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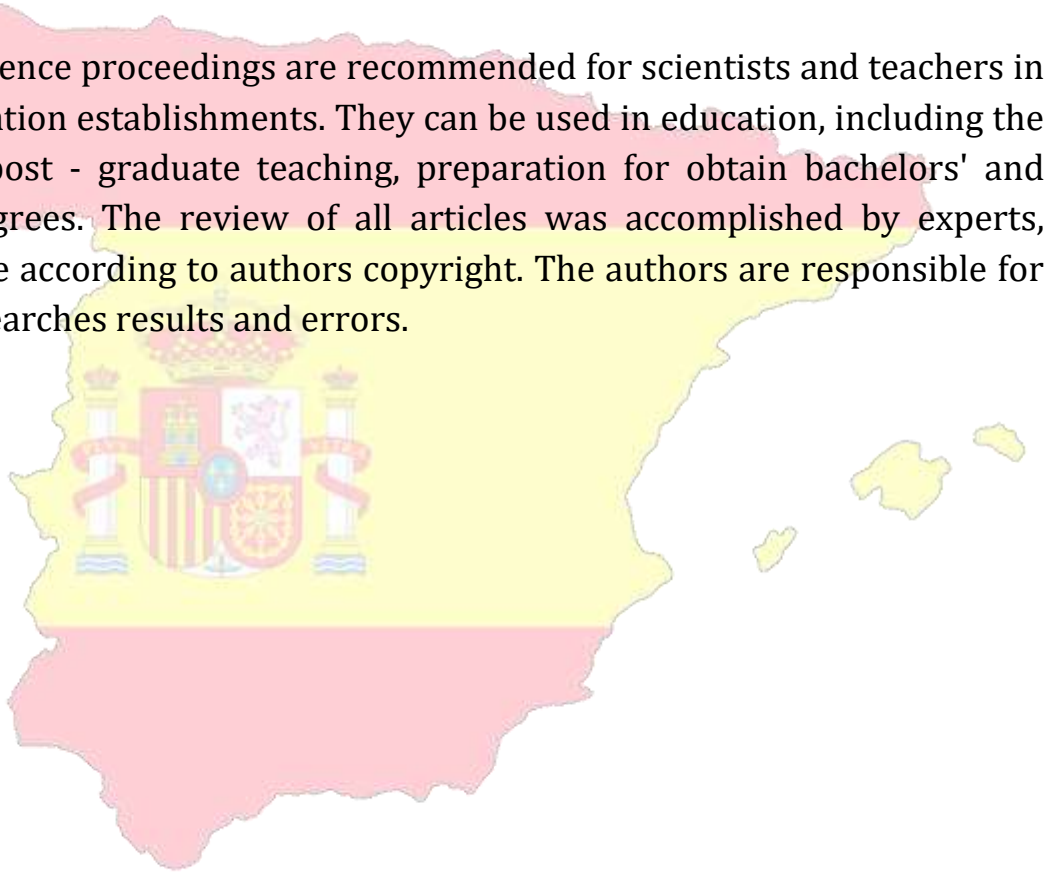

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Etiology, pathogenesis, and clinical characteristics of acute respiratory viral infections in children

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Abstract: Acute respiratory viral infections (ARVI) - represent the most common group of infectious diseases in childhood and constitute a major public health burden worldwide. These infections are caused by a wide spectrum of respiratory viruses and are characterized by high transmissibility, seasonal variability, and diverse clinical manifestations. Despite substantial advances in molecular diagnostics and virology, ARVI continues to pose challenges due to viral diversity, frequent genetic mutations, and limited availability of universal antiviral therapies. This article presents a theoretical and analytical overview of the etiology, pathogenesis, and clinical characteristics of ARVI in children based on contemporary scientific literature and epidemiological data. Emphasis is placed on viral classification, mechanisms of host–virus interaction, immunopathogenesis, and age-related susceptibility. Statistical trends illustrating global incidence, morbidity patterns, and healthcare impact are also summarized. The review does not include individual patient data or clinical case descriptions; instead, it focuses exclusively on population-level analyses, theoretical frameworks, and evidence-based concepts. Understanding the biological mechanisms underlying ARVI and their clinical expression in pediatric populations is essential for improving preventive strategies, optimizing supportive care, and guiding future research aimed at reducing disease burden.

Keywords: Acute respiratory viral infections, children, etiology, pathogenesis, respiratory viruses, immunity, epidemiology, clinical features, inflammation, pediatrics, transmission, prevention

Introduction: Acute respiratory viral infections (ARVI) - are a heterogeneous group of infectious diseases affecting the upper and lower respiratory tract and are predominantly caused by viral pathogens. In pediatric populations, ARVI accounts for the majority of infectious episodes encountered in outpatient and hospital settings. Global epidemiological analyses indicate that children experience an average of 4–8 episodes of ARVI per year, a rate significantly higher than that observed in adults. This elevated susceptibility is primarily related to the immaturity of the immune system, limited immunological memory, and frequent exposure to communal environments such as schools and childcare institutions.

The public health significance of ARVI extends beyond individual morbidity. These infections are responsible for substantial healthcare utilization, including outpatient visits, hospital admissions, and indirect economic losses related to parental work absence. According to international surveillance systems, ARVI

contributes to millions of hospitalizations annually among children under five years of age, particularly in low- and middle-income countries. Mortality rates are relatively low in otherwise healthy children but increase significantly in infants, immunocompromised individuals, and those with chronic comorbidities.

From a biological perspective, ARVI encompasses infections caused by diverse viral families, including Orthomyxoviridae, Paramyxoviridae, Coronaviridae, Adenoviridae, Picornaviridae, and others. The high genetic variability of many respiratory viruses, particularly RNA viruses, facilitates rapid evolution and periodic emergence of new strains. This variability complicates long-term immunity and contributes to recurrent infections throughout childhood.

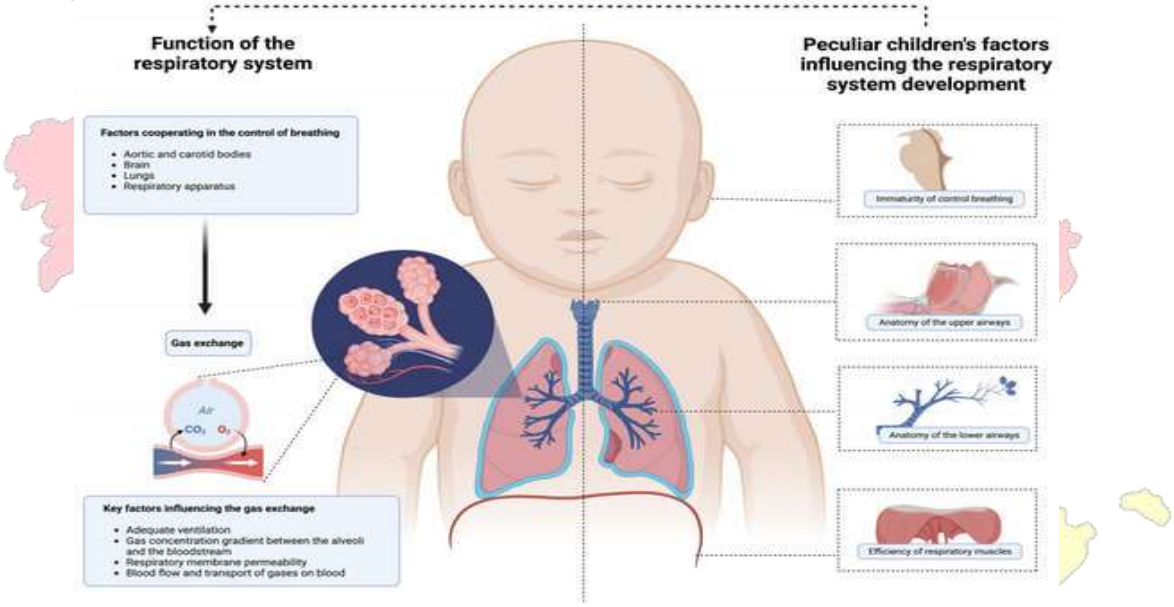

Figure 1. Pathophysiology of acute respiratory failure in infants and young children.

Clinically, ARVI exhibits a broad spectrum of manifestations, ranging from mild rhinorrhea and cough to severe lower respiratory tract involvement, including bronchiolitis and viral pneumonia. While many infections are self-limiting, their cumulative impact on child health, growth, and development is substantial.

Repeated episodes of ARVI during early childhood have been associated with increased risk of wheezing disorders, impaired lung function, and secondary bacterial infections.

Understanding ARVI in children requires an integrated approach that considers etiological diversity, pathogenetic mechanisms, and age-specific clinical features. Advances in molecular virology and immunology have provided new insights into virus–host interactions, innate immune responses, and inflammatory pathways involved in respiratory viral infections. However, translation of this knowledge into universally effective preventive and therapeutic strategies remains incomplete.

This article aims to synthesize theoretical, clinical, and statistical data on ARVI in children, with a focus on etiology, pathogenesis, and clinical characteristics. By emphasizing scientific analysis rather than individual clinical cases, the review

seeks to provide a comprehensive conceptual framework for understanding ARVI as a complex and dynamic group of pediatric infectious diseases.

Materials and Methods: This article is based on a structured analysis of peer-reviewed scientific literature obtained from major international biomedical databases, including PubMed, Scopus, Web of Science, and Google Scholar. The search strategy employed combinations of key terms such as “acute respiratory viral infection,” “children,” “etiology,” “pathogenesis,” “clinical features,” and “epidemiology.” Publications from the last two decades were prioritized, while seminal older works of high scientific relevance were also considered.

Eligible sources included original research articles, systematic reviews, meta-analyses, epidemiological reports, and doctoral dissertations focusing on pediatric ARVI. Only publications written in English and presenting clearly defined methodologies were included. Studies addressing bacterial respiratory infections without a viral component were excluded.

The selection process involved an initial screening of titles and abstracts, followed by full-text evaluation to assess relevance and scientific rigor. Emphasis was placed on studies providing theoretical insights into viral biology, immune responses, and clinical patterns rather than those centered on specific therapeutic interventions or individual patient case series.

Extracted information was organized into thematic categories: viral etiology, mechanisms of pathogenesis, age-related immune responses, and clinical manifestations. Epidemiological data were synthesized to identify global trends in incidence, seasonal distribution, and disease burden.

The analytical approach was narrative and integrative, allowing comparison and synthesis of findings across diverse sources. No new experimental or clinical data were generated. The review adheres to principles of scientific neutrality and aims to present a balanced representation of current knowledge.

Results: Analysis of the selected literature confirms that ARVI in children is caused predominantly by a limited number of viral groups, with rhinoviruses, respiratory syncytial virus, influenza viruses, parainfluenza viruses, adenoviruses, and coronaviruses accounting for the majority of cases. Rhinoviruses alone are responsible for approximately 30–50% of all upper respiratory infections in children.

Epidemiological data indicate that children under five years of age experience the highest incidence rates, with peak occurrence during autumn and winter in temperate climates. Hospitalization rates for ARVI-related lower respiratory tract infections are highest among infants under one year of age.

Pathogenetic studies consistently demonstrate that viral replication within respiratory epithelial cells initiates a cascade of innate immune responses, including interferon production and pro-inflammatory cytokine release. Excessive or dysregulated inflammatory responses are associated with more severe clinical presentations.

Clinical data synthesized from large observational studies show that nasal congestion, rhinorrhea, cough, and fever are the most common manifestations. Lower respiratory involvement occurs in a significant proportion of cases, particularly with respiratory syncytial virus and influenza viruses.

Collectively, these findings highlight the multifactorial nature of ARVI in children, integrating viral characteristics, host immunity, and environmental factors.

Discussion: The etiology of ARVI in children is notable for its remarkable diversity. Unlike many infectious diseases caused by a single dominant pathogen, ARVI represents a syndrome produced by numerous viruses with distinct biological properties. This diversity contributes to the persistence of ARVI as a global health problem despite decades of research.

RNA viruses dominate the etiological spectrum of ARVI. Their high mutation rates enable continuous genetic evolution, facilitating immune evasion and repeated infections. For children, whose immunological memory is still developing, this results in frequent exposure to novel viral antigens. DNA viruses, such as adenoviruses, exhibit greater genetic stability but possess mechanisms for immune modulation and prolonged persistence.

Pathogenesis begins with viral entry through the nasal or oral mucosa, followed by attachment to specific cellular receptors on respiratory epithelial cells. Viral replication disrupts epithelial integrity and impairs mucociliary clearance, reducing the effectiveness of a primary mechanical defense mechanism. This local damage facilitates viral spread along the respiratory tract.

Innate immunity plays a central role in early defense. Pattern recognition receptors detect viral components and trigger interferon signaling pathways. Interferons inhibit viral replication and activate natural killer cells and macrophages. In children, these responses are functional but often quantitatively and qualitatively different from those in adults, contributing to variable disease expression.

Adaptive immune responses, including virus-specific T lymphocytes and antibody production, develop over several days. Secretory IgA antibodies are particularly important for mucosal protection, but their production is limited in early childhood. Consequently, children rely heavily on innate immunity, which may be insufficient to prevent symptomatic infection.

Inflammation represents a double-edged sword in ARVI. While necessary for viral clearance, excessive inflammatory responses contribute to tissue damage and symptom severity. Cytokines such as interleukin-6 and tumor necrosis factor-alpha are associated with fever, malaise, and systemic symptoms. In the lower respiratory tract, inflammation can lead to airway obstruction, particularly in small-caliber bronchioles.

Clinical characteristics of ARVI reflect this underlying pathophysiology. Upper respiratory tract involvement predominates, but lower respiratory tract disease is common in younger children. The anatomical and physiological features of

pediatric airways—smaller diameter, higher resistance, and greater susceptibility to edema—predispose infants to more severe manifestations.

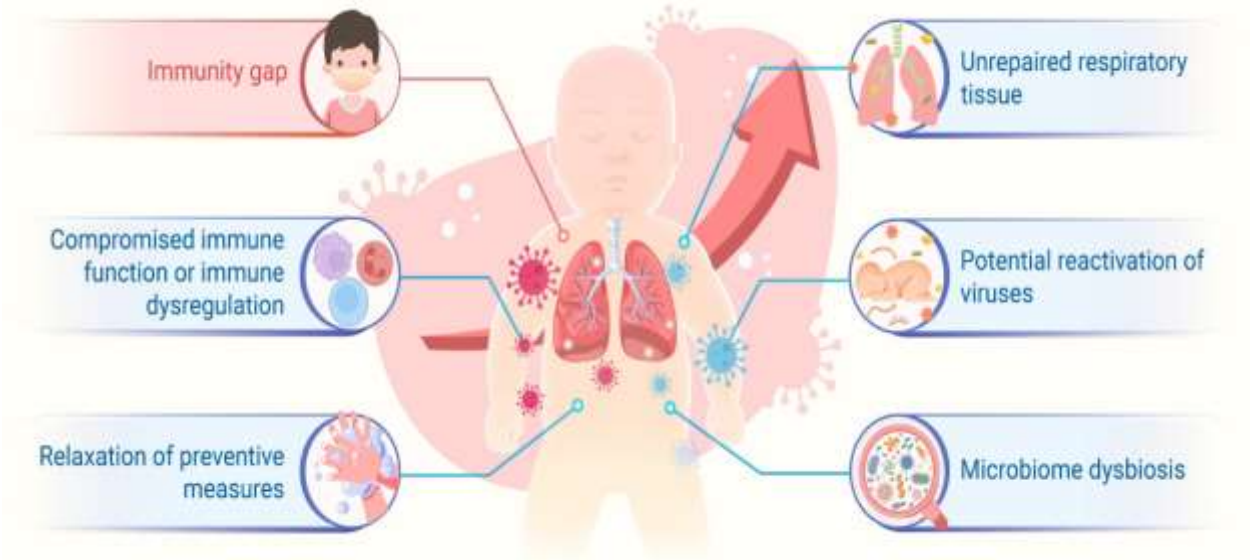

Figure 2. The possible reasons for surging of respiratory infection post-COVID-19.

Age-related differences are a consistent theme in ARVI research. Neonates and young infants demonstrate higher rates of hospitalization and complications, while older children typically experience milder disease. This pattern underscores the importance of immune maturation in determining disease outcome.

Statistical analyses reveal that ARVI incidence has remained relatively stable over recent decades, despite advances in diagnostics and preventive measures. Vaccination against influenza has reduced disease burden in immunized populations, but coverage remains variable, and vaccines are virus-specific rather than universally protective.

Environmental and social factors also influence ARVI transmission. Crowded living conditions, indoor air pollution, and attendance at childcare facilities are associated with increased risk. Seasonal variation is driven by changes in temperature, humidity, and human behavior.

From a theoretical standpoint, ARVI can be viewed as an inevitable consequence of human–virus coexistence. Complete eradication is unlikely, given the vast reservoir of respiratory viruses and their evolutionary adaptability. Therefore, the primary goals of public health strategies are to reduce severity, prevent complications, and protect high-risk groups.

The synthesis of etiological, pathogenetic, and clinical data highlights the complexity of ARVI in children. No single factor determines disease outcome; rather, it is the result of dynamic interactions between virus, host, and environment. Continued research into immune mechanisms, viral evolution, and preventive strategies is essential for future progress.

Conclusion: Acute respiratory viral infections in children represent a complex group of diseases characterized by etiological diversity, multifaceted pathogenesis,

and variable clinical expression. High incidence rates are driven by the biological properties of respiratory viruses, the immaturity of pediatric immune systems, and environmental exposure. Pathogenesis involves viral replication in respiratory epithelium, activation of innate and adaptive immunity, and inflammatory responses that determine symptom severity. Clinical manifestations range from mild upper respiratory symptoms to significant lower respiratory involvement, particularly in younger children. Theoretical and statistical evidence underscores the persistent global burden of ARVI despite medical advances. A comprehensive understanding of etiological agents, immune mechanisms, and age-related factors is fundamental for developing effective preventive and supportive strategies aimed at reducing morbidity and improving child health outcomes.

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