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"Free Fatty Acid Elovanoid Precursors Modulate Allergen-induced NLRP10 Expression in Human Nasal Epithelial Cells"

House dust mites (HDM) are a common indoor source of allergen-induced inflammation in the nose. The two prominent allergens that compose HDM are *Dermatophagoides pteronyssinus* (*D. ptero*) and *Dermatophagoides farinae* (*D. fari*). Elovanoids (ELVs), derived from omega-3 very-long-chain polyunsaturated fatty acids (VLC PUFA), are pro-homeostatic lipid mediators that have been shown to provide cells protection from damage and death. NLRP10 is a pro-inflammatory protein that when expressed, forms an inflammasome complex. Literature study has shown that the NLRP10 initiated inflammasome complex leads to the expression of the protease caspase-1 and cytokine IL-18. In this study, we explored the effects of common indoor allergens on the expression of NLRP10 and whether ELV precursors 32:6 and 34:6 free fatty acids (FFAs) could modulate the allergen-induced expression. In addition, we plan to silence NLRP10 expression using an NLRP10 specific silencer and look for the expressions of NLRP10, caspase-1, and IL-18. This analysis is in progress and will hopefully be included in the final data. We used human nasal epithelial cells (HNEpC) as an in vitro model and challenged them with allergens *D. fari* (30µg/mL), *D. ptero* (30µg/mL), and HDM (30µg/mL) [*D. fari* (15µg/mL) + *D. ptero* (15µg/mL)]. Cells were then treated with ELV precursors 32:6 and 34:6 FFA (500nM) to counteract the HDM induced allergic effect. An NLRP10 specific siRNA will be used to minimize NLRP10 expression in HNEpC cells. An unbiased and time-efficient Jess (Protein Simple) Western blot (WB) analysis will be used to detect the expression of NLRP10 and associated proteins in the post treatment cell extracts (with and without siRNA). Our results show that NLRP10 expression is upregulated in HNEpC when challenged with HDM, *D. fari*, and *D. ptero* compared to a control group. Along with these results, we found that when the same groups were treated with 32:6 and 34:6 FFA, NLRP10 expression was downregulated across all three conditions, with the difference being the greatest in the HDM condition. Furthermore, like NLRP10, expressions of both caspase-1 and IL-18 were upregulated with HDM, *D. fari*, and *D. ptero* compared to controls. Moreover, we found a downregulation in the expressions of both proteins when the allergen-challenged cells were treated with 32:6 and 34:6 FFA. Collectively, these findings show that HDM can induce an increase in NLRP10 inflammasome formation, and the formation of the inflammasome is compromised by ELV precursors 32:6 and 34:6 FFA in HNEpC. Together, our findings displayed for the first time that ELV precursors mediate HNEpC protection from inflammation and may be a potential therapeutic target for allergens.