Healthy human lung epithelial cells beas-2b cells exhibit pro-inflammatory reactions.

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Abstract

An immune response to infection that involves both the innate and adaptive immune systems is called inflammation. If inflammation is allowed to persist unchecked, however, it can lead to a number of pathologies. Antioxidant-rich natural molecules have the ability to target the main causes of inflammation and have positive effects on health. In order to show the enhanced antioxidant and anti-inflammatory properties of the combination, human normal bronchial (Beas-2B) and prostate (HPrEpiC) epithelial cell lines were exposed to infectious stimulation and treated with phycocyanin (PC) and palmitoylethanolamide (PEA). The treatment significantly reduced the production of radical oxygen species (ROS), several inflammatory cytokines, and protected against cytotoxicity. By modifying three key factors, oxidative stress and inflammation were reduced.

Keywords: Phycocyanin, Palmitoylethanolamide, Glutathione, Mitochondrial respiratory complexes, Human lung epithelial cells.

Introduction

The return to homeostasis and complete recovery to a state of good health are the objectives of the inflammatory process. The inflammatory phenomenon is a defensive mechanism controlled by biological molecules that results from the removal of the infectious agent and the repair of the damaged tissues (neuropeptides, hormones, cytokines, growth factors). But if it fails, the pathology may advance, functional and structural damage may begin, and a chronic inflammatory process may result-often fatally. Inflammation causes immune cells to secrete a variety of cytokines and chemokines in order to attract more immune cells to the infection site. A stress-inflammatory response is activated when the homeostatic capacity is too low and mechanisms fail to resolve the insult [1].

The cyclooxygenase-2 enzyme is inhibited which results in a reduction in the signalling that produces ROS; glutathione synthesis is increased, which strengthens the body's natural antioxidant defences; and the infection-driven mitochondrial respiratory burst, which causes oxidative stress, is reduced. The current study reveals novel mechanisms of action and increased efficacy of PC and PEA, supporting the potential application of this combination in treating human disorders. This study was motivated by the growing interest in using nutraceuticals as adjuvants in clinical practise [2].

In addition to being caused by exposure to the environment or to drugs, a metabolic disorder in the mitochondria also produces too many ROS. OS and tissue damage are brought on by increased ROS generation that is sparked by immune cells at the site of inflammation. The availability of antioxidants or radical scavengers and the production of ROS are in fact out of balance, which leads to OS. The primary antioxidant defence of the tissues is represented by glutathione [3].

Previous descriptions of the interaction between glutathione and COX-2 have been provided. According to reports, COX-2 activity and expression are both suppressed by the mutual regulation of glutathione-dependent glutathione peroxidase (GPx) and COX-2 by the local removal of hydroperoxides from GPx. When glutathione levels are low, the excess ROS production either oxidises biomolecules or structurally modifies proteins and genes to activate transcription factors and pro-inflammatory genes to start signalling cascades. These occurrences can trigger the onset and development of inflammatory illnesses like cancer, neurodegenerative diseases, and ageing. In an ongoing effort to reduce inflammation, various pharmacological strategies that either target COX-2 activity or ROS production are being developed [4].

Palmitoylethanolamide is a different molecule that recent studies have suggested as an anti-inflammatory compound (PEA). PEA has inflammatory-blocking, analgesic, and neuroprotective effects. Peroxisome proliferator-activated receptor (PPAR) activation by PEA reduces inflammation by influencing the TLR/NF-B axis, lowering NF-B activity,

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suppressing the expression of pro-inflammatory cytokines like TNF- and limiting the recruitment of immune cells. According to a recent study, PEA combined with paracetamol reduces inflammation caused by NF-B, which in turn reduces the production of inflammatory prostaglandins brought on by COX-2 activity. Therefore, it was noted that PEA's impact on COX-2 was indirect and mediated by NF-B. A decrease in PEA levels is thought to contribute to the inflammatory response and evidence points to disturbed PEA metabolism during inflammation [5].

Conclusion

This *in vitro* study backs up the efficacy of concomitant PC and PEA administration in preventing cytotoxicity and reducing both oxidative stress and inflammation. These findings imply that PC and PEA therapy strengthens the tissue's natural antioxidant and anti-inflammatory defences by raising glutathione levels, enabling the cell to react to the inflammatory stimulus more forcefully. The following mechanisms are used to produce the advantageous effects: (1) less inflammatory cytokine secretion, and (2) lessened ROS levels and inhibition of the ROS/COX-2/inflammatory cytokine self-amplifying loop due to decreased mitochondrial respiratory burst brought on by bacterial or viral stimulus. Additionally, PC and PEA shield cells from oxidative stress by influencing three major pathways: (1) inhibition of COX-2 and a resulting reduction in signalling that produces ROS; (2) increased glutathione synthesis and a corresponding bolstering of the tissue's natural antioxidant defences; and (3) a reduction in the infection-driven mitochondrial respiratory burst, which causes oxidative stress.

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