

# A Microbiological Study on Asthma and Allergy Management

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**ABSTRACT:** The compare of allergic disorders between populations in different countries by sing standard validated questions, thereby providing a framework for research into possible modifiable lifestyle and environmental factors affecting these disorders that may ultimately lead to a reduction in the personal burden of allergic diseases. Many centres from developing countries were able to participate in the simplicity. The data provides a unique illustration of the prevalence of current symptoms of asthma, rhino conjunctivitis and eczema in children and adolescents worldwide.

**KEY WORDS:** Antimicrobial peptides;Immunomodulatory;Lymphopietin;Antibiotics.

## I. INTRODUCTION

There is a wide consensus that the prevalence of asthma and allergies are on the increase among children worldwide. In some industrialized countries, asthma and allergies have reached alarming proportions, affecting up to one-third of children within the general population. In addition, few of the studies of prevalence were completed in developing countries. A Study of Asthma and Allergies has developed a standardized methodology to describe the prevalence and severity of symptoms of asthma, rhino conjunctivitis and eczema in the world. In the development of both asthma and allergic rhinoconjunctivitis, there is a complex interaction of genetic and environmental factors. A possible explanation is the "hygiene hypothesis". This suggests that increased hygiene and the resulting lack of exposure to various microorganisms in early life affect the immune system so that individuals' ability to fight off certain diseases is weakened and they are more susceptible to autoimmune diseases.

Good management of asthma can control the disorder and enable people to enjoy a high quality of life. Early diagnosis and appropriate treatment lead to much better disease control and outcomes. Allergy can be progressive, and neglecting its symptoms may lead to a worsening of the disease. Medication is not the only way to control asthma and allergies. It is also important to avoid triggers that irritate and inflame the airways. Primary prevention to reduce the level of exposure to common risk factors, particularly tobacco smoke, frequent lower respiratory infections during childhood and air pollution (indoor and outdoor), is an important step.

## II. REVIEW OF LITERATURE

The identification of key microorganisms that are linked with asthma development or asthma pathogenesis raises the important question of which properties of these microorganisms promote asthma. Some of these microorganisms may be found systemically in more severe disease, as has been shown for RVs73. A key feature of a microorganism in promoting asthma is the ability to induce lung inflammation, injury, or repair and remodeling. Many of the innate receptors that

# International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

Vol.3, Issue 6, June 2014

recognize respiratory fungi, viruses or bacteria, including TLRs, RIG-I-like helicases and NOD-like receptors activate the nuclear factor- $\kappa$ B (NF- $\kappa$ B) family of transcription factors, which induce more than 100 pro-inflammatory and host response genes<sup>74</sup>. RSV and RVs induce a range of pro-inflammatory cytokines, chemokine's, growth factors, adhesion molecules and mucins. Mucins cause mucous plugging of the airway and may provide a substrate for bacterial colonization, whereas cytokines facilitate cellular chemo taxis, and activation and proliferation of immune cells in the infected airway<sup>45, 75</sup>. Microorganisms may damage and compromise the integrity of the airway by infecting airway epithelial cells, causing cell death<sup>76</sup> and shedding<sup>77</sup>. They can also affect epithelial permeability, leading to increased airway inflammation and creating opportunities for increased infection<sup>78</sup>, allergen uptake<sup>79</sup> or exposure to environmental pollutants, all of which are important contributing factors in asthma.

## Microorganisms and asthma treatment

Asthma treatment often requires daily treatment with a range of immunomodulatory or immunosuppressive agents, such as macrolide antibiotics or glucocorticoids, suggesting another way that microorganisms interact with asthma. Surprisingly, scant attention has been paid to the possible effects of such treatments on an individual's micro biota and their antimicrobial responses. However, some studies provide indirect evidence for an immunosuppressive effect. For example, increases in bacterial pneumonia have been noted in patients with COPD who are treated with glucocorticoid therapy<sup>84</sup>. Furthermore, an increased incidence of upper respiratory tract infections following treatment with the leukotriene receptor antagonist montelukast has been reported in young children with asthma<sup>85</sup>. It is also interesting to speculate whether the fact that asthma is a risk factor for IPI, as previously discussed, is directly attributable to asthma therapies such as glucocorticoids. Conversely, there is some experimental evidence suggesting that the action of glucocorticoids confers benefits to antimicrobial immunity. *In vitro*, glucocorticoids have been shown to induce a number of antimicrobial peptides, pentraxins, and collections and complement proteins.

## III. OBJECTIVE AND SCOPE

An increasing trend in the prevalence of asthma and allergies is particularly apparent in urban areas, where children have been found to have more allergic reactions to outdoor and indoor allergens.

Asthma phenotype	Description
Atopic (allergic) asthma	<ul style="list-style-type: none"><li>• Reactivity to one or more known allergens (determined by skin prick test)</li><li>• Higher than normal IgE levels</li><li>• Specific IgE to allergen (determined by radioallergosorbent assay*)</li><li>• Eosinophils in the airways (common but not always)</li></ul>
Non-allergic asthma	<ul style="list-style-type: none"><li>• No known allergy</li><li>• No or low serum IgE</li><li>• No reactivity to allergen (determined by skin prick test or radioallergosorbent assay*)</li></ul>
Mild asthma	<ul style="list-style-type: none"><li>• Occasional symptoms, but well controlled by therapy (low-dose glucocorticoids and/or short-acting <math>\beta</math>2 agonists)</li></ul>
Stable asthma	<ul style="list-style-type: none"><li>• Well-controlled symptoms</li><li>• No dysregulated lung function or asthma exacerbations</li></ul>
Severe asthma	<ul style="list-style-type: none"><li>• Poor control of symptoms despite using maximal therapy (high-dose glucocorticoids and/or <math>\beta</math>2 agonists, or other therapies)</li></ul>
Asthma exacerbations	<ul style="list-style-type: none"><li>• Mild or severe asthma with sudden symptom onset, often requiring medical consultation or hospitalization and possible oral glucocorticoid intervention</li></ul>
Neutrophilic asthma	<ul style="list-style-type: none"><li>• Increased number of neutrophils in bronchoalveolar lavage or sputum samples (can be allergic or non-allergic asthma)</li></ul>

# International Journal of Innovative Research in Science, Engineering and Technology

(An ISO 3297: 2007 Certified Organization)

Vol.3, Issue 6, June 2014

Allergic bronchopulmonary aspergillosis	• Invasive colonization of the airway with <i>Aspergillus</i> spp., an opportunistic pathogen; <i>Aspergillus</i> growth promotes allergic sensitization to at least 12 known fungal allergens
Virus-induced wheeze or bronchiolitis	• Common in young children (<2 years of age) • Asthma caused by a viral infection, mucus and inflammation in young children, giving a typical 'wheezy' episode • Mild, severe, acute onset or febrile
High TH2-associated atopic asthma	• TH2 cell-associated gene expression is typical (for example, expresses <i>IL13</i> and <i>IL-13</i> -inducible genes) • Responds well to glucocorticoids • Prone to asthma exacerbations <sup>135</sup>
Low TH2-associated atopic asthma	• A mixed phenotype of asthma • Less prominent TH2 type cytokine responses • More refractory to glucocorticoid treatment than patients with high TH2-associated atopic asthma <sup>135</sup>

There seems to be a parallel development between climate change and the increasing prevalence of asthma and allergies in children. As warmer temperatures and early spring are related to increased airborne pollen, sensitization to pollen allergens is likely to have doubled during the last three decades, particularly in young people in many areas.

## IV. RESEARCH METHODOLOGY

### Microbiological involved in asthma and their niches

A cross-section of the human lower respiratory tract, showing sites of infection for different microbiological effects that they have on airway function. This would also be facilitated by an increase in tertiary lymphoid tissue in the lung parenchyma, which is a common feature following respiratory tract infection<sup>104</sup>, in human asthma<sup>105</sup> and in experimental mouse models of asthma<sup>106</sup>.

Successive infections may also alter the lung environment and affect the delicate balance between host, pathogen and chronic airway disease. Bacterial infections are known to follow influenza virus infections; indeed, many of the individuals who died in the influenza pandemics exhibited coexisting bacterial pneumonia<sup>107</sup>. This has implications for asthma: what is assumed to be a virus-induced asthma exacerbation may in fact include a secondary bacterial infection. Data suggesting that this does occur have been reported for *C. pneumoniae*<sup>69</sup>, but further studies are required to determine whether secondary bacterial infections commonly follow viral infections in asthma. Thus, by causing barrier disruption, viruses may initiate a cascade that, as described above, allows environmental bacteria better access to the underlying tissue.

### Respiratory viruses interact with allergens in an additive or synergistic manner, promoting asthma

Following sensitization, allergen presentation by airway dendritic cells (DCs) facilitates the promotion of T helper 2 (TH2) cells. Viruses infect epithelial cells, stimulating the release of TH2 cell-promoting chemokines CC-chemokine ligand 17 (CCL17) and CCL22, and cytokines thymic stromal lymphopoietin (TSLP), interleukin-25 (IL-25) and IL-33. The TH2 type chemokines attract TH2 cells into the airway, and these in turn secrete IL-4, IL-5 and IL-13. IL-5 promotes eosinophilia, and the resultant eosinophils release the inflammatory mediator's major basic protein (MBP), eosinophil cationic protein (ECP) and transforming growth factor- $\beta$  (TGF $\beta$ ), inducing inflammation in the airway smooth muscle (ASM). IL-4 and IL-13 cause antibody class switching to immunoglobulin E in B cells, so that B cells secrete allergen-specific IgE. This antibody then binds mast cells, and crosslinking of the allergen on mast cell-bound IgE causes mast cell degranulation and release of preformed mediators, including histamine, prostaglandin (PGD<sub>2</sub>) and leukotrienes (LTC<sub>4</sub>, LTD<sub>4</sub> and LTE<sub>4</sub>). These mediators cause bronchoconstriction and further airway inflammation. Mast cells also produce the TH2 cytokines IL-4 and IL-13, as well as other cytokines, including TGF $\beta$  and tumour necrosis factor (TNF), promoting further TH2 type immune responses and inflammation.

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Vol.3, Issue 6, June 2014

## V. DISCUSSION

Although several studies support the use of macrolide antibiotics for the treatment of atypical pathogens in patients with asthma exacerbations<sup>88</sup>, it is important to consider how the microbiological is affected by macrolides or other antibiotics and how this might affect asthma and the host responses to infection. Treatment of mice with the antibiotics vancomycin, neomycin, metronidazole and ampicillin greatly changes their airway and gut microbiological and impairs their antiviral responses to influenza viruses<sup>35</sup>. In human studies, it is currently unclear whether the genera that seems to colonize the airways of patients with asthma or and of individuals without asthma or differ because of treatment-specific effects<sup>34</sup>. More research is needed to understand whether the microbiological of patients with asthma is actually a consequence of active therapy. Further large clinical trials with such treatments are required to understand the relationships between microbiological and asthma treatments.

## VI. CONCLUSION

The empirically driven realization that allergy and asthma are intertwined demand a greater understanding of the cellular components of these and of the mechanisms that regulate them. Whether adenosine is the key component that links allergy and asthma remains to be determined.

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