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Review Article

Molecular diversity and evolutionary dynamics of Nipah virus: implications for surveillance and control

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ABSTRACT

The Nipah virus (NiV) is a highly dangerous zoonotic virus that continues to threaten global health. Its alarming case fatality rate, ability to spread from person to person, and potential to jump from wildlife to humans make it a serious concern. To effectively monitor and control this virus, it's crucial to understand its molecular diversity and how it evolves. We carried out an extensive review of existing genomic, phylogenetic, and epidemiological research on NiV. Our focus was on understanding its genetic diversity, patterns of recombination, and traits specific to different lineages. We also looked into the current shortcomings in surveillance, diagnostics, and public health responses through a One Health lens. We identified two main lineages—NiV-Malaysia (NiV-MY) and NiV-Bangladesh (NiV-BD)—which show unique transmission patterns and genetic characteristics. Our phylogenomic analysis uncovered lineage-specific mutations in the glycoprotein and polymerase genes, some of which are under positive selection. Additionally, recombination events and mutations within hosts indicate that the virus is adapting. However, there's still a lack of data from wildlife reservoirs, and many diagnostic tools do not account for genetic variations. These insights underscore the urgent need for improved surveillance strategies, outbreak readiness, and vaccine development. To prevent the emergence of NiV, we urgently need to enhance genomic surveillance, integrate one health approaches across sectors, and conduct functional studies on mutations that define different lineages. Adapting diagnostics and vaccine strategies to keep pace with viral evolution will be crucial in reducing the risk of future outbreaks.

Keywords: Nipah virus, Phylogenetics, Molecular diversity, Zoonosis, Genomic surveillance, One health, Outbreak control

INTRODUCTION

The Nipah virus (NiV) is a serious zoonotic virus that falls under the Henipavirus genus within the Paramyxoviridae family. It was first spotted during an outbreak in Malaysia between 1998 and 1999, and since then, it has emerged as a major public health concern due to its alarming case-fatality rate, which ranges from 40% to 75%. This virus has a wide range of hosts and can spread from person to person.

Fruit bats of the Pteropus genus are the primary carriers, and humans can become infected through intermediate hosts like pigs or by coming into contact with food contaminated by bats.[1]

Visual workflow chart description

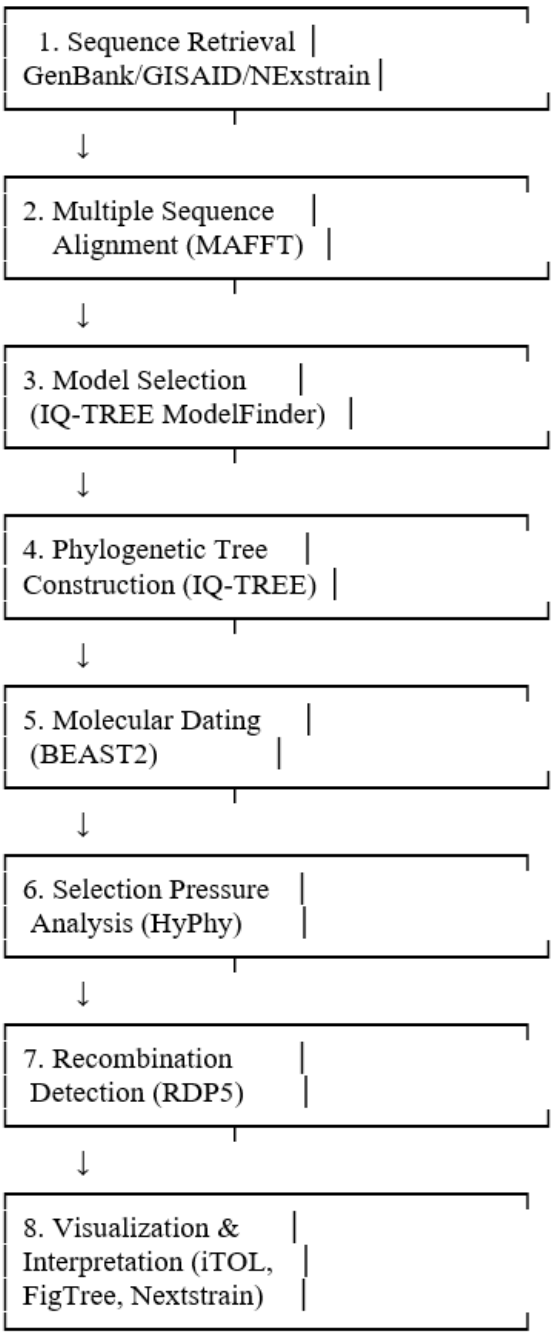

Figure 1: Workflow for molecular and evolutionary analysis of Nipah virus.

In the last twenty years, NiV has triggered repeated outbreaks that have spread across Bangladesh, India, and various regions in South and Southeast Asia. Interestingly, the virus shows different transmission patterns and levels of severity depending on its strain, which raises intriguing questions about how it evolves and its potential to spread even further.[2]

To effectively predict future outbreaks, enhance diagnostic methods, and create successful vaccines and treatments, it's crucial to understand the molecular diversity and evolutionary patterns of NiV. Previous genomic research has revealed two main lineages—Malaysia and Bangladesh/India that exhibit significant genetic differences and unique epidemiological traits. This article sets out to: Provide an overview of what we currently know about NiV's genomic structure and its sequence variability.[2]

Explore the evolutionary processes at play, including mutation, selection, and recombination.

Discuss how these insights can inform genomic surveillance, bolster public health readiness, and aid in developing control strategies within a one health framework.[3]

NiV is a single-stranded RNA virus with a negative-sense genome that's about 18.2 kb long. It belongs to the Henipavirus genus, which is part of the paramyxoviridae family.[4] This genome encodes six structural proteins: nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), glycoprotein (G), and large polymerase (L), all arranged in the 3′–N–P–M–F–G–L–5′ order.[2] Additionally, the P gene produces three nonstructural proteins—C, V, and W—through alternative open reading frames and RNA editing. These proteins are known to help the virus evade the immune system by interfering with interferon responses.[4,5]

The N protein wraps around the viral RNA genome, creating the ribonucleoprotein (RNP) complex, which is vital for the virus's replication and transcription.[6] The P protein acts as a cofactor for the polymerase and assists the N protein, while also helping to suppress the host's innate immune response.[6] Meanwhile, the M protein is crucial for the assembly and budding of the virus at the host cell membrane.[7]

The F and G glycoproteins are key players in how the virus enters host cells. The G protein helps the virus attach to specific receptors on host cells, especially ephrin-B2 and ephrin-B3, which are found in high amounts in endothelial and neuronal tissues.[8] Once attached, the F protein facilitates the fusion of membranes, a critical step for the virus to enter the cell and form syncytia.[9] Variations in the amino acid sequences of the G and F glycoproteins across different strains can influence how well they bind to receptors, their cell tropism, and their virulence.[10]

Genomic comparisons show that NiV shares about 92% nucleotide identity with the Hendra virus (HeV), which is another member of the Henipavirus genus.[11] However, the differences in how these two viruses cause disease and spread are partly due to variations in their surface glycoproteins and accessory proteins.[12] It is interesting to note that the NiV-Bangladesh lineage tends to spread more easily from person to person compared to the Malaysia lineage. This could be due to some structural

differences in the G protein.[13] While NiV is relatively stable compared to other RNA viruses, it still shows enough genetic flexibility to require ongoing molecular monitoring. This is crucial, especially given its potential to jump from animals to humans and spread quickly across different regions.[14]

Molecular diversity of Nipah virus strains, we find two main genetic lineages: the Malaysia lineage (NiV-MY) and the Bangladesh/India lineage (NiV-BD/IN). These lineages are quite distinct, especially in the glycoprotein and phosphoprotein gene areas.[2-15]

Sequence comparisons reveal that NiV-BD/IN strains share about 91–92% nucleotide identity with the original Malaysian strains, but they also have key amino acid changes that could affect how the virus spreads and its severity.[3-16] The NiV-MY lineage, which was first identified during the outbreak in Malaysia and Singapore in 1998–1999, is more genetically stable and is mainly linked to transmission from pigs to humans.[4-17] On the other hand, the NiV-BD/IN lineage, which appeared in Bangladesh in 2001 and has caused outbreaks in eastern India, shows greater genetic variability and is associated with more frequent human-to-human transmission.[5,6,18] This difference might be a result of ecological and evolutionary adaptations to various host animals and environmental factors.[7-19]

Table 1: Key genetic mutations in Nipah virus and their functional implications.

Gene	Mutation/ region	Lineage/strain	Reported/predicted effect	Reference
G (glycoprotein)	R533Q	Niv-BD	Alters receptor binding affinity	Harcourt et al, 2005
G (glycoprotein)	Y581H	NiV-BD (India, 2021)	Increases surface expression and syncytia formation	Yadav et al, 2022
F (fusion protein)	S447P	NiV-MY vs. NiV-BD	May alter fusion and entry	Wong et al, 2002
N (nucleoprotein)	K258N	NiV-BD	Could influence replication efficiency	Lo et al, 2012
L (polymerase)	T1834I	NiV-BD	May affect replication fidelity	Escaffre et al, 2013
M (matrix protein)	A152T	NiV-BD	Involved in assembly and budding	Freiberg et al, 2008
Whole genome	Intergenic regions	NiV-BD	May influence transcription hierarchy	Mathieu

Phylogenetic analyses of the whole genome show that NiV strains cluster distinctly based on their geographic origin and the year they were isolated, indicating localized evolutionary paths and potential hidden transmission chains.[8] For example, phylogenetic trees based on the G or N genes reveal that Bangladeshi strains collected between 2004 and 2014 form a separate monophyletic group from the Malaysian strains.[20] The mean nucleotide substitution rate is estimated to be around 6.5×10^{-4} substitutions per site per year.[9] This rate aligns with what we see in other RNA viruses, indicating a moderate pace of evolution. It provides enough flexibility for adaptation while still maintaining the integrity of structural proteins.[10-21]

When it comes to differences in glycoprotein gene sequences—especially in the G protein receptor-binding domain—these variations have been linked to a stronger binding affinity to ephrin-B2/B3 receptors found in both human and bat cells.[11-22] Studies conducted in vitro and in vivo suggest that these mutations could be a factor in the increased neurovirulence and the direct spillover events from bats to humans that were noted during the NiV-BD/IN outbreaks.[23] Genomic data also point to variations within lineages. For instance, Indian strains from the 2018 and 2021 outbreaks in Kerala, while classified under the NiV-BD lineage, displayed unique SNP patterns in the N, G, and L genes. This suggests microevolutionary changes likely influenced by local ecological and host factors.[13] However, the relatively small sample size and the limited temporal and spatial coverage of the available sequences highlight the urgent need for more extensive genomic surveillance, especially in wildlife reservoirs like Pteropus medius.[24]

Thanks to advanced sequencing techniques, such as metagenomic next-generation sequencing (mNGS) and amplicon-based deep sequencing, we can now detect NiV diversity in real-time during both outbreak and inter-outbreak periods.[25] By combining genomic data with epidemiological metadata, we can significantly improve our understanding of transmission networks, spillover patterns from reservoirs, and the potential emergence of new variants.

PHYLOGENETICS AND EVOLUTIONARY DYNAMICS OF THE NIPAH VIRUS

It offers fascinating insights into its evolutionary paths and how it spreads across different regions. Early research, which focused on partial genome sequences, identified two distinct genetic lineages: the Malaysia (NiV-MY) and Bangladesh (NiV-BD) lineages. These lineages form well-supported clades on phylogenetic trees.[2-26] Thanks to recent advancements in whole-genome sequencing, we now have a clearer picture of the phylogenetic landscape, revealing sub-lineage diversification, particularly within the NiV-BD group.

This group shows distinct temporal and regional patterns in strains collected from Bangladesh and India.[3-27] By utilizing time-calibrated phylogenetic trees created with Bayesian methods like BEAST, researchers estimate that the most recent common ancestor (tMRCA) of current NiV strains likely existed between 1947 and 1985, depending on the specific model and gene region examined.[4,5,28]

The average evolutionary rate for NiV is estimated to be around $6.5–8.0\times10^{-4}$ substitutions per site per year, which aligns with rates observed in other non-segmented negative-sense RNA viruses.[6] Even though the mutation rates are relatively slow, analyses of selection pressure reveal instances of episodic positive selection at certain codons, particularly in the glycoprotein (G) and polymerase (L) genes. This suggests that the virus may be undergoing adaptive evolution during cross-species transmission.[7] Phylogenomic studies that employ maximum-likelihood and Bayesian inference consistently indicate that geographic location plays a significant role in how strains cluster.

For example, NiV isolates from southern India, such as those from the outbreaks in Kerala in 2018 and 2021, form a distinct subclade within the broader NiV-BD lineage. This reflects local microevolution, likely driven by repeated zoonotic introductions and ongoing low-level circulation in populations of Pteropus medius bats.[8-29] Additionally, while recombination events are rare, some NiV genomes have shown signs of these occurrences, as identified using tools like RDP5 and SimPlot. However, these findings should be interpreted with caution due to the potential for sequencing artifacts and sampling, the significance of full-genome surveillance in spotting emerging variants with unique genomic signatures, despite the limited sampling we have.[30] Molecular clock analyses indicate that different lineages have their own substitution rates, with the NiV-BD lineage evolving a bit quicker than NiV-MY. This could be due to more replication cycles happening during human-to-human transmission.[31] Coalescent-based skyline plots show changes in effective population size over time, which points to episodic outbreaks followed by periods of reduced activity—similar patterns have been seen in other zoonotic viruses like Ebola and Marburg.[32]

Phylogeographic studies have pinpointed the likely origins of NiV spillovers to certain ecological hotspots in Bangladesh and northeastern India. These areas are marked by close human-bat interactions, seasonal fruiting, and a lack of biosecurity during date palm sap collection.[33] Such findings highlight the need to combine viral genomic data with ecological and behavioral information to better understand the factors driving emergence and spread. Thanks to portable sequencing technologies like the Oxford Nanopore MinION and open platforms such as Nextstrain, we can now track NiV evolution in real-time. This advancement paves the way for early warning systems and targeted interventions in high-risk regions.[15-34] When it comes to surveillance and public health control, the insights gained from the molecular diversity and evolutionary dynamics of the NiV are crucial. The presence of multiple lineages—each with its own genetic, epidemiologic, and clinical traits—calls for lineage-informed surveillance and specialized diagnostic tests that can accurately detect both NiV-MY and NiV-BD strains with high sensitivity and specificity.[1,2]

The identification of microevolutionary changes within NiV-BD strains, especially in southern India and Bangladesh, emphasizes the urgent need for active genomic surveillance in areas where the virus is endemic or poses a risk.[3] Real-time sequencing platforms, like portable nanopore devices, have become essential tools for decentralized surveillance. They enable local labs to spot genetic variants and track the movement of viruses over different spaces and times.[35] By incorporating these platforms into national outbreak response strategies, we could significantly reduce the time it takes to get diagnostic results and implement immediate containment measures. Moreover, the cross-species transmission events, especially from pteropus medius to humans, underscore the need for ecological surveillance and the One health approach, which brings together human, animal, and environmental health.[5] Surveillance programs focused on fruit bat populations, particularly during the date palm sap harvesting season and peak fruiting times, can help pinpoint potential spillover opportunities.[36] Keeping an eye on viral load, seroprevalence, and shedding patterns in bat populations is vital for predicting outbreak risks before they affect humans. Public health strategies also need to take into account the differences in transmission among NiV lineages. NiV-BD has shown a tendency for frequent human-to-human transmission, including hospital outbreaks with high secondary attack rates and case fatality ratios that can exceed 70%.[37] This reality calls for stringent infection prevention and control (IPC) measures, such as contact tracing, the use of personal protective equipment, and isolation facilities during outbreaks in areas where the virus is endemic.[8] When it comes to diagnostics, the molecular variations in the N and G genes mean that we need to regularly validate and update PCR primers and probes to ensure diagnostic accuracy.[9]

Likewise, serological tests must be validated against the local strains that are currently circulating, especially when they are used in surveillance or sero-epidemiological studies involving wildlife and human populations.[40] In terms of preparedness, we currently face some gaps, including a lack of genomic data from wildlife reservoirs, the absence of real-time data-sharing protocols, and insufficient public awareness in high-risk areas. Rural communities face significant challenges, and tackling these issues calls for global teamwork and investment in genomic infrastructure, bioinformatics capabilities, and strategies that engage the community.[41]

Currently, there is not a licensed vaccine available, but several candidates, like the ChAdOx1-NiV and HeV-sG subunit vaccines, are making their way through preclinical and early clinical development. Understanding genetic stability and immune escape mutations, particularly in surface glycoproteins, will be crucial for guiding vaccine design and assessing how well they protect against different lineages.[42] In conclusion, combining genomic surveillance with focused ecological monitoring and community-driven interventions creates a comprehensive approach for early detection, swift containment, and long-term prevention of NiV outbreaks. As molecular tools become more accessible, it's essential to weave them into national and regional preparedness plans to help reduce the risk of future zoonotic spillovers.[43]

Future directions and research gaps

Even with the progress we've made in molecular virology and phylogenetics, there are still some significant gaps in our understanding of the NiV that make it tough to manage effectively. To really tackle this issue, future research needs to fill these gaps so we can develop better prevention strategies, treatments, and ways to predict outbreaks.

Expanded genomic surveillance in reservoir hosts

One of the biggest challenges we face is the lack of viral genomic data from bat reservoirs, especially from species like Pteropus medius and P. vampyrus, across their habitats. Most of the genomic data we have, comes from human outbreak samples, which creates a bias and makes it hard to trace how the virus spreads between species. We need to systematically sample across different seasons, bat species, and ecological environments to uncover hidden viral lineages, keep an eye on recombination events, and understand how the virus and its hosts evolve together.[44]

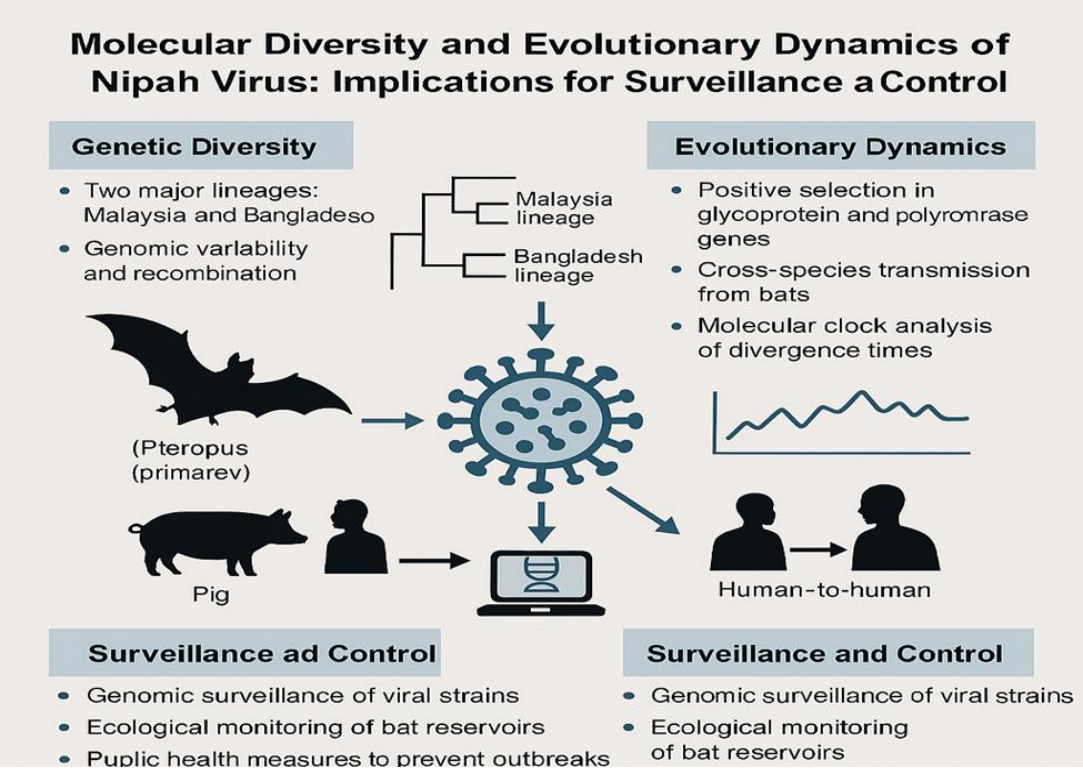

Figure 2: Molecular diversity and evolutionary dynamics of Nipah virus: implications for surveillance and control.

Longitudinal studies on viral evolution and host immunity

There is a real shortage of long-term data on how the virus evolves within and between hosts. It's crucial to know how long the virus sticks around, how it evades the immune system, and what mutations happen within a host. This information is key for predicting transmission risks and designing effective vaccines.

Additionally, population-level serosurveys can help us understand the true impact of asymptomatic or mild infections, particularly among bat handlers, pig farmers, and date palm collectors.[45]

Functional genomics and viral fitness studies

While molecular phylogenetics has pointed out mutations that define different lineages, there haven't been many

studies that experimentally confirm how these mutations affect viral replication, transmissibility, or virulence.

Using reverse genetics systems and in vitro models with primary bat or human cells could shed light on how specific mutations in the G, F, and L proteins impact viral fitness or help the virus escape the immune response. These findings would be invaluable for targeting therapies and assessing the risks of new variants.[46]

Cross-sectoral One health integration

One significant issue we face is the limited implementation of the One health approach. Many surveillance systems operate in isolation, failing to effectively combine wildlife virology, human health data, and ecological modeling.[43] To tackle this, future frameworks should focus on fostering collaboration between ministries, establishing data-sharing protocols, and providing cross-disciplinary training. This will help us detect zoonotic threats early at the intersection of human, animal, and environmental health.

Diagnostic and vaccine development gaps

Right now, our diagnostic methods depend on conserved regions of the viral genome. However, it's crucial to regularly reassess the specificity of primers and probes because of the mutations that keep piling up, especially in areas showing signs of recombination or positive selection.[47] Additionally, while there are some promising vaccine candidates in the pipeline (like ChAdOx1-NiV, mRNA platforms, and subunit vaccines), we have not yet seen any human efficacy trials, and we still need to test their immunogenicity across different lineages.[45-48]

Predictive modeling and outbreak forecasting

When it comes to predicting NiV outbreaks, we're just getting started. Most existing models do not take into account genomic data, vector ecology, or climate factors. By developing strong, data-driven models that integrate environmental influences, human movement, and virus evolution, we could significantly improve our preparedness for outbreaks and better allocate resources in areas where the virus is endemic.[9-49]

CONCLUSION

The molecular diversity and evolutionary dynamics of the NiV highlight its persistent threat as a zoonotic pathogen with the potential for a pandemic. The existence of distinct genotypes—mainly NiV-MY and NiV-BD that exhibit different levels of virulence, transmission patterns, and ecological reservoirs calls for careful, lineage-specific surveillance strategies. Over the last two decades, the accumulation of genomic data has greatly improved our understanding of NiV evolution, showing signs of positive selection, recombination, and cross-species transmission, especially at the human-bat interface.

Nevertheless, despite these advancements, we still need to ensure that our surveillance and response strategies are robust and adaptable to the evolving nature of this virus. Understanding the viral diversity among bat populations is still quite limited, and the lack of frequent validation for genomic mutations makes it tough for us to predict how these viruses might emerge or evade vaccines. On top of that, our diagnostic tools and vaccine development need to keep pace with the viruses ever-changing genetic makeup, all while being backed by comprehensive One health surveillance systems.

To reduce the chances of future outbreaks, we really need to enhance real-time genomic surveillance, invest in long-term studies that explore host-pathogen interactions, and encourage collaboration across different sectors. By using molecular insights to guide ecological monitoring, public health strategies, and applied research, we can significantly lower the risk of NiV spillover and boost our ability to respond to outbreaks in both endemic and emerging areas.

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