An Update on the Recently Published Peer-Reviewed Scientific Literature on Bisphenol A (BPA)

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In 2010, as part of its concurrence with the rule proposed by Maine Department of Environmental Protection to designate bisphenol A (BPA) as a Priority Chemical and to regulate BPA in certain children's products, the Maine Center for Disease Control and Prevention reviewed the literature concerning the health effects of exposure to BPA, as well as evidence for human exposure. This document updates the literature since the 2010 review, focusing on evidence of human exposure to BPA from products and evidence for adverse effects in the human population as a result of environmental exposure to BPA. Results from occupational studies are included in support of environmental studies.

Recent studies on human exposure to BPA

Prior to 2010, many studies of consumer products identified sources of human BPA exposures including canned foods, plastics, dental sealants, and food contact papers (Vandenberg et al., 2007). Since then, additional studies have performed detailed analyses of canned foods (Cao et al., 2010; Schecter et al., 2010), receipts and other thermal papers (Liao and Kannan, 2011b; Liao et al., 2012), money (Liao and Kannan, 2011a), air and dust (Fu and Kawamura, 2010; Loganathan and Kannan, 2011), among others. Importantly, all sources of human exposure are not yet identified, and controversy remains over which sources are the highest contributors to daily BPA intake.

There is evidence that food packaging is a major source of BPA exposure (Muncke, 2011). In a study of pregnant women, consumption of canned vegetables but not fresh fruits or vegetables or canned fruit was associated with higher urinary BPA levels (Braun et al., 2011a). These findings are consistent with estimates that canned vegetables contribute 10-40% of daily BPA intake (von Goetz et al., 2010). A study representative of the general US population found that increased consumption of soda, school lunches, and meals not prepared at home was predictive of higher urinary levels of BPA (Lakind and Naiman, 2011). These variables were examined specifically because it was hypothesized that food products from outside the home were more likely to be packaged in contact with BPA.

Studies performed at the US CDC have shown age-related associations with BPA exposures, where younger individuals typically have higher levels of BPA in their bodies relative to older individuals (Calafat et al., 2008). Similar results have also been reported in population-based studies from Canada (Bushnik et al., 2010). Perhaps most

concerning is a recent comparison of the effects of age and country of residence on BPA exposure levels; BPA concentrations were higher in every age group examined in US residents compared to Canadian, Chinese and German residents (Vandenberg, 2011). These results may be indicative of divergent exposure sources, as well as differences in consumer habits, among Americans compared to other populations.

Two studies have examined the effects of manipulating exposure to packaged food on urinary BPA concentrations. In a randomized cross-over study, adults ate fresh or canned soup for five days, after which treatment assignments were reversed (Carwile et al., 2011). Consumption of canned soup increased urinary BPA levels by over 1000% regardless of whether canned soup was eaten first or second in the treatment assignment. In the second study examining the effects of dietary interventions, subjects switched from their usual diet to one comprised of fresh foods that were not canned or packaged in plastic; the participants then returned to their usual diet (Rudel et al., 2011). BPA levels decreased by 66% with three days of eating fresh food, and rebounded 202% with the resumption of the subjects' typical diet.

Because BPA is found in high quantities in thermal papers, and is transferred from these papers to skin (Biedermann et al., 2010), non-oral exposures cannot be ignored. Cashiers who handled BPA-containing receipts had higher levels of urinary BPA than individuals who did not handle BPA-containing paper (Braun et al., 2011a). In an experimental study, it was also determined that BPA passed freely across human skin explants and entered the simulated blood circulation (Zalko et al., 2011).

Recent studies on effects of BPA on infants and children

Some of the most concerning and compelling evidence linking BPA exposure to effects in humans concerns the effects of developmental exposures on behavior. A 2009 study reported that prenatal exposure was associated with an increase in hyperactivity and aggression in two-year-old girls (Braun et al., 2009). In a follow-up assessment of this cohort of children, average maternal BPA levels were associated with an increase in anxiety and hyperactivity, and poorer emotional control and inhibition in three-year-old girls (Braun et al., 2011b). These results suggest that the behavior of BPA-exposed girls was masculinized. This is perhaps most revealing when considered in the context of animal studies, which have indicated that BPA can masculinize behaviors of female rodents, and may feminize the behaviors of male rodents (Adewale et al., 2011; Patisaul et al., 2006; Patisaul et al., 2009; Rubin et al., 2006).

Other studies link maternal BPA exposures to an increase in premature births, as well as small for gestational age babies (Cantonwine et al., 2010; Chou et al., 2011; Miao et al., 2011b). Maternal or paternal exposure to BPA during pregnancy was also associated with decreased anogenital distance in sons (Miao et al., 2011a), suggesting feminization of male offspring. Maternal BPA levels also influenced newborn hormone levels that are associated with lipid metabolism (Chou et al., 2011). These results are consistent with a study in mice documenting disruption of glucose homeostasis in mothers and male offspring as a function of increased BPA exposure (Alonso-

Magdalena et al., 2010b). Offspring may therefore be at risk for diabetes or obesity later in life.

Finally, BPA exposure may also influence the developing immune system. Early prenatal exposure, but not later prenatal exposure or neonatal exposure, was associated with an increase in child wheeze at six months of age (Spanier et al., 2012). Additionally, BPA levels were associated with antibody titers to a common pathogen (cytomegalovirus), although the relationship was reversed for individuals younger vs. older than 18 years old (Clayton et al., 2011).

Recent studies on reproductive effects of BPA (in human adults)

Increased BPA levels are associated with decreased sperm quality following environmental (Meeker et al., 2010b) and occupational (Li et al., 2011) exposure. Higher BPA levels were also associated with poorer sexual function in occupationally or environmentally exposed men, including decreased sexual desire and decreased erection and orgasmic function (Li et al., 2010a; Li et al., 2010b). Several studies indicate that environmental exposures to BPA (i.e. those experienced by typical adults) affect testosterone levels in men (Galloway et al., 2010; Meeker, 2010; Mendiola et al., 2010), and are associated with changes in estrogenic gene expression in adult males (Melzer et al., 2011). In women receiving *in vitro* fertilization, higher BPA concentrations were associated with poorer oocyte quality, decreased estradiol levels, and decreased implantation success (Bloom et al., 2011; Ehrlich et al., 2012; Fujimoto et al., 2011; Mok-Lin et al., 2010).

Recent studies on non-reproductive health effects of BPA in adults

In 2008, the first study showing an association between urinary BPA levels and heart disease was published; individuals with higher BPA exposures were more likely to report cardiovascular diseases (Lang et al., 2008). In 2010, another cross-sectional study representative of the US population found that higher BPA levels were associated with an increased incidence of coronary heart disease (Melzer et al., 2010). This study was followed by a longitudinal study, in which BPA exposures were measured in adults free of coronary heart disease, and these individuals were then followed for 10.8 years (Melzer et al., 2012). Individuals with higher urinary BPA levels at time zero were more likely to develop coronary heart disease at the end of the study compared to individuals with low urinary BPA concentrations at time zero. This study thus addresses the issue of causation, and suggests that BPA exposures could cause heart disease (and refutes the suggestion that heart disease causes increases in BPA exposure.)

Similar to what was reported in 2008 (Lang et al., 2008), studies of additional adult populations indicate that increased urinary BPA levels are associated with obesity in adults (Carwile and Michels, 2011). Additional longitudinal studies are needed to address the possibility of reverse causality in these cross-sectional studies (i.e. obese individuals have higher levels of BPA as a result of dietary or other lifestyle variables).

However, BPA activates the human pregnane X receptor (Sui et al., 2012), which is involved in lipid homeostasis in addition to steroid and xenobiotic chemical metabolism. BPA may affect other endocrine parameters in addition to reproductive hormones and possibly metabolic homeostatis. Specifically, higher BPA levels were associated with decreased thyroid hormone levels in adults (Meeker et al., 2010a; Meeker and Ferguson, 2011).

Human studies are supported by hundreds of animal studies

This document has focused on the studies that are directly relevant to human exposures, sources of exposure, and known human health concerns. However, it should be noted that in the last few years there have been rigorous and compelling studies that have described the effects of BPA on various endpoints in animals. These studies include several that report effects on the mammary gland (Betancourt et al., 2010a; Betancourt et al., 2010b; Jones et al., 2010; Lamartiniere et al., 2011; Tharp et al., 2012; Weber Lozada and Keri, 2011), fertility and development of the reproductive organs (Aldad et al., 2011; Arase et al., 2011; Cabaton et al., 2011; Lawson et al., 2011; Signorile et al., 2010), metabolic endpoints (Alonso-Magdalena et al., 2010a; Alonso-Magdalena et al., 2010b), and brain development and behavioral endpoints (Bai et al., 2011; Goncalves et al., 2010; Hajszan and Leranth, 2010; Monje et al., 2010; Nakamura et al., 2010; Poimenova et al., 2010; Tian et al., 2010), among others.

There truly is no question that BPA produces adverse effects in rodents, documented in hundreds of studies [see reviews by (Richter et al., 2007; Vandenberg et al., 2009; vom Saal and Hughes, 2005)]. There is considerable evidence that BPA interferes with male and female reproduction, brain development, the adult brain, metabolic processes, and development of the mammary gland (Vandenberg et al., 2012). Effects in numerous studies were observed at blood levels consistent with levels in humans in the general population (Vandenberg et al., 2007; vom Saal et al., 2007).

Nonetheless, it has been argued that the rodent studies are not relevant to human exposure to BPA because of differences in metabolism in humans compared to rats and mice (US FDA, 2010), which are purported to result in higher circulating blood BPA levels in rodents for an equivalent oral intake of BPA in humans. In order for BPA to be eliminated from blood and excreted from the body, it must be metabolized to a conjugated form, which allows the BPA to be excreted. It is the free (unconjugated) form of BPA that is estrogenically active, so differences in metabolism could conceivably result in the rodent being a poor model for humans.

However, the argument that the rodent studies are not relevant to humans fails for a number of reasons.

1) Free (unconjugated) BPA was detected in the vast majority of studies of the general population in which it was measured [for reviews of these studies, and further discussion of this controversy, see (Vandenberg et al., 2010a; Vandenberg et al., 2010b)]. Free BPA has been detected in placenta, amniotic

fluid, breast milk and follicular fluid (the fluid surrounding the egg). The finding of free BPA in multiple population studies stands in contradiction to a laboratory study in which 20 adults who were fed diets purported to be high in BPA and monitored for 24 hours, in which BPA was undetectable in most blood **and urine** samples (Teeguarden et al., 2011). This study was recently critiqued (Vom Saal et al., 2012), with the authors identifying serious flaws in the Teeguarden study, including the fact that the amount of ingested BPA was not measured and likely to be low based on the diet of the volunteers. vom Saal *et al*. also point out that in the real world, there are undoubtedly other sources of exposure to BPA in addition to cans and polycarbonate containers that are unrecognized and unreported.

2) A recent study reported that adult female mice, monkeys, and humans metabolized BPA at "virtually identical rates" (Taylor et al., 2011). Further, this study demonstrated that the blood concentrations of conjugated BPA in these three species would be the same following the same oral intake of BPA. These data suggest that regardless of any differences in metabolism and excretion, blood levels in humans would be the same as those in rodents following an equivalent oral exposure.

3) Numerous tissues in the body are capable of deconjugating BPA: in other words, the conjugated (inactive) form can pass from blood into a specific organ and be reactivated to free BPA (Ginsberg and Rice, 2009; Nishikawa et al., 2010). Enzymes that perform this task are present in numerous tissues in the body, including placenta and fetal liver. It is also noteworthy that in a study of 1469 adults representative of the U.S. population (Stahlhut et al., 2009), BPA levels did not decrease rapidly following fasting, suggesting alternate (non-oral) sources of exposure as well as conjugation-deconjugation cycling. In any event, the possibility for deconjugation in specific tissues, including fetal tissues, suggests that the circulating concentration of free BPA in blood may underestimate exposure and therefore the potential for adverse effects.

4) Finally, there is increasing evidence in humans for adverse effects associated with the blood concentration of BPA. As delineated in the "Rationale for Concurrence by MeCDC on the Designation of Bisphenol A as a Priority Chemical by MeDEP", and as described in some detail above, BPA exposure in humans is associated with effects on male and female reproduction, metabolic processes, heart disease, immune function, and brain development and children's behavior. These effects are consistent with effects observed in rodent studies following BPA administration. This congruence of findings strongly suggests that the plethora of studies in rodents documenting BPA toxicity predicts similar effects in the human population environmentally exposed to BPA, notwithstanding any differences in metabolism that may be present between humans and rodents.

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Spanier, A.J., Kahn, R.S., Kunselman, A.R., Hornung, R., Xu, Y., Calafat, A.M. and Lanphear, B.P. (2012) Prenatal Exposure to Bisphenol A and Child Wheeze from Birth to Three Years. Environ Health Perspect.
Stahlhut, R.W., Welshons, W.V. and Swan, S.H. (2009) Bisphenol A data in NHANES suggest longer than expected half-life, substantial non-food exposure, or both. Environ Health Perspect 117, 784-9.
Sui, Y., Ai, N., Park, S.H., Rios-Pilier, J., Perkins, J.T., Welsh, W.J. and Zhou, C. (2012) Bisphenol A and its analogues activate human pregnane X receptor. Environ Health Perspect 120, 399-405.
Taylor, J.A., vom Saal, F.S., Welshons, W.V., Drury, B., Rottinghaus, G., Hunt, P.A. and Vandevoort, C.A. (2011) Similarity of Bisphenol A Pharmacokinetics in Rhesus Monkeys and Mice: Relevance for Human Exposure. Environ Health Perspect 119, 422-30.
Teeguarden, J.G., Calafat, A.M., Ye, X., Doerge, D.R., Churchwell, M.I., Gunawan, R. and Graham, M.K. (2011) Twenty-four hour human urine and serum profiles of bisphenol a during high-dietary exposure. Toxicol Sci 123, 48-57.
Tharp, A.P., Maffini, M.V., Hunt, P.A., Vandevoort, C.A., Sonnenschein, C. and Soto, A.M. (2012) Bisphenol A alters the development of the rhesus monkey mammary gland. Proc Natl Acad Sci U S A 109, 8190-5.
Tian, Y.H., Baek, J.H., Lee, S.Y. and Jang, C.G. (2010) Prenatal and postnatal exposure to bisphenol a induces anxiolytic behaviors and cognitive deficits in mice. Synapse 64, 432-9.
US FDA (2010) Update on bisphenol A (BPA) for use in food: January 2010. In.
Vandenberg, L.N. (2011) Exposure to bisphenol A in Canada: invoking the precautionary principle. CMAJ online Feb 22, doi:10.1503/cmaj.101408.
Vandenberg, L.N., Chahoud, I., Heindel, J.J., Padmanabhan, V., Paumgartten, F.J.R. and Schoenfelder, G. (2010a) Urine, serum and tissue biomonitoring studies indicate widespread exposure to bisphenol A. Environ Health Perspect 118, 1055-70.
Vandenberg, L.N., Chahoud, I., Padmanabhan, V., Paumgartten, F.J.R. and Schoenfelder, G. (2010b) Biomonitoring studies should be used by regulatory agencies to assess human exposure levels and safety of Bisphenol A. Environ Health Perspect 118, 1051-4.
Vandenberg, L.N., Colborn, T., Hayes, T.B., Heindel, J.J., Jacobs, D.R., Lee, D.-H., Shioda, T., Soto, A.M., Vom Saal, F.S., Welshons, W.V., Zoeller, R.T. and Myers, J.P. (2012) Hormones and endocrine disrupting chemicals: low dose effects and non-monotonic dose responses. Endocrine Reviews online 2012 Mar 14.
Vandenberg, L.N., Hauser, R., Marcus, M., Olea, N. and Welshons, W.V. (2007) Human exposure to bisphenol A (BPA). Reprod Toxicol 24, 139-77.
Vandenberg, L.N., Maffini, M.V., Sonnenschein, C., Rubin, B.S. and Soto, A.M. (2009) Bisphenol-A and the great divide: a review of controversies in the field of endocrine disruption. Endocrine Reviews 30, 75-95.
vom Saal, F.S., Akingbemi, B.T., Belcher, S.M., Birnbaum, L.S., Crain, D.A., Eriksen, M., Farabollini, F., Guillette, L.J., Jr., Hauser, R., Heindel, J.J., Ho, S.M., Hunt, P.A., Iguchi, T., Jobling, S., Kanno,

J., Keri, R.A., Knudsen, K.E., Laufer, H., LeBlanc, G.A., Marcus, M., McLachlan, J.A., Myers, J.P., Nadal, A., Newbold, R.R., Olea, N., Prins, G.S., Richter, C.A., Rubin, B.S., Sonnenschein, C., Soto, A.M., Talsness, C.E., Vandenbergh, J.G., Vandenberg, L.N., Walser-Kuntz, D.R., Watson, C.S., Welshons, W.V., Wetherill, Y. and Zoeller, R.T. (2007) 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. Reprod Toxicol 24, 131-8.

vom Saal, F.S. and Hughes, C. (2005) An extensive new literature concerning low-dose effects of bisphenol A shows the need for a new risk assessment. Environ Health Perspect 113, 926-933.

Vom Saal, F.S., Prins, G.S. and Welshons, W.V. (2012) Report of very low real-world exposure to bisphenol A is unwarranted based on a lack of data and flawed assumptions. Toxicol Sci 125, 318-20; author reply 321-5.

von Goetz, N., Wormuth, M., Scheringer, M. and Hungerbuhler, K. (2010) Bisphenol a: how the most relevant exposure sources contribute to total consumer exposure. Risk Anal 30, 473-87.

Weber Lozada, K. and Keri, R.A. (2011) Bisphenol A Increases Mammary Cancer Risk in Two Distinct Mouse Models of Breast Cancer. Biol Reprod.

Zalko, D., Jacques, C., Duplan, H., Bruel, S. and Perdu, E. (2011) Viable skin efficiently absorbs and metabolizes bisphenol A. Chemosphere 82, 424-30.

Curriculum Vitae

Laura N. Vandenberg
Biology Department
& Center for Regenerative and Developmental Biology
Tufts University
200 Boston Ave Suite 4600
Medford, MA 02155
Telephone (617) 627-4094
Email: laura.vandenberg@tufts.edu

EDUCATION & PROFESSIONAL EXPERIENCE

Tufts University, Department of Biology and Center for Regenerative & Developmental Biology
Postdoctoral Fellow, 2008 – present
Mentor: Dr. Michael Levin

Harvard University School of Dental Medicine
Research Associate in Developmental Biology, 2008

The Forsyth Institute Center for Regenerative & Developmental Biology
Postdoctoral Fellow, 2007 – 2008
Mentor: Dr. Michael Levin

Tufts University School of Medicine, Sackler School of Graduate Biomedical Sciences,
PhD, Cell, Molecular & Developmental Biology, 2007
Mentor: Dr. Ana Soto

Cornell University, Ithaca NY
BS, Biology, Concentration in Genetics & Developmental Biology, 2003
Undergraduate research mentor: Dr. Mariana Wolfner

PEER-REVIEWED PUBLICATIONS

Vandenberg LN, Colborn T, Hayes T, Heindel JJ, Jacobs D, Lee DH, Shioda T, Soto AM, vom Saal FS, Welshons WV, Zoeller RT, Myers JP. 2012 Hormones and endocrine disrupting chemicals: low dose effects and non-monotonic dose responses. *In press, Endocrine Reviews. doi:10.1210/er.2011-1050*

Vandenberg LN, Adams DS, Levin M. 2012 Normalized Shape and Location of Perturbed Craniofacial Structures in the *Xenopus* Tadpole Reveal an Innate Ability to Achieve Correct Morphology. *Developmental Dynamics. 241(5): 863-78.*

Vandenberg LN, Levin M. 2012. Planar cell polarity and apical-basal polarity are required for early orientation of the left-right axis and twin-twin instruction in Xenopus. *genesis, The Journal of Genetics & Development. 50(3): 219-34.*

Vandenberg LN. 2012. Laterality defects are influenced by timing of treatments and animal model. *Differentiation 83(1): 26-37.*

Vandenberg LN. 2011. Exposure to bisphenol A in Canada: invoking the precautionary principle. *Canadian Medical Association Journal (Epub Feb 22). doi: cmaj.101408v1-cmaj.101408.*

Vandenberg LN, Pennarola B, Levin M. 2011. Low frequency vibrations alter patterning of the left-right axis in developing Xenopus embryos. *PLoS ONE 6(8): e23306.*

Vandenberg LN, Morrie RD, Adams DS. 2011. V-ATPase-dependent ectodermal voltage and pH regionalization are required for craniofacial morphogenesis. *Developmental Dynamics 240: 1889-904.*

Vandenberg LN, Chahoud I, Padmanabhan V, Paumgartten FJR, Schoenfelder G. 2010. One database should be used by regulatory agencies to assess human exposure levels and safety of bisphenol A. *Environmental Health Perspectives 118: 1051-4.*

Vandenberg LN, Chahoud I, Heindel JJ, Padmanabhan V, Paumgartten F, Schoenfelder G. 2010. Urinary, circulating, and tissue biomonitoring studies indicate widespread exposure to bisphenol A. *Environmental Health Perspectives 118: 1055-70.*

vom Saal FS, Akingbemi BT, Belcher SM, Crain DA, Crews D, Guidice LC, Hunt PA, Leranth C, Myers JP, Nadal A, Olea N, Padmanabhan V, Rosenfeld CS, Schneyer A, Schoenfelder G, Sonnenschein S, Soto AM, Stahlhut RW, Swan SH, **Vandenberg LN**, Wang HS, Watson CS, Welshons WV, Zoeller RT. 2010. Flawed experimental design reveals the need for guidelines requiring appropriate positive controls in endocrine disruption research. *Toxicol Sci 115 (2): 612-3.*

Vandenberg LN, Levin M. 2010. Far from solved: a perspective on what we know about early mechanisms of left-right asymmetry. *Developmental Dynamics 239: 3131-46.*

Blackiston DJ, **Vandenberg LN**, Levin M. 2010. High throughput *Xenopus laevis* immunohistochemistry using agarose sections. *Cold Spring Harbor Protocols 2010(12): pdb.prot5532.*

Vandenberg LN, Levin M. 2010. Consistent left-right asymmetry cannot be established by late organizers in Xenopus unless the late organizer is a conjoined twin. *Development 137: 1095-1105.*

Vandenberg LN, Maffini MV, Sonnenschein C, Rubin BS, Soto AM. 2009. Bisphenol-A and the great divide: a review of controversies in the field of endocrine disruption. *Endocrine Reviews 30: 75-95.*

Myers JP, vom Saal FS, Akingbemi BT, Arizono K, Belcher S, Colborn T, Chahoud I, Crain DA, Farabollini F, Guillette LJ Jr., Hassold T, Ho S-M, Hunt PA, Iguchi T, Jobling S, Kanno J, Laufer H, Marcus M, McLachlan JA, Nadal A, Oehlmann J, Olea N, Palanza P, Parmigiani S, Rubin BS, Schoenfelder G, Sonnenschein C, Soto AM, Talsness CE, Taylor JA, **Vandenberg LN**, Vandenbergh JG, Vogel S, Watson CS, Welshons WV, Zoeller RT. 2009. Why public health agencies cannot depend upon 'Good Laboratory

Practices' as a criterion for selecting data: the case of bisphenol-A. *Environmental Health Perspectives 117: 309-15.*

Vandenberg LN, Levin M. 2009. Perspectives and open problems in the early phases of left-right patterning. *Seminars in Cell and Developmental Biology 20: 456-63.*

Myers JP, vom Saal FS, Akingbemi BT, Arizono K, Belcher S, Colborn T, Chahoud I, Crain DA , Farabollini F, Guillette LJ Jr, Hassold T, Ho SM, Hunt PA, Iguchi T, Jobling S, Kanno J, Laufer H, Marcus M, McLachlan JA, Nadal A, Oehlmann J, Olea N, Palanza P, Parmigiani S, Rubin BS, Schoenfelder G, Sonnenschein C, Soto AM, Talsness CE, Taylor JA, **Vandenberg LN**, Vandenbergh JG, Vogel S, Watson CS, Welshons WV, Zoeller RT. 2009. Re: Good laboratory practices and safety assessments. [Response to Becker et al]. *Environmental Health Perspectives 117: A482-3.*

Vandenberg LN, Maffini MV, Schaeberle CM, Ucci AA, Sonnenschein C, Rubin BS, Soto AM. 2008. Perinatal exposure to the xenoestrogen bisphenol-A induces mammary intraductal hyperplasias in adult CD-1 mice*. Reproductive Toxicology 26: 210-9.*

Soto AM, **Vandenberg LN**, Maffini MV, Sonnenschein C. 2008. Does breast cancer start in the womb? *Basic and Clinical Pharmacology & Toxicology*, 102: 125-33.

Vandenberg LN, Maffini MV, Wadia PR, Sonnenschein C, Rubin BS, Soto AM. 2007. Exposure to environmentally relevant doses of the xenoestrogen bisphenol-A alters development of the fetal mouse mammary gland. *Endocrinology 148: 116-27.*

Wadia PR, **Vandenberg LN**, Schaeberle CM, Rubin BS, Sonnenschein C, Soto AM. 2007. Perinatal bisphenol-A exposure increases estrogen sensitivity of the mammary gland in diverse mouse strains. *Environmental Health Perspectives 115: 592-8.*

Vandenberg LN, Hauser R, Marcus M, Olea N, Welshons WV. 2007. Human exposure to bisphenol A (BPA). *Reproductive Toxicology 24: 139-177.*

vom Saal FS, Akingbemi BT, Belcher SM, Birnbaum LS, Crain DA, Eriksen M, Farabollini F, Guillette LJ Jr, Hauser R, Heindel JJ, Ho SM, Hunt PA, Iguchi T, Jobling S, Kanno J, Keri RA, Knudsen KE, Laufer H, Leblanc GA, Marcus M, McLachlan JA, Myers JP, Nadal A, Newbold RR, Olea N, Prins GS, Richter CA, Rubin BS, Sonnenschein C, Soto AM, Talsness CE, Vandenbergh JG, **Vandenberg LN**, Walser-Kuntz DR, Watson CS, Welshons WV, Wetherill Y, Zoeller RT. 2007. 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. *Reproductive Toxicology 24: 131-8.*

Rubin BS, Lenkowski JR, Schaeberle CM, **Vandenberg LN**, Ronsheim PM, Soto AM. 2006. Evidence of altered brain sexual differentiation in mice exposed perinatally to low, environmentally relevant levels of bisphenol A. *Endocrinology 147: 3681-91.*

Vandenberg LN, Wadia PR, Schaeberle CM, Rubin BS, Sonnenschein C, Soto AM. 2006. The mammary gland response to estradiol: monotonic at the cellular level, non-monotonic at the tissue-level of organization? *Journal of Steroid Biochemistry and Molecular Biology 101: 263-74.*

Heifetz Y, **Vandenberg LN**, Cohn HI, Wolfner MF. 2005. Two cleavage products of the Drosophila accessory gland protein ovulin can independently induce ovulation. *PNAS 18: 743-8.*

BOOK CHAPTERS

Schug TT, Vogel S, **Vandenberg LN**, Braun JM, Hauser R, Taylor JA, vom Saal FS, Heindel JJ. Bisphenol A. In: Dioxins and Other Persistent Organic Pollutants and Health. Edited by Arnold Schecter, Published by Wiley-Blackwell.

Vandenberg LN. Bisphenol A and diseases of aging: evidence from animal models and human studies. In: Aging and Vulnerability to Environmental Chemicals. Edited by Bernard Weiss, Published by Royal Society of Chemistry (Cambridge, UK). *(in press)*

PUBLICATIONS UNDER REVIEW

Schug, TT, Abagyan R, Blumberg B, Collins TJ, Crews D, DeFur PL, Dickerson SM, Edwards TM, Gore AC, Guillette LJ, Hayes T, Heindel JJ, Moores AR, Patisaul HB, Tal TL, Thayer KA, **Vandenberg LN**, Warner J, Watson CS, vom Saal FS, Zoeller RT, O'Brien KP, Myers JP. Designing endocrine disruption out of the next generation of chemicals. *Submitted to The Green Chemistry Journal.*

Heindel JJ, Zoeller RT, Jobling S, Iguchi T, **Vandenberg LN.** What is endocrine disruption all about? *Submitted to the World Health Organization.*

Vandenberg LN, Lemire JM, Levin M. Serotonin has early, cilia-independent roles in Xenopus left-right patterning. S*ubmitted to Disease Models and Mechanisms.*

Vandenberg LN, Stevenson C, Levin M. Low frequency vibrations disrupt patterning and development of Xenopus laevis embryos. S*ubmitted to PLoS ONE.*

PUBLICATIONS IN PREPARATION

Vandenberg LN, Schaeberle CM, Rubin BS, Sonnenschein C, Soto AM. The male mammary gland is affected by perinatal exposure to bisphenol A. *To be submitted to Reproductive Toxicology.*

Vandenberg LN, Morrie RD, Seebohm G, Lemire JM, Levin M. The role of Rab GTPases in patterning of the left-right axis. *To be submitted to Mechanisms of Development.*

Pai VP, **Vandenberg LN**, Blackiston DJ, Aw S, Levin M. Eye development in Xenopus laevis embryos exhibits a consistent physiological left-right asymmetry. *To be submitted to Developmental Biology.*

Vandenberg LN, Belcher SM, Tanguay R, Schug T, Newbold RR, vom Saal FS, Heindel JJ. Low dose effects of bisphenol A in cells, rodents and aquatic animals. *To be submitted to Environmental Health Perspectives.*

ADDITIONAL REPORTS

Vandenberg LN, Zoeller RT, Myers JP. Environmental Chemicals: Large Effects from Low Doses. *San Francisco Medicine, v 85 (5). June 2012.*

Vandenberg LN. Fixing a deformed frog face. Radio interview, *Living on Earth. PRI's Environmental News Magazine, May 18, 2012.* http://www.loe.org/shows/segments.html?programID=12-P13-00020&segmentID=1

Vandenberg LN. The dose doesn't always make the poison. Radio interview, *Living on Earth. PRI's Environmental News Magazine, March 16, 2012.* *http://www.loe.org/shows/segments.html?programID=12-P13-00011&segmentID=1*

Vandenberg LN. Opinion: There are no safe doses for endocrine disruptors. *Environmental Health News (invited opinion piece), March 15, 2012.* http://www.environmentalhealthnews.org/ehs/news/2012/opinion-endocrine-disruptors-low-level-effects

Vandenberg LN. The BPA show. Radio interview, *Green Street with Patti & Doug Wood, September 28, 2010.* http://www.greenstreetradio.com/092810.html

Vandenberg LN. Formaldehyde in baby shampoos; polycarbonate plastic and bisphenol A. Radio interview, *World News Network, January 14, 2010*. http://wn.com/vandenberg_bpa

Vandenberg LN and Maffini MV. The chemical in your baby's bottle. *Boston Globe Op/Ed, March 23, 2009.* http://www.boston.com/bostonglobe/editorial_opinion/oped/articles/2009/03/23/the_chemical_in_your_babys_bottle/

Soto AM, Maffini MV, **Vandenberg LN**, Rubin BS, Sonnenschein C. Comments for the Meeting of the NTP Board of Scientific Counselors. *CERHR website, May 2008.* http://cerhr.niehs.nih.gov/chemicals/bisphenol/pubcomm/BPA(38)Sotoetal22May2008.pdf

Vandenberg LN, Maffini MV, Rubin BS, Sonnenschein C, Soto AM. Response to the final draft of the NTP-CERHR report on the reproductive and developmental toxicity of bisphenol A. *CERHR website, January 2008.* http://cerhr.niehs.nih.gov/chemicals/bisphenol/pubcomm/Soto_BPA_PanelRptCms_Jan08.pdf.

Vandenberg LN, Maffini MV, Rubin BS, Soto AM. Response to the interim draft of the NTP-CERHR report on the reproductive and developmental toxicity of bisphenol A. *CERHR website, June 2007.* http://cerhr.niehs.nih.gov/chemicals/bisphenol/pubcomm/Soto_comments_BPA_interim.pdf

Additional interviews with Huffington Post, NY Times, Martha Stewart Living Magazine, Glamour Magazine, Time Magazine, NPR's Here and Now, USA Today, The Boston Globe, Environmental Health News, and Men's Health (among others).

FELLOWSHIPS & FUNDING

Gerber Foundation Grant (submitted 2011)
Requested $300,000 over 3 years

Wrote R01 supplement (NIEHS) for BPA Biomonitoring Round-robin study (PI: T Woodruff)
PI awarded $200,000, shared between 6 investigators

NIH NRSA Postdoctoral Fellowship, 2009-2011
Total award: $97264 dispersed over 2 years

Science Communication Fellowship, Environmental Health News, 2010
Total award: $5000

Sackler School Dean's Fellowship in Cancer Research, 2005-2006
Total award: $44,000 for stipend, tuition and laboratory supplies

Cell Molecular & Developmental Biology Training Grant, Tufts University, 2004-2005
Total award: $88,000 for stipend, tuition and laboratory supplies

Howard Hughes Research Scholar, Cornell University, 2002-2003

TEACHING EXPERIENCE (FULL SEMESTER COURSES)

Instructor, Tufts University, Department of Biology, Spring 2011.
Experiments in Cell Biology
Responsible for course content, assessments & organizing laboratory exercises for a semester-long senior level course.

Instructor, Tufts University, Department of Biology, Spring 2010.
Experiments in Cell Biology
Developed course content, assessments & laboratory activities for a semester-long senior level course.

TEACHING EXPERIENCE (INVITED COURSES)

Guest Lecturer, Simmons College, Chemistry Department, Fall 2011.
Mechanistic Toxicology
Provided 3 hours of lecture on the endocrine system, endocrine disruptors, and testing for these compounds that can be performed by chemists/environmental health scientists.

Guest Lecturer, University of Massachusetts - Lowell, Graduate Program in Community Health & Sustainability, Fall 2011.
Risk Assessment
Provided 3 hours of lecture on BPA risk assessments.

Guest Lecturer, Tufts University School of Medicine, Graduate Program in Pharmacology, Fall 2011.
Translational Physiology

Provided 2.5 hours of lecture on reproductive physiology, wrote exam questions.

Guest Lecturer, Tufts University School of Medicine, Graduate Program in Pharmacology, Fall 2010.
Translational Physiology
Provided 2.5 hours of lecture on reproductive physiology, wrote exam questions.

Lecturer, Continuing Education Seminars, Fall 2009 – Spring 2010.
Association of Women's Health, Obstetric and Neonatal Nurses
Gave lectures (10-15 hours total) & wrote exam questions for a series of seminars provided around the state.

Guest Lecturer, Tufts University, Department of Biology, Spring 2009.
Experiments in Cell Biology
Organized content and laboratory exercises for a single class (3 hours).

Teaching Assistant, Cornell University, Education Department, Spring 2003.
Community & Learning Partnerships
Led weekly group sessions, served as discussion leader.

Teaching Assistant, Cornell University, College of Human Ecology, Fall 2002.
Design & Environmental Analysis
Led weekly laboratory sessions, graded assignments.

MENTORING EXPERIENCE

Mr. Brian Pennarola (graduated 2011, attending medical school in fall 2012). Mentored for 1.5 years of independent research projects.
Mr. Ryan Morrie (graduated 2012, attending graduate school in fall 2012). Mentored for 3 years including an undergraduate thesis that was rated "highest honors"
Ms. Claire Stevenson (graduated 2010, attending graduate school in fall 2012). Mentored for 1.5 years of independent research projects.
Ms. Minori Keefe (visiting high school student). Mentored for 1 semester of independent research projects.
Mr. Tanzeel Ahmed (graduated 2011). Mentored for 1 semester of independent research projects.
Mr. Chris Bredie (graduated 2012, plans to attend medical school in fall 2013). Mentored for 1 year of independent research projects.

INVITED SEMINARS

Vandenberg LN. Hormones and endocrine disrupting chemicals: low dose effects and non-monotonic dose responses. European Food Safety Authority Scientific Colloquium on low dose response in toxicology and risk assessment, *Parma, Italy, June 2012.*

Vandenberg LN. When the dose doesn't make the poison: low dose effects & endocrine disrupting chemicals. University of Nebraska Medical Center, College of Public Health Grand Rounds, *Omaha, NE, May 2012.*

Vandenberg LN. Non-monotonicity in endocrine disrupting chemical studies: examples and mechanisms. Pew Health Group meeting on Non-Monotonic Doses, *Washington, DC, April 2012.*

Vandenberg LN. Demonstrating low dose effects using a weight of the evidence approach: examples and mechanisms. Pew Health Group meeting on Non-Monotonic Doses, *Washington, DC, April 2012.*

Vandenberg LN. BPA biomonitoring and round-robin approaches to validation of assays. NIH/NIEHS BPA grantees meeting, *Raleigh-Durham, NC, January 2012.*

Vandenberg LN. Low doses and non-monotonicity in the recent BPA literature: trends & new directions. NIH/NIEHS BPA grantees meeting, *Raleigh-Durham, NC, January 2012.*

Vandenberg LN. BPA: how much is in humans, and should we be worried? 15th Annual Green Chemistry & Engineering Conference, *Washington, DC, June 2011.*

Vandenberg LN. BPA is a model endocrine disruptor. e.hormone conference, *New Orleans LA, October 2010.*

Vandenberg LN. Overview of human biomonitoring studies. National Institute of Environmental Health Sciences Grantees Meeting, *Research Triangle Park, September 2010. (Speaker & Discussion Leader)*

Vandenberg LN. The case of human exposure to bisphenol-A. Gordon Research Conference - Environmental Endocrine Disruptors, *Les Diablerets, Switzerland, June 2010.*

Vandenberg LN. Low doses have large effects: the case of bisphenol A. 14th Annual Green Chemistry & Engineering Conference, *Washington, DC, June 2010.*

Vandenberg LN. BPA and the fragile fetus: fetal origins of adult disease. Partners in Perinatal Health Seminar, *Norwood, MA, May 2010.*

Vandenberg LN, Maffini MV. Bisphenol A: Information for Public Health Agencies. Massachusetts Department of Public Health, *Boston, MA, February 2009.*

Vandenberg LN. Does breast cancer start in the womb? The case of bisphenol A. Partners in Perinatal Health Seminar, *Marlboro, MA, May 2009.*

Vandenberg LN. Xenoestrogens and the breast cancer link: The tale of Bisphenol-A. Pardon Our Appearance: Massachusetts Breast Cancer Coalition Educational Workshop, *Arlington, MA, April 2007.*

Vandenberg LN, Atkinson JC, Calafat AM, Eichmiller F, Kingman A, Marcus M, Olea N, Thayer KA, Hauser R, and Welshons WV. Bisphenol-A: Human exposure panel report. NIEHS BPA Workshop, *Research Triangle Park, NC, November 2006.*

ADDITIONAL PRESENTATIONS

Vandenberg LN, Jacobs DR, Lee DH. Examples of non-monotonicity in epidemiologic studies. *Selected Speaker,* Pew Health Group meeting on Non-Monotonic Doses, *Washington, DC, April 2012.*

Vandenberg LN. The Xenopus tadpole has an innate ability to correct craniofacial defects. *Speaker,* Tufts University Biology Department, Research Associate Seminar, *Medford, MA, 2011.*

Vandenberg LN, Levin M. The Par6 complex is required for both early and late orientation of the left-right axis in Xenopus. *Selected Speaker,* Society for Developmental Biology Annual Meeting, *Albuquerque, NM, 2010.*

Vandenberg LN and Levin M. Consistent left-right asymmetry cannot be established by late organizers in Xenopus. *Poster presenter,* Society for Developmental Biology Annual Meeting, *San Francisco, CA, 2009.*

Vandenberg LN, Soto AM, and Sonnenschein C. It's not in your genes but the company you keep. Phenotype, a view from the bench. *Selected Speaker,* International Society for the History, Philosophy, and Social Studies of Biology Annual Meeting, *Exeter, England, 2007*.

Vandenberg LN, Maffini MV, Schaeberle CM, Rubin BS, Sonnenschein C, Soto AM. Early exposure to the xenoestrogen bisphenol-A has long-lasting effects on the mammary gland in both male and female mice. *Selected speaker,* Endocrinology Annual Meeting, *Toronto, Canada, 2007.*

Vandenberg LN, Maffini MV, Wadia PR, Sonnenschein C, Rubin BS, and Soto AM. Exposure to bisphenol-A alters growth and morphology of the fetal mammary gland. *Poster presenter,* Endocrinology Annual Meeting, *Boston, MA, 2006.*

Vandenberg LN, Maffini MV, Wadia PR, Sonnenschein C, Rubin BS, and Soto AM. In utero exposure to environmentally relevant levels of bisphenol-A alters growth and morphology of the fetal mouse mammary gland. *Poster presenter and selected speaker,* Gordon Research Conference- Environmental Endocrine Disruptors, *Il Ciocco, Italy, 2006.*

PROFESSIONAL SERVICE

Reviewer -- Environmental Research, Toxicology & Applied Pharmacology, Reproductive Toxicology, Human & Experimental Toxicology, Environmental Health Perspectives, Archives of Environmental Contamination & Toxicology, Environmental Health, Fertility & Sterility, Science Translational Medicine, Proceedings of the National Academy of Sciences (PNAS), Molecular & Cellular Endocrinology, Food & Chemical Toxicology, Journal of Toxicology, Environmental Science & Technology, Pediatrics, Environmental International, Chemical Research in Toxicology

Grant Reviewer (ad hoc) – Medical Research Council of South Africa, 2011.

Invited member, German Umweltbundesamt Panel, BPA assessment, 2009.

Invited member, German Federal Institute for Risk Assessment, Endocrine Disrupting Chemicals and Plant Assessment Expert Panel, 2009.

Board of Directors, Massachusetts Odyssey of the Mind, 2008 – present

Senior Advisory Council Member, Coalition for a Safe & Healthy Connecticut, 2008

Judge, Boston High School Science Fair, and Massachusetts State Science & Engineering Fairs (Middle & High School), 2007-2009

Mentor, Great Neck Breast Cancer Coalition High School Scholars Program, 2006-2007

Mentor, Boston Latin High School Science Outreach Program, 2006

AWARDS

Top 20 Downloaded Citations, *Reproductive Toxicology 2007-2008.*

Top 10 Cited Manuscripts, *Reproductive Toxicology 2007-2008.*

Endocrine Society Travel Award, *2007*.

Outstanding Trainee Award, Gordon Conference, Environmental Endocrine Disruptors, *2006*.

Cornell University, graduation *Magna Cum Laude*, awarded *Distinction in Research, 2003.*

PUBLISHING METRICS

H-index of 14, as of May 2012.

ENVIRONMENTAL HEALTH NEWS: SCIENCE SUMMARIES & MEDIA REVIEWS

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