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IT'S IN OUR BLOOD: A CRITIQUE OF THE FDA'S RELUCTANCE TO REGULATE THE USE OF BISPHENOL A IN THE FOOD SUPPLY

RYAN J. CARRA, PH.D.[1]

INTRODUCTION

"Better Living Through Chemistry" is a mantra that the chemical industry has repeated for almost a hundred years, and it cannot be disputed that the discipline has improved our lives in many respects.[2] During that

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1. University of Virginia School of Law, J.D. Expected, Spring 2011; University of Illinois at Urbana-Champaign, Ph.D. in Organic Chemistry, 2008; College of William & Mary, B.S. in Chemistry, 2002. The author wishes to thank Professor Mimi Riley for her guidance.

2. The phrase was used as a slogan by DuPont starting in 1935. *See* John Kenly Smith, *DuPont: The Enlightened Organization*, DUPONT,

time, synthetic estrogens and progestins have revolutionized birth control,[3] antiviral therapies have significantly increased the life expectancy of people diagnosed with HIV,[4] and improvements in semiconductor and dopant technology have contributed to exponential increases in computing power.[5] This era has been fueled by an explosion in chemical research and manufacturing. As of 1997, over 12,000 different chemicals were used in commerce in quantities of over one million pounds per year.[6]

This enormous quantity of production has resulted, perhaps inevitably, in the proliferation of man-made chemicals throughout our ecosystem. Recent studies have demonstrated that no living creature is safe from these chemicals' effects. For example, polychlorinated biphenyls (PCBs), a class of coolant whose manufacture was banned worldwide in 2004,[7] have been detected in the milk, fatty tissue, and blood of arctic polar bears.[8] These chemicals are implicated in the discovery of hermaphroditic polar bears on an island in the remote Barents Sea in 1998.[9] Even the unborn are not safe

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http://www2.dupont.com/Heritage/en_US/Enlightened/Enlightened.html (last visited Feb. 4, 2011) (tracing the significant chemical advancements that led to a heightened standard of living for Americans post World War II).

3. For a brief discussion of the social impacts of oral contraception, see Elaine Tyler May, *Mother's Other Little Helper: The Pill*, WASH. POST, May 9, 2010, at B2 (discussing the social and scientific factors that led to over 80% of women taking birth control pills at some point in their lives). In 2002, oral contraceptives were the leading method of birth control in the United States, used by 11.6 million women. William D. Mosher et al., Div. of Vital Statistics, U.S. Ctrs. for Disease Control & Prevention, *Use of Contraception and Use of Family Planning Services in the United States: 1982-2002*, 350 ADVANCE DATA FROM VITAL & HEALTH STAT., Dec. 10, 2004, at 1, 7, *available at* http://www.cdc.gov/nchs/data/ad/ad350.pdf.

4. Between 1996 and 2005, improvements in antiviral therapies increased the life expectancy of a person starting treatment at age 20 by 13 years. Steven Reinberg, *HIV Patients Living Longer*, WASH. POST, July 25, 2008, http://www.washingtonpost.com/wp-dyn/content/article/2008/07/24/AR2008072403289.html.

5. *See Moore's Law: Raising the Bar*, INTEL CORP. (2005), http://download.intel.com/museum/Moores_Law/Printed_Materials/Moores_Law_Backgrounder.pdf (discussing improvements to transistors, and predicting that the semiconductor industry will soon surpass all other industries). *See also* Michelle Y. Simmons, *Scanning Probe Spectroscopy: Probing Dopants at the Atomic Level*, 4 NATURE PHYSICS 165, 165 (2008) (discussing recent research on dopant atoms in semiconductors that suggests that dopants might lead to "significant improvement in computing power").

6. John C. Dernbach, *The Unfocused Regulation of Toxic and Hazardous Pollutants*, 21 HARV. ENVTL. L. REV. 1, 28 (1997).

7. United Nations Environment Programme (UNEP): Stockholm Convention on Persistent Organic Pollutants, May 22, 2001, 40 I.L.M. 532. The Stockholm Convention on Persistent Organic Pollutants covered twelve substances in total, and provided for the immediate cease in PCB production. Miquel Porta & Ekhine Zumeta, *Implementing the Stockholm Treaty on Persistent Organic Pollutants*, 59 OCCUPATIONAL & ENVTL. MED. 651, 651 (2002).

8. Espen O. Henriksen et al., *Monitoring PCBs in Polar Bears: Lessons Learned from Svalbard*, 3 J. ENVTL. MONITORING 493, 493–94 (2001).

9. Øystein Wiig et al., *Female Pseudohermaphrodite Polar Bears at Svalbard*, 34 J. WILDLIFE DISEASES 792, 792, 795 (1998).

from industrial chemicals. In 2004, tests of the umbilical cord blood of ten babies revealed an average of 200 industrial chemicals per baby, including PCBs and perfluorooctanoic acid (PFOA), which has been classified as a likely human carcinogen.[10]

One particularly troublesome class of chemical pollutants affects the function of the body's delicate endocrine system by acting as natural hormones. Many of these endocrine disruptors are furtively dangerous because they bear no structural resemblance to natural hormones, making their effects difficult for scientists to predict. Furthermore, they can cause severe health problems, including reproductive abnormalities, cancer, and liver problems, at low concentrations.[11] Young people are especially sensitive to these compounds because of their effects on hormonally-regulated maturation processes.[12] Famous examples include the now-banned pesticide dichlorodiphenyltricholoroethane (DDT), which is best known domestically for endangering the bald eagle by causing thinning of its eggs,[13] and the aforementioned PCBs. One endocrine disruptor, whose health effects have been under increasing scrutiny over the last fifteen years, is the chemical bisphenol A (BPA). Like DDT and PCBs, BPA has been shown to have hormone-disrupting effects in laboratory animals.[14] Unlike DDT and PCBs, however, BPA is still actively produced and used worldwide. The United States alone had a production volume of 2.3 billion pounds in 2004, up from 16 million pounds in 1991.[15] BPA is primarily used as a monomer in the production of polycarbonate plastics, which can

10. JANE HOULIHAN ET AL., ENVTL. WORKING GRP., BODYBURDEN – THE POLLUTION IN NEWBORNS 13–14 (2005).

11. *See, e.g.*, *infra* notes 59–67 and accompanying text (collecting the results of empirical research on BPA, an endocrine disruptor).

12. *See, e.g.*, Yueliang L. Guo et al., *Growth Abnormalities in the Population Exposed* in Utero *and Early Postnatally to Polychlorinated Biphenyls and Dibenzofurans*, 103 ENVTL. HEALTH PERSP. (SUPP. 6) 117, 117–18 (1995) (describing a study of the developmental abnormalities of children whose mothers ingested PCB-contaminated rice oil before, or shortly after, their births). *See also* Cynthia F. Bearer, *Environmental Health Hazards: How Children are Different from Adults*, 5 FUTURE CHILD., Summer/Fall 1995 at 11, 11, 18 (explaining the unique impact that PCBs and other environmental contaminants have on children because of their physical, biological, and social characteristics).

13. Erik Stokstad, *Can the Bald Eagle Still Soar After it is Delisted?*, 316 SCIENCE 1689, 1689 (2007).

14. *See infra* notes 59–67 and accompanying text (discussing BPA studies on pregnant female rats).

15. CTR. FOR THE EVALUATION OF RISKS TO HUMAN REPROD., NAT'L TOXICOLOGY PROGRAM, NTP-CERHR-BPA-07, NTP-CERHR EXPERT PANEL REPORT ON THE REPRODUCTIVE AND DEVELOPMENTAL TOXICITY OF BISPHENOL A 3 (2007) [hereinafter NTP REPORT], *available at* http://cerhr.niehs.nih.gov/evals/bisphenol/BPAFinalEPVF112607.pdf. Worldwide production of BPA in 2003 was over 4.4 billion pounds. Iain A. Lang et al., *Association of Urinary Bisphenol A Concentration with Medical Disorders and Laboratory Abnormalities in Adults*, 300 JAMA 1303, 1303 (2008).

be used to make baby and water bottles, and in the epoxy resins which line aluminum food and drink cans.[16]

Despite the fact that BPA's many food contact applications fall under the jurisdiction of the Food and Drug Administration (FDA), the FDA has thus far declined to exert its regulatory power. As recently as 2008, FDA officials were insisting that current uses with food were safe,[17] but after a change in administrations and mounting reports of the harmful effects of BPA on animals and humans, the agency expressed "some concern" about BPA's effects in early 2010.[18] In the meantime, the chemical sparked a heated political battle, with Congressional Democrats and environmental groups making accusations of industry capture and the chemical industry scrambling to protect its interests.[19] The heat has been turned up both by the potentially disproportionate effects of BPA on children on one hand, and the chemical's continued utility and profitability on the other.[20] The fact that an intriguing, though inconclusive, case of the harmful effects of BPA on humans can be made has not helped settle the issue. As more studies about the potential effects of BPA were reported in the media, some industrial companies have attempted to curtail their use of the chemical. These companies include Similac, which now sells 91% of its baby formula BPA-free,[21] and Nalgene, which no longer uses BPA in the manufacture of

16. NTP REPORT, *supra* note 15, at 3, 11.

17. U.S. FOOD & DRUG ADMIN., DRAFT ASSESSMENT OF BISPHENOL A FOR USE IN FOOD CONTACT APPLICATIONS 36 (Aug. 14, 2008) [hereinafter DRAFT ASSESSMENT], *available at* http://www.fda.gov/ohrms/dockets/AC/08/briefing/2008-0038b1_01_02_FDA BPA Draft Assessment.pdf).

18. Denise Grady, *F.D.A. Concerned About Substance in Food Packaging*, N.Y. TIMES, Jan. 16, 2010, http://www.nytimes.com/2010/01/16/health/16plastic.html?_r=1.

19. *See* Denise Grady, *In Feast of Data on BPA Plastic, No Final Answer*, N.Y. TIMES, Sept. 7, 2010, at D1 (discussing concerns on both sides of the BPA debate).

20. *See* U.S. ENVTL. PROT. AGENCY, BISPHENOL A ACTION PLAN 18 (2010), *available at* http://www.epa.gov/opptintr/existingchemicals/pubs/actionplans/bpa_action_plan.pdf (detailing the Environmental Protection Agency's (EPA) plan to evaluate BPA's potential disproportionate impact on children). Meanwhile, studies show BPA is still being used in forty-six out of fifty grocery store cans that have been tested. Todd Zwillich, *Study by Consumer Groups Shows Bisphenol A is in 46 out of 50 Cans Tested*, WEBMD HEALTH NEWS (May 18, 2010), http://www.webmd.com/food-recipes/news/20100518/canned-food-may-expose-people-to-bpa?print=true.

21. Press Release, Abbott Laboratories, Abbott Reports on "BPA Free" Status (July 9, 2009), *available at* http://abbottnutrition.com/news/pressReleaseDetail.aspx?ContentTitle=Abbott-Leads-by-Achieving-BPA-Free-Status-for-its-Infant-Formulas&year=2009. According to the Environmental Working Group, Similac's powder and ready-to-eat formula is BPA free, but its concentrated liquid formulas "are still sold in BPA-lined metal cans." Jane Houlihan et al., *Timeline: BPA from Invention to Phase-Out*, ENVTL. WORKING GRP. (Aug. 4, 2010), http://www.ewg.org/reports/bpatimeline.

its water bottles.[22] However, the FDA has still not acted to regulate BPA's use.

Depending on one's point of view, BPA makes a fascinating case study of industry capture or industry self-regulation. This paper will analyze the factors that led to BPA becoming a political football and the lessons that can be learned from the BPA story. Ultimately, this paper recommends that the FDA ban the chemical from the manufacture of food contact devices designed for children and to keep the public aware of BPA's potential dangers. Part I of this paper will discuss BPA's current uses, and the resulting human exposure.[23] Part II will discuss the studies purporting to evaluate the dangers of BPA to public health, and the controversy surrounding them.[24] Part III will discuss the FDA's increasingly controversial refusal to regulate BPA's use.[25] Finally, Part IV will discuss the FDA's policy options, including the extent of its authority to regulate BPA.[26]

I. BISPHENOL A, ITS USES, AND THE RESULTING HUMAN EXPOSURE

BPA, a man-made chemical, was first synthesized in the laboratory in 1891.[27] In 1936, despite the fact that it bears no structural similarities to natural hormones, BPA was discovered to have estrogenic properties.[28] Ideas of using BPA to control pregnancies were set aside when another synthetic chemical with even more powerful estrogenic properties, diethylstilbestrol (DES), was discovered.[29] In an illustration of the potential dangers of synthetic hormones, almost all uses of DES were discontinued in 1971, when it was linked to vaginal cancer in the daughters of mothers who had ingested it.[30]

In the 1940s, the chemical industry began using BPA in the manufacture of plastics,[31] and its use has grown rapidly within the past twenty years. Between 1991 and 2004, the production volume of BPA in

22. *FDA Forms Agency-Wide Task Force to Review Safety of Bisphenol A*, FDA WK., Apr. 25, 2008.

23. *See infra* Part I.

24. *See infra* Part II.

25. *See infra* Part III.

26. *See infra* Part IV.

27. Houlihan et al., *supra* note 21.

28. Edward C. Dodds & Wilfrid Lawson, *Synthetic Estrogenic Agents Without the Phenanthrene Nucleus*, 137 NATURE 996, 996 (1936).

29. *See* Houlihan et al., *supra* note 21 (noting that BPA's use as a pharmaceutical hormone was precluded by the invention of DES).

30. Hervy E. Averette, *Cancers of the Female Reproductive System*, *in* THE MERCK MANUAL OF MEDICAL INFORMATION – HOME EDITION 1401, 1408 (Mark H. Beers et al. eds., 2d ed. 2003).

31. Houlihan et al., *supra* note 21.

the United States increased by a factor of over one hundred.[32] In 2003, it was estimated that worldwide demand of BPA was still growing by 6% to 10% annually.[33]

BPA's primary industrial use is as a monomer used in the manufacture of two classes of polymers: polycarbonate plastics and epoxy resins.[34] Polycarbonate plastics are used to make compact discs, medical devices, and food containers such as water and baby bottles.[35] Epoxy resins are used in adhesives, PVC plastic, and protective coatings, including those used on the inside of aluminum food and drink cans.[36] Of the 1.9 billion pounds of BPA consumed in the United States in 2003, approximately 74% was used in the manufacture of polycarbonate plastics and 21% was used in the manufacture of epoxy resins.[37] Since the "highest potential for human exposure to [BPA] is from products that directly contain food," this paper will focus on exposure from food contact applications.[38]

Human exposure to BPA comes primarily from either trace amounts of unreacted monomer contained within a manufactured polymer or monomer generated by the breakdown of a polymer.[39] Examples of both routes of exposure have been demonstrated in several independent studies measuring the migration of BPA from baby bottles to food simulants.[40] In these studies, which were summarized in a 2007 report by the National Toxicology Program (NTP),[41] measurements of BPA migration[42] were

32. *See* NTP REPORT, *supra* note 15, at 3 (reporting that the production volume of BPA in the United States increased from 16 million pounds in 1991 to approximately 2.3 billion pounds in mid-2004.) As of 2007, six plants manufacture BPA in the United States and three out of the country's four polycarbonate plants are located within BPA plants. *Id.* There are also 13 epoxy plants in the United States, but it is not clear how many of them use BPA. *Id.*

33. Lang et al., *supra* note 15, at 1303.

34. NTP REPORT, *supra* note 15, at 3.

35. *Id.*

36. *Id.* at 3, 11–12.

37. *Id.* at 3.

38. *Id.* at 6. By "food," NTP clearly meant both food and beverages: "[e]xamples of food contact materials that can contain bisphenol A include food and beverage containers with internal epoxy resin coatings and polycarbonate tableware and bottles." *Id.*

39. *Id.* at 4, 6. In addition, babies can be exposed to BPA through their mother's breast milk. *Id.* at 6. One study of 32 women found an average concentration of 1.4 micrograms per liter of BPA in breast milk. *Id.*; Antonia M. Calafat et al., *Human Exposure Assessment to Environmental Chemicals Using Biomonitoring*, 29 INT'L J. ANDROLOGY 166, 168 (2005).

40. *See* NTP REPORT, *supra* note 15, at 7–10 (showing tabulated and summarized results of studies measuring the migration of BPA from containers into foods).

41. *Id.* The National Toxicology Program is an interagency program within the Department of Health and Human Services. *About the NTP – National Toxicology Program*, U.S. DEP'T OF HEALTH & HUMAN SERVS., http://ntp.niehs.nih.gov/?objectid=7201637B-BDB7-CEBA-F57E39896A08F1BB (last visited Feb. 6, 2011).

42. Food simulants used to measure BPA migration included water, ethanolic solutions, acetic acid solutions, n-heptane, and fractionated coconut oil as well as actual fruit juice and infant

taken both before and after repeated washings of the bottles.[43] Some studies showed decreased BPA migration after repeated washings, suggesting that the bulk of exposure to a child would come from unreacted BPA on the surface of the bottle.[44] Other studies gave the opposite results, suggesting that subjecting the baby bottles to the washing conditions caused breakdown of the polymer, releasing BPA to the inside surface of the bottle.[45] The large variability of the experimental conditions between studies caused NTP not to announce any conclusions from this data.[46]

Studies of BPA migration from the lining of aluminum cans also have demonstrated reason for concern but a frustrating amount of data variability.[47] In one study, empty cans were filled with different foods, sealed, processed at different temperatures, and stored for various times, and half of the cans were dented after processing.[48] The study found that 80 to100% of BPA migration occurred immediately after processing; the condition of the can, temperature during processing, and length of storage had little effect.[49] These results conflicted with the results of other studies that showed increased leaching when cans were heated.[50]

Perhaps most troubling of all of the research discrepancies are those seeking to estimate total BPA intake in humans. In 2006, a study attempted to estimate exposures of 257 children in North Carolina and Ohio by measuring BPA concentration in indoor and outdoor air, food, dust, and

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formula. NTP REPORT, *supra* note 15, at 7–10. Transfer conditions ranged from microwaving for 30 seconds and letting stand for 20 minutes to heating at 49° C for ten days. *Id.*

43. *Id.* Conditions simulating washing also varied by study, included brushing, immersing in boiling water, and using a commercial dishwasher. *Id.*

44. *Id.* at 7 (citing reports that demonstrated decreased BPA concentrations as bottles were used and washed, suggesting that migration was more likely the result of the monomer present on the surface of the product rather than polymer breakdown over time).

45. *Id.* The most alarming results showed a BPA migration concentration of 8.4 micrograms per liter after 51 cycles in a dishwasher. *Id.* at 9. *Compare also id.* at 357 nn.40–46 (collecting studies of BPA concentrations in baby bottles).

46. *Id.* at 352–53 (discussing the inconsistent results of BPA studies and citing a need for more data relating to human exposure assessments). However, following the release of the NTP's report, a study found that college students who drank most of their cold beverages from polycarbonate drinking bottles for one week showed a 69% increase in urinary BPA concentration. Jenny L. Carwile et al., *Polycarbonate Bottle Use and Urinary Bisphenol A Concentrations*, 117 ENVTL. HEALTH PERSP. 1368, 1368, 1370 (2009).

47. *See* NTP REPORT, *supra* note 15, at 11–13 (demonstrating the varied results of BPA concentrations from studies using canned foods).

48. *Id.* at 11 (citing A. Goodson et al., *Migration of Bisphenol A from Can Coatings – Effects of Damage, Storage Conditions and Heating*, 21 FOOD ADDITIVES & CONTAMINANTS 1015, 1017 (2004)).

49. *Id.* at 11.

50. *Id.* (citing Yuji Takao et al., *Release of Bisphenol A from Food Can Lining upon Heating*, 48 J. HEALTH SCI. 331, 333 (2002)).

soil.[51] The calculated medium BPA exposure was 0.07 micrograms per kilogram body mass per day (μg/kg/d) in North Carolina and 0.06 μg/kg/d in Ohio.[52] In another study, the BPA concentration of the urine of approximately 1400 men and women was measured.[53] Based on the results, the authors estimated that "exposure among the general US population is likely to exceed the 50-μg/kg per day reference dose currently recommended by the US Environmental Protection Agency."[54] Thus, the two studies' estimates of BPA exposure to two population groups in the United States differ by an astounding factor of over 800.[55]

From these studies, on which the NTP had few summarizing thoughts of its own, a few comments may be made. First, it is undeniable that BPA has the potential to leach into baby formula and canned goods.[56] Second, babies are at risk to get a double dose of BPA if their mothers buy formula in aluminum cans and transfer it to BPA-containing polycarbonate baby bottles. Third, it can be frustrating for a regulatory body to cull meaningful conclusions from different studies. The studies discussed above used different sources of baby bottles and aluminum cans, and vastly different experimental conditions.[57] They also showed widely varying results, with many finding BPA leaching below the limits of detection.[58] With this type of data, it is difficult to draw a meaningful, intellectually honest conclusion. Conversely, it is easy to "cherry pick" data so that a biased observer may

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51. *Id.* at 25 (citing Nancy K. Wilson et al., *An Observational Study of the Potential Exposures of Preschool Children to Pentachlorophenol, Bisphenol-A, and Nonylphenol at Home and Daycare*, 103 ENVTL. RES. 9, 10–11 (2007)).

52. *Id.* (citing Wilson et al., *supra* note 51, at 16).

53. Lang et al., *supra* note 15, at 1305.

54. *Id.* at 1308. The authors state that "near-complete urinary excretion has been shown to occur within 24 hours of a single high dose" of BPA. *Id.*

55. *Compare* Lang et al., *supra* note 15, at 1308 (finding that "exposure among the U.S. population is likely to exceed 50-μg/kg per day"), *with* Wilson et al., *supra* note 51, at 16 (finding median BPA exposure not to exceed 0.07-μg/kg per day). Clearly, one or both studies are seriously flawed. The study less likely to be flawed is the one which is actually measuring BPA coming out of the body—the one reported by Lang et al.—because it carries with it one fewer level of assumptions (i.e. how much BPA in the atmosphere is actually taken in to the body).

56. *See infra* Part IV (discussing the issue of whether the magnitude of leaching observed in the most alarming studies would present health problems).

57. *See, e.g.*, *supra* notes 42–48 and accompanying text (describing the different food stimulants and transfer conditions for each study).

58. Limits of detection varied by more than an order of magnitude among some studies. For instance, one study showed that storage of baby bottles in water for 39 weeks caused BPA migration to increase from around 0.3 micrograms per liter to 4.7 μ/L. J.E. Biles et al., *Determination of Bisphenol-A in Reusable Polycarbonate Food-Contact Plastics and Migration to Food-Simulating Liquids*, 45 J. AGRIC. FOOD CHEMISTRY 3541, 3544 tbl.5 (1997). Another study, conducted by the plastics industry, showed no migration up to the limit of detection, which was 5 μ/L. Susan R. Howe & Lester Borodinsky, *Potential Exposure to Bisphenol A from Food-Contact Use of Polycarbonate Resins*, 15 FOOD ADDITIVES & CONTAMINANTS 370 (1998).

support whatever claim he or she wishes. Data on the effects of BPA in living organisms, which will be discussed in Part II, is similarly inconclusive. Unsurprisingly, serious accusations of bias about data interpretation have arisen.

II. CONNECTION BETWEEN BPA EXPOSURE AND PUBLIC HEALTH

Beginning in 1997, studies of the effects of BPA on laboratory animals began to trickle out of academia.[59] These studies were characterized by the significant effects they had on the physiology of laboratory animals at doses well below that of the safety standard of 50 μg/kg/d set by the EPA in 1988.[60] Low doses of BPA given to pregnant female rats were found to significantly affect the prostate size of their male offspring[61] and the speed of puberty of their female offspring.[62] The nursing characteristics of female mice who were either exposed to BPA neonatally or in adulthood were also found to be affected.[63] BPA has also been found to stress the livers of rats,[64] and to increase plasma insulin, resulting in the chance that "BPA

59. *See, e.g.*, John B. Colerangle & Deodutta Roy, *Profound Effects of the Weak Environmental Estrogen-like Chemical Bisphenol A on the Growth of the Mammary Gland of Noble Rats*, 60 J. STEROID BIOCHEMISTRY & MOLECULAR BIOLOGY 153, 154–55 (1997) (describing a study performed to investigate the effect on BPA on cellular growth using Noble rats and the results). Results from agency and industry research began to emerge in 1995. *Consumer Bisphenol A Exposure & Safety Information*, BISPHENOL A, http://www.bisphenol-a.org/human/consafety.html (last visited Feb. 8, 2011).

60. *See* Laura N. Vandenberg et al., *Human Exposure to Bisphenol A (BPA)*, 24 REPRODUCTIVE TOXICOLOGY 139, 140–41 (2007) (noting that there are more than 150 published studies of the low-dose effect of BPA on animals and that more than 40 of the studies involved dosages below 50 μg/kg/d). *See also* Aurora A. Saulo, *Bisphenol A*, FOOD SAFETY & TECH., May 2008, at 1, 1–2 (stating that the EPA set the 50 μg/kg/d standard in 1988). This safety standard was still being cited by the FDA as late as 2008. DRAFT ASSESSMENT, *supra* note 17, at 5–6.

61. Susan C. Nagel et al., *Relative Binding Affinity-Serum Modified Access (RBA-SMA) Assay Predicts the Relative* In Vivo *Bioactivity of the Xenoestrogens Bisphenol A and Octylphenol*, 105 ENVTL. HEALTH PERSP. 70, 74 (1997). Researchers fed BPA to pregnant mice at a quantity of 2 μg/kg/d. *Id.* Upon reaching adulthood, the male offspring of exposed females showed a 30% increased prostate weight relative to a control group. *Id.*

62. Kembra L. Howdeshell et al., *Exposure to Bisphenol A Advances Puberty*, 401 NATURE 763, 763 (1999). The female offspring of mothers who were fed 2.4 μg/kg/d BPA during pregnancy grew at an increased rate, and showed a "significantly reduced number of days" between vaginal opening and first ovulation. *Id.*

63. Paola Palanza et al., *Exposure to a Low Dose of Bisphenol A during Fetal Life or in Adulthood Alters Maternal Behavior in Mice*, 110 ENVTL. HEALTH PERSP. (SUPP. 3) 415, 417–18 & figs.1–2 (2002). Compared to the control group, female mice exposed to BPA as fetuses or during pregnancy spent significantly less time nursing, *id.* at 417, and more time out of the nest. *Id.* at 418. The relevant dose of BPA was 10 μg/kg/d. *Id.* at 417 fig.1.

64. V. Bindhumol et al., *Bisphenol A Induces Reactive Oxygen Species Generation in the Liver of Male Rats*, 188 TOXICOLOGY 117, 123 (2003). Results indicated that the livers of rats that were fed BPA at a rate of 20 μg/kg/d and 40 μg/kg/d experienced oxidative stress due to a decrease in antioxidant enzymes. *Id.* at 118, 123.

exposure may enhance the risk of developing type II diabetes."[65] Finally, the female offspring of rats exposed to BPA during pregnancy were shown to have an increased rate of precancerous breast lesions[66] and changes to mammary cell structure.[67]

Animal studies, of course, leave open the question of how similar BPA's effects in humans will be. A clue to the answer was given in 2008, when a study found a significant correlation between the quantity of BPA in urine and incidence of cardiovascular disease and diabetes.[68] Of course, because of the delay between exposure to BPA and injury, and the many possible causes of diabetes and heart disease, these associations are not necessarily causal.[69]

In contrast to the many academic studies which found low-dose physiological effects caused by BPA exposure, three industry-funded studies performed over the last ten years showed no significant effects in rodents.[70] In these studies, multiple generations of rats were dosed with various amounts of BPA and effects on reflexes, reproductive capabilities,

65. *See* Ana B. Ropero et al., *Bisphenol-A Disruption of the Endocrine Pancreas and Blood Glucose Homeostasis*, 31 INT'L J. ANDROLOGY 194, 198 (2008) (concluding that a BPA dose as low as 10 μg/kg could result in "a rapid increase in plasma insulin").

66. Milena Durando et al., *Prenatal Bisphenol A Exposure Induces Preneoplastic Lesions in the Mammary Gland in Wistar Rats*, 115 ENVTL. HEALTH PERSP. 80, 85 (2007) (demonstrating a correlation between exposure to 25 μg/kg/d BPA in pregnant rats and an increase in preneoplastic lesions).

67. Raquel Moral et al., *Effect of Prenatal Exposure to the Endocrine Disruptor Bisphenol A on Mammary Gland Morphology and Gene Expression Signature*, 196 J. ENDOCRINOLOGY 101, 102, 104–05 (2008) (finding modifications to the mammary glands of pregnant rats exposed to either a low dose (25 μg/kg/d) or a high dose (250 μg/kg/d) of BPA).

68. Lang et al., *supra* note 15, at 1305.

69. *See id.* at 1308–09 (noting that health effects are most often associated with long-term exposure, that other studies have found associations between toxins and diseases such as diabetes, and that more study is needed to determine if the association between BPA and disease in humans is causal).

70. Makoto Ema et al., *Rat Two-Generation Reproductive Toxicity Study of Bisphenol A*, 15 REPROD. TOXICOLOGY 505, 522 (2001); Rochelle W. Tyl et al., *Three-Generation Reproductive Toxicity Study of Dietary Bisphenol A in CD Sprague-Dawley Rats*, 68 TOXICOLOGICAL SCI. 121, 144 (2002) [hereinafter Tyl et al., *Three-Generation*]; Rochelle W. Tyl et al., *Two-Generation Reproductive Toxicity Study of Dietary Bisphenol A in CD-1 (Swiss) Mice*, 104 TOXICOLOGICAL SCI. 362, 382 (2008) [hereinafter Tyl et al., *Two-Generation*]. The Ema et al., *supra* at 522, study was funded by the Japanese Ministry of Health and Welfare; the Tyl et al., *Three-Generation*, *supra* at 144, study was funded by The Society of the Plastics Industry, Inc.; and Tyl et al., *Two-Generation*, *supra* at 383, study was funded by the Polycarbonate/BPA Global Group.

sperm parameters, anogenital distance, and organ weight were measured.[71] The only significant abnormalities occurred at high doses of BPA.[72]

These industry-funded studies were criticized by the Environmental Working Group (EWG) in 2008 for having "critical methodological flaws."[73] EWG's complaints included a lack of a suitable positive control[74] and a failure to study the most sensitive targets of BPA.[75] For instance, while academic studies showed BPA caused precancerous lesions in the breasts of rats, the industry studies did not collect mammary tissue in a way that would have allowed the detection of lesions.[76] Also, while academic studies showed that BPA affects weaning behavior, two of the industry studies did not address behavioral affects at all,[77] and the third did not address the loss of sex-specialized behavior.[78]

To summarize the scientific data currently available, industry-funded studies have demonstrated that, at low doses, BPA does not have immediate, acute physiological effects. However, these studies were carried out in such a way as to minimize the observance of chronic effects that other studies suggest BPA may have. As such, it would be a mistake to rely only on the industry-funded studies when making scientific conclusions. Part III of the paper will discuss the FDA's analysis of the scientific data available, and the resulting outcry from Congress, nonprofit groups, and the public.

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71. Ema et al., *supra* note 70, at 506 (indicating a dosage range of 0.2 μg/kg/day to 200 μg/kg/day); Tyl et al., *Three-Generation*, *supra* note 70, at 123 (indicating a dosage range of 0.001 mg/kg/day to 500 mg BPA/kg/day); Tyl et al., *Two-Generation*, *supra* note 70, at 364, 366 tbl.1 (indicating a dosage range of 0.003 mg/kg/day to 600 mg BPA/kg/day).

72. Tyl et al., *Three-Generation*, *supra* note 70, at 131; Tyl et al., *Two-Generation*, *supra* note 70, at 382. The Japanese study involved comparatively low doses of BPA, and found no adverse affects. Ema et al., *supra* note 70, at 522.

73. Letter from Anila Jacob & Sonya Lunder, Envtl. Working Grp., to Martin Philbert, BPA Subcomm. Chair, U.S. Food & Drug Admin. 5 (Sept. 12, 2008) [hereinafter EWG Letter], *available at* http://www.ewg.org/files/BPA_091208.pdf. *See infra* Part III.B (discussing further criticisms included in the EWG letter, for example the FDA's reliance on industry studies, and its disregard for academic studies that found significant physiological effects of BPA on rodents).

74. EWG Letter, *supra* note 73, at 6. The EWG explained that, in this context, a positive control would involve dosing a group of rats with a hormone known to have physiological effects. *Id.* As such, the EWG asserted that two of the three studies in question wholly lacked a positive control. *Id.* Moreover, the third used such a large dose of estrogen that EWG claimed it did not validate the study's ability to evaluate low-dose effects. *Id.*

75. *Id.*

76. *See id.* at 7 (explaining that the industry-funded studies did not show increased rates of cancer in BPA-dosed rats because the rats were sacrificed at one year of age, "which is not sufficiently old to expect tumor formation").

77. *Id.*

78. *Id.*

III. WHAT IS SAFE? FDA'S ANALYSIS OF CLINICAL DATA, AND THE RESULTING BLOWBACK

A. FDA's Position on BPA

As discussed in the introduction, the lack of data comprising a slam-dunk case indicating that BPA is safe or unsafe makes it tempting for an interested party to present only the most favorable data. One might expect industry to promote most heavily studies showing that their product is safe and, conversely, consumer groups to cite studies showing that their membership should be concerned. As late as 2008, however, it appears likely that even the FDA was engaging in such scientific "cherry picking," resulting in accusations of bias and conflicts of interest.[79]

Between 1999 and 2008, as more studies demonstrated the potentially harmful effects of BPA, the FDA's failure to regulate it as a food additive came under increased scrutiny.[80] In early 2008, House Energy and Commerce Committee Chair John Dingell (D-MI) and Oversight Subcommittee Chair Bart Stupak (D-MI) sent a letter to the FDA demanding to know what information the FDA was relying on to conclude that BPA was safe.[81] The FDA responded to these inquiries in August 2008 with a Draft Assessment justifying its continued refusal to regulate BPA.[82]

Much to the consternation of the Democratic Congress and nonprofit consumer and environmental groups, the Draft Assessment reiterated the FDA's stance that BPA was safe for use in food contact applications.[83] In supporting this conclusion, the only studies to which the FDA gave full weight were the industry-funded studies discussed above.[84] Academic

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79. *See, e.g.*, Susanne Rust & Meg Kissinger, *Critics Slam Chemical Report: Scientists Note Flaws in Bisphenol A Study; Lawmaker Wants Ban*, MILWAUKEE J. SENTINEL, Oct. 24, 2008, at A1 (reporting on an investigation that revealed significant industry influence in the commission and execution of the FDA's draft assessment of BPA, and detailing the conflict of interest accusations that have come from a wide range of sources).

80. *See infra* Part IV. See also Federal Food, Drug, and Cosmetic Act, 21 U.S.C. § 321(s) (2006), which establishes that the FDA is not required to regulate a compound as a "food additive" if the substance "is not generally recognized, among experts qualified by scientific training and experience to evaluate its safety, as having been adequately shown through scientific procedures . . . to be safe under the conditions of its intended use."

81. *House Democrats Probe FDA Role in Infant Formula Packaging*, FDA WK., Jan. 25, 2008, at 8.

82. *See* DRAFT ASSESSMENT, *supra* note 17, at 2 (finding that levels of BPA exposure for infants and adults fall within adequate margins of safety).

83. *See id.* at 36 ("FDA concludes that an adequate margin of safety exists for BPA at current levels of exposure from food contact uses.").

84. *See id.* at 15–16, 22–23 (discussing the FDA's criteria for its use of published studies in making food safety evaluations and noting that it opted to conduct a full review of two industry-funded BPA studies, one sponsored by the American Plastics Council and the other by the Society of Plastics Industry). *See also* Christopher Lee, *FDA Draft Report: No Risk from BPA in Food*

studies were given little or no weight due to small sample size, limited dose groups, lack of positive control, or use of a dosing method other than oral.[85] To justify this narrow view of available data, the FDA cited its regulations for Good Laboratory Practices (GLP), which are designed to "assure the quality and integrity of... safety data."[86] GLP regulate how data is reported to the FDA, as well as the treatment of laboratory animals, and standards for equipment and personnel.[87] While both of the industry studies the FDA cited were performed using GLP, none of the academic studies discussed above were.[88] Thus, the FDA did not give full weight to any study which showed the harmful effects of BPA at low doses.[89]

B. Complaints About FDA's Exclusion of Academic Research

Scientists and nonprofit groups voiced their displeasure at the FDA's exclusion of non-GLP studies from its Draft Assessment. One group of scientists complained that the FDA was excluding non-GLP studies based on two misguided assumptions: 1) that GLP ensured good science, and 2) that non-GLP studies never constitute good science.[90] Most of the non-GLP

Containers, WASH. POST, Aug. 16, 2008, at A2 (reporting that both the FDA and the EPA have deemed BPA safe, "largely on the strength of two industry-funded studies that found no problems").

85. *See* DRAFT ASSESSMENT, *supra* note 17, at 16 (stating that two common limitations in studies rejected by the FDA are "lack of a positive control" and "internal dose measurement"). *See also* EWG Letter, *supra* note 73, at 7 (stating that the FDA dismissed the results of academic BPA studies that demonstrated adverse affects at low doses).

86. 21 C.F.R. § 58.1(a) (2010); *see also* DRAFT ASSESSMENT, *supra* note 17, at 15 (stating that the FDA's guidelines for studies for submission to the FDA follow the good laboratory practices laid out in 21 C.F.R. § 58).

87. DRAFT ASSESSMENT, *supra* note 17, at 15–16; *Scientists Blast FDA Emphasis on Studies Done Under Good Lab Practices*, FDA WK., Nov. 7, 2008.

88. *See* Memorandum from Yan Gu, Toxicology Group II, Div. of Food Contact Notifications to Michelle Twaroski, Team Leader, Toxicology Group I, Div. of Food Contact Notifications (July 18, 2007) (stating that the Society of Plastics Industry-sponsored study adhered to GLP regulations); Memorandum from Mary E. Shackelford, Toxicology Group II, Div. of Food Contact Notifications to Michelle Twaroski, Team Leader, Toxicology Group 1, Div. of Food Contact Notifications (July 24, 2007) (stating that the American Plastics Council-sponsored study followed GLP guidelines); *cf.* Frederick S. vom Saal, *Comments on NTP April 2008 Draft Report on Bisphenol A (BPA)*, NAT'L TOXICOLOGY PROGRAM (May 26, 2008) http://cerhr.niehs.nih.gov/evals/bisphenol/pubcomm/BPA(40)vomsaal26May2008.pdf (stating that, due to paper work and high expense, only industry conducts GLP experiments).

89. *See* EWG Letter, *supra* note 73, at 1 (noting the FDA rejected 12 studies showing low toxicity of BPA).

90. *See* John Peterson Myers et al., *Why Public Health Agencies Cannot Depend on Good Laboratory Practices as a Criterion for Selecting Data: The Case of Bisphenol A*, 117 ENVTL. HEALTH PERSP. 309, 310–11 (2009) (arguing that the FDA's acceptance of only GLP studies implies that non-GLP studies are "not reliable or valid," and that the FDA has blindly accepted GLP studies that have been "harshly criticized in peer-reviewed publications"). The scientist

work that the FDA excluded in its analysis was funded by the National Institutes of Health (NIH) or its foreign equivalents.[91] The scientists asserted that NIH-funded studies actually carry stronger guarantees of quality than GLP studies because of the strict grant requirements.[92] Addressing the FDA's concern about small sample size in non-GLP studies, the group complained that NIH and GLP animal use guidelines are mutually exclusive.[93] Thus, a study funded by NIH cannot possibly meet the FDA's GLP standards, no matter how scientifically sound it is. Finally, the scientists also pointed out the bitter irony that GLP, used here to exclude independent science in favor of industry science, was originally instituted in response to a "2-year federal investigation into sloppy laboratory practices of a number of private research companies."[94]

The EWG also protested the exclusion of non-GLP studies.[95] Responding to the FDA's assertion that subcutaneous dosing was improper, the EWG cited a study in which scientists administered BPA to newborn mice both orally and subcutaneously and found no significant differences in BPA plasma levels between the two groups.[96]

The EWG makes a compelling case that the FDA improperly excluded good scientific research when analyzing the safety of BPA. It was improper to give little or no weight to non-GLP studies for several reasons. Most of the best life scientists in the country are funded by the NIH,[97] but scientists

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authors pointed out many perceived flaws in the industry-funded studies, including "the use of insensitive, out-of-date protocols and assays," and flawed dissection procedures. *Id.* at 311–13.

91. *See* Letter from Sen. Chuck Grassley to Andrew C. von Eschenbach, M.D., Comm'r, U.S. Food & Drug Admin. (Sep. 16, 2008) (addressing reports that the FDA's BPA study ignored many NIH funded studies). Members of a science-based nonprofit have charged that the FDA ignored assessments performed by the National Toxicology program, which has reviewed numerous foreign studies on BPA in making its assessments. *The FDA Declares that Bisphenol A is Safe, Despite Scientific Evidence*, UNION OF CONCERNED SCIENTISTS, http://www.ucsusa.org/scientific_integrity/abuses_of_science/bisphenol-a.html (last visited Feb. 9, 2011); *see also* NAT'L TOXICOLOGY PROGRAM, U.S. DEP'T OF HEALTH & HUMAN SERVS., NIH PUB. NO. 08-5994, NTP-CERHR MONOGRAPH ON THE POTENTIAL HUMAN REPRODUCTIVE AND DEVELOPMENTAL EFFECTS OF BISPHENOL A 45 (2008) (referencing numerous foreign studies on BPA including those conducted by the European Union).

92. *See* Myers et al., *supra* note 90, at 311. According to the group of scientists, NIH studies must be led by a principal investigator with "demonstrated competence to conduct the research," be published in peer-reviewed journals, and be subjected to independent replication of results. *Id.*

93. *See id.* at 313–14 (noting that large animal groups are considered "good science" by the FDA but "arbitrarily large" animal groups violate NIH guidelines).

94. *Id.* at 309–10.

95. *See* EWG Letter, *supra* note 73, at 11 (noting that the FDA's assessment failed to include a "rich body or research" that indicated harmful effects from BPA exposure).

96. EWG Letter, *supra* note 73, at 8.

97. *See About the National Institutes of Health*, NAT'L INSTS. HEALTH, http://www.nih.gov/about/index.html (last visited Feb. 9, 2011) (stating that NIH has supported

cannot use these funds to do GLP studies. Thus, a strict fidelity to GLP data by the FDA necessarily results in the exclusion of some of the country's best scientific work. In addition, automatic exclusion of independent scientific research violates the stated purpose of the GLP rules.[98] The FDA should evaluate the quality of both GLP and non-GLP data individually when deciding how much weight to grant it.[99]

C. Accusations of Conflict of Interest at FDA

In July 2008, a retired medical supply manufacturer[100] made a $5 million donation to a research center co-directed by FDA scientist Martin Philbert.[101] The donation was almost 25 times the size of the annual budget of the research center.[102] Further, this donation occurred around the time that Philbert was chosen to chair the BPA Subcommittee in charge of evaluating the Draft Assessment discussed above.[103] By the time the donation was made public by the Milwaukee Journal Sentinel in October 2008,[104] the Draft Assessment had already been completed, but Philbert still had not revealed his conflict of interest to his FDA colleagues.[105] Democratic members of the House Energy and Commerce Committee sent a letter to FDA Commissioner Andrew von Eschenbach demanding to know whether the donation gave rise to a conflict of interest.[106] Two members of the House Appropriations Agriculture Subcommittee called on

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the research of "more than 130 Nobel Prize winners" whose studies have led to important discoveries including the "development of MRI").

98. *See* 21 C.F.R. § 58.1(a) (2010) ("Compliance with this part is intended to assure the quality and integrity of the safety data.").

99. Federal Regulations do not require the FDA to ignore non-GLP work; in fact, the FDA has acted on non-GLP data in the past. *See* EWG Letter, *supra* note 73, at 5 ("FDA issued immediate guidance to patients and initiated changes to drug labeling when an academic article suggested problems with the drug Procrit in patients with chronic kidney disease.").

100. BPA is a component of some medical supplies. *See* Jarred A. Favole & Alicia Mundy, *FDA Seeks Data on Medical Devices, Drugs Containing BPA*, WALL ST. J., Oct. 15, 2008, http://online.wsj.com/article/SB122409377059237095.html.

101. Susanne Rust & Meg Kissinger, *Donation Raises Questions for Head of FDA's Bisphenol A Panel*, MILWAUKEE J. SENTINEL, Oct. 12, 2008, http://www.jsonline.com/news/32431234.html.

102. *Id.* The research center is the University of Michigan Risk Science Center, of which Philbert is founder and co-director. *Id.*

103. *See id.* (stating that the head of the FDA's science board believed Philbert was appointed chair of the BPA subcommittee in July of 2008, which was the same month that the donation came in).

104. *Id.*

105. The Draft Assessment was released on August 14, 2008 while Philbert's conflict of interest was not revealed until the publication of an article in the Milwaukee Sentinel two months later, in October 2008. DRAFT ASSESSMENT, *supra* note 17; Rust & Kissinger, *supra* note 101.

106. *House Dems Request Interview with Commissioner on BPA*, FDA WK., Oct. 17, 2008.

Philbert to recuse himself.[107] Less than three weeks after the disclosure of the donation, Philbert's subcommittee reported that the Draft Assessment was seriously flawed and the FDA's position on BPA should be reevaluated.[108]

D. Congress' Response to FDA's Inaction

In Federalist 10, James Madison argued that the bigger a government is the harder it is for special interests to control.[109] The response by House Democrats to the FDA's position on BPA is a prime illustration of Madison's argument. The FDA's refusal to change its position on BPA in the face of scientific evidence of its danger is an example of industry capture, but the House Democrats have remained uninfluenced by those special interests, and have continued to challenge the FDA and industry.[110]

In May 2008, Democratic Energy and Commerce Subcommittee members sent letters to manufacturers of infant formula, asking them to remove BPA from their products.[111] The next month, Representative Edward Markey (D-MA) introduced legislation which would have, within 180 days of passage, prohibited food and beverage manufacturers from using BPA in packaging.[112] In May and June 2008, the FDA Associate Commissioner for Science Norris Alderson testified at a Senate commerce hearing and a House Energy and Commerce hearing, defending the FDA's position on BPA. Commissioner Alderson faced stiff criticism from Senator John Kerry (D-MA) at this hearing.[113]

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107. *Id.*

108. *Science Board: FDA Should Consider Non-GLP BPA Studies*, FDA WK., Nov. 7, 2008.

109. THE FEDERALIST NO. 10 at 21–22 (James Madison) (Roy P. Fairfield ed., Johns Hopkins Univ. Press 1981).

110. It could be plausibly argued that House Democrats were simply trying to score political points by attacking the FDA under the unpopular Bush Administration, or were acting under the influence of consumer or environmental groups.

111. *FDA's BPA Report Due Next Week; Devices Not Part of Review*, FDA WK., Aug. 15, 2008.

112. *Markey Introduces Bill to Ban BPA Use; FDA Insists Chemical is Safe*, FDA WK., June 13, 2008. The proposed bill was supported by Consumers Union, the Environmental Working Group, and the Public Interest Research Group. *Id.*

113. *Id. See also Bisphenol A (BPA) in Packaging*, BARRY SANEL'S PACKAGING BLOG (May 19, 2008, 01:57 PM), http://barrysanel.blogspot.com/2008_05_01_archive.html (describing the Senate committee hearing and noting that Dr. Alderson was "defensive" in fielding questions from Sen. Kerry, who reportedly "tongue lash[ed]" the doctors who testified). For video of the hearing, see *Plastic Additives in Consumer Products*, HEARINGS—U.S. SENATE COMM. ON COMMERCE, SCI., & TRANSP., http://commerce.senate.gov/public/index.cfm?p=Hearings&ContentRecord_id=d8894142-44e0-4a06-999c-05811a11938c&ContentType_id=14f995b9-dfa5-407a-9d35-56cc7152a7ed&Group_id=b06c39af-e033-4cba-9221-de668ca1978a&MonthDisplay=5&YearDisplay=2008 (last visited Feb. 16, 2011).

E. Response of State and Local Governments

Filling the federal vacuum, state and local governments are catering to public demand by restricting BPA use within their jurisdictions. As of Spring 2010, the EWG reports that bills regulating the use of BPA are in play in ten states and the District of Columbia.[114] Recently, the Maryland governor signed a bill banning BPA in bottles and cups for children;[115] this ban will take effect in 2012.[116] Similar laws were passed in Connecticut and Minnesota in 2009.[117]

F. Industry Response to Popular Pressure

In the face of Congressional pressure, and a flood of jurisdictions banning BPA in various forms, the chemical industry has begun to react. In 2008, Nalgene Outdoor Products announced that all of its plastic bottles would soon be BPA-free,[118] and in September 2009, the water bottle manufacturer Sigg followed suit.[119] In July 2009, Similac announced that 91% of the formula it sold would be BPA-free.[120] Although plastic BPA-free baby bottles are currently available, the EWG still recommends that mothers use glass bottles.[121] Eden Foods has been using BPA-free cans for its canned beans since 1999; instead of BPA-containing epoxy linings, the cans are lined with a vegetable resin.[122] However, the vast majority of food cans on the market are still lined with epoxy resin that contains BPA.[123]

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114. Houlihan et al., *supra* note 21.

115. Press Release, Envtl.Working Grp., BPA Ban Now Law in Maryland (Apr. 14, 2010), *available at* http://www.ewg.org/BPA_Ban_Now_Law_In_Maryland. The provisions went into effect on July 1, 2010 and are codified in the Heath-General Article of the Maryland Code. MD. CODE ANN., HEALTH-GEN. § 24-304; S.B. 213 (LexisNexis 2011), 2010 Leg., 427th Sess. (Md. 2010).

116. § 24-304(b); *Maryland: Chemical Banned from Cups*, N.Y. TIMES, Feb. 26, 2010, at A13.

117. *Maryland: Chemical Banned from Cups*, *supra* note 116. *Compare* § 24-304, *with* CONN. GEN. STAT. § 21a-12c (2010), *and* MINN. STAT. §§ 325F.172-325F.173 (2010).

118. Press Release, Nalgene, Nalgene to Phase Out Production of Consumer Bottles Containing BPA (Apr. 18, 2008), *available at* http://www.nalgene-outdoor.com/PDFs/08NAL_BPA_PR.pdf.

119. Steve Wasik, *SIGG CEO: I'm Sorry*, HUFFINGTON POST (Sept. 7, 2009, 02:38 PM), http://www.huffingtonpost.com/steve-wasik/sigg-ceo-im-sorry_b_278291.html.

120. Press Release, Abbott Laboratories, *supra* note 21.

121. *EWG's Guide to Infant Formula and Baby Bottles*, ENVTL. WORKING GRP., http://www.ewg.org/book/export/html/25570 (last visited Feb. 17, 2011).

122. *Bisphenol-A (BPA) Free Can Lining*, EDEN FOODS, INC., http://www.edenfoods.com/articles/view.php?articles_id=178 (last visited Feb. 7, 2011). Eden claims the BPA free cans cost them 14% more than cans lined with BPA-containing epoxy. *Id.*

123. Lyndsey Layton, *Replacing BPA in Cans Gives Foodmakers Fits; FDA Safety Concerns Prompt Scramble to Remove the Chemical*, WASH. POST, Feb. 23, 2010, at A1.

G. *FDA Signals a Change in Position*

In June 2009, the FDA announced that it was reconsidering its prior opinion that BPA is safe as currently used in food contact applications.[124] In January 2010, FDA announced that it had "some concern"[125] about BPA due to "subtle effects of low doses of BPA in laboratory animals."[126] The agency now recommends that parents use powdered formula instead of liquid and only gently heat plastic baby bottles.[127] Yet, the agency has taken no regulatory action, and in January 2010, stated uncertainty about its legal authority to regulate BPA absent legislative action.[128] Part IV of this paper will begin with an analysis of whether the FDA possesses that authority.

IV. FDA'S REGULATION OF BPA

A. May the FDA Regulate BPA as a Food Additive?

The FDA does not currently regulate BPA as a food additive,[129] although at least one polymer approved as a food additive does contain BPA as an ingredient.[130] Declaring BPA to be a food additive would provide the FDA with the regulatory power to dictate the conditions upon which it may be used in food contact applications.[131] However, the FDA expressed uncertainty about its authority to declare BPA to be a food additive within the statutory definition.[132] This section of the paper analyzes whether the FDA has such authority, and concludes that it does.

To declare BPA to be a food additive under the Federal Food, Drug, and Cosmetic (FD&C) Act, the FDA would need to make two factual

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124. Jared A. Favole, *FDA to Revisit Decision on Safety of BPA*, WALL ST. J., June 3, 2009, http://online.wsj.com/article/SB124405286248681991.html.

125. Grady, *supra* note 18.

126. *Bisphenol A (BPA) Information for Parents*, U.S. DEP'T OF HEALTH & HUMAN SERVS., http://www.hhs.gov/safety/bpa/ (last visited Feb. 17, 2011).

127. *Id.* The FDA also claims that at least 90% of the baby bottles on the market in the United States are BPA-free. *Id.*

128. Alyah Kahn, *FDA Seeking Bolstered Regulatory Authority for BPA*, FDA WK., Jan. 22, 2010.

129. *See* DRAFT ASSESSMENT, *supra* note 17, at 3 (explaining that BPA is not considered a food additive but discussing the ways in which BPA nevertheless affects food additives).

130. *See* 21 C.F.R. § 177.1555 (2010) ("Polyarylate resins . . . may be safely used as articles or components of articles intended for use in contact with food . . . .") Polyarylate resins contain BPA as an ingredient. *Id.* § 177.1555(a).

131. *See* 21 C.F.R. § 170.15(a) (2010) (providing that once a substance is determined to be a food additive the Commissioner has authority to initiate the adoption of regulations for safe use).

132. Khan, *supra* note 128.

findings.[133] First, it would have to find that, when used as intended in food contact applications (such as packaging), BPA "may reasonably be expected to result . . . in its becoming a component or otherwise affecting the characteristics of any food."[134] The question is thus, how much of a packaging substance must migrate to food before it becomes a "component" of the food?

This issue was addressed by the D.C. Circuit in *Monsanto Co. v. Kennedy*.[135] In that case, the FDA, acting on concerns about the deleterious effects of the chemical acrylonitrile, declared a polymer in which acrylonitrile was an ingredient to be a food additive.[136] This finding was challenged by the manufacturer of the polymer.[137] The FDA did not have data that showed that acrylonitrile actually migrated from the polymer into food. Instead, the FDA presented evidence that other polymers leached their component monomers into food under similar conditions of intended use.[138] The court found that the FDA had failed to satisfy the "component" requirement, because it had no *actual* evidence of migration.[139] The court stated that once actual migration has been demonstrated, the statute affords the FDA Commissioner "latitude" in finding whether or not the migration is significant enough to meet the component requirement.[140] Although the court explicitly stated that this requirement does not require that the magnitude of migration to be "toxicologically significant,"[141] one can

133. *See* 21 U.S.C. § 321(s) (2006) (defining "food additive" generally as an additive that (1) becomes a component or otherwise affects food, and (2) has not had its safety scientifically demonstrated). Specifically, "food additive" is defined in the FD&C Act as "any substance the intended use of which results or may reasonably be expected to result, directly or indirectly, in its becoming a component or otherwise affecting the characteristics of any food (including any substance intended for use in producing, manufacturing, packing, processing, preparing, treating, packaging, transporting, or holding food; and including any source of radiation intended for any such use), if such substance is not generally recognized, among experts qualified by scientific training and experience to evaluate its safety, as having been adequately shown through scientific procedures . . . to be safe under the conditions of its intended use . . . ." *Id.*

134. *Id.*

135. 613 F.2d 947 (D.C. Cir. 1979).

136. *Id.* at 951 .

137. *See id.* at 950–52 (stating that, after the FDA Commissioner found that the polymer was a food additive, Monsanto introduced evidence that the polymer did not in fact migrate into food).

138. *Id.* at 951–52. The Court noted that acrylonitrile drew FDA's attention when it found evidence of possible "significant" migration in a container made of a "somewhat different substance." *Id.* at 951. From this evidence, the Commissioner concluded that the polymer was a "food additive." *Id.* Additionally, the Court noted that, at the administrative hearing, Monsanto introduced evidence that acrylonitrile in particular did not migrate into food. *Id.* at 952.

139. *Id.* at 952–53 (remanding the agency decision for further consideration because "the Commissioner had made a projection of migration from 3.3 ppm RAN containers without the support of any actual data showing that migration had occurred from such containers.").

140. *Id.* at 955.

141. *Id.*

hardly think of a more important criteria upon which the Commissioner should base his judgment.

It is likely that the FDA could successfully defend a finding that at least some BPA-containing cans and bottles meet the "component" requirement. The scientific literature abounds with studies that show that BPA migrates from both epoxy resins and polycarbonate plastics into food in detectible quantities.[142] Thus, the FDA has discretion to determine if the migration is significant enough to either render BPA a "component" of the food, or to "affect[] the characteristics" of the food. A court would likely find it proper for the Commissioner to weigh the amount of BPA likely to have a physiological effect against the amount likely to leach from a container.

Studies have shown that as little as 2 μg/kg/d BPA can have significant physiological effects in rodents.[143] Other studies have shown that BPA can leach out of baby bottles at a concentration of up to 8.4 μg/L.[144] Following the rule of thumb that a child should be offered 2.5 fluid ounces of formula per pound of body weight per day,[145] a 15 pound child may drink around 37.5 fluid ounces, or 1.1 liters, of formula per day. Consequently, such a child might consume approximately 9.2 μg of BPA per day, or, factoring in body weight, 1.35 μg/kg/d of BPA *from the bottle alone*. Factoring in the amount of BPA that may be in the formula may push the baby's intake above the 2 μg/kg/d threshold. Even if intake does not reach this threshold level, the FDA would be justified in finding that BPA may be expected to migrate in significant enough quantities to render it a "component" of food, given the much larger margins of safety agencies usually feel comfortable working with.[146]

The food industry, if it were to challenge the finding, would likely point to other studies that showed that BPA does not migrate to food in detectible quantities, and that it does not have significant physiological effects at quantities much greater than 2 μg/kg/d.[147] If the FDA uses only

142. *See* NTP REPORT, *supra* note 15, at 7–13 (describing numerous studies which found that BPA migrates from epoxy resins and polycarbonate plastic into food).

143. Nagel et al., *supra* note 61, at 70, 74.

144. *See* NTP REPORT, *supra* note 15, at 9 (detailing results from a Norwegian study performed on twelve infant bottles).

145. BabyCenter Medical Advisory Board, *How to Tell How Much Formula Your Baby Needs*, BABYCENTER, http://www.babycenter.com/0_how-to-tell-how-much-formula-your-baby-needs_9136.bc (last updated Sept. 2008).

146. *See* 21 U.S.C. § 321(s) (2006) (indicating that a food "component" can include, *inter alia*, "substances intended for use in . . . packaging, . . . or holding food" that "affect the characteristics of any food").

147. Ema et al., *supra* note 70, at 505; Tyl et al., *Three-Generation*, *supra* note 70, at 121; Tyl et al., *Two-Generation*, *supra* note 70, at 362.

the studies cited above, the industry likely will accuse the FDA of performing the same data "cherry picking" that environmental groups accused the FDA of using in 2008. However, the gap between demonstrated migration and demonstrated physiological effects is not likely large enough that a court would find the FDA's conclusion that BPA migration "affect[s] the characteristics" of food arbitrary and capricious.

The second finding that the FDA would be required to make in order to regulate BPA as a food additive would be that the substance "is not generally recognized, among experts qualified by scientific training and experience to evaluate its safety, as having been adequately shown through scientific procedures . . . to be safe."[148] Thus, this requirement is met unless the product has affirmatively been shown to be safe. The FDA will likely be able to successfully meet this safety-related requirement as well. Although the food industry has commissioned several studies that did not find significant physiological impacts in rodents exposed to low doses of BPA,[149] a large group of academic scientists found these studies to be flawed.[150] In addition, academic research has demonstrated the physiological effects BPA has at low doses in rodents,[151] and medical research has correlated elevated urinary BPA levels with heart disease and diabetes.[152] Finally, a group of scientists (many of the same who criticized the industry research) have concluded that BPA is "a chemical of high concern."[153] A court would thus be unlikely to find that the FDA was acting contrary to Congress' intent by asserting that BPA is not "generally recognized, among experts . . . to be safe."[154]

If the FDA declares BPA to be a food additive, it would clear the way for the agency to issue regulations prescribing the conditions under which it may be used.[155] For example, it could forbid the use of BPA in baby bottles

148. 21 U.S.C. § 321(s).

149. Ema et al., *supra* note 70, at 153; Tyl et al., *Three-Generation*, *supra* note 70, at 121; Tyl et al., *Two-Generation*, *supra* note 70, at 362.

150. *See* EWG Letter, *supra* note 73, at 5 (discussing the "methodological flaws" of several studies).

151. *See supra* notes 59–67 and accompanying text (detailing various studies that found that low doses of BPA have physiological effects on rodents).

152. *See* Lang et al., *supra* note 15, at 1304–05 (finding higher concentrations of BPA in the urine of humans who reported that they suffered cardiovascular disease and diabetes, suggesting the possibility that BPA might have a causal relationship with these diseases).

153. Frederick S. vom Saal et al., *Chapel Hill Bisphenol A Expert Panel Consensus Statement: Integration of Mechanisms, Effects in Animals and Potential to Impact Human Health at Current Levels of Exposure*, 24 REPRODUCTIVE TOXICOLOGY 131, 131, 136 (2007).

154. 21 U.S.C. § 321(s) (2006).

155. *Id.* § 348(d) ("The Secretary may at any time, upon his own initiative, propose the issuance of a regulation prescribing, with respect to any particular use of a food additive, the conditions under which such additive may be safely used, and the reasons therefore. After the

but allow its use in aluminum cans. If the FDA did not issue such a regulation, a food manufacturer would have two choices. First, it could notify the FDA pursuant to 21 U.S.C. § 348(a)(3)(B) that it would continue to use BPA because it believed the chemical to be safe.[156] The FDA would then have 120 days to object; otherwise, the manufacturer could resume using BPA.[157] Alternatively, the manufacturer could file a proposed method of use pursuant to 21 U.S.C. § 348(b). The FDA would then have 180 days to establish a regulation or deny the request.[158]

In summary, enough scientific evidence of BPA's danger exists that the FDA has the authority to regulate its use as a food additive. The agency could use that authority to do anything from ban the use of the chemical in the manufacture of one or two items to banning the chemical from use in all food contact applications in which it can be shown that BPA migrates to food. The next section of the paper will address whether, and to what extent, the FDA should exercise that authority.

B. Should the FDA Regulate Bisphenol A as a Food Additive?

When determining the extent to which to regulate BPA, the FDA should compare the costs and benefits of using the chemical in each food contact application. This will likely require investigation into chemical alternatives to BPA. The availability, safety, and cost of the alternatives should all be considered. The food industry's public stance is that replacing BPA for use in hard plastics will not be difficult, but that replacing BPA in resins lining aluminum cans will be.[159] The manufacturers of plastic bottles have already found polypropylene to be a reasonable alternative to BPA-containing polycarbonates.[160] However, thus far, alternatives to BPA in cans have not matched up to BPA's ability to cost-effectively keep food fresh for long periods of time without interfering with taste.[161]

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thirtieth day following publication of such a proposal, the Secretary may by order establish a regulation based upon the proposal."). Any adversely affected party could challenge the regulation by demanding a public hearing, then appealing to the D.C. Circuit or the circuit in which the person lives or has his principal place of business. §§ 348(f)–(g).

156. § 348(a)(3)(B); § 348(h)(1). Such course of actoin would only be available if BPA properly falls under the definition of a "food contact substance" included in 21 C.F.R. § 170.3(e)(3). § 348(a)(3)(B). The regulation provides that "any substance that is intended for use as a component of materials used in manufacturing, packing, packaging, transporting, or holding food if such use is not intended to have any technical effect in such food." 21 C.F.R. § 170.3(e)(3) (2009).

157. 21 U.S.C. § 348(h)(2)(A).

158. *Id.* § 348(b)–(c)

159. *See* Layton, *supra* note 123 (stating that makers of plastic bottles found an easy substitute for BPA, whereas, canned-food makers have not found a viable alternative).

160. *Id.*

161. *Id.*

Banning BPA completely, while perhaps the best option from a public-health perspective, will likely cause an uproar among the public, who will worry about the economy, as well as their access to canned foods.[162] Therefore, such a broad ban is unlikely to happen. Instead, the FDA should act now to protect the population group most vulnerable to BPA: young children. Research has demonstrated that this group is the most sensitive to endocrine disruptors and may suffer a double dose of BPA if their mothers use both canned formula and a BPA-containing bottle.[163] This regulation would also cause few problems, because BPA-free baby bottles are already on the market and powdered infant formula is easily packaged in BPA-free containers.[164] Thus, the FDA should ban the use of BPA in products designed for young children.

Another benefit of this type of regulation is that it would keep the chemical in the public eye. This would have two positive effects. First, it would make people more aware of what foods contain BPA, perhaps encouraging them to moderate their consumption of canned goods. Second, it would maintain industry's impetus to continue searching for alternatives to BPA-containing liners in aluminum cans. Ultimately, what will solve the BPA problem is the development of a safe, effective, and cost-efficient alternative. Public awareness of BPA and its potentially harmful effects will encourage the food industry to keep searching for that alternative.

CONCLUSION

BPA's potential for low-dose toxicity in humans and its abundant use in food contact applications are a distressing combination. Although the public's increasing awareness of BPA's dangers have caused industry to remove it from most food containers designed for use by children, it is still contained in nearly every can of food we buy.[165] Right now, the food industry claims it is researching BPA-free liners to aluminum cans.[166] In order to maintain the industry's impetus to search for alternatives, as well as to make the public aware of a potential health danger, the FDA should work to keep the chemical in the news. This can be accomplished, in part, by prohibiting BPA's use in the packaging of baby formula and in all containers designed to hold children's food.

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162. *See id.* (noting that BPA-free linings for canned foods are more expensive and that one company has passed the increased manufacturing costs onto customers).

163. Guo et al., *supra* note 12, at 117–18, 120.

164. *Bisphenol A (BPA) Information for Parents*, *supra* note 126.

165. Layton, *supra* note 123.

166. *See id.* (stating that industry is having trouble finding a BPA-free alternative for canned products).

The FDA's first step should be declaring BPA to be a "food additive" under the FD&C Act. In justifying this finding, the FDA should embrace many of the academic studies it failed to consider in its 2008 Draft Assessment. Further, the FDA should take steps to ensure that the GLP standards, which were designed to fight sham science, do not elevate industry-funded science to the only game in town. A policy should be instituted whereby every potentially relevant study is evaluated on a case-by-case basis by the quality of the researcher, the quality of the experimental design, and potential conflicts of interest.