

Using our quantum biology algorithm, with near certainty, the links between phthalates and breast cancer or other chronic diseases can be understood.

For discussion with qualified bioinformatics professionals, the document affixed to this article explains the fact that endocrine disruption by phthalates is caused by mutation of cell receptors that are crucial for the uptake of cell defenses.

In our opinion, the use of phthalates in drugs or anywhere in the food chain is unthinkable!

<https://ascopubs.org/doi/abs/10.1200/JCO.18.02202?journalCode=jco>

## **Phthalate Exposure and Breast Cancer Incidence: A Danish Nationwide Cohort Study**

[Thomas P. Ahern](#), PhD, MPH<sup>1</sup>; [Anne Broe](#), MD, PhD<sup>23</sup>; [Timothy L. Lash](#), DSc<sup>45</sup>; [Deirdre P. Cronin-Fenton](#), PhD<sup>5</sup>; [Sinna Pilgaard Ulrichsen](#), MSc<sup>5</sup>; [Peer M. Christiansen](#), MD, DMSci<sup>5</sup>; ...[Show More](#)

4-26-2019

### **PURPOSE**

Phthalate exposure is ubiquitous and especially high among users of drug products formulated with phthalates. Some phthalates mimic estradiol and may promote breast cancer. Existing epidemiologic studies on this topic are small, mostly not prospective, and have given inconsistent results. We estimated associations between longitudinal phthalate exposures and breast cancer risk in a Danish nationwide cohort, using redeemed prescriptions for phthalate-containing drug products to measure exposure.

### **METHODS**

We ascertained the phthalate content of drugs marketed in Denmark using an internal Danish Medicines Agency ingredient database. We enrolled a Danish nationwide cohort of 1.12 million women at risk for a first cancer diagnosis on January 1, 2005. By combining drug ingredient data with the Danish National Prescription registry, we characterized annual, cumulative phthalate exposure through redeemed prescriptions. We then fit multivariable Cox regression models to

estimate associations between phthalate exposures and incident invasive breast carcinoma according to tumor estrogen receptor status.

## **RESULTS**

Over 9.99 million woman-years of follow-up, most phthalate exposures were not associated with breast cancer incidence. High-level dibutyl phthalate exposure ( $\geq 10,000$  cumulative mg) was associated with an approximately two-fold increase in the rate of estrogen receptor–positive breast cancer (hazard ratio, 1.9; 95% CI, 1.1 to 3.5), consistent with in vitro evidence for an estrogenic effect of this compound. Lower levels of dibutyl phthalate exposure were not associated with breast cancer incidence.

## **CONCLUSION**

Our results suggest that women should avoid high-level exposure to dibutyl phthalate, such as through long-term treatment with pharmaceuticals formulated with dibutyl phthalate.

**MCFIP – Research has established phthalates as being endocrine disruptor. With that being said, when we subjected phthalates to our quantum biology modeling, the following factors were identified:**

- Phthalates impact G-protein coupled receptor 30 (GPCR30)
- GPCR30 and GPER (G-protein estrogen receptors) are bioidentical
- GPCR30 provide uptake for the super family of cytokines; i.e. IL-1 – IL-18 and Il-33.
- The autophagy mechanism for conversion and recycling of lipids (fat) is a byproduct of IL-18

## **Summary**

Phthalates mutate GPCR30/GPER receptors. This disruption impacts estrogen activity (a fact determined through research) as well as the ability to convert fat within cells as a result of disruption of IL-18 uptake into cells.

Our modeling of endocytosis established the fact that many forms of reception of signaling exists; GPCR30/GPER being different that phagocytosis.

<http://www.mcfip.net/upload/Endocytosis%20Modeling%208-24-15.pdf>

We theorize that the above referenced scenario accounts for how and why phthalates disrupt estrogen activity as well as leading to obesity.

<https://www.sciencedaily.com/releases/2016/04/160420125551.htm>

## Chemical exposure could lead to obesity, study finds

April 20, 2016

University of Georgia

*Date:*

*Source:*

*Summary:*

Exposure to chemicals found in everyday products could affect the amount of fat stored in the body, according to a study. Phthalates are chemicals found in everything from plastic products to soap to nail polish -- they give plastic its bendy stretch.

Exposure to chemicals found in everyday products could affect the amount of fat stored in the body, according to a study by University of Georgia researchers.

Phthalates are chemicals found in everything from plastic products to soap to nail polish -- they give plastic its bendy stretch. But growing research shows that these chemicals could be harming people's health, said the study's lead author Lei Yin, an assistant research scientist in the UGA College of Public Health's department of environmental health science.

"Phthalate exposure can be closely associated with the rise of different types of disease development," Yin said.

Because levels of phthalates were found in human fluids in previous studies, the researchers wanted to see if a specific phthalate, benzyl butyl phthalate, or BBP, had an effect on the accumulation of fat in cells. Their findings were published in *Toxicology in Vitro*.

The researchers used mouse cells to create in vitro models to analyze how exposure to BBP affected the way oils and fats, known as lipids, accumulated within the cells.

"Obesity is one of the big issues in humans now, and of course genetic components can contribute to the development of obesity," said study co-author Xiaozhong "John" Yu, an assistant professor of environmental health science. "However, environmental exposure may also contribute to obesity."

Some phthalates have proven to cause reproductive toxicity at high levels of exposure, but the link between low-level exposure and BBP hadn't yet been thoroughly explored, Yin explained.

"It could be that some chemicals at a very low dose and over a long period time, which is known as chronic exposure, can cause more harmful diseases or effects," she said.

The researchers quantified lipid droplet accumulation using traditional staining approaches, in which the cells are stained and therefore can be visually assessed under a microscope, and a newer approach called cellomics high-content analysis. This high-content screening uses "image processing algorithms, computer machine learning and can measure the multiple parameters in a fast and objective way," Yin said.

The results of BBP's effects were compared with bisphenol A, or BPA, an environmental endocrine disruptor that is known for its role in adipogenesis, or how fat cells develop.

BBP caused a response in the cells that is similar to BPA: Both chemicals prompt the accumulation of lipid droplets. However, the droplets from BBP-treated cells were larger, something that suggests BBP exposure may lead to obesity.

Although the findings cannot be directly generalized to the human population -- Yu notes the cells used were mouse cells and a "human is not a big mouse" -- they do give an indication of a possible link between exposure to BBP and obesity, something that could affect human health.

Calling obesity research a very exciting area to be studying, Yin said she would like to explore the relationships between other environmental chemicals and obesity in future studies. She is also interested in learning if certain plant-based chemicals could counterbalance the negative effects of exposure to more harmful chemicals.

---

#### Story Source:

The above post is reprinted from [materials](#) provided by [University of Georgia](#). The original item was written by Leigh Beeson. *Note: Materials may be edited for content and length.*

---

#### Journal Reference:

1. Lei Yin, Kevin Shengyang Yu, Kun Lu, Xiaozhong Yu. **Benzyl butyl phthalate promotes adipogenesis in 3T3-L1 preadipocytes: A High Content Cellomics and metabolomic analysis.** *Toxicology in Vitro*, 2016; 32: 297 DOI: [10.1016/j.tiv.2016.01.010](https://doi.org/10.1016/j.tiv.2016.01.010)