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How Nutrition might Modify the Possible Effects of Air Pollution on Cardiovascular Diseases' Risk?

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Air pollution is a heterogeneous and complex mixture of gases, liquids, and particulate matters. Over the last decade, however, a growing body of epidemiological and clinical evidence has led to a heightened concern about the potential deleterious effects of ambient air pollution on the risk of cardiovascular diseases (CVDs). Recent evidences from the World Health Organization (WHO) revealed that China and India are two of the most affected countries in terms of indoor and outdoor air pollution exposures with approximately 6.5 million associated deaths each year [1]. The health effects of pollutant exposure not only pose a problem for the individual, but also contribute to the global burden of disease [2].

Several environmental air pollutants including carbon monoxide, oxides of nitrogen, sulfur dioxide, ozone, lead, and particulate matter [3] are associated with increased hospitalization [4]. It is proposed that even brief exposures to air pollution can be associated with increase in cardiovascular mortality [5, 6], particularly in susceptible populations [7]. In particular, PM was associated with CVDs mortality, development of chronic CVDs conditions, including hypertension and ischemic heart disease, as well as acute events like myocardial infarction [8].

Circulating nutrients, toxicants, and their metabolites can modulate vascular responses that can either be pro- or anti-atherogenic. One of the key events in the progression of atherosclerosis is endothelial cell dysfunction. It was documented that exposure to PM air pollution can disrupt the vasculature, resulting in endothelial cell dysfunction [8]. For instance, coplanar polychlorinated biphenyls (PCBs), a class of persistent organic pollutants (POPs) increase downstream inflammatory responses by binding to the aryl hydrocarbon receptor, which can up-regulate the transcription of cytochrome P450 1A1 (Cyp1a1) gene [9, 10]. Cyp1a1 is primarily involved in xenobiotic detoxification; however, in the presence of certain PCBs, it results in the production of reactive oxygen species (ROS) via an uncoupling mechanism [11]. This production of ROS yields an increase in oxidative stress

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because of the imbalance in the cellular redox status [10]. This pro-oxidative cellular status contributes to a state of chronic inflammation, which is a hallmark of many diseases, including atherosclerosis, diabetes, and other metabolic disorders [12]. More recent evidences suggest that a dioxin-like pollutant exposure, e.g., PCB-126, might up-regulates the enzyme flavin-containing monooxygenase 3 and subsequently increases circulating levels of trimethylamine-*N*-oxide, a biomarker strongly associated with CVDs [13].

It is well understood that a nutritious diet, rich in foods containing bioactive components such as polyphenols and anti-inflammatory fatty acids might protect against chronic inflammation, diabetes, metabolic syndrome, and CVDs [14]. Diets rich in these bioactive food components often contain greater levels of antioxidant and anti-inflammatory compounds. They consequently have the capacity to attenuate the inflammatory and oxidative properties of pollutant exposure [15- 21], for instance, diets similar to the Mediterranean diet, which are rich in omega-3 fatty acids, fruits, vegetables, and whole grains [22, 23].

On the other hand it is revealed that poor nutrition might increase CVDs risk. For instance, diets containing certain fatty acid profiles might promote the development of CVDs. For example, consumption of pro-inflammatory fatty acids such as linoleic and trans-fatty acids can contribute to endothelial cell dysfunction, a beginning stage of atherosclerosis [24, 25]. There is increasing experimental evidence that poor nutrition and pollutant exposure can interact and synergistically increase the risk of CVDs [26].

A number of food items are shown to be protective against CVDs. Curcumin, a principal component of the spice turmeric, has been demonstrated to suppress or inhibit the expression of tumor necrosis factor, as well as cyclooxygenase 2, the target of non-steroidal anti-inflammatory drugs [27, 28]. Additionally, consumption of table grapes that are rich in polyphenolic compounds, including resveratrol and anthocyanins, has been shown to attenuate systemic inflammatory responses, hepatic lipogenesis, and adiposity in mice fed with a high-fat diet [29]. Because many of the environmental pollutants are lipophilic and accumulate within the adipose tissue of exposed individuals, examining nutritional means to promote the excretion and reduction of body burden is also an important facet. It has been noted that individuals with a vegetarian or vegan diet tended to have lower amounts of organochloride body burden [30].

As a conclusion, environmental pollutant exposure is associated with numerous health complications ranging from metabolic syndrome and type 2 diabetes to CVDs [2]. While efforts to reduce and remediate pollution are underway, means of immediate protection at an individual level is of upmost importance [1]. Nutrition can have a drastic impact on the development of or protection against non-communicable diseases, such as those associated with pollutant exposure [1]. Western diets, abundant in processed foods, caloric content, and pro-inflammatory fatty acids may contribute to the development of obesity and cardiovascular diseases, they might also enhance the pollutant toxicity. Conversely, diets rich in bioactive food components, such as polyphenols and omega-3 fatty acids (i.e., the Mediterranean diet) are associated with reduced risk of inflammatory diseases and can attenuate the negative health effects of pollutant exposure [2]. Future studies examining the possible interaction between dietary foods or nutrients intakes and air pollutants in association with CVDs development might allow us to understand how certain foods may be used to alter specific epigenetic tags to reduce the risk of pollutant-associated diseases [2].

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