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Science 2.0

What Do We Really Know About BPA And Fertility?

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Last week, a study published in the journal Human Reproduction reported that bisphenol-A (BPA), a compound widely used to make polycarbonate plastic and epoxy resins, altered maturation of human oocytes in vitro.

Specifically, at high concentrations of BPA, oocyte maturation decreased while the incidence of oocyte degeneration increased. In an accompanying press release, the authors suggested that BPA "may cause a significant disruption to the fundamentals of the human reproductive process and may play a role in human infertility."

But does this study really support such a far-reaching implication, and are the results consistent with other studies that directly and comprehensively examined the potential for BPA to affect reproduction? In vitro studies such as this are useful, as a preliminary study to generate a hypothesis, or to examine the mechanism of an established effect. However, it would overstate the results of this study to say that it establishes an effect in humans. In the case of BPA, robust data is available to help us interpret these findings and, in particular, demonstrate why this study is of limited utility in identifying and evaluating health risks in people.

Perhaps most importantly, the study is of questionable physiological relevance since the BPA concentrations showing effects on maturation are vastly higher than an average person's daily intake of BPA. Extensive biomonitoring data from CDC and others show that typical human exposure to BPA from all sources is extremely low, on the order of a couple micrograms per day for a typical adult.

Image credit: BevNet

Since BPA is rapidly metabolized and eliminated from the body with a half-life of only a few hours (Doerge et al., 2010), it does not accumulate in the body and peak concentrations of BPA in serum are undetectable (< 0.3 ppb) even with an atypically high dietary intake of BPA (Teeguarden et al., 2011). The authors point to a 2002 study from Japanese authors claiming low ppb levels of BPA in serum and follicular fluid (Ikezuki et al. 2002), but neglected to note that the analytical method used in that study was subsequently found to be invalid for measurement of BPA in human samples (Fukata et al., 2006). In contrast, the concentration showing a clear effect on oocyte maturation in this study was 20,000 ppb, with no apparent effect on maturation at 20 ppb and borderline significance at 200 ppb.

The authors do note that the study may have been impacted by the use of "clinically discarded" oocytes, mostly from infertile patients, that had not matured in vivo even after hormonal stimulation. They acknowledge that this "potentially compromised material," could "reduce the relevance of our findings to a more normal population."

Although this new study has significant limitations to clinical relevance, the potential for BPA to affect reproduction has been carefully examined with multi-generation studies in multiple species and strains of laboratory animals (for example, Ema et al., 2001; Tyl et al., 2002; Tyl et al., 2008).

These studies, published in the peer-reviewed scientific literature but not cited in this new study, consistently have concluded that BPA does not affect fertility or other reproductive parameters at any dose even remotely close to actual human exposure levels.

Based on the weight of scientific evidence from these studies and others, regulators have repeatedly concluded that BPA is not a reproductive toxicant. In fact, the FDA recently updated its position on BPA, stating its "current assessment is that BPA is safe at the very low levels that occur in some foods."