International Conference on Cancer Research 2019

Website: www.CancerConf.org

June 10-11, London, UK


# Influence of Microalgae Extracts on the Toxicity of a Carcinogenic Polycyclic Aromatic Hydrocarbon, Benzo(a)pyrene, in Endothelial Cells

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**Abstract**

Exposure to environmental contaminants is known to cause cancer in humans. Polycyclic aromatic hydrocarbons (PAHs) generated during the incomplete combustion of organic matter, constitute an ubiquitous family of environmental contaminants whose main source of exposure is from human activities (industrial activities, cigarette smoke, food processing...). Among them, benzo(a)pyrene(B(a)P), found in all mixtures humans are exposed to, is classified in group 1, i.e. carcinogenic to humans, by the International Agency for Research on Cancer. Most of B(a)P deleterious effects (i.e. apoptosis, induction of pro-inflammatory cytokines and CYP450 enzymes) are mediated by the activation of a ligand-activated transcription factor : aryl hydrocarbon receptor (AhR).

Marine microalgae are photosynthetic microorganisms, known to be a source of bioactive molecules of interest to human health, such as n-3 polyunsatured fatty acids (n-3 PUFAs) and pigments (carotenoids: fucoxanthin, violaxanthin…). The fact that some of the natural compounds are known to exhibit antiinflammatory, antioxidant, anti-proliferative, apoptosis-inducing effects demonstrates their potential interest in preventing cancers.

In this context, we have used two microalgae extracts in order to evaluate and compare their potential effects towards B(a)P-induced toxicity in endothelial HMEC-1 cells (human microvascular endothelial cells-1). Endothelial cells were chosen because they express AhR eceptor and CYP450s and they are well known targets of PAHs.

The cytotoxicity, apoptosis, expression of CYP1B1 (P450 isoenzyme involved in B(a)P bio-activation) and of pro-inflammatory cytokines have been studied during exposure of B(a)P or microalgae extracts alone and upon co-exposure using MTT assay, Hoechst fluorescent staining and mRNA expression analysis, respectively

The microalgae extracts are derived from *Ostreoccoccus tauri* (OT), a green microalgae rich in docosahexaenoic acid (DHA, 22 :6 n-3) and *Phaeodactylum tricornutum* (PT), a diatom rich in eicosapentanenoic acid (EPA, 20 :5 n-3). The lipid and pigment composition of the extracts were determined.

Our results show that OT and PT extracts can significantly decrease B(a)P-induced CYP1B1 mRNA levels. MTT assay, as expected under our experimental conditions, revealed B(a)P toxicity, but does not show any toxicity for the extracts alone, nor increased toxicity upon co-exposure. Interestingly, using Hoechst staining, we have observed that OT extract, unlike PT extract, significantly decreased B(a)P-induced apoptosis. Concerning the effects of OT and PT extracts on mRNA expression of two pro-inflammatory cytokines, IL-8 and IL-β, we have observed that both OT and PT extracts inhibited B(a)P-induced IL-8 and IL-1β mRNA expression.

Taken together, our data indicate that microalgae extracts can influence the toxicity of B(a)P at least by decreasing the induction of CYP1B1 mRNA and of pro-inflammatory cytokines (IL-8 and Il1-β), and only for OT extract, the B(a)P-induced-apoptosis. The differential effects obtained between the two extracts on the toxicity of B(a)P is probably related to their different composition, such as EPA and DHA among others. Indeed, it has been recently reported that EPA and DHA may differently affect B(a)P metabolism.

In conclusion, these results are promising concerning the possible use of microalgae extracts to counteract the B(a)P-induced toxicity but remain to be confirmed by further study *in vivo.*

**Keywords:**

Microalgae,

Benzo(a)pyrene,

Endothelial Cells,

PAHs.