One Health Outlook

REVIEW

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Ecological determinants driving orthohantavirus prevalence in small mammals of Europe: a systematic review

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Abstract

Orthohantaviruses are emerging zoonotic pathogens that cause severe human disease and are considered an emerging public health threat globally. Mammalian orthohantaviruses are naturally maintained in rodent species and occasionally in other mammals. The abundance and density of natural orthohantavirus reservoir species are affected by multi annual and seasonal population cycles, community composition, ecosystem variables and climate. Horizontal transmission between host species is mostly density-driven and occurs via contact with infected host excreta, thus, fluctuations in populations and environmental variables often determine the prevalence of hantavirus in natural hosts. Given the zoonotic potential of hantaviruses, ecological factors influencing their spread and persistence in their natural reservoir and population dynamics influencing horizontal transmission require critical evaluation for human infection risk assessment. The present review paper discusses the impacts of natural host population cycles and ecosystem diversity, environmental conditions, and abiotic factors on the epidemiology of rodent-borne hantavirus infections in Europe. While significant efforts have been made to understand the drivers of hantavirus prevalence in natural hosts, we highlight key challenges in evaluating viral prevalence and assessing the role of environmental and population variables in determining hantavirus prevalence in host species.

Introduction

Orthohantaviruses are emerging zoonotic viruses with a major global impact on public health. As per the most recent taxonomical review, the genus Orthohantavirus is one of the four belonging to the subfamily *Mammantavirinae*, order *Bunyavirales*, family *Hantaviridae* and includes sixty different viruses [1]. Like other *Bunyaviridae* members, hantaviruses are enveloped and pleomorphic, with genomes comprising three negative-sense, single-stranded RNA segments encoding structural and non-structural proteins. Orthohantaviruses infect wild rodents, specifically species within *Muridae* (mice), *Cricetidae* (voles), and *Soricidae* (shrews) families in Europe. Occasionally, bats may serve as hosts [1].

Upon direct or indirect contact with infected hosts, hantaviruses can infect humans causing a mild-to-severe, sometimes fatal disease [2–4]. The presence of the virus within the host, both human and rodent, is concentrated in the kidneys, lungs and, at times, may affect the brain [5–8]. Chronic viral proliferation within target organs is often asymptomatic in natural hosts due to immune suppression [9–11] but several clinical signs may manifest if human infection takes

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place. Hantavirus replication in humans occurs in macrophages and vascular endothelial cells of lung and kidney tissues and may lead to many symptoms including fever, pain, nausea and in severe cases renal conditions, shock, haemorrhagic manifestations, and haemodialysis [4, 12–16].

Hantavirus species are divided into Old World and New World based on their distribution and pathogenicity. Old World hantaviruses, which are found in Asia and Europe, predominantly cause haemorrhagic fever with renal syndrome (HFRS). New World hantaviruses, located in the Americas, are major causes of haemorrhagic cardio-pulmonary or pulmonary syndrome (HCPS/HPS).

Approximately thirty thousand people are affected each year, however, owing to widespread underreporting, the actual number of affected individuals is likely to be much greater [2, 17, 18]. The highest case numbers occur in the Old World, particularly in China, with over 10,000 cases reported annually between 2004 and 2016 [19], and in Russia, with approximately 7,000 cases per year [20]. In Europe, cases average around 3,000 annually (mean: 3,152 from 2010–2020, ranging from 1,647 cases in 2020 to 4,686 in 2012 [21–23]). Scandinavia has the highest case numbers within Europe, followed by Central Europe and the Balkans [21–23]. In the Americas, reported cases are much lower, at about 300 annually [4].

Hantavirus transmission primarily occurs through the inhalation of contaminated aerosols derived from the secretions of infected hosts, but may also occur through direct contact with infected host saliva [1, 9, 23–28]. Forestry workers, outdoor enthusiasts, and rural inhabitants are generally considered at greater risk due to their prolonged contact with potentially infected hosts and their habitat [19, 29–36].

Given the high coevolution between viruses and host species, hantaviruses often rely on a single species as the main reservoir [2, 17] although spillover has been extensively reported, the role of non-target hosts remains unclear [9, 37, 38]. In Western Europe, *Orthohantavirus puumalaense* (PUUV) accounts for over 95% of reported human hantavirus infections [23]. Predominant in Boreal and Continental Europe, PUUV causes "nephropathia epidemica" (NE), a milder, often asymptomatic form of HFRS. Its main reservoir host is the bank vole (*Myodes glareolus* syn. *Chletrionomys glareolus*) [11], a common rodent in many forested areas [39]. The second most prevalent hantavirus in Europe, *Orthohantavirus dobravaense* (DOBV), can cause a severe form of HFRS with up to 12% fatality [2]. DOBV is common in the Balkans and Eastern Europe, with the yellow-necked mouse (*Apodemus flavicollis*) as its primary host, often found in mature deciduous forests in mountainous areas of Central and Southern Europe. Less frequently recorded hantaviruses in Europe include *O. tulaense*, *O. seoulense*, *O. hantanense*, *O. brugesense*, and *O. asikkalaense*, linked to various rodent hosts (Table 1), with few or no human cases reported to date [2, 4, 40, 41]. In the Americas, *sigmodontinae*-borne hantaviruses cause the most severe cases of hantavirus cardiopulmonary syndrome (HCPS), with fatality rates reaching 60% [42].

In Europe, human hantavirus infection is a concerning issue aggravated by the lack of available and effective vaccines, an increasing trend in the number of reported human cases, and an increase in the endemicity of natural host species [2, 3, 17, 23]. This trend is likely intensified by ongoing climate change, which is expected to increase the frequency of rodent-borne zoonosis outbreaks by promoting more frequent population surges [48–51].

Table 1 ICVT currently recognised Orthohantavirus species in Western Europe [1], the main reservoir host and broad geographic distribution

***Orthohantavirus* species**	**Abbreviation**	**Main host spp.**	**Location**	**Citation**
O. asikkalaense	ASIV	Pygmy shrew (*Sorex minutus*)	Boreal & Continental Europe	[43, 44]
O. brugesense		European mole (*Talpa europaea*)	Central Europe	[45]
O. dobravaense	DOBV	Yellow-necked mouse (*Apodemus flavicollis*) Striped field mouse (*Apodemus agrarius*)	Balkans Baltics Continental Europe	[46]
O. puumalaense	PUUV	Bank vole (*Clethrionomys glareolus*)	Boreal & Continental Europe	[45]
O. seoulense	SEOV	Brown rats (*Rattus norvegicus*)	Continental Europe	[47]
O. tulaense	TUUV	Common vole (*Microtus arvalis*)	Baltics Continental Europe	[45]

Several studies link human hantavirus cases with location through large-scale climate datasets and virus prevalence surveys in reservoir hosts [48, 50, 52–62]. However, patterns of human cases do not consistently correspond with virus distribution within host populations, and peak prevalence in hosts does not necessarily coincide with peak human incidence [59, 61, 63–65]. Predicting and monitoring hantavirus prevalence in hosts is challenging due to factors such as winter survival rates, ecological conditions, and climate, which influence host population dynamics, distribution, and indirect transmission [65–71]. Moreover, forecasting host population trends across Europe remains difficult, given the scarcity of large-scale data on host abundance and the challenges in obtaining unbiased local data [72].

This review focuses on the environmental, climate and host factors that influence the prevalence of hantavirus in small mammals in Europe. Understanding host population variations, ecological drivers, and the effects of variable interactions on population diversity and indirect transmission is essential for grasping pathogen epidemiology in reservoir hosts, thereby supporting efforts to assess and mitigate human risk.

Methods

The "Preferred Reporting Items for Systematic Reviews and Meta-Analyses" (PRISMA) method was adopted for this review [73]. The searches covered entries from January 2003 to January 2024. The query was applied to four digital databases (CABI Digital Library, PubMed, JSTOR, and SpringerLink) using the Boolean query: Hantavirus AND Europe AND "Small Mammals". After duplicateremoval (=51), 364 entries remained.

We included studies using live trapping data from small mammal reservoir hosts, assessing hantavirus prevalence (regardless of species) and analysing environmental or population dynamics factors. Studies focusing on human case data, genetic diversity, non-European reservoir species, or experimental infections were excluded, though relevant conclusions from experimental studies are discussed. Scientific papers in languages other than English were translated using commercially available software. Out of 364 entries, 51 were excluded for lacking relevance to European hantavirus dynamics (see Fig. 1).

Papers were further filtered after the title; abstract and full text were read. Articles were also removed from the selected pool when no statistics-based method was provided even if all other criteria were fulfilled. After applying the selection criteria, 20 scientific journals were selected for inclusion in the review. A further twenty research papers, identified through reference analysis of the selected articles, were considered significant and included in the review. A complete list of the articles included in this review as well as the main effects they describe can be found in Supplementary Material 1.

Results

Of the 40 articles reviewed, 4 examined multiple hantavirus species across different hosts at the same site. Most studies (37/40) analysed biotic and abiotic factors affecting PUUV prevalence and its primary host, the bank vole (*Clethrionomys glareolus*). Six articles assessed hantavirus prevalence in *Apodemus* spp. (4 with DOBV and 1 with PUUV antibodies), while four focused on TULV in its main host, the field vole. Based on European climate macro areas, 16 studies analysed data from Boreal Europe and 24 from Continental Europe, with articles from the Balkans (2), Pannonian region (2), and Alpine region (1) included in the Continental group (see Supplementary Material 2).

The analysis highlighted five key focus areas. Twenty-one articles examined host characteristics (e.g., gender, age, reproductive maturity), and another 21 analysed population dynamics, specifically abundance and density. Eight studies addressed abiotic factors, such as local or regional climates. Eleven articles explored habitat characteristics, including vegetation, landscape changes, and masting patterns of deciduous trees (periodic production of seeds, nuts, or fruits by deciduous trees). Finally, seven studies investigated interactions with other species, including non-host rodents and predators (Fig. 2). A graphical summary of factors affecting hantavirus prevalence in hosts is presented in Fig. 3.

Host factors

Effects of hantavirus on host fitness and behaviour

While usually asymptomatic, hantavirus infections can affect the survival and fitness of carrier hosts. These viruses have co-evolved with their natural hosts, typically causing chronic, subclinical infections that minimally impact short-term fitness [9–11]. However, host mortality may increase under certain ecological conditions or when prolonged infection significantly affects fitness [74–76]. For example, PUUV-infected bank voles may show lower winter survival than uninfected voles, perhaps due to the energy demands of managing infection under harsh winter conditions and food scarcity [76]. Gender effects on winter survival are unclear, with some studies reporting higher mortality in infected females [75], others in infected males [76], and others only in immature voles regardless of gender [74]. Interestingly, infected hosts may experience increased survival between spring and summer [69]. No mortality changes were observed in infected *A. flavicollis* [75]. Multiple factors—species, fitness, environmental conditions, and reproductive status— may affect hantavirus infection

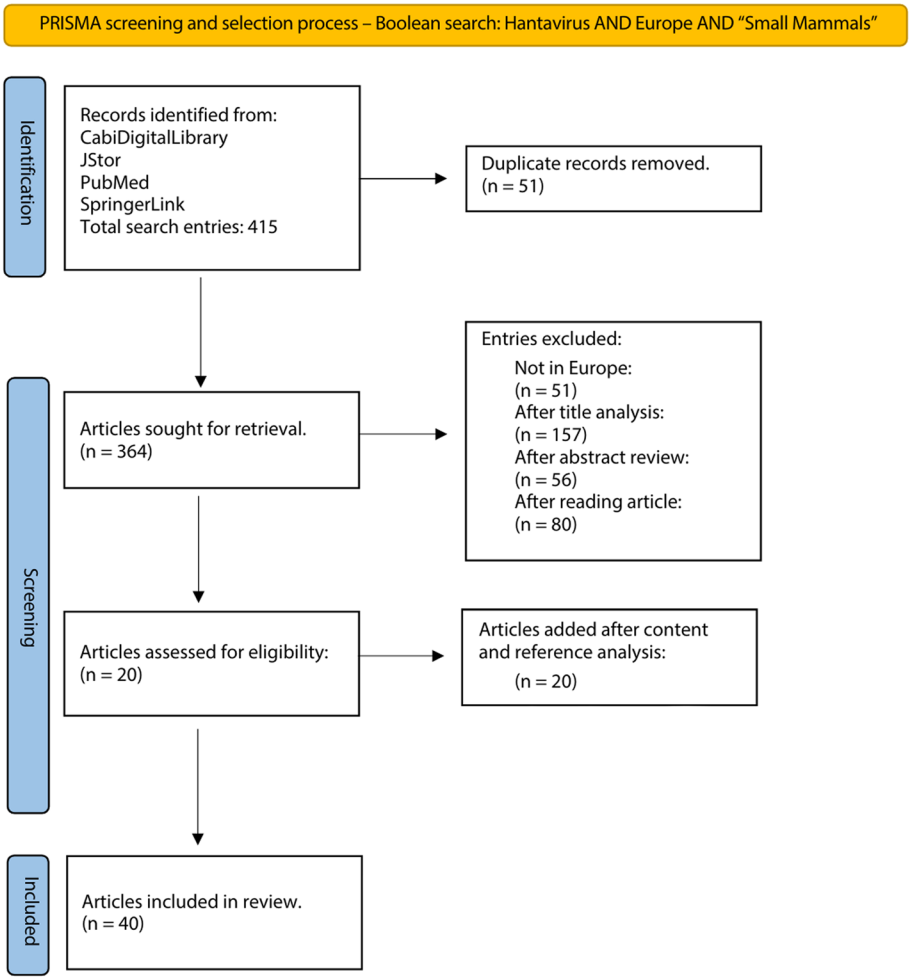

Fig. 1 The PRISMA screening and selection process is illustrated in the figure. Entries on the right indicate the number of articles discarded or included and the reasons for these decisions. Out of 415 entries obtained from search engines, 40 were ultimately selected for this review. The full details and factor breakdown of each selected article are contained in Supplementary Material 1

outcomes, and assessments rely on recapture to evaluate survival, potentially overlooking the effects of dispersal or migration [69, 74–76].

Chronic hantavirus infection in bank voles is linked to co-infections with pathogens such as *Leptospira* spp. and parasitic helminths [77, 78]. PUUV-infected voles are more likely to be co-infected, although whether this is due to individual traits or diminished fitness from infections remains unclear. In contrast, no significant relationship was found between ectoparasite occurrence and DOBV in *Apodemus* spp. [79].

Infected hosts can also exhibit behavioural changes that affect fitness and energy allocation. PUUV-infected female bank voles show age-dependent behavioural shifts during the reproductive season. Young, infected females may be more likely to reach breeding maturity and prioritise litter size and birth frequency, which could compromise long-term immune function and increase pathogen susceptibility [74, 76, 80]. Older females, on the other hand, tend to reduce litter production, likely due to worsened body condition and fitness from prolonged immunological stress [80]. It remains unclear whether this pattern occurs in other small mammal species infected with different hantavirus species [76, 80].

Effects of age on hantavirus prevalence in the host

Most studies suggest that hantavirus prevalence in natural hosts increases with age, using weight or reproductive maturity as indicators [59, 65, 66, 70, 78–90]. Few studies found age to have no significant effect [76, 91]. Similar age-related patterns have been observed for other pathogens, such as *Leptospira* spp., and co-infections are frequently reported [66, 77, 92–94].

Viral proliferation peaks within the first month after infection, maximising infectivity [24, 28]. Although viremia declines over time, it is never fully eliminated,

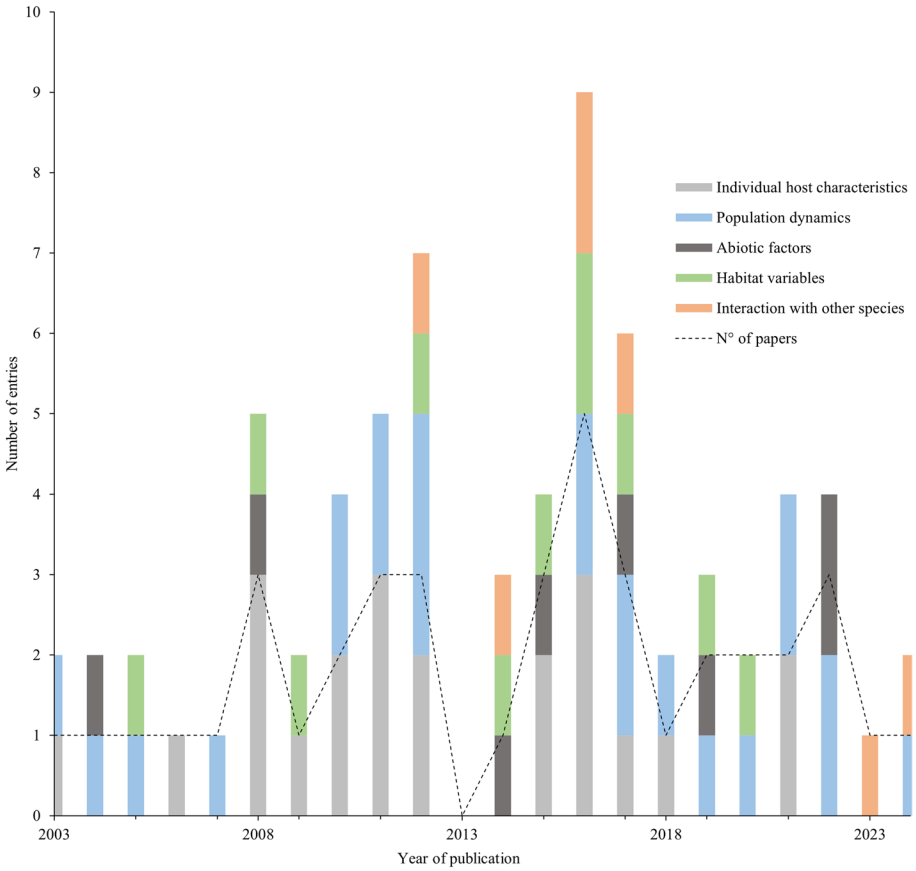

Fig. 2 Publication timeline and topic covered by articles included in the review. The colours of the cumulative bar chart indicate the main topic. The dotted line represents the number of selected articles published in the year

leading to continued shedding [26, 28, 95]. As hosts age, the likelihood of infection increases due to more frequent encounters with infected conspecifics, more aggressive adult behaviour, and greater opportunities for indirect transmission [26, 65, 70].

Newborn bank voles born to hantavirus-infected females inherit maternal antibodies, providing immunity for up to 80 days [26, 88]. This phenomenon, observed across hantavirus species and hosts (including humans) [96–99], contributes to seasonal fluctuations in hantavirus prevalence. This vertical transmission contributes to seasonal declines in hantavirus prevalence, as the influx of young, immune individuals during the reproductive period lowers overall viral prevalence [26, 88, 100–102].

Effect of the host gender on hantavirus prevalence

Gender influences hantavirus prevalence in small mammal hosts, with males more frequently infected than females, likely due to behavioral and physiological factors during the reproductive season [59, 65, 69, 70, 84, 87, 88, 90, 103, 104]. However, several studies instead found no significant effect of gender on prevalence [66, 75, 76, 79, 81, 82, 86, 89, 92].

During the breeding season, mature males allocate energy to reproductive behaviours, which may lead to immune suppression and facilitate infection [105, 106]. These behaviours, including mate-seeking and territoriality, increase the chances of horizontal transmission by bringing males into frequent contact with females and other males, further spreading the virus [69, 84, 101, 107, 108].

Breeding males also mark their territory with hormonally attractive, infectious urine [25, 74]. This drives investigation by both females and competing males, thus promoting the indirect transmission of the pathogen [25, 27, 109]. The behaviour and susceptibility of adult male bank voles play a pivotal role in hantavirus transmission, with higher infection rates among males likely acting as a primary driver of rapid increases in virus prevalence within the host population [101].

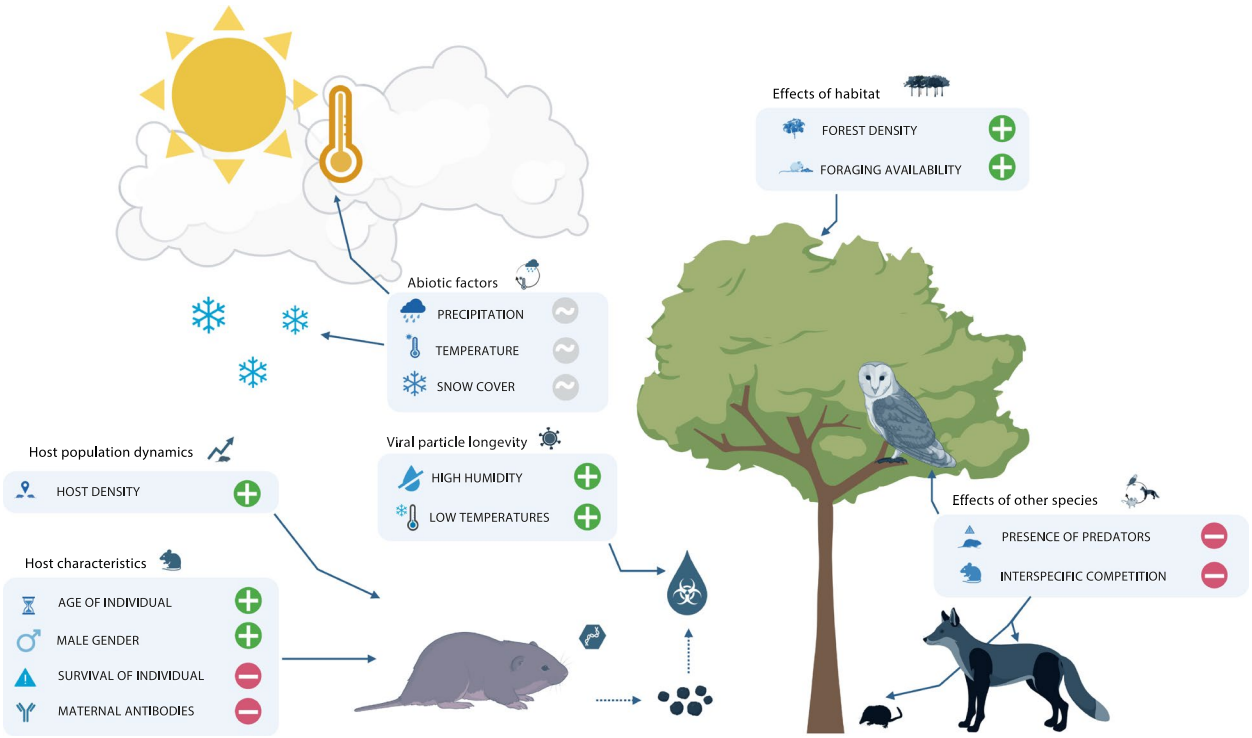

Fig. 3 Graphical representation of key factors influencing Orthohantavirus prevalence in natural reservoir hosts. These factors include host biology, environmental abiotic and biotic conditions, habitat characteristics, and determinants of viral particle survival outside the host. Symbols indicate the direction of influence: a green plus sign represents a positive effect, a red minus sign indicates a negative effect, and a grey tilde denotes variable influence

Genetic immunity to hantavirus

Genetic predisposition plays a key role in PUUV susceptibility in bank voles. Specific immune genes or mutations influence infection tolerance, with related individuals being more susceptible or resistant to infection due to shared genetic traits [83, 107, 110–113]. In several studies, infected hosts from genetically related clusters were observed [84, 113], also likely due in part to communal wintering burrow behaviour [114] that facilitates direct and indirect virus transmission [25].

An experimental study found that bank voles from PUUV-endemic regions had higher antiviral gene expression (e.g., *Tnf*, *Mx2*) than those from non-endemic areas [115]. Genes such as *Drb* (MHC class II), *Tlr-4*, and *Tlr-7* are frequently associated with tolerance strategies in these populations [83, 110, 111, 113, 115, 116].

While immune responses in Central European bank vole populations may reflect postglacial colonisation patterns [117], not all studies agree. For example, a Finnish study found only weak associations between the *Mx2* gene and infection, suggesting limited evidence for strong genetic selection [116]. Further research is needed to identify and understand potential hantavirus resistance genes in host species.

Effects of host demography fluctuations

For horizontally transmitted pathogens, such as hantaviruses, elevated host abundances and densities increase the transmission rate due to higher frequency of contact and greater indirect transmission, leading to a swift increase in pathogen prevalence. Most studies in this review confirm a positive correlation between host abundance and hantavirus prevalence [59, 66, 68–70, 85, 86, 100, 100, 102, 118, 119]. Other studies have reported a delayed effect of host abundance, where prevalence is significantly linked to host abundance several months earlier [65, 67, 104, 120–122], and few found no significant relationship [75, 89, 123], with one even observing a negative impact of current host abundance on seroprevalence [104].

The effect of density should be considered within the context of cyclic fluctuations that occur on both multiannual and seasonal temporal scales, influencing population trends and hantavirus prevalence [124]. In boreal ecosystems, multiannual cyclic peaks occur every three to five years due to predator–prey dynamics [125–128],

whereas in continental ecosystems, peaks appear every two to three years, mainly driven by winter food availability, particularly the seed production of deciduous trees, or mast [48, 58, 124, 129]. These cycles include a "low" phase, a growth phase, and a peak phase, after which populations crash [56, 65, 100, 108, 130].

Seasonal fluctuations further shape small mammal population dynamics. Populations are lowest in winter but increase throughout the reproductive season, peaking in fall. The highest yearly prevalence often occurs at the end of winter, although the intensity of this peak is lower during the "low" phase. The lowest infection prevalence often occurs in early September during the "increase" and "peak" phases, while in the "low" phase, it is usually lowest in early winter, marking the absolute lowest level of hantavirus prevalence. The concept of "virus refugia" [131] may explain how the virus survives during low-density phases by persisting in locations that support viral particle survival outside the host, as discussed further in the chapter titled "Environmental drivers of hantavirus transmission".

Highest hantavirus prevalence in small mammals occurs either in late winter or early in the reproductive season [59, 65–67, 70, 84, 86, 88, 90, 102, 108, 122, 132]. During winter, host populations primarily consist of susceptible adults, as the mating season has ended, and maternal antibodies are no longer present. These adults remain active throughout winter without hibernating, showing reduced territoriality and engaging in communal burrowing, behaviours that facilitate horizontal transmission and contribute to elevated hantavirus prevalence [26, 70, 84, 85, 102, 114, 132]. Additionally, the cold winter conditions help viral particles in excreta maintain infectivity, further aiding transmission [25].

In summer and autumn, when reproductive activity peaks, host abundance and density rise, but the proportion of adults decreases as more juveniles enter the population. During the reproductive season, mature individuals engage in mate-seeking, territorial defence, and foraging behaviours, which increase encounters with potentially infected individuals in high-density environments, further facilitating virus transmission [107, 108]. However, high densities in the summer do not always correlate with increased hantavirus prevalence. The influx of temporarily immune juvenile individuals may reduce overall prevalence by increasing the proportion of immune hosts [88, 100, 102, 120]. Nevertheless, transmission rates remain high due to frequent interactions among mature individuals [108], and one study found the highest prevalence overall in the summer [92]. Notably, maternal antibodies in juveniles were not accounted for in this study, which may result in positive antibody results in uninfected individuals.

In autumn, as the reproductive season ends, hantavirus prevalence may increase in the host population [65, 66]. As reproductive effort declines this would lead to an increased proportion of susceptible adults in the population. Combined with higher densities and abundance, transmission is facilitated [80]. Several studies have indeed reported highest hantavirus prevalence in autumn compared to other seasons [63, 85, 104].

The cyclic nature of annual and multi-annual host population fluctuations influences hantavirus transmission. The abundance of susceptible adults in winter is linked to the density of individuals in the preceding autumn, which plays a crucial role in determining hantavirus prevalence at the start of the reproductive season in spring [65, 67, 104, 120]. Unlike the spring peak, the autumn peak lacks density-dependent characteristics, as many juveniles remain protected by maternal antibodies [69, 88]. While some studies found a relationship between population density and autumn seroprevalence [119], others noted that autumn prevalence can be negatively affected by the population density observed in spring [76, 88]. At a fine temporal scale, the lag between population abundance and hantavirus prevalence ranges from 3 to 4 and a half months, decreasing to 2 months during peak phases [65]. This lag coincides with the window of immune protection from maternal antibodies, and the differences in peak timings may be attributed to density-dependent horizontal transmission dynamics.

Effect of tree mast cycle on transmission dynamics

Cyclic increases in mast production, particularly from oak (*Quercus* spp.) and beech (*Fagus* spp.) trees, lead to a delayed rise in small mammal abundance and density, subsequently affecting hantavirus transmission [48, 59, 63, 81, 118]. These fluctuations in seed production provide abundant autumn resources, which enhance winter survival [2, 48, 57, 59, 65] and lead to larger spring populations, associated with higher levels of hantavirus prevalence throughout the following year [59, 118]. Small mammal populations are affected by masting at both at patch level and landscape level, with masting events being considered one of the primary causes of multi-annual population fluctuations in continental Europe [48, 63, 118].

Effects of inter-species competition

The richness and diversity of non-susceptible species can suppress hantavirus prevalence in the target host by reducing its density through competition for ecological niches and resources, leading to a decrease in viral transmission [133]. In continental Europe, studies have shown that PUUV prevalence in bank voles declines as the proportion of *Apodemus* spp. increases [89, 120]. High shrew

(*Sorex* spp.) abundance also negatively impacts hantavirus prevalence in bank voles, as they compete for similar habitats. Over time, an increase in the abundance of other generalist species may result in reduced vole density, lowering the horizontal transmission rate to susceptible individuals [70, 100, 102, 120]. Host densities may also decline in habitats dominated by specialist species, such as grasslands, where field voles outcompete bank voles, reducing host populations and viral transmission [102]. Seasonal effects have also been observed, with a negative impact of small mammal species diversity on bank vole hantavirus prevalence in spring, but not in fall [70].

The "dilution effect" from interspecific competition does not always occur and can vary depending on the location, habitat, and resource availability. For example, studies conducted in Belgium revealed that the prevalence of *Apodemus* species did not significantly affect bank vole densities or hantavirus prevalence [59, 89]. In contrast, while no substantial impact of other vole or shrew species on PUUV prevalence in bank voles was found, species diversity was linked to changes in bank vole abundance [71]. Additionally, research has also suggested that local small mammal diversity may increase PUUV prevalence in vole hosts, possibly due to interspecific interactions driving more territorial behaviour [123].

Effect of predation

The presence of predators may reduce hantavirus prevalence in host species by inducing behavioural changes or through selective predation of infected individuals. For example, in the presence of foxes (*Vulpes vulpes*), bank voles alter their behaviour to minimise interactions with conspecifics, decreasing pathogen transmission and reducing overall prevalence within the population [71]. Predators may also reduce prevalence by preferentially targeting infected individuals. In boreal ecosystems, owl species (*Aegolius funereus*), along with weasels (*Mustela nivalis*) and foxes, selectively prey on infected voles, affecting hantavirus transmission dynamics [71, 91, 102]. This effect is amplified during high predator density periods in autumn, which decreases infected individuals and overall abundance, leading to a lagged reduction in hantavirus prevalence by spring [65, 67, 104]. The increased predation risk may be due to infected individuals inhabiting suboptimal patches with reduced vegetation cover [68, 107], combined with a loss of immunological fitness induced by the pathogen [74, 76, 80].

Environmental drivers of hantavirus transmission

Outside-host hantavirus particle survival

Viral particle survival outside the host is essential for hantavirus transmission and persistence within host populations. Indirect transmission, primarily via contaminated excreta, is the main pathway for hantavirus spread among reservoir hosts [11]. For PUUV, infectivity in faeces and urine can persist for several days, facilitating aerosol transmission without direct contact [24, 25]. Low temperatures and high moisture enhance viral survival in the environment [25, 134].

Laboratory studies show that particles from *O. hantanense* remains infective for up to 9 days at 20 °C, while TULV and PUUV persist for 5 days at 23 °C [25, 134]. Under optimal conditions, *O. hantanense* retains infectivity for up to 96 days at 4 °C [134] but is inactive within 24 h in dry conditions [25, 134].

Persistent viral infectivity is crucial during high-density phases, such as the reproductive season, and prevents virus fadeout during low-density periods [27, 67, 107, 108, 128]. Hosts often interact with contaminated environments during dispersal, foraging, and mating, further facilitating transmission [27, 109]. Climate analyses show that factors promoting viral survival, such as low temperatures and high rainfall, may correlate with hantavirus prevalence in small mammal populations [68, 71, 102, 108, 122]. A negative relationship between bank vole territorial range and population density also supports the role of indirect transmission in maintaining the virus in low-density scenarios [135].

Habitat influence on viral infection and transmission

Forest patches often show higher hantavirus transmission rate and prevalence, serving as refuges for outside-host viral particles due to shade and humidity, while also providing optimal habitats for hosts [68, 107, 131, 132, 136]. Normalized differential vegetation index (NDVI), obtained from satellite imagery, estimates vegetative production and correlates with increased hantavirus prevalence in bank vole populations, using both historical and seasonal data [89, 108].

Densely vegetated areas offer shelter and foraging resources year-round, supporting high small mammal densities and facilitating hantavirus transmission [70, 108, 131]. This effect is particularly pronounced in winter, when increased trophic availability reduces mortality, leading to higher host densities and increased prevalence in spring [67, 87, 104, 123, 131]. Shaded, humid conditions in winter further enhance viral persistence, promoting indirect transmission [25, 70, 123]. In both boreal and continental European ecosystems, the presence of woody debris and hollow areas on the forest floor may increase PUUV prevalence in bank voles [119, 123, 131].

In Boreal ecosystems, forest strand age may influence bank vole populations and hantavirus prevalence. Old forest strands (over 100 years old) have the highest populations, but the highest PUUV prevalence is found

in 25-30-year-old stands [70]. A more recent study, using landscape metrics and a larger dataset, found that PUUV prevalence in bank voles was linked to old forest strands, while increased forest cutting was associated with lower hantavirus prevalence [132]. Whether forest strand age affects other species or produces similar results in continental Europe remains unclear.

One article analysed also found that the largest populations and highest prevalence of PUUV were found in moist forest environments [119]. The role of habitat moisture was further emphasized by other authors and linked to indirect transmission [25, 131]. Similarly, another study using large spatial datasets found a significant association between proximity to waterlogged soils and hantavirus prevalence in bank voles [71].

When optimal habitats exceed their carrying capacity, small mammals may disperse to lower-quality neighbouring patches, driven by high population densities and limited resources [137]. This typically occurs during population peaks, when abundance rises rapidly, and available niches become crowded [68]. In fragmented landscapes, this dispersal helps spread hantavirus across patches [107]. Dispersing individuals may acquire the virus either before or during movement, as high densities and competition reduce their fitness and increase the likelihood of infection [25, 27, 107, 109]. Additionally, higher densities and hantavirus prevalence may also be observed in isolated patches, possibly due to dispersal constraints [68, 91].

The link between hantavirus infection and dispersal may result in high prevalence in lower-quality habitats, where individuals allocate more energy to survival than to immune function, increasing susceptibility to the virus [70, 91]. These habitats also have higher mortality rates, which may lead to stochastic fadeout events of the virus or the host population [76, 107, 135]. In contrast, high-quality, vegetated patches maintain hantavirus presence by supporting susceptible hosts and trophic availability, even at low population densities [70, 76, 132].

Effects of climate on hantavirus prevalence

In Continental Europe, studies have shown varying effects of temperature and precipitation on hantavirus prevalence in host populations. Some report a positive relationship between winter temperatures and viral prevalence, likely due to improved small mammal fitness and increased vegetative production, which enhances horizontal transmission [56, 79, 108]. In contrast, other studies suggest that colder temperatures increase viral particle longevity, leading to higher hantavirus prevalence in host [68, 89, 123]. Finally, some studies found no effect of temperature on PUUV prevalence in bank voles [60].

In Boreal Europe, winter temperature outcomes are influenced by regional factors. Prolonged snow cover provides thermal insulation, encouraging hosts to reduce activity outside burrows, which may increase horizontal transmission within these sheltered environments [122]. Conversely, in areas with little snow, exposure to harsher winter conditions may cause higher mortality rates, indirectly lowering hantavirus prevalence [53].

Regarding precipitation, some studies found no significant correlation with hantavirus prevalence [68, 71], while others linked winter [108], annual [60, 79], or spring rainfall [90] to increased viral prevalence in host populations. However, a few authors observed that higher winter and spring precipitation negatively affected bank vole density, thus reducing hantavirus prevalence [68]. In Boreal ecosystems, temperature and precipitation during November were associated with PUUV prevalence in bank voles the following spring [122]. Habitat humidity and water bodies also play a role in viral prevalence [60, 71], likely by improving viral survival in moist environments [25]. Further investigation is needed to better understand the timing of rainfall and host sampling frequencies [71].

Conclusion

Humans are increasingly exposed to rodent-transmitted zoonoses, with growing endemicity, more frequent outbreaks, and the breakdown of human-animal barriers. A review of the ecological factors affecting hantaviruses in European reservoir species is of public health importance, as it sheds light on viral transmission dynamics and fluctuations, helping to reduce human risk.

This review examines the complex interactions underlying hantavirus prevalence in rodent populations in Europe. Key drivers include host demographic patterns, which influence transmission among susceptible individuals, as well as environmental factors that prolong indirect transmission, affect host density, and enhance survival. Densely vegetated environments provide optimal conditions for both the virus and its hosts, increasing viral infectivity while offering shelter and resources that boost host fitness and density.

Predicting hantavirus fluctuations in host populations may be possible by monitoring mast years, as food abundance directly affects small mammal winter survival and correlates with higher viral prevalence in spring. With climate change leading to more frequent mast years and milder winters, coupled with a loss of species richness, hantavirus distribution and prevalence are expected to increase across Europe. This review calls for further research, especially on climate and habitat factors, which have produced conflicting results in previous studies. Additionally, preserving small mammal biodiversity and natural

predator–prey dynamics can help mitigate infection peaks by reducing intraspecific interactions and promoting selective predation of infected individuals.

Supplementary Information

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Supplementary Material 1.

Supplementary Material 2.

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Authors' contributions
DF concept and design, writing, and data acquisition. MM concept, writing, and review. VT review. ML review. AO review. PB project coordinator, design and review.

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Data availability
Not applicable.

Declarations

Ethics approval and consent to participate
Not applicable.

Competing interests
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References

1. Bradfute SB, Calisher CH, Klempa B, Klingström J, Kuhn JH, Laenen L, et al. ICTV Virus Taxonomy Profile: Hantaviridae 2024: This article is part of the ICTV Virus Taxonomy Profiles collection. J Gen Virol. 2024;105. https://doi.org/10.1099/jgv.0.001975. Available from: https://www.microbiologyresearch.org/content/journal/jgv/. Cited 2024 May 20.
2. Heyman P, Thoma BR, Marié JL, Cochez C, Essbauer SS. In search for factors that drive hantavirus epidemics. Front Physiol. 2012;3. https://doi.org/10.3389/fphys.2012.00237/full. Available from: https://www.frontiersin.org/journals/physiology/articles/. Cited 2024 May 15.
3. Klempa B. Hantaviruses and climate change. Clin Microbiol Infect. 2009;15:518–23.
4. 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:e371–82.
5. Easterbrook JD, Klein SL. Immunological mechanisms mediating hantavirus persistence in rodent reservoirs. PLOS Pathog. 2008;4:e1000172.
6. Madai M, Horváth G, Herczeg R, Somogyi B, Zana B, Földes F, et al. Effectiveness regarding hantavirus detection in rodent tissue samples and urine. Viruses. 2021;13: 570.
7. Perley CC, Brocato RL, Kwilas SA, Daye S, Moreau A, Nichols DK, et al. Three asymptomatic animal infection models of hemorrhagic fever with renal syndrome caused by hantaviruses. PLoS One. 2019;14:e0216700.
8. Schountz T, Prescott J. Hantavirus immunology of rodent reservoirs: current status and future directions. Viruses. 2014;6:1317–35.
9. Forbes KM, Sironen T, Plyusnin A. Hantavirus maintenance and transmission in reservoir host populations. Curr Opin Virol. 2018;28:1–6.
10. Vaheri A, Vapalahti O, Plyusnin A. How to diagnose hantavirus infections and detect them in rodents and insectivores. Rev Med Virol. 2008;18:277–88.
11. Vapalahti O, Mustonen J, Lundkvist Å, Henttonen H, Plyusnin A, Vaheri A. Hantavirus infections in Europe. Lancet Infect Dis. 2003;3:653–61.
12. Avšič-Županc T, Saksida A, Korva M. Hantavirus infections. Clin Microbiol Infect. 2019;21:e6-16.
13. Krautkrämer E, Zeier M, Plyusnin A. Hantavirus infection: an emerging infectious disease causing acute renal failure. Kidney Int. 2013;83:23–7.
14. Muranyi W, Bahr U, Zeier M, van der Woude FJ. Hantavirus Infection. J Am Soc Nephrol. 2005;16:3669.
15. Rasmuson J, Andersson C, Norrman E, Haney M, Evander M, Ahlm C. Time to revise the paradigm of hantavirus syndromes? Hantavirus pulmonary syndrome caused by European hantavirus. Eur J Clin Microbiol Infect Dis. 2011;30:685–90.
16. Sargianou M, Watson DC, Chra P, Papa A, Starakis I, Gogos C, et al. Hantavirus infections for the clinician: From case presentation to diagnosis and treatment. Crit Rev Microbiol. 2012;38:317–29.
17. Vaheri A, Henttonen H, Voutilainen L, Mustonen J, Sironen T, Vapalahti O. Hantavirus infections in Europe and their impact on public health. Rev Med Virol. 2013;23:35–49.
18. 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;40:261–72.
19. Wang Q, Yue M, Yao P, Zhu C, Ai L, Hu D, et al. Epidemic Trend and Molecular Evolution of HV Family in the Main Hantavirus Epidemic Areas From 2004 to 2016, in P.R. China. Front Cell Infect Microbiol. 2021;10. Available from: https://www.frontiersin.org/journals/cellular-and-infection-microbiology/articles/10.3389/fcimb.2020.584814/full. Cited 2024 Aug 27.
20. Tkachenko EA, Ishmukhametov AA, Dzagurova TK, Bernshtein AD, Morozov VG, Siniugina AA, et al. Hemorrhagic fever with renal syndrome. Russia Emerg Infect Dis. 2019;25:2325–8.
21. European Centre for Disease Prevention and Control. Hantavirus infection - Annual Epidemiological Report for 2014. 2016. Available from: https://www.ecdc.europa.eu/en/publications-data/hantavirus-infection-annual-epidemiological-report-2014. Cited 2024 Aug 27.
22. European Centre for Disease Prevention and Control. Hantavirus infection - Annual epidemiological report for 2016. 2018. Available from: https://www.ecdc.europa.eu/en/publications-data/hantavirus-infection-annual-epidemiological-report-2016. Cited 2024 Aug 27.
23. European Centre for Disease Prevention and Control. Hantavirus infection - Annual Epidemiological Report for 2020. 2023. Available from: https://www.ecdc.europa.eu/en/publications-data/hantavirus-infection-annual-epidemiological-report-2020. Cited 2024 May 15.
24. Hardestam J, Karlsson M, Falk KI, Olsson G, Klingström J, Lundkvist Å. Puumala hantavirus excretion kinetics in bank voles (*Myodes glareolus*). Emerg Infect Dis. 2008;14:1209–15.
25. Kallio ER, Klingström J, Gustafsson E, Manni T, Vaheri A, Henttonen H, et al. Prolonged survival of Puumala hantavirus outside the host: evidence for indirect transmission via the environment. J Gen Virol. 2006;87:2127–34.
26. Kallio ER, Poikonen A, Vaheri A, Vapalahti O, Henttonen H, Koskela E, et al. Maternal antibodies postpone hantavirus infection and enhance individual breeding success. Proc R Soc B Biol Sci. 2006;273:2771–6.
27. Sauvage F, Langlais M, Yoccoz NG, Pontier D. Modelling hantavirus in fluctuating populations of bank voles: the role of indirect transmission on virus persistence. J Anim Ecol. 2003;72:1–13.
28. Voutilainen L, Sironen T, Tonteri E, Bäck AT, Razzauti M, Karlsson M, et al. Life-long shedding of Puumala hantavirus in wild bank voles (Myodes glareolus). J Gen Virol. 2015;96:1238–47.

29. Acke S, Couvreur S, Bramer WM, Schmickler M-N, Schryver AD, Haagsma JA. Global infectious disease risks associated with occupational exposure among non-healthcare workers: a systematic review of the literature. Occup Environ Med. 2022;79:63–71.
30. Andreychev A, Boyarova E, Brandler O, Tukhbatullin A, Kapustina S. Terrestrial and subterranean mammals as reservoirs of zoonotic diseases in the central part of European Russia. Diversity. 2023;15:39.
31. Haagsma JA, Tariq L, Heederik DJ, Havelaar AH. Infectious disease risks associated with occupational exposure: a systematic review of the literature. Occup Environ Med. 2012;69:140–6.
32. Kallio-Kokko H, Laakkonen J, Rizzoli A, Tagliapietra V, Cattadori I, Perkins SE, et al. Hantavirus and arenavirus antibody prevalence in rodents and humans in Trentino, Northern Italy. Epidemiol Infect. 2006;134:830–6.
33. Piechotowski I, Brockmann SO, Schwarz C, Winter CH, Ranft U, Pfaff G. Emergence of hantavirus in South Germany: rodents, climate and human infections. Parasitol Res. 2008;103:131–7.
34. Princk C, Drewes S, Meyer-Schlinkmann KM, Saathoff M, Binder F, Freise J, et al. Cluster of human Puumala orthohantavirus infections due to indoor exposure?—An interdisciplinary outbreak investigation. Zoonoses Public Health. 2022;69:579–86.
35. Riccò M, Peruzzi S, Ranzieri S, Magnavita N. Occupational hantavirus infections in agricultural and forestry workers: a systematic review and metanalysis. Viruses. 2021;13:2150.
36. Tagliapietra V, Rosà R, Rossi C, Rosso F, Hauffe HC, Tommasini M, et al. Emerging rodent-borne viral zoonoses in Trento. Italy EcoHealth. 2018;15:695–704.
37. Forbes KM, Huitu O, Sironen T, Voutilainen L, Stuart P, Niemimaa J, Vapalahti O, Henttonen H. Experimental investigation of a hantavirus host-switch between arvicoline rodents Lemmus lemmus and Myodes glareolus. J Vector Ecol. 2013;38(2):408–10. https://doi.org/10.1111/j.1948-7134.2013.12058.x.
38. Klingström J, Heyman P, Escutenaire S, Sjölander KB, Jaegere FD, Henttonen H, et al. Rodent host specificity of European hantaviruses: evidence of Puumala virus interspecific spillover. J Med Virol. 2002;68:581–8.
39. Hutterer R, Kryštufek B, Yigit N, Mitsainas G, Palomo L, Henttonen H, et al. Myodes glareolus (amended version of 2016 assessment). The IUCN red list of threatened species 2021:e. T4973A197520967. 2021.
40. Dupinay T, Pounder KC, Ayral F, Laaberki M-H, Marston DA, Lacôte S, et al. Detection and genetic characterization of Seoul virus from commensal brown rats in France. Virol J. 2014;11:32.
41. Reynes JM, Carli D, Boukezia N, Debruyne M, Herti S. Tula hantavirus infection in a hospitalised patient, France, June 2015. Euro Surveill Bull Eur Sur Mal Transm Eur Commun Dis Bull. 2015;20:20.
42. Jonsson CB, Figueiredo LTM, Vapalahti O. A global perspective on hantavirus ecology, epidemiology, and disease. Clin Microbiol Rev. 2010;23:412–41.
43. Radosa L, Schlegel M, Gebauer P, Ansorge H, Heroldová M, Jánová E, et al. Detection of shrew-borne hantavirus in Eurasian pygmy shrew (*Sorex minutus*) in Central Europe. Infect Genet Evol. 2013;19:403–10.
44. Ling J, Sironen T, Voutilainen L, Hepojoki S, Niemimaa J, Isoviita V-M, et al. Hantaviruses in Finnish soricomorphs: evidence for two distinct hantaviruses carried by *Sorex araneus* suggesting ancient host-switch. Infect Genet Evol. 2014;27:51–61.
45. Laenen L, Vergote V, Kafetzopoulou LE, Wawina TB, Vassou D, Cook JA, et al. A novel hantavirus of the European mole, bruges virus, is involved in frequent Nova virus coinfections. Genome Biol Evol. 2018;10:45–55.
46. Olsson GE, Leirs H, Henttonen H. Hantaviruses and their hosts in europe: reservoirs here and there, but not everywhere? Vector-Borne Zoonotic Dis. 2010;10:549–61.
47. Ling J, Verner-Carlsson J, Eriksson P, Plyusnina A, Löhmus M, Järhult JD, et al. Genetic analyses of Seoul hantavirus genome recovered from rats (Rattus norvegicus) in the Netherlands unveils diverse routes of spread into Europe. J Med Virol. 2019;91:724–30.
48. Clement J, Vercauteren J, Verstraeten WW, Ducoffre G, Barrios JM, Vandamme A-M, et al. Relating increasing hantavirus incidences to the changing climate: the mast connection. Int J Health Geogr. 2009;8:1.
49. Imholt C, Reil D, Eccard JA, Jacob D, Hempelmann N, Jacob J. Quantifying the past and future impact of climate on outbreak patterns of bank voles (Myodes glareolus). Pest Manag Sci. 2015;71:166–72.
50. Roda Gracia J, Schumann B, Seidler A. Climate variability and the occurrence of human Puumala hantavirus infections in Europe: a systematic review. Zoonoses Public Health. 2015;62:465–78.
51. Semenza JC, Suk JE, Estevez V, Ebi KL, Lindgren E. Mapping climate change vulnerabilities to infectious diseases in Europe. Environ Health Perspect. 2012;120:385–92.
52. Drewes S, Turni H, Rosenfeld UM, Obiegala A, Straková P, Imholt C, et al. Reservoir-driven heterogeneous distribution of recorded human Puumala virus cases in South-West Germany. Zoonoses Public Health. 2017;64:381–90.
53. Khalil H, Olsson G, Ecke F, Evander M, Hjertqvist M, Magnusson M, et al. The importance of bank vole density and rainy winters in predicting nephropathia epidemica incidence in Northern Sweden. Stoddart CA, editor. PLoS One. 2014;9:e111663.
54. Milhano N, Korslund L, Evander M, Ahlm C, Vainio K, Dudman SG, et al. Circulation and diagnostics of Puumala virus in Norway: nephropatia epidemica incidence and rodent population dynamics. APMIS. 2017;125:732–42.
55. Moirano G, Botta A, Yang M, Mangeruga M, Murray K, Vineis P. Land-cover, land-use and human hantavirus infection risk: a systematic review. Pathog Glob Health. 2023;0:1–15.
56. Monchatre-Leroy E, Crespin L, Boué F, Marianneau P, Calavas D, Hénaux V. Spatial and temporal epidemiology of nephropathia epidemica incidence and hantavirus seroprevalence in rodent hosts: identification of the main environmental factors in Europe. Transbound Emerg Dis. 2017;64:1210–28.
57. Swart A, Bekker DL, Maas M, de Vries A, Pijnacker R, Reusken CBEM, et al. Modelling human Puumala hantavirus infection in relation to bank vole abundance and masting intensity in the Netherlands. Infect Ecol Epidemiol. 2017;7:1287986.
58. Tersago K, Verhagen R, Servais A, Heyman P, Ducoffre G, Leirs H. Hantavirus disease (nephropathia epidemica) in Belgium: effects of tree seed production and climate. Epidemiol Infect. 2009;137:250–6.
59. Tersago K, Verhagen R, Vapalahti O, Heyman P, Ducoffre G, Leirs H. Hantavirus outbreak in Western Europe: reservoir host infection dynamics related to human disease patterns. Epidemiol Infect. 2011;139:381–90.
60. Vanwambeke SO, Zeimes CB, Drewes S, Ulrich RG, Reil D, Jacob J. Spatial dynamics of a zoonotic orthohantavirus disease through heterogenous data on rodents, rodent infections, and human disease. Sci Rep. 2019;9:2329.
61. Weber De Melo V, Sheikh Ali H, Freise J, Kühnert D, Essbauer S, Mertens M, et al. Spatiotemporal dynamics of Puumala hantavirus associated with its rodent host, Myodes glareolus. Evol Appl. 2015;8:545–59.
62. Zeimes CB, Quoilin S, Henttonen H, Lyytikäinen O, Vapalahti O, Reynes JM, et al. Landscape and regional environmental analysis of the spatial distribution of hantavirus human cases in Europe. Front Public Health. 2015;3. https://doi.org/10.3389/fpubh.2015.00054/full. Available from: https://www.frontiersin.org/journals/public-health/articles/. Cited 2024 Jun 28.
63. Binder F, Drewes S, Imholt C, Saathoff M, Below DA, Bendl E, et al. Heterogeneous Puumala orthohantavirus situation in endemic regions in Germany in summer 2019. Transbound Emerg Dis. 2020;67:502–9.
64. Khismatullina NA, Karimov MM, Khaertynov KS, Shuralev EA, Morzunov SP, Khaertynova IM, et al. Epidemiological dynamics of nephropathia epidemica in the Republic of Tatarstan, Russia, during the period of 1997–2013. Epidemiol Infect. 2016;144:618–26.
65. Voutilainen L, Kallio ER, Niemimaa J, Vapalahti O, Henttonen H. Temporal dynamics of Puumala hantavirus infection in cyclic populations of bank voles. Sci Rep. 2016;6:21323.
66. Jeske K, Jacob J, Drewes S, Pfeffer M, Heckel G, Ulrich RG, et al. Hantavirus– *Leptospira* coinfections in small mammals from central Germany. Epidemiol Infect. 2021;149:e97.
67. Khalil H, Ecke F, Evander M, Bucht G, Hörnfeldt B. Population dynamics of bank voles predicts human Puumala hantavirus risk. EcoHealth. 2019;16:545–57.
68. Linard C, Tersago K, Leirs H, Lambin EF. Environmental conditions and Puumala virus transmission in Belgium. Int J Health Geogr. 2007;6:55.
69. Reil D, Rosenfeld UM, Imholt C, Schmidt S, Ulrich RG, Eccard JA, et al. Puumala hantavirus infections in bank vole populations: host and virus dynamics in Central Europe. BMC Ecol. 2017;17:9.

70. Voutilainen L, Savola S, Kallio ER, Laakkonen J, Vaheri A, Vapalahti O, et al. Environmental change and disease dynamics: effects of intensive forest management on puumala hantavirus infection in boreal bank vole populations. PLoS One. 2012;7:e39452 Vijaykrishna D, editor.
71. Wang YXG, Voutilainen L, Aminikhah M, Helle H, Huitu O, Laakkonen J, et al. The impact of wildlife and environmental factors on hantavirus infection in the host and its translation into human risk. Proc R Soc B Biol Sci. 2023;290:20222470.
72. Monchatre-Leroy E, Murri S, Castel G, Calavas D, Boué F, Henaux V, et al. First insights into Puumala orthohantavirus circulation in a rodent population in Alsace. France Zoonoses Public Health. 2018;65:540–51.
73. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71.
74. Tersago K, Crespin L, Verhagen R, Leirs H. Impact of Puumala virus infection on maturation and survival in bank voles: a capture-mark-recapture analysis. J Wildl Dis. 2012;48:148–56.
75. Madai M, Németh V, Oldal M, Horváth G, Herczeg R, Kelemen K, et al. Temporal dynamics of two pathogenic hantaviruses among rodents in Hungary. Vector-Borne Zoonotic Dis. 2020;20:212–21.
76. Kallio ER, Voutilainen L, Vapalahti O, Vaheri A, Henttonen H, Koskela E, et al. Endemic hantavirus infection impairs the winter survival of its rodent host. Ecology. 2007;88:1911–6.
77. Obiegala A, Albrecht C, Dafalla M, Drewes S, Oltersdorf C, Turni H, et al. Leptospira spp. in small mammals from areas with low and high human hantavirus incidences in South-West Germany. Vector-Borne Zoonotic Dis. 2017;17:312–8.
78. Salvador AR, Guivier E, Xuéreb A, Chaval Y, Cadet P, Poulle ML, et al. Concomitant influence of helminth infection and landscape on the distribution of Puumala hantavirus in its reservoir. Myodes glareolus BMC Microbiol. 2011;11:30.
79. Rizzoli A, Tagliapietra V, Rosà R, Hauffe HC, Marini G, Voutilainen L, et al. Recent increase in prevalence of antibodies to Dobrava-Belgrade virus (DOBV) in yellow-necked mice in northern Italy. Epidemiol Infect. 2015;143:2241–4.
80. Kallio ER, Helle H, Koskela E, Mappes T, Vapalahti O. Age-related effects of chronic hantavirus infection on female host fecundity. J Anim Ecol. 2015;84:1264–72.
81. Augot D, Muller D, Demerson JM, Boué F, Caillot C, Cliquet F. Dynamics of Puumala virus infection in bank voles in Ardennes department (France). Pathol Biol. 2006;54:572–7.
82. Augot D, Sauvage F, Boue F, Bouloy M, Artois M, Demerson JM, et al. Spatial and temporal patterning of bank vole demography and the epidemiology of the Puumala hantavirus in northeastern France. Epidemiol Infect. 2008;136:1638–43.
83. Deter J, Bryja J, Chaval Y, Galan M, Henttonen H, Laakkonen J, et al. Association between the DQA MHC class II gene and Puumala virus infection in Myodes glareolus, the bank vole. Infect Genet Evol. 2008;8:450–8.
84. Deter J, Chaval Y, Galan M, Gauffre B, Morand S, Henttonen H, et al. Kinship, dispersal and hantavirus transmission in bank and common voles. Arch Virol. 2008;153:435–44.
85. Dobly A, Yzoard C, Cochez C, Ducoffre G, Aerts M, Roels S, et al. Spatiotemporal dynamics of Puumala hantavirus in suburban reservoir rodent populations. J Vector Ecol. 2012;37:276–83.
86. Heroldová M, Pejčoch M, Bryja J, Jánová E, Suchomel J, Tkadlec E. Tula virus in populations of small terrestrial mammals in a rural landscape. Vector-Borne Zoonotic Dis. 2010;10:599–603.
87. Heyman P, Mele RV, Smajlovic L, Dobly A, Cochez C, Vandenvelde C. Association between habitat and prevalence of hantavirus infections in bank voles (Myodes glareolus) and wood mice (Apodemus sylvaticus). Vector-Borne Zoonotic Dis. 2009;9:141–6.
88. Kallio ER, Begon M, Henttonen H, Koskela E, Mappes T, Vaheri A, et al. Hantavirus infections in fluctuating host populations: the role of maternal antibodies. Proc R Soc B Biol Sci. 2010;277:3783–91.
89. Tersago K, Schreurs A, Linard C, Verhagen R, Van Dongen S, Leirs H. Population, environmental, and community effects on local bank vole (Myodes glareolus) Puumala virus infection in an area with low human incidence. Vector-Borne Zoonotic Dis. 2008;8:235–44.
90. Tersago K, Verhagen R, Leirs H. Temporal variation in individual factors associated with hantavirus infection in bank voles during an epizootic: implications for Puumala virus transmission dynamics. Vector-Borne Zoonotic Dis. 2011;11:715–21.
91. Khalil H, Ecke F, Evander M, Hörnfeldt B. Selective predation on hantavirus-infected voles by owls and confounding effects from landscape properties. Oecologia. 2016;181:597–606.
92. Kurucz K, Madai M, Bali D, Hederics D, Horváth G, Kemenesi G, et al. Parallel survey of two widespread renal syndrome-causing zoonoses: leptospira spp. and hantavirus in urban environment, Hungary. Vector-Borne Zoonotic Dis. 2018;18:200–5.
93. Tadin A, Turk N, Korva M, Margaletić J, Beck R, Vucelja M, et al. Multiple co-infections of rodents with hantaviruses, *Leptospira,* and *Babesia* in Croatia. Vector-Borne Zoonotic Dis. 2012;12:388–92.
94. Tadin A, Tokarz R, Markotić A, Margaletić J, Turk N, Habuš J, et al. Molecular survey of zoonotic agents in rodents and other small mammals in Croatia. Am J Trop Med Hyg. 2016;94:466–73.
95. Korva M, Duh D, Saksida A, Trilar T, Avšič-Županc T. The hantaviral load in tissues of naturally infected rodents. Microbes Infect. 2009;11:344–51.
96. Borucki MK, Boone JD, Rowe JE, Bohlman MC, Kuhn EA, DeBaca R, et al. Role of maternal antibody in natural infection ofPeromyscus maniculatus with Sin Nombre Virus. J Virol. 2000;74:2426–9.
97. Dohmae K, Nishimune Y. Maternal transfer of hantavirus antibodies in rats. Comp Med. 1998;48:395–7.
98. Hofmann J, Führer A, Bolz M, Waldschläger-Terpe J, Meier M, Lüdders D, et al. Hantavirus infections by Puumala or Dobrava-Belgrade virus in pregnant women. J Clin Virol. 2012;55:266–9.
99. Schmaljohn C, Hjelle B. Hantaviruses: a global disease problem. Emerg Infect Dis. 1997;3:95–104.
100. Ecke F, Angeler DG, Magnusson M, Khalil H, Hörnfeldt B. Dampening of population cycles in voles affects small mammal community structure, decreases diversity, and increases prevalence of a zoonotic disease. Ecol Evol. 2017;7:5331–42.
101. Kallio ER, Henttonen H, Koskela E, Lundkvist Å, Mappes T, Vapalahti O. Maternal antibodies contribute to sex-based difference in hantavirus transmission dynamics. Biol Lett. 2013;9:20130887.
102. Khalil H, Ecke F, Evander M, Magnusson M, Hörnfeldt B. Declining ecosystem health and the dilution effect. Sci Rep. 2016;6:31314.
103. Papa A, Rogozi E, Velo E, Papadimitriou E, Bino S. Genetic detection of hantaviruses in rodents, Albania: Hantaviruses in rodents. Albania J Med Virol. 2016;88:1309–13.
104. Schmidt S, Reil D, Jeske K, Drewes S, Rosenfeld UM, Fischer S, et al. Spatial and temporal dynamics and molecular evolution of Tula orthohantavirus in German vole populations. Viruses. 2021;13:1132.
105. Mills SC, Grapputo A, Jokinen I, Koskela E, Mappes T, Poikonen T. Fitness trade-offs mediated by immunosuppression costs in a small mammal. Evol Int J Org Evol. 2010;64:166–79.
106. Nelson RJ, Demas GE, Klein SL, Kriegsfeld LJ. Seasonal patterns of stress, immune function, and disease. New York, NY, US: Cambridge University Press; 2002. p. xv, 291.
107. Guivier E, Galan M, Chaval Y, Xuéreb A, Ribas Salvador A, Poulle ML, Voutilainen L, Henttonen H, Charbonnel N, Cosson JF. Landscape genetics highlights the role of bank vole metapopulation dynamics in the epidemiology of Puumala hantavirus. Mol Ecol. 2011;20(17):3569–83. https://doi.org/10.1111/j.1365-294X.2011.05199.x.
108. Monchatre-Leroy E, Sauvage F, Boué F, Augot D, Marianneau P, Hénaux V, et al. Seroprevalence and incidence of Puumala orthohantavirus in its bank vole (Myodes glareolus) host population in northeastern France: between-site and seasonal variability. Epidemics. 2022;40:100600.
109. Hughes NK, Helsen S, Tersago K, Leirs H. Puumala hantavirus infection alters the odour attractiveness of its reservoir host. Oecologia. 2014;176:955–63.
110. Charbonnel N, Pagès M, Sironen T, Henttonen H, Vapalahti O, Mustonen J, et al. Immunogenetic factors affecting susceptibility of humans and rodents to hantaviruses and the clinical course of hantaviral disease in humans. Viruses. 2014;6:2214–41.
111. Guivier E, Galan M, Salvador AR, Xuéreb A, Chaval Y, Olsson GE, et al. *Tnf-a* expression and promoter sequences reflect the balance of tolerance/resistance to Puumala hantavirus infection in European bank vole populations. Infect Genet Evol. 2010;10:1208–17.
112. Guivier E, Galan M, Henttonen H, Cosson J-F, Charbonnel N. Landscape features and helminth co-infection shape bank vole

immunoheterogeneity, with consequences for Puumala virus epidemiology. Heredity. 2014;112:274–81.
113. Rohfritsch A, Galan M, Gautier M, Gharbi K, Olsson G, Gschloessl B, et al. Preliminary insights into the genetics of bank vole tolerance to Puumala hantavirus in Sweden. Ecol Evol. 2018;8:11273–92.
114. Sipari S, Haapakoski M, Klemme I, Palme R, Sundell J, Ylönen H. Population sex-ratio affecting behavior and physiology of overwintering bank voles (Myodes glareolus). Physiol Behav. 2016;159:45–51.
115. Dubois A, Castel G, Murri S, Pulido C, Pons J-B, Benoit L, et al. Bank vole immunoheterogeneity may limit Nephropatia Epidemica emergence in a French non-endemic region. Parasitology. 2018;145:393–407.
116. Dubois A, Galan M, Cosson J-F, Gauffre B, Henttonen H, Niemimaa J, et al. Microevolution of bank voles (*Myodes glareolus*) at neutral and immune-related genes during multiannual dynamic cycles: consequences for *Puumala hantavirus* epidemiology. Infect Genet Evol. 2017;49:318–29.
117. Drewes S, Ali HS, Saxenhofer M, Rosenfeld UM, Binder F, Cuypers F, et al. Host-associated absence of human Puumala virus infections in Northern and Eastern Germany. Emerg Infect Dis. 2017;23:83–6.
118. Bjedov L, Svoboda P, Tadin A, Habuš J, Štritof Z, Labaš N, et al. Influence of beech mast on small rodent populations and hantavirus prevalence in Nacional Park „Plitvice lakes" and Nature Park „Medvednica". Šumar List. 2016;140:455–63.
119. Olsson GE, White N, Hjältén J, Ahlm C. Habitat factors associated with bank voles (Clethrionomys glareolus) and concomitant hantavirus in Northern Sweden. Vector-Borne Zoonotic Dis. 2005;5:315–23.
120. Bujnoch FM, Reil D, Drewes S, Rosenfeld UM, Ulrich RG, Jacob J, et al. Small mammal community composition impacts bank vole (Clethrionomys glareolus) population dynamics and associated seroprevalence of Puumala orthohantavirus. Integr Zool. 2024;19:52–65.
121. Olsson GE, Ahlm C, Elgh F, Verlemyr A-C, White N, Juto P, et al. Hantavirus antibody occurrence in bank voles (Clethrionomys glareolus) during a vole population cycle. J Wildl Dis. 2003;39:299–305.
122. Sipari S, Khalil H, Magnusson M, Evander M, Hörnfeldt B, Ecke F. Climate change accelerates winter transmission of a zoonotic pathogen. Ambio. 2022;51:508–17.
123. Thoma B, Müller J, Bässler C, Georgi E, Osterberg A, Schex S, et al. Identification of factors influencing the puumala virus seroprevalence within its reservoir in a montane forest environment. Viruses. 2014;6:3944–67.
124. Andreassen HP, Sundell J, Ecke F, Halle S, Haapakoski M, Henttonen H, et al. Population cycles and outbreaks of small rodents: ten essential questions we still need to solve. Oecologia. 2021;195:601–22.
125. Hanski I, Henttonen H. Population Cycles of Small Rodents in Fennoscandia. In: Berryman A, editor. Popul Cycles Case Trophic Interact. Oxford University Press; 2002. p. 0. https://doi.org/10.1093/oso/9780195140989.003.0007. Cited 2024 Aug 28.
126. Korpela K, Helle P, Henttonen H, Korpimäki E, Koskela E, Ovaskainen O, et al. Predator–vole interactions in northern Europe: the role of small mustelids revised. Proc R Soc B Biol Sci. 2014;281:20142119.
127. Oli MK. Population cycles in voles and lemmings: state of the science and future directions. Mammal Rev. 2019;49:226–39.
128. Vaheri A, Henttonen H, Mustonen J. Hantavirus research in Finland: highlights and perspectives. Viruses. 2021;13:1452.
129. Pinot A, Barraquand F, Tedesco E, Lecoustre V, Bretagnolle V, Gauffre B. Density-dependent reproduction causes winter crashes in a common vole population. Popul Ecol. 2016;58:395–405.
130. Schmidt-Chanasit J, Essbauer S, Petraityte R, Yoshimatsu K, Tackmann K, Conraths FJ, et al. Extensive host sharing of Central European Tula Virus. J Virol. 2010;84:459–74.
131. Khalil H, Olsson G, Magnusson M, Evander M, Hörnfeldt B, Ecke F. Spatial prediction and validation of zoonotic hazard through micro-habitat properties: where does Puumala hantavirus hole – up? BMC Infect Dis. 2017;17:523.
132. Magnusson M, Ecke F, Khalil H, Olsson G, Evander M, Niklasson B, et al. Spatial and temporal variation of hantavirus bank vole infection in managed forest landscapes. Ecosphere. 2015;6:art163.
133. Ostfeld RS, Keesing F. Effects of host diversity on infectious disease. Annu Rev Ecol Evol Syst. 2012;43:157–82.
134. Hardestam J, Simon M, Hedlund KO, Vaheri A, Klingström J, Lundkvist Å. Ex vivo stability of the rodent-borne hantaan virus in comparison to that of arthropod-borne members of the Bunyaviridae family. Appl Environ Microbiol. 2007;73:2547–51.
135. Guzzetta G, Tagliapietra V, Perkins SE, Hauffe HC, Poletti P, Merler S, et al. Population dynamics of wild rodents induce stochastic fadeouts of a zoonotic pathogen. Fenton A, editor. J Anim Ecol. 2017;86:451–9.
136. Escutenaire S, Chalon P, de Jaegere F, Karelle-Bui L, Mees G, Brochier B, et al. Behavioral, physiologic, and habitat influences on the dynamics of Puumala virus infection in bank voles (Clethrionomys glareolus). Emerg Infect Dis. 2002;8:930–6.
137. Armansin NC, Stow AJ, Cantor M, Leu ST, Klarevas-Irby JA, Chariton AA, et al. Social barriers in ecological landscapes: the social resistance hypothesis. Trends Ecol Evol. 2020;35:137–48.

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