Born and Dying in a Toxic World:

Testing for Environmental Pollutants During Weight Loss, Part 1

By Chris D. Meletis, N.D.
DATE PLACEHOLDER

Obesity and being overweight are known to cause a vast number of health problems. Yet, in some cases, an obesity paradox has been reported, where obese and overweight individuals have a better prognosis and lower mortality than people who have an ideal body weight.[1] For example, it has been found that obesity can protect against dementia.[1]

Mortality is also lower in obese patients with high levels of toxins.[2] In obese people with high levels of polychlorinated biphenyls, those with the highest fat mass actually had lower mortality compared to those with lower fat mass.[2] According to Hong and colleagues, "Although weight loss may be beneficial among the obese elderly with low POP concentrations, weight loss in the obese elderly with higher serum concentrations of POPs may carry some risk."[2]

It is believed that the reason for this obesity paradox is the sequestering of lipophilic chemicals known as persistent organic pollutants (POPs) and other environmental toxins in adipose tissue.[2] Thus, the toxins are prevented from reaching and harming critical organs.[2] However, they are not completely harmless while stored in the adipose tissue. POPs can alter adipose tissue function, thereby increasing inflammation in the fat tissue.[3]

In the first part of this article, I will address the evidence that weight loss causes the release of toxins from adipose tissue and the damaging effects this can have on certain aspects of health. In the second part, I will delve further into the implications of toxin

release during weight loss as well as explain in more detail the ways that the US BioTek Comprehensive Urinary Metabolic Profile (organic acids test) with an Environmental Pollutants Profile can be used to detect toxin levels in people planning to undergo a weight loss regimen.

The Toxic Burden of Weight Loss

A substantial amount of evidence indicates that during weight loss, a heavy burden of toxins is released from adipose tissue into the bloodstream. For example, researchers studied serum samples from 63 participants before and one year after bariatric surgery.[4] The samples were analyzed for POPs such as organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs), and brominated flame retardants (BFRs). After a year, the subjects lost a mean of more than 70 pounds. There was a 46.7% to 83.1% rise in concentrations of all the measured POPs in serum during the study.

Another trial investigated the effect of weight loss on serum POP levels in 94 obese adolescents.[5] During the study, the participants lost 4 to 42 kg of body weight. Due to this weight loss, there was a pronounced increase in serum levels of most POPs. Serum levels of POPs increased by 1% to 3.5% per kilogram of weight loss.

A third study of 1,099 adults investigated the association between self-reported weight change over 1 and 10 years and serum levels of seven POPs.[6] Serum levels of most POPs were greater in participants with long-term weight loss and lower in subjects who gained weight over time. The increase in POPs associated with weight loss through dieting is more pronounced in people losing visceral adipose tissue compared to subcutaneous fat.[7]

The release of POPs from adipose tissue leaves these toxins free to roam about the body, eventually being deposited in places where they can potentially do harm such as the pancreas, liver, heart, and brain. (I will address the effects of POPs on these organs in Part Two of this article.) Breast and ovarian health may also suffer after blood-borne POPs are deposited in those areas.[8] Furthermore, there is indication that the release of POPs from adipose tissue during weight loss and subsequent deposition in other parts of the body could interfere with energy metabolism, thus leading to regaining of the weight previously lost.[9]

Other environmental toxins such as phthalates and BPA are known as obesogens and can cause weight gain.[10] These toxins can also accumulate in adipose tissue.[11] BPA acts like a POP and is often grouped with POPs in discussions involving these toxins.[12]

Could Weight Loss Cause Cancer?

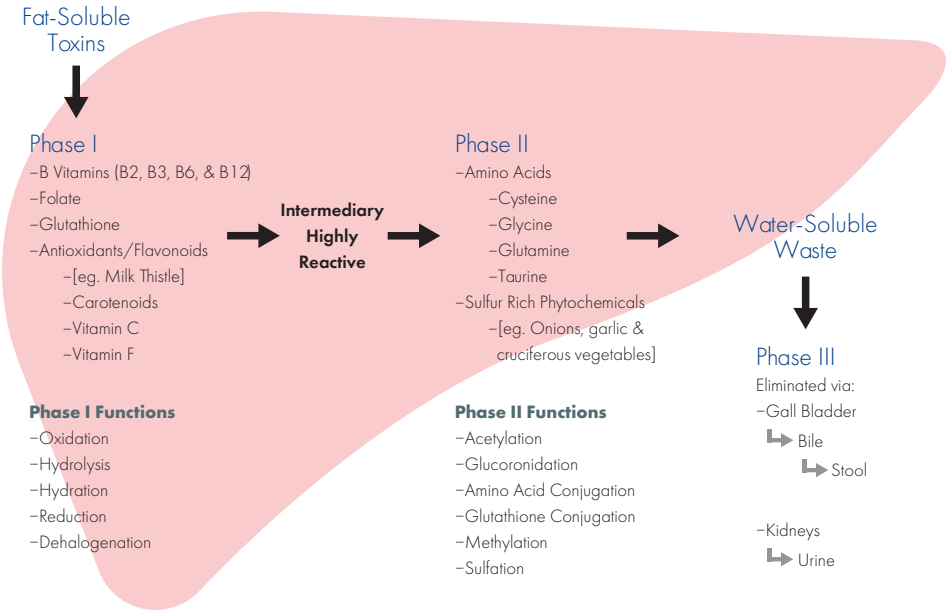
Accumulating evidence strongly suggests that POPs play a role in cancer development. We know that breast cancer patients who have undergone weight loss have high levels of POPs in their serum.[13] In addition, at the developmental stages of breast cells, exposure to environmental toxins with hormonal effects may increase the incidence of breast cancer later in life.[14]

Several studies have also established a possible connection between exposure to polychlorinated biphenyls and increased risk of developing breast, prostate, testicular, ovarian, and uterine cancers as well as colorectal polyps and colon cancer.[8,15]

POPs are known to act as estrogen mimics by binding and activating the estrogen receptor.[16] As an environmental estrogen, they are therefore prone to be cancer-causing chemicals. Furthermore, uterine leiomyomatas, endometriosis, and ovarian cysts are all associated with POPs.[17]

Given the known role of POPs in cancer development, by allowing these hormone-disrupting chemicals to lodge in various organs of the body, weight loss could potentially lead to carcinogenesis. This is one of the reasons why ordering the US BioTek Comprehensive Urinary Metabolic Profile (organic acids test) with an Environmental Pollutants Profile is critical to the health of anyone undergoing a weight loss program.

Phase I, II, & III Detoxification



Case Study: 58-Year-Old Patient Develops Ovarian Cancer After Vigorous Weight-Loss Program

When a 58-year-old patient whom I will call Laura came to my clinic for the first time, she had already implemented a weight-loss program that resulted in a considerable decline in adipose tissue. Unfortunately, prior to losing the weight she had not determined her toxic burden. Nor had she implemented a detoxification protocol. Nine months after losing the weight, she received a diagnosis of ovarian cancer. Laura arrived at my clinic after this diagnosis.

I immediately ordered Laura a US BioTek Comprehensive Urinary Metabolic Profile with an Environmental Pollutants Profile. The environmental pollutants panel indicated high levels of phthalates, styrene, and xylene as well as likely sources where she had been contaminated with those pollutants. It also indicated these toxicities were causing mitochondrial dysfunction. I have placed Laura on a detoxification protocol to eliminate high levels of the indicated toxins. This protocol includes supplementation with glycine, fiber to help bind and eliminate toxins, mitochondria support with PQQ, NAD and Mitoquinone (MitoQ®), while supporting all phases of detoxification I, II and III (see diagram) with a combination detoxification powder; encouraging sweating via sauna or exercise followed by a tepid shower to prevent reabsorption of toxins released in sweat. Her regimen also includes mitochondrial support. In addition, she is undergoing chemotherapy/radiation.

It would have been ideal if Laura could have been tested for environmental pollutants prior to undertaking weight loss. She could have then implemented a detoxification regimen to prevent her organs from becoming overwhelmed with weight-loss related toxic burden. This could have tipped the scales, leading to her health crisis.

References:

1 Lee YM, Kim KS, Jacobs DR Jr., et al. Persistent organic pollutants in adipose tissue should be considered in obesity research. *Obes Rev.* 2017 Feb;18(2):129-39.

2 Hong NS, Kim KS, Lee IK, et al. The association between obesity and mortality in the elderly differs by serum concentrations of persistent organic pollutants: a possible explanation for the obesity paradox. *Int J Obes* (Lond). 2012 Sep;36(9):1170-5.

3 La Merrill M, Emond C, Kim MJ, et al. Toxicological function of adipose tissue: focus on persistent organic pollutants. *Environ Health Perspect.* 2013 Feb;121(2):162-9.

4 Jansen A, Polder A, Müller MHB, et al. Increased levels of persistent organic pollutants in serum one year after a great weight loss in humans: Are the levels exceeding health based guideline values? *Sci Total Environ.* 2018 May 1;622-3:1317-26.

5 Malarvannan G, Van Hoorenbeeck K, Deguchtenaere A, et al. Dynamics of persistent organic pollutants in obese adolescents during weight loss. *Environ Int.* 2018 Jan;110:80-7.

6 Lim JS, Son H-K, Park S-K, et al. Inverse associations between long-term weight change and serum concentrations of persistent organic pollutants. *International Journal of Obesity.* 2011;35:744-7.

7 Dirinck E, Dirtu AC, Jorens PG, et al. Pivotal Role for the Visceral Fat Compartment in the Release of Persistent Organic Pollutants During Weight Loss. *J Clin Endocrinol Metab.* 2015 Dec;100(12):4463-71.

8 Roveda AM, Veronesi L, Zoni R, et al. Exposure to polychlorinated biphenyls (PCBs) in food and cancer risk: recent advances. [Article in Italian, Abstract in English.] *Ig Sanita Pubbl.* 2006 Nov-Dec;62(6):677-96.

9 Cheikh Rouhou M, et al. Adverse effects of weight loss: Are persistent organic pollutants a potential culprit? *Diabetes Metab.* 2016 Sep;42(4):215-23.

10 Yin L, Shengyang Yu K, Lu K, Yu X. Benzyl butyl phthalate promotes adipogenesis in 3T3-L1 preadipocytes: A High Content Cellomics and metabolomic analysis. *Toxicology in Vitro.* 2016;32:297.

11 Lind PM, Roos V, Rönn M, et al. Serum concentrations of phthalate metabolites are related to abdominal fat distribution two years later in elderly women. *Environ Health.* 2012 Apr 2;11:21.

12 Haffner D, Schecter A. Persistent Organic Pollutants (POPs): A Primer for Practicing Clinicians. *Curr Environ Health Reports.* 2014;1(2):123-31.

13 Pestana D, Faria G, Sá C, et al. Persistent organic pollutant levels in human visceral and subcutaneous adipose tissue in obese individuals—depot differences and dysmetabolism implications. *Environ Res.* 2014 Aug;133:170-7.

14 Bachelet D, Verner MA, Neri M, et al. Breast Cancer and Exposure to Organochlorines in the CECILE Study: Associations with Plasma Levels Measured at the Time of Diagnosis and Estimated during Adolescence. *Int J Environ Res Public Health*. 2019 Jan 18;16(2). pii: E271.

15 Lee YM, Kim SA, Choi GS, et al. Association of colorectal polyps and cancer with lowdose persistent organic pollutants: A case-control study. *PLoS One*. 2018 Dec 6;13(12):e0208546.

16 Hunter DS, Hodges LC, Eagon PK, et al. Influence of exogenous estrogen receptor ligands on uterine leiomyoma: evidence from an in vitro/in vivo animal model for uterine fibroids. *Environ Health Perspect*. 2000 Oct;108 Suppl 5: 829-34.

17 Lambertino A, Turyk M, Anderson H, et al. Uterine leiomyomata in a cohort of Great Lakes sport fish consumers. *Environ Res*. 2011 May;111(4):565-72.