Hantavirus: Rare, Severe, and Mostly Preventable

SOURCETRACER PUBLIC HEALTH REVIEW

Hantavirus: Rare, Severe, and Mostly Preventable

Hantaviruses are uncommon but potentially severe rodent-associated viruses. This review summarizes their transmission, clinical significance, and public-health risk in practical terms, with emphasis on environmental prevention, early recognition, and the distinction between typical rodent-associated exposure and Andes virus person-to-person outbreak risk.

David R. Graham, PhD
Graham Scientific, LLC
Germantown, MD
Email: david.graham@grahamscientific.us

Published Saturday, May 9, 2026
Published by Graham Scientific on the SourceTracer™ website
Prepared using SourceTracer PubMed evidence retrieval and citation review. Vancouver-style citations are numbered in order of first appearance.

The Practical Risk

Hantaviruses are globally distributed orthohantaviruses maintained primarily in small mammal reservoir hosts. In humans, they cause two major clinical patterns: hantavirus cardiopulmonary syndrome, also called hantavirus pulmonary syndrome, which predominates in the Americas, and hemorrhagic fever with renal syndrome, which is classically associated with Old World hantaviruses in Europe and Asia.[1-4] This distinction is useful for clinical orientation, but the syndromes overlap mechanistically. Both are vascular leak diseases shaped by endothelial dysfunction, thrombocytopenia, immune activation, and organ-specific involvement.[1,4,5]

The dominant public-health message is straightforward: most human infections follow environmental exposure to infected rodents or contaminated rodent material, not casual contact between people.[2,6] The highest-risk scenario is disturbance of rodent-contaminated dust in enclosed or poorly ventilated spaces such

as cabins, sheds, barns, crawlspaces, storage areas, trailers, and peridomestic structures.[7,8] For the public, prevention is therefore more important than panic: exclude rodents, ventilate closed spaces, wet disinfect contaminated material, avoid dry sweeping or vacuuming, wear gloves and appropriate respiratory protection in higher-risk settings, and seek medical care promptly if compatible symptoms follow plausible exposure.

> **Low incidence should not be confused with low consequence: hantavirus infection is uncommon, but severe cardiopulmonary disease can progress rapidly.**

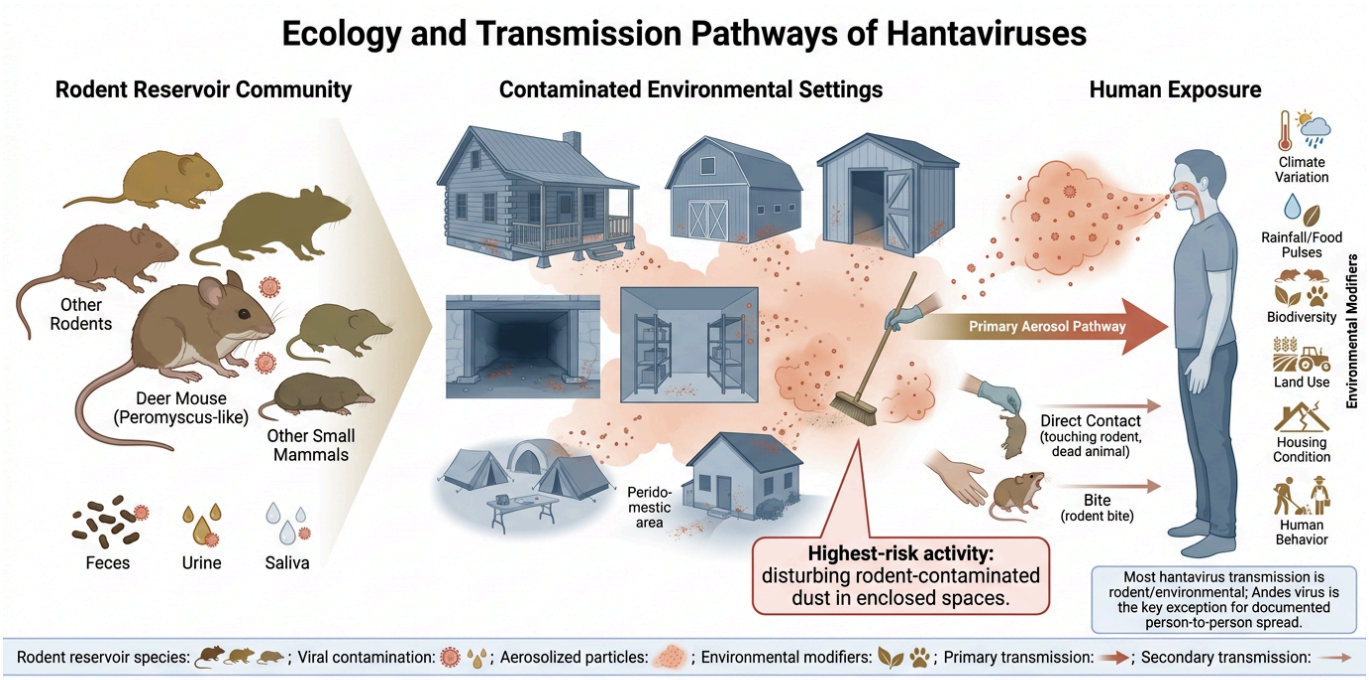


Figure 1. Ecology and environmental transmission pathways of hantaviruses. Hantaviruses are maintained in reservoir host communities and spill over to humans when infectious rodent excreta contaminate enclosed or peridomestic environments. The figure emphasizes the primary aerosol pathway from feces, urine, saliva, and nesting material to inhaled contaminated dust, while distinguishing secondary routes such as direct rodent contact or bites. Environmental modifiers, including climate variation, rainfall and food pulses, biodiversity, land use, housing condition, and human behavior, alter reservoir abundance and human exposure opportunities.[2,6,9,10]

U.S. Sin Nombre Risk

In the United States, Sin Nombre virus is the principal cause of hantavirus cardiopulmonary syndrome. The classic reservoir is the deer mouse, but newer evidence argues against an overly narrow one-host model. A 2025 study found Sin Nombre virus circulating and being shed by multiple small-mammal species in New Mexico, including evidence of infectious virus in tissues and feces from alternative or novel reservoirs.[11] That finding does not displace the importance of deer mice, but it supports a broader surveillance posture in endemic regions.

U.S. case data show a strongly regional pattern. A retrospective analysis of U.S. orthohantavirus disease from 2008 to 2020 found that most New World hantavirus cases occurred west of the Mississippi, with a major concentration in the Four Corners region, and with many detections occurring between March and August.[12] California environmental investigations from 1993 to 2020 similarly show that many cases were linked to visual evidence of rodent activity, direct rodent contact, or cleaning of rodent-affected spaces; probable exposure sites were often indoor or peridomestic rather than remote wilderness settings.[7] Risk modeling also identifies western U.S. landscapes, arid conditions, social vulnerability, rodent richness, and open low-level development as important long-term exposure correlates.[8]

Why the Disease Can Be Dangerous

Early hantavirus disease is clinically treacherous because it often begins as a nonspecific febrile illness. Fever, malaise, myalgia, headache, gastrointestinal symptoms, and fatigue can precede abrupt cardiopulmonary decline.[1,13] In severe HCPS, patients can develop pulmonary edema, shock, myocardial dysfunction, hypoxemia, and respiratory failure. In HFRS, renal involvement, thrombocytopenia, hemorrhagic manifestations, and renal support needs are more prominent, although renal and pulmonary features can overlap across syndromes.[1,4,13]

The shared mechanism is vascular permeability. Hantaviruses infect endothelial cells and are associated with endothelial barrier disruption, immune activation, platelet activation or depletion, and capillary leak rather than simple direct tissue destruction.[4,5,13] This explains why severe disease requires early recognition and rapid escalation to supportive care. There is no broadly accepted curative antiviral therapy that replaces critical care.[1,14,15] Severe HCPS management may require intensive hemodynamic and respiratory support and, in selected cases, extracorporeal membrane oxygenation.[16-18]

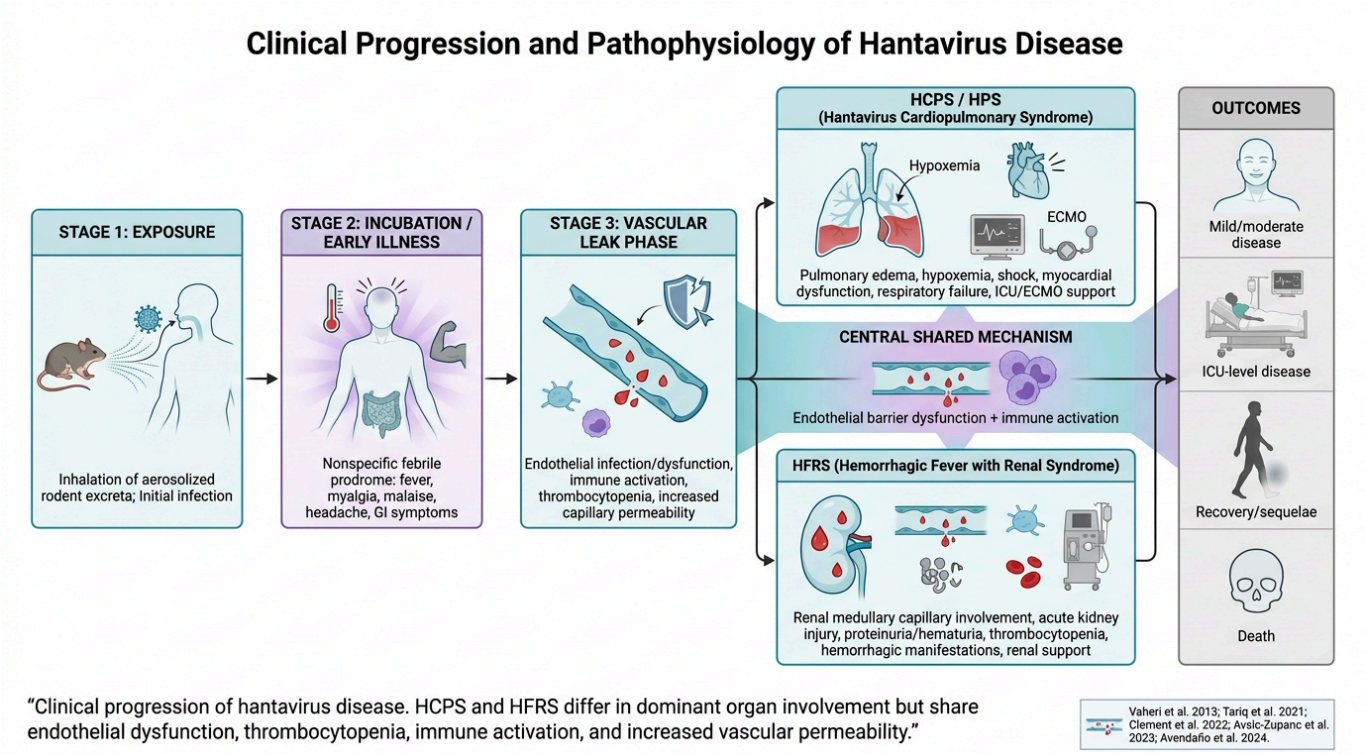


Figure 2. Clinical progression and pathophysiology of hantavirus disease. Hantavirus infection can progress from environmental exposure and nonspecific febrile illness to a vascular leak phase marked by endothelial dysfunction, immune activation, thrombocytopenia, and increased capillary permeability. HCPS/HPS and HFRS differ in dominant organ involvement, with pulmonary edema and shock prominent in HCPS and renal medullary capillary injury more prominent in HFRS, but the syndromes share core vascular and immune mechanisms. Severe disease may require ICU-level supportive care, including renal replacement therapy, mechanical ventilation, or ECMO in selected cases.[1,4,13,16]

The Andes Virus Exception

Most hantavirus prevention focuses on rodent-environment exposure. Andes virus is the important exception because person-to-person transmission has been documented in outbreak investigations. Full-genome sequencing confirmed person-to-person transmission in an Argentina cluster in 2014.[19] A larger 2018-2019 outbreak in Argentina involved 34 confirmed infections and 11 deaths; transmission was associated with symptomatic persons attending crowded events or having extensive contacts, and public-health measures including isolation and self-quarantine were associated with reduced spread.[20] A 2025 characterization of the Epuyen-associated strain further supported concern for sustained person-to-person transmission in that outbreak context.[21]

This point must be communicated carefully. Andes virus evidence does not mean all hantaviruses spread efficiently between people. A systematic review found that the evidence base for human-to-human transmission is heterogeneous and that stronger studies controlling for shared rodent exposure remain needed.[22] The defensible public-health posture is therefore precise: Sin Nombre virus risk in the U.S. is

mainly environmental and rodent-driven, while Andes virus outbreaks require additional contact tracing, isolation, quarantine, and symptom monitoring when indicated.

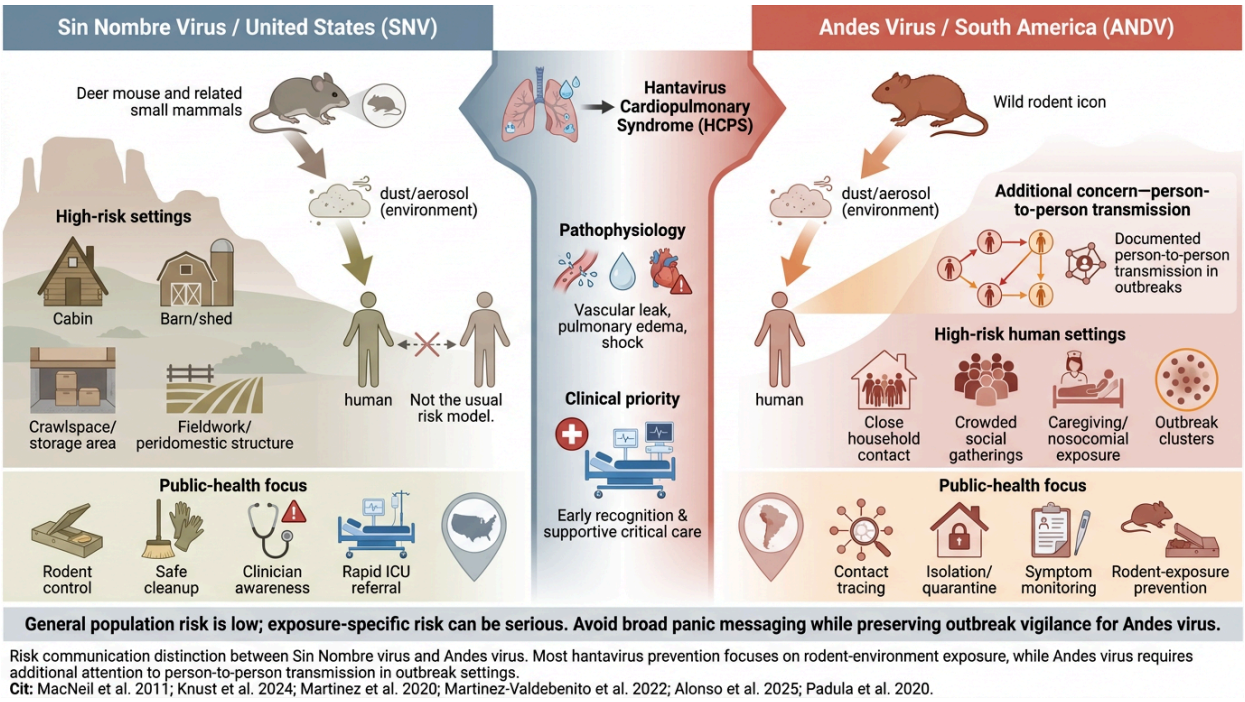


Figure 3. Risk stratification: Sin Nombre environmental exposure versus Andes virus person-to-person transmission. The figure separates the usual U.S. Sin Nombre risk model from Andes virus outbreak dynamics. Sin Nombre prevention centers on rodent control, safe cleanup, clinician awareness, and rapid referral for suspected severe disease. Andes virus shares rodent-environment exposure risk but carries an additional outbreak concern: documented person-to-person transmission in some South American clusters. The central panel highlights shared HCPS pathophysiology and the common clinical priority of early recognition and supportive critical care.[12,19-22]

Public Health Message

The public-facing message should be calm, specific, and action-oriented. Hantavirus is rare, but severe disease can progress quickly. Risk is not distributed evenly across the population; it is concentrated in people who enter, clean, work in, or sleep in rodent-contaminated spaces. The key behaviors are to keep rodents out, avoid aerosolizing contaminated material, disinfect before cleaning, use protection when exposure is likely, and report rodent exposure when seeking care for compatible symptoms.[2,7,14]

Clinicians should consider hantavirus when a compatible febrile illness follows rodent exposure, particularly if thrombocytopenia, pulmonary symptoms, hypotension, renal involvement, or rapid deterioration appear.[1,4,13] Public-health authorities should avoid both overstatement and complacency. Hantavirus is not a generalized pandemic-style respiratory threat, but it is a high-consequence zoonosis at the human-rodent-environment interface.

For the public

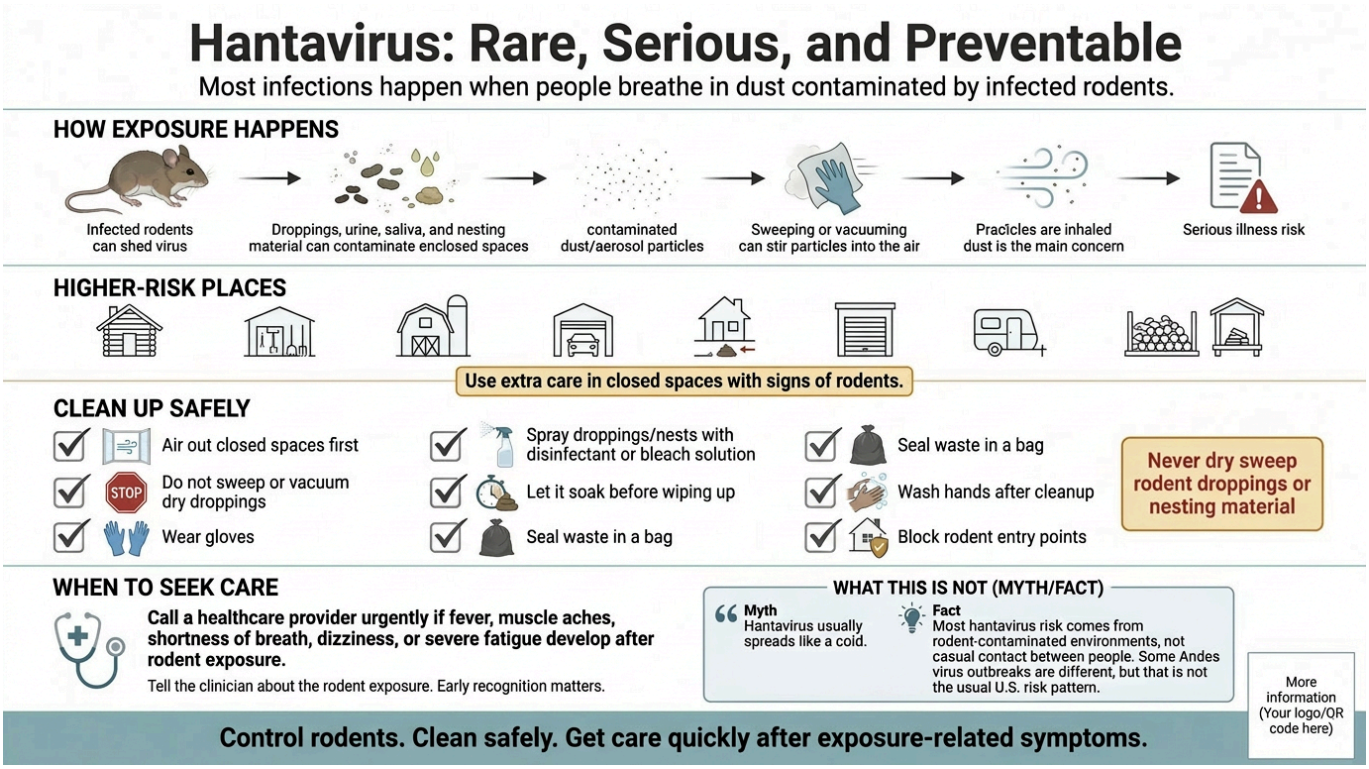
Focus on exposure control: keep rodents out, ventilate closed spaces, disinfect before cleaning, and avoid aerosolizing contaminated material.[2,7,14]

For clinicians

Ask about rodent exposure when febrile illness is followed by thrombocytopenia, pulmonary symptoms, hypotension, renal findings, or rapid deterioration.[1,4,13]

For communicators

Separate typical U.S. Sin Nombre environmental risk from Andes virus outbreak settings, where person-to-person transmission requires added vigilance.[12,19-22]



Public health infographic. Hantavirus: rare, serious, and preventable. This 8.5 x 11 inch handout translates the review evidence into practical prevention guidance for the general public. It highlights the primary exposure pathway from infected rodents to contaminated dust, identifies higher-risk enclosed and peridomestic settings, and prioritizes safe cleanup steps: air out closed spaces, avoid dry sweeping or vacuuming, wet disinfect droppings and nesting material, wear gloves, seal waste, wash hands, and block rodent entry points. The myth/fact panel distinguishes ordinary U.S. rodent-environment risk from the less common Andes virus person-to-person outbreak context.[2,7,12,14,22]

Bottom Line

Hantavirus is rare but serious. In the U.S., prevention centers on avoiding aerosolized rodent-contaminated dust; for Andes virus, outbreak vigilance remains important. The practical priorities are rodent exclusion, safe cleanup, exposure-aware diagnosis, and rapid escalation for severe disease.

References

1. Hantavirus in humans: a review of clinical aspects and management. *Lancet Infect Dis*. 2023. PMID: 37105214.
2. Epidemiology of Hantavirus infections in humans: a comprehensive, global overview. *Crit Rev Microbiol*. 2014. PMID: 23607444.
3. Hantavirus diseases pathophysiology, their diagnostic strategies and therapeutic approaches: A review. *Clin Exp Pharmacol Physiol*. 2021. PMID: 32894790.
4. The kidney in hantavirus infection: epidemiology, virology, pathophysiology, clinical presentation, diagnosis and management. *Clin Kidney J*. 2022. PMID: 35756741.
5. Immunological mechanisms mediating hantavirus persistence in rodent reservoirs. *PLoS Pathog*. 2008. PMID: 19043585.
6. Hantaviruses. *Clin Lab Med*. 2010. PMID: 20513542.
7. Epidemiologic and Environmental Investigations of Reported Hantavirus Cases Inform Exposure Risk in California, 1993-2020. *Am J Trop Med Hyg*. 2025. PMID: 41115422.
8. Hantavirus is Associated With Open Developed Areas and Arid Climates, Highlighting Increased Risk in the Western United States. *Transbound Emerg Dis*. 2025. PMID: 41141578.
9. Dynamics and drivers of hantavirus prevalence in rodent populations. *Vector Borne Zoonotic Dis*. 2014. PMID: 25072983.
10. Hantaviruses and a neglected environmental determinant. *One Health*. 2018. PMID: 29911161.
11. A human pathogenic hantavirus circulates and is shed in taxonomically diverse rodent reservoirs. *PLoS Pathog*. 2025. PMID: 39836687.
12. Human Orthohantavirus disease prevalence and genotype distribution in the U.S., 2008-2020: a retrospective observational study. *Lancet Reg Health Am*. 2024. PMID: 39100240.
13. Hantavirus infections for the clinician: from case presentation to diagnosis and treatment. *Crit Rev Microbiol*. 2012. PMID: 22553984.
14. Hantavirus Infections: Treatment and Prevention. *Curr Treat Options Infect Dis*. 2020. PMID: 33144850.

15. Hantavirus: an overview and advancements in therapeutic approaches for infection. *Front Microbiol*. 2023. PMID: 37901807.

16. Critical care management of hantavirus cardiopulmonary syndrome. A narrative review. *J Crit Care*. 2024. PMID: 39024823.

17. Severe Hantavirus pulmonary syndrome: a new indication for extracorporeal life support? *Crit Care Med*. 1998. PMID: 9468156.

18. An early aggressive strategy for the treatment of Hanta virus cardiopulmonary syndrome: a perspective from an extracorporeal membrane oxygenation center. *Clin Infect Dis*. 2014. PMID: 24771336.

19. Person-to-Person Transmission of Andes Virus in Hantavirus Pulmonary Syndrome, Argentina, 2014. *Emerg Infect Dis*. 2020. PMID: 32186494.

20. "Super-Spreaders" and Person-to-Person Transmission of Andes Virus in Argentina. *N Engl J Med*. 2020. PMID: 33264545.

21. Virological characterization of a new isolated strain of Andes virus involved in the recent person-to-person transmission outbreak reported in Argentina. *PLoS Negl Trop Dis*. 2025. PMID: 40523041.

22. Evidence for Human-to-Human Transmission of Hantavirus: A Systematic Review. *J Infect Dis*. 2022. PMID: 34515290.