

NGO's BPA Report Intended to Frighten, Not Enlighten

By ACSH Staff — May 20, 2010

Raising the specter of “endocrine disruption” is always a good way to promote anxiety and concern, as the National Workgroup for Safe Markets must know very well.

Its [report](#) ^[1] declaring it had found trace levels of the chemical Bisphenol A (BPA) in cans of food prompted scary news stories. “Before you open a can of soup or green beans,” [one report ominously intoned](#) ^[2], “you might want to hear this.”

But conclusions about the risks of environmental toxicants must be based on good science and the principles of teratology (the study of birth defects) and toxicology, not hype and hysteria.

BPA has been commonly used as a liner in food cans for 50 years. It prevents the food from interacting with the metal and deteriorating.

What's in the can is somewhat irrelevant because the can can't develop cancer, get birth defects or have its endocrines disrupted.

What's important is the human exposure to BPA and the range of serum concentrations —something that's been exhaustively studied.

A colleague of mine, developmental toxicologist Shelly Tyl, performed one of those studies. A few years ago she performed a modern, up-to-date in vivo animal toxicology study, titled “[Three-generation reproductive toxicity study of dietary bisphenol A in CD Sprague-Dawley rats](#) ^[3].”

This was, practically speaking, a very scientific study into the effects of BPA upon several generations of rats. The rodents were placed in seven groups and given BPA-laced food in a 500,000-fold range of exposures — from one part per billion to 500 parts per million.

Three generations of rats were evaluated on a huge host of factors: mating; fertility; gestational indices; ovarian primordial follicle counts; estrous cyclicity; precoital interval; gestational length; offspring sex ratios; postnatal survival; nipple/areolae retention in pre-weanling males; epididymal sperm number, motility, morphology; daily sperm production; and efficiency of daily sperm production.

No rats showed any sign of exposure on those counts, although there was total and some organ reduction in weight for rodents in the highest exposure groups.

In order to perform a risk analysis for BPA in the human population you need two more pieces of

information that are not present in Tyl's study. But other studies have filled in the gaps.

Japanese researcher Hideto Yamada and his colleagues [studied levels](#) [4] of BPA concentrations in the blood and amniotic fluid of pregnant mothers in their early second trimester. The average concentration of bisphenol A was 0.32 parts per billion of serum, with a range of 0.0 to 1.6 parts per billion.

It appears these levels are far below the threshold exposure determined in the Tyl study. But you cannot be certain because we don't have the same pharmacokinetic data in rats that Yamada and his colleagues reported in humans. We don't know the threshold serum levels in rats.

We also don't know whether the metabolism and effects of BPA are similar in humans and rats.

All this shows how difficult risk analysis can be if the researcher wishes to base the risk analysis and conclusion on the most accurate and complete set of data — but the important point is that human serum concentrations of BPA are very, very low, far below any expected toxic effects.

Combining studies of Tyl et al and Yamada et al may appear to be a rare occurrence in the field of risk analysis. Fortunately, it is occurring more often.

However, the National Workgroup for Safe Markets publication wasn't intended to educate the public about risks, but to frighten unsophisticated scientists and the public. We should respond to such garbage with good science.

I testified before Sen. Barbara Boxer's committee dealing with chemical risks. Her knowledge of toxicology is primitive, but she is committed to banning this chemical without any scientific evidence that it poses a risk to the public. I wish that there was not one toxic chemical in the environment. But they are there and we have to deal with the chemicals on the basis of the risk that they may represent. There are very few environmental chemicals that pose a hazard because the exposure level is low. Toxic exposures, meaning high-level exposures, have occurred with lead, methyl mercury, carbon monoxide, arsenic and others. That is why it's so important to determine what exposure levels are toxic and which exposures are not toxic.

The overwhelming scientific evidence points to the conclusion that at current human exposure levels, BPA is not toxic — and specifically is not linked to the myriad diseases outlined in the National Workgroup for Safe Markets report released earlier this week.

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Links

[1] <http://www.uspirg.org/home/reports/report-archives/healthy-communities/healthy-communities/no-silver-lining-an-investigation-into-bisphenol-a-in-canned-foods>

[2] <http://www.keyc.com/node/37455>

[3] <http://www.ncbi.nlm.nih.gov/pubmed/12075117>

[4] http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6TC0-471FGYH-1&_user=10&_coverDate=12%2F31%2F2002&_rdoc=1&_fmt=high&_orig=search&_sort

[5] <http://www.otispregnancy.org/>