

May 13, 2010

Ms. Cynthia Oshita
Office of Environmental Health Hazard Assessment
P.O. Box 4010, MS-19B
Sacramento, California 95812-4010

Dear Ms. Oshita:

Thank you for giving us the opportunity to provide comments to you regarding bisphenol A (BPA). As organizations that represent health affected communities, environmental health groups, public health organizations and workers, we are pleased that OEHHA is taking the necessary steps towards examining this chemical. We agree with OEHHA's initial assessment that BPA meets the criteria for listing as known to the State to cause reproductive toxicity under Proposition 65, based on findings of the National Toxicology Program's Center for the Evaluation of Risks to Human Reproduction.

We appreciate that you are receiving public comment to fully evaluate the science on this matter and agree that science should dictate this decision, rather than politics. However, with the politicization of science that has occurred on this issue, it is impossible to separate the scientific inquiry from the historical and political context.

As you know, scientists have known since at least the 1930s of BPA's ability to interfere with hormones. It was developed to be one of the first synthetic estrogens but shelved for pharmacological use in favor of the more potent DES (diethylstilbestrol). However, polymer scientists began to use BPA in consumer products as early as the 1950s. Today, BPA is one of the most pervasive chemicals in modern life with an annual national production exceeding two billion pounds and can be found in the bodies of 93% of Americans.

To date, over 200 studies have demonstrated the harm that comes from extremely low doses of BPA. All of these studies have been peer reviewed and demonstrate clear harm. Since the NTP's 2008 statement regarding BPA, additional research has come out that highlights the links between low-dose exposure to BPA and reproductive harm. A study published in 2010 showed that exposure of human placental cells to low doses of BPA may cause detrimental effects, leading *in vivo* to adverse pregnancy outcomes such as preeclampsia, intrauterine growth restriction, prematurity and pregnancy loss.ⁱ Animal studies conducted in 2009 suggested that BPA causes long-term adverse reproductive and carcinogenic effects if exposure occurs during critical periods of differentiation and neonatal exposure to BPA altered reproductive parameters and hypothalamic-pituitary function in female rats.^{ii,iii}

Due to this clear and compelling evidence, regulatory agencies in the United States are beginning to take action on the concerns surrounding BPA. In January 2010, the U.S. Food and Drug Administration (FDA) reversed its much-criticized position on BPA safety, stating its concern about the chemical's effects on fetuses, infants and children. FDA is now in agreement with a National Toxicology Program 2008 position that there is "some concern" regarding BPA. In March 2010, the United States Environmental Protection Agency announced that it will be working closely with other regulatory agencies on research to better assess and evaluate the potential health consequences of BPA exposures, including health concerns from non-food packaging exposures that fall outside of the FDA's reach but within EPA's regulatory authority. Finally, in January 2010, NIEHS announced plans to fund nearly 50 million dollars

of American Recovery and Reinvestment Act funds to produce data that will allow for a comprehensive assessment of BPA's possible human health effects.

Clearly there is growing scientific consensus that BPA is harmful. The Canadian Health Ministry recently listed it as a toxic substance, has banned its use in baby bottles and has announced its plans to regulate BPA in food and infant formula containers. Denmark has also banned the use of BPA in children's products. In addition, legislation to regulate BPA has been introduced in more than 29 states and localities. Five of those states have passed legislation regulating and banning BPA especially in relation to its uses in children's feeding devices and infant formula.

The case of BPA is reminiscent of the tobacco industry's campaign to deny the health hazards of smoking. For years, state agencies and scientific bodies were unsure about how to act on tobacco due to the "dueling science" that confronted decision makers. We now know that much of the science demonstrating no adverse effect from tobacco products was produced by the tobacco industry as a way to manufacture doubt in regulators' minds long enough to sell their product for a little while longer.

We are seeing the same scenario play out with BPA. Time and again, industry declares that they cannot replicate the findings of the independent scientists' studies and states that the chemical is safe. But further examination of their studies show serious flaws such as using rats that are predisposed to not be affected by synthetic estrogens or feeding the animals a diet that would mask the effects of BPA or even, as in the case of a recent study by Rebecca Tyl, downplaying the results of data clearly demonstrating an effect.

Too often, we give chemicals the same rights as people—demanding that we have absolute certainty of harm beyond all doubt, rather than relying on credible evidence of harm to take action. As a result, doubt is often manufactured through industry funded studies and inconsistencies in outcome from government funded studies are used as an excuse to not take action.

The job of the government is to protect public health. OEHHA's role in protecting public health is to determine if there is enough evidence to warrant informing the public of a risk to their health. The science is in and the evidence is clear. Public health, particularly the health of fetuses, infants and children is compromised by exposure to BPA. We urge OEHHA to use the evidence before it and not be swayed by industry tactics or their manufactured doubt and list BPA as a reproductive toxicant on the Proposition 65 list.

Sincerely,

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i Benachour N, Aris A (2010). Toxic effects of low doses of Bisphenol-A on human placental cells. *Toxicology and Applied Pharmacology* 241: 322–328.

ii Newbold RR, Jefferson WN, Padilla-Banks E (2009). Prenatal exposure to bisphenol A at environmentally-relevant doses adversely affects the murine female reproductive tract later in life. *Environmental Health Perspectives* 117:879-885.

iii Fernández M, Bianchi M, Lux-Lantos V, Libertun C (2009). Neonatal exposure to bisphenol A alters reproductive parameters and gonadotropin releasing hormone signaling in female rats. *Environmental Health Perspectives* 117:757-762.