Journal of Biosciences and Medicines, 2026, 14(8), 244-253
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Analysis of Risk Factors for Wheezing Episodes Triggered by Rhinovirus Respiratory Tract Infection in Children

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How to cite this paper: Xiang, K.F. and Chen, L. (2026) Analysis of Risk Factors for Wheezing Episodes Triggered by Rhinovirus Respiratory Tract Infection in Children. *Journal of Biosciences and Medicines*, **14**, 244-253.
https://doi.org/10.4236/jbm.2026.148021

Received: July 22, 2026
Accepted: August 22, 2026
Published: August 25, 2026

Abstract

Acute respiratory infection (ARI) is the most common disease in pediatrics. Among the implicated respiratory pathogens, respiratory syncytial virus (RSV) and rhinovirus (RV) are the major pathogens causing wheezing in children. Pediatric wheezing is defined as a high-pitched, continuous, abnormal breath sound produced predominantly during the expiratory phase due to airway narrowing or obstruction that leads to airflow limitation; it typically manifests as a whistling or wheezing sound. With the continuous progress of molecular diagnostic techniques, human rhinovirus (HRV) infection has become increasingly prominent in wheezing disorders among preschool children. The risk factors for wheezing episodes following HRV infection involve multiple dimensions. This article will explore their roles from three perspectives: viral factors, host factors, and environmental factors. In terms of viral factors, it analyzes the virulence differences among different serotypes/genotypes. Regarding host factors, it examines the roles of age differences, atopic predisposition, and genetic susceptibility in triggering wheezing. With respect to environmental factors, it analyzes the differential effects of air quality, season, and allergen exposure on the induction of wheezing. This article aims to provide a reference for the early clinical identification of high-risk children and the optimization of intervention strategies, and to explore the relationship between RV infection and wheezing episodes in children, thereby offering strong support for optimizing treatment strategies and for the prevention and control of wheezing symptoms triggered by rhinovirus respiratory tract infection in children.

Keywords

Human Rhinovirus, Pediatric Wheezing, Risk Factors, Host Susceptibility, Viral Properties, Environmental Exposures, Immune Response

1. Introduction

Wheezing is one of the most common respiratory symptoms in childhood, with more than half of children experiencing at least one wheezing episode before the age of 6. Among the numerous pathogens, viral respiratory tract infections are the major factor inducing wheezing in children, and human rhinovirus (HRV) is one of the primary triggers of acute wheezing episodes in children [1]. According to relevant literature, in children over 2 years of age, HRV infection is one of the most common viral causes of wheezing, and among preschool children, HRV infection is the most common trigger of acute wheezing illnesses [2] [3]. However, the development of wheezing episodes following HRV infection is not a universal phenomenon; the majority of HRV infections manifest only as upper respiratory catarrhal symptoms, and only a subset of affected children present with wheezing. This disparity in clinical outcomes suggests the existence of specific risk factors and susceptibility mechanisms. Identifying these factors is of significant clinical importance for the early recognition of high-risk children and for guiding precise interventions.

In this review, HRV infection-induced wheezing is defined as HRV infection confirmed by nasopharyngeal swab nucleic acid testing, accompanied by wheezing symptoms. The inclusion criteria comprised a diagnosis of wheezing by the attending physician or the presence of wheeze and stridor on auscultation.

In recent years, the incidence of wheezing and asthma in children has shown a sustained upward trend worldwide. HRV-induced early wheezing has been confirmed as one of the important risk factors for the development of asthma in later childhood [4], and the interrelationship between HRV infection-induced wheezing and asthma has become a hot topic of research in the relevant field. According to previous studies, wheezing following HRV infection is not solely caused by direct viral injury, but rather results from the interplay of multiple factors, including host, viral, and environmental factors.

Host factors are one of the key contributors to wheezing episodes following HRV infection. For example, children with an atopic predisposition, such as those with a history of allergy, are more likely to develop more severe wheezing symptoms after HRV infection. Relevant studies have shown that among children with HRV infection, the proportion with a history of allergy is higher in those with HRV-C detection than in those with HRV-A or HRV-B infection [5]. Moreover, related studies have demonstrated that the majority of children with acute wheezing exhibit atopic manifestations, and sensitization to aeroallergens and food allergens is inextricably linked to the risk of recurrent wheezing in children [6].

Regarding viral factors, different HRV serotypes also differ in their contribution to the occurrence and severity of wheezing. Multiple studies have demonstrated that HRV-C strains exhibit higher pathogenicity than other serotypes. In a study conducted in Peru, the detection rate of HRV-C among hospitalized children reached as high as 73.31%, and it was significantly associated with symptoms including cough and wheezing [7]. Similarly, a study in Mexico found that HRV-C

infection was more strongly associated with more severe clinical manifestations and symptoms such as dyspnea [8].

Environmental factors also play a relatively important role in the occurrence and exacerbation of wheezing triggered by HRV infection. According to relevant studies, the detection rate of rhinovirus varies by season. In most regions of China, for instance, rhinovirus detection rates are predominantly concentrated in summer and autumn; in the Changchun area of Jilin Province, the detection rates were 46.43% in June 2019 and 48.78% in September 2020, respectively [9]. Meanwhile, air pollution, such as particulate matter ($PM_{2.5}$ and PM_{10}), is also correlated with HRV infection. An *in vitro* experiment demonstrated that exposure to diesel exhaust particles increased HRV replication in airway epithelial cells, and when combined with inhaled corticosteroids, this proviral replication effect exhibited supra-additivity [10]. These findings suggest that environmental factors may exacerbate airway injury and trigger wheezing episodes following HRV infection.

Host innate and adaptive immune responses to HRV are associated with the development of wheezing following infection. According to studies, among children who develop wheezing symptoms after HRV infection, serum levels of IL-23 and IL-17 are higher than those in non-wheezing children and healthy controls, and these cytokine levels decline during the convalescent phase, indicating that the severity of symptoms is correlated with the levels of IL-23 and IL-17 [11]. In addition, IgE levels are positively correlated with the severity of wheezing after RV infection [12].

In summary, wheezing episodes in children following HRV infection result from the interplay of multi-dimensional factors, including host genetic susceptibility, viral differences, and environmental exposure. Although a substantial body of research has identified these risk factors, a comprehensive framework for their systematic integration and analysis is still lacking. This review aims to sort out the risk factors for wheezing episodes after HRV infection in children, exploring them from four dimensions—host, viral, environmental, and immune—so as to provide a scientific basis for early clinical intervention.

2. Risk Factors

2.1. Host Factors

2.1.1. Age and Gender

The peak incidence of human rhinovirus (HRV)-associated wheezing episodes occurs in infancy and early childhood. According to epidemiological data, among children hospitalized for acute respiratory infection, the detection rate of HRV infection is highest in infants aged 0 - 5 months [7], and wheezing symptoms are relatively common in children aged two years and younger. This phenomenon may be related to the particularities of their airway anatomy and the immaturity of their immune system, leading to insufficient defense against viral infection. In addition, sex differences are associated with wheezing episodes. A birth cohort study conducted in Bangladesh found that among children under two years of age,

the number of boys with wheezing symptoms was higher than that of girls [13].

2.1.2. Genetic Susceptibility

The occurrence of wheezing following HRV infection is also closely associated with genetic factors. Genome-wide association studies (GWAS) have identified multiple genetic polymorphisms associated with an increased risk of HRV-related wheezing. Among these, genetic variants in the ORMDL3 gene are significantly associated with a markedly increased number of HRV-induced wheezing episodes in children; this gene influences viral replication and airway inflammation by regulating pathways such as sphingolipid metabolism, endoplasmic reticulum stress, and glycolysis [14]. In addition, the rs6967330-A variant of the cadherin-related family member 3 (CDHR3) gene has been confirmed as a risk factor for HRV-C infection and is associated with a high incidence of HRV-C-induced wheezing [5]. These genetic backgrounds collectively determine the host's susceptibility to HRV infection and the propensity for subsequent wheezing episodes.

2.1.3. Atopic Constitution

Atopic predisposition is an associated predictor of post-HRV wheezing in children. Children with a history of eczema, food allergy, or a family history of asthma are at significantly increased risk of developing wheezing following HRV infection. Studies have found that the proportion of children with a history of allergy and family history of asthma is higher among those with HRV-C infection than among those with HRV-A or HRV-B infection [5]. It should be noted, however, that the above evidence is derived primarily from cross-sectional or case-control studies, and large-scale prospective cohort studies are still lacking to confirm the independent predictive value of atopic predisposition for post-HRV wheezing episodes after adjusting for other factors. The synergistic interaction between allergen sensitization and viral infection is a key mechanism, and children sensitized to inhalant allergens are more susceptible to viral infections [15].

2.2. Viral Factors

2.2.1. Serotypes and Genotypes of HRV

HRV belongs to the genus Enterovirus within the family Picornaviridae and exhibits extremely high genetic diversity. Based on genomic, phylogenetic, and molecular characteristics, HRVs are primarily classified into three species: HRV-A, HRV-B, and HRV-C. Significant differences in pathogenicity exist among different serotypes and genotypes of HRV. RV-A and RV-C are relatively more likely than RV-B to cause severe illness and wheezing [16]. Multiple studies have consistently demonstrated that HRV-C is the major serotype responsible for severe wheezing and acute asthma exacerbations in children. In a study conducted in Peru, HRV-C had the highest detection rate among HRV-positive children and was significantly associated with symptoms such as cough, wheezing, and conjunctival injection [7]. Similarly, among hospitalized children in China, the proportions of children with a history of allergy, a family history of asthma, and the

presence of wheezing and asthma were significantly higher in those with HRV-C infection than in those with HRV-A or HRV-B infection [5].

However, the aforementioned studies are primarily analyses of hospitalized cases, mainly reflecting the association between HRV-C detection and severe disease. They cannot yet fully demonstrate that HRV-C directly predicts the onset of post-infectious wheezing—because HRV-C is also detectable in asymptomatic children, and wheezing outcomes are modulated by host and environmental factors. Future community-based longitudinal studies are needed to clarify the independent predictive role of HRV-C in the development of post-infectious wheezing.

2.2.2. Co-Infection with Other Viruses

Co-infection of HRV with other respiratory pathogens is an important associated factor that exacerbates the risk of wheezing. The most common co-infection pattern is simultaneous infection with HRV and respiratory syncytial virus (RSV). A systematic review has shown that the two most frequently detected viruses in children with wheezing symptoms are RSV and HRV, with detection rates of approximately 31.0% and 35.6%, respectively [1]. Simultaneous infection with these two viruses can further aggravate airway injury through synergistic inflammatory responses, leading to more severe clinical symptoms and higher hospitalization rates [17]. Furthermore, studies have found that among children experiencing their first wheezing episode induced by HRV, those co-infected with human bocavirus (HBoV) exhibit a distinct cytokine response profile, with decreased expression of inflammatory cytokines such as IL-1β and TNF-α during the acute phase, suggesting that HBoV may suppress the immune response induced by HRV [18]. However, the association between co-infection and wheezing severity is confounded by the fact that co-infections are frequently detected in hospitalized children, making it difficult to exclude detection bias due to inherently more severe illness. Currently, there is insufficient evidence to suggest that co-infection independently predicts wheezing episodes following HRV infection, and the causal relationship still requires further validation.

2.3. Environmental Factors

2.3.1. Allergen Exposure

Indoor allergen exposure is an important environmental cofactor for wheezing episodes following HRV infection. Common indoor allergens, such as dust mites, pet dander, and cockroach allergens, can enhance the HRV-induced Th2-type immune response. A prospective cohort study found that among HRV-positive patients with asthma, dust mite-specific IgE and total IgE levels were significantly higher than those in HRV-negative asthma patients, and the magnitude of dust mite sIgE elevation was positively correlated with the severity of HRV-induced asthma [12]. Therefore, a close relationship exists between HRV infection and allergens, which can induce sensitization or amplify pre-existing atopy, thereby promoting the development and progression of asthma [6]. These findings are largely

derived from observational studies, and a causal relationship between allergen exposure and post-HRV-infection wheezing has not yet been definitively established; however, controlling indoor allergen exposure is still regarded as one of the potential preventive strategies.

2.3.2. Air Pollution

Air pollution, particularly traffic-related and industrial pollution ($PM_{2.5}$, PM_{10}), NO_2, and O_3, has been demonstrated to be an independent risk factor for wheezing episodes following HRV infection. Air pollutants can impair airway epithelial barrier function through multiple mechanisms, including disruption of tight junction proteins, induction of oxidative stress, and inflammatory responses. A study conducted in Bangladesh found that longer durations of daily $PM_{2.5}$ concentrations exceeding 50 μg/m^3 were significantly associated with an elevated risk of wheezing in children [13]. In a study from Suzhou, China, the hospitalization rate for wheezing illnesses in children peaked in winter, which was associated with reduced indoor ventilation, accumulation of air pollutants, and increased viral transmission during winter [19]. Although these associations are consistent, most studies are ecological or time-series in design and cannot exclude confounding factors; only a few *in vitro* experiments have provided direct evidence that pollutants promote viral replication. Nevertheless, improving household ventilation, controlling humidity, and reducing mold growth may be important public health measures to lower the risk of wheezing in children.

2.4. Immune Response Characteristics

2.4.1. Th2-Skewed Immune Response

Th2-skewed immune response is a core immunological feature underlying wheezing episodes in atopic children following HRV infection. Compared with healthy children, atopic children exhibit significantly elevated levels of Th2-type cytokines (e.g., IL-4, IL-5, IL-13) and relatively decreased levels of Th1-type cytokines (e.g., IFN-γ) in peripheral blood and airways after HRV infection [20]. This immune imbalance leads to eosinophil recruitment and activation, as well as mast cell degranulation, thereby inducing airway hyperresponsiveness and wheezing symptoms. Studies have found that serum levels of IL-23 and IL-17 are significantly elevated in children who develop wheezing after HRV infection, suggesting that the IL-23/Th17 pathway is also involved in this process [11]. Furthermore, IgE-mediated stimulation can further enhance monocyte-driven Th2 differentiation by suppressing virus-induced type I interferons and inducing IL-10, thereby creating a positive feedback loop that promotes allergic inflammation [21].

2.4.2. Innate Immune Deficiency

Functional deficiencies of the innate immune system represent another important host factor that increases the risk of wheezing following HRV infection.

Upon HRV infection, airway epithelial cells recognize viral RNA through pattern recognition receptors (PRRs) and initiate type I and type III interferon sig-

naling pathways to restrict viral replication. Functional deficiencies in these receptors can impair the host's early recognition and clearance of HRV, leading to sustained viral replication and excessive inflammatory responses. Relevant studies have shown that children with deficiencies in type I and type III interferon responses at birth have a higher risk of subsequently developing lower respiratory tract infections and persistent wheezing or asthma compared with those without such deficiencies [22].

2.4.3. Airway Inflammatory Markers

Airway inflammatory markers can serve as one of the biomarkers for predicting the risk of HRV-related wheezing. Existing studies have confirmed that HRV infection can induce a pro-inflammatory response in airway epithelial cells, leading to the increased release of multiple cytokines, such as IL-1, IL-6, IL-8, and IL-11, which disrupt the epithelial barrier function and exacerbate clinical symptoms in affected children [23]. Furthermore, fractional exhaled nitric oxide (FeNO) is a non-invasive biomarker reflecting Th2-type airway inflammation. Although a study found that FeNO levels during the acute phase of wheezing were lower in children at high risk of asthma than in healthy controls [15], the value of FeNO in predicting future wheezing recurrence and asthma development is still widely recognized. Sputum eosinophilia is one of the direct pieces of evidence of Th2-type airway inflammation and is associated with the severity of wheezing after HRV infection. The combined detection of these markers can help identify high-risk children more accurately and guide individualized prevention and treatment strategies.

3. Limitations

This review has the following limitations in its analysis of risk factors for wheezing episodes triggered by respiratory rhinovirus infection. Most of the included studies employed PCR methods to detect HRV; however, HRV infection can also be detected in asymptomatic children, making it difficult to establish a definitive causal relationship between HRV positivity and wheezing. The definition of wheezing varied across studies—for instance, some relied on parental reports even though the child presented without wheezing symptoms or without audible wheeze or stridor on lung auscultation at the time of the visit, and auscultatory findings also differed among clinicians, thereby limiting the comparability of the results. A history of asthma or atopic predisposition often acted as a confounding factor, but some studies did not adequately adjust for it, resulting in residual confounding that may affect the determination of independent risk factors. Therefore, future prospective cohort studies with standardized criteria, large sample sizes, and multicenter designs are needed to overcome the aforementioned limitations.

4. Conclusions and Future Perspectives

This review systematically collates and summarizes, based on previous studies, the

multi-level risk factors for wheezing episodes in children following human rhinovirus (HRV) infection, encompassing host susceptibility, viral characteristics, environmental exposure, and immune response features. The available evidence indicates that these factors do not act independently, but rather jointly influence the risk of wheezing episodes through their interactions. Most of the current evidence derives from observational studies, and the associations demonstrated do not equate to causal relationships. Therefore, in clinical practice, particular attention should be paid to infants and young children with an atopic predisposition and exposure to adverse environmental conditions, and the frequency of wheezing episodes should be effectively reduced through early interventions such as allergen avoidance, environmental improvement, and immunomodulation.

When reconciling the perspectives and findings across different studies, it is important to note the variability of atopic predisposition among different populations, and the consequences of the interplay among the aforementioned factors remain to be fully elucidated. Future research should focus on developing clinical prediction tools that integrate multiple factors to improve the accuracy of identifying high-risk populations. Exploration of targeted therapies directed against HRV-C or the Th2 pathway to block the inflammatory cascade is warranted; however, these interventions, such as immunomodulators, anti-HRV-C monoclonal antibodies, and Th2 pathway inhibitors, are currently in the preclinical or early clinical trial stages, and their efficacy and safety in preventing HRV-related wheezing still require validation through large-scale randomized controlled trials. Large-scale longitudinal cohort studies should be conducted to validate the interactions among various risk factors and to clarify the optimal timing of intervention, thereby reducing the progression from HRV-related wheezing to asthma and improving long-term respiratory health outcomes in children through the precise identification of high-risk populations and the implementation of individualized preventive strategies.

Author Contributions

Conceptualization, K.F.X. and L.C.; methodology, K.F.X. and L.C.; validation, K.F.X. and L.C.; investigation, K.F.X. and L.C.; resources, K.F.X. and L.C.; writing—original draft preparation, K.F.X.; writing—review and editing, L.C.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Kengne–Nde, C., Kenmoe, S., Modiyinji, A.F. and Njouom, R. (2020) Prevalence of Respiratory Viruses Using Polymerase Chain Reaction in Children with Wheezing, a Systematic Review and Meta-Analysis. *PLOS ONE*, **15**, e0243735. https://doi.org/10.1371/journal.pone.0243735

[2] Jackson, D.J., Gangnon, R.E., Evans, M.D., Roberg, K.A., Anderson, E.L., Pappas, T.E., *et al.* (2008) Wheezing Rhinovirus Illnesses in Early Life Predict Asthma Devel-

opment in High-Risk Children. *American Journal of Respiratory and Critical Care Medicine*, **178**, 667-672. https://doi.org/10.1164/rccm.200802-309oc

[3] Basnet, S., Palmenberg, A.C. and Gern, J.E. (2019) Rhinoviruses and Their Receptors. *Chest*, **155**, 1018-1025. https://doi.org/10.1016/j.chest.2018.12.012

[4] Jackson, D.J. and Gern, J.E. (2022) Rhinovirus Infections and Their Roles in Asthma: Etiology and Exacerbations. *The Journal of Allergy and Clinical Immunology*: *In Practice*, **10**, 673-681. https://doi.org/10.1016/j.jaip.2022.01.006

[5] Fu, H., De, R., Sun, Y., Yao, Y., Zhu, R., Chen, D., *et al.* (2025) Association between Cadherin-Related Family Member 3 rs6967330-A and Human Rhinovirus-C Induced Wheezing in Children. *Virology Journal*, **22**, Article No. 29. https://doi.org/10.1186/s12985-025-02644-7

[6] Yuan, X.H., Xin, R. and Ye, X.H. (2025) Research Progress on Rhinovirus Infection. *Chinese Journal of Viral Diseases*, **15**, 403 410. (In Chinese)

[7] Castañeda-Ribeyro, A., Martins-Luna, J., Verne, E., Aguila-Luis, M.A., Silva-Caso, W., Ugarte, C., *et al.* (2022) High Prevalence and Clinical Characteristics of Respiratory Infection by Human Rhinovirus in Children from Lima-Peru during Years 2009-2010. *PLOS ONE*, **17**, e0271044. https://doi.org/10.1371/journal.pone.0271044

[8] Galindo-Fraga, A., Guerra-de-Blas, P.d.C., Ortega-Villa, A.M., Mateja, A., Ruiz Quiñones, J.A., Ramos Cervantes, P., *et al.* (2022) Different Clinical Presentations of Human Rhinovirus Species Infection in Children and Adults in Mexico. *Open Forum Infectious Diseases*, **9**, ofac303. https://doi.org/10.1093/ofid/ofac303

[9] Xia, B.C., Ma, Y.W., Sun, L.W., *et al.* (2022) Molecular Typing and Infection Status of Rhinovirus among Children with Acute Respiratory Tract Infections in Changchun, 2019-2021. *Disease Surveillance*, **37**, 469-474. (In Chinese)

[10] Huff, R.D., El-Bittar, N., Watts, E.R., Yoon, M., Afshar, T., Wiehler, S., *et al.* (2025) Increased Rhinovirus Replication Following Corticosteroid and Air Pollution Exposures. *Environmental Research*, **281**, Article ID: 121970. https://doi.org/10.1016/j.envres.2025.121970

[11] Teng, S., Yu, M., Qi, Z., Zhao, S. and Wang, H. (2025) Expression Analysis of the IL-23/Th17 Pathway in Children with Rhinovirus Infection. *Italian Journal of Pediatrics*, **51**, Article No. 230. https://doi.org/10.1186/s13052-025-02068-7

[12] Kantor, D.B., Stenquist, N., McDonald, M.C., Schultz, B.J., Hauptman, M., Smallwood, C.D., *et al.* (2016) Rhinovirus and Serum Ige Are Associated with Acute Asthma Exacerbation Severity in Children. *Journal of Allergy and Clinical Immunology*, **138**, 1467-1471.e9. https://doi.org/10.1016/j.jaci.2016.04.044

[13] Shoshi, H.R., Pyash, A.S., Biswas, M.A.A.J., Riaj, M.A.A., Rahman, M., Haque, R., *et al.* (2026) Incidence, Viral Etiology, and Risk Factors of Wheezing in Children under Two Years of Age: Findings from a Low-Income Urban Community in Dhaka, Bangladesh. *BMC Pulmonary Medicine*, **26**, Article No. 237. https://doi.org/10.1186/s12890-026-04281-4

[14] Zhang, Y. (2021) Orosomucoid-Like Protein 3, Rhinovirus and Asthma. *World Journal of Critical Care Medicine*, **10**, 170-182. https://doi.org/10.5492/wjccm.v10.i5.170

[15] Wang, T., Dong, H., Jiang, W., Li, Y., Sun, H., Huang, L., *et al.* (2020) Viral Etiology and Atopic Characteristics in High-Risk Asthmatic Children Hospitalized for Lower Respiratory Tract Infection. *Translational Pediatrics*, **9**, 541-550. https://doi.org/10.21037/tp-20-165

[16] Choi, T., Devries, M., Bacharier, L.B., Busse, W., Camargo, C.A., Cohen, R., *et al.*

(2021) Enhanced Neutralizing Antibody Responses to Rhinovirus C and Age-Dependent Patterns of Infection. *American Journal of Respiratory and Critical Care Medicine*, **203**, 822-830. https://doi.org/10.1164/rccm.202010-3753oc

[17] Comte, A., Bour, J., Darniot, M., Pitoiset, C., Aho-Glélé, L.S. and Manoha, C. (2020) Epidemiological Characteristics and Clinical Outcomes of Human Rhinovirus Infections in a Hospitalized Population. Severity Is Independently Linked to RSV Coinfection and Comorbidities. *Journal of Clinical Virology*, **125**, Article ID: 104290. https://doi.org/10.1016/j.jcv.2020.104290

[18] Hurme, P., Sahla, R., Rückert, B., Vahlberg, T., Turunen, R., Vuorinen, T., *et al.* (2023) Human Bocavirus 1 Coinfection Is Associated with Decreased Cytokine Expression in the Rhinovirus-Induced First Wheezing Episode in Children. *Clinical and Translational Allergy*, **13**, e12311. https://doi.org/10.1002/clt2.12311

[19] Huang, J.Q., Zhang, J., Hao, C.L. and Chen, Z.R. (2022) Association of Children Wheezing Diseases with Meteorological and Environmental Factors in Suzhou, China. *Scientific Reports*, **12**, Article No. 5018. https://doi.org/10.1038/s41598-022-08985-5

[20] Price, A.S. and Kennedy, J.L. (2022) T-Helper 2 Mechanisms Involved in Human Rhinovirus Infections and Asthma. *Annals of Allergy, Asthma & Immunology*, **129**, 681-691. https://doi.org/10.1016/j.anai.2022.08.015

[21] Rowe, R.K., Pyle, D.M., Farrar, J.D. and Gill, M.A. (2020) IgE-Mediated Regulation of IL-10 and Type I IFN Enhances Rhinovirus-Induced Th2 Differentiation by Primary Human Monocytes. *European Journal of Immunology*, **50**, 1550-1559. https://doi.org/10.1002/eji.201948396

[22] Read, J.F. and Bosco, A. (2020) Decoding Susceptibility to Respiratory Viral Infections and Asthma Inception in Children. *International Journal of Molecular Sciences*, **21**, Article 6372. https://doi.org/10.3390/ijms21176372

[23] Gern, J.E. (2015) How Rhinovirus Infections Cause Exacerbations of Asthma. *Clinical & Experimental Allergy*, **45**, 32-42. https://doi.org/10.1111/cea.12428