International Conference on "Novel Approaches in Agro-ecology, Forestry, Horticulture, Aquaculture, Animal Biology and Food Sciences for Sustainable Community Development"
(Agro-tech-2017)

Potential Role of Lipoxygenases and their Metabolites in Asthma

Younus Ahmad Bhat[1], Rakhshinda Rehman[1], Ulaganathan Mabalirajan[1] and Anurag Agrawal[1]

CSIR-Institute of Genomics and Integrative Biology, Mall Road, Delhi, India
E-mail: bhatunice@gmail.com

Abstract—Asthma is a chronic inflammatory disease of airways of lungs that is symptomized by recurring periods of wheezing, chest tightness, coughing and shortness of breath. It is a multicellular process involving many eosinophils, lymphocytes, and mast cells. Lipoxygenases are a group of enzymes that catalyze conversion of polyunsaturated fatty acids to peroxides. 15-Lipoxygenase (LOX) and its metabolites, such as 13-(S)-hydroxyoctadecaenoic acid (13-(S)-HODE) and 12(S)-HETE, are known to cause mitochondrial degradation. 13-(S)-HODE is implicated to bind and activate TRPV1, transient receptor potential vanilloid subtype-1, which is known to increase intracellular calcium. This can alter calcium signalling and cause apoptosis. In this context, we hypothesize that 13-S-HODE may accelerate apoptosis in allergic mice and inhibition of this lipid metabolite or its downstream signaling pathway by inhibiting TRPV1 may alleviate asthma features by reducing epithelial apoptosis. In this study we show that exogenous administration of 13-S-HODE causesairway inflammation, airway hyperresponsiveness, and apoptosis in the bronchial epithelia. We were able to explicitly demonstrate that neutralization of 13-S-HODE and knockdown of TRPV1 receptor alleviates symptoms of asthma. However, 13-S-HODE does not play any role in mucus metaplasia and sub-epithelial fibrosis. In conclusion, this study revealed few novel and crucial functions of 13-S-HODE in allergic airway and emphasize that the neutralization of 13-S-HODE could be an efficient and promising therapeutic strategy in respiratory diseases.

Keywords*: Asthma, Apoptosis, Metaplasia, Fibrosis, Inflammation.*

ISBN: 978-93-85822-60-5
Pages No. 76-76