Proceedings of the Nutrition Society (2022), **81 (OCE4)**, E135 doi:10.1017/S0029665122001641

Irish Section Conference 2022, 15–17 June 2022, Impact of nutrition science to human health: past perspectives and future directions

Anti-inflammatory and antioxidant activities of polar lipids *in vitro* and implications for neurodegenerative disease

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The platelet-activating factor (PAF) molecule is a pro-inflammatory phospholipid mediator that functions by binding its receptor PTAFR. PAF is known to have a key role in the mechanism of chronic inflammation(1). The pathology of cardiovascular disease, cancer, rheumatoid arthritis, and neurodegenerative diseases has been linked to chronic inflammation(2). Studies have shown that polar lipids have a significant anti- inflammatory and antithrombotic effect and thus present a potential source of nutraceuticals that protect against the detrimental effects of pro-inflammatory processes(3). This has important implications for disease pathologies linked to chronic inflammation. The goal of our study is to determine if polar lipids derived from salmon can attenuate chronic inflammation and oxidative stress, in the context of neurodegenerative disease. We performed bioassays to study the anti-inflammatory and antioxidative role of salmon lipids. Using DI TNC1 rat astrocytes as an *in vitro* model, we stimulated one set of cells with 1 μM amyloid-beta protein (Aβ) to induce inflammation in the cells. Astrocytes stimulated with LPS (1mg/ml) were included as a positive control. After 24 h incubation with either Aβ or LPS, the cells were treated with salmon-derived polar lipid extract (SPL). We also treated astrocytes with lipid alone without any induced inflammation. Subsequently, RNA was purified from the cells to analyze PTAFR gene expression levels by qRT-PCR. To understand effects on oxidative stress levels, cells were stained with CellROX Green dye to detect intracellular ROS and imaged using the IMX micro confocal system. All experiments were performed in technical triplicate. Statistical analysis was performed using one-way ANOVA and Tukey's post hoc test. Our results show that SPL produce a statistically significant reduction in PTAFR expression compared to cells stimulated with Aβ alone (p-value 0.0021) and LPS alone (p-value 0.00027). In addition, after inducing oxidative stress (OS), SPL cause a statistically significant reduction in intracellular ROS compared to cells not treated with lipid (p-value 0.0057). We conclude that specific lipids attenuate oxidative stress and downregulate pro-inflammatory signaling in cells relevant for mediating immune responses. Mechanistically, lipids may exert their activity via inhibiting PTAFR pro-inflammatory signaling in astrocytes. Future experiments will aim at investigating the properties of lipids from different sources, for example, dairy lipids. These findings could inform highly effective dietary preventive strategies against chronic inflammation.

Acknowledgments

I would like to sincerely thank my advisors Dr Zabetakis and Dr Grabrucker and also my colleagues for their continuous support and guidance during these investigations.

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