HHS Public Access

Author manuscript

Food Front. Author manuscript; available in PMC 2021 March 01.

Published in final edited form as:

Food Front. 2020 March ; 1(1): 13–14. doi:10.1002/fft2.6.

Healthful nutrition as a prevention and intervention paradigm to decrease the vulnerability to environmental toxicity or stressors and associated inflammatory disease risks

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Many non-communicable diseases, such as atherosclerosis and other cardiometabolic diseases, are defined as inflammatory diseases, which suggests that multifactorial interactions linked to exacerbated disease pathology include pro-inflammatory chemical and non-chemical stressor. Recent data suggest that genetic and lifestyle factors are independently associated with susceptibility to cardiovascular diseases. Thus, potential biological interactions between chemical and non-chemical stressors and buffers and other lifestyle factors will determine disease outcome. Chemical stressors include environmental pollutants with pro-oxidant and pro-inflammatory properties, such as air pollutants, both gaseous and particulate matter, and persistent organic pollutants such as dioxin-like polychlorinated biphenyls (PCBs). Many persistent organic pollutants also have an affinity for the aryl hydrocarbon receptor (AhR). AhR ligands lead to induction of cytochrome P450s and free radical formation, followed by inflammation. For example, dioxin-like PCBs can increase endothelial cell dysfunction, activation and inflammation (e.g., increased adhesion molecule expression and cytokine production).

It is unlikely that pollutant remediation alone will be sufficient to address the health impacts associated with chronic pollutant exposure, because most environmental toxicants such as persistent organic pollutants (POPs) are ubiquitous in the ecosystem. Thus, there is a need to explore preventive measures of environmental exposure and disease risk through positive lifestyle changes such as healthful nutrition. Our data indicate that plant-derived diets enriched with fiber or bioactive food components such as phytochemicals can prevent or decrease toxicant-induced inflammation. In fact, our data suggest that nutrition, or the type of diet we eat, can modulate environmental insults and disease outcome. For example, fats/oils high in omega-6 fatty acids can act as pro-oxidative and pro-inflammatory stressors, thus exacerbating an inflammatory response. Mechanistically, many environmental pollutants and pro-atherosclerotic nutrients/diets can both activate nuclear factor-κB (NF-κB) signaling leading to further increases in oxidative stress and inflammation. In contrast, many anti-inflammatory nutrients and/or bioactive phytochemicals can decrease inflammation by activating nuclear factor erythroid 2–related factor 2 (Nrf2) signaling. This is important, because foods rich in omega-3 fatty acids and plant-derived phytochemicals (e.g., polyphenols) or bioactive compounds can act as non-chemical buffers to reduce inflammation and thus prevent disease outcome associated with exposure to environmental

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toxicants. Our data, including metabolomic profiling, suggest that the pathology of inflammatory diseases initiated or caused by exposure to environmental pollutants (e.g., dioxin-like PCBs) is complex and may involve disturbances in the gut microbiota, liver and vascular tissues. The literature, including our data, also suggest that nutritional intervention can modulate the gut microbiota. This suggests that positive lifestyle changes, such as healthful nutrition, can have a disease-reducing impact already at the level of the gut microbiota. Of special interest are meaningful and translational approaches of prevention/intervention to lower disease outcome linked to complex interactions of chemical stressors to modify the outcome of cardiometabolic diseases, i.e., diseases linked to major causes of death especially in developed countries. Therefore, adopting healthy lifestyle changes, such as consuming healthy diets rich in plant-derived bioactive nutrients may reduce the vulnerability to diseases linked to environmental toxic insults. This nutritional paradigm in environmental toxicology requires further study in order to improve our understanding of the relationship between nutrition or other lifestyle modifications and toxicant-induced diseases.

Acknowledgements:

Supported in part by NIEHS/NIH grant P42ES007380 and the Kentucky Agricultural Experiment Station.

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