

Role of Mycobacterial Species in Asthma

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DESCRIPTION

Asthma is a significant human disease that causes significant morbidity and mortality globally. Asthma is caused by the influence of environmental, immunological, and host genetic factors. Furthermore, epidemiological studies shows a strong link between asthma and infection with respiratory pathogens such as rhinoviruses, human respiratory syncytial virus, adenoviruses, coronaviruses, and influenza viruses, as well as bacteria (including atypical bacteria) and fungi. Inhaled Corticosteroids (ICSs) are generally used as treatment for asthma and other pulmonary diseases. It is known that Inhaled Corticosteroids (ICS) increase the risk of developing other mycobacterial diseases like Tuberculosis (TB) or Non-Tuberculous Mycobacterial Pulmonary Disease (NTM-PD). Studies have also predicted that the use of bacterial components like Toll-Like Receptor (TLR) ligands and other pathogen-associated molecular patterns can be used as potential asthma therapies.

Studies have found an association between severe lower respiratory infections caused by Human Respiratory Syncytial Virus (RSV) or Human Rhinoviruses (RVs) in childhood and the development of recurrent wheeze followed by asthma later in life. RSV is a Paramyxoviridae family negative-sense single-stranded RNA (ssRNA) virus. Some children with bronchiolitis experience recurrent respiratory symptoms for unknown reasons. Human Rhinoviruses (RVs) belong to the Picornaviridae family, have a positive-sense ssRNA genome of about 7.1-7.5 kb, and are classified into major and minor groups based on their host receptors: major group viruses bind Inter Cellular Adhesion Molecule 1 (ICAM1), whereas minor group viruses bind Low-Density Lipoprotein Receptor (LDLR). Human Rhinoviruses (RVs) can also be divided into three groups based on nucleotide sequence identity (RV-A, RV-B and RV-C). The RV-C group viruses have distinct sequences at the Inter Cellular Adhesion Molecule 1 (ICAM1) and Low-Density Lipoprotein Receptor (LDLR) binding sites, indicating that they use a different (but as yet unknown) receptor.

The onset of Asthma

Hypothesis of hygiene: According to the hygiene hypothesis, repeated exposure to various common infections (particularly bacteria, food-borne and orofaecal parasites, and hookworms) and exposure to environmental micro biota during childhood are strongly associated with immune system maturation and protection from the development of asthma and allergies later in life. A molecular analysis of the microbiome has recently improved the understanding of the importance of microorganisms in allergy and asthma protection. Populations living in environments with diverse microbiological flora have a lower incidence of asthma, whereas populations living in environments with low microbiological diversity have a higher incidence of asthma. The prevalence of bacteria and their components in various environments have been studied using culture-dependent and molecular methods, and both qualitative and quantitative differences between countries with a high and low incidence of atopy that have been discovered. Experiments in mice show that intranasal exposure to *Escherichia coli* or *Acinetobacter lwoffii* str. protect against allergic inflammation of the airways, leading to the hygiene hypothesis.

CONCLUSION

Advances in molecular microbiology are opening up new avenues for studying the roles of microorganisms in asthma pathogenesis and exacerbation. Asthma is now widely recognized as a chronic inflammatory condition that is initiated, triggered, and maintained by the respiratory microbiota. However, it is unclear about the apt cause of the disease but it is predicted that most of the asthma exacerbations might undoubtedly be caused by viruses, microorganisms, especially different mycobacterial strains and some non-viral pathogens. Further research is needed to identify microbial targets that can be eradicated with vaccines or antimicrobial drugs, thereby reducing asthma.

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