

РОЗДІЛ 1. Емерджентні хвороби та Єдине здоров'я
SECTION 1. Emergent diseases and One Health

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CHARACTERISTICS OF THE PATHOGEN, EPIZOOTOLOGICAL FEATURES, CLINICAL
SIGNS, DIAGNOSTICS AND PREVENTION OF NIPAH VIRUS DISEASE
(REVIEW ARTICLE)

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Abstract. *The results of an analysis of international scientific literature on the epizootic situation of the emerging zoonotic disease Nipah virus disease are presented. The virological, serological, and molecular-genetic characteristics of Nipah virus (NiV), which belongs to the family Paramyxoviridae, subfamily Orthoparamyxovirinae, genus Henipavirus, are described. The structure of its genome is outlined; it consists of six genes (N, P, M, F, G, and L) encoding the nucleoprotein (NP), phosphoprotein (P), matrix protein (M), fusion protein (F), glycoprotein (G), and large RNA-dependent RNA polymerase (L), respectively.*

The pathogen is shown to replicate efficiently in various primary and continuous mammalian cell cultures. In Vero cell culture, a cytopathic effect (CPE) is observed on days 3–6 of the first passage; after prior viral adaptation, CPE appears within 24–48 hours.

The main natural reservoir hosts of the virus are fruit bats of the genus Pteropus, including Pteropus vampyrus, Pteropus hypomelanus, Pteropus medius (formerly P. giganteus), and Pteropus lylei; Pteropus poliocephalus has been infected experimentally. The virus is capable of infecting pigs, humans, cattle, goats, cats, dogs, and horses. Under natural conditions, pigs are most commonly infected, and transmission to humans primarily occurs from infected pigs.

Transmission from bats to other hosts occurs through viral shedding in saliva and urine; contamination of food and water sources creates conditions for infection of other animals and humans. Fewer than 10% of patients are capable of transmitting the virus to other individuals; most cases of person-to-person transmission occur in the later stages of the disease, particularly in patients with respiratory symptoms. The case fatality rate during major outbreaks has ranged from 38% to approximately 75%.

The development of vasculitis indicates that endothelial cells of blood vessels are the primary target cells; the central nervous system, lungs, and kidneys are severely affected. Histopathological findings include hemorrhagic or necrotizing alveolitis, pulmonary edema, aspiration pneumonia, intra-alveolar inflammatory cells, and occasionally multinucleated giant cells.

Diagnosis is based on epidemiological data, clinical signs, pathological findings, and laboratory methods (virological, serological, and molecular-genetic assays). Based on global experience, measures for control, prevention, and surveillance of this disease are substantiated.

Keywords: Nipah virus disease, Paramyxoviridae, epizootic situation, factors of pathogen spread, diagnosis, control measures, prevention, surveillance.

Nipah virus disease is an emerging zoonotic infection that may manifest as a chronic infection with viral persistence or as a respiratory syndrome and encephalitis in pigs and humans. The case fatality rate in humans may reach 40–75%.

New infectious diseases reported over the past 25–30 years unfortunately possess significant zoonotic potential. A considerable proportion of them are transmitted by bats. One such infection is Nipah virus disease (NiV). The causative agent belongs to the recently classified genus of *paramyxoviruses* within the family *Paramyxoviridae*.

Nipah virus disease virus causes fatal encephalitis in humans. The virus is carried subclinically by fruit bats of the genus *Pteropus*, a host to which it appears to be well adapted. The virus was apparently transmitted from bats to pigs around 1996 and subsequently persisted in pig populations.

In 1998, outbreaks were reported in Malaysia among pig farmers. The first outbreaks occurred in the Kinta district of Perak, followed by additional outbreaks in three districts of Negeri Sembilan, including Sungai Nipah. The outbreaks continued until February 1999, with later cases associated with the movement of infected pigs. The previously unknown disease caused mild illness in pigs; however, approximately 100 out of 300 infected people died. To control the outbreak, more than one million pigs were culled (Chua, 2003; Chua, 2010).

In March 1999, a similar disease was reported at 11 abattoirs in Singapore, where one person died. The pathogen had been introduced with pigs imported from Malaysia (Paton, 1999). Investigation of these events led to the discovery of Nipah virus in March 1999. The pathogen was named “Nipah virus” after the first isolation from a human patient in the Sungai Nipah area (Wong, 2002). Other species, including cats, dogs, and goats, were also affected.

The Malaysian outbreaks were controlled in both animals and humans by culling more than one million pigs. In addition, pig farming was permanently banned in certain high-risk areas. Since then, no further cases of Nipah virus–associated encephalitis have been documented in Malaysia, although serological evidence of human exposure exists in some regions.

However, sporadic clinical cases have been reported in Bangladesh and India since 2001. Many affected individuals appear to have been infected directly from bats through consumption of raw date palm sap, a widely consumed local delicacy, after contamination by bats visiting unprotected collection sites. In addition, person-to-person transmission has occasionally occurred during close, unprotected contact, particularly from patients with respiratory symptoms and/or in the late stages of disease.

One outbreak occurred in Philippines, affecting individuals who slaughtered or consumed undercooked horse meat from animals with encephalitis. Evidence of Nipah virus circulation has also been detected in bats in several Asian countries where no clinical cases have ever been reported, suggesting that the risk of disease may be more widespread (CFSPH, 2025).

Thus, although Nipah virus disease has officially been reported in only four countries worldwide (Malaysia, Singapore, India, and Bangladesh), its potential for spread remains considerable due to the involvement of fruit bats as reservoir hosts (Kornienko, 2020).

The causative agent of **Nipah virus disease (NiV)** is an enveloped virus containing a non-segmented, single-stranded negative-sense RNA genome. It belongs to the family *Paramyxoviridae*, subfamily *Orthoparamyxovirinae*, genus *Henipavirus*.

The genus *Henipavirus* and the closely related genus *Parahenipavirus* include Hendra virus, a highly pathogenic virus, as well as several other recognized (e.g., *Cedar* virus) or proposed members that may cross-react with Nipah virus in certain diagnostic assays. Some of these recently described viruses appear to be non-pathogenic; one (*Langya* virus) has been proposed as a human pathogen, while the clinical significance of others remains unclear.

Nipah virus strains identified in humans in Bangladesh and India appear to be more virulent than the strains responsible for the pig-associated outbreak in Malaysia. The virus detected during the outbreak in Philippines was most closely related to the Malaysian strains (CFSPH, 2025).

The nucleocapsid of Nipah virus (NiV) measures approximately 18 nm in diameter. The genome consists of six genes—N, P, M, F, G, and L—encoding the nucleoprotein (NP), phosphoprotein (P), matrix protein (M), fusion protein (F), glycoprotein (G), and large RNA-dependent RNA polymerase (L), respectively (Chan, 2001).

It is assumed that the G and F glycoproteins of NiV mediate viral entry into host cells and also play a major role in inducing neutralizing antibodies. Infection of the host cell requires coordinated interaction between these two glycoproteins (Harcourt, 2000; Harcourt, 2001).

The nonstructural C protein regulates viral RNA synthesis and the expression of proinflammatory cytokines, thereby coordinating chemokine-induced immune responses and potentially serving as an important virulence factor by influencing the fatal outcome of infection.

The V protein of Nipah virus (*NiV*) plays a key role in regulating interferon (*IFN*) signaling. It is also known to form high-molecular-weight complexes in the cytoplasm, thereby inhibiting host cell signaling functions. In addition, the W protein of *NiV* has been reported to possess immunosuppressive properties.

The phosphoprotein (*P*) is the only essential product of the *P* gene required for genome replication; additional gene products are not necessary for viral replication in vitro but may contribute to virulence in vivo (Lamb, 2005).

A slight difference was identified between the genomes of Malaysian and Bangladeshi *NiV* isolates: the former contains 18,246 nucleotides, whereas the latter have 18,252 nucleotides. This variation has been attributed to viral passage in the human population over the course of a year (Harcourt, 2005).

Nipah virus (*NiV*) is antigenically closely related to Hendra virus (*HeV*). *NiV* is completely neutralized by antibodies against Hendra virus. It shares approximately 70–78% nucleotide homology with *HeV* in three major genes—*N*, *P*, and *M* (Daniels et al., 2001).

NiV replicates efficiently in developing chicken embryos, which serve as a useful model for studying the vascular and neuronal tropism of the virus (Tanimura et al., 2006). However, due to its high pathogenicity, research on *NiV* requires biosafety level 4 (BSL-4) laboratory containment (Lo, 2008).

The virus also replicates well in various primary and continuous mammalian cell cultures. Studies using Vero cells have shown that cytopathic effects (*CPE*) appear on days 3–6 during the first passage and within 24–48 hours after prior viral adaptation.

Experimental infection has been successfully achieved in guinea pigs, hamsters, ferrets, and non-human primates (squirrel monkeys and African green monkeys). In contrast, mice and rats are not susceptible to infection (Wong, 2003; Torres-Velez, 2008; Geisbert, 2010; Marianneau, 2010).

Environmental stability of the virus remains insufficiently studied; according to unpublished data, *NiV* may survive for several days in fruit juice or urine. Like other members of the family *Paramyxoviridae*, *NiV* is readily inactivated by soaps, detergents, and disinfectants. Regular cleaning and disinfection with standard, high-quality disinfectants are effective against the virus (Lam, 2002). For example, sodium hypochlorite was successfully used on pig farms in Malaysia (Mohd Nor, 2000).

In general, as with other paramyxoviruses, Nipah virus is expected to be easily inactivated on surfaces by soap, detergents, and many common disinfectants. Agents proven effective against *NiV* on surfaces include 40–80% ethanol, sodium hypochlorite, and certain quaternary ammonium compounds. Additionally, 4% paraformaldehyde and 10% neutral buffered formalin have been effective for inactivating the virus in infected cells and tissues. Sodium hypochlorite has been specifically recommended for disinfection of pig farms in Malaysia.

Nipah virus (*NiV*) was not completely inactivated in artificial palm sap heated to 70 °C (158°F) for 1 hour; however, it was inactivated at 100 °C (212 °F) after 15 minutes of heating. Ultraviolet light has also been shown to effectively inactivate the pathogen (CFSPH, 2025).

Epizootiological data. Fruit bats of the genus *Pteropus* (flying foxes) are the principal reservoir hosts of Nipah virus (*NiV*). Species known to carry the virus include *Pteropus vampyrus*, *Pteropus hypomelanus*, *Pteropus medius* (formerly *P. giganteus*), and *Pteropus lylei*, while other flying foxes such as *Pteropus poliocephalus* have been successfully infected experimentally.

Viral RNA and/or antibodies have also been detected in several other genera of frugivorous or insectivorous bats; however, their significance as reservoir hosts remains unclear. Some bats, for example *Rousettus aegyptiacus*, appear resistant to experimental infection.

Nipah virus can also be maintained in pig populations, and several other mammals have been infected during outbreaks, most likely as incidental (spillover) hosts. Clinical cases have been reported in dogs, cats, goats, and horses, and infection of sheep and cattle has been suggested. The role of horses in the 2014 human outbreak in Philippines has not been fully clarified, although epidemiological data suggest that human cases were associated with sick horses (CFSPH, 2025).

The virus has the capacity to infect numerous mammalian species. The primary natural reservoir host is fruit bats (Yob, 2001). The pathogenic spectrum of *NiV* includes pigs, humans, cattle, goats, cats, dogs, and horses, and antibodies to the virus have been detected in all of these species (Tamin, 2009).

During the first outbreaks in Malaysia, pigs were the main naturally infected species, and transmission to humans occurred primarily from infected pigs (Chua, 1999). Subsequently, infections were identified in dogs, cats, and horses that had contact with infected pigs.

Experimental infection has been reproduced in cats (Middleton et al., 2002), golden hamsters, guinea pigs (Torres-Velez, 2008), African green monkeys (*Chlorocebus aethiops*) (Geisbert, 2010), squirrel monkeys (*Saimiri sciureus*) (Marianneau et al., 2010), ferrets, non-human primates, and golden hamsters (*Mesocricetus auratus*). Some laboratory mouse strains

can also be infected; however, wild mice appear to be resistant, and no evidence of infection in rats was found during the Malaysian outbreak (CFSPH, 2025).

Following the initial report of Nipah virus (*NiV*) infection in Malaysia during 1998–1999 (CDC, 1999; Chua, 1999), a single case was reported in Singapore in 1999 (Paton, 1999), multiple human outbreaks occurred in Bangladesh between 2001 and 2015 (Kulkarni, 2013), and two outbreaks were documented in India (Chhadha, 2006; Arankalle, 2011).

Cases with typical clinical presentations in humans have been reported in Malaysia (1998–1999), Singapore (1999), India (2001 and 2007), and Bangladesh (2001–2015). In Bangladesh, multiple outbreaks since 2001 have been associated with significant human morbidity and nearly 70% mortality. No other countries have reported human cases. Fatalities in pigs were recorded in Malaysia, and one case of transmission occurred via infected pigs imported to a Singaporean abattoir. Nevertheless, bats, which serve as *NiV* reservoirs, inhabit several countries ranging from West Africa to South, Southeast, and East Asia (Kulkarni, 2013). Despite the presence of Hendra virus reservoir bats in Queensland, Nipah virus has not been detected in Australia (Breed, 2013).

Fruit bats are often considered orchard pests. They are hunted for crop protection, sport, food, and traditional medicine. Bats are the only mammals capable of sustained flight over hundreds of kilometers and can travel more than 2,000 km per year. This long-range mobility may have significant implications for disease spread, as bats are reservoirs for numerous high-risk pathogens, including *NiV* (Breed, 2006).

Pteropus bats from Southeast or South Asia have been found to carry antibodies against Henipaviruses. East African bats have also shown substantial population seropositivity for *NiV*. Studies indicate that of 23 bat species examined, at least 10 tested positive for *NiV* antibodies. These bats are widely distributed in countries including Bangladesh, China, India, Cambodia, Thailand, Indonesia, Papua New Guinea, Madagascar, the Gulf of Guinea, Cameroon, Nigeria, and parts of West Africa.

NiV has been isolated from urine samples of *Pteropus vampyrus* and *Pteropus hypomelanus*, as well as from partially eaten fruit in Malaysia (Chua, 2002); from *Pteropus lylei* in Cambodia (Reynes, 2005); from urine of *P. hypomelanus* and *P. lylei*, and from saliva of *P. lylei* in Thailand (Wacharapluesadee, 2005). Among *Pteropus* species in South and Southeast Asia, *Pteropus giganteus*, widely distributed across India and Bangladesh, is considered the principal species responsible for *NiV* transmission in these countries (Sharma, 2020). *NiV* RNA has been detected in liver homogenates of *P. giganteus* in West Bengal, India (Yadav, 2012). Similarly, antibodies to *NiV* have been reported in bats in India (Epstein, 2008), Indonesia (Sendow, 2013), Madagascar (Lehlé, 2007), China (Li, 2008), and Vietnam (Hasebe, 2012), and *NiV* RNA was detected in *Eidolon helvum* in Ghana (Drexler, 2009).

Bats are naturally resistant to *NiV* infection and do not become ill after experimental infection with the virus, indicating possible co-evolution of *NiV* with its hosts (currently reservoir species) over many centuries. They are asymptomatic carriers but have the potential for continuous virus shedding (at certain intervals) through their secretions and excretions. Researchers also point to the fact that their travel over certain distances facilitates virus transmission not only to animals of their own species but also ensures interspecies transmission. (Middleton, 2007; Chong, 2009; FAO, 2011; Middleton, 2012).

The transmission of *NiV* from bats to other hosts occurs according to a specific pattern. Bats excrete the virus in their saliva and urine, which contaminates food and water sources, becoming vectors for the virus and creating opportunities for transmission to other animals. During the Malaysian outbreak in 1998, it was concluded that fruit bats eat fruit on plants, partially eating the fruit (not completely), leaving fruit residues contaminated with saliva (which contains the virus), and when they fall from trees, they are consumed by pigs, which leads to the infection of the latter. The disease spreads rapidly among pigs, as urine, saliva, and secretions from the throat and respiratory tract infect other pigs.

Clinical cases in humans and/or domestic animals have only been reported in Malaysia, Bangladesh, India, and the Philippines, as well as a few cases in slaughterhouse workers in Singapore who had been in contact with pigs imported from Malaysia. No evidence of endemic viruses has been found in Singapore. Most cases on the Indian subcontinent are confined to certain regions, but infected bats are also found outside these areas, and human cases are reported from time to time.

The Nipah virus has also been detected in bats in Cambodia, Thailand, and East Timor, and it is possible that this virus is endemic throughout most of Southeast Asia. Observations in Cambodia and Thailand have found no evidence of human infection, although this may be due to fruit and palm juice harvesting practices that reduce the opportunity for exposure to bats. Seropositive bats, other animals, and/or humans have been found in some other Asian countries (e.g., China) and on other continents (Africa, South America); However, viral and serological data suggest that at least some of these antibodies may be caused by contact with other *henipaviruses*. (CFSPH, 2025).

During the analysis of outbreaks in Malaysia, it was proven that human infection from pigs occurs through the respiratory tract (CDC, 1999; Chua, 2000). Researchers also pointed out that when there were no infected pigs, dogs were not a secondary reservoir of NiV and were not considered a source of infection (Mills, 2009). In Bangladesh, humans were infected without the involvement of pigs. People climbed trees and either had direct contact with bats or collected virus-contaminated fruit (Hsu, 2004; Montgomery, 2008). In most of Bangladesh and the neighboring state of West Bengal (India), people collect fresh palm sap. Scratches on the trunk can collect 1–2 liters of juice overnight in a tied clay pot. The pots are mostly open and easily accessible to fruit bats, so the juice can be contaminated with their saliva and excrement, which contain the virus. When consuming such juice, humans become infected with the Nipah virus (Luby, 2006; Yadav, 2012). It is not yet fully understood how widely NiV circulates in bat populations; however, NiV, its RNA, or antibodies to the virus have been detected in many countries where no clinical cases in humans or animals have been reported.

In bats of the genus *Pteropus*, the Nipah virus has been detected in urine, oropharyngeal swabs, rectal swabs, and birth fluids, although detection of the virus in urine is most consistent. Domestic animals are likely to become infected with this virus from bats by consuming fruit or water contaminated with their secretions and excretions, or by eating aborted bat fetuses and birth products. They can also become infected by eating the tissues of other infected mammals, which is suspected to be an important route of infection for dogs and cats, as well as through intranasal inoculation in laboratory experiments (Sendow, 2013).

During the outbreak in Malaysia, the Nipah virus appeared to spread among pigs on a farm through aerosols and direct contact, while transmission between farms was usually associated with the movement of pigs. The reuse of needles during vaccination (iatrogenic route) may also have contributed to its transmission. Pigs are known to shed the virus in respiratory secretions, and are suspected to shed it in urine. Viral RNA has been detected in feces, although the significance of this finding is still unclear, and transmission may be possible via semen. Virus shedding in respiratory secretions and urine has also been demonstrated in some incidental hosts, including experimentally infected cats and ferrets, but whether any species other than pigs readily spreads Nipah virus is currently unknown. Serological studies in Malaysia showed that the pathogen did not spread horizontally among dogs during this epidemic, and studies conducted on ferrets kept in the same room found that there was no transmission of the virus from animal to animal, possibly because the ferrets refused social contact after becoming clinically ill (Middleton, 2012).

Intrauterine transmission of the virus has been demonstrated in experimentally infected cats and suspected in naturally infected pigs.

Humans can become infected through direct contact with infected pigs, probably through the mucous membranes, but possibly also through skin scratches. Infection also occurs through the consumption of contaminated date palm juice, undercooked horse meat, or other food products. Like other species, humans can shed the Nipah virus in respiratory secretions, saliva, and urine, and viral RNA has been detected in semen for several weeks. People who have close contact with a patient or his/her biological fluids sometimes become infected; however, human-to-human transmission is inconsistent, occurring during some outbreaks but not during others. Some data suggest that this is most likely to occur in the terminal stages of the disease and is particularly common in cases with respiratory symptoms.

Nosocomial transmission has been documented in hospitals where infection control measures are inadequate and/or untrained family members perform certain caregiving tasks.

The Nipah virus can remain viable in the environment for several minutes to several days, surviving longer at neutral pH than at acidic pH. It has been reported to persist for up to 3–7 days or longer in some fruits or fruit juices, including artificial date palm juice (13% sucrose and 0.21%

BSA in water) at room temperature (22°C/72°F). Date palm juice may be a particularly favorable environment for this virus because it is harvested during the coldest months of the year, has a near-neutral pH, and is typically consumed within a few hours before fermentation. Although the Nipah virus survived for less than 30 minutes in normal *P. vampyrus* urine (pH 2) at 22°C, it persisted for more than 4 days in the same urine with the pH adjusted to neutral. It remained viable for at least a week at 20 °C (68 °F) in sealed tubes containing small amounts of blood or culture medium, but at this time the virus was not detected in unsealed tubes stored at 20 ° or 30 °C (86 °F), or in closed tubes at 30 °C. It degrades fairly rapidly on surfaces, remaining viable on non-porous polystyrene (a material expected to maximize virus survival under drying conditions) for < 15 minutes at 37°C (99°F) in the laboratory and decreasing in titer by more than two orders of magnitude in 1 hour at 22°C (CFSPH, 2025).

Domestic animals become infected through contact with infected pigs. Experimental infections have also been established in cats after nasal and oral infection. Horizontal transmission has not been demonstrated between cats, but it is theoretically possible. Vertical transmission of this virus in cats has been demonstrated in experimental conditions (Mungall, 2007). Data from experimental studies in dogs have not been published, but serological monitoring conducted in Malaysia suggests the possibility of virus spread in dogs (Middlton, 2002).

Overall, Nipah virus infections appear to be relatively common among flying fox populations in endemic regions, even where human cases have not been reported. Although seroprevalence may be high, only a few bats in a colony may shed the virus at any given time, and shedding from a colony may be sporadic. Several studies have noted periodic peaks in virus shedding that can last for weeks, although the reasons for these peaks are not fully understood and may be multifactorial. Only one outbreak has been reported in pigs. It occurred in 1996–1999 in Malaysia and affected approximately 6% of all pig farms, with up to 90% or more of animals affected by the virus on some farms. The incidence of Nipah virus in pigs is estimated to be approximately 70–100%, but the mortality rate is low (e.g., 1–5% in animals aged 1–6 months), except for young piglets. In the latter age group in Malaysia, the mortality rate was approximately 40%, but neglect of sick sows that remained indoors may also have played a role (Yadav, 2012).

Information on other hosts is limited; however, studies in laboratory animals (e.g., hamsters) indicate that some individuals of the same species may die from the disease, while others survive even after exposure to high doses of the virus. All natural clinical cases in dogs and cats to date have been described during outbreaks in pigs or horses. However, only two cases of the disease in dogs in Malaysia have been confirmed (Nipah virus isolated). It has also been reported that a significant number of dogs died on infected farms, and farmers also reported illness in cats and goats. Serological studies subsequently found seroprevalence rates of 15–55% in dogs, 4–6% in cats, and 1.5% in goats in the outbreak area. Four cats and one dog also died shortly after eating the tissues of sick horses during an outbreak in the Philippines, while antibodies to the Nipah virus were detected in dogs living nearby but not in cats. Although the horses were thought to be the source of the virus for humans, tissue from the latter was not available for confirmation, and relatively little has been published about the outbreak. Nipah virus infections in horses appeared to be rare during outbreaks in pigs in Malaysia: only five horses out of more than 3,200 tested positive in serological tests, and viral antigens were detected in one horse that died with signs of meningitis (Hasebe, 2012).

Several studies have investigated the possibility of direct transmission from bats to incidental hosts, with one study finding that no wild cats living near an infected bat colony on Tioman Island, Malaysia, in 2004 had antibodies to the Nipah virus. There are no reports of confirmed cases in domestic animals from Bangladesh or India, and no unusual outbreaks of disease in animals have been reported during human outbreaks in this region. However, the possibility of undiagnosed cases cannot be ruled out, and one study in Bangladesh found antibodies to Nipah virus or other henipaviruses in 1% of cattle, 3% of dogs, and 5% of cats in villages where human cases had been observed (CFSPH, 2025).

The Nipah virus is zoonotic. Most clinical cases in India and Bangladesh appear to be caused by the consumption of raw date palm juice contaminated by bats, or are transmitted from person to person. Consumption of fermented date palm juice (containing approximately 4% alcohol) has been identified as a risk factor in several cases, while in some cases no obvious source of infection has been identified. An outbreak in Malaysia was caused by contact with

infected pigs, and an outbreak in the Philippines was linked to the consumption of undercooked meat from sick horses or participation in their slaughter (CFSPH, 2025).

People become infected through direct contact with infected pigs. Infection can occur through mucous membranes and skin. Researchers have confirmed that drinking unpasteurized date palm juice contaminated with the virus is a factor in transmission. However, after the disease develops, an infected person can transmit the virus to others through direct and indirect contact (Gurley, 2007). In sick people, NiV is found in significant amounts in saliva, urine, and respiratory secretions (Chua, 2000; Harcourt, 2005). There have been reports of people getting infected with this virus after coming into contact with the bodies of people who died from *Nipah* (Sazzad, 2013).

The Nipah virus has repeatedly appeared in human populations in Southeast Asia, where more than 700 cases had been reported as of 2024. The first known cases were reported in Malaysia (and among slaughterhouse workers in Singapore) in 1998–1999, although retrospective analysis shows that human infections also occurred in 1997. Approximately 300 cases of encephalitis were reported during these outbreaks. Most people became infected through contact with pigs, and after that, seropositive animals were culled. However, since 2001, sporadic cases and outbreaks have been reported for many years from Bangladesh, occasionally from India, while an outbreak linked to sick horses was observed in the Philippines in 2014. Some cases in endemic regions probably remain undiagnosed due to their similarity to other diseases and/or the limited use of diagnostic tests for this virus.

Outbreaks in Bangladesh are seasonal and occur mainly between December and May, which is the harvest period for date palm sap. Human-to-human transmission through close contact has also been observed in Bangladesh and India, often in family clusters, and nosocomial outbreaks have occurred in hospitals where barrier precautions were inadequate as patients were placed in shared wards and/or inadequately trained family members acted as caregivers. Transmission varied between outbreaks, with some individuals infecting a significant number of their contacts, but others did not play such a role and no visible disease occurred even among “high-risk” contacts who had direct, unprotected contact with such individuals or their body fluids (Pernet, 2012).

Overall, it is estimated that less than 10% of patients transmit the virus to others, with most transmission believed to occur in the late stages of the disease, particularly from those with respiratory symptoms.

The case fatality rate during major outbreaks has ranged from 38% in Malaysia to approximately 70–75% in some cases in India and Bangladesh, with a case fatality rate of 8% in Singapore. Mortality rates of up to 90–100% have been documented, but these usually occurred in clusters where only a few people were affected. One such outbreak occurred in India, where a person with an unrecognized severe case infected a number of other patients through close contact in a hospital, as well as family members who cared for her or transported her between medical facilities. The case fatality rate appears to be higher in Bangladesh and India than in Malaysia, but it is unclear how much this is related to strain variability, differences in healthcare, or limited recognition of milder cases. In Malaysia, late onset or recurrent encephalitis was observed in <5% and <10% of patients, respectively, with an overall mortality rate of 18% (Daszak, 2013).

It is still unclear how many people are infected asymptotically or develop only mild symptoms, but the subclinical infection rate was estimated at 8–15% during the outbreak in Malaysia and 5% in Singapore. Approximately 11% of the population of Malaysia living in rural or semi-urban areas, who may have contact with infected bats, were found to have antibodies to the Nipah virus, although no clinical cases have been reported in this region. Studies of two outbreaks showed that antibodies were rare (1%) or absent among asymptomatic contacts of patients in Bangladesh and India (CFSPH, 2025).

In January 2026, an outbreak of Nipah virus was reported in the Indian state of West Bengal. Among those affected were medical personnel who had contact with the first patient. Nearly 100 people have been instructed to remain in home quarantine, and infected patients are being treated in hospitals in Kolkata and its surroundings, with one patient in critical condition. Due to the threat of its spread, a number of Asian countries have tightened controls on passengers arriving from India.

Pathogenesis. The virus uses leukocytes as a means of spreading throughout the body. Virus replication also occurs in dendritic cells. The cellular tropism of NiV correlates well with the

expression pattern of *Ephrin B2*. It acts as a receptor for the entry of *NiV G-glycoprotein* into endothelial cells, neurons, and smooth muscle cells surrounding small arteries and arterioles. After primary replication of the virus in these cells, viremia occurs, leading to systemic spread of the virus, thrombosis, vascular occlusion, and ischemia, which leads to severe damage to the central nervous system. Proinflammatory cytokines such as *TNF- α* and *IL-1 β* increase the permeability of the blood-brain barrier and contribute to the induction of neuronal damage, thereby disrupting the blood-brain barrier, after which neurological signs become apparent. The vasculitis that occurs in this disease indicates that the target cells for the virus are blood vessels. The central nervous system is severely affected, although the lungs, kidneys, and other organs are also infected (Wong, 2002; Negrete, 2006; Mathieu, 2011; Pernet, 2012).

Pathological studies reveal hemorrhagic or necrotic alveolitis, pulmonary edema, and aspiration pneumonia. Intra-alveolar inflammatory cells, sometimes multinucleated giant cells, are also found in the affected alveolar tissues (Lo, 2010).

Clinical signs and course in animals and humans. The incubation period in humans after *NiV* infection varies from 2 to 30 days (most cases manifest in less than two weeks). Late encephalitis develops months or years after the initial mild or subclinical infection, with one case reportedly occurring 11 years later.

In humans, mortality was around 40% during the Malaysian outbreaks and 70% in India and Bangladesh. A small proportion of victims may have neurological dysfunction for several months to several years.

Although some cases of Nipah virus infection in humans may be asymptomatic or mild, most reported cases have been severe. Initial symptoms are usually flu-like, with fever, headache, sore throat, myalgia, and in some cases nausea, vomiting, and/or nonproductive cough. Acute symptoms also included fever and muscle pain, inflammation of the brain leading to disorientation or coma. This prodromal syndrome is sometimes followed by either encephalitis or respiratory syndrome, which may include atypical pneumonia or acute respiratory distress; or respiratory and neurological signs. Meningitis, as well as encephalitis, have been reported in the Philippines. Septicemia, signs of vasculitis, bleeding tendency (e.g., *melena*, *hemoptysis*), renal failure, myocarditis, and other complications are possible in severely ill patients. Acute encephalitis was accompanied by drowsiness, depression, and coma. Patients with encephalitis who recovered may have had mild or severe residual neurological disorders. In several cases, recovery from encephalitis was followed by recurrent lesions several months later.

Recurrent encephalitis or late-onset encephalitis was observed in several cases and occurred several months or years after asymptomatic infection or neurological disease. Clinical signs usually developed acutely, with symptoms that could include headache, seizures, and focal neurological signs; however, fever was less common than in encephalitis occurring during the initial stage of the disease (Goh, 2000; Chua, 2001; Wong, 2002; Chadha, 2006; CFSPH, 2025).

The incubation period in pigs is 7–14 days in most cases, although in some individuals it can be as short as 4 days. In experimentally infected cats, clinical signs developed after 6–8 days, and in experimentally infected ferrets, after 6–10 days (CFSPH, 2025).

Although many pigs become subclinically infected, some individuals develop acute febrile diseases. Most cases are self-limiting, except in young piglets; however, fulminant infections and sudden deaths are sometimes observed. Typical signs in piglets are open-mouth breathing, twitching, weakness of the legs with muscle tremors, and a significant number of affected piglets die. Death from starvation may contribute to mortality if the sow is also ill. Young pigs aged 1–6 months sometimes develop respiratory signs, which may be characterized by fever, nasal discharge, open-mouth breathing, rapid and labored breathing, and a loud barking cough. In severe cases, hemoptysis may be observed. Various neurological symptoms may also be observed, such as tremors, twitching, muscle spasms, myoclonus, weakness in the hind legs, spastic paresis, lameness, uncoordinated gait when the pig is moving or rushing, and general pain, which is particularly noticeable in the rear of the body. One experiment showed that secondary bacterial meningitis may be a contributing factor in some cases, especially when neurological symptoms develop in the later stages of the disease. Similar lesions were sometimes found in sows and boars, and some sows aborted during outbreaks of the Nipah virus, usually in the first trimester of pregnancy. Overall mortality is low, except in young piglets (CFSPH, 2025).

In general, clinical signs among pigs during Malaysian outbreaks were mainly characterized by respiratory and neurological (encephalitic) syndromes. The respiratory

syndrome was also called “barking pig syndrome.” The incidence among pigs was approximately 80%, and mortality was less than 5%. It should be noted that mortality among piglets was higher than among adult animals. The incubation period lasted about 1–2 weeks. The clinical manifestation of the disease in pigs also depended significantly on the involvement of the central nervous or respiratory systems and the age of the pigs. Pigs developed febrile respiratory lesions with a loud cough. Neurological lesions prevailed in adult animals, while respiratory lesions prevailed in piglets. In the respiratory form in pigs, an acute febrile reaction is observed, including fever, labored breathing, dry cough, and in severe cases, bloody sputum. In the neurological form, fever, whole-body tremors, muscle twitching and spasms, impaired coordination of movements, weakness of the hind limbs in the early stages with subsequent spastic or flaccid paresis were also recorded. In some individuals, nervous and respiratory symptoms were recorded simultaneously. In adult animals with respiratory forms of the disease, increased salivation, lacrimation, and nasal discharge were observed. Nervous forms include nystagmus, trismus, tetanus-like spasms, and convulsions. Pregnant sows mostly aborted (Mohd, 2000; Chua, 2003; Wong, 2011).

Horses believed to have been infected with the Nipah virus in the Philippines developed acute, fatal neurological symptoms, or some of them died suddenly without obvious prior signs of illness.

Non-productive cough, poor growth, severe respiratory symptoms, and death were reported in naturally infected goats in Malaysia. Goats infected with Nipah virus in Bangladesh had febrile neurological syndrome (but it was not confirmed that it was this particular virus).

There are not many descriptions of clinical cases in naturally infected dogs or cats in the specialized literature, as most animals of these species were found dead or dying. One dog had clinical signs of carnivore plague, with fever, respiratory distress, and mucous discharge from the nose and conjunctiva. One cat, in agonal state, was described as having terminal bleeding from the nose and mouth. Experimentally infected cats had severe respiratory symptoms and depression, while experimentally infected ferrets developed both respiratory and neurological symptoms, including tremors and hind limb paresis, before death.

Experimentally infected African green monkeys (*Chlorocebus aethiops*) and common (white-faced) squirrel monkeys (*Saimiri sciureus*) may also be severely affected, but crab-eating macaques (long-tailed macaques, *Javanese macaques*) (*Macaca fascicularis*) either remained asymptomatic or had a mild, self-limiting respiratory disease, depending on the viral strain.

Infections in fruit bats, natural carriers of the Nipah virus, are mostly asymptomatic (CFSPH, 2025).

After experimental infection of guinea pigs, hamsters, ferrets, and non-human primates, and their subsequent death, autopsy revealed significant vascular and parenchymal damage in the central nervous system and other organs, such as the liver, lungs, kidneys, muscles, and lymphoid organs (Korniienko, 2020).

Pathological and anatomical changes. Massive lesions in pigs can be found either in the lungs or in the brain. In pigs, varying degrees of hemorrhage (petechiae, ecchymosis) are found in the lungs. Lung lesions, ranging from mild to severe, may include varying degrees of consolidation, petechial or ecchymotic hemorrhages, and emphysema with frothy, sometimes bloody, fluid in the bronchi and trachea of some animals. Congestion of the cerebral blood vessels and meningeal edema may be observed in the brain. Lymphadenopathy may also be noted, and although the kidneys are often normal, they may sometimes be congested, with petechiae in the renal capsule and cortical substance.

During the autopsy of dogs that died from the infection, exudate was found in the trachea and bronchi, vasculitis in the lungs, and glomerular and tubular necrosis with syncytium formation with severe hemorrhaging in the kidneys, non-purulent meningitis, signs of degeneration of the cerebral and hepatic vessels, as well as necrosis and inflammation of the adrenal glands (Mohd et al., 2000).

The main macroscopic lesions in experimentally infected cats were hydrothorax, consolidation and edema in the lungs, edema of the pulmonary lymph nodes, and foam in the bronchi, while histopathology also revealed meningitis in some cases. More subtle lesions in the early stages of the disease included numerous small hemorrhagic nodules in the lungs, scattered hemorrhagic nodules on the visceral pleura, and, in one animal, edema of the serous membrane of the bladder with dilation of the serous lymphatic vessels. Generalized vasculitis was observed

in a naturally infected cat in Malaysia, particularly in the brain, kidneys, liver, and, to a lesser extent, in the lungs (Middleton, 2002; CFSPH, 2025).

Non-purulent meningitis was reported in an infected horse in Malaysia (CFSPH, 2025).

Histological examination of the lungs reveals interstitial pneumonia with hemorrhages and syncytial formations in the endothelium of blood vessels. Generalized vasculitis with fibrinoid necrosis, hemorrhages, mononuclear cell infiltration, and thrombosis are found in the kidneys and brain. In some cases, meningeal inflammatory infiltrates on the meninges may also be noted. The demonstration of high concentrations of NiV antigens in the endothelium of blood vessels and in the lungs can be taken as evidence of the ability of such an infected pig to shed significant amounts of virus through the respiratory tract (Chua, 2000; Wong, 2003).

Diagnostics. Nipah virus infections can be diagnosed by isolating the virus, detecting nucleic acids or antigens, and serological testing. Most laboratories use “in-house” tests with varying levels of validation, but several commercial PCR tests may also be available.

In pigs, the Nipah virus was detected in respiratory secretions and blood, as well as in various tissues during autopsy, including the lungs, spleen, bronchial and submandibular lymph nodes, kidneys, and brain. It was detected in similar tissues of dogs and experimentally infected cats, particularly in the lungs and spleen, as well as in the urine, blood, and respiratory secretions of cats. Thus, samples of cerebrospinal fluid, brain tissue, and lungs are taken from carcasses for examination. Swabs from the larynx, nasal and oral secretions, blood, and urine are taken from sick animals (CFSPH, 2025).

The laboratory conducts laboratory tests using molecular genetic methods – PCR (*RT-PCR*) and its modifications. Direct immunofluorescence (*DIF*) and enzyme-linked immunosorbent assay (*ELISA*) tests have also been developed for the diagnosis of the disease (*OIE*, 2010). It has been shown that *IgM ELISA* is better to use in the early stages of infection, and *IgG ELISA* in the later stages (Kulkarni, 2013).

Polymerase chain reaction (*RT-PCR*) is often used to diagnose clinical cases, and viral antigens can be detected in tissues during necropsy using immunoperoxidase or immunofluorescence analysis. Additional tests for the detection of antigens (e.g., *rapid lateral flow, ELISA*) or nucleic acids (loop-mediated isothermal amplification, recombinase polymerase amplification) are described in the specialized literature.

The virus can be isolated in cell culture. It is often isolated in Vero cells, but many other cell lines or chicken embryos can also be used. After NiV isolation, a virus neutralization test is performed. It should be noted that working with this pathogen requires BSL-4 biosafety. Serology can also be useful, especially in pigs, which are often subclinically infected. However, reports of low titers in some experimentally infected pigs may raise concerns about the sensitivity of some tests, especially when the virus strain used in the test does not match the strain in the infected animal. Serological tests used in animals include both virus neutralization and *ELISA*; however, virus neutralization has limited availability because it requires high biosafety laboratories (Kulkarni, 2013).

Histopathology also takes part in diagnostics.

If an unknown pathogen has been handled in a BSL-3 facility and Nipah virus has been detected, all samples are immediately transferred to a BSL-4 laboratory using the appropriate transport protocol for hazardous pathogens (Kulkarni, 2013; CFSPH, 2025).

Nipah virus infections in humans can be diagnosed by *RT-PCR*, antigen detection in tissues, virus isolation, and serology, as in animals. Serological tests available for humans include *ELISA* for *IgM* or *IgG* and serum neutralization, with clinical cases diagnosed by detection of *IgM* or serum titer elevation and/or detection of antibodies in cerebrospinal fluid (*CSF*). The virus is most likely to be detected in clinical samples early in the course of the disease. In humans, it has often been detected in blood, throat or nose swabs, *CSF*, and urine, as well as in various postmortem tissues. Virus isolation from *CSF* is a poor prognostic sign (CFSPH, 2025).

Differential diagnosis. The disease has not been reported in pigs since 1999, but the presence of the virus in bat populations requires veterinarians to keep them informed of clinical and diagnostic studies on NiV and its differentiation from swine influenza, porcine reproductive and respiratory syndrome (PRRS), enzootic pneumonia of pigs, and *Aujeszky's* disease. In humans, Japanese encephalitis, other arboviral diseases, and bacterial meningitis are also ruled out (Korniienko, 2020). The Nipah virus can be distinguished from the Hendra virus or other henipaviruses using genetic methods (e.g., *real-time PCR*), comparative immunostaining, or

differential neutralization analysis; However, virus neutralization or immunostaining alone is insufficient due to cross-reactivity between these viruses (CFSPH, 2025).

Treatment. No specific treatment has been developed for animals. Although most infected pigs recover, infected animals are usually slaughtered to prevent transmission of the virus to humans who care for them (CFSPH, 2025). There are currently no approved or licensed therapeutic agents or effective treatments for NiV infection (Broder, 2012a and b). Treatment in humans is mostly based on supportive care, but antiviral drugs (e.g., ribavirin, acyclovir, favipiravir) or monoclonal antibodies have been tried in a limited number of human cases or tested in animals. In animal models, these interventions appear to be most effective when started early (CFSPH, 2025).

Prevention and control. Good biosecurity is important for preventing infections on pig farms; strategies should target routes of contact with other pigs, as well as with fruit bats. To avoid attracting bat colonies, fruit tree orchards should be removed from areas where pigs are kept. Wire mesh can reduce bat contact when pigs are raised in open pens. Roof drainage into pens should also be prevented. Fruit or date palm juice that may have been contaminated by bats should not be fed to pigs or other animals. Early detection of infected pigs can help protect other animals and humans. Due to the high contagiousness of the virus in pigs, mass culling of seropositive pig populations may be necessary. Most farms in Malaysia appear to have been infected with the virus through imported pigs, and quarantine is expected to prevent transmission of the virus between farms during an outbreak. Objects and equipment should be cleaned and disinfected, and other animals, including dogs and cats, should be protected from contact with infected pigs or movement between farms. There are currently no vaccines available for any animal species (CFSPH, 2025). No vaccines have been developed to prevent the disease in humans either. Recombinant vaccines for the prevention of the disease in pigs have been studied in experimental conditions (Weingartl et al., 2006). A subunit vaccine made from a recombinant soluble and oligomeric form of Hendra virus *G-glycoprotein* (*HeV-sG*) provides protection against Hendra virus (*HeV*) and Nipah virus (*NiV*).

NiV infections can be prevented by preventing contact (direct or indirect) between pigs and bats in endemic areas and by avoiding the consumption of raw palm juice. Active educational work should be organized in these areas, primarily among medical and veterinary workers and the general public.

It is now known that populations of fruit bats, which are reservoir carriers of this virus, exist in several countries. Their range extends from western Africa to South Asia, Southeast Asia, and East Asia. The constant mixing of *NiV*-carrying bat populations with intact ones leads to the spread of the virus in most countries. Climate change, such as global warming, floods, fires, etc., can force bats to migrate and indirectly expand the circulation of the virus and the risk of side effects, i.e., cause a change in geographical distribution as a direct effect. After all, a reduction in local food resources or extreme weather conditions expose them to physiological stress, leading to immunosuppression and prolonged release of the virus into the external environment. Food shortages may prompt these bats to consume crops grown by humans, thereby increasing the risk of contact between animals and humans (Daszak, 2013). In cases such as immediate response to disease outbreaks, local residents and sometimes even government officials may contribute to a general sentiment of eliminating fruit bats. However, it should be remembered that the pollination of many plants depends partly or entirely on bats pollinating their flowers or spreading their seeds, while other bats also help in biological pest control by consuming insects. Humans must take care to protect food and water sources that may be contaminated by bats. The practice of using palm juice requires strict control (Korniienko, 2020).

Pigs were important carriers of the Nipah virus during outbreaks in Malaysia, and preventing infections in this species may reduce the risk of infection for humans. Sick animals of any species should not be used for food, even if the meat is cooked at high temperatures, as the slaughter process can cause viruses to enter the tissue.

Close contact with fruit bats, their secretions, and excrement should also be avoided. Consumption of raw date palm juice appears to be the most common source of infection in regions where bats sometimes contaminate juice collection pots with urine and saliva. The risk can be significantly reduced, though not completely eliminated, by using protective covers (such as bamboo juice skirts) to keep bats away from juice collection sites. In addition, some types of juice collection containers, such as those used in parts of Cambodia, are less accessible to bats than

the large pots used in Bangladesh. Fruit should be thoroughly washed, peeled, or cooked before consumption. In addition, netting can be used to keep bats away from ripe fruit, which is more likely to attract bats. People who handle infected animals or potentially infected transmission factors (fomites) should practice good hygiene (e.g., washing hands) and wear currently recommended PPE to prevent contact of mucous membranes or broken skin with animal secretions and excretions or viruses that may be transmitted by airborne droplets (FAO, 2011).

Since Nipah virus can be transmitted from person to person, patients should be isolated and barrier nursing measures should be used. Although the results of the study are not conclusive (human-to-human transmission does not always result in infection, even among high-risk contacts), one study reported that 8 healthcare workers who had close, unprotected contact with a patient infected with Nipah virus and who were given a high prophylactic dose of ribavirin within 72 hours did not become infected.

Nipah virus is classified as a risk group 4/BSL pathogen, and only laboratories with secure facilities for hazardous pathogens can work with this virus (CFSPH, 2025).

Conclusions. An analysis of current foreign scientific literature has yielded the following results: the main reservoir of the Nipah virus is the fruit bat, and the pathogen has the ability to infect pigs, humans, cattle, goats, cats, dogs, and horses. The pathogen is mainly prevalent in Southeast Asia and India. The main foci include Bangladesh and India (in particular, the states of Kerala and West Bengal), where regular outbreaks occur. Cases have also been reported in Malaysia, Singapore, and the Philippines. In pigs, the disease should be differentiated from swine flu, porcine reproductive and respiratory syndrome (PRRS), enzootic pneumonia of pigs, and Aujeszky's disease. In humans, Japanese encephalitis, other arbovirus diseases, and bacterial meningitis should also be ruled out. Given the lack of specific treatment for animals and humans, it is important to implement a system of epidemiological surveillance during import operations involving susceptible animals.

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ХАРАКТЕРИСТИКА ЗБУДНИКА, ЕПІЗООТОЛОГІЧНІ ОСОБЛИВОСТІ, КЛІНІЧНІ ОЗНАКИ, ДІАГНОСТИКА ТА ПРОФІЛАКТИКА ХВОРОБИ НІПА (ОГЛЯДОВА СТАТТЯ)

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Резюме. Описано результати аналізу закордонних наукових літературних джерел щодо епізоотичної ситуації емерджентного зоонозного захворювання – хвороби Ніпа. Представлено вірусологічні, серологічні та молекулярно-генетичні властивості вірусу, який належить до родини *Paramyxoviridae* підродини *Paramyxovirinae* рід *Nipavirus*. Висвітлено будову геному, що складається із шести генів: *N*, *P*, *M*, *F*, *G* і *L*; це білки: нуклеопротеїн (*np*), фосфопротеїн (*php*), матричний білок (*mp*), злиття (*fp*), глікопротеїн (*gp*), велика РНК-полімераза (*rnp*). Розкрито здатність збудника добре розмножуватись на різних первинних і перещеплюваних лініях культур клітин ссавців. Дослідження з культурою Vero дають можливість отримати в першому пасажі ЦПД на 3–6 добу, після попередньої адаптації – через 24–48 год. Основними резервуарними господарями для вірусу Ніпа є фруктові кажани *P. vampyrus*, *P. hypomelanus*, *P. medius* (раніше *P. giganteus*) та *P. lylei*, тоді як *P. rolіoscephalus* заражались експериментально. Вірус має здатність інфікувати свиней, людей, велику рогату худобу, кіз, кішок, собак, коней. Природньо інфікуються переважно свині, саме від них збудник передається людям. Передача збудника хвороби від кажанів іншим господарям відбувається зараунок виділення вірусу зі слиною й сечею, останні забруднюють їжу і джерела води, які стають чинниками передачі вірусу і створюються можливості для передачі збудника іншим тваринам. Менше 10% пацієнтів передають вірус іншим людям, причому більшість

випадків передачі, як вважається, відбувається на пізніх стадіях захворювання, особливо від тих, хто має респіраторні ознаки. Рівень летальності під час значних спалахів коливався від 38% до приблизно 75%. Васкуліти які виникають за цього захворювання показують, що клітинами-мішенями для вірусу є судини, сильному ураженню піддається центральна нервова система, легені, нирки. Під час патогістологічних досліджень виявляють геморагічний або некротичний альвеоліт, легеневий набряк і аспіраційну пневмонію. Внутрішньоальвеолярні запальні клітини, іноді багатоядерні гігантські клітини також виявляються в уражених альвеолярних тканинах. Діагноз ставлять на основі епідеміологічних, клінічних, патолого-анатомічних та лабораторних методах досліджень. Із урахуванням світового досвіду обґрунтовано заходи боротьби, профілактики та контролю з цим захворюванням.

Ключові слова: хвороба Ніпа, *Paramyxoviridae*, епізоотична ситуація, фактори розповсюдження збудника, діагностика, заходи боротьби, профілактика та контроль.

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