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An understanding of the MUC5AC gene expression in asthma

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Asthma is a chronic disease that affects the respiratory system and causes inflammation in the lungs, consequently leading to complications in breathing. One of the causes of asthma is airway obstruction due to an over-production of the mucin glycoprotein (mucin 5AC) which is a principal component of mucus. Elevation in the expression of mucin 5AC (MUC5AC) is caused by the cytokine, interleukin-4 (IL-4) which is produced by T helper type 2 cells (Th2). Th2 cells are involved in type 2 immune responses that work towards eliminating extracellular infections as in the case of asthma. IL-4 elicits allergen-induced airway inflammation and excess mucus production leading to respiratory tract blockage. After IL-4 production is triggered, it binds with the receptors present on the surface of the cell membranes which causes the signalling molecule inositol trisphosphate (IP3) to bind with the inositol trisphosphate receptor (IP3R) that is present on the surface of the endoplasmic reticulum (ER). As a result, calcium moves out of the ER, and this decrease in calcium concentration, in turn, activates the stromal interaction molecule 1 (STIM1) proteins present on the surface of ER. They form a complex with the transient receptor potential channel 1 (TRPC1) located on the surface of the plasma membrane which is responsible for allowing an influx of calcium ions. This calcium influx activates the protein complex called nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB), which is involved in the transcription of the MUC5AC gene expression. Overexpression of this gene is responsible for causing asthma. Therefore, novel strategies can be developed to selectively inhibit the effect of IL-4 and provide alternative treatments for people with asthma.

Keywords: Asthma, Mucus, Calcium, Interleukin-4, Mucin 5AC

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