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Phonetic spelling of your (full) name

Sod

Presenting Author Category

Undergraduate Student

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GBD Audio File

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Headshot

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Short Bio (100 words)

Oxylipins such as EpOMEs and DiHOMEs are abundant in our airway, but their functions are not fully understood.

EpOMEs and DiHOMEs are shown to delay airway epithelial wound recovery likely through a partial inhibition in cell proliferation.

The purpose of this research is to determine whether these oxylipins act through a TRPA1/TRPV1 ion channel dependant mechanism. Because impaired epithelial repair is associated with diseases such as asthma, understanding this mechanism may help explain how oxylipins contribute to airway disease.

Research Category

Basic Science

Abstract Title

EpOMEs and DiHOMEs affect Airway Epithelial Wound Healing

Authors

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Background

Oxylipins are oxidized signalling lipids derived from polyunsaturated fatty acids. Some oxylipins, like epoxyoctadecamonoenoic acids (EpOMEs) and dihydroxyoctadecamonoenoic acids (DiHOMEs), are abundant in our airways and yet, a physiologic function has not been elucidated. In airway epithelium, we have shown these oxylipins delay wound healing, but the mechanism is unknown. In airway smooth muscle, these oxylipins act through ion channels Transient Receptor Potential Ankyrin 1 (TRPA1) and Vanilloid 1 (TRPV1).

Objective

Validate that EpOME and DiHOME impairs airway epithelial proliferation and determine dependency on TRPV1 and TRPA1 activation.

Methods

Calu-3 human airway epithelial cells were cultured on transwell inserts and serum-deprived for 24 hours prior to EpOME and DiHOME treatments. Transepithelial electrical resistance (TEER) was used to measure resistance as an inference of wound recovery for 72-hours. For measurement of proliferation, cells were stimulated with EpOME and DiHOMEs along with TRPV1 or TRPA1 channel inhibitors. Luciferase activity was measured to infer Ki67 protein content and quantify proliferation at 3 and 24hrs after stimulation. Data were analyzed using a two-way ANOVA with repeated measures in GraphPad Prism.

Results

Wound healing was significantly delayed in response to oxylipin exposure, ranging from a 13% reduction with 9,10 DiHOME to a maximum of 38% with 12,13 EpOME relative to control. Ki67 abundance was reduced by ~25% with oxylipin exposure at 3hrs, but Ki67 signal was noisy 24hrs post stimulation. The inhibitor for TRPA1 and TRPV1 reversed some of these effects, dependent on the oxylipin, although a sample size prohibits strong conclusions.

Conclusion

EpOMEs and DiHOMEs inhibit airway epithelial wound healing by preventing cell proliferation. Activation of TRPV1 and TRPA1 channels may be involved, but we cannot conclusively rule out other pathways. Identifying the physiological role of these EpOMEs and DiHOMEs provides a framework for understanding changes in abundance may relate to airway pathophysiology.