DOI: 10.2478/10004-1254-62-2011-2176

Letter to the Editor

BISPHENOL-A IN CANNED FOOD PRODUCTS: IS IT REALLY REQUIRED?

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Bisphenol-A (BPA) is usually associated with plastics leachate and most of the research has been primarily focused on identifying ways and means to prevent it. However, there are new indications from recent studies that consumption of canned products, soups in particular, can increase urinary BPA levels in humans by more than 1200 % (1). The potential health effects of BPA on human body are still under debate in scientific communities at this point.

BPA exposure to humans through consumer products

BPA is a chemical that is produced in the world in very high volumes up to an amount of nearly 2.7 billion kilograms per year. Around 450 thousand kilograms or more per year is manufactured or imported in the United States alone (2). It is used in the plastic industry, metal food containers industry, and as composites and sealants in dentistry (3-5). It is used for the manufacture of epoxy resins and for application in metal food cans as a protection against rusting and corrosion. There are documented studies about potential migration of BPA leaching from epoxy resin coatings to food in metal cans (6).

This is not the only exposure route to humans, others being through drinking water, composites and sealants in dental industry, skin exposure, and dust inhalation. It is estimated that nine out of ten persons in the world are exposed to BPA (7).

HUMAN HEALTH EFFECTS FROM BPA DIETARY INTAKE EXPOSURE

The last decade of studies

Studies indicate that human urine is the most appropriate body fluid for BPA exposure assessment (8) and the higher the BPA concentration in urine, the more adverse are the health effects on humans. This hypothesis is based on animal and laboratory experimental evidence, which calculates the half-life for renal clearance of BPA after oral ingestion to be 5.3 h in adult men and women (8). In 2008, the US National Health and Nutrition Survey (NHANES) released epidemiological data from a study of urinary BPA concentrations and their health effects on a large-scale population. The study showed that the higher the urinary BPA concentration, the more adverse are the health problems associated with cardiovascular diseases and diabetes (6).

Animal experimental studies have demonstrated that high BPA doses result in estrogen-like effects on uterine and prostate organ weights. At low doses, they decrease sperm production, increase the prostate volume, change the mammary gland, change vaginal morphology and estrous cycles, disrupt sexual differentiation in the brain, and accelerate growth and puberty (9-12). Presently the low-dose effects of BPA are being contested by some researchers (13), but other

recent studies clearly indicate various pathways through which even low-dose BPA can stimulate cellular responses (14).

In contrast, there are research studies that do not agree with the above conclusions and indicate that there is no immunologic, neurologic, genotoxic or carcinogenic risk for humans by oral ingestion of BPA (15).

The debate

A lot of studies have been carried out since 1999 that used different techniques and experimental methods to measure unconjugated BPA in human serum and found it anywhere in the range between 0.2 ng mL^{-1} and 20 ng mL^{-1} (6). This strongly contests the theory of oral ingestion as the only accountable cause of exposure in humans. It fails to take in account other possibilities of contamination by non-oral routes such as bathing, inhaling or using implanted medical devices (14). In fact, experiments indicate that BPA has regularly been detected in human blood in low levels, which supports other routes of exposure (16).

Studies in 2008

Bontempo et al. (17) found apoptotic effects of BPA in three different acute myeloid leukemias. Soto et al. (18) found a clear correlation between fetal exposure to BPA and increased incidence of breast cancer over the last five decades in the US and Europe. It concluded that fetal environment is sensitive to BPA exposure and can lead to breast cancer in adulthood. In addition, Dairkee et al. (19) suggest that prior exposure to BPA is associated with aggressive tumor in high-risk breast tissues.

Studies in 2009

Benachour and Aris (20) clearly identified the connection between low-dose exposure to BPA and adverse effects on human placental cells, leading *in vivo* to adverse pregnancy outcomes such as preeclampsia, intrauterine growth restriction, prematurity, and pregnancy loss.

Studies in 2010

Cavalieri and Rogan (21) connected the risk of cancer initiation in human body by direct and/or indirect mechanisms, including DNA mutations, with exposure to BPA. A few other significant studies clearly established the ability of BPA to induce neoplastic transformation in human breast epithelial cells, the role of BPA in causing neuroblastoma cell proliferation, and the toxic effects of BPA exposure on placental and fetal development (22-24)

Studies in 2011

A recent study (25) on mice has confirmed the risk of mammary cancer in mice through BPA exposure. In addition, there is experimental evidence that exposure to BPA leads to adverse reproductive effects in both male and female mice (26).

BPA IN METAL-CANNED FOOD PRODUCTS

Recent study

A recent study carried out by the Harvard School of Public Health (1) has established an increase of more than 1200 % in urinary BPA levels in daily canned soup consumers compared with the consumption of fresh soup. The study participants were asked to eat soup directly out of metal cans for five days to rule out any external contamination with BPA. Normal adult urinary BPA levels average around 2 $\mu g\ L^{-1}$. However, in this study, the urinary BPA levels rose above 20 $\mu g\ L^{-1}$or 1221 % compared to the study participants who consumed soup from fresh ingredients.

Another study by Rudel et al. (27) evaluated the contribution of food packaging to human BPA exposure. It measured urinary BPA and phthalate metabolites before, during, and after dietary intervention with fresh foods. Urine analysis showed lower levels of BPA and Bis(2-ethylhexyl)phthalate (DEHP) metabolites during fresh-food intervention (27).

CONCLUSION AND RECOMMENDATION

Recent studies have plainly shown that the use of epoxy coatings in metal cans is responsible for high urinary BPA concentrations in humans. However, health effects of this exposure are still unclear and debatable. This does not suggest in any way that we can have a can of BPA-contaminated food product every morning and evening and expect to lead a healthy life. Quite clearly, the data collected so far in the field of environmental toxicology are good enough

to raise concern about the future impact of BPA on human health and development.

As Benjamin Franklin rightly said "An ounce of prevention is better than a pound of cure", so as a precaution for human health and development, it is highly advisable to regulate the use of BPA in metal food containers, which is finding its way onto our dining tables through canned food products like fish, beans, corn, soups, and dry milk, especially when such products are widely available for consumption by communities of all ages, including the healthy, sick, pregnant, at-risk, and hospital communities. Based on the experimental evidence and scientific theories available to us, the regulatory agencies should replace chemicals like BPA with safer options that are not likely to harm normal development of humans. Such abnormally high levels of BPA in human urine clearly urge for BPA-free food products. This would be possible only by exploring the possibilities of a BPA-free packaging for a healthy future of consumers.

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