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Epidemic of Obesity Link to Environmental & Food Toxins

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Abstract

Almost every American is aware of, or can relate to, the rise in obesity over the past several decades. There is a clear correlation between this epidemic of obesity and exposure to toxins that disrupt the internal systems efficient flow of functions. The link has become so well-known that there are specific toxins referred to as "obesogens" and there are scientific papers of profound depth and detail on explicit chemicals, or endocrine disruptors, and each one's relation to obesity. Regardless of this information, the majority of the public is unaware and not well-informed about what could be damaging their body's systems in regards to their environment and health issues. Fortunately, as a result of the recent work by medical professionals to the likes of Dr. Mark Hyman the amount of evidence is being compiled into scientific studies and presented to the public in a way that would be better understood or more easily attainable. However, there are still many questions to be answered about the detrimental links amongst obesity, environmental toxins, and dietary toxins.

Introduction

The prevalence of toxins in the daily environment of people today makes it difficult for anyone to avoid toxic exposure multiple times, if not at least once, throughout the day. This is best understood when recognizing that people are exposed to environmental toxins through the food and water, alone, as methylmercury, arsenic, persistent organic pollutants (POPs) such as bisphenol A (BPA) and phthalates. To date, there is evidence linking BPA to increases in the risk of diabetes, heart disease, and abnormal liver function, as well as a striking correlation between POPs, arsenic, and obesity. Many people are unaware or do not find any reason to be concerned about how they are being exposed, nor question or research the possible effects of these toxins.

A large part of this exposure is coming from our environment, as 'even today in the age of chronic diseases there remains an important connection between population health and the built environment" (Perdue et. al., 2003). As the environment has developed more over the decades since the Industrial Revolution, there has also been a parallel increase in the prevalence of adult obesity, as well as an increase in body mass index (BMI), childhood obesity, diabetes, and medical costs for obesity. Obesity is a complex chronic illness that when tainted by toxins involves the neuro-endocrine-immune system, and occurs from a mix of genetic, environmental and lifestyle factors. Obesity has become a big issue as the "prevalence of obesity more than doubled between 1980 and 2008 worldwide, so that by 2008, 10% of men and 14% of women in the world were obese (body mass index, BMI $\geq$ 30 kg/m^2) and at least 2.8 million people die each year as a result of being overweight or obese…the estimated annual medical costs of obesity in 2008 were $147 billion in the USA alone" (Sharpe and Drake, 2013). With this illness costing people so much money and affecting so many, it is critical to know how people are exposed and this happens in two primary ways – through the environment as external toxins, and the gut as internal toxins that breakdown metabolizing products. Thus, this paper seeks to determine if the epidemic of obesity can be linked to an increase in toxins, such as environmental pollutants and nutritional deficiencies.

Methods

The search methods used to retrieve the over 15 credible resources included in this paper were obtained through internet search engines, academic databases, print and web literature. The search engines and academic databases utilized include LIRN, Google Scholar, ProQuest and Gale, in which the following specific terms were explored in a multitude of combinations: obesity, nutrition deficiency, and chronic diseases in relation to (searched with "AND") environmental toxins, environmental pollutants, genetically altered or genetically modified, as well as those nutrition, organic foods, and genetically modified organism (GMO). Of the tens of thousands of sources retrieved, those selected include only full-text reports published since 1990 in English that did not require payment.

Results

Until the latter half of this past decade, there was little evidence available about the effects of toxins on the gut microbes, which is hard to turn a blind eye to considering "gut microbes outnumber human cells by a factor of 10" (Snedeker and Hay, 2012). This makes it hard to identify what specific toxins, like POPs, are dangerous and at what point they become toxic to humans. Thus, there are no studies with information to prove how variations of gut ecology affect human absorption, distribution, metabolism, and excretion (ADME) capacity regarding environmental toxins. But there are a number of studies that link the groups of environmental toxins to specific disorders and Snedeker and Hay (2012) compile a great amount of evidence to suggest "that microbes may affect obesity and diabetes by altering the ADME of environmental chemicals." Their review did not, however, include the work of Dr. Mark Hyman who sensibly and scientifically is able to put the pieces of this puzzle together. Though Dr. Hyman (2010) openly acknowledges how "clearly, our sedentary, high-stress lifestyle and our high glycemic, trans fat- and saturated fat-rich, low-fiber, phytonutrient-poor diet contributes to the epidemic of diabetes and obesity," he emphasizes that the "environmental toxins interfere with glucose and cholesterol metabolism and induce insulin resistance" then continues to explain the biological issues of these toxins as inducing "obesity and insulin resistance through multiple mechanisms, including inflammation, oxidative stress, mitochondrial injury, altered thyroid metabolism, and impairment of central appetite regulation."

In another study done a few years earlier, Dr. Hyman (2007) identified environmental toxins as causers of interference with metabolism, overloading hepatic detoxification systems, disrupting central weight-control systems, promoting insulin resistance, altering circadian rhythms, activating the stress response, interfering with thyroid function, increasing inflammation, damaging mitochondria, and leading to obesity. This study more importantly notes the internal toxins of the gut that occur due to the breakdown of metabolizing products. The internal toxins are a part of our refined diet of today that "places an extra burden on detoxification systems through excessive consumption of sugar, high-fructose

corn syrup (the two most important causes of elevated liver function tests), trans fatty acids, alcohol, caffeine, aspartame, foods made with genetically modified organisms (GMOs), and the various plastics, pathogens, hormones, and antibiotics found in our food supply" (Hyman, 2007). Environmental toxins that are parts of the standard American diet (SAD) of today including heavy metals, like lead and mercury, and chemicals, such as dopamine. The connection was made by Sharpe and Drake (2013) when noting that because the main factor for obesity is the "'Western' fast-food, high fat, energy-rich diet" and people are exposed to obesogens via this diet, then "consumption of this diet would increase the risk of becoming obese at the same time as increasing exposure to environmental obesogens." This makes it hard to differentiate how much more one factor may play over the other, but emphasizes the importance on information on both means of exposure through the environment and the food.

Discussion

Though other studies observe a large number of influential factors that play a role in whether or not a person develops obesity, like chronic reductions in sleep, stress, and viral exposure, the global relevance of chemical exposure via the environment and the 'Western'-ized food to obesity has caused these toxins to become well-known as "obesogens." According to Grün and Blumberg (2009), obesogens "can be defined as chemicals that inappropriately alter lipid homeostasis to promote adipogenesis and lipid accumulation." The variety of resources collected present not only a correlation between environmental and food toxins to obesity, but go into great detail and explanation to identify obesogens and explore how they may be affecting individual health. A thoroughly written minireview by Grün and Blumberg (2009) cleverly compiles a multitude of studies regarding plausible links to obesogens and categorizes the specifics by the susceptible pathways (metabolic sensors, sex steroid hormones, hypothalamic-pituitary-adrenal axis, neurological disorders and pharmaceutical treatments) and endocrine disruptors (BPA, xenoestrogens, organotins, perfluorooctanoic acid (PFOA), and phthalates). There are, however, even more studies with depth and detail on explicit chemicals, or endocrine disruptors, and each one's relation to obesity that were overlooked; though, vital in expanding upon our understanding of this issue. Recent examples of this are the works of Elobeid and Allison (2008) with endocrine disruptors, and Aitlhadj et. al. (2011) with Caenorhabditis elegans (C. elegans), dopamine and heavy metals.

Yet, "in spite of the accumulating substantial evidence for an obesity epidemic, our knowledge about the effect of environmental chemicals on weight gain and the magnitude of human or wildlife exposure to these chemicals is limited" (Elobeid and Allison, 2008). Some missing puzzle pieces that would greatly expand upon these studies include but are not limited to determining the role a poor diet (or the SAD diet, specifically) plays in obesogen exposure, how much of past or current toxin exposure levels pose a risk to humans, identify predictive biomarkers of impaired obesogenic chemical ADME, if the circulation of environmental chemicals is affected by variable populations of gut microbes, and assessing interactions between developmental obesogens and microbiota such as intergenerational effects.

Conclusions & Recommendations

In the words of Dr. Hyman (2010), 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." The results of missing puzzle pieces discussed above would allow for a more personalized approach in the prevention and treatment of obesity, as well as diabetes. For a larger impact to occur around the world, though, it would be most beneficial to implement carefully coordinated changes into a global policy that reduces "exposure to environmental toxins combined with funding for further investigation into effective diagnosis of and treatment for elevated body burdens" of environmental toxins in individuals (Hyman, 2010). This level of involvement would ease the burden of social, personal, and economic suffering for many countries, as well as permit clearer identifications of obesogens roles as carcinogens, autogens, and neurotoxins. These various roles of obesogens would not only provide better insight into the effects of environmental and dietary toxins, but allow for their influences on other diseases, such as dementia, depression, attention deficit disorder (ADD), and autism spectrum disorders, to be understood by professionals seeking to aid patients in alleviating their symptoms and signs.

The mix of these resources establishes a neglected issue that "can no longer be the neglected step-child of modern medicine" and demonstrates the need for further research to establish a solid foundation around which individual care, or health and wellness, plans can be developed (Hyman, 2010). The functional medicine work being done by medical and/or health professionals, such as Dr. Hyman, that are addressing treatment-resistant and complex chronic disease, like obesity, may provide a roadmap on how to tackle this global burden within clinical care.

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