

## Respiratory Infections by Virus: A Review Properties

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### ABSTRACT

The understanding of the causes and risk factors of respiratory diseases can go to the early treatments and prevention of the complications that are so prevalent in these pathologies. There are a variety of types of respiratory virus that are capable of respiratory disease begin. Rhinovirus, influenza, respiratory syncytial virus (RSV), adenovirus and coronavirus are capable of beginning of a common influenza to pneumonia, bronchitis and asthma. One of the important questions in the actual days is to relation the respiratory viral infection with the bronchial hyperresponsiveness? The mechanisms that cause sibilance, the relation between autonomic nervous system and viral infection, the association virus and respiratory diseases and the treatment there is in nowadays are relevant matters. In this work it was pretend to explore explanations about these themes in the scientific literature.

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### Introduction

The relation of viral infections and respiratory diseases is a theme of great controversy. There are many interest areas, as epidemiologic data, infections clinic characteristics, physio pathological means, the first bronchospasm episode, exacerbations and respiratory difficulties persistence. Many respiratory diseases affected some individuals with complications as a short time as long time [1]. The understanding of the causes and risk factors of these diseases can go to the early treatments and prevention of the complications that are so prevalent in these pathologies. Some information more recently compare the viral infection, manly by the respiratory virus in the first months of life, with atopy and allergy [2, 3].

### Respiratory Virus

There are a variety of types of respiratory virus that are capable of respiratory disease begin. Rhinovirus, influenza, respiratory syncytial virus (RSV), adenovirus and coronavirus are capable of beginning of a common influenza to pneumonia, bronchitis and asthma [4]. Recent works evidenced that the human metapneumovirus and the bocavirus are also capable of produce sibilance [5].

In majority, the respiratory infections in infancy are consequence of the infection by RSV [6], that causes approximately 50% of the bronchospasm and 80% of the cases of bronchiolitis. Approximately 70% of the children are infected by RSV in the first life year and almost all of them until the 3 years old.

On the children have more age, the rhinovirus is capable of provoke 60% of the acute respiratory infections [7]. The influenza virus occurs in the epidemics and the compromise grade has a relation to the type of viral injury and to the host resistance.

The clinic could vary of a simple respiratory tract superior infection to a serious pulmonary disease, with high mortality risk [8]. The parainfluenza virus affect any age group and it is particularly associated to the croup (laryngotracheobronchitis) in small child [9, 10]. The adenovirus cause, common colds, but could be associated the pneumonia. The coronavirus causes about of 15% of common colds and rarely are associated to pulmonary manifestations [12].

### Sibilance and Viral Infection Association

The respiratory virus is capable of produce exacerbations of the asthma, manly the rhinovirus [13]. It is evident that in the cold months of the year, when the viral infections are more frequency, occur a great number of sibilancy boards. It is also in this period that it was observed high rate of hospital care and admissions, especially of children. One of the important questions in the actual days is to relation the respiratory viral infection with the bronchial hyperresponsiveness [14], this is, the virus that produce bronchospasm in a child with pre-existing bronchial hyperresponsiveness or are capable to produce hyperresponsiveness by direct lesion in the bronchial epithelium?

One of the hypotheses more interesting was proposed by Holgate: The virus is capable of assault and destroy the bronchial epithelium, with cytokines release identified by epithelium injured cells [15]. Other irrelevant data of these study are the capacity of the virus of by attract cells as eosinophils and lymphocytes, the capacity of to produce specific IgE, to favour the liberation of histamine and mastocytes leukotrienes and to provoke the bradykinin release [15, 16].

In the last two decades, various investigators searched to correlate respiratory infection and sibilance. The conclusions were that viral respiratory infections, but not the bacterial, developed asthma [17-20]. McIntosh evaluated of prospective form 32 children between

1 and 5 years old, searching to determine if the exacerbations of asthma had relation with the viral respiratory infection [21]. During the 2 years were confirmed 102 viral respiratory infections and 139 sibilance episodes. Fifty-eight episodes (42%) had direct relation with viral respiratory infection, and the prevalent virus was the RSV.

Martinez, studying child since infant until 7/8 years-old and after 13 years, observed that the majority of the episodes of sibilance that occur during these years had relation with viral respiratory infection [22]. Four phenotypes were settled down: those who sibilance temporally (transitory sibilance), those who persisted with asthma (persistence sibilance), other with later begin asthma and finally those who not presented any sibilance episode until that day. In an early work, Martinez had published that the infant that sibilate with viral respiratory infection presented respiratory functional test alterations before of the first sibilance episode [23]. This happens mainly in boys. More recently, tried a relationship between this functional alterations with mother smoke.

In a study of 11 years in pediatric patients in the North Caroline, 6165 patients with inferior tract respiratory infection were evaluated and 1851 (30%) presented bronchospasm. The majority of the cases happened in child with less than 2 years-old [24, 25]. Some works evident factors that confirm the appearance of sibilance during respiratory viral infection: Children with less than 5 years, familiar history of atopy, macrobian agents like as RSV, parainfluenza, rhinovirus, influenza and Mycoplasma pneumoniae, symptoms coexistence as rhinorrhea, general malaise and increase of mucus secretion, occurrence in boys, pre-existence bronchial hyperactivity [26].

### Mechanisms of Viral Cause Sibilance

The actual knowledge about the viral injuries' repercussion to the superior respiratory tract is based in experimental infections by rhinovirus in human beings and in animal models [27]. The respiratory viruses can infect as well as the respiratory inferior or superior tract. After the bronchiolitis by respiratory syncytial virus, the viral antigen can be detected in the bronchial mucous [28]. The rhinovirus (RV), particularly the RV16, can be detected in the bronchial mucous by hybridization in situ in biopsies after the experimental infection.

The relevant data of these experiments show that the infected and lesioned epithelial cells secreted cytokines and chemokines (IL-1, IL-6, IL-11, GM-CSF, IL-8, RANTES e MIP1 $\alpha$ ) and other substances pro-inflammatory, as IFN- $\alpha$ , IFN- $\gamma$  e TNF- $\alpha$ , that keeps the cells recruitment (eosinophils, T-Cells, macrophages, neutrophils) to the infection local [29]. The viruses are capable also of increase the expression of adhesion molecules or MHC class 1 [30].

The epithelial lesion during the viral infection leads to the exposition of sensorial nerve terminals with release of neurotransmitters that have the fundamental goal in the inflammatory process, such as the P-substance (PS) and neurokinin (NKA, NKB) [31-33]. In consequence of participation of these mechanisms described, can be observed yet a major liberation of mastocytes mediators, mainly of histamines and leukotrienes (LKT4), increase the mucous secretion, bronchial mucosa edema and, as a result, major obstruction of airways [34]. The diminished of the nitric oxide production, sometimes detected during the viral infections of the inferior respiratory tract, can be other aggravating of the clinic board. This diminish can permit the prolongation of the viral replication, possibly causing more serious viral cause of sibilance [35].

### Autonomic Nervous System and Viral Infection

The viruses are capable of provoke beta-adrenergic block, but it seems not be an important event. The principal abnormality would be the intense parasympathetic predominance, perhaps due of the receptor M2-Muscarinic destruction or disfunction [36, 37]. Some virus destroys the receptors calico acid residues and, thus take along to this alteration. Another relevant data is consequence of the action of neurotransmitters not adrenergic no cholinergic as the substance P, strong inflammatory mediator.

### Association Virus and Respiratory Diseases

The RSV is capable of provoke allergic sensibilization? Some authors relate the viral aggression, the tissue lesion and the inhalers antigen penetration with the change of TH, para-TH, with the specific IgE antibody production [38-40]. Various works and studies come trying show the association between respiratory viral infection and asthma development. One of the first works, just cited previously, was published by Fernando Martinez (Tucson Children's Respiratory Study) [41]. Other study, as the SIDRIA, ALSPAC and PLAMA, published after, also related the asthma persistence in those with sibilance on the first years of life, associated to respiratory viral infection and asthma in the parents, high levels of IgE by occasion of the sibilance aspects [42].

### Treatment

There is much controversies yet about how would be the ideal treatment for the bronchospasm during the respiratory viral infections. Looks consensus the utilization of bronchodilators as the  $\beta_2$  agonists of short action, and the association to the Ipratropium Bromide in nebulization [43]. Based in the actual concepts of that the inflammatory process it is the event more important, it could not leave to valorisation the indication of the inhaled corticoid as inter crisis therapy by a period not inferior the 3 months [44]. Some works show that the cellular infiltrate, due of the action of cytokines, diminished, as well as the bronchial hyperresponsiveness [45-47]. Worth resalt that it is not indicate the association of  $\beta_2$  agonist of long action to inhaled corticoid in children with less than 4 years.

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