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The impact of annexin A5 on asthma exacerbation following nanoparticles exposure

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Introduction & Aims: Annexin A5 (ANXA5) has a potential role in cellular signal transduction, inflammation and fibrosis. However, the exact role of ANXA5 in asthma remains to be clarified. The aims of the present study were to investigate ANXA5 protein expression in a mouse model of asthma and pollutant exposure and to elucidate the relationships between clinical variables and plasma ANXA5 levels in patients with asthma.

Methods: A murine model of asthma induced by Ovalbumin (OVA) and titanium dioxide (TiO₂) nanoparticles has been established using BALB/c mice and we examined ANXA5 expression and lung fibrosis using this model. Moreover, we also compared ANXA5 plasma levels in patients with controlled vs. exacerbated asthma.

Results: ANXA5 protein levels were lower in lung tissue from OVA+OVA mice than in control mice. Lung ANXA5, Connective Tissue Growth Factor (CTGF) and Transforming Growth Factor β 1 (TGF- β 1) protein levels were higher in OVA+TiO₂ exposed mice than in control or OVA+OVA mice. Although Dermatophagoides pteronyssinus (Derp1) treatment increased lung ANXA5 protein levels in MRC-5 cells and A549 epithelial cells, it decreased lung ANXA5 levels in NHBE cells. Treatment with TiO₂ nanoparticles increased lung ANXA5, CTGF and TGF- β 1 protein levels in MRC-5 cells, A549 epithelial cells and NHBE cells. Plasma ANXA5 levels were lower in asthmatic patients than in healthy controls and they were significantly enriched in patients with exacerbated asthma compared with those with controlled asthma (P<0.05). ANXA5 levels were correlated with pulmonary function as assessed by spirometry.

Conclusion: Our results imply that ANXA5 plays a potential role in asthma pathogenesis and may be a promising marker for exacerbated bronchial asthma and exposure to air pollutants.

Biography

Pureun-Haneul Lee works for Genome Research Center for Allergy and Respiratory Diseases Soon Chun Hyang University Bucheon Hospital, Republic of Korea. He has contributed his work on Claudin 5 transcripts following acrolein exposure affected by epigenetic enzyme in the *Journal of Clinical Toxicology*.

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