Endocrine disruptors and obesity, diabetes and heart disease: What does epidemiological research tell us?

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1. Background

2. Bisphenol A (BPA)

3. Phthalates (results only)

4. Conclusions
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Studies based on cross-sectional datasets have been used to draw causal inferences regarding environmental chemical exposures and adverse health outcomes.

**BPA, phthalates tied to kids' weight, diabetes risk**
Reuters Health; Aug 19, 2013;
http://uk.reuters.com/article/2013/08/19/us-bpa-kids-weight-idUKBRE97I02E20130819

**BPA, Phthalates Linked With Teen Obesity, Insulin Resistance**
HuffPost Healthy Living
http://www.huffingtonpost.com/2013/08/19/bpa-teen-obesity-insulin-resistance-phthalates_n_3781248.html
Bisphenol A and indicators of obesity, glucose metabolism/type 2 diabetes and cardiovascular disease: A systematic review of epidemiologic research
Methods:

Followed the current methodological guidelines for systematic reviews

Particular attention paid to study design and exposure assessment: cited as main areas of weakness in BPA epidemiologic research

Study results were categorized qualitatively as positive, negative, null, or mixed
Results:

For all outcomes, results across studies were inconsistent.

Some studies used the same data and the same or similar statistical methods: when the methods varied slightly, even studies that used the same data produced different results.

Why?

Nearly all studies used cross-sectional design

Single measure of BPA exposure – exposure misclassification
Conclusions:

Study design issues severely limit our understanding of potential health effects associated with BPA exposure.

Considering the methodological limitations of the existing body of epidemiology literature, current epi data **neither support nor refute** the hypothesis that BPA causes obesity, CVD or diabetes.
Do phthalates act as obesogens in humans?
A systematic review of the epidemiology literature
Results:

26 epidemiology publications; 18 independent data sources.

No inter- or intra-study consistency for any phthalate metabolite for any of the indicators of overweight/obesity, DM or CVD.

Most reported associations were not statistically significantly different from the null, some were positive, and others were inverse.
Conclusion:

Considering the methodological limitations of the existing body of epidemiology literature, the current epidemiological data neither support nor refute the hypothesis that phthalates cause obesity, CVD or diabetes.
Not drawing conclusions as to whether or not BPA/phthalates are risk factors for obesity, heart disease or diabetes.

Stating the opposite: using cross-sectional datasets like NHANES to draw conclusions about exposure to short-lived environmental chemicals and chronic complex diseases is inappropriate.
Need more resources for appropriately designed epidemiologic studies and toxicological explorations to understand whether these types of chemicals play a causal role in chronic diseases.