either the CPSA or the FHSA, if it chooses to promulgate safety standards pursuant to the CPSA for a product that may be regulated under the FHSA, it must promulgate a rule finding that it is in the public interest to regulate the risk of injury under the CPSA.[71] Since the CPSA and the FHSA differ with regard to the rule-making procedures, the precise nature of the acts prohibited with regard to products or substances that fail to comply with safety standards, and other issues, there are advantages and disadvantages to promulgating standards under the CPSA versus the FHSA. For example, injunctive enforcement authority for States attorneys general is expressly provided by the FHSA, but not by the CPSA; the apparent ambiguity of the CPSA on this point has led to clarifying provisions in both the Senate and House versions of H.R. 4040, the CPSC reform legislation now in conference. Therefore, if a phthalates standard were to be promulgated under the FHSA, States attorneys general would have the authority to enjoin violations of the federal standard, whereas arguably they would not have similar authority to enforce a similar standard under the CPSA.

Additionally, critics allege problems with the ability of the CPSC to establish safety standards in a timely fashion. For example, some assert that the rulemaking

---

[68] FFDCA section 601; 21 U.S.C. 361.

[69] FDA, "Phthalates and Cosmetic Products," February 7, 2008, at [http://www.cfsan.fda.gov/~dms/cos-phth.html].

[70] Ibid.

[71] CPSA §30(d) (15 U.S.C. §2079).

procedures under the CPSA and other acts under the CPSC's jurisdiction, such as the FHSA, are unnecessarily onerous, requiring protections beyond those required by the Administrative Procedures Act. Also, the CPSA requires the CPSC to rely on voluntary standards where an adequate standard exists with which the industry widely and substantially complies. The voluntary nature of some safety standards limits the action that can be taken by the CPSC for violations. Under both the CPSA and the FHSA, the CPSC may order a recall and/or other remedies for products that violate a safety standard under the pertinent act and may inspect factories where products are made.[72] Products violating a safety standard may be denied importation.

Although the European Union and some states have enacted safety standards regarding phthalates in children's products, as discussed below, the CPSC has not promulgated such standards. In 1983, the CPSC determined that DEHP in children's products might result in substantial exposure of children to an animal carcinogen.[73] The CPSC is not permitted to initiate rule-making relating to risks of cancer, birth defects or gene mutations unless it first establishes a Chronic Hazard Advisory Panel to study the issue and make recommendations. The panel appointed to study the risks of DEHP concluded that it could put children at risk of cancer from mouthing of products containing DEHP. Accordingly, the CPSC worked with the children's products industry to reach a voluntary agreement banning the use of DEHP from pacifiers, rattles, and teethers.[74] Although other children's products were not included in the agreement between CPSC and the industry, most manufacturers substituted other phthalates for DEHP in other children's products. DINP was the substitute. Despite studies conducted by the industry in the late 1990s linking DINP to liver toxicity and cancer in rodents, the CPSC concluded that the risk to children from mouthing children's products was minimal. However, the CPSC achieved a voluntary agreement with the industry banning DINP and dioctyl phthalate from pacifiers and bottle nipples. The CPSC appointed a Chronic Hazard Advisory Panel on DINP and conducted other studies in response to a petition to initiate rule-making regarding phthalates in children's products. The panel concluded in 2001 that DINP posed a minimal or nonexistent risk of cancer to humans. After further consideration of the panel report and other studies, in 2003 the CPSC denied the petition to establish a safety standard for PVC containing phthalates in children's products intended for children five years of age and younger.[75]

---

[72] For a general discussion of the statutes administered and enforced by the CPSC, see CRS Report RL34399, *Consumer Product Safety Commission Reform: Senate and House Versions of H.R. 4040*, by Margaret Mikyung Lee.

[73] See the discussion of scientific studies for various phthalates above and the discussion of CPSC actions in *Young Children and Plastic Toys* and *Phthalates in Plastic Toys*, 8 Consumer Product Safety Review 3-5 (2003).

[74] Although the CPSC has promulgated safety standards for pacifiers and rattles at 15 C.F.R. parts 1510 and 1511, these primarily concern choking/mechanical hazards, not hazards from phthalates or other chemical constituents.

[75] Denial letter from the CPSC to the National Environmental Trust, dated February 26, 2003, available at [http://www.cpsc.gov/LIBRARY/FOIA/FOIA03/petition/Ageunder.pdf] (last visited July 11, 2008).

Regulation in the European Union (EU) and the States

European Union. Proposed federal legislation and several state statutes concerning phthalates apparently were modeled on EU laws. Under Council Directive 76/769/EEC,[76] as amended by Council Directive 2005/84/EC,[77] the EU currently prohibits the use (at concentrations greater than 0.1% by mass of the plasticized material) of DEHP, DBP, and BBP in toys and child-care articles and of DINP, DIDP, and DnOP in toys and child-care articles that can be mouthed by children. It also prohibits the sale of toys and child-care articles containing phthalates at a concentration exceeding the permitted level. It defines "childcare article" as meaning "any product intended to facilitate sleep relaxation, hygiene, the feeding of children or sucking on the part of children."

Council Directive 2005/84/EC notes the existence of Commission Decision 1999/815/EC[78] that banned phthalates in toys and child-care articles as a renewable emergency measure in response to phthalate studies conducted by various Member States and assessed by the Scientific Committee on Toxicity, Ecotoxicity and the Environment.[79] The language of the Decision differs from that of the Directive in that it limits the ban on the six phthalates to products that can be mouthed by children under three years of age, while the Directive does not contain the age limit. This Decision apparently was last extended in 2004 until September 20, 2005, but Council Directive 2005/84/EC refers to the Decision as being renewed regularly. Regardless of whether the Decision continues to be renewed, EU Member States were required to take necessary measures to comply with the standards described in the Decision, therefore, those measures would continue in effect. It appears that the Decision may not have been renewed in anticipation of Council Directive 2005/84/EC and the Regulation (EC) No. 1907/2006,[80] which was recently enacted and will repeal Council Directive 76/769/EEC effective June 1, 2009, while retaining the same phthalate in children's product standard, effective June 1, 2009. The new Regulation also retains the Directive's requirement that the European Commission re-evaluate this standard by January 16, 2010, in light of new scientific information and amend the standard accordingly, if justified.

---

[76] 1976 O.J. (L 262) 201 (originally enacted July 27, 1976).

[77] 2005 O.J. (L 344) 40 (enacted December 14, 2005). This Directive amended the 1976 Directive to add the restrictions on phthalates in children's products.

[78] O.J. (L 315) 46 (issued December 7, 1999). This Decision in turn was preceded by Commission Recommendation 98/485/EC of July 1, 1998, O.J. (L 217) 35, inviting Member States to take measures to ensure a high level of child health protection with regard to child-care articles and toys intended to be placed in the mouth by children less than three years of age and made of PVC containing any of the six phthalates.

[79] Information received via email communications with the Delegation of the European Commission in Washington, D.C., July 11, 2008.

[80] Arts. 139, 141, Annex XVII.51 & 52, 2006 O.J. (L 396) 1 (enacted December 18, 2006) (popularly referred to as REACH, the acronym for Registration, Evaluation, Authorisation, and Restriction of Chemicals).

While the Directive is binding law on the Member States of the EU, requiring them to take necessary measures to bring their respective national laws into compliance with the Directive standards, the new Regulation is directly binding on the Member States. This means they are obligated to comply with the standard and enforce it without any implementing legislation or rule at the national level.

One group of phthalates, DEHP, DBP, and BBP, is banned without limitation because they are classified as reproductive toxicants that present an unacceptable risk given the general safety requirements of the European Union. The other group, DINP, DIDP and DnOP, are banned only for products that can be placed in the mouth by children. Despite inconclusive scientific evidence of harm, these phthalates were banned under the precautionary principle of the European Union given the potential risk posed to children.[81]

States. California, Vermont and Washington have recently enacted legislation establishing safety standards for phthalate content in children's articles. These standards all appear to have been at least partly modeled on current or earlier versions of the EU regulations. Aside from the age limit specified for the phthalate standard or for subcategories of children's products, if any, the existing state statutes do not define children's products generally in terms of an age ceiling for "child" or "children." Hawaii's Senate has adopted a resolution requesting the Hawaii Department of Health to monitor research being conducted regarding the risks posed by phthalates and bisphenol-A in consumer products and to report recommendations and proposed legislation before the 2009 legislative session.[82] Oregon's legislature has adopted a joint memorial urging Congress to regulate phthalates at the federal level as a substance in cosmetics, personal care products, and children's toys.[83] Additionally, several other states have introduced legislation concerning phthalates.

Beginning January 1, 2009, the California statute[84] will prohibit the manufacture, sale, or distribution in commerce of any toy or child-care article that contains DEHP, DBP, or BBP in concentrations exceeding 0.1% and of any toy or child-care article, intended for use by children under three years of age that can be mouthed, that contains DINP, DIDP or DnOP in concentrations exceeding 0.1%. The statute requires manufacturers to use the least toxic alternative when replacing phthalates in such products and also prohibits them from replacing phthalates with certain carcinogens (including substances known, likely to be, or suggestive of being human carcinogens) or reproductive toxicants identified in accordance with federal or California laws. "Toys" are defined as "all products designed or intended by the manufacturer to be used by children when they play" and "child care article" is defined as "all products designed or intended by the manufacturer to facilitate sleep, relaxation, or the feeding of children, or to help children with sucking or teething."

---

[81] Information received via email communications with the Delegation of the European Commission in Washington, D.C., July 11, 2008.

[82] S.Res. 68, 24th Leg. (Haw. 2008).

[83] S. Jt. Memorial 8, 74th Leg. (Or. 2007).

[84] Cal. Health & Safety Code §§108935-108939 (current on LexisNexis).

This statute appears to be partly modeled on an earlier version of the EU Council Directive 76/769/EEC with regard to the threshold concentration level for the ban and the definition of child-care articles and toys. It does not include the amendment made by EU Directive 2005/84/EC (December 14, 2005) adding "hygiene" to the scope of child-care articles, which effectively included items such as lotion, powder, baby oil, etc. It also includes the three-year-old age limit regarding products that can be placed in the mouth from the EU Commission Decision 1999/815/EC (December 7, 1999). It added "teething" to the scope of child-care articles, for which the EU includes "sucking" but not teething.

The Vermont statute[85] appears to be modeled on the California statute, but with some differences. Beginning July 1, 2009, it will prohibit the manufacture, sale, or distribution in commerce of any toy or child-care article intended for use by a child under three years of age that contains DEHP, DBP, or BBP in concentrations exceeding 0.1% and of any toy or child-care article intended for use by a child under three years of age that can be placed in the mouth and that contains DINP, DIDP, or DnOP in concentrations exceeding 0.1%. The Vermont statute provides for an under- three-years-old age limit for the first group of phthalates, unlike the California statute. The Vermont law adopts the California statutory definition of "child care article" and "toy" and additionally defines "phthalate" as "any one of a group of chemicals used as plasticizers to provide flexibility and durability to plastics such as polyvinyl chloride (PVC)." Like the California statute, it requires manufacturers to use the least toxic alternative to phthalates and prohibits them from substituting carcinogens (including substances known, likely to be, or suggestive of being human carcinogens) or reproductive toxicants identified by the EPA under federal law, but not under state laws, apparently because Vermont does not have such environmental laws identifying carcinogens or reproductive toxicants.

The Vermont law provides that a violation of the phthalates law shall be deemed a violation of the Vermont Consumer Fraud Act and that the provisions of that act concerning the enforcement authority of the Vermont Attorney General and the rights of private parties shall apply to violations of the phthalates law. It further clarifies that nothing in the phthalates law regulates firearms, ammunition, shooting ranges, or hunting/fishing equipment.

The Washington provision banning phthalates in children's products is part of a broader statute[86] concerning chemicals in children's products generally. It covers lead and cadmium content and also provides, among other things, for the identification of chemicals of "high concern" to children and children's products by the Washington Department of Ecology, for notification to the Department by a manufacturer that its children's product contains a chemical of high concern, and for a product safety education campaign to promote awareness of unsafe children's products. With regard to phthalates, beginning July 1, 2009, it prohibits a manufacturer, wholesaler, or retailer from manufacturing, knowingly selling, offering

---

[85] Act of May 24, 2008, Act 171, 2008 Vt. Adv. Legis. Serv. 171 (LexisNexis) (relating to phthalates in products for young children).

[86] Act of June 12, 2008, ch. 288, 2008 Wash. Adv. Legis. Serv. 288 (LexisNexis) (relating to the children's safe products act).

for sale, or distributing for sale or for use in the state a children's product or product component containing phthalates, individually or in combination, at a concentration exceeding 0.1% by weight (a thousand parts per million). It defines phthalates as meaning the six phthalates discussed in this report. It adopts the California definition of "toy," but does not refer to "child-care articles." Instead, it defines "children's product," which includes the California definition of a child-care article, expanding it to include children's clothing, and also includes toys, children's cosmetics (for children under the age of twelve), children's jewelry (for children under the age of twelve), and car seats (it also includes a list of items not considered "children's products").

Proposed Legislation

On July 28, 2008, the House and Senate conferees for H.R. 4040, the Consumer Product Safety Improvement Act of 2008, announced that an agreement on a final text had been reached resolving the differences between the Senate and House versions of the bill. As of the date of this report, it remains uncertain whether a conference agreement will be completed in time for a floor vote in both chambers before the August recess. The conference committee and Senator Pryor published summaries of some of the agreed provisions. Because a final text for the agreed provisions was not made public, however, the details of the agreed provisions generally remain unclear. The summary below is based on details published by the conference and by the media.

The conference agreement would permanently ban the three phthalates whose toxicity is not disputed and would temporarily ban three other phthalates pending a review by a Chronic Hazard Advisory Panel (CHAP). It would prohibit children's toys or child care articles that contain more than 0.1 percent DEHP, DBP, or BBP. The sale of children's toys or child care articles containing concentrations of more than 0.1 percent of DINP, DIDP, or DnOP would be prohibited on an interim basis until a review by a CHAP. After the CPSC receives the report from the CHAP, it would determine, by rule, whether to continue the interim ban.

In March 2008, the Senate approved an amendment to H.R. 4040, which would have restricted the use of six phthalates in certain toys and child-care products. The House-passed version of H.R. 4040 had no phthalate amendment; the House Committee on Energy and Commerce noted in its report that it became aware of the potential dangers posed by phthalates in toys late in the legislative process and intended to take up the issue later.[87]

Section 40 of the Senate version of H.R. 4040, also referred to as the Feinstein-Boxer Amendment for the two California senators who introduced this specific amendment to the bill, was modeled on the California statute, with some changes, but placed the provisions in the context of the Federal Hazardous Substances Act (FHSA) framework. It adopted the California statutory definition of "child care article" and did not adopt its definition of "toy," but instead defined "children's product" as "a toy or any other product designed or intended by the manufacturer for

---

[87] H.Rept. 110-501, at 47 (2007).

use by a child when the child plays," which effectively included the same products as the California statute.

The provision would have treated as a banned hazardous substance under the FHSA any children's product or child-care article which (1) contains in any part any combination of DEHP, DBP or BBP in concentrations exceeding 0.1% or (2) is intended for use by a child that can be placed in a child's mouth and either contains any combination of DINP, DIDP or DnOP in concentrations exceeding 0.1% or contains any combination of any of the six phthalates in concentrations exceeding 0.1%. Any prohibitions under FHSA §4 (15 U.S.C. §1263) would have applied to such products, including the introduction, receipt or delivery into interstate commerce of such products; failure to permit inspections of any factory, warehouse, or other establishment where such products are manufactured, processed, packed, or held; etc. Like the California statute, the provision would have prohibited a manufacturer from replacing phthalates with certain carcinogens (including substances known, likely to be, or suggestive of being human carcinogens) or reproductive toxicants identified by the EPA. However, unlike the California statute, the provision would not have required manufacturers to use the least toxic alternative as a substitute for phthalates.

Section 40 would have provided that neither it nor FHSA §18(b)(1)(B) (15 U.S.C. §1261 note), concerning preemption of non-identical State or local regulations of banned hazardous substances, would preempt State or local laws applying to a phthalate other than the six described in the bill; applying to a phthalate described in the bill that is not otherwise regulated by the bill; requiring a warning of risk, illness, or injury regarding any phthalate; or prohibiting the use of alternatives to phthalates not prohibited under this bill. Generally, non-identical State or local regulations are preempted unless an exemption is granted by the CPSC upon request by the State or locality.

Conclusion

When the House and Senate consider the conference report accompanying H.R. 4040, Members might be expected to consider two central questions about phthalates and the proposed amendment. First, what is the scientific basis for health concerns about exposure to these chemicals? Second, would the provision reduce the risks without generating greater risks? These issues are discussed briefly here, based on the scientific and legal information presented above.

Scientific Basis for Health Concerns

The scientific basis for concerns about risks to human health appears to be strong in the case of some phthalates, adequate with respect to others, and weak for the remaining chemicals. The strongest evidence with respect to developmental effects has been produced since about the year 2000. Many of these studies were not available to the NTP or to CPSC when they reviewed the phthalate literature in 2000 or 2001. At that time, regulators focused on carcinogenic effects, rather than effects on fetal development. This more recent animal evidence strongly supports a claim

that DEHP, DBP, and DBB can harm reproduction and damage fetal and juvenile development in rats.

The structure-activity relationship (between the molecular structure of the phthalates and developmental damage) is well understood, such that scientific concern focuses now on DEHP, DBP, and DBB.[88] DINP also is a developmental toxicant, but is only about 15% as potent as the most potent phthalates. To the extent that it is still studied, it is generally studied together with other more potent toxic phthalates to evaluate additivity of effects. Scientific evidence regarding the other phthalates mentioned in the amendment to H.R. 4040 is lacking. Limited evidence indicates that DIDP, DINP, and DnOP might have effects on the immune system, but these phthalates appear to be much less toxic to developing rodents.

Data indicate that people are exposed to many phthalates, especially DEHP, DBP (di-n-butyl), and DBB. Children appear to be most heavily exposed. Data are insufficient to judge exposure for DINP, DIDP, and DnOP. Individuals such as newborn babies in the intensive care units of hospitals face multiple and continuous phthalate exposures.

Scientists are just beginning to explore the additive effects of exposure to multiple phthalates, as well as to phthalates in combination with certain pesticides.[89] To date, studies suggest there may be additive effects of multiple phthalate exposures.[90] The National Academy of Sciences is evaluating the risk of aggregate human exposure to multiple phthalates, and is expected to report before the end of 2008.

---

[88] According to Dr. Paul Foster, Acting Toxicology Branch Chief of the National Toxicology Program (personal communication, July 9, 2008), the structure that is known to produce toxic effects is a benzene ring of 6 atoms with two linear side chains in the ortho position of between 4 and 6 carbon atoms each. Longer or shorter side chains are not developmentally toxic or are much less toxic.

[89] Rider, C.V., J. Furr, V.S. Wilson, et al. 2008. A mixture of seven antiandrogens induces reproductive malformations in rats. *International Journal of Andrology* v. 31, n. 2, p. 249-262.

Fowler, Paul A., David R. Abramovich, Neva E. Haites, et al. 2007. Human fetal testis Leydig cell disruption by exposure to the pesticide dieldrin at low concentrations. *Human Reproduction*, v. 22, n. 11, p. 2919-2928.

[90] Hotchkiss, A.K., L.G. Parks-Saldutti, J.S. Ostby, et al. 2004. A mixture of the "antiandrogens" linuron and butyl benzyl phthalate alters sexual differentiation of the male rat in a cumulative fashion. *Biology of Reproduction*, v. 71, p. 1852-1861.

Howdeshell, Kembra L., Vickie S. Wilson, Johnathan Furr, et al. April 14, 2008. A mixture of five phthalate esters inhibits fetal testicular testosterone production in the Sprague Dawley rat in a cumulative, dose additive manner. *Toxicological Sciences Advance Access*. Reprint received from the author.

Rider, Cynthia V., Johnathan Furr, Vickie S. Wilson, et al. 2008, A mixture of seven antiandrogens induces reproductive malformations in rats. *International Journal of Andrology*, v. 31, p. 249-262.

Would the Amendment Reduce the Risks Without Generating Greater Risks?

By eliminating the use of six phthalates in child-care items and toys, the proposed amendment would codify the voluntary agreements reached by CPSC with product manufacturers (to keep DEHP and DINP out of nipples, pacifiers, and teething toys, and DEHP out of toys that might be mouthed) and reduce exposure to DINP, the only phthalate currently used in the United States to produce toys. The effect of banning use of the other phthalates is less clear, because their child-related uses are not known. However, because the law would prohibit their use as substitutes for DINP in toys, toys would be eliminated as a source of exposure.

For each use, different chemicals might be used in lieu of the six phthalates. Acetates might be used in some applications, phthalates other than the six specified might be used in others. The risks of such chemicals may be known or unknown. Given the lack of legal authority to require testing for a chemical proposed for most uses, and the cost of testing, new formulations of products might pose either more or less risk than the current formulations.