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New Science 2.0 Article

Does BPA Make You Fat?

http://www.science20.com/steve_hentges/does_bpa_make_you_fat-131831

The article discusses the recent LaKind et al. paper on BPA epidemiology studies that report links between BPA exposure and obesity, diabetes, and cardiovascular disease (see my e-mail of January 6).

As with most questions related to the common chemical bisphenol A (BPA), the answer to that question may depend on who you ask. The question is a particularly relevant one in recent years with the advent of the "environmental obesogen" hypothesis, which refers to "…chemicals that inappropriately alter lipidhomeostasis and fat storage, metabolic setpoints, energy balance, or the regulation of appetite and satiety to promote fat accumulation and obesity."

The hypothesis further suggests that exposure to so-called obesogens can cause an array of medical conditions that result in increased risk for obesity, type 2 diabetes mellitus (DM) and cardiovascular disease (CVD).

Not surprisingly, given the controversy that occasionally swirls around it, BPA has been suggested to be an obesogen.

If BPA makes you fat, then not using BPA will be like a diet. Right? In the world of talk show "correlomics" you just assume a correlation (BPA causes obesity) and imply causation (there is more obesity, right?) and then let people make the connection. That isn't science, though. Credit and link: Examiner.com

But getting away from opinion and into the realm of science, hard data and the scientific process will ultimately provide the most reliable answer. One form of data that has become plentiful in recent years comes from epidemiology studies aimed at testing the hypothesis that BPA is a risk factor for obesity, CVD and DM. Taken at face value, one study at a time, these studies seem to suggest that BPA may be linked to these health outcomes.

With more than 40 published epidemiology studies that examined potential links between BPA and obesity, CVD and DM, a group of researchers recently set out to evaluate the consistency and quality of these studies. Their results don't answer the title question, but are very informative to understanding why the available studies are incapable of answering the question.

In a recent paper published in the peer-reviewed journal Critical Reviews in Toxicology, the researchers describe their use of current methodological guidance for systematic reviews to evaluate the BPA studies. In particular, the evaluation used two independent researchers to identify, review and summarize the relevant studies, which numbered 45 at the time the evaluation was conducted.

Particular attention was paid to study design and exposure assessment, which have previously been identified as weaknesses in BPA epidemiology studies.

The researchers found that a quantitative meta-analysis, which would involve pooling results across studies, was not feasible. In part this was because many of the studies were based on the same data and the methods used to analyze the data were too heterogeneous to allow pooling of results. Instead, the researchers evaluated the strength of evidence from each individual study and characterized the outcomes as positive, inverse, null, or mixed.

Overall, results across studies were inconsistent for the health outcomes examined. Even studies that used the same data with slightly different statistical methods produced different results. Most informative from the evaluation is the researchers' assessment of the limitations in the available studies and why the current body of literature cannot provide an answer to the title question.

Although the number of BPA epidemiology studies has been growing at a seemingly exponential rate in the last few years, the quality of the evidence provided has not improved. In particular, most of the studies use a cross-sectional design in which health outcome and exposure data are collected at a single point in time.

The main limitation of cross-sectional studies is the inability to establish a temporal sequence between exposure and outcome, which is of critical importance for a cause-effect relationship. This is not only a scientific issue since the findings from cross-sectional studies are often interpreted in the popular media as demonstrating a cause-effect link between BPA and a health outcome. Such a link cannot be established with a cross-sectional study design.

A second critical issue is that almost all of the studies relied upon single exposure measurements, typically measurement of BPA metabolites in single urine samples. Because of the very short half-life of BPA in the body, and high variation in BPA levels over short time periods, single measures of exposure do not provide robust estimates of long-term exposure. This is a particular issue for health outcomes that may initiate and develop over many years.

So what does it all mean? Does BPA make you fat? The conclusion says it all: “study design and

data quality issues severely limit the utility of the existing epidemiology research for improving our understanding of potential chronic health effects associated with BPA exposure…. Considering the methodological limitations of the current epidemiology literature, assertions for or against a causal link between BPA and obesity, CVD or DM based on this body of research are unsubstantiated."

That's about as close to a definitive no as you'll get.

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