

## Infants with RSV Infection Immunity and the Microbiome

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**Received:** December 30, 2019; **Published:** February 29, 2020

### Abstract

Upper respiratory infections in infants is one of the most serious disease which infant may encounter Prevention and management are crucial for healthy development of the growing infant.

**Keywords:** *Viral Lower Respiratory Tract Infection; RSV; Asthma; Microbiota*

### Introduction

Respiratory Syncytial Virus (RSV) is the most important cause of childhood acute respiratory morbidity and hospitalization. This disease is rated as the highest among children aged 6 weeks to 6 months. Severe RSV disease in the first year of life is a significant risk factor for subsequent asthma and recurrent wheeze in children later in life [1,2].

Infants and children with high risk for severe RSV illness are usually premature infants, under 6 months of age, or under 2 years of age with the following conditions: chronic lung disease, chronic heart disease, fragile immune system, neuromuscular disorders, difficulty in swallowing or unable to clear mucus secretions [3-5].

RSV causes frequent emergency visits in most common chronic reactive airway disease of childhood and it is a significant healthcare burden and emotional stress on the parents [6]. Children who develop severe RSV disease in early childhood are more prone to have difficult asthma if they encounter asthma later in childhood. There is a clear association between the age during which severe RSV disease occurred and the subsequent risk of asthma in children who develop their first attack episode of severe RSV disease in the first 2 years of life. Although burden of RSV is highest in children aged less than 6 months, but the burden of subsequent asthma is higher in children who develop RSV disease at ages older than 6 months [3]. Factors which contribute to severity of RSV are: multiparty of the mother, mode of delivery, maternal smoking during pregnancy, small for gestational age and association with other viral respiratory infection such as Influenzas' virus, Rhino virus, Para influenza virus and other seasonal viruses [7].

An effective maternal vaccine will play a pivotal role in lowering the burden of RSV-associated lower respiratory tract infections in children aged below 6 months [8]. An effective vaccine for children aged more than 6 months will have a beneficial impact on the long-term consequences of RSV disease sine it may ameliorate the development of asthma following early RSV infection.

RSV plays short-term and long-term alterations (damage) in the airway physiology and in the airway immune response [9]. The extent of airway damage is probably linked to the severity of the first episode of RSV disease. The severity of RSV disease in young infants aged

below 6 months is probably with highest risk, owing to the incomplete lung and immune system maturation. Maternal antibody against RSV has a half-life of 76 - 81 days and provides protection against severe disease during the first RSV season [10]. Babies born to mothers with high levels of IgG antibody to respiratory syncytial virus were protected against infection with this virus during the first month of life when the risk of severe disease was greatest [11].

There is a relationship between increased rates of hospitalization for asthma and increasing age at first episode of severe RSV disease, which may be due to the waning level of maternal antibody. The first 6 months of life is the time for rapid lung alveolar multiplication and airway remodeling. The alteration of the lower airway due to severe RSV disease in the first 6 months of life is transient and improves as the pathogen clears. Lung alveolarization is completed by age 2 - 3 years; hence, severe RSV disease beyond infancy will result in persistent adverse impact on lung development and function. Multiple hospitalizations for RSV disease in the first 2 years of life were not associated with an increased risk of subsequent asthma, compared with a single hospitalization for RSV disease. Passive immunization through maternal vaccination and active immunization in the first 2 years of life could lower the burden of acute and chronic childhood respiratory diseases associated with RSV later in life [12].

During infancy, the type of bacterial pathogens presents in the nasal area (nasal phalangeal microbiome) at the time of upper respiratory viral infections contributes as risk factor for the spread of infection to the lower airways causing inflammatory reaction. It may contribute directly and indirectly to the development of persistent asthma later, which is always associated with frequent episodes of infection of the nasal phalangeal microbiome [13].

We can prevent RSV infections and its exacerbation in high-risk asthmatic children by immune prophylaxis or vaccines. Other bacterial vaccination such as those against pneumococcal or H.B influenza may also aid in prevention [14].

Severe viral lower respiratory infections in developing countries such as RSV-bronchiolitis induce significant mortality. Susceptibility to severe RSV-bronchiolitis is governed by gene–environmental interactions that affect the host response to RSV infection. The composition and diversity of the microbiota, which in humans stabilizes in the first year of life, critically affects the development and function of the immune system and have a profound impact on the host response to RSV and susceptibility to childhood asthma. This knowledge will be harnessed for the prevention and treatment of severe viral bronchiolitis as a strategy to prevent the onset and development of asthma [15].

### Conclusion

The microbiome plays a critical role in protecting against asthma and allergies, particularly in early-life. Although, RSV-bronchiolitis predominantly affects infants under 6 months of age, it has an influence on the gut microbial colonization and microbial metabolites especially in severe RSV-bronchiolitis. Identifying the link between the gut microbiota and the development of lung mucosal immunity could have the potential to find new therapeutic ways in treatment of RSV-bronchiolitis and may prevent and ameliorate the potential current high rates of future allergic asthma among this group of infants [16].

### Acknowledgement

To Miss Lubna Sino, IBR Secretary, for read proving and editing.

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**Volume 9 Issue 3 March 2020**

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