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Review

Reinfection Mechanisms of Various Viruses and Their Societal Implications

Ryusuke Kimura [1,2,†], Yuriko Hayashi [3,†], Yuka Sato-Fujimoto [4], Kei Miyakawa [5], Kazuya Shirato [6], Koo Nagasawa [7], Fuminori Mizukoshi [6], Takeshi Tsugawa [8], Akihide Ryo [4] and Hirokazu Kimura [2,3,*]

[1] Department of Bacteriology, Graduate School of Medicine, Gunma University, Maebashi-shi 371-8511, Gunma, Japan
[2] Advanced Medical Science Research Center, Gunma Paz University, Takasaki-shi 370-0006, Gunma, Japan
[3] Department of Health Science, Graduate School of Health Sciences, Gunma Paz University, Takasaki-shi 370-0006, Gunma, Japan
[4] Faculty of Healthcare, Tokyo Healthcare University, Setagaya-ku 141-8648, Tokyo, Japan
[5] Research Center for Influenza and Respiratory Viruses, National Institute of Infectious Diseases, Musashimurayama-shi 208-0011, Tokyo, Japan
[6] Department of Virology III, Infectious Disease Surveillance Center, National Institute of Infectious Diseases, Musashimurayama-shi 208-0011, Tokyo, Japan
[7] Department of Pediatrics, Chiba University Hospital, Chiba-shi 260-8670, Chiba, Japan
[8] Department of Pediatrics, Sapporo Medical University School of Medicine, Sapporo-shi 060-8543, Hokkaido, Japan
* Correspondence: h-kimura@paz.ac.jp
† These authors contributed equally to this work.

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Abstract: Viral infections involve numerous pathogens, some of which allow reinfection while others, such as measles virus, provide lifelong immunity. The differences in reinfection mechanisms can be attributed to variations in viral antigenicity and host immune responses. Measles virus exhibits highly conserved hemagglutinin (HA) proteins, where neutralizing antibody-binding regions overlap with host receptor-binding sites, resulting in effective immune protection against reinfection. In contrast, influenza viruses undergo rapid antigenic evolution driven by immune selection pressures, leading to immune escape variants that facilitate annual reinfections. SARS-CoV-2, similarly, shows frequent mutations in its spike protein receptor-binding domain (RBD), contributing to reinfection despite prior immunity from vaccination or infection. Respiratory syncytial virus (RSV) and human respirovirus type 3 (HRV3) are monoserotype viruses capable of lifelong reinfections. Structural analyses indicate that their conformational epitopes do not align with neutralizing antibody-binding sites, undermining the effectiveness of immune responses. To better understand these mechanisms highlights the interplay between viral evolution and host defenses, providing essential insights for developing targeted vaccines and therapeutic strategies to combat respiratory virus reinfections. Moreover, understanding of the reinfection mechanisms regarding various virus infections may significantly influence public health policies, emphasizing the need for effective vaccination strategies, risk communication, and consideration of cultural factors to address challenges in vaccine adoption, health behaviors, and societal stigma.

Keywords: reinfection; respiratory viruses; measles virus; epitope; vaccine

1. Introduction

Despite significant advancements in virology, the number and pathogenic details of pathogens responsible for respiratory infections remain unclear. Previous studies have suggested that at least hundreds of viruses are involved in these diseases [1]. Moreover, it is well known that many respiratory viral infections allow reinfection with the same pathogen [2]. However, some viral infections, such as measles, are characterized as "No Reinfection," where reinfections are either absent or exceedingly rare [3–8]. To elucidate these differences, the study was conducted using pioneering bioinformatics techniques, focusing on both the evolution of viral antigens and host immune defenses. Recent advances in in silico analyses, including bioinformatics, have facilitated not only the study of viral genome evolution but also enabled the prediction of viral antigen protein structures/antigenicity [9]. These analyses further contribute to discussions on the immunogenicity of viruses in hosts and the efficacy of vaccines [9]. This review may provide an overview of the mechanisms underlying reinfection with several viruses based on these two perspectives using authentic bioinformatics technologies. Moreover, this review also explores the significance of reinfection mechanisms in the context of social science and public health.

2. Interactions Among Viral Antigens, Cellular Receptors, Neutralizing Antibodies, and Epitope Motifs

Most viruses initiate infections by binding specifically to various molecules (viral receptors) on the host cell surface. Proteins found inside or on the surface of viral particles, which bind to these receptors, act as foreign antigens that stimulate the host's innate and humoral immune responses, resulting in the production of IgM, IgG, or IgA antibodies [10]. Neutralizing antibodies, which can effectively inhibit viral infection, are induced when these antibodies target the receptor-binding sites or their surrounding regions on viral antigens. The motifs on antigenic proteins that induce antibody production are referred to as epitopes. Epitopes are classified into linear epitopes, determined by the amino acid sequence, and conformational epitopes, which arise from the three-dimensional structure of the protein [10]. Previous reports suggests that the majority of epitopes on viral antigens are conformational [11]. These molecular interactions play a crucial role in viral infection and immune defense, providing a "key" to understanding the reinfection mechanisms observed in many viruses [9].

3. Mechanisms Behind Measles Virus What Is Known as "No Reinfection"

Measles virus, an RNA virus from the genus *Morbillivirus* of the family *Paramyxoviridae*, is known for its high infectivity. Although over 20 genotypes of measles virus exist, these are considered to be monoserotype [12]. Epidemiologically, natural measles infections are associated with lifelong immunity [12]. The major antigens of the measles virus are hemagglutinin protein (HA) and fusion protein (F) [13]. The HA protein plays a critical role in the early stages of infection by facilitating adsorption and entry into host cells via the SLAM receptor (CD150) [14,15]. Neutralizing antibodies against HA inhibit the binding of HA to SLAM, effectively preventing infection [16]. Molecular studies on HA proteins from 24 genotypes showed minimal amino acid substitutions in the neutralizing antibody-binding regions and SLAM-binding sites, suggesting highly conserved antigenicity. This conservation likely explains why measles infections are rarely followed by reinfection (Figure 1).

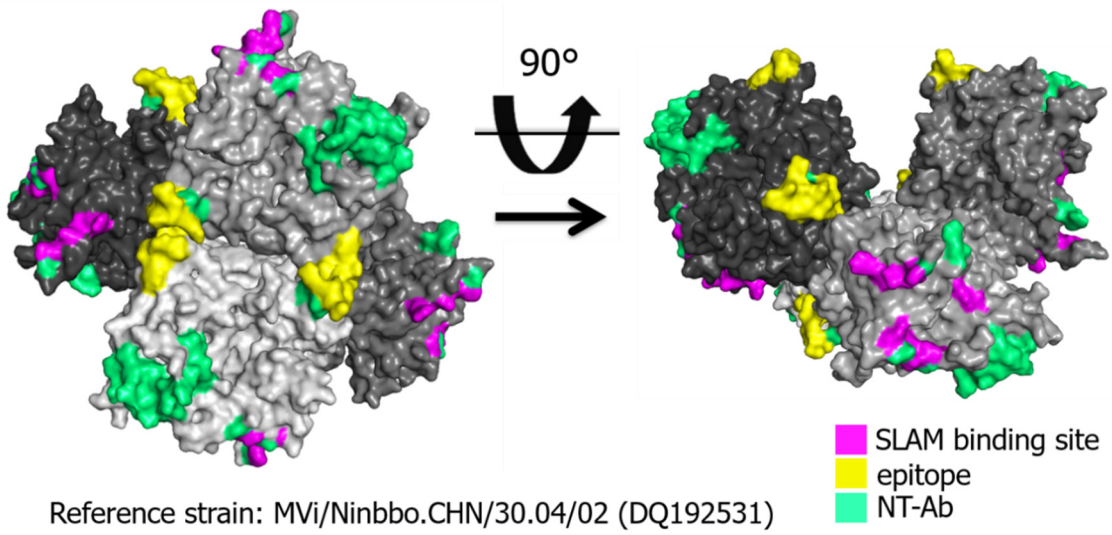

Figure 1. Mapping of the cellular receptor (SLAM) binding site, conformational epitope motifs, and neutralizing antibody binding sites on the structural model of the measles virus H1 genotype H protein tetramer. Each peptide

chain of the tetramer is represented in distinct shades: dark gray (chain A), gray (chain B), light gray (chain C), and off-white (chain D). The conformational epitopes, cellular receptor binding sites, and neutralizing antibody binding sites are shown in magenta, yellow, and lime green, respectively. The figure presented in this review is original.

4. Mechanisms of Influenza Virus Reinfection

Influenza viruses, classified into the genus *Influenzavirus* of the family *Orthomyxoviridae*, circulate annually as seasonal flu, primarily in the form of subtype AH1, subtype AH3, and subtype B. Similar to the measles virus, the HA protein is essential for host cell infection, binding to sialic acid receptors. However, unlike measles, influenza HA evolves rapidly (over 10^{-3} nucleotide/site/year, s/s/y), driven by positive selection pressures from host immune defenses (Figure 2) [17]. This rapid evolution generates immune escape variants, enabling reinfections. Structural analyses of the HA protein reveal numerous amino acid substitutions, especially in receptor-binding and immune interaction sites, underscoring its capacity for antigenic drift [18].

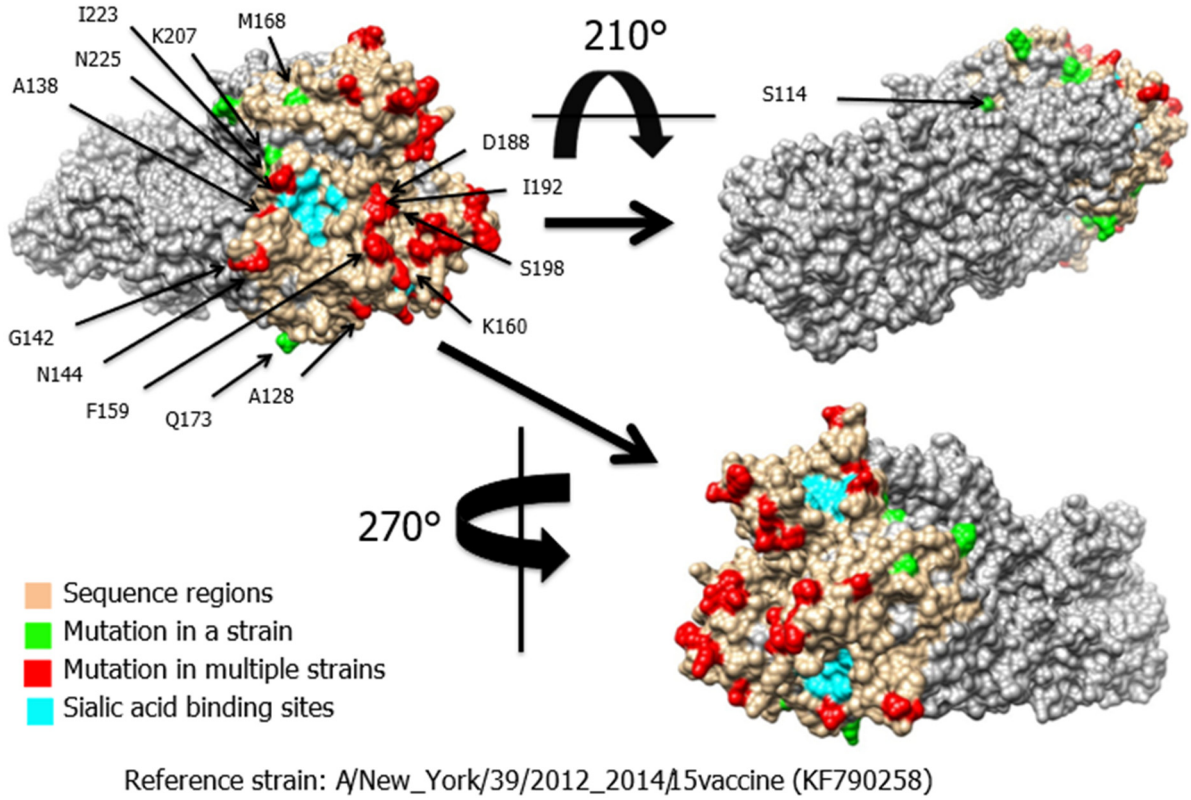

Figure 2. Mapping of amino acid mutation sites, sialic acid-binding sequence regions, and amino acid mutation sites specific to subtype AH3 on the structural model of influenza virus H3 subtype H protein. Amino acid mutation sites common across other subtypes, the sialic acid-binding site of the cellular receptor, and AH3-specific amino acid mutation sites are depicted in light orange, green, red, and light blue, respectively. The figure presented in this review is an original creation.

5. Mechanisms of SARS-CoV-2 Reinfection

SARS-CoV-2 belongs to the genus *Betacoronavirus*, has been shown to cause reinfections in short intervals [19]. The spike (S) protein of the virus, which binds to the ACE2 receptor, acts as the primary antigen [20]. Molecular evolutionary analyses showed a relatively high mutation rate (around 7.7×10^{-4} s/s/y) for the spike gene, particularly in the receptor-binding domain (RBD) [4,21]. Positive selection pressures further drive mutations in the RBD, facilitating immune escape and diminishing the neutralizing effects of antibodies from prior infections or vaccinations (Figure 3).

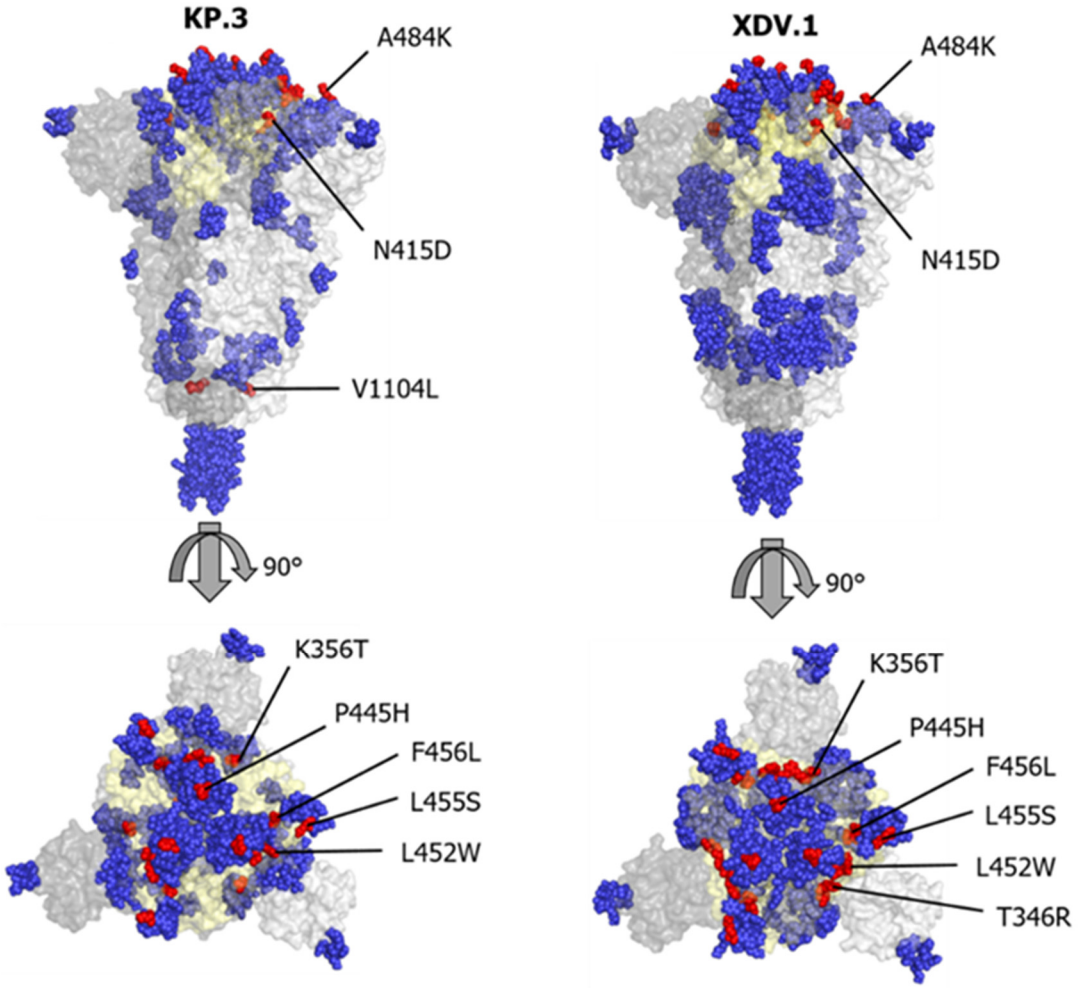

Figure 3. Mapping of amino acid mutation sites, conformational epitope motifs, and receptor-binding domains (RBD) on the trimeric structural model of the SARS-CoV-2 spike (S) protein. The peptide chains of the trimeric structure are depicted in dark gray (chain A), gray (chain B), and light gray (chain C). Conformational epitopes, major amino acid mutation sites, and the RBD are shown in blue, red, and light yellow, respectively. Among the major amino acid mutations, those inferred to be under positive selection pressure are indicated in red text. This figure has been adapted from Reference 4 under the terms of the CC BY license.

6. Mechanisms of RSV and HRV3 Reinfection

Respiratory syncytial virus (RSV) belongs to the family *Pneumoviridae* and the genus *Orthopneumovirus* [2]. It causes pneumonia in infants and the elderly and is also known to induce lifelong reinfection. Human respirovirus type 3 (HRV3; formerly known as parainfluenza virus type 3) belongs to the family *Paramyxoviridae* and the genus *Respirovirus*. Similar to RSV and HRV3 exhibit pathogenicity and induces reinfection [2]. The major antigenic proteins of RSV are the fusion (F) protein and the attachment glycoprotein (G protein), whereas HRV3′s major antigens are the F protein and the hemagglutinin-neuraminidase (HN) protein. Despite having a single serotype, both RSV and HRV3 are known to cause reinfections, much like measles virus [22]. Neutralizing antibodies effective for infection prevention are thought to be induced by the F protein for RSV and by both the F protein and HN protein in HRV3. Moreover, the RSV F protein undergoes a significant structural transition from a prefusion to a postfusion state before and after binding to the cellular receptor [23]. It has also been suggested that antibodies targeting the prefusion form of the F protein may serve as potent neutralizing antibodies for infection prevention. In the 1950s, inactivated vaccines for RSV and HRV3 were developed and underwent clinical trials [24,25]. However, following wild-type virus infection, vaccinated individuals not only showed insufficient infection prevention compared to unvaccinated groups but also experienced exacerbated clinical symptoms. This led to a long hiatus in vaccine development for these viruses. To better understand the mechanisms of reinfection, molecular evolutionary analyses of the F and HN proteins of RSV and HRV3 were conducted [6,7]. Additionally, given that the cellular receptor for the RSV F protein is Toll-like receptor 4 (TLR4), which plays a key role in innate immunity, the molecular interactions between the RSV F protein and TLR4 were analyzed in detail. Figure

4 illustrates the three-dimensional structure of the RSV F protein in its prefusion form, highlighting conformational epitopes, neutralizing antibody binding sites, and amino acid substitution (mutation) sites. Notably, conformational epitopes did not align with the neutralizing antibody binding sites [8]. A similar observation was made for the F protein of HRV3, where conformational epitopes and neutralizing antibody binding sites did not coincide [6]. In contrast, the three-dimensional structure of the HRV3 HN protein revealed that, while some conformational epitopes aligned with neutralizing antibody binding sites, the majority did not [7]. These findings suggest that in the major antigens of RSV and HRV3, conformational epitopes are not located at or near the neutralizing antibody binding sites. This misalignment may contribute to the ability of these viruses to cause reinfection [8].

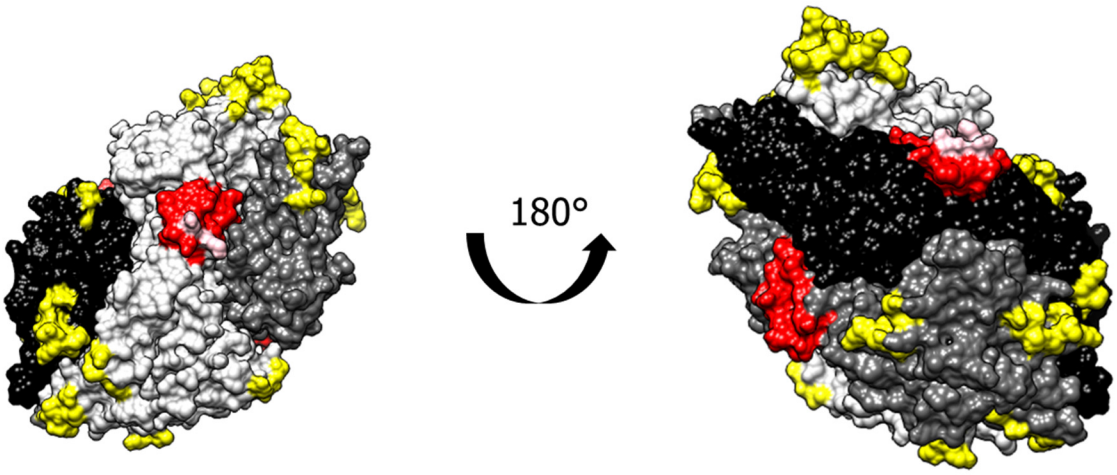

Figure 4. Mapping of conformational epitopes and neutralizing antibody binding sites on the structural model of RSV F protein. Conformational epitopes are shown in yellow, while neutralizing antibody binding sites are depicted in red. This figure is an original creation presented in this review.

7. Relationships Between Respiratory Virus Reinfection and Genome Recombination/Reassortment

Genome reassortment or viral genome recombination has been reported to occur in some respiratory viruses, such as influenza viruses and SARS-CoV-2 [26,27]. If recombinant variants with increased genetic diversity adapt successfully, they have the potential to become widespread. Antigenic changes driven by genome recombination are among the key factors contributing to pandemics. These antigenic changes can also compromise the efficacy of existing vaccines. For example, in the case of SARS-CoV-2, the emergence and widespread transmission of subvariants such as XBB and XEC highlight the role of recombination [27]. Similarly, for influenza viruses, the emergence and global spread of the AH1pdm09 virus in 2009 exemplifies the significant impact of genome reassortment [28]. Therefore, genome reassortment and recombination are critical factors, not only in influencing viral outbreaks but also in guiding the development and/or improvement of vaccines [26,27].

8. Current Status and Challenges of Vaccination Strategies in the World

Vaccination strategies vary significantly across countries and regions, each facing unique challenges that necessitate tailored solutions [29]. In the United States, although the country has demonstrated leadership in vaccine development and distribution, regional disparities and the spread of misinformation have impeded efforts to improve vaccination rates [30]. These challenges underscore the need for region-specific campaigns and enhanced educational initiatives. Similarly, Western Europe benefits from robust public health systems, which have enabled high overall vaccination rates [31]. Nonetheless, lower vaccine uptake among younger populations and rural communities remains a pressing issue. To address these gaps, targeted digital media outreach and the deployment of mobile clinics can play critical roles in improving accessibility. In Asia, countries like China and India possess substantial vaccine production capacities. However, logistical barriers and cultural challenges in rural and remote areas continue to hinder vaccine uptake [31]. Strengthening logistical networks and developing culturally sensitive outreach initiatives are essential to overcoming these obstacles. In Africa, reliance on international organizations for vaccine supply poses a significant challenge, further exacerbated by inadequate healthcare infrastructure and insufficient cold-chain facilities [31]. Addressing these issues requires increased investment in infrastructure, targeted public awareness campaigns, and expanded international support. Despite regional differences, several common strategies have emerged. These include designing flexible, region-specific policies, leveraging technological innovations to enhance logistics and healthcare infrastructure, and combating

misinformation through public education and trust-building initiatives. By fostering international collaboration and implementing comprehensive, locally adapted measures, global public health efforts can strive toward achieving equitable and effective vaccination outcomes.

9. Public Health and the Socially Scientific Impact of Reinfections

Insights into viruses that cause reinfection play a crucial role in the formulation of public health policies. For instance, preventive strategies must account for the timing and frequency of vaccinations and the feasibility of achieving herd immunity against rapidly evolving viruses such as influenza and SARS-CoV-2 [32,33]. Furthermore, vulnerable populations, including the elderly, children, and immunocompromised individuals, face heightened susceptibility to severe outcomes from viral reinfections due to weakened or immature immune systems. Investigating the interaction between viral characteristics and the specific predispositions of these groups is crucial for developing targeted interventions. Such measures may include tailored vaccination strategies and enhanced healthcare surveillance to reduce the impact of reinfections and guide the formulation of effective public health policies [34]. Additionally, the societal evaluation of how outbreaks caused by viruses with high reinfection risk affect healthcare systems and economic activities is essential to establishing sustainable healthcare infrastructure [35]. Moreover, even if the mechanisms of reinfection are elucidated, the absence of sufficient public trust and understanding regarding vaccine efficacy and safety could hinder the improvement of vaccination coverage rates. Particularly for cases like RSV, where vaccine development was once halted, public anxiety and misconceptions may spread [36]. Therefore, discussions on effective risk communication and science-based information dissemination are imperative. Finally, health behaviors related to viral reinfection, such as handwashing, mask-wearing, and vaccination, are strongly influenced by cultural and social factors. For example, in certain regions or communities, traditional treatments or beliefs may conflict with modern medicine, making it challenging to adopt preventive measures [37]. Moreover, societal stigma related to viruses with frequent reinfections may emerge. Designing social approaches that consider such cultural factors is critical.

10. Conclusions

The mechanisms underlying reinfections by various viruses are intricately tied to the dynamic interplay between viral antigenic properties and host immune responses. Viruses such as the measles virus possess highly conserved antigenic sites, enabling effective and long-lasting immunity after a single infection. In contrast, viruses like influenza and SARS-CoV-2 undergo rapid antigenic evolution under immune selection pressure, leading to the emergence of immune escape variants that facilitate reinfection. Additionally, the structural complexities of proteins in viruses such as respiratory syncytial virus (RSV) and human respirovirus 3 (HRV3), where conformational epitopes often fail to align with neutralizing antibody-binding regions, highlight further obstacles in achieving sustained immune protection. These findings underscore the critical need to deepen our understanding of antigenic evolution and host-pathogen interactions to inform the development of effective vaccines and therapeutic strategies aimed at mitigating the global burden of respiratory virus reinfections. The mechanisms underlying reinfections caused by other significant respiratory viruses, such as human rhinoviruses and human metapneumoviruses (hMPV), remain poorly understood. Further studies are required to elucidate these mechanisms. Currently, the nucleotide sequences of numerous respiratory virus strains (big data) can be utilized to infer past outbreaks through phylodynamic analyses, such as Bayesian skyline plot analysis. However, these approaches face considerable challenges in predicting future outbreaks [38]. Addressing these limitations and developing more accurate forecasting methods are critical areas for future research. Moreover, these advancements are essential for the development and refinement of vaccines targeting various respiratory viruses. Continuous global viral surveillance, coupled with the sharing of information such as nucleotide sequences of detected strains, is indispensable for achieving these objectives. Such efforts will not only enhance our understanding of reinfection mechanisms but also contribute to improving public health responses to emerging respiratory virus threats. Finally, the review may attribute to viral reinfection mechanisms from both biological and social science perspectives. The key areas include the impact on public health policies, challenges in vaccine adoption, and cultural influences on health behaviors. Integrating these insights aims to enhance strategies for managing reinfections and improving global health outcomes.

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Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

The data presented in this study are available on request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

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