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Can Air Pollution Biologically Hinder Efforts to Lose Body Weight?

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As the incidence of obesity increased sharply, controlling body weight is one of the most important public health concerns in the 21st century. However, losing weight is often difficult, and maintaining it is even more challenging. Thus, development of strategies for successful weight loss has been on high priority in research agendas.

Chin et al. [1] and Ustulin et al. [2] conducted studies involving users of a popular smartphone application designed for weight loss. They reported the value of smartphone application for successful weight reduction and maintenance among overweight or obese individuals and also implicated the importance of climate variables such as temperature and wind speed on weight loss [1,2]. In this issue of *Diabetes & Metabolism Journal*, they further evaluated whether air pollution could affect efforts to lose weight among a subset of the original cohort [3].

This study was performed in two stages. First, the data from a cohort registered to the smartphone application in 10 large cities worldwide was linked to the annual air pollution levels that were measured as particulate matter (PM) 10 and PM2.5. Second, the finding from the first-stage analyses was validated using daily air pollution data in the United States. Their analyses revealed the possibility that air pollution may hinder the efforts to lose body weight. A stronger effect was observed with PM2.5 than with PM10.

Now, the question is whether this association can be explained by any biological mechanism. The answer would be "Yes," because there is growing evidence that exposure to various environmental pollutants can contribute to the development of obesity [4]. They are called as obesogens and act at low doses to which humans are usually exposed in daily life [4]. A wide range of chemicals such as pesticides, herbicides, plastics, detergents, flame retardants, and personal care products are suspected to be obesogens, and the list is rapidly growing [4]; various obesogens are attached to gaseous and particulate outdoor air phases such as PM10 and PM2.5 [5]. Many known or suspected obesogens are classified as endocrine disrupting chemicals (EDCs). Similar to EDCs, *in utero* and/or neonatal period is the most sensitive period to obesogens and these effects can be transmitted to their descendants [6].

Obesogens have numerous mechanisms of action, including increasing the number of adipocytes, increasing the ability to store fat, and modulating hormones that regulate appetite, satiety, and energy metabolism [7]. Therefore, the exposure to obesogens can hinder an individual's efforts to lose weight by limiting calorie intake and increasing physical activity, which biologically supports the findings of Ustulin et al. [3].

Currently, obesogens are considered an emerging public health concern. However, one important aspect is often disregarded by researchers in the field of obesogens. In the modern society, the role of healthy adipose tissue has become important because it is impossible to live without being exposed to pollutants; adipose tissue can provide a relatively safe storage site for lipophilic chemicals with long half-lives [8]. In fact, obesogens can contribute to secure sufficient healthy adipose tissue through promoting adipogenesis.

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Adipose tissue expansion is featured by both hypertrophy (increase in cell size) and hyperplasia (increase in cell size). Hypertrophic adipose expansion is associated with harmful phenomena such as proinflammatory cytokine release and impaired insulin sensitivity, but hyperplasic adipose expansion is linked to beneficial phenomena such as decreased proinflammatory cytokine release and improved insulin sensitivity [9,10]. For example, metabolically healthy obese persons have a higher proportion of relatively small adipocytes in the adipose tissues, suggesting hyperplasia-dominant obesity [11]. Similarly, the antidiabetic drugs, thiazolidinedione derivatives, promote adipogenesis by acting as peroxisome proliferator-associated receptor gamma (PPARγ) ligands [12].

Obesogens promote adipogenesis via altering the programming of fat cell development [6]. Many obesogens are PPARγ agonists [13]. Although obesogens can cause various dysfunctions of adipocytes [14], it would be difficult to see that the increased adipogenesis caused by exposure to obesogens is harmful if obesogens can contribute to the expansion of healthy adipose tissue.

Finally, the release of lipophilic chemicals from the adipose tissue during weight loss has also been neglected by researchers, clinicians, and the general public. Weight loss has become an obsession in the modern society, but the release of such chemicals may counteract the benefits of weight loss [8]. The negative effects of lipophilic chemicals released from the adipose tissue during weight loss, such as alterations in resting metabolic rate and thyroid hormone, have been reported [15,16]. The null effect of an intensive lifestyle intervention focusing on weight loss in a randomized controlled study among overweight or obese patients with type 2 diabetes mellitus [17] may be partly explained by the dynamics of lipophilic chemicals in the adipose tissue [8]. Understanding the complicated interrelationships between the pollutants and the adipose tissue can present a new perspective to the field of weight management.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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