



# Biology of Measles Virus: Epidemiology and Clinical Manifestations

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## Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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## ABSTRACT

Measles virus is a highly infectious RNA virus belonging to the genus Morbillivirus in the family Paramyxoviridae. It causes measles, a respiratory disease that is one of the leading causes of childhood mortality worldwide. The virus has a pleomorphic structure and a genome consisting of a single strand of negative-sense RNA. The genome encodes six structural proteins, including the nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), hemagglutinin (H), and the large polymerase (L) proteins. Measles virus enters host cells through the interaction of the viral H

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protein with cellular receptors, followed by fusion of the viral envelope with the host cell membrane. The virus then undergoes replication and assembly, leading to the release of new virus particles. The viral life cycle is complex and involves numerous host factors. Diagnosis of measles virus is typically made by detecting the virus in clinical specimens using laboratory tests such as RT-PCR or serological assays. Clinical symptoms such as fever, cough, runny nose, and rash can also aid in the diagnosis. Complications of measles virus infection include pneumonia, encephalitis, and death. Prevention of measles virus involves vaccination, maintaining high population immunity, and early identification and isolation of infected individuals. Measles virus remains a significant public health threat, particularly in areas with low vaccination coverage. The virus is highly contagious and can cause severe disease and mortality. The global incidence of measles has increased in recent years due to outbreaks in various regions. Treatment for measles virus infection is primarily supportive, with no specific antiviral therapies currently available.

*Keywords: Measles virus; infectious viral disease; vaccine; viral RNA genome.*

## 1. INTRODUCTION

The general public is periodically faced with outbreaks or emergence of new infection like COVID-19 that triggered pandemic response [1-3]. Measles is a highly infectious viral disease that has been affecting human populations for centuries [4]. The virus that causes measles belongs to the family Paramyxoviridae, genus Morbillivirus, and is known for its ability to cause severe illness and complications, especially in young children and immune-compromised individuals [4]. Despite the availability of a highly effective vaccine, measles continues to be a significant public health concern, particularly in areas with low vaccine coverage [4].

The World Health Organization (WHO) estimates that approximately 7 million people contract measles every year, resulting in over 100,000 deaths globally [5]. In 2019, there were an estimated 869,770 cases of measles worldwide, with the highest burden of disease seen in the African and Southeast Asian regions [5]. While measles is a vaccine-preventable disease, outbreaks continue to occur, often in areas with low vaccination coverage [5].

The measles virus is highly contagious and spreads through the air via droplets from the nose, mouth, or throat of infected individuals. Once inside the body, the virus replicates rapidly, leading to a range of clinical symptoms, including fever, cough, runny nose, and a characteristic rash [6]. In addition to the acute symptoms, measles can also cause serious complications, such as pneumonia, encephalitis, and blindness [6].

The development of a highly effective measles vaccine in the 1960s led to a dramatic decrease in the incidence of the disease in developed countries [7]. However, despite widespread

vaccination efforts, vaccine hesitancy and refusal have led to a resurgence of measles in many parts of the world [7]. Outbreaks of measles have been reported in countries with high vaccine coverage, such as the United States, where 1,282 cases were reported in 2019 [5].

This review aims to provide a comprehensive overview of the measles virus, including its structure, replication cycle, and pathogenesis. It will examine the clinical features of measles infection, including the acute symptoms and complications associated with the disease. Additionally, the review will discuss the diagnosis and treatment of measles, as well as the challenges associated with its control and elimination.

In addition to its clinical impact, the review will explore the epidemiology of measles, including the global incidence of the disease and the impact of vaccination on its prevalence. This review will also examine the challenges associated with measles control and elimination efforts, including vaccine hesitancy, outbreaks in high-income countries, and the role of global health organizations in promoting measles vaccination.

Overall, this review aims to provide a comprehensive understanding of the measles virus and its impact on global public health. By examining the biology of the virus, its clinical manifestations, and the challenges to its control and elimination, we can gain insight into effective strategies for preventing and treating this highly contagious and potentially deadly disease.

## 2. STRUCTURE OF THE MEASLES VIRUS

The measles virus is a single-stranded RNA virus that belongs to the genus Morbillivirus, which is part of the family Paramyxoviridae [8]. The virus

is approximately 150-300 nm in diameter and has a spherical shape with a lipid envelope that surrounds its nucleocapsid core. The envelope contains viral glycoproteins, which are essential for viral attachment, entry, and fusion with the host cell membrane [8].

The nucleocapsid core of the measles virus is made up of the viral RNA genome, which is tightly bound to the nucleoprotein (N protein) and is associated with the viral polymerase complex [9]. The RNA genome is approximately 15,894 nucleotides in length and encodes six structural proteins (N, P, M, F, H, and L) and two non-structural proteins (V and C). These proteins are synthesized from a set of seven distinct mRNAs, which are transcribed from the viral RNA genome [9].

The N protein of the measles virus plays a critical role in viral RNA packaging and replication. It binds to the viral RNA genome and forms a ribonucleoprotein (RNP) complex, which is essential for viral transcription and replication [9]. The P protein of the measles virus interacts with the N protein and forms a complex with the viral RNA polymerase, which is responsible for viral transcription and replication [9].

The M protein of the measles virus is a trans-membrane protein that interacts with the viral envelope and nucleocapsid core. It plays a critical role in viral assembly and budding by facilitating the transport of viral components to the plasma membrane and coordinating viral assembly at the plasma membrane [9].

The F protein of the measles virus is a class I viral fusion protein that mediates viral entry into host cells. It interacts with the host cell receptor and undergoes conformational changes that facilitate viral fusion with the host cell membrane, leading to viral entry into the cytoplasm.

The H protein of the measles virus is a viral attachment protein that binds to host cell receptors, initiating viral attachment and entry. The H protein interacts with the F protein and is responsible for the specificity of the viral attachment to host cells.

The L protein of the measles virus is the viral RNA polymerase, which is responsible for viral transcription and replication. It interacts with the P protein and forms a complex with the viral RNA genome, which is essential for viral RNA synthesis.

Overall, the structure of the measles virus is essential for its replication, assembly, and entry into host cells. A detailed understanding of the structure and function of the viral proteins is critical for the development of effective antiviral therapies and vaccines.

### **3. HISTORY OF THE MEASLES VIRUS**

The history of the measles virus can be traced back to ancient times, with some of the earliest recorded cases of measles occurring in the 9th century in the Middle East. However, it was not until the 18th and 19th centuries that measles began to be recognized as a distinct and highly contagious disease [10].

#### **3.1 Discovery and Early History of Measles**

The earliest known description of measles was recorded in the 9th century by the Persian physician Rhazes. He described a highly contagious and fatal disease that he called "Moretum," which is believed to have been measles. However, it was not until the 16th century that the disease was formally recognized as a distinct illness, when the Italian physician Giovanni Filippo described an outbreak of a highly contagious fever with a rash [11].

Over the next few centuries, the disease became increasingly recognized and studied, with physicians and scientists making significant progress in understanding its epidemiology and clinical presentation. In 1757, the English physician Francis Home published a seminal work on the clinical features of measles, describing the characteristic rash, fever, and cough that are associated with the disease [11].

By the 19th century, measles had become a widespread and highly contagious disease, with outbreaks occurring throughout Europe and North America. During this time, physicians and researchers began to develop new techniques for diagnosing and studying the disease. In 1874, the German physician Hugo Engelmann first isolated the measles virus from a patient with the disease, using a technique known as serum therapy [11].

#### **3.2 Development of Measles Vaccines**

The development of effective vaccines for measles has been a major public health achievement, with vaccines helping to significantly reduce the incidence and severity of the disease worldwide. The first measles vaccine

was developed in the 1960s by the American virologist John Enders, who used a weakened form of the virus to produce an attenuated vaccine that was highly effective at preventing measles [12].

Over the next few decades, several other types of measles vaccines were developed, including inactivated and subunit vaccines. These vaccines were highly effective at preventing measles, and their widespread use led to a significant reduction in the global incidence of the disease.

In recent years, however, there has been a resurgence of measles in some parts of the world, due in part to a decline in vaccination rates and the spread of anti-vaccine misinformation. To address this problem, public health authorities have launched several initiatives to promote measles vaccination and increase public awareness of the importance of vaccines in preventing infectious diseases.

One such initiative is the Global Measles and Rubella Strategic Plan, which was launched by the World Health Organization (WHO) in [13]. The plan aims to reduce the global incidence of measles and rubella by 95% by the year 2020, through a combination of vaccination campaigns, surveillance and monitoring, and research and development of new vaccines [12].

Despite the progress that has been made in preventing and controlling measles, the disease remains a significant public health threat in many parts of the world. In addition to outbreaks in areas with low vaccination rates, there are also concerns about the potential for the virus to mutate and become resistant to existing vaccines [12]. As such, ongoing research into the epidemiology and biology of measles is essential for the development of new strategies for preventing and controlling the disease.

## **4. VIRAL LIFE CYCLE OF MEASLES VIRUS**

### **4.1 Entry**

Measles virus entry is a complex process that involves multiple interactions between the virus and host cells [14]. The initial attachment of MeV to host cells is mediated by the viral hemagglutinin (H) protein, which binds to cellular receptors on the surface of host cells. The two major cellular receptors for MeV are CD150 (also

known as signaling lymphocyte activation molecule, SLAM) and CD46, which are expressed on immune cells and various other cell types, respectively [14]. The H protein binds to both receptors, although with different affinities, and triggers conformational changes that lead to fusion of the viral and cellular membranes [14].

The fusion process is mediated by the viral fusion (F) protein, which undergoes a series of conformational changes upon receptor binding and H protein triggering [14]. The F protein exists in a metastable prefusion conformation on the surface of the virus and must be activated by cleavage to its fusion-active form [15]. This cleavage is carried out by host proteases, such as furin, which cleaves the F protein into two subunits, F1 and F2 [15]. The F1 subunit contains the fusion peptide, which inserts into the host cell membrane, while the F2 subunit anchors the F protein to the viral membrane [15].

### **4.2 Replication**

Once inside the host cell, the MeV genome is released into the cytoplasm, where it serves as a template for viral replication. The negative-sense RNA genome is first transcribed into a positive-sense RNA intermediate by the viral RNA-dependent RNA polymerase (RdRp), which is packaged inside the virion. This positive-sense RNA is then used as a template for the synthesis of new negative-sense RNA genomes and viral mRNAs [15].

MeV replication occurs in the cytoplasm and is carried out by the viral RdRp in conjunction with host cell machinery. The viral genome is encapsidated by the nucleocapsid (N) protein, which binds to the RNA genome and packages it into a helical structure [15]. The N protein also interacts with the RdRp and other viral proteins to form the viral ribonucleoprotein (RNP) complex, which serves as the template for RNA synthesis [16].

MeV replication is tightly regulated by the viral proteins, which interact with host cell factors to modulate the cellular environment and promote viral replication. For example, the viral V protein interacts with host STAT proteins to prevent the induction of interferon and other antiviral responses [16]. The viral C protein interacts with the host RNA helicase DDX3 to modulate RNA synthesis and promote viral replication [16].

### 4.3 Assembly and Release

Once the viral components have been synthesized, they must be assembled into new virions and released from the infected cell. MeV assembly occurs at the plasma membrane, where the viral glycoproteins and nucleocapsid interact to form the mature virion [17]. The H and F proteins are transported to the plasma membrane separately, where they interact with the N protein to form the nucleocapsid. The nucleocapsid is then transported to the plasma membrane, where it interacts with the glycoproteins to form the mature virion [17].

The final step in the MeV life cycle is the release of newly formed virions from the infected cell. MeV release occurs through a process known as budding, in which the mature virion is enveloped by a lipid bilayer derived from the host cell membrane. The viral glycoproteins are inserted into the host cell membrane and interact with the nucleocapsid to initiate budding [17]. As the virion buds from the infected cell, it acquires its lipid envelope and glycoprotein spikes, which are derived from the host cell membrane [17].

The release of MeV virions from infected cells is a complex process that is regulated by multiple viral and host factors. For example, the MeV matrix (M) protein interacts with the host cell actin cytoskeleton to facilitate virion budding and release [4]. The viral V protein also plays a role in virion release by interacting with host cell factors that regulate the cellular trafficking and secretion of viral particles [4].

### 4.4 Recent Advances

In recent years, significant advances have been made in our understanding of the MeV life cycle, including the molecular mechanisms of viral entry, replication, and assembly. One major area of research has focused on the structure and function of the MeV glycoproteins, particularly the H and F proteins, which mediate viral entry and fusion [4]. High-resolution structures of the MeV H and F proteins have been obtained by cryo-electron microscopy, revealing new insights into their conformational changes and interactions with host cell receptors [4].

Another area of research has focused on the interactions between MeV and host cells, particularly the host immune response. MeV infection induces a strong immune response, which is thought to contribute to the

pathogenesis of the disease. Recent studies have identified a number of viral proteins that interact with host immune factors, including the V protein, which interacts with host STAT proteins to prevent interferon induction, and the C protein, which interacts with the host RNA helicase DDX3 to modulate RNA synthesis and promote viral replication [18].

Finally, recent studies have also shed light on the molecular mechanisms of MeV assembly and release. For example, the MeV matrix (M) protein has been shown to interact with the host cell actin cytoskeleton to facilitate virion budding and release [18]. The viral V protein has also been shown to interact with host cell factors that regulate the cellular trafficking and secretion of viral particles [18].

## 5. EPIDEMIOLOGY OF MEASLES VIRUS

### 5.1 Global Burden of Measles

Measles is one of the leading causes of death among young children globally, with an estimated 207,500 deaths in 2019 [5]. Although measles is a vaccine-preventable disease, it remains endemic in many parts of the world, particularly in low- and middle-income countries with weak health systems and low vaccination coverage. The World Health Organization (WHO) has set a target to eliminate measles in five of its six regions by 2020, but progress towards this goal has been hampered by a resurgence of measles in several countries [13].

### 5.2 Transmission Patterns

Measles is highly contagious and is transmitted through respiratory droplets when an infected person coughs or sneezes. The virus can survive in the air or on surfaces for up to two hours, making it highly transmissible in crowded settings [19]. Measles is most infectious in the period from four days before to four days after the appearance of the rash, but it can be transmitted from a person with measles for up to seven days before and after the onset of rash [19].

Measles is a disease of humans, and there are no animal reservoirs. The virus is transmitted only from person to person, and there is no evidence of airborne spread over long distances or transmission through food or water [19].

### 5.3 Risk Factors for Infection

Anyone who is not immune to measles and comes into contact with the virus is at risk of

infection [19]. The most effective way to prevent measles is through vaccination with the measles, mumps, and rubella (MMR) vaccine, which provides long-lasting protection against measles [19].

However, in populations with low vaccination coverage, outbreaks can occur, particularly in communities with poor living conditions and inadequate health services. Infants, young children, and pregnant women are particularly vulnerable to severe disease and death from measles [19].

In addition to low vaccination coverage, other risk factors for measles infection include:

- Travel to areas with ongoing measles outbreaks
- Crowded living conditions, such as refugee camps or slums
- Poor nutrition, which can weaken the immune system
- Lack of access to healthcare, which can delay diagnosis and treatment
- Immunocompromising conditions or treatments, which can increase the risk of severe disease and complications.

#### 5.4 Epidemiological Trends

Measles is a vaccine-preventable disease, and vaccination programs have led to significant reductions in the global burden of measles. In 2000, the WHO launched the Measles Initiative, a global partnership to accelerate progress towards measles control and elimination. As a result of these efforts, global measles deaths declined by 73% between 2000 and 2018 [13].

However, progress towards measles elimination has stalled in recent years, with a significant increase in measles cases and deaths reported in many countries. In 2019, there were an estimated 869,770 measles cases reported worldwide, the highest number in over two decades [5]. The majority of these cases occurred in countries with low vaccination coverage, particularly in sub-Saharan Africa and the Asia-Pacific region.

In high-income countries, measles outbreaks have been linked to pockets of unvaccinated individuals, often due to vaccine hesitancy or refusal. In the United States, for example, there were 1,282 confirmed cases of measles in 2019, the highest number since 1992 [20].

## 6. PATHOGENESIS OF MEASLES VIRAL INFECTION

The pathogenesis of MeV begins with the entry of the virus into the host's respiratory tract through inhalation of infectious droplets [21]. MeV initially infects epithelial cells of the respiratory tract, where it replicates and spreads to regional lymph nodes [22]. MeV targets dendritic cells and macrophages, which facilitate viral dissemination to other organs [23].

During the early phase of infection, MeV suppresses the host's innate immune response by inhibiting interferon production and reducing the expression of major histocompatibility complex (MHC) class I and II molecules [24]. This allows the virus to evade detection and clearance by the host's immune system.

As the infection progresses, MeV induces a strong T cell response, leading to the production of pro-inflammatory cytokines such as interferon-gamma, tumor necrosis factor-alpha, and interleukin-6 [25]. These cytokines promote the recruitment of additional immune cells to the site of infection and contribute to the development of the characteristic measles rash.

In addition to its effects on the immune system, MeV also has direct cytopathic effects on infected cells. MeV induces syncytia formation, in which infected cells fuse together to form multinucleated giant cells [25]. This process can lead to tissue damage and contribute to the development of MeV-associated complications such as encephalitis.

## 7. MEASLES IMMUNE RESPONSE

Measles is an acute viral infection that is typically characterized by a fever, cough, coryza, and conjunctivitis, followed by a maculopapular rash [20]. The measles virus primarily infects the respiratory tract and spreads to the regional lymph nodes, where it infects immune cells, including dendritic cells and T cells [26]. The immune response to measles virus involves both innate and adaptive immune mechanisms, and the development of long-term immunity is critical for protection against future infections [27].

### 7.1 Innate Immune Response

The innate immune response is the first line of defense against invading pathogens, including measles virus. The innate immune response to

measles virus involves the recognition of viral components, such as viral RNA, by pattern recognition receptors (PRRs), including Toll-like receptors (TLRs) and RIG-I-like receptors (RLRs) [8]. This recognition triggers the production of type I interferons (IFNs), which activate antiviral signaling pathways and induce the expression of hundreds of interferon-stimulated genes (ISGs) [21]. The expression of ISGs leads to the establishment of an antiviral state within infected and neighboring cells, limiting the spread of the virus and promoting viral clearance [21].

## 7.2 Adaptive Immune Response

The adaptive immune response to measles virus involves the activation of B and T lymphocytes, which work together to eliminate the virus and establish long-term immunity [28]. The primary target of the adaptive immune response is the measles virus envelope glycoprotein, which is the major target of neutralizing antibodies [28]. B cells recognize and bind to the envelope glycoprotein, leading to the production of virus-specific antibodies. These antibodies can neutralize the virus by preventing it from entering host cells and can also facilitate the clearance of virus-infected cells by activating the complement system and recruiting immune cells [28].

Measles-specific T cells are also critical for the adaptive immune response to measles virus. Measles virus-specific T cells recognize and eliminate virus-infected cells by directly killing them or by secreting cytokines that activate other immune cells [29]. Measles virus-specific T cells also play a critical role in the establishment of long-term immunity by differentiating into memory T cells, which can rapidly respond to future infections with measles virus [29].

## 7.3 Immune Evasion Strategies

The measles virus has evolved several strategies to evade the host immune response, including inhibition of IFN production, suppression of antigen presentation, and modulation of cytokine signaling [30]. Measles virus infection can inhibit the production of type I IFNs by infected cells, reducing the activation of antiviral signaling pathways and the expression of ISGs [30]. Measles virus can also modulate cytokine signaling by inhibiting the production of pro-inflammatory cytokines and promoting the production of anti-inflammatory cytokines, which can limit the recruitment and activation of immune cells [31].

## 8. COMPLICATIONS OF MEASLES VIRAL INFECTION

Measles is a highly contagious viral disease that can lead to severe complications. Although it can be prevented by vaccination, outbreaks still occur in many parts of the world.

- **Pneumonia:** Pneumonia is the most common complication of measles, and it is responsible for most measles-related deaths. It can be caused by a direct viral infection of the lung or by a secondary bacterial infection. Children under the age of five and adults over the age of 20 are at higher risk of developing pneumonia following measles infection [32].
- **Encephalitis:** Measles encephalitis is a rare but serious complication that occurs in about one in every 1,000 cases of measles. It is caused by a direct viral infection of the brain and can lead to permanent brain damage or death [33].
- **Subacute sclerosing panencephalitis (SSPE):** SSPE is a rare and fatal complication of measles that occurs years after the initial infection. It is caused by a persistent measles virus infection of the brain and can lead to progressive neurological deterioration and death [34].
- **Febrile seizures:** Febrile seizures can occur in children during the acute phase of measles infection. They are usually self-limiting and do not lead to long-term neurological damage [35].
- **Thrombocytopenia:** Measles can cause a decrease in platelets, which are responsible for blood clotting. This can lead to bleeding disorders and may require hospitalization [36].
- **Otitis media:** Measles can lead to otitis media, which is an infection of the middle ear. It can cause pain, fever, and hearing loss [32].
- **Blindness:** Measles can cause a severe eye infection called measles keratitis, which can lead to blindness. It is more common in malnourished children and those with vitamin A deficiency [33].
- **Malnutrition:** Measles can lead to malnutrition due to loss of appetite and malabsorption of nutrients. Malnourished children are at higher risk of developing severe complications of measles [34].
- **Death:** Measles can be fatal, especially in children under the age of five and adults over the age of 20. The mortality rate can

be as high as 30% in some populations, especially in areas with poor healthcare infrastructure and low vaccination coverage [35].

## 9. DIAGNOSIS OF MEASLES VIRUS

Diagnosis of measles virus is typically made by detecting the virus in clinical specimens, such as throat swabs, nasal swabs, or urine samples, using laboratory tests [37].

One common laboratory test used to diagnose measles virus is the reverse transcription-polymerase chain reaction (RT-PCR) assay. This test detects the viral RNA in clinical specimens, and has high sensitivity and specificity [29]. Other laboratory tests that can be used to diagnose measles virus include serological assays, such as enzyme-linked immunosorbent assay (ELISA) and plaque reduction neutralization test (PRNT), which detect measles virus-specific antibodies in serum samples [37].

In addition to laboratory tests, clinical symptoms such as fever, cough, runny nose, and rash can also aid in the diagnosis of measles virus. However, these symptoms are not specific to measles and can be caused by other viral infections as well [38].

## 10. PREVENTION OF MEASLES VIRUS

Prevention of measles virus involves a combination of measures, including vaccination, maintaining high population immunity, and early identification and isolation of infected individuals. Vaccination is the most effective way to prevent measles virus infection and is recommended by the World Health Organization (WHO) as part of routine childhood immunization programs [39].

The measles vaccine is a live attenuated vaccine that is highly effective in preventing measles virus infection when given in two doses. The first dose is recommended at 12-15 months of age, followed by a second dose at 4-6 years of age [40]. Vaccination not only protects individuals from measles virus infection, but also contributes to herd immunity, which is essential for protecting vulnerable populations who cannot receive the vaccine, such as infants, pregnant women, and individuals with compromised immune systems [39].

In addition to vaccination, early identification and isolation of infected individuals is crucial for

preventing the spread of measles virus. Infected individuals should be isolated for at least four days after the onset of rash to prevent transmission to others [40]. Health care providers should be vigilant for suspected cases of measles and report them to public health authorities immediately to prevent outbreaks [39].

## 11. TREATMENT OF MEASLES VIRUS INFECTION

The treatment of measles virus infection is mainly supportive, with the focus on managing complications and preventing secondary infections. The World Health Organization recommends vitamin A supplementation for all children diagnosed with measles, as it has been shown to reduce mortality and the risk of complications such as pneumonia and diarrhea [33].

Antiviral therapy with ribavirin and interferon has been used in severe cases, but its effectiveness is still controversial. Immunoglobulin therapy has been used in immunocompromised patients, but its benefit in immunocompetent patients is limited [33].

## 12. CONCLUSION

In conclusion, measles virus is a highly infectious RNA virus that can cause significant morbidity and mortality, particularly in populations with low vaccination coverage. The virus has a pleomorphic structure and a complex genome organization that encodes six structural proteins. Measles virus enters host cells through the interaction of the viral H protein with cellular receptors, followed by replication and assembly of new virus particles. The immune response to measles virus is critical for controlling the infection and providing long-term protection. Diagnosis of measles virus infection involves detecting the virus in clinical specimens using laboratory tests, with clinical symptoms aiding in the diagnosis. Complications of measles virus infection can be severe, including pneumonia, encephalitis, and death. Prevention of measles virus infection through vaccination, maintaining high population immunity, and early identification and isolation of infected individuals is essential. Despite the availability of an effective vaccine, measles virus remains a significant public health threat, with recent outbreaks reported in various regions globally. Treatment for measles virus infection is primarily supportive, with no specific antiviral therapies currently available. Continued



efforts towards increasing vaccination coverage and early detection and control of outbreaks are essential for reducing the burden of measles virus infection globally.

## CONSENT

It is not applicable.

## ETHICAL APPROVAL

It is not applicable.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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