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Andes Hantavirus Beyond Endemic Borders — Why Vigilance Matters More Than Panic: A Rapid Mini Review

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Abstract

Background: In May 2026, the World Health Organization (WHO) reported a multi-national cluster of Andes hantavirus (ANDV) infections linked to the cruise vessel M/V Hondius. The outbreak generated international concern because ANDV is associated with hantavirus pulmonary syndrome (HPS), a severe zoonotic disease with reported case-fatality rate (CFR) of 35-50%, despite limited person-to-person transmission. This rapid mini-review aimed to examine the virologic, epidemiologic, and public health significance of the ANDV outbreak while highlighting why evidence-based vigilance is more appropriate than disproportionate societal panic.

Methods: We conducted a rapid mini-review of the virologic, epidemiologic, phylogenetic, clinical, and public health dimensions of hantavirus infections with a focus on ANDV using literature identified through Google Scholar and publicly available reports from the WHO, the United States Centers for Disease Control and Prevention (CDC), and international public health agencies up to 14 May 2026.

Results: As of 13 May 2026, 11 ANDV outbreak-associated cases, including three deaths (CFR, 27%), had been reported. Eight cases were laboratory-confirmed for ANDV infection, two were classified as probable, and one remained under investigation. Current evidence indicates that ANDV transmission remains epidemiologically constrained by low secondary attack rates, prolonged close-contact requirements, limited asymptomatic spread, and absence of sustained community spread. The review identified several major public health implications, including the importance of early clinical recognition of HPS, implementation of infection-control measures, coordinated multi-national surveillance, rapid diagnostic preparedness, and timely risk communication capable of balancing vigilance with avoidance of disproportionate societal alarm.

Conclusions: The M/V Hondius ANDV outbreak highlights that pathogens capable of causing severe disease are not necessarily capable of sustaining efficient transmission. Although ANDV warrants international surveillance and preparedness because of its substantial clinical severity and limited person-to-person transmission, currently available evidence suggests that the broader public health risk remains relatively low. Nevertheless, ongoing epidemiologic monitoring and international coordination remain essential as additional data emerge. Effective ANDV outbreak management therefore depends upon coordinated public health response, transparent risk communication, and evidence-based vigilance while avoiding disproportionate societal alarm.

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Abbreviations

AIC: Akaike Information Criterion; aLRT-SH: Approximate likelihood-ratio test Shimodaira–Hasegawa; ANDV: Andes hantavirus; BIC: Bayesian Information Criterion; CDC: United States Centers for Disease Control and Prevention; CFR: Case-fatality rate; COVID-19: Coronavirus disease 2019; DOBV: Dobrava-Belgrade virus; ECDC: European Centre for Disease Prevention and Control; Gn and Gc: Hantavirus envelope glycoproteins; GTR: General Time Reversible model; HFRS: Hemorrhagic fever with renal syndrome; HNTV: Hantaan virus; HPS: Hantavirus pulmonary syndrome; IHR: International Health Regulations; L: Large gene segment; M: Medium gene segment; MAFFT: Multiple alignment program for amino acid or nucleotide sequences; MEGA: Molecular Evolutionary Genetics Analysis; ML: Maximum-likelihood; PUUV: Puumala virus; RdRp: RNA-dependent RNA polymerase; RNA: Ribonucleic Acid; RT-PCR: Reverse-transcriptase polymerase chain reaction; S: Small gene segment; SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2; SEOV: Seoul virus; SMS: Smart Model Selection; SNP: Single nucleotide polymorphism; SNV: Sin Nombre virus; WHO: World Health Organization

Introduction

Emerging infectious diseases often generate public fear and anxiety disproportionate to their actual epidemiologic potential [1-4]. Inevitably, the recent reporting of a lethal hantavirus zoonotic virus aboard an international cruise ship evoked comparisons with previous global outbreaks [5]. Such reactions are particularly understandable in the post-coronavirus disease 2019 (COVID-19) era, during which confined maritime settings became emblematic of rapid pathogen transmission and international dissemination [6-9]. However, the ability of a pathogen to cause severe disease in individual patients does not necessarily mean the capability for its sustained population-level transmission [10]. Evolutionary models of pathogen fitness, including the trade-off hypothesis, suggest that maximum transmissibility is often achieved at intermediate levels of virulence, balancing efficient host-to-host spread against host death [11, 12]. Consequently, the pandemic potential is not directly related to pathogen virulence, but is determined by a complex interplay among transmissibility, host adaptation, timing of infectiousness, asymptomatic spread, environmental stability, and the ability to sustain repeated chains of community transmission [13].

In May 2026, the World Health Organization (WHO) and the United States Centers for Disease Control and Prevention (CDC) reported a multi-national cluster of Andes hantavirus (ANDV) infections linked to individuals aboard the M/V Hondius cruise ship [14, 15]. As of 8 May 2026, WHO had reported eight cases (six laboratory-confirmed and two probable cases) of Andes hantavirus (ANDV) infection, including three deaths, corresponding to an observed case-fatality rate (CFR) of approximately 38% [14]. By 13 May 2026, the reported outbreak had expanded to 11 cases while the number of deaths remained unchanged at three, reducing the observed CFR to approximately 27% [16]. All confirmed cases were confirmed as ANDV infection through reverse-transcriptase polymerase chain reaction (RT-PCR) testing and/or viral sequencing [14, 16]. Several patients required international medical evacuation and intensive care management in South Africa, the Netherlands, Switzerland, and Germany, while large-scale contact tracing and symptom monitoring operations were initiated across multiple jurisdictions, including St Helena, South Africa, Switzerland, and other transit countries [14].

Hantaviruses are enveloped, segmented, negative-sense RNA viruses in the order Bunyavirales, family Hantaviridae, and genus Orthohantavirus carried by rodents, shrews, and bats [17-19]. The term hantavirus derives from the Hantan River, which runs between North and South Korea near Songnaeri, where the eminent researcher Ho Wang Lee established a field research station during investigations of Korean hemorrhagic fever in the late 1970s [20-23]. During the Korean War (1951-1954), more than 3000 soldiers developed a severe febrile illness characterized by hemorrhage, shock, and acute renal failure, with reported mortality approaching 10% [20-23]. Subsequent investigations led to the isolation of Hantaan virus (HNTV) from the lungs of the striped field mouse (Apodemus agrarius coreae), establishing the rodent reservoir basis of hantavirus transmission [23-25].

Hantaviruses cause two severe zoonotic syndromes worldwide; hantavirus pulmonary syndrome (HPS) in the Americas and hemorrhagic fever with renal syndrome (HFRS) in Europe and Asia [26-28]. HFRS occurs predominantly in Eurasia, whereas HPS remains largely confined to the Americas, reflecting the phylogeographic distribution of rodent reservoirs and the long-term coevolution of hantaviruses with their specific hosts [23, 26, 29].

Accordingly, distinct hantavirus species exhibit characteristic geographic distributions and organ tropisms [30, 31]. The Old World hantaviruses, which circulate predominantly in Europe and Asia, are classically associated with HFRS. In Asia, HNTV and Seoul virus (SEOV) predominantly target the kidneys and are major causes of HFRS [32-34]. In Europe, Dobrava-Belgrade virus (DOBV), Tula virus, and Puumala virus (PUUV) are associated with generally milder forms of HFRS, including nephropathia epidemica [35-38]. By contrast, the New World hantaviruses circulating in the Americas particularly, ANDV and Sin Nombre virus (SNV) primarily involve the pulmonary microvasculature, causing HPS, a severe respiratory syndrome associated with substantial mortality [39-41].

First characterization of ANDV was reported during a 1995 outbreak of HPS in the Andean region of Patagonia in Argentina, from which the virus derives its name [42]. Since then, the ANDV remained a major cause of HPS in Chile and Argentina because of its documented ability for limited person-to-person transmission [41, 43, 44]. Nevertheless, the epidemiologic evidence supporting sustained human transmission remains limited and methodologically controversial. A systematic review by Toledo et al., which evaluated 22 studies, concluded that — with the exception of a single prospective cohort study from Chile with a risk of bias — comparative epidemiologic evidence did not provide robust support for sustained human-to-human transmission of ANDV [45].

The HPS is characterized by rapidly progressive non-cardiogenic pulmonary edema resulting primarily from endothelial hyperpermeability and dysregulated host immune activation, with reported CFR ranging from approximately 35% to 50% [46, 47]. Despite this substantial mortality burden, epidemiologic investigations during previous ANDV outbreaks in Argentina and Chile demonstrated relatively inefficient secondary transmission

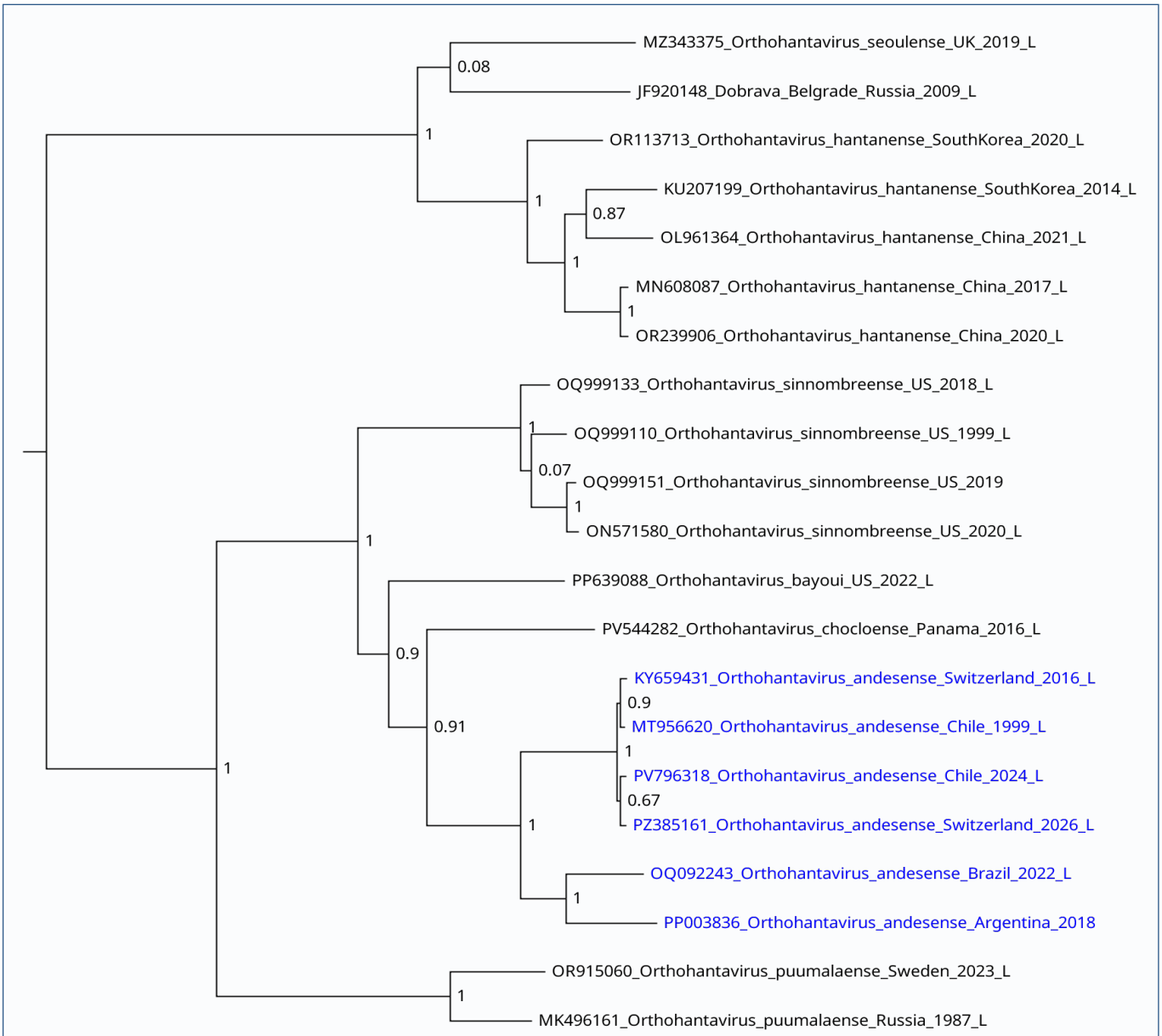

Figure 1: Maximum-likelihood phylogenetic analysis of Orthohantaviruses based on the near-complete 6320-base open reading frame of the L-segment RNA-dependent RNA polymerase (*RdRp*) gene from 21 human-derived GenBank viral sequences. Andes virus (ANDV) strains, highlighted in blue, clustered within the New World hantavirus lineage and remained genetically distinct from Old World Orthohantaviruses circulating in Europe and Asia. Branch support values are shown as approximate likelihood-ratio test Shimodaira–Hasegawa (aLRT-SH) statistics. The phylogenetic tree was reconstructed using PhyML version 3.0, midpoint-rooted, and visualized using FigTree version 1.4.4.

as opposed to the highly transmissible respiratory viruses such as influenza viruses or severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [48-50]. Thus, although human infection occurs primarily through inhalation of aerosolized rodent excreta, available evidence suggests that person-to-person transmission, when it occurs, is uncommon and generally requires prolonged close exposure to symptomatic individuals [51].

Importantly, both WHO and CDC assessed the broader public health risk associated with the M/V Hondius ANDV outbreak as low [14, 15]. This distinction is epidemiologically important. Contemporary outbreak management requires avoidance of two opposing failures; complacency, which may delay recognition and containment, and alarmism, which can amplify misinformation, erode public trust, and provoke disproportionate societal responses [52]. The M/V Hondius ANDV outbreak may therefore represent a relatively contained zoonotic event that also offers a broader lesson in risk interpretation during an era of heightened global pandemic sensitivity [9, 53, 54].

This rapid mini-review aimed to synthesize current evidence regarding the virologic characteristics, transmission dynamics, epidemiologic implications, and public health significance of the M/V Hondius ANDV outbreak. Particular emphasis was placed on understanding why pathogens capable of causing severe disease are not necessarily capable of sustaining global pandemics, and why evidence-based vigilance remains more appropriate than panic. Relevant literature and public health reports from Google Scholar

search, WHO, CDC, and related international surveillance sources available up to 14 May 2026 were reviewed to contextualize the evolving epidemiologic and public health dimensions of the ANDV outbreak [55].

Virology and Transmission Dynamics of Hantaviruses

Hantaviruses are enveloped, negative-sense single-stranded RNA viruses belonging to the family Hantaviridae [23, 51]. Their tripartite genome consists of small (S), medium (M), and large (L) RNA segments [56]. These segments encode the nucleocapsid protein, envelope glycoproteins (Gn and Gc), and viral RNA-dependent RNA polymerase (RdRp), respectively [57-59]. Distinct hantavirus species are maintained within specific rodent reservoirs through persistent asymptomatic infection, reflecting long-standing virus-host co-evolution and phylogeographic adaptation [29, 60]. Humans are incidental hosts and typically acquire infection through inhalation of aerosolized urine, saliva, or fecal particles shed by infected rodents, particularly in enclosed or poorly ventilated environments such as cabins, storage facilities, barns, garbage dumps, and military shelters [18, 30, 61, 62].

In this review, to characterize the genetic diversity of Orthohantaviruses infecting Homo sapiens, 21 complete L-segment RdRp gene sequences were retrieved from GenBank [63]. Multiple sequence alignment was performed using the Multiple alignment program for amino acid or nucleotide sequences (MAFFT) version 7 online platform [64], and subsequently inspected in Molecular Evolutionary Genetics Analysis (MEGA) software (version 6.0) to verify open reading frames and alignment quality [65]. Maximum-likelihood (ML) phylogenetic reconstruction was conducted using PhyML version 3.0 following model selection with Smart Model Selection (SMS) [66, 67]. Based on Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) optimization, the General Time Reversible model with among-site rate heterogeneity (GTR+R) was identified as the best-fit nucleotide substitution model for the dataset [68]. Branch support was assessed using approximate likelihood-ratio test Shimodaira–Hasegawa (aLRT-SH) statistics [69]. The final phylogenetic tree was visualized and annotated using FigTree version 1.4.4 [70] (Figure 1).

Phylogenetic and genomic analyses additionally provided important insights into the probable origin and transmission dynamics of the ANDV outbreak. Sequencing performed by the Swiss National Reference Center for Emerging Viral Infections and the Institute of Medical Virology at the University of Zurich demonstrated that the ANDV outbreak-associated sequences exhibited a very high degree of genetic similarity, differing by at most a single nucleotide polymorphism (SNP) among analyzed cases [71, 72]. This limited genomic diversity supports the hypothesis that the ANDV outbreak most likely originated from a single zoonotic spillover event, or from a very limited number of closely related spillover events, followed by subsequent limited human-to-human transmission [72].

Hantavirus infection causes two principal clinical syndromes with distinct geographic distributions. In the Americas, HPS is characterized by fever, thrombocytopenia, endothelial dysfunction, and rapidly progressive noncardiogenic pulmonary edema that may culminate in refractory hypoxemic respiratory failure and cardiogenic shock [26-28, 73]. Reported CRF range from approximately 35% to 50%, particularly among patients requiring intensive care support [46, 47, 73]. In contrast, HFRS, observed predominantly in Europe and Asia, is characterized by acute kidney injury, vascular instability, and hemorrhagic manifestations, with mortality varying substantially according to viral species, from less than 1% for PUUV to approximately 15% for HNTV [30, 32, 74, 75].

The cardinal pathophysiologic feature of severe hantavirus disease is diffuse capillary leak resulting from endothelial hyperpermeability rather than direct viral cytopathic destruction [76-78]. Experimental and clinical studies suggest that immune-mediated mechanisms play a central role, including excessive activation of cytotoxic T lymphocytes, dysregulated cytokine release, increased vascular endothelial growth factor signaling, and disruption of endothelial barrier integrity [79-82]. These processes contribute to plasma extravasation, pulmonary edema, hypotension, and multi-organ dysfunction [77, 78]. The severity of HPS therefore reflects an exaggerated host inflammatory response occurring within the pulmonary microvasculature [18].

Among hantaviruses, ANDV has a unique epidemiologic position because it has been reported as the only hantavirus associated with limited person-to-person transmission [41, 43, 44, 83]. First recognized during outbreaks in Argentina and Chile, secondary ANDV transmission has been documented primarily among household contacts, intimate partners, and caregivers following prolonged close exposure to symptomatic patients [41, 43, 44]. Nevertheless, such transmission remains relatively inefficient compared with highly transmissible respiratory pathogens [45, 49].

Thus, this transmission profile of ANDV reflects important biologic and epidemiologic constraints. Efficient pandemic pathogens typically exhibit several characteristics, including pre-symptomatic infectiousness, short serial intervals, efficient airborne dissemination, and a substantial proportion of asymptomatic or minimally symptomatic infections [84-87]. Available evidence suggests that ANDV fulfills few of these criteria since person-to-person transmission, when it occurs, is limited and generally requires prolonged close exposure to symptomatic individuals [45, 49]. Moreover, the rapid progression of severe HPS may paradoxically further reduce transmission potential by limiting patient mobility and social interaction during peak infectivity. Thus, although hantaviruses possess substantial pathogenic potential at the individual level, they remain primarily zoonotic viruses with limited capacity for sustained human adaptation and efficient population-level transmission [88].

The M/V Hondius ANDV Outbreak: A Test of International Preparedness

The ANDV outbreak linked to the cruise vessel M/V Hondius illustrates the operational challenges of containing zoonotic infections in an era characterized by rapid international mobility and highly interconnected travel networks. According to reports issued by the WHO and the CDC in May 2026, the vessel carried passengers and crew members from multiple countries, several of whom had already disembarked across different jurisdictions before recognition of the ANDV outbreak [14, 15]. This multi-national dispersion rapidly transformed a geographically localized outbreak into a complex transnational public health event requiring coordinated surveillance, diagnostic harmonization, international contact tracing, and real-time information exchange among national health authorities.

The epidemiologic significance of the ANDV outbreak derived from the severity of HPS [89]. Additional concern arose from the involvement of the ANDV which is the only hantavirus reported to

be associated with limited person-to-person transmission. Although available evidence suggested that the risk of sustained secondary transmission remained low [41, 43, 44], the prolonged incubation period of ANDV — reported to range from 7 to 39 days — raised concern for delayed case emergence and necessitated expanded international contact tracing and monitoring of exposed individuals [90].

The WHO subsequently activated coordinated response measures under the framework of the International Health Regulations (IHR) [14, 91]. These interventions included deployment of outbreak specialists aboard the vessel, comprehensive clinical assessment of passengers and crew, multi-national contact tracing operations, operational guidance for safe disembarkation and onward travel, and laboratory coordination across affected countries [14]. In addition, approximately 2500 diagnostic kits are prepared to be shipped from Argentina to reference laboratories in five countries to strengthen testing capacity and accelerate case identification [92]. The ANDV outbreak therefore represented an important real-world demonstration of the continuing relevance of the IHR framework for managing rapidly evolving cross-border infectious threats.

Simultaneously, the CDC issued a Health Alert Network advisory urging heightened clinician awareness for compatible febrile and respiratory illness among exposed travelers while emphasizing that broader spread within the United States was considered "extremely unlikely" [93]. This dual message — heightened vigilance without disproportionate alarm — reflected an evidence-based risk communication strategy grounded in the known transmission dynamics of ANDV.

Cruise ships occupy a distinctive position in the modern epidemiology of infectious diseases because they combine several conditions conducive to outbreak amplification. These conditions include confined environments, multi-national passenger movement, delayed recognition of illness, shared ventilation systems, and intense global media scrutiny [94-97]. Following the COVID-19 pandemic, maritime outbreaks — exemplified by the COVID-19 outbreak aboard the Diamond Princess cruise ship — acquired substantial symbolic significance within the public imagination, often amplifying societal anxiety beyond the actual epidemiologic characteristics of the pathogen involved [98-101]. However, confined settings alone do not determine pandemic potential. The decisive factor remains the intrinsic transmission efficiency of the pathogen itself. The M/V Hondius ANDV outbreak therefore provides a critical lesson in contemporary outbreak interpretation. A pathogen capable of causing severe disease and international concern is not necessarily capable of sustaining efficient global transmission [102].

Why High CFR Does Not Necessarily Predict Pandemic Potential

A misconception in both public discussion and media reporting is the assumption that pathogens associated with high CFRs inevitably possess substantial pandemic potential [103-105]. In reality, virulence and transmissibility are biologically distinct properties that may even evolve in opposing directions. The capacity of a pathogen to sustain global spread depends less on the severity of illness it causes than on the efficiency of transmission within largely susceptible populations [106-109].

Several highly lethal pathogens have historically shown limited pandemic capacity because their routes and timing of transmission impose strong epidemiologic constraints. Ebola virus disease is instructive; despite CFRs that may exceed 50% in some outbreaks, transmission occurs mainly through direct contact with infectious body fluids, often during advanced symptomatic illness or after death, rather than through efficient casual respiratory spread [110]. This biology permits explosive local amplification, especially in healthcare or funeral settings, but also makes outbreaks more amenable to interruption through early case recognition, isolation, contact tracing, clinical care, and community engagement [111].

Andes hantavirus appears to follow a similar epidemiologic pattern, although through different virologic and transmission mechanisms. Hantavirus pulmonary syndrome is clinically severe, but severity alone does not generate pandemic fitness. Human infection usually follows inhalation of aerosolized rodent excreta, and reported person-to-person transmission of ANDV appears uncommon, clustered, and dependent on prolonged close exposure, particularly among household members, intimate partners, or caregivers [43, 45]. Thus, the ANDV high CFR coexists with inefficient secondary transmission — a combination that warrants vigilance but argues against sustained pandemic spread.

By contrast, globally disseminated pandemic viruses frequently exhibit comparatively lower mortality but markedly greater transmission efficiency. For example, SARS-CoV-2 achieved sustained worldwide spread because it combined efficient respiratory transmission with pre-symptomatic infectiousness, environmental adaptability, and a substantial burden of asymptomatic or minimally symptomatic infection instead of high virulence [112-114]. Influenza viruses similarly exploit rapid replication, airborne dissemination, and frequent transmission during mild illness [115]. These characteristics enable repeated chains of community transmission before public health detection or intervention becomes possible.

Available data indicates that ANDV transmission occurs predominantly after symptom onset rather than during asymptomatic or presymptomatic phases, thereby limiting opportunities for unrecognized dissemination [40, 44, 45]. In addition, severe HPS frequently progresses rapidly to respiratory compromise and hospitalization, which may paradoxically reduce transmission potential by restricting mobility and social interaction during peak infectivity [116]. Epidemiologic investigations from previous ANDV outbreaks in Argentina and Chile have consistently demonstrated clustered transmission patterns, primarily among close contacts, without evidence of prolonged self-sustaining community spread [44, 48].

The relationship between virulence and transmissibility may therefore be partially paradoxical and called the trade-off hypothesis [11, 108, 109, 117]. Pathogens that rapidly incapacitate hosts may inadvertently restrict their own opportunities for dissemination, particularly when transmission requires close physical proximity [118]. This principle does not diminish the clinical severity of ANDV infection since HPS remains among the most lethal viral respiratory syndromes recognized in the Americas [46, 47, 119]. However, severity at the bedside does not necessarily translate into pandemic capability at the population level.

Importantly, WHO, the European Centre for Disease Prevention and Control (ECDC), CDC, and multiple national public health agencies have continued to classify the broader public health risk associated with the M/V Hondius ANDV outbreak as "low" or "very low" [14, 15, 92, 93, 120]. This assessment reflects not complacency,

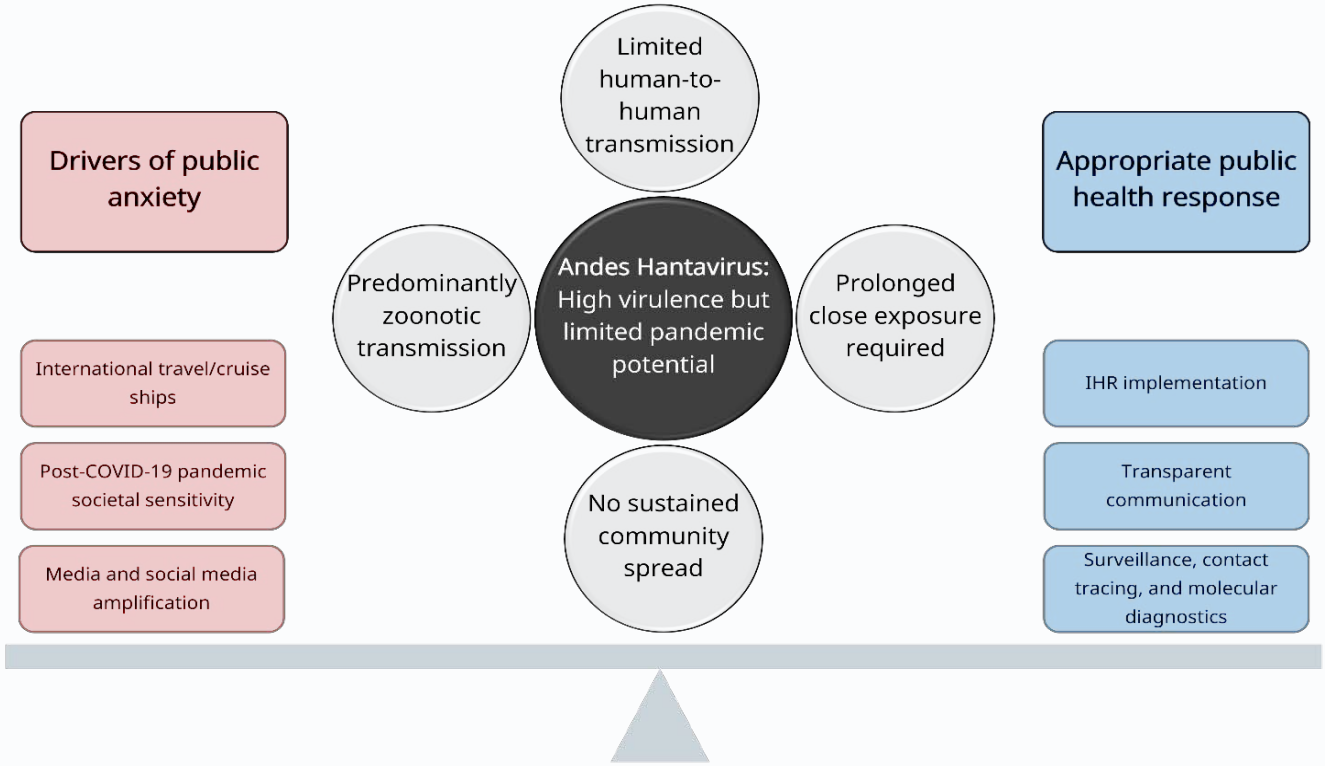

Figure 2: Framework for interpreting pandemic risk during the M/V Hondius Andes hantavirus (ANDV) outbreak. The central framework illustrates the major biologic and epidemiologic constraints limiting the pandemic potential of ANDV. Red panels represent principal drivers of public anxiety and risk amplification. Blue panels represent appropriate evidence-based public health responses. IHR: International Health Regulations.

but interpretation grounded in the established transmission biology of hantaviruses. The outbreak therefore highlights a critical distinction in emerging infectious diseases; pathogens capable of causing severe disease and international concern are not invariably capable of sustaining efficient global transmission.

Risk Communication: The Necessity of Vigilance Without Panic

Modern outbreak management depends as much upon effective risk communication as upon virologic containment. In the contemporary information milieu, public anxiety and misinformation may disseminate more rapidly than pathogens themselves [121-124]. As stated earlier, the post-COVID-19 era has substantially heightened global sensitivity toward emerging infectious threats, particularly those associated with international travel, cruise ships, or unexplained respiratory illness [9, 125, 126]. Under such conditions, geographically limited outbreaks may rapidly acquire disproportionate global visibility through continuous media coverage, social media amplification, and collective societal memory of previous pandemics [127, 128].

The M/V Hondius ANDV outbreak therefore represents an epidemiologic concern as well as a test of institutional risk communication. The appropriate response should be neither dismissal nor alarmism. Vigilance remains essential because zoonotic spillover events represent continuing opportunities for viral adaptation, ecological expansion, and unexpected epidemiologic behavior. Robust surveillance systems, diagnostic preparedness, infection-control capacity, and international coordination remain indispensable components of global health security. As illustrated in Figure 2, effective outbreak management requires a calibrated balance between legitimate epidemiologic vigilance and avoidance of disproportionate societal alarm.

At the same time, disproportionate panic may itself become a public health hazard [1, 129]. Inappropriate alarm can promote stigmatization of affected individuals, dissemination of misinformation, inappropriate resource allocation, unnecessary travel disruption, and erosion of public trust in health authorities [52, 130, 131]. Effective outbreak communication therefore requires calibrated transparency; acknowledging scientific uncertainty and clinical seriousness while simultaneously contextualizing the actual probability of sustained transmission [132]. The WHO Director-General Tedros Adhanom Ghebreyesus reflected this balance by emphasizing both the seriousness of the ANDV outbreak and the currently low public health risk posed by the outbreak [92]. Such messaging is epidemiologically important because overstatement and understatement are equally capable of undermining public confidence [133]. Credible public health institutions must communicate proportionately to the available evidence rather than react to the emotional intensity surrounding a pathogen [134].

The M/V Hondius ANDV outbreak also underlines the enduring importance of international cooperation in infectious disease control. Pathogens do not recognize national borders, and containment increasingly depends upon transparent reporting, rapid data sharing, coordinated surveillance, and equitable diagnostic access [135, 136]. The IHR were designed for such cross-border events, providing a framework through which countries can coordinate responses to emerging health threats while minimizing unnecessary interference with travel and trade [137, 138]. Ultimately, the lesson of the M/V

Hondius ANDV outbreak is not that highly pathogenic zoonotic viruses should be trivialized, but rather that they must be interpreted through rigorous epidemiologic analysis rather than reflexive fear. Vigilance grounded in evidence remains more sustainable, more credible, and more effective than panic.

Clinical and Public Health Implications of the M/V Hondius ANDV Outbreak

Several important clinical and public health lessons emerge from the M/V Hondius ANDV outbreak. First, clinicians should maintain heightened diagnostic suspicion for hantavirus infection in travelers presenting with acute febrile illness, thrombocytopenia, unexplained pulmonary infiltrates, or rapidly progressive respiratory compromise following exposure to rodent-infested environments or recognized outbreak settings [15, 92, 120]. Early recognition is particularly important because HPS may deteriorate abruptly after the onset of cardiopulmonary manifestations, frequently progressing within hours to severe hypoxemia, non-cardiogenic pulmonary edema, shock, and intensive care requirement [139]. Delayed diagnosis may additionally increase the risk of unrecognized exposure among healthcare personnel and close household contacts, particularly in the context of ANDV infection, for which limited person-to-person transmission has been reported [140].

Second, infection-control measures should remain evidence-based and proportionate to the known transmission dynamics of ANDV. Current epidemiologic evidence supports active surveillance, prompt diagnostic evaluation, and targeted isolation of symptomatic high-risk contacts, particularly among individuals with prolonged close exposure to confirmed cases [14, 15, 120]. However, implementation of broad quarantine measures in the absence of evidence for efficient sustained transmission may generate substantial societal and economic disruption while providing limited epidemiologic benefit [141, 142]. Public health responses that exceed the biologic realities of a pathogen risk misallocation of healthcare resources, amplification of public anxiety, and unintended erosion of confidence in health institutions.

Third, the ANDV outbreak reinforces the continuing importance of zoonotic surveillance within an increasingly interconnected ecological landscape [143]. Climate change, deforestation, urban expansion, biodiversity disruption, and shifting rodent population dynamics continue to alter patterns of human–wildlife interaction, thereby increasing opportunities for spillover of rodent-borne pathogens [144-146]. Hantavirus ecology is particularly influenced by environmental fluctuations affecting reservoir abundance and behavior, including rainfall variability, food availability, and habitat displacement [147, 148]. Consequently, future emergence patterns may become less geographically predictable as ecological pressures reshape reservoir distribution and human exposure risk [149]. The ANDV outbreak also highlights the necessity of maintaining international laboratory preparedness and diagnostic surge capacity [91, 150]. Because hantavirus infections remain uncommon in many regions outside endemic areas, delayed recognition may occur in healthcare systems unfamiliar with the clinical syndrome. Rapid access to molecular diagnostics, standardized laboratory protocols, and coordinated international reporting mechanisms therefore remains essential for timely case identification, outbreak containment, and accurate epidemiologic assessment.

Equally important is the management of public uncertainty. Scientific credibility during outbreaks depends not upon projecting absolute certainty, but upon communicating evolving evidence transparently, accurately, and proportionately [133]. Public trust may be undermined both by minimization of legitimate risks and by exaggerated speculation unsupported by epidemiologic data [151]. Effective outbreak communication therefore requires continual recalibration as new evidence emerges, particularly during rapidly evolving multi-national events associated with intense media visibility and heightened post-pandemic public sensitivity.

Finally, the M/V Hondius ANDV outbreak serves as a broader reminder that preparedness for emerging infectious diseases cannot be restricted solely to pathogens with obvious pandemic potential. Even epidemiologically constrained zoonotic viruses may generate substantial clinical burden, international disruption, and public anxiety when introduced into highly mobile global populations [152, 153]. The challenge for modern public health systems is therefore to distinguish rigorously between pathogens requiring sustained pandemic concern and those warranting focused containment without disproportionate societal alarm.

Conclusion

The ANDV outbreak linked to the M/V Hondius represents a clinically severe yet epidemiologically constrained zoonotic event. Although ANDV warrants close international monitoring because of its reported potential for limited person-to-person transmission and the substantial mortality associated with HPS, current evidence does not support a high risk of sustained global dissemination. Rather, the ANDV outbreak highlights a fundamental principle in infectious disease epidemiology; the ability of a pathogen to cause severe illness does not necessarily confer the capacity to generate a pandemic. Pandemic potential is determined less by virulence alone than by the interaction among transmissibility, timing of infectiousness, host adaptation, environmental stability, and the ability to sustain repeated chains of community transmission. In contrast to highly transmissible respiratory viruses such as influenza viruses and SARS-CoV-2, ANDV transmission appears biologically constrained by the requirement for prolonged close exposure, limited evidence for asymptomatic dissemination, relatively inefficient secondary transmission, and rapid progression to severe symptomatic disease. These virologic and epidemiologic characteristics substantially reduce the likelihood of uncontrolled international spread despite the high CFR associated with HPS. At the same time, the M/V Hondius ANDV outbreak highlights the importance of global preparedness for emerging zoonotic threats in an era of unprecedented international mobility. Rapid multi-national coordination, timely diagnostic deployment, transparent surveillance systems, and implementation of the IHR remain essential components of effective outbreak containment. Equally important is the maintenance of evidence-based risk communication capable of informing the public without amplifying disproportionate fear or misinformation. The appropriate response to outbreaks of this nature is therefore neither complacency nor alarmism, but disciplined vigilance grounded in epidemiologic evidence and scientific proportionality. In an era increasingly shaped by both emerging pathogens and accelerated information dissemination, maintaining perspective may itself represent an essential component of global health security.

Declarations

Acknowledgments

The authors declare that Generative AI was used in the creation of

this manuscript. ChatGPT-5 was employed for language refinement (improving grammar, sentence structure, and readability of the manuscript) and technical writing assistance (providing suggestions for structuring complex technical descriptions more effectively). We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results. The final decisions and interpretations presented in this article were solely made by the authors.

Author contributions

Conceptualization, Malik Sallam; methodology, Malik Sallam, Johan Snygg, Mohammed Sallam; validation, Malik Sallam, Johan Snygg, Mohammed Sallam; data curation, Malik Sallam, Johan Snygg, Mohammed Sallam; writing—original draft preparation, Malik Sallam; writing—review and editing, Malik Sallam, Johan Snygg, Mohammed Sallam; visualization, Malik Sallam; supervision, Malik Sallam, Johan Snygg, Mohammed Sallam; project administration, Malik Sallam. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical approval

Not applicable.

Availability of data and materials

The authors confirm that the data supporting the findings of this study are available within the article.

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References

1. Pappas G, Kiriaze IJ, Giannakis P, Falagas ME. Psychosocial consequences of infectious diseases. Clin Microbiol Infect. 2009; 15(8): 743-7. doi: 10.1111/j.1469-0691.2009.02947.x.
2. Asmundson GJG, Taylor S. Coronaphobia: Fear and the 2019-nCoV outbreak. J Anxiety Disord. 2020; 70: 102196. Epub 20200210. doi: 10.1016/j.janxdis.2020.102196.
3. Shultz JM, Althouse BM, Baingana F, Cooper JL, Espinola M, Greene MC, et al. Fear factor: The unseen perils of the Ebola outbreak. Bull At Sci. 2016; 72(5): 304-10. Epub 20160811. doi: 10.1080/00963402.2016.1216515.
4. Joffe H. Public apprehension of emerging infectious diseases: are changes afoot? Public Underst Sci. 2011; 20(4): 446-60. doi: 10.1177/0963662510391604.
5. Carmo A, UN news. Hantavirus outbreak on cruise ship not 'another COVID', WHO says 2026 [updated 7 May 2026; cited 2026 11 May 2026]. Available from: https://news.un.org/en/story/2026/05/1167458.
6. VaccinesWork. Hantavirus, COVID, norovirus, legionnaires': why are cruise ships so prone to disease outbreaks? VaccinesWork: Gavi, the Vaccine Alliance; 2026 [updated 5 May 2026; cited 2026 11 May 2026]. Available from: https://www.gavi.org/vaccineswork/hantavirus-covid-norovirus-legionnaires-why-are-cruise-ships-so-prone-disease.
7. Tokuda Y, Sakihama T, Aoki M, Taniguchi K, Deshpande GA, Suzuki S, et al. COVID-19 outbreak on the Diamond Princess Cruise Ship in February 2020. J Gen Fam Med. 2020; 21(4): 95-7. Epub 20200729. doi: 10.1002/jgf2.326.
8. Jimi H, Hashimoto G. Challenges of COVID-19 outbreak on the cruise ship Diamond Princess docked at Yokohama, Japan: a real-world story. Glob Health Med. 2020; 2(2): 63-5. doi: 10.35772/ghm.2020.01038.
9. Sallam M. Six Years on: Persistent Structural Gaps in COVID-19 Pandemic Policy and Implications for Future Global Health Crises. WebLog Journal of Public Health and Epidemiology. 2026; 2(1): d1802. doi: 10.5281/zenodo.19765318.
10. Lipsitch M, Moxon ER. Virulence and transmissibility of pathogens: what is the relationship? Trends Microbiol. 1997; 5(1): 31-7. doi: 10.1016/s0966-842x(97)81772-6.
11. Alizon S, Hurford A, Mideo N, Van Baalen M. Virulence evolution and the trade-off hypothesis: history, current state of affairs and the future. J Evol Biol. 2009; 22(2): 245-59. doi: 10.1111/j.1420-9101.2008.01658.x.
12. Brown NF, Wickham ME, Coombes BK, Finlay BB. Crossing the line: selection and evolution of virulence traits. PLoS Pathog. 2006; 2(5): e42. doi: 10.1371/journal.ppat.0020042.
13. McCloskey B, Dar O, Zumla A, Heymann DL. Emerging infectious diseases and pandemic potential: status quo and reducing risk of global spread. Lancet Infect Dis. 2014; 14(10): 1001-10. Epub 20140901. doi: 10.1016/s1473-3099(14)70846-1.
14. World Health Organization (WHO). Hantavirus cluster linked to cruise ship travel, Multi-country WHO: WHO; 2026 [updated 8 May 2026; cited 2026 11 May 2026]. Available from: https://www.who.int/emergencies/disease-outbreak-news/item/2026-DON600.
15. CDC: Centers for Disease Control and Prevention. CDC Provides Update on Hantavirus Outbreak Linked to M/V Hondius Cruise Ship 2026 [updated 8 May 2026; cited 2026 11 May 2026]. Available from: https://www.cdc.gov/media/releases/2026/2026-cdc-provides-update-on-hantavirus-outbreak-linked-to-m-v-hondius-cruise-ship.html.
16. World Health Organization (WHO). Disease Outbreak News: Hantavirus cluster linked to cruise ship travel, Multi-country WHO: WHO; 2026 [updated 13 May 2026; cited 2026 15 May 2026]. Available from: https://www.who.int/emergencies/disease-outbreak-news/item/2026-DON601.
17. Laenen L, Vergote V, Calisher CH, Klempa B, Klingström J, Kuhn JH, et al. Hantaviridae: Current Classification and Future Perspectives. Viruses. 2019; 11(9): 788. Epub 20190827. doi: 10.3390/v11090788.
18. Vaheri A, Strandin T, Hepojoki J, Sironen T, Henttonen H, Mäkelä S, et al. Uncovering the mysteries of hantavirus infections. Nat Rev Microbiol. 2013; 11(8): 539-50. doi: 10.1038/nrmicro3066.
19. Omoga DCA, Tchouassi DP, Venter M, Ogola EO, Rotich G, Muthoni JN, et al. Divergent Hantavirus in Somali Shrews (Crocidura somalica) in the Semi-Arid North Rift, Kenya. Pathogens. 2023; 12(5): 685. Epub 20230507. doi: 10.3390/pathogens12050685.
20. Mir MA. Hantaviruses. Clin Lab Med. 2010; 30(1): 67-91. doi: 10.1016/j.cll.2010.01.004.
21. Lee HW, Baek LJ, Johnson KM. Isolation of Hantaan virus, the etiologic agent of Korean hemorrhagic fever, from wild urban rats. J Infect Dis. 1982; 146(5): 638-44. doi: 10.1093/infdis/146.5.638.
22. Shin M. Becoming an International Scientist in South Korea: Ho Wang Lee's Research Activity about Epidemic Hemorrhagic Fever. Uisahak. 2017; 26(1): 95-124. doi: 10.13081/kjmh.2017.26.95.
23. Johnson KM. Hantaviruses: history and overview. Curr Top Microbiol Immunol. 2001; 256: 1-14. doi: 10.1007/978-3-642-56753-7_1.
24. Narayan KG, Sinha DK, Singh DK. Hantaviruses. In: Narayan KG, Sinha DK, Singh DK, editors. Handbook of Management of Zoonoses. Singapore: Springer Nature Singapore; 2024. p. 291-303.
25. Lee HW, Lee PW, Johnson KM. Isolation of the etiologic agent of Korean Hemorrhagic fever. J Infect Dis. 1978; 137(3): 298-308. doi: 10.1093/infdis/137.3.298.
26. Peters CJ, Simpson GL, Levy H. Spectrum of hantavirus infection: hemorrhagic fever with renal syndrome and hantavirus pulmonary syndrome. Annu Rev Med. 1999; 50: 531-45. doi: 10.1146/annurev.

med.50.1.531.

27. Chen RX, Gong HY, Wang X, Sun MH, Ji YF, Tan SM, et al. Zoonotic Hantaviridae with Global Public Health Significance. Viruses. 2023; 15(8): 1705. Epub 20230808. doi: 10.3390/v15081705.

28. Lee HW, Vaheri A, Schmaljohn CS. Discovery of hantaviruses and of the Hantavirus genus: personal and historical perspectives of the Presidents of the International Society of Hantaviruses. Virus Res. 2014; 187: 2-5. Epub 20140108. doi: 10.1016/j.virusres.2013.12.019.

29. de Oliveira RC, Guterres A, Fernandes J, D'Andrea PS, Bonvicino CR, de Lemos ER. Hantavirus reservoirs: current status with an emphasis on data from Brazil. Viruses. 2014; 6(5): 1929-73. Epub 20140429. doi: 10.3390/v6051929.

30. Afzal S, Ali L, Batool A, Afzal M, Kanwal N, Hassan M, et al. Hantavirus: an overview and advancements in therapeutic approaches for infection. Front Microbiol. 2023; 14: 1233433. Epub 20231012. doi: 10.3389/fmicb.2023.1233433.

31. Jeyachandran AV, Irudayam JI, Dubey S, Chakravarty N, Daskou M, Zaiss A, et al. Differential tropisms of old and new world hantaviruses influence virulence and developing host-directed antiviral candidates. PLoS Pathog. 2025; 21(8): e1013401. Epub 20250826. doi: 10.1371/journal.ppat.1013401.

32. Sehgal A, Mehta S, Sahay K, Martynova E, Rizvanov A, Baranwal M, et al. Hemorrhagic Fever with Renal Syndrome in Asia: History, Pathogenesis, Diagnosis, Treatment, and Prevention. Viruses. 2023; 15(2): 561. Epub 20230218. doi: 10.3390/v15020561.

33. Mir S. Hantavirus Induced Kidney Disease. Front Med (Lausanne). 2021; 8: 795340. Epub 20220118. doi: 10.3389/fmed.2021.795340.

34. Clement J, LeDuc JW, Lloyd G, Reynes JM, McElhinney L, Van Ranst M, et al. Wild Rats, Laboratory Rats, Pet Rats: Global Seoul Hantavirus Disease Revisited. Viruses. 2019; 11(7): 652. Epub 20190717. doi: 10.3390/v11070652.

35. Essbauer SS, Schmidt-Chanasit J, Madeja EL, Wegener W, Friedrich R, Petraityte R, et al. Nephropathia epidemica in metropolitan area, Germany. Emerg Infect Dis. 2007; 13(8): 1271-3. doi: 10.3201/eid1308.061425.

36. Plyusnin A, Krüger DH, Lundkvist A. Hantavirus infections in Europe. Adv Virus Res. 2001; 57: 105-36. doi: 10.1016/s0065-3527(01)57002-5.

37. Klempa B, Meisel H, Räth S, Bartel J, Ulrich R, Krüger DH. Occurrence of renal and pulmonary syndrome in a region of northeast Germany where Tula hantavirus circulates. J Clin Microbiol. 2003; 41(10): 4894-7. doi: 10.1128/jcm.41.10.4894-4897.2003.

38. Mustonen J, Vapalahti O, Henttonen H, Pasternack A, Vaheri A. Epidemiology of hantavirus infections in Europe. Nephrol Dial Transplant. 1998; 13(11): 2729-31. doi: 10.1093/ndt/13.11.2729.

39. Levis S, Rowe JE, Morzunov S, Enria DA, St Jeor S. New hantaviruses causing hantavirus pulmonary syndrome in central Argentina. Lancet. 1997; 349(9057): 998-9. doi: 10.1016/s0140-6736(05)62895-4.

40. Martinez VP, Bellomo C, San Juan J, Pinna D, Forlenza R, Elder M, et al. Person-to-person transmission of Andes virus. Emerg Infect Dis. 2005; 11(12): 1848-53. doi: 10.3201/eid1112.050501.

41. Martínez VP, Di Paola N, Alonso DO, Pérez-Sautu U, Bellomo CM, Iglesias AA, et al. "Super-Spreaders" and Person-to-Person Transmission of Andes Virus in Argentina. N Engl J Med. 2020; 383(23): 2230-41. doi: 10.1056/NEJMoa2009040.

42. López N, Padula P, Rossi C, Lázaro ME, Franze-Fernández MT. Genetic identification of a new hantavirus causing severe pulmonary syndrome in Argentina. Virology. 1996; 220(1): 223-6. doi: 10.1006/viro.1996.0305.

43. Alonso DO, Pérez-Sautu U, Bellomo CM, Prieto K, Iglesias A, Coelho R, et al. Person-to-Person Transmission of Andes Virus in Hantavirus Pulmonary Syndrome, Argentina, 2014. Emerg Infect Dis. 2020; 26(4): 756-9. doi: 10.3201/eid2604.190799.

44. Martinez-Valdebenito C, Calvo M, Vial C, Mansilla R, Marco C, Palma RE, et al. Person-to-person household and nosocomial transmission of andes hantavirus, Southern Chile, 2011. Emerg Infect Dis. 2014; 20(10): 1629-36. doi: 10.3201/eid2010.140353.

45. Toledo J, Haby MM, Reveiz L, Sosa Leon L, Angerami R, Aldighieri S. Evidence for Human-to-Human Transmission of Hantavirus: A Systematic Review. J Infect Dis. 2022; 226(8): 1362-71. doi: 10.1093/infdis/jiab461.

46. Enria DA, Briggiler AM, Pini N, Levis S. Clinical manifestations of New World hantaviruses. Curr Top Microbiol Immunol. 2001 ;256: 117-34. doi: 10.1007/978-3-642-56753-7_7.

47. Macneil A, Nichol ST, Spiropoulou CF. Hantavirus pulmonary syndrome. Virus Res. 2011; 162(1-2): 138-47. Epub 20110917. doi: 10.1016/j.virusres.2011.09.017.

48. Padula PJ, Edelstein A, Miguel SD, López NM, Rossi CM, Rabinovich RD. Hantavirus pulmonary syndrome outbreak in Argentina: molecular evidence for person-to-person transmission of Andes virus. Virology. 1998; 241(2): 323-30. doi: 10.1006/viro.1997.8976.

49. Ferres M, Vial P, Marco C, Yanez L, Godoy P, Castillo C, et al. Prospective evaluation of household contacts of persons with hantavirus cardiopulmonary syndrome in chile. J Infect Dis. 2007; 195(11): 1563-71. Epub 20070416. doi: 10.1086/516786.

50. Wells RM, Sosa Estani S, Yadon ZE, Enria D, Padula P, Pini N, et al. An unusual hantavirus outbreak in southern Argentina: person-to-person transmission? Hantavirus Pulmonary Syndrome Study Group for Patagonia. Emerg Infect Dis. 1997; 3(2): 171-4. doi: 10.3201/eid0302.970210.

51. Jonsson CB, Figueiredo LT, Vapalahti O. A global perspective on hantavirus ecology, epidemiology, and disease. Clin Microbiol Rev. 2010; 23(2): 412-41. doi: 10.1128/cmr.00062-09.

52. Abuhaloob L, Purnat TD, Tabche C, Atwan Z, Dubois E, Rawaf S. Management of infodemics in outbreaks or health crises: a systematic review. Front Public Health. 2024; 12: 1343902. Epub 20240315. doi: 10.3389/fpubh.2024.1343902.

53. Selvam KP, Kosalram K, Chinnaiyan S. Post-COVID pandemic: The new normal and aftermath. J Family Med Prim Care. 2024; 13(10): 4308-14. Epub 20241018. doi: 10.4103/jfmpc.jfmpc_313_24.

54. Akingbola A, Adegbesan A, Tundealao S, Benson AE, Makinde AO, Shekoni M, et al. Post-pandemic era: global trends, benefits, and barriers in integrating artificial intelligence into public health education. Front Public Health. 2025; 13: 1648970. Epub 20251013. doi: 10.3389/fpubh.2025.1648970.

55. Sallam M, Snygg J, Sallam M. Can Google Scholar Serve as a Sufficient Source for Systematic Reviews? Reconsidering the Need for Multiple Database Searches. WebLog Journal of Medical Sciences. 2026; 2(1): d3001. doi: 10.5281/zenodo.19764716.

56. Elliott RM. Molecular biology of the Bunyaviridae. J Gen Virol. 1990; 71(Pt 3): 501-22. doi: 10.1099/0022-1317-71-3-501.

57. Boshra H. An Overview of the Infectious Cycle of Bunyaviruses. Viruses. 2022; 14(10): 2139. Epub 20220928. doi: 10.3390/v14102139.

58. Muyangwa M, Martynova EV, Khaiboullina SF, Morzunov SP, Rizvanov AA. Hantaviral Proteins: Structure, Functions, and Role in Hantavirus Infection. Front Microbiol. 2015; 6: 1326. Epub 20151127. doi: 10.3389/fmicb.2015.01326.

59. Cifuentes-Muñoz N, Salazar-Quiroz N, Tischler ND. Hantavirus Gn and Gc envelope glycoproteins: key structural units for virus cell entry and virus assembly. Viruses. 2014; 6(4): 1801-22. Epub 20140421. doi: 10.3390/v6041801.

60. Meyer BJ, Schmaljohn CS. Persistent hantavirus infections: characteristics and mechanisms. Trends Microbiol. 2000; 8(2): 61-7. doi: 10.1016/s0966-842x(99)01658-3.

61. Watson DC, Sargianou M, Papa A, Chra P, Starakis I, Panos G. Epidemiology of Hantavirus infections in humans: a comprehensive, global overview. Crit Rev Microbiol. 2014; 40(3): 261-72. Epub 20130422. doi: 10.3109/1040841x.2013.783555.

62. Singh S, Numan A, Sharma D, Shukla R, Alexander A, Jain GK, et al. Epidemiology, virology and clinical aspects of hantavirus infections: an overview. Int J Environ Health Res. 2022; 32(8): 1815-26. Epub 20210422. doi: 10.1080/09603123.2021.1917527.

63. Sayers EW, Cavanaugh M, Clark K, Ostell J, Pruitt KD, Karsch-Mizrachi I. GenBank. Nucleic Acids Research. 2020; 48(D1): D84-D6. doi: 10.1093/nar/gkz956.

64. Katoh K, Rozewicki J, Yamada KD. MAFFT online service: multiple sequence alignment, interactive sequence choice and visualization. Briefings in Bioinformatics. 2019; 20(4): 1160-6. doi: 10.1093/bib/bbx108.

65. Tamura K, Stecher G, Peterson D, Filipski A, Kumar S. MEGA6: Molecular Evolutionary Genetics Analysis version 6.0. Mol Biol Evol. 2013; 30(12): 2725-9. Epub 20131016. doi: 10.1093/molbev/mst197.

66. Guindon S, Dufayard JF, Lefort V, Anisimova M, Hordijk W, Gascuel O. New algorithms and methods to estimate maximum-likelihood phylogenies: assessing the performance of PhyML 3.0. Syst Biol. 2010; 59(3): 307-21. Epub 20100329. doi: 10.1093/sysbio/syq010.

67. Guindon S, Gascuel O. A simple, fast, and accurate algorithm to estimate large phylogenies by maximum likelihood. Syst Biol. 2003; 52(5): 696-704. doi: 10.1080/10635150390235520.

68. Lefort V, Longueville JE, Gascuel O. SMS: Smart Model Selection in PhyML. Mol Biol Evol. 2017; 34(9): 2422-4. doi: 10.1093/molbev/msx149.

69. Anisimova M, Gil M, Dufayard JF, Dessimoz C, Gascuel O. Survey of branch support methods demonstrates accuracy, power, and robustness of fast likelihood-based approximation schemes. Syst Biol. 2011; 60(5): 685-99. Epub 20110503. doi: 10.1093/sysbio/syr041.

70. Rambaut A, Drummond AJ. Molecular evolution, phylogenetics and epidemiology: FigTree v.1.4.4. 2018 [updated 26 November 2018; cited 2026 14 May 2026]. Available from: https://tree.bio.ed.ac.uk/software/figtree/.

71. Bloemberg G, Chudzinski V, Cordey S, Huber M, Laubscher F, Pérez-Rodríguez F-J, et al. Complete sequence of Orthohantavirus andesense virus: Swiss resident 2026 Virological2026 [updated 8 May 2026; cited 2026 15 May 2026]. Available from: https://virological.org/t/complete-sequence-of-orthohantavirus-andesense-virus-swiss-resident-2026/1023.

72. Palacios G. Preliminary analysis of Orthohantavirus andesense virus sequences from a cruise-ship related cluster, May 2026 Virological2026 [updated 11 May 2026; cited 2026 15 May 2026]. Available from: https://virological.org/t/preliminary-analysis-of-orthohantavirus-andesense-virus-sequences-from-a-cruise-ship-related-cluster-may-2026/1029.

73. Vial PA, Ferrés M, Vial C, Klingström J, Ahlm C, López R, et al. Hantavirus in humans: a review of clinical aspects and management. Lancet Infect Dis. 2023; 23(9): e371-e82. Epub 20230424. doi: 10.1016/s1473-3099(23)00128-7.

74. Avšič-Županc T, Saksida A, Korva M. Hantavirus infections. Clinical Microbiology and Infection. 2019; 21: e6-e16. doi: 10.1111/1469-0691.12291.

75. Klein SL, Marks MA, Li W, Glass GE, Fang LQ, Ma JQ, et al. Sex differences in the incidence and case fatality rates from hemorrhagic fever with renal syndrome in China, 2004-2008. Clin Infect Dis. 2011; 52(12): 1414-21. doi: 10.1093/cid/cir232.

76. Gavrilovskaya IN, Gorbunova EE, Mackow NA, Mackow ER. Hantaviruses direct endothelial cell permeability by sensitizing cells to the vascular permeability factor VEGF, while angiopoietin 1 and sphingosine 1-phosphate inhibit hantavirus-directed permeability. J Virol. 2008; 82(12): 5797-806. Epub 20080326. doi: 10.1128/jvi.02397-07.

77. Hepojoki J, Vaheri A, Strandin T. The fundamental role of endothelial cells in hantavirus pathogenesis. Front Microbiol. 2014; 5: 727. Epub 20141222. doi: 10.3389/fmicb.2014.00727.

78. Gavrilovskaya I, Gorbunova E, Matthys V, Dalrymple N, Mackow E. The Role of the Endothelium in HPS Pathogenesis and Potential Therapeutic Approaches. Adv Virol. 2012; 2012: 467059. Epub 20120628. doi: 10.1155/2012/467059.

79. Mori M, Rothman AL, Kurane I, Montoya JM, Nolte KB, Norman JE, et al. High levels of cytokine-producing cells in the lung tissues of patients with fatal hantavirus pulmonary syndrome. J Infect Dis. 1999; 179(2): 295-302. doi: 10.1086/314597.

80. Klingström J, Plyusnin A, Vaheri A, Lundkvist A. Wild-type Puumala hantavirus infection induces cytokines, C-reactive protein, creatinine, and nitric oxide in cynomolgus macaques. J Virol. 2002; 76(1): 444-9. doi: 10.1128/jvi.76.1.444-449.2002.

81. Ennis FA, Cruz J, Spiropoulou CF, Waite D, Peters CJ, Nichol ST, et al. Hantavirus pulmonary syndrome: CD8+ and CD4+ cytotoxic T lymphocytes to epitopes on Sin Nombre virus nucleocapsid protein isolated during acute illness. Virology. 1997; 238(2): 380-90. doi: 10.1006/viro.1997.8827.

82. Noack D, Goeijenbier M, Reusken C, Koopmans MPG, Rockx BHG. Orthohantavirus Pathogenesis and Cell Tropism. Front Cell Infect Microbiol. 2020; 10: 399. Epub 20200804. doi: 10.3389/fcimb.2020.00399.

83. Velavan TP, Schmidt-Chanasit J. When Rare Zoonoses Travel: Andes virus, Hantavirus Cardiopulmonary Syndrome, and the Preparedness Gap. International Journal of Infectious Diseases. 2026: 108778. doi: 10.1016/j.ijid.2026.108778.

84. Shaikh N, Swali P, Houben R. Asymptomatic but infectious - The silent driver of pathogen transmission. A pragmatic review. Epidemics. 2023; 44: 100704. Epub 20230612. doi: 10.1016/j.epidem.2023.100704.

85. Adalja AA, Watson M, Toner ES, Cicero A, Inglesby TV. Characteristics of Microbes Most Likely to Cause Pandemics and Global Catastrophes. Curr Top Microbiol Immunol. 2019; 424: 1-20. doi: 10.1007/82_2019_176.

86. Ward J, Gressani O, Kim S, Hens N, Edmunds WJ. The epidemiology of pathogens with pandemic potential: A review of key parameters and clustering analysis. Epidemics. 2026; 54: 100882. Epub 20251213. doi: 10.1016/j.epidem.2025.100882.

87. Fraser C, Riley S, Anderson RM, Ferguson NM. Factors that make an infectious disease outbreak controllable. Proc Natl Acad Sci U S A. 2004; 101(16): 6146-51. Epub 20040407. doi: 10.1073/pnas.0307506101.

88. Krüger DH, Schönrich G, Klempa B. Human pathogenic hantaviruses and prevention of infection. Hum Vaccin. 2011; 7(6): 685-93. Epub 20110601. doi: 10.4161/hv.7.6.15197.

89. Ulloa-Morrison R, Pavez N, Parra E, Lopez R, Mondaca R, Fernandez P, et al. Critical care management of hantavirus cardiopulmonary syndrome. A narrative review. J Crit Care. 2024; 84: 154867. Epub 20240717. doi: 10.1016/j.jcrc.2024.154867.

90. Vial PA, Valdivieso F, Mertz G, Castillo C, Belmar E, Delgado I, et al. Incubation period of hantavirus cardiopulmonary syndrome. Emerg Infect Dis. 2006; 12(8): 1271-3. doi: 10.3201/eid1208.051127.

91. World Health Organization (WHO). International Health regulations (2005) as amended 2014, 2022 and 2024 WHO: WHO; 2025 [cited 2026 15 May 2026]. Available from: https://apps.who.int/gb/bd/pdf_files/IHR_2014-2022-2024-en.pdf.

92. World Health Organization (WHO). WHO's response to hantavirus cases linked to a cruise ship: WHO; 2026 [updated 7 May 2026; cited 2026 12 May 2026]. Available from: https://www.who.int/news/item/07-05-2026-

who-s-response-to-hantavirus-cases-linked-to-a-cruise-ship.

93. CDC: Centers for Disease Control and Prevention. 2026 Multi-country Hantavirus Cluster Linked to Cruise Ship: CDC; 2026 [updated 8 May 2026; cited 2026 12 May 2026]. Available from: https://www.cdc.gov/han/php/notices/han00528.html.

94. Abdelrazec A. Modeling Infectious Disease Dynamics on Cruise Ships:An Enhanced SIR Framework with Risk Index Assessment. Research Square. 2026; Preprint. doi: 10.21203/rs.3.rs-8747573/v1.

95. Li H, Meng S, Tong H. How to control cruise ship disease risk? Inspiration from the research literature. Mar Policy. 2021; 132: 104652. Epub 20210705. doi: 10.1016/j.marpol.2021.104652.

96. Zhang N, Miao R, Huang H, Chan EY. Contact infection of infectious disease onboard a cruise ship. Sci Rep. 2016; 6: 38790. Epub 20161208. doi: 10.1038/srep38790.

97. Jernigan DB, Hofmann J, Cetron MS, Genese CA, Nuorti JP, Fields BS, et al. Outbreak of Legionnaires' disease among cruise ship passengers exposed to a contaminated whirlpool spa. Lancet. 1996; 347(9000): 494-9. doi: 10.1016/s0140-6736(96)91137-x.

98. Rocklöv J, Sjödin H, Wilder-Smith A. COVID-19 outbreak on the Diamond Princess cruise ship: estimating the epidemic potential and effectiveness of public health countermeasures. J Travel Med. 2020; 27(3). doi: 10.1093/jtm/taaa030.

99. Yamahata Y, Shibata A. Preparation for Quarantine on the Cruise Ship Diamond Princess in Japan due to COVID-19. JMIR Public Health Surveill. 2020; 6(2): e18821. Epub 20200511. doi: 10.2196/18821.

100. Radic A, Lück M, Ariza-Montes A, Han H. Fear and Trembling of Cruise Ship Employees: Psychological Effects of the COVID-19 Pandemic. Int J Environ Res Public Health. 2020; 17(18): 6741. Epub 20200916. doi: 10.3390/ijerph17186741.

101. Zhou J, Chen S-LP, Shi WW, Kanrak M, Ge J. The impacts of COVID-19 on the cruise industry based on an empirical study in China. Marine Policy. 2023; 153: 105631. doi: 10.1016/j.marpol.2023.105631.

102. van Seventer JM, Hochberg NS. Principles of Infectious Diseases: Transmission, Diagnosis, Prevention, and Control. In: Quah SR, editor. International Encyclopedia of Public Health (Second Edition). Oxford: Academic Press; 2017. p. 22-39.

103. Brown D, Yoo J, Johnson T. Spreading Ebola Panic: Newspaper and Social Media Coverage of the 2014 Ebola Health Crisis. Health Communication. 2018; 34: 1-7. doi: 10.1080/10410236.2018.1437524.

104. Chew C, Eysenbach G. Pandemics in the age of Twitter: content analysis of Tweets during the 2009 H1N1 outbreak. PLoS One. 2010; 5(11): e14118. Epub 20101129. doi: 10.1371/journal.pone.0014118.

105. Garfin DR, Holman EA, Fischhoff B, Wong-Parodi G, Silver RC. Media exposure, risk perceptions, and fear: Americans' behavioral responses to the Ebola public health crisis. Int J Disaster Risk Reduct. 2022;77. Epub 20220520. doi: 10.1016/j.ijdrr.2022.103059.

106. Choo K, Williams P, Day T. Host mortality, predation and the evolution of parasite virulence. Ecology Letters. 2003; 6: 310-5. doi: 10.1046/j.1461-0248.2003.00425.x.

107. Morozov A, Best A. Predation on infected host promotes evolutionary branching of virulence and pathogens' biodiversity. J Theor Biol. 2012; 307: 29-36. Epub 20120509. doi: 10.1016/j.jtbi.2012.04.023.

108. Cressler CE, Mc LD, Rozins C, J VDH, Day T. The adaptive evolution of virulence: a review of theoretical predictions and empirical tests. Parasitology. 2016; 143(7) :915-30. Epub 20150825. doi: 10.1017/s003118201500092x.

109. Acevedo MA, Dillemuth FP, Flick AJ, Faldyn MJ, Elderd BD. Virulence-driven trade-offs in disease transmission: A meta-analysis. Evolution. 2019; 73(4): 636-47. Epub 20190214. doi: 10.1111/evo.13692.

110. Rewar S, Mirdha D. Transmission of Ebola Virus Disease: An Overview. Annals of Global Health. 2014; 80(6): 444-51. doi: 10.1016/j.aogh.2015.02.005.

111. Hasan S, Ahmad SA, Masood R, Saeed S. Ebola virus: A global public health menace: A narrative review. J Family Med Prim Care. 2019; 8(7): 2189-201. doi: 10.4103/jfmpc.jfmpc_297_19.

112. Meyerowitz EA, Richterman A, Gandhi RT, Sax PE. Transmission of SARS-CoV-2: A Review of Viral, Host, and Environmental Factors. Ann Intern Med. 2021; 174(1) :69-79. Epub 20200917. doi: 10.7326/m20-5008.

113. Balloux F, Tan C, Swadling L, Richard D, Jenner C, Maini M, et al. The past, current and future epidemiological dynamic of SARS-CoV-2. Oxf Open Immunol. 2022; 3(1): iqac003. Epub 20220620. doi: 10.1093/oxfimm/iqac003.

114. Johansson MA, Quandelacy TM, Kada S, Prasad PV, Steele M, Brooks JT, et al. SARS-CoV-2 Transmission From People Without COVID-19 Symptoms. JAMA Netw Open. 2021; 4(1): e2035057. Epub 20210104. doi: 10.1001/jamanetworkopen.2020.35057.

115. Neumann G, Eisfeld Amie J, Kawaoka Y. Viral factors underlying the pandemic potential of influenza viruses. Microbiology and Molecular Biology Reviews. 2025; 89(2): e00066-24. doi: 10.1128/mmbr.00066-24.

116. Carnehl C, Fukuda S, Kos N. Epidemics with behavior. Journal of Economic Theory. 2023; 207: 105590. doi: 10.1016/j.jet.2022.105590.

117. de Roode JC, Yates AJ, Altizer S. Virulence-transmission trade-offs and population divergence in virulence in a naturally occurring butterfly parasite. Proc Natl Acad Sci U S A. 2008; 105(21): 7489-94. Epub 20080520. doi: 10.1073/pnas.0710909105.

118. Engering A, Hogerwerf L, Slingenbergh J. Pathogen-host-environment interplay and disease emergence. Emerg Microbes Infect. 2013; 2(2): e5. Epub 20130206. doi: 10.1038/emi.2013.5.

119. Schmaljohn C, Hjelle B. Hantaviruses: a global disease problem. Emerg Infect Dis. 1997; 3(2): 95-104. doi: 10.3201/eid0302.970202.

120. European Centre for Disease Prevention and Control (ECDC). Andes hantavirus outbreak in cruise ship, 13 May 2026 ECDC: ECDC; 2026 [cited 2026 14 May 2026]. 13 May 2026. [Available from: https://www.ecdc.europa.eu/en/infectious-disease-topics/hantavirus-infection/surveillance-and-updates/andes-hantavirus-outbreak.

121. Chowdhury N, Khalid A, Turin TC. Understanding misinformation infodemic during public health emergencies due to large-scale disease outbreaks: a rapid review. Z Gesundh Wiss. 2023; 31(4): 553-73. Epub 20210501. doi: 10.1007/s10389-021-01565-3.

122. Cinelli M, Gesualdo F. Infodemic Versus Viral Information Spread: Key Differences and Open Challenges. JMIR Infodemiology. 2025; 5: e57455. Epub 20250507. doi: 10.2196/57455.

123. Bhattacharya S, Singh A. Unravelling the infodemic: A systematic review of misinformation dynamics during the COVID-19 pandemic. Frontiers in Communication. 2025; 10: 1560936. doi: 10.3389/fcomm.2025.1560936.

124. Tentolouris A, Ntanasis-Stathopoulos I, Vlachakis PK, Tsilimigras DI, Gavriatopoulou M, Dimopoulos MA. COVID-19: time to flatten the infodemic curve. Clinical and Experimental Medicine. 2021; 21(2): 161-5. doi: 10.1007/s10238-020-00680-x.

125. Chu C. Global health security in the post-COVID-19 era: threats, preparation, and response. Osong Public Health Res Perspect. 2025; 16(2): 116-25. Epub 20250404. doi: 10.24171/j.phrp.2025.0037.

126. Khubchandani J, Wiblishauser M, Webb FJ, Yockey RA, Batra K. Reforming public health organizations in the post-COVID era. Journal of Medicine, Surgery, and Public Health. 2025; 5: 100187. doi: 10.1016/j.glmedi.2025.100187.

127. Williams BA, Jones CH, Welch V, True JM. Outlook of pandemic preparedness in a post-COVID-19 world. NPJ Vaccines. 2023; 8(1): 178.

Epub 20231120. doi: 10.1038/s41541-023-00773-0.

128. Lv H, Cao X, Chen S, Liu L. Public and Private Information Sharing under "New Normal" of COVID-19: Understanding the Roles of Habit and Outcome Expectation. Int J Environ Res Public Health. 2022; 19(9): 5552. Epub 20220503. doi: 10.3390/ijerph19095552.

129. Wei C, Xu J, Xu Z. The manifestation and causes of public panic in the early stage of COVID-19 in China: a framework based on consciousness-attitude-behavior. Front Public Health. 2024; 12: 1324382. Epub 20241203. doi: 10.3389/fpubh.2024.1324382.

130. Denniss E, Lindberg R. Social media and the spread of misinformation: infectious and a threat to public health. Health Promot Int. 2025; 40(2). doi: 10.1093/heapro/daaf023.

131. Borges do Nascimento IJ, Pizarro AB, Almeida JM, Azzopardi-Muscat N, Gonçalves MA, Björklund M, et al. Infodemics and health misinformation: a systematic review of reviews. Bull World Health Organ. 2022; 100(9): 544-61. Epub 20220630. doi: 10.2471/blt.21.287654.

132. Cruickshank S, McKee M, Pagel C. Effective communication and public engagement strategies to counter misinformation about infectious diseases. Immunol Cell Biol. 2026; 104(2): 92-105. Epub 20251212. doi: 10.1111/imcb.70073.

133. Sallam M. The fragility of public health in the face of controversial leadership. BMJ Glob Health. 2025; 10(7). Epub 20250701. doi: 10.1136/bmjgh-2024-018536.

134. Niederdeppe J, Boyd AD, King AJ, Rimal RN. Strategies for Effective Public Health Communication in a Complex Information Environment. Annu Rev Public Health. 2025; 46(1): 411-31. Epub 20241210. doi: 10.1146/annurev-publhealth-071723-120721.

135. Ellwanger JH, Veiga ABG, Kaminski VL, Valverde-Villegas JM, Freitas AWQ, Chies JAB. Control and prevention of infectious diseases from a One Health perspective. Genet Mol Biol. 2021;44(1 Suppl 1): e20200256. Epub 20210129. doi: 10.1590/1678-4685-gmb-2020-0256.

136. Mulumba B, N'gattia A, Belizaire MR. One Health, Many Gaps: Rethinking Epidemic Intelligence in Resource-Limited Settings to Prepare for the Global Threat of Disease X. Microorganisms. 2025; 13(11): 2615. doi: 10.3390/microorganisms13112615.

137. The Lancet Global Health. The future of the International Health Regulations. Lancet Glob Health. 2022; 10(7): e927. doi: 10.1016/s2214-109x(22)00254-6.

138. Wilson K, von Tigerstrom B, McDougall C. Protecting global health security through the International Health Regulations: requirements and challenges. Cmaj. 2008; 179(1): 44-8. Epub 20080612. doi: 10.1503/cmaj.080516.

139. Brorstad A, Oscarsson KB, Ahlm C. Early diagnosis of hantavirus infection by family doctors can reduce inappropriate antibiotic use and hospitalization. Scand J Prim Health Care. 2010; 28(3): 179-84. doi: 10.3109/02813432.2010.506058.

140. Bi Z, Formenty PB, Roth CE. Hantavirus infection: a review and global update. J Infect Dev Ctries. 2008; 2(1): 3-23. Epub 20080201. doi: 10.3855/jidc.317.

141. Arndt C, Davies R, Gabriel S, Harris L, Makrelov K, Robinson S, et al. Covid-19 lockdowns, income distribution, and food security: An analysis for South Africa. Glob Food Sec. 2020; 26: 100410. Epub 20200717. doi: 10.1016/j.gfs.2020.100410.

142. Adebiyi BO, Roman NV, Chinyakata R, Balogun TV. The Negative Impacts of COVID-19 Containment Measures on South African Families - Overview and Recommendations. The Open Public Health Journal. 2021; 14: 233-8. doi: 10.2174/1874944502114010233.

143. Sharan M, Vijay D, Yadav JP, Bedi JS, Dhaka P. Surveillance and response strategies for zoonotic diseases: a comprehensive review. Sci One Health. 2023; 2: 100050. Epub 20231101. doi: 10.1016/j.soh.2023.100050.

144. Hudu SA, Jimoh AO, Amin-Nordin S, Syed Hassan S. Environmental degradation as a recipe for emerging viral diseases: implications for global health. One Health Outlook. 2026; 8(1). Epub 20260318. doi: 10.1186/s42522-026-00203-w.

145. Esposito MM, Turku S, Lehrfield L, Shoman A. The Impact of Human Activities on Zoonotic Infection Transmissions. Animals (Basel). 2023; 13(10): 1646. Epub 20230515. doi: 10.3390/ani13101646.

146. Pfenning-Butterworth A, Buckley LB, Drake JM, Farner JE, Farrell MJ, Gehman AM, et al. Interconnecting global threats: climate change, biodiversity loss, and infectious diseases. Lancet Planet Health. 2024; 8(4): e270-e83. doi: 10.1016/s2542-5196(24)00021-4.

147. Tian H, Stenseth NC. The ecological dynamics of hantavirus diseases: From environmental variability to disease prevention largely based on data from China. PLoS Negl Trop Dis. 2019; 13(2): e0006901. Epub 20190221. doi: 10.1371/journal.pntd.0006901.

148. Guterres A, de Lemos ERS. Hantaviruses and a neglected environmental determinant. One Health. 2018; 5: 27-33. Epub 20180102. doi: 10.1016/j.onehlt.2017.12.002.

149. Guo J, Semenza JC, Ecke F, Rizzoli A, Dagostin F, Ulrich RG, et al. A pan-European assessment of multi-sector drivers of human hantavirus risk: climate, biodiversity, and socio-economic factors as key determinants. Environmental Research. 2026; 298: 124282. doi: 10.1016/j.envres.2026.124282.

150. Wolford T, Sutton J, Mangal CN. Laboratory Response to Pandemic Threats: Challenges, Needs, and Solutions. Health Secur. 2023; 21(S1): S56-s9. Epub 20230809. doi: 10.1089/hs.2022.0123.

151. Jennings B, Gostin LO. The crisis of public trust and legitimacy in public health: being successful in a world in turmoil. J Public Health (Oxf). 2025; 47(Supplement_1): i5-i7. doi: 10.1093/pubmed/fdaf099.

152. Rahman MT, Sobur MA, Islam MS, Ievy S, Hossain MJ, El Zowalaty ME, et al. Zoonotic Diseases: Etiology, Impact, and Control. Microorganisms. 2020; 8(9): 1405. Epub 20200912. doi: 10.3390/microorganisms8091405.

153. Salajegheh Tazerji S, Magalhães Duarte P, Gharieb R, Szarpak L, Pruc M, Rahman MT, et al. Migratory Wave due to Conflicts: Risk of Increased Infection From Zoonotic Diseases. Transbound Emerg Dis. 2025; 2025: 5571316. Epub 20250130. doi: 10.1155/tbed/5571316.