# Nipah virus as an emerging threat: mutational dynamics, pathogenesis, and advances in vaccine development-a systematic review

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**ABSTRACT**

Nipah virus (NiV) is an emerging zoonotic pathogen with significant pandemic potential. Large outbreaks, such as in Malaysia, required the culling of over one million pigs to control transmission. However, the epidemiology of NiV among animal hosts, including pigs, horses, and bats, remains incompletely understood. NiV infection primarily affects the respiratory and nervous systems, causing severe pneumonia, vasculitis, and meningitis, while encephalitis may be mild or infrequent in some cases. This systematic review summarizes current evidence on NiV mutational variation, pathogenesis, treatment strategies, and vaccine development up to 2022. Data were collected from major databases, including PubMed, PMC, and Cochrane Library. Due to limited therapeutic options, NiV management relies mainly on supportive care, as no approved vaccines or specific antiviral treatments are available for humans or livestock. Preventive strategies focus on reducing zoonotic transmission, particularly by minimizing contact between livestock and bat-contaminated food sources, and improving farm management practices. Early detection and continuous surveillance of high-risk populations and animal reservoirs are essential for outbreak control. Current vaccine research targets viral antigens using subunit and vector-based approaches. Overall, further studies are urgently needed to develop effective vaccines and antiviral therapies for NiV infection.



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## 1. INTRODUCTION

Emerging and re-emerging zoonotic diseases such as SARS, Ebola, Zika, Nipah, and Hendra virus continue to pose significant global public health challenges [1]. These pathogens originate in animal reservoirs and have the ability to cross species barriers, leading to outbreaks in human populations. Past epidemics, including severe acute respiratory syndrome (SARS) and influenza A (H1N1), demonstrate the potential of zoonotic viruses to trigger widespread health crises and global emergencies [2]. As a result, understanding the transmission dynamics and control of zoonotic diseases has become a critical priority.

Among these emerging pathogens, Nipah virus (NiV) has attracted considerable attention due to its high case fatality rate, which can reach approximately 79% [3]. Since its first identification in Malaysia in 1999, outbreaks have been reported in several countries, particularly in South and Southeast Asia, including Bangladesh and India [3]. The World Health Organization has classified NiV as a priority pathogen because of its high mortality rate, potential for human-to-human transmission, and the absence of effective vaccines or antiviral therapies [4]. Consequently, NiV is considered a serious threat with pandemic potential (Table 1).

Table 1. Overview of the pandemic situation of the Nipah virus in Asian countries

| Year | Country | Location | Number of cases | Number of deaths | Case fatality (%) |
|---|---|---|---|---|---|
| Sep 1998 – Apr 1999 | Malaysia | Perak, Selangor, Negeri Sebilan | 265 | 105 | 40 |
| Mar 1999 | Singapore | Singapore | 11 | 1 | 9 |
| Jan – Feb 2001 | India | Siliguri | 66 | 45 | 68 |
| Apr – May 2001 | Bangladesh | Meherpur | 13 | 9 | 69 |
| Jan 2003 | | Naogaon | 12 | 8 | 67 |
| Jan 2004 | | Rajbari | 31 | 23 | 74 |
| Apr 2004 | | Faridpur | 36 | 27 | 75 |
| Jan – Mar 2005 | | Tangail | 12 | 11 | 92 |
| Jan – Feb 2007 | | Thakurgaon | 7 | 3 | 43 |
| Mar 2007 | | Kushtia, Pabna, Tatore | 8 | 5 | 63 |
| Apr 2007 | | Naogaon | 3 | 1 | 33 |
| Apr 2007 | India | Nadia | 5 | 5 | 100 |
| Feb 2008 | Bangladesh | Manikgon | 4 | 4 | 100 |
| Apr 2008 | | Rajbari, Faridpur | 7 | 5 | 71 |
| Jan 2009 | | Gaibandha, Rangpur, Nilphamari | 3 | 0 | 0 |
| Jan 2009 | | Rajbari | 1 | 1 | 100 |
| Feb – Mar 2010 | | Faridpur, Rajbari, Gopalganj, Madaripur | 16 | 14 | 87.5 |
| | | Lalmohirhat, Dinajpur, Comilla, Nilphamari, Rangpur | 44 | 40 | 91 |
| Feb 2012 | | Joypurhat, Rajshahi, Tatore, Rajbari, Gopalganj | 12 | 10 | 83 |
| Jan – Feb 2013 | | Gaibandha, Natore, Rajshahi, Rajbari, Pabna, Jhenaidah, Mymensingh | 24 | 21 | 87.5 |
| Feb 2014 | | Manikganj, Magura, Faridpur, Rangpur, Shaariatpur, Kushtia, Rajshahi, Tatore, Dinajpur, Chapai Nawabganj, Naogaon | 18 | 9 | 50 |
| Mar – May 2014 | Philippines | Tinalon, Midtungok | 17 | 9 | 53 |
| Feb 2015 | Bangladesh | Nilphamari, Pnchoghor, Faridpur, Magura, Naugaon, Rajbari | 9 | 6 | 67 |
| May 2018 | India | Kozhikode, Malappuram | 19 | 17 | 89 |
| Overall Total | | | 643 | 379 | 58.9 |
| Malaysia/Philippines/ Singapore total | | | 293 | 115 | 39.2 |
| Bangladesh/India total | | | 350 | 264 | 75.4 |

NiV is a bat-borne virus, with fruit bats recognized as the primary reservoir. Transmission to humans can occur directly or through intermediate hosts, such as pigs, which played a key role in the Malaysian outbreak that resulted in the culling of approximately 1.1 million pigs [5], [6]. The epidemiological factors influencing viral transmission among bats, livestock, and humans remain under investigation [7]. NiV infection primarily affects the respiratory and central nervous systems, causing severe pneumonia, encephalitis, vasculitis, and meningitis. While encephalitis may be mild or infrequent in some cases, vasculitis and meningitis are often severe [8]. Neurological involvement, particularly of the brainstem, is strongly associated with mortality, and survivors may experience long-term neurological complications such as encephalopathy, behavioral abnormalities, and motor dysfunction [7].

Current research has explored various aspects of NiV, including viral genetics, mutational variation, transmission dynamics, and pathogenesis. Diagnostic approaches mainly rely on serological and molecular techniques, while vaccine development has focused on subunit vaccines and viral vector-based platforms targeting key viral antigens. However, no licensed vaccines or specific antiviral treatments are currently available, and patient management remains largely supportive [1]. Preventive strategies primarily aim to reduce zoonotic transmission by limiting contact between livestock and bat-contaminated food sources, improving farm management practices, and enhancing public awareness [9].

Despite these advances, significant gaps remain in understanding the epidemiological patterns, mutational evolution, and host–pathogen interactions of NiV. In particular, there is a lack of comprehensive integration of viral genetics, disease mechanisms, and translational strategies for vaccine and drug development. These limitations hinder effective preparedness and response to potential outbreaks. Considering these challenges, NiV is increasingly recognized as a potential future pandemic agent [10]. Therefore, this systematic review aims to provide a comprehensive overview of NiV, focusing on mutational variation, pathogenesis, current vaccine development, and treatment approaches. By synthesizing existing evidence, this study seeks to enhance understanding of NiV and support the development of effective prevention and control strategies.

## 2. METHOD

To perform the systematic review, we followed the preferred reporting items for systematic reviews (PRISMA) standards in four phases: database search, article evaluation, data extraction, and summary. Seven writers performed the search for relevant publications, and six authors independently evaluated and extracted data from the articles. Seven writers contributed to the final draft, while six authors worked together to assemble the data and perform the systematic review (Figure 1).

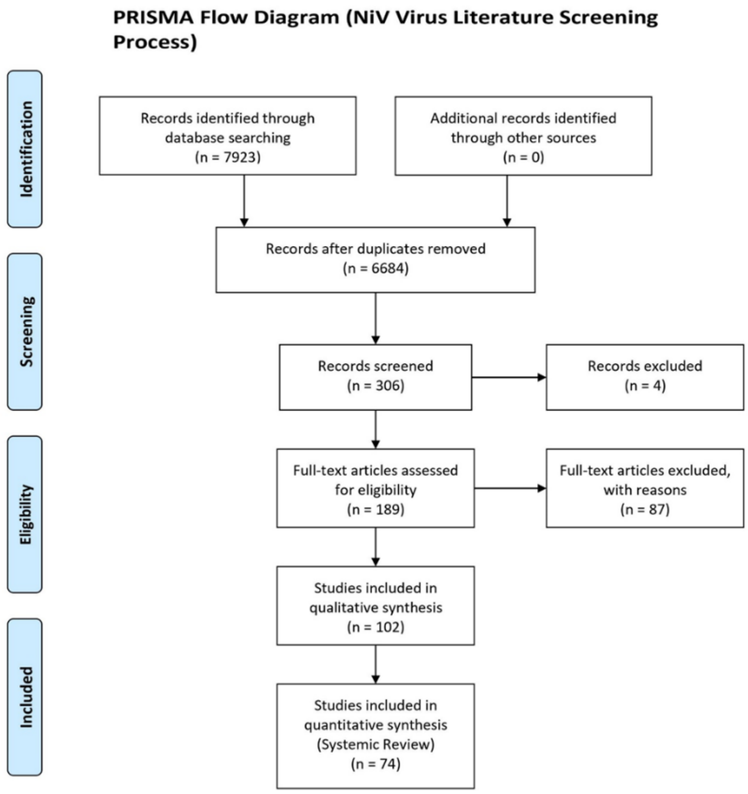


Figure 1. Prisma flowchart for Nipah virus scientific literature screening

To learn more about bats and the zoonotic diseases they may spread, including Nipah virus, we looked through articles in PubMed, PMC, and Cochrane. From 1999 to 2022, we looked through all the research publications and reviews that presented data from the field on bat-borne zoonotic diseases, including Nipah virus in Asian and African nations. MeSH terms were used: 'NiV Bangladesh', 'NiV Mutation', 'NiV Pathogenesis', 'NiV Epidemiology', and 'NiV Vaccine'.

For this data extraction, we looked exclusively at scientific reports that included bats and zoonotic illnesses. Information such as the location of the epidemic, the year the sample was taken, the number of people who were tested, the number of people who died, and any potential correlates such as the disease-causing agent, its treatment, and its pathogenicity. were culled from the data.

## 3. RESULT

### 3.1. Mutational variations of Nipah virus (NIV)

Mutation is the process by which the likelihood of altering genetic information from one to the next generation, and mutation effects the genetic diversity [11]. To understand the evolution of viruses, it is highly crucial to estimate the rates of viral mutation [12]. In our research regarding Nipah virus, it is one sort of RNA virus which includes an extremely virulent zoonotic illness, and it has the potential for fast spontaneous mutations as like the present COVID-19. Because of its quicker mutation, this virus may quickly adapt to the environment, hosts, or other circumstances, and multiple epidemics happened by the Nipah virus in recent decades. From the following table, we can witness the prior epidemic in Asian countries [13].

Basically, Nipah virus contains two main strains NiV-MY (The Malaysian strain of NiV) and NiV-BD (The Bangladesh strain of NiV) [14]. In addition to, NiV-BD carries the larger genomic size (18,252 nucleotides in length) than NiV-MY (18,246 nucleotides in length) [15]. Because of this greater genomic size, NiV-BD is slightly more pathogenic than NiV-MY, and it spreads interhost transmission more rapidly than NiV-MY [16]. From different studies, it was clinically proven that NiV-BD caused rapid oral shedding and productive infection and a highly infected respiratory tract [17].

Nipah virus transmission occurred rapidly from bats to people in Bangladesh and this occurrence was identified by three ways as like – ingestion of fresh date palm sap, domestic animal and direct contact with NiV infected bat secretions [18]. Besides this, person-to-person transmission also occurred by NiV in Bangladesh and chronic infections were shown in respiratory tract [18]. The transmission happened quickly and acted as like the chain of spreading of NiV. This way, the spreading chain included 5 generations and 34 peoples were infected by this pathogenic virus (NiV) [19]. In the exploration about Nipah virus, two envelop glycoproteins (the receptor binding G proteins and the fusion protein F) are encoded by NiV and the host cell protease activates the viral fusion proteins which is crucial step for viral life cycle [19]. Additionally, trypsin and trypsin-like protease which is similar as like paramyxovirus F proteins with containing monobasic cleavage sites can be activated the fusion protein F [20]. In mutagenesis and structural analysis, G glycoprotein plays a key role for receptor binding and the same thing is not approached in viral ephrin-B2 receptor [14]. However, ephrin-B2 (which is critical of binding NiV) was identified by the performance of alanine-scanning mutagenesis analysis [9]. Further analysis of ephrin-B2, the binding affinity of the mutant receptor with NiV-G (responsible for raising the tendency of infections) was increased by L124A ephrin-B2 mutants [21]. Therefore, L124A mutants gained the ability to support the NiV infection and arranging a new target cell platform [22], [23].

### 3.2. Pathogenesis of Nipah virus in humans

Non-specific flu-like symptoms such as fever, headache, disorientation, muscle stiffness, and watery stool are common in the early stages of a NiV infection [23]. In certain outbreaks, mild or show no symptoms infections have been documented, but the overall frequency is low and seems to be strain dependant, with the Malaysian variant producing less severe disease, a lower case mortality rate, and a greater proportion of asymptomatic infections than the Bangladesh strain [24]. An encephalitic condition occurs in around 60% of patients after an incubation period of seven to forty days. There is no information on how long it takes for the illness to proceed from the onset of symptoms to the onset of the encephalitic condition. About a third of individuals have neurological symptoms such as meningismus (inflammation of the central nervous system) and seizures. An individual's mental status may deteriorate in as little as seven days (in the case of recent outbreaks in Bangladesh) or as long as 16 days (in general) (Malaysia outbreak). About 20-30% of those who have survived NiV encephalitis have long-term neurologic dysfunction marked by seizures that continue, exhaustion that is incapacitating, and aberrant behavior. In addition, a late-onset or recurring neurological illness may develop in people with initially moderate, non-encephalitic condition. Some patients also have chronic respiratory distress, and respiratory involvement has been more prevalent in the Bangladesh outbreaks than in the Malaysian outbreaks [25].

### 3.3. Treatment criteria for Nipah virus (NIV) infection

The actual therapy for Nipah virus infection has not yet been developed (drug or vaccination). Supportive therapy, which includes ensuring enough rest, drinking more water, and symptomatic maintenance of acute encephalitis syndrome, is the primary treatment [24]. It is possible to prevent the transmission of this virus from individual to individual by using good nursing procedures. All suspected Nipah virus-infected animals and humans should be quarantined [26]. Infection control techniques should not be replaced by specific pharmaceutical approaches [26]. The intensive care unit is the appropriate setting for monitoring patients who are very unwell. Patients diagnosed with severe pneumonia and acute respiratory failure are required to have mechanical ventilation administered. This incubation phase is thought to last anywhere from three to fourteen days [27].

Fever, headache, dizziness, disorientation, and vomiting are some of the early signs of an infection caused by the Nipah virus. Necrosis of endothelial cells in the vasculature and damage to neural tissue are two of the many adverse effects that may be caused by this virus. Endothelial cells in humans are responsible for vascular permeability; this progressively damages the cell, which results in vessel leakage, and the final outcome is hypovolemic shock. The Nipah virus caused severe symptoms such as encephalitis, convulsions, and coma in those who were infected. However, the epidemic in Malaysia has been linked to a greater number of complications, such as septicemia, intestinal hemorrhage, and renal impairment [28].

In the early stages of Nipah virus infections in Singapore and Malaysia, two medications, ribavirin and acyclovir (Figure 2), were administered to patients. The open-label research in Malaysia indicated that ribavirin reduced mortality by 36% in patients infected with Nipah virus encephalitis, with 45 of 140 patients (32%) in the ribavirin group dying and 29 of 54 (54%) patients developing their symptoms [28], [30]. Serology, histopathology, immunohistochemistry, real-time polymerase chain reaction (PCR), and viral isolation techniques are all laboratory methods for diagnosing Nipah virus infection [28].

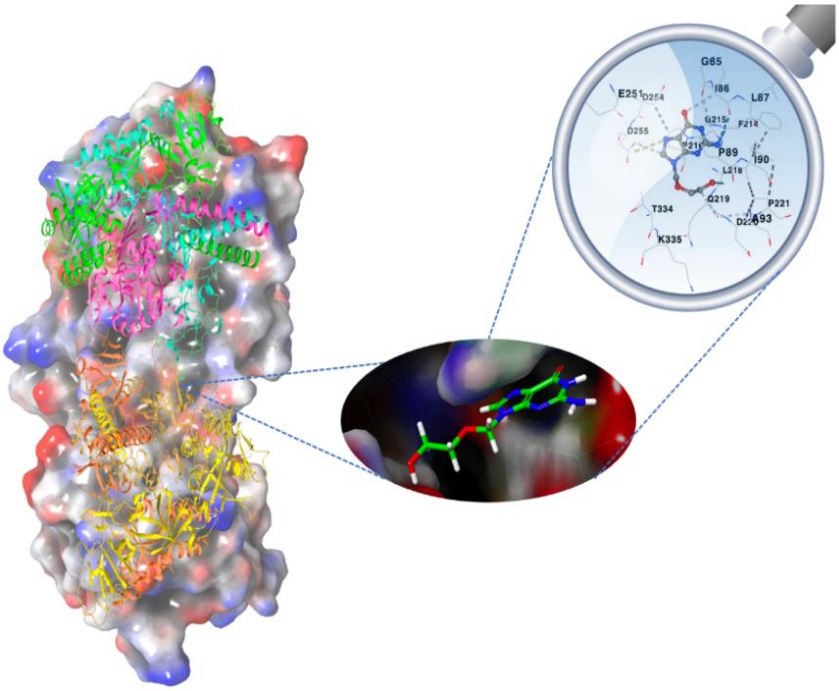


Figure 2. Molecular docking representation between acyclovir and Nipah virus fusion glycoprotein

For Nipah virus infection, the dose form of ribavirin has not yet been specified, however during the first stage, WHO recommends Lassa fever dosage amounts of 30mg/kg for children and 2000 mg/kg for adults, with a maintenance period of roughly 10 days [25]. It is possible to administer ribavirin orally since it does not bind to plasma protein. Ribavirin's side effects include neutropenia (80%-40%), anemia (11%-35%), lymphocytopenia (12%-14%), and the potential for suicidal thoughts.

However, ribavirin has not been shown to be teratogenic in humans but has been reported to be teratogenic in mice and rabbits (Table 2) [29]-[39] . Ribavirin has a lengthy half-life; thus, it must be taken at least seven months before pregnancy to be safe [36]. The short-term safety profile of Ribavirin is impressive, given its promising in-vivo and in-vitro results. Although a controlled study is missing, the status is still unresolved. When it comes to treating nipah virus infection, Ribavirin was recommended without reservation [27]. A study in animals found that ribavirin therapy delayed mortality from hamster infection by around five days, but it did not deter demise. The antimalarial medicinal agent chloroquine works in vitro in a manner similar to ribavirin. For this reason, chloroquine and ribavirin were tested in a golden hamster model for the treatment of Nipah virus infection [27].

Table 2. Drugs with potential antiviral activity

| Drug molecule | Description | Experimental model | Reference |
|---|---|---|---|
| Favipiravir | Purine analogue | In vitro (Syrian hamster) | [29] |
| Ribavirin | Guanosine analogue | In vitro | [30] |
| Acyclovir | Guanosine analogue | Historical review | [31] |
| Remdesivir (GS-5734) | Adenosine analogue | In vitro (African green monkey) | [32], [33] |
| Balapiravir ($R_{1419}$) | Cytidine analogue | In vitro | [34] |
| Chloroquinine | 4-aminoquinoline | In vitro | [35] |
| Poly (I)- Poly ($C_{12}$U) | Interferon inducer | In vitro (Hamster) | [36] |
| Human mAb m102.4 | G glycoprotein fusion inhibitor | Ferrets (African green monkey) | [37] |
| Human mAb h5B3.1 | F glycoprotein fusion inhibitor | Ferrets | [38], [39] |

### 3.3.1. Monoclonal antibody

An antibody that targets the ephrin-B2 and ephrin-B3 receptors, which are neutralized by the antibody in vitro through binding with the area of the Henipavirus G envelope glycoprotein. Nipah virus may be prevented in ferrets by this antibody [36]. In a research, m102.4 protected African green monkeys against infection and death after the injection of a fatal amount (AGM) [37].

### 3.3.2. Favipiravir

An RNA polymerase inhibitor for viral RNA. Favipiravir is an antiviral drug designed to keep influenza at bay. Nipah virus infection in Syrian hamsters may be reduced or inhibited by the use of the antiviral favipiravir drug [40]. In an emergency, anti-G and anti-F monoclonal antibodies are often employed in India. Only if the RT-PCR test is negative on a throat swab or blood sample may the patient be discharged. And it's not clear how long it will last, but we may presume that it will last for 21 days. After the infection has been confirmed, released patients are encouraged to remain in isolation for a minimum of 21 days [29].

The primary component of therapy for a NiV infection is palliative care, which may include the administration of anticonvulsants, the administration of fluids, the treatment of secondary infections, and mechanical ventilation [29]. There are presently no medicines that are authorized that can be used in humans that are effective in treating NiV infection. During the NiV Malaysia epidemic in 1998-1999, the antiviral medication ribavirin was given to 140 patients, which resulted in a 36 percent decrease in death when compared to the mortality rate of 52 control patients who were not treated [29]. On the other hand, the medication distribution was not done in a random way; there is a possibility that the patients who were treated had superior care in general. This makes the result difficult to predict. Subsequent animal challenge trials conducted in hamsters revealed that ribavirin, although it did not prevent mortality after NiV infection, did postpone the onset of the disease. After transmission of African Green Monkeys with the similarly related Hendra Virus (HeV; see also Hendra Virus), researchers found comparable results [65].

## 4. CURRENT STATE OF NIPAH VIRUS VACCINE (NIV) DEVELOPMENT

Undoubtedly, Nipah virus is an emerging, unapparent & mild disease, but there is no approved vaccine for humans or any livestock. The approach of the NiV vaccine focused on antigens that are delivered as subunit vaccines or using viral vectors (Table 3) [41]-[55]. Nowadays, the most studied vaccine participant is the soluble form of the G protein that is highly related to the Hinda virus [56]. But currently, there are no drugs or vaccines for the Nipah virus Research and Development Blueprint. They advise treating severe respiratory and neurologoic complicatis axons [57]. Although NiV is an infectious & pathogenic disease, there is no licensed human vaccine; there is potent proof that an effective vaccine is feasible. The recent evidence suggests that the average incubation period for Niv is above the 5-7 days' threshold, and so has a strong potential for the development of a successful vaccine [58].

Vaccine developed by Auro Vaccine and Public Health Vaccines is the first in the world to reach clinical trial. All R&D activities for NiV vaccines are in the pre-clinical stage, having been tested in the hamster, ferret, and/or AGM preclinical challenge models (Table 4, see in Appendix) [2], [3], [59]–[65]. Suggestion by a number of factors suggest that developing a safe and effective human prophylactic vaccine against NiV is scientifically possible. There is a broad agreement that neutralizing antibodies confer protect againt NiV infection [66]. The pigs suffered during the Malaysian outbreak caused by the NiV virus. The severity of symptoms of NiV infections in pig differ with age. An adjuvanted Hev SG protein subunit- based vaccine has been licensed in Australia to protect horses against Hev and to reduce the zoonotic risk to humans. But unfortunately, this vaccine failed to treat pigs from experimental challenge with NiV [67].

Among all studies of various animals, during initial studies, mice were found to be highly resistant to infection with NiV. Recently, two mouse models were given for the experiment [50]. According to that experiment, it showed that aged animals are susceptible to NiV infection via the intranasal route. So far, vaccine research has concentrated on the viral F and G envelope glycoproteins, either produced in recombinant virus or subunit immunogens, to protect against the henipavirus.

The immunogen HeV recombinant soluble G glycoprotein (sG) has also been used in many research studies (HeVsG). Cats were able to withstand a deadly NiV challenge with no clinical indications, and the results indicated that this animal model had developed a sterilizing immunity. Reisolated virus was found in the brains of six cats that had received the second greatest doses of vaccination, or viral RNA was found in the brains of one vaccinated cat.

PHV02 is an Rvsv vector vaccine candidate expressing the Nipah virus glycoprotein in the Bangladesh strain of Nipah virus vaccine (PHV02). It is a live, attenuated Rvsv vector vaccine candidate. It will also be used to find the development of critical research tools, such as assays and antibody standards, for forthcoming Phase II clinical trials of Nipah vaccinations, which are presently expected to begin in mid-to-late 2022. At this time, the field considers the ferret and AGM models to be the most appropriate animal models for reproducing henipavirus infection and illness, as well as for the assessment of novel anti-henipavirus treatments and vaccines. In earlier research, the authors and others, including others, have put the HeV-sG recombinant subunit immunogen through its paces as a potential vaccine candidate in cats, ferrets, horses, and other animal models of the disease [68].

Table 3. Animals tested NiV vaccine candidates

| Reference | Vaccine description | Animal(s) immunized | Vaccination route/Regimen/Challenge (Strain3) | NiV neutralization titers (Pre-challenge)2 |
|---|---|---|---|---|
| [41] | Vaccinia virus (VV) vector expressing NiV G or F | Hamsters | SC/2 vaccinations 1 month apart/challenge 3 months after last vaccination (M) | ~ 1:10 – 1:25 |
| [42] | Canarypox vector (ALVAC) expressing NiV G or F | Pigs | IM/2 vaccinations 14 days apart/challenge on day 28 post 2nd vaccination (NiV strain not specified) | 1:200 – 1:1280 |
| [43] | Venezuelan equine encephalitis virus (VEEV) expressing NiV G or F | Mice | Footpad inoculation/3 vaccinations on week 0, 5 and 18/no challenge | ~ 1:215 – 1:217 * |
| [44] | Newcastle disease virus (NDV) vector expressing NiV F or G | Pigs | IM/2 vaccinations 4 weeks apart/no challenge | ~ 1:27 – 1:212* |
| [45] | Single-cycle replication VSV-ΔG vector expressing NiV G or F | Ferrets | IM/one vaccination/challenge on day 20 post-vaccination (M). | ~1:40 – 1:160 |
| [46] | Adeno-associated virus (AAV) vector expressing NiV G | Hamsters | IM/one vaccination/challenge at 5 weeks post-vaccination (M). | < 1:10 to 1:160 |
| [47] | Measles virus vaccine vector expressing NiV G glycoprotein | Hamsters, AGM* | Hamster: IP/2 vaccinations 21 days apart/challenge 7 days post 2nd Vaccination AGM. SC/2 vaccinations 28 days apart/challenge 2 weeks post 2nd vaccination (NiV strain not specified). | Hamster: Not reported AGM: 1:1600 – 1:3200 |
| [48] | Replication-competent VSV vector expressing NiV G or F | Hamsters | IP/one vaccination/challenge on day 28 post-vaccination (M). | 1:80 - ≥ 1:640 |
| [49] | Replication-defective VSV-ΔG vector expressing NiV G or F. | Hamsters | IM/ one vaccination/challenge at day 32 post-vaccination (M) | ~ 5 x103 – 1 x 104 |
| [50] | Canarypox vector (ALVAC) expressing HeV G or F | Ponies (horses) | IM/2 vaccinations 21 days apart/ no challenge | ~ 1:2128* |
| [51] | Live-attenuated VSV vector expressing NiV G | AGM1 | IM/one vaccination/challenge on day 29 post-vaccination (M). | 1:80 – 1:160 |
| [52] | Live attenuated VSV vector expressing NiV G | Hamsters | IP/one vaccination/challenge one day post- vaccination (100% survival) (M). | Not reported |
| [53] | Live-attenuated rabies virus vaccine vector (RABV) expressing NiV G | Mice | IM/2 vaccinations 28 days apart/no challenge | ~1:10 to 1:600 (no challenge) |
| [54] | Single-cycle replication VSV-ΔG vector expressing NiV G or F | AGM1 | IM/one vaccination/challenge on day 28 post-vaccination (B). | 1:160 – 1:640 |
| [55] | Chimpanzee adenovirus (ChAd) vector expressing NiV G | Hamsters | IM/one or two vaccinations (28 days apart)/challenge 70 days post-prime or 42 days post- boost (M and B). | ~1:40 - ~1:100 |

## 5. CONCLUSION

Nipah virus (NiV) remains a significant emerging zoonotic pathogen with substantial pandemic potential. Accumulating evidence suggests that NiV is capable of adapting to new host species and transmission pathways beyond its primary reservoir, *Pteropus* fruit bats. Since its initial identification, NiV has demonstrated the ability to spread to humans through contact with infected animals, particularly pigs, as well as through human-to-human transmission. Repeated spillover events and subsequent human transmission may substantially increase the overall disease burden, highlighting the importance of controlling both zoonotic and interpersonal transmission pathways. Despite efforts to reduce exposure through physical and environmental interventions, the risk of NiV transmission persists, particularly in regions with close interactions between humans, livestock, and wildlife. Current research demonstrates promising progress in vaccine development, with several candidate vaccines showing efficacy in animal models such as hamsters, ferrets, and African green monkeys. These models have been instrumental in replicating key features of human NiV infection and pathogenesis. In addition, advances in immunological assays and the identification of neutralizing antibodies are expected to support the establishment of correlates of protection, although assay standardization remains in its early stages. However, significant challenges remain in translating these findings into effective human interventions. There are currently no licensed vaccines or specific antiviral therapies for NiV, and treatment remains largely supportive. Therefore, the development of safe and effective vaccines and antiviral agents is urgently needed. In parallel, strengthening public health strategies—such as surveillance of high-risk populations, improved livestock management, and community education on hygiene and zoonotic transmission—is essential to reduce the risk of future outbreaks. In conclusion, a comprehensive and integrated approach combining biomedical research, vaccine development, and public health interventions is critical to mitigate the threat posed by NiV. Continued interdisciplinary research and global collaboration will be essential to enhance preparedness and prevent future epidemics.


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| C : **C**onceptualization | I : **I**nvestigation | Vi : **Vi**sualization |
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## CONFLICT OF INTEREST STATEMENT

Authors state no conflict of interest.

## DATA AVAILABILITY

Data availability does not apply to this paper as no new data were created or analyzed in this study.

## APPENDIX

Table 4. NiV submits animal-tried vaccine candidates

| Reference | Vaccine description | Animal(s) immunized | Vaccination route/Regimen/Challenge (Strains) | NiV Neutralization Titers (Pre-challenge)2 |
|---|---|---|---|---|
| [59] | SGNiV or sGHeV adjuvanted with Montanide/QuiIA/DEAE dextran | Cats | SC/3 vaccinations 2 weeks apart/challenge l5 weeks after the first vaccination (M). | 1:2560-1:2O48O |
| [60] | Recom bina nt soluble HeVG glycoprotei n adjuva nted with CpG + AlhydrogelTM | Cats | IM/ 2 vaccinations 21 days apart/challenge onday 42 post 1st vaccination (M). | 1:32-1:512 |
| [61] | Virus like particles (VLPs) comprising NiV M, G and F | Mice | SC/ 3 vaccinations on days 0, 15 a nd 29/no challenge | 1:5->1:8O |

Table 4. NiV submits animal-tried vaccine candidates (continued)

| Reference | Vaccine description | Animal(s) immunized | Vaccination route/Regimen/Challenge (Strains) | NiV Neutralization Titers (Pre-challenge)2 |
|---|---|---|---|---|
| [62] | Recombinant soluble HeVG glycoprotein adjuvanted with CpG + AlhydrogelTM | AGM1 | IM/ 2 vaccinations 21days apart/challenge 2ldays post 2nd vaccination (M). | 1:67-1:379 |
| [63] | Recombinant soluble HeVG glycoprotein adjuvanted with CpG | Ferrets | SC/2 vaccinations 20 days a part/cha Ilenge 20days or 14 months post 2 nd vaccination (B) | 1:16-1:128 |
| [64] | Recombinant soluble HeV G glycoprotein in a proprietary adjuvant (Zoetis, Inc.) | Pigs | IM/ 2 vaccinations 21 days apart/challenge 35 days post 1st vaccination (Strain not specified) | 1:25-1:450 |
| [65] | Virus-like panicles (VLPs) containing NiVM, F and G | Hamsters | Single dose trial: TM/ one dose/challenge on day 28 post vaccination (M)<br>3 dose trial: 3 doses on days 0, 21 and 42/challenge on days 58 (M). | 3-dose trial 1:200-1:2500<br><br>2-dose trial 1:10-1:200 |

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