Science of the Total Environment 491–492 (2014) 11–16

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Modulation of persistent organic pollutant toxicity through nutritional intervention: Emerging opportunities in biomedicine and environmental remediation☆

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HIGHLIGHTS

- Nutrition modulates vulnerability to disease risks associated with exposure to environmental pollutants.
- Healthful nutrient polyphenols are protective by upregulating antioxidant and anti-inflammatory pathways.
- Polyphenols can be used in sensing, capture, and remediation technologies.
- Healthy nutrition may provide a cost-effective and environmentally friendly means of modulating environmental toxicity.

ARTICLE INFO

Article history:
Received 4 November 2013
Received in revised form 27 January 2014
Accepted 28 January 2014
Available online 13 February 2014

Keywords:
POP toxicity
Oxidative stress
Polyphenol
Antioxidant response
Nutrition
Sustainable remediation

ABSTRACT

Environmental pollution is increasing worldwide, and there is evidence that exposure to halogenated persistent organic pollutants (POPs) such as polychlorinated biphenyls can contribute to the pathology of inflammatory diseases such as atherosclerosis, diabetes, and cancer. Pollutant removal from contaminated sites and subsequent pollutant degradation are critical for reducing the long-term health risks associated with exposure. However, complete remediation of a toxicant from the environment is very difficult and cost-prohibitive. Furthermore, remediation technologies often result in the generation of secondary toxicants. Considering these circumstances, environmentally-friendly and sustainable remediation technologies and biomedical solutions to reduce vulnerability to environmental chemical insults need to be explored to reduce the overall health risks associated with exposure to environmental pollutants. We propose that positive lifestyle changes such as healthful nutrition and consumption of diets rich in fruits and vegetables or bioactive nutrients with antioxidant and/or anti-inflammatory properties will reduce the body's vulnerability to environmental stressors and thus reduce toxicant-mediated disease pathologies. Interestingly, emerging evidence now implicates the incorporation of bioactive nutrients, such as plant-derived polyphenols, in technologies focused on the capture, sensing and remediation of halogenated POPs. We propose that human nutritional intervention in concert with the use of natural polyphenol sensing and remediation platforms may provide a sensible means to develop primary and long-term prevention strategies of diseases associated with many environmental toxic insults including halogenated POPs.

1. Introduction

1.1. Combining environmental science and biomedical strategies to better protect against POP toxicity

Contaminant remediation and biological modulation of environmentally persistent pollutants are two crucial means of reducing the human health risks of Superfund chemicals/toxicants and related hazardous materials. The Superfund program, designed to clean up sites contaminated with a variety of hazardous substances, was formed

☆ Funding: This work was supported by the National Institute of Environmental Health Sciences at the National Institutes of Health [P42ES007380] and the University of Kentucky Agricultural Experiment Station.
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http://dx.doi.org/10.1016/j.scitotenv.2014.01.109

as the United States' response to this growing environmental and public health concern (EPA, 2013), but these pollutants are of worldwide concern. Although complete remediation of a hazardous site may continue to be an ultimate goal of human exposure risk reduction, it is important to design and implement other biologically relevant means of buffering against toxicant exposure before, during, and after remediation activities. New data now implicate the importance of an individual's nutritional status and the use of protective bioactive food components to decrease the overall toxicity of environmental pollutants to biological systems (Petriello et al., 2013). Nutrition is being substantiated as an important modulator of inflammatory and antioxidant pathways, especially with regard to environmental insults. Interestingly, novel research now shows that bioactive food components that are environmentally friendly can also be integrated into remediation technologies, which in turn allow for more sustainable, inexpensive, and effective pollutant removal and detoxification (Newsome et al., 2014a,b). Implementing bioactive molecules in both biomedical and environmental science settings, together, will allow for decreased overall body burden and human toxicity of a multitude of pollutants (see Fig. 1 for overview). In this review article, we discuss the relevant literature concerning this novel paradigm and point to the possible future uses of bioactive food components in healthful nutrition as well as their emerging uses in environmentally friendly remediation strategies for ultimately reducing human risks to persistent environmental pollutants.

Thus, this review is subdivided into 1) topics related to healthful nutrition (e.g., increased consumption of protective bioactive food components) as a means of reducing the disease vulnerability associated with exposure to environmental pollutants, and in particular pollutants found at Superfund sites, and 2) topics relevant to the emerging field pertaining to the utilization of environmentally friendly bioactive food components (e.g., polyphenols) to increase the efficiency and effectiveness for pollutant sensing, binding, and remediation efforts.

1.2. Exposure to POPs, risks for non-communicable diseases, and mechanisms of toxicity

Halogenated persistent organic pollutants (POPs), including polychlorinated biphenyls (PCBs) and trichloroethylene, are highly prevalent in the environment in soil, air, and water (Petriello et al., 2013). For example, a major source of human exposure to PCBs is through dietary intake of contaminated foods and through inhalation of airborne pollutants (Crinnion, 2011). Because most halogenated POPs, including PCBs, are lipid soluble, they easily accumulate in human tissues, leading to a perpetually increasing disease risk throughout a life span, especially in overweight populations (Kim et al., 2011). Exposure to halogenated POPs, and in particular pollutants that are ligands of the aryl hydrocarbon receptor (AhR), can lead to inflammatory events tied to diseases such as atherosclerosis, diabetes, obesity, cancer, etc. (Petriello et al., 2013). The pathology of many inflammatory diseases, especially of non-communicable or chronic diseases, develops over a long period of time and thus can be easily modulated by environmental exposures, specifically to persistent organic pollutants.

Mechanistically, an increase in cellular oxidative stress often precedes an inflammatory response (Schulze and Lee, 2005). PCBs, and in particular coplanar PCBs, have been shown to cause oxidative stress primarily through a cytochrome P450 (CYP1A1)-mediated uncoupling mechanism (Schlezinger et al., 2006). CYP1A1 induction allows for the detoxification of multiple xenobiotics, but when in the presence of PCB, can become inefficient and leaky (i.e., uncoupled) and produce detrimental reactive oxygen species (Schlezinger et al., 2006). A hallmark of the pathology of vascular diseases, including atherosclerosis, includes a change in the cellular redox status and a resultant increase in oxidative stress, which favors chronic and low level inflammation (Libby, 2012). Such changes in redox status and oxidative stress levels can be driven in part by pro-oxidative and pro-inflammatory environmental pollutants that are persistent and which can be easily stored in adipose tissue. Thus, an increased body burden of persistent environmental pollutants is a particular risk factor during obesity, a disease characterized by excessive adipose tissue (Kim et al., 2011). There is sufficient evidence that POPs contribute to inflammation by activating oxidative stress-sensitive transcription factors such as nuclear factor kappa-light-chain-enhancer of activated B cells (NFκB) (Hennig et al., 2002). For example, our studies suggest that PCBs, and in particular coplanar PCBs, can increase cellular oxidative stress and induce inflammatory parameters such as inflammatory cytokines, chemokines, and adhesion molecules in the vascular endothelium, which are metabolic events that foster an inflammatory response and atherosclerosis (Eske et al., 2013; Hennig et al., 2002; Lim et al., 2007; Majkova et al., 2009). Through these pro-inflammatory mechanisms, PCBs and related environmental toxicants have been correlated with an increased risk of multiple human chronic disease phenotypes including diabetes and heart disease (Carpenter, 2011; Goncharov et al., 2008; Silverstone et al., 2012; Uemura, 2012). Since many populations susceptible to toxicant-induced disease are often also afflicted by diet-induced diseases, future human studies and integrated risk assessments should better investigate the interaction between nutrition and toxicology (Hennig et al., 2012).

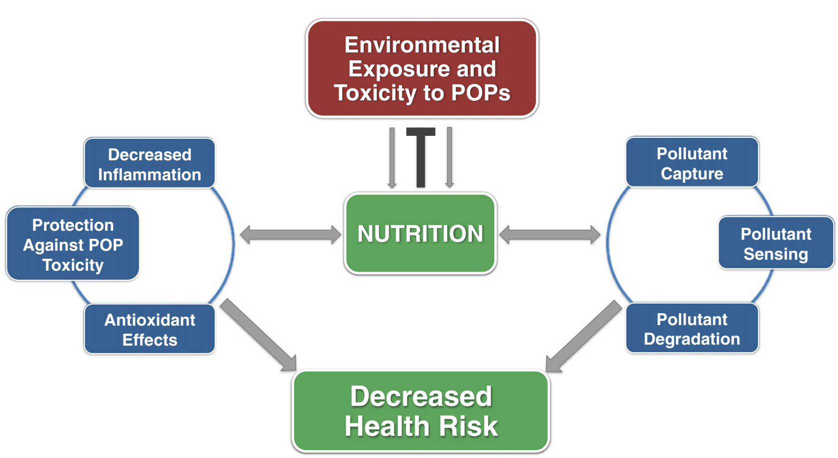

Fig. 1. Illustration of nutrition as 1) a modulator of health risks associated with environmental exposure and toxicity of persistent organic pollutants (POPs) and as 2) an environmentally sustainable tool to capture, sense and remediate POPs. We propose that nutrition, and in particular diet-derived bioactive compounds such as polyphenols, can protect against POP toxicity through their antioxidant and anti-inflammatory properties. Polyphenols also can participate in a remediation platform that includes their roles in POP capture and sensing technologies, thus allowing for environmentally friendly and sustainable remediation of POPs.