The inviolable assumption that obesity is simply a thermodynamic problem of calories in/calories out is being dissembled by the science linking environmental toxins to obesity and diabetes. Exposure to environmental toxins in the absence of increased caloric intake induces weight gain and insulin resistance. Stated simply, toxins are an invisible, unappreciated cause of obesity and diabetes.
Clearly, our sedentary, high-stress lifestyle and our highlyglycemic, trans fat– and saturated fat–rich, low-fi ber, phytonutrient-poor diet contributes to the epidemic of diabetes and obesity.

But the increasing burden of environmental toxins, including persistent organic pollutants and heavy metals, can no longer be ignored as a key etiologic factor in the epidemic of obesity and diabetes, or what should be called “diabesity,” the continuum of metabolic dysfunction mild insulin resistance to end-stage diabetes.

Diabesity is our modern plague. The many conditions that exist under the umbrella of diabesity affect more than one billion people worldwide. More than 100 million Americans (including 50% of those over age 65) suffer from the condition in its various forms.

The Centers for Disease Control and Prevention estimates that nearly 24 million Americans (including 23% of those over age 60) suffer from type 2 diabetes.1 More than 50% of people over 65 have diabesity. Nearly 6 million diabetics are undiagnosed.

Diabetes and the resultant cardiovascular mortality is a global problem outpacing infectious disease as a cause of morbidity and mortality in developing countries.2 It now affects more than 240 million people worldwide and is projected to affect 380 million by the year 2030. This is nearly 10 times the number of people affected by HIV/AIDS worldwide.3 Diabesity has been identified as one of the leading causes of cardiovascular disease,4 dementia5 and cancer.6 Consider this: From 1983 to 2008, the number of people in the world with diabetes increased seven-fold, from 35 million to 240 million. Shouldn’t our main question be why this is happening, rather than what new drug we can find to treat it?
Diabesity also places a large economic burden on our society.

The direct and indirect costs of diabetes in America in 2007 amounted to $174 billion. The cost of obesity is also significant, amounting to $113 billion every year. During the past 10 years, these two conditions cost America alone a total of $3 trillion.

That’s three times the estimated cost of fixing our entire health care system! Between 2009 and 2034, the number of people with diagnosed and undiagnosed diabetes will increase from 23.7 million to 44.1 million in the United States. During the same period, annual diabetes-related spending is expected to increase from $113 billion to $336 billion (2007 dollars).7

The parallel increase in our environmental toxic burden and obesity must be addressed on a policy level and in the clinical treatment of diabesity. Environmental toxins interfere with glucose and cholesterol metabolism and induce insulin resistance.8

Toxins induce obesity and insulin resistance through multiple other mechanisms, including inflammation, oxidative stress, mitochondrial injury, altered thyroid metabolism, and impairment of central appetite regulation.9

The most recent example of how toxins induce obesity is the dramatic increase in obesity in babies. In 2006, scientists at the Harvard School of Public Health found that rates of obesity in infants less than 6 months old have risen 73% since 1980.10 This epidemic of obesity in 6-month-olds is not related to diet or lack of exercise. Babies live on breast milk or formula and love. They don’t say, “Hey, Mom, take me out for a 1200-calorie McDonald’s breakfast or a giant tub of buttered popcorn.” Clearly, watching too much television is not a risk factor. So what is the cause? It appears it may be the load of environmental toxins in their little bodies.

Mounting evidence points to a unique and unappreciated trigger for obesity—exposure to small traces of environmental chemicals in the environment. The average newborn has 287 chemicals in the umbilical cord blood, 217 of which are neurotoxic.
The chemicals these infants are exposed to include pesticides, phthalates, bisphenol A, flame retardants, and heavy metals such as mercury, lead, and arsenic. These chemicals have a broad range of negative effects on human biology. They are neurotoxic, carcinogenic, and now it seems, obesogenic.

A 2008 study in JAMA found that bisphenol A, a petrochemical that lines water bottles and canned food containers, increases the risk of diabetes, heart disease, and abnormal liver function. Data from the government's National Health and Nutrition Examination Survey 1999-2002 found a very striking correlation between blood levels of six common persistent organic pollutants (petrochemical toxins) and diabetes. Those who had the highest serum levels of pollutants had a dramatically higher risk for diabetes.

Studies of Air Force veterans of the Vietnam War found that those who had been exposed to Agent Orange (dioxin) had a much higher risk for diabetes. Canadian Aboriginals and Great Lakes sport fishermen both have higher rates of diabetes from eating contaminated seafood.

The ubiquitous exposure to persistent organic pollutants (POPs) such as p,p'-diphenyldichloroethene (DDE), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) has been linked to obesity and diabetes. Toxins interfere with and slow metabolism and contribute to weight gain and diabetes. A larger population study published recently in Environmental Health has confirmed this finding. In the study, higher levels of organochlorine pesticides were found in those who suffered from diabetes. Heavy metals such as mercury, lead, and arsenic also cause diabesity. A recent article in JAMA linked arsenic exposure to increases in the risk of type 2 diabetes. Other data link mercury from fish consumption, dental amalgams, and vaccines through multiple mechanisms including enzymatic disruption, impaired glucose transport, oxidative stress, induction of inflammatory cytokines, and mitochondrial injury. This suggests a new model of potential treatment for diabetes and obesity. A comprehensive
Toxins induce insulin resistance by interfering with the function of a class of nuclear receptors called PPARs (peroxisome proliferator-activated receptors) needed for optimal insulin function, glucose control, fatty oxidation, and regulation of inflammation. 

Using new techniques of genetic and metabolic analysis, researchers have shown how toxins cause increases in glucose, cholesterol, and fatty liver. While observational data are suggestive, newer experimental data confirm causality between environmental toxins and obesity. New evidence shows that weight gain can occur in the absence of excess calorie intake. In a recent study, rats given toxic chemicals gained weight and increased their fat storage without increased caloric intake or decreased exercise. In 6 months, these rats were 20% heavier and had 36% more body fat than rats that had not been exposed to those chemicals. This is no longer something that can be ignored. The data are clear enough. The body burden of persistent organic pollutants (plastics, pesticides, industrial petrochemicals) and heavy metal toxins must be addressed in public health policy and in any treatment program for diabesity.

This is not a fringe idea of radical environmentalists. The National Institutes of Health, the US Food and Drug Administration, the Environmental Protection Agency, and the National Academy of Sciences recently convened to examine this new phenomenon of obesogens—toxins that cause obesity.

The sustainability of our environment is directly connected to the sustainability of the health of the global population. The connection between toxins and their effects on metabolism are well established. The link between our addiction to energy,
industrialization, and the millions of pounds of chemicals and heavy metals released every year into our environment and the epidemics of chronic illness from which we now suffer should make us all stop and think about how we live and the choices we make every day in the food we eat and the products we purchase.

There is a role for a new ecological intelligence in the production and consumption of products and services.24

TREATMENT OF TOXIN BURDEN

A few simple clinical strategies can help reduce the body burden of toxins. Dietary phytonutrients, antioxidants, and nutritional supplements that induce phase 1 and phase 2 detoxification combined with exercise and hyperthermic therapy are safe and effective strategies for reducing the body burden of toxins.

Medical therapy with heavy metal chelators may also be necessary to lower the body burden of mercury, lead, arsenic, and other toxic metals.

DIETARY SUPPORT FOR DETOXIFICATION

Plants have evolved many protective defenses against pests and infection in the form of phytonutrients. These defenses are better developed in organic foods because of selective adaptive pressures necessary to resist pests, infections, and variable climate conditions. Organic food provides higher concentrations of protective detoxifying, antioxidant, and antiinflammatory phytonutrients.

Specific foods contain higher concentrations of unique phytonutrients such as glucosinolates or catechins, which support detoxification.

The most effective foods to support detoxification include cruciferous vegetables (cabbage, broccoli, collards, kale, Brussels sprouts, Chinese cabbage, bok choy, arugula, radish, wasabi,
watercress, kohlrabi, mustard greens, rutabaga, and turnips), curcuminoids (turmeric and curry), green tea (increases glutathione-S-transferases), and sulfur-containing proteins and foods (eggs, garlic, and onions).

SUPPLEMENTS TO ENHANCE DETOXIFICATION

Vitamins, minerals, amino acids, and phytonutrients from the foods noted above are needed as cofactors for phase 1 and phase 2 detoxification and to protect against the inflammatory, oxidative stress, and mitochondrial injury induced by toxins.

The most critical endogenous molecule for detoxification is glutathione. Optimal methylation is required to generate glutathione through the methylation/transsulfuration cycle, making B6, folic acid, and B12 essential. Zinc and selenium also facilitate detoxification as cofactors in the enzymes metallothionein and glutathione peroxidase.

N-acetyl-cysteine increases glutathione and historically has been used to treat depleted glutathione and liver failure from acetaminophen overdose. Milk thistle has long been used in liver disease and increases glutathione. Buffered ascorbic acid (vitamin C) is also critical in detoxification and has been associated with a reduction in lead levels.

HYPERTHERMIC THERAPY (SAUNAS)

Sauna and heat therapies are an ancient method of cleansing.

The Native American sweat lodge was a tool for physical and spiritual purification. The Environmental Protection Agency has
shown that sauna therapy increases excretion of heavy metals (lead; mercury; cadmium; and fat-soluble chemicals such as PCBs, PBBs, and HCBs).27

CONCLUSION

Global awareness of climate change’s impact on the environment must be linked to awareness of the impact of environmental toxins on human health and the global epidemic of obesity (>1 billion) and diabetes (280 million). The burden of social, economic, and personal suffering may be mitigated through coordinated global policy changes that reduce exposure to environmental toxins combined with funding for further investigation into effective diagnosis of and treatment for elevated body burdens of persistent organic pollutants and heavy metal toxins in individual patients.

These toxins are not only obesogens but also carcinogens and autogens, which trigger autoimmune and inflammatory disorders and cardiovascular mortality. They are also neurotoxic and have been linked to increasing rates of depression, autism spectrum disorders, attention deficit disorder, and dementia. Environmental medicine can no longer be the neglected step-child of modern medicine; it must come to the forefront of clinical care for us to effectively treat the global burden of chronic disease.

REFERENCES


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