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Hantavirus infections in fluctuating host populations: the role of maternal antibodies

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Infected females may transfer maternal antibodies (MatAbs) to their offspring, which may then be transiently protected against infections the mother has encountered. However, the role of maternal protection in infectious disease dynamics in wildlife has largely been neglected. Here, we investigate the effects of Puumala hantavirus (PUUV)-specific MatAbs on PUUV dynamics, using 7 years' data from a cyclic bank vole population in Finland. For the first time to our knowledge, we partition seropositivity data from a natural population into separate dynamic patterns for MatAbs and infection. The likelihood of young of the year carrying PUUV-specific MatAbs during the breeding season correlated positively with infection prevalence in the overwintered parent population in the preceding spring. The probability of PUUV infection varied between seasons (highest in spring, lowest in late summer) and depended on population structure, but was also, in late autumn, notably, negatively related to summer MatAb prevalence, as well as to infection prevalence earlier in the breeding season. Hence, our results suggest that high infection prevalence in the early breeding season leads to a high proportion of transiently immune young individuals, which causes delays in transmission. This suggests, in turn, that MatAb protection has the potential to affect infection dynamics in natural populations.

Keywords: bank vole; infection transmission; maternal antibody; Puumala hantavirus; transient immunity

1. INTRODUCTION

A common expectation for infectious diseases transmitted directly (without a vector) and horizontally (but not sexually or congenitally) is that infection prevalence will increase with host abundance, and there will be a threshold host density below which the pathogen will not persist (e.g. Mills *et al.* 1999; McCallum *et al.* 2001; Begon *et al.* 2002; Hudson *et al.* 2002; Lloyd-Smith *et al.* 2005), but this expectation is not always borne out. Davis *et al.* (2005) and Adler *et al.* (2008) have recently reviewed studies on the relationship between the prevalence of rodent-borne diseases and host abundance. For example, studies on hantaviruses (genus *Hantavirus*, family *Bunyaviridae*), carried by specific rodent or insectivore hosts and causing several human diseases in Eurasia and the Americas (Vaheri *et al.* 2008), have shown great variation in this relationship. Seroprevalence for Sin Nombre hantavirus has shown either a positive, a negative or no significant relationship with the abundance of its main host, the deer mouse, *Peromyscus maniculatus* (Davis *et al.* 2005; Adler *et al.* 2008), while the relationship between Puumala hantavirus (PUUV) seroprevalence and the density of its host, the bank vole (*Myodes glareolus*), has sometimes been positive (Brummer-Korvenkontio *et al.* 1982; Niklasson *et al.* 1995; Linard *et al.* 2007), but in other cases has shown no clear correlation (Escutenaire *et al.* 2000; Sauvage *et al.* 2002; Olsson *et al.* 2005; Linard *et al.* 2007; Tersago *et al.* 2008).

Often, the absence of a positive correlation has been associated with the relationship being followed over different seasons (Mills *et al.* 1999; Davis *et al.* 2005). Relatively few studies have followed PUUV in bank vole populations over the course of a breeding season, but in those cases, the seroprevalence has peaked in spring, at the beginning of breeding season, and decreased, together with increasing host population density, during the breeding season (e.g. Verhagen *et al.* 1986; Niklasson *et al.* 1995; Escutenaire *et al.* 2000; Sauvage *et al.* 2002). Similar results have also been reported from other hantavirus–host systems (Calisher *et al.* 1999, 2005; Douglass *et al.* 2001). A high influx of newborns during the breeding season may decrease hantavirus prevalence, and this 'juvenile dilution effect' has been proposed to be the mechanism through which the expected positive relationship between prevalence and abundance is lacking or delayed (Mills *et al.* 1999; Davis *et al.* 2005; Adler *et al.* 2008).

Newborn individuals may, however, induce an additional effect in the relationship between infection

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prevalence and host abundance. Infected females commonly transfer maternal antibodies (MatAbs) to their offspring, providing temporary protection against the infection(s) the mother has encountered (Grindstaff *et al.* 2003; Bouliner & Staszewski 2008; Hasselquist & Nilsson 2009). Hantavirus-infected female rodents transfer MatAbs to their offspring *in utero* and during lactation (Dohmae *et al.* 1993), and the temporary immunity against PUUV lasts for up to 80 days in bank voles (Gavrilovskaya *et al.* 1990; Kallio *et al.* 2006*a*), a relatively long period when compared with their average lifespan, three to five months (Innes & Millar 1994). The presence of MatAbs among young individuals has been acknowledged in several hantavirus studies (e.g. Mills *et al.* 1997, 1999; Borucki *et al.* 2000; Escutenaire *et al.* 2000; Calisher *et al.* 2007; Dearing *et al.* 2009). However, their role in infection dynamics has largely been neglected in studies on hantaviruses and on natural populations generally, despite recent acknowledgement of the potential of MatAbs to influence disease dynamics in wildlife populations through altering the amount of susceptible individuals (Gasparini *et al.* 2001; Staszewski *et al.* 2007; Bouliner & Staszewski 2008), and their recent inclusion in theoretical studies (Fouchet *et al.* 2006, 2007, 2008).

Here, we test whether the transmission dynamics of infectious pathogens in our wildlife host populations are influenced by the presence of MatAb-protected individuals in the population. We hypothesize that the proportion of MatAb-protected young individuals in the population should itself be determined by earlier infection prevalences (the proportion of the population, and specifically females that are infected) in the host population. Therefore, high infection prevalence at the beginning of a breeding season may lead to high numbers of maternally protected young individuals during the breeding season, delaying the influx of susceptible individuals until the maternal protection disappears. Consequently, the expected positive relationship between host abundance and infection prevalence by the end of the breeding season may be lacking despite an earlier increase in host abundance. Here, we test these ideas using a 7 year dataset on PUUV and bank vole dynamics in Central Finland. For the first time to our knowledge, we partition data on seropositivity from a natural population into separate dynamic patterns for MatAbs and infection. Our aim is to elaborate seasonal and host density-dependent patterns in PUUV infection dynamics, and in particular to evaluate the evidence on effects of MatAbs on the transmission of PUUV.

2. MATERIAL AND METHODS

(a) *Study species*

PUUV is carried by the bank vole (Brummer-Korvenkontio *et al.* 1980), in which the infection does not cause clinical illness (Gavrilovskaya *et al.* 1990; Bernshtein *et al.* 1999) but may have deleterious fitness consequences (Kallio *et al.* 2006*a*, 2007). The infection is chronic (Meyer & Schmaljohn 2000), transmission is horizontal (e.g. Niklasson *et al.* 1995; Kallio *et al.* 2006*b*) and the virus is excreted during the first two months after becoming infected (Hardestam *et al.* 2008). Also, the transmission may take place indirectly via a contaminated environment, where the shed virus remains infectious for prolonged time periods depending on the temperature (Kallio *et al.* 2006*b*).

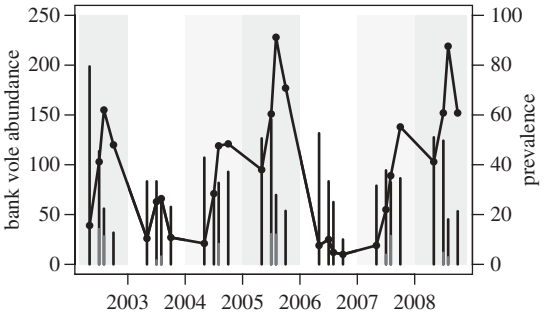

Figure 1. Bank vole abundance (black line; dots indicate the trappings), PUUV seroprevalences (black bars) and MatAb prevalences (grey bars) 2002–2008 in the Konnevesi area in Central Finland. (Note that infection prevalence = seroprevalence − MatAb prevalence.) Phases of the cycles are indicated with different background colours (white, low phase; light grey, increase phase; darker grey, peak phase). The time labels are located at the beginning of the year.

The bank vole is a common species in most of Europe. Seasonal fluctuations with occasional mast-driven outbreaks characterize the populations of continental temperate Europe (e.g. Tersago *et al.* 2009), but in northern Fennoscandia, bank vole populations show multi-annual cyclic fluctuations (Henttonen *et al.* 1985). The length of the vole cycle varies from 3 to 5 years, being synchronous over large areas and determined predominantly by specialist mammalian predators and the voles' winter food resources (Hansson & Henttonen 1988; Hanski *et al.* 1991; Korpimäki *et al.* 2005; Huitu *et al.* 2007).

(b) *Data collection*

Voles were trapped in the Konnevesi area in Central Finland (62°8379′ N, 26°8209′ E) four times per year from May 2002 to October 2008. The first trapping ('spring') was carried out early in the breeding season in May. The second ('summer') was conducted typically during the first week of July, in the middle of the breeding season. The third ('late summer') was typically in late breeding season in late August, and the fourth ('late autumn') was carried out in late October to early November, after the breeding season. Based on these annual trappings, monthly bank vole incidences were computed using linear interpolation (for details and justification, see Kallio *et al.* 2009). The bank vole populations in the study area fluctuate cyclically, and this study period covered three 'peak' years (2002, 2005 and 2008), two 'crash/low' years (2003 and 2006) and two 'increase' years (2004 and 2007) (figure 1) (Kallio *et al.* 2009). The trappings were carried out in 20 permanent trapping sites and in each site four Ugglan Special multiple-capture live traps (Grahnab, Hillerstorp, Sweden) were set over two nights (altogether 160 trapping nights in each trapping session).

Captured voles were taken to a laboratory where their sex, breeding status and body mass were recorded and blood or tissue samples taken (Kallio *et al.* 2009) for antibody detection using immunofluorescence assay (IFA) (Vapalahti *et al.* 1995). Pregnant females gave birth in the laboratory and their postpartum weight was measured. To diminish the influence of pregnancy on body mass, the postpartum

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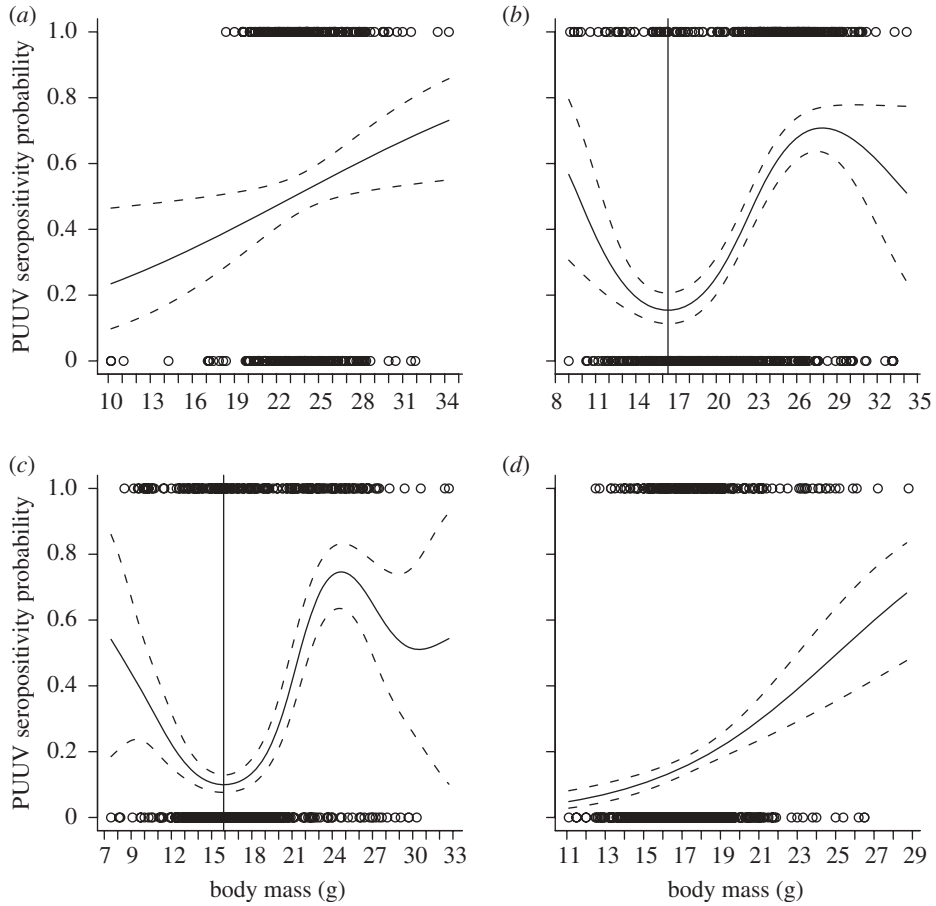

Figure 2. Predicted probability, at the individual level, of being PUUV antibody positive in relation to body mass (g) in (*a*) spring, (*b*) summer, (*c*) late summer and (*d*) late autumn. Cut-off weight values below which the seropositive result is considered as maternal antibody positivity are indicated with vertical lines. In summer (*b*), the cut-off value was 16.4 g and in late summer (*c*), the cut-off was 15.9 g. Observed data are shown with dots (PUUV seropositive = 1, PUUV seronegative = 0).

weight (when it was lower than the trapping weight) was used in the analyses. As the exact age of the trapped individuals could not be determined, we used body mass as a proxy for age (see, e.g. Telfer *et al.* 2008).

Seasonally reproducing rodents may mature at different ages and weights at different cyclic phases. Maturity and related hormonal and immunological changes may, therefore, sometimes explain, better than weight does, the probabilities of being infected. Unfortunately, reproductive status could not be recorded for all animals. Therefore, to test for the effects of maturity, we used subsets of the data where maturity status had been determined, distinguishing two categories, immature versus mature, where 'mature' describes an animal that is or has previously been reproductively active.

(c) *Data analyses*

(i) *Separation between maternal antibodies and genuine infection*

Because we used a serological diagnostic method and all individuals were sampled only once, it was not feasible to distinguish individuals that carried MatAbs directly from those that were infected (both detected as seropositive by IFA). However, we analysed the probability of being seropositive in relation to individuals' weight, using a generalized additive mixed model approach (gamm4 function in gamm4 library in R software, available under GNU licence at http://www.r-project.org) with binomial distributions and with the models fitted using the Laplace approximation method. Splines were fitted using thin plate regression, and the model with the lowest *k*-value (least complex) within 2 of the model with the lowest Akaike information criterion (AIC) value was selected. To control for the correlation among individuals that were captured at the same trapping site in the same trapping session, *trapping year* ∗ *trapping site* was included as a random effect in the models. These models were fitted separately for each trapping season. This allowed us to detect any tendency for seroprevalence first to decline with increasing weight (declining prevalence of MatAbs) and then to increase (increasing prevalence of infection). When this relationship indicated a U-shaped curve, the weight at the lowest probability was used as a cut-off value (§3, figure 2): when a seropositive individual's weight was below the cut-off, the individual was recorded as carrying MatAbs, whereas with weight above the cut-off, the individual was recorded as genuinely infected. In principle, young but genuinely infected individuals may be misclassified as having MatAbs by this procedure, and the oldest individuals with MatAbs misclassified as infected. However, the effects of this should be negligible, because the period in an individual's life of possible misclassification is short—from the age of *ca* 45 days (first appearance of antibody following weaning and infection) to *ca* 60 days (by which time 95% have lost MatAb; Kallio *et al.* 2006*a*)—and so the numbers will themselves be small. Consequently, infection prevalence has been calculated as the number infected (PUUV seropositives–MatAb positives) divided by the total number of animals tested. MatAb prevalence has been calculated as the number of MatAb positives also divided by the total number of

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animals tested, since it is used as a possible explanatory variable for infection prevalence in the whole population.

(ii) *Probability of carrying MatAbs*

The likelihood of carrying MatAbs was investigated among the individuals with body mass below or equal to the detected cut-off value (above). The presence of MatAb (binomial response variable) was studied in relation to gender, weight, the trapping season (summer or late summer, i.e. when MatAb-positive individuals were detected, see §3) and the infection prevalence in the bank vole population during the spring and summer trappings. Instead of using infection prevalence among breeding females only, we used infection prevalence in the whole population because the numbers of captured females were lower than males, and because there was no significant difference in the mean infection prevalence of males and females in spring and summer. In addition, current bank vole abundance (N_t) and bank vole abundances two (N_{t-2}), four (N_{t-4}) and six (N_{t-6}) months earlier (natural log-transformed data) were used as predictors. The analyses were carried out using a generalized linear mixed model (GLMM) with a logit link function and binomial errors, and the models were fitted using the Laplace approximation method (lmer function in lme4 library in the R software package). To control for the correlation among individuals that were captured at the same trapping site in the same trapping session, *trapping year* $*$ *trapping site* was included as a random effect in the models. Starting from a full model, terms were omitted if they did not reduce the AIC by more than two units when included (see electronic supplementary material, table S1, for the model selection) (Burnham & Anderson 2002).

(iii) *Probability of being PUUV-infected*

To study the probability of an individual being genuinely infected, we first analysed whether there was any seasonal pattern in the dependent variable using the entire dataset with a GLMM (as described above) with *trapping session* $*$ *trapping site* as a random effect in the models. Because the trapping season had a significant influence on the probability of being infected (§3), and because our unequal trapping interval meant that explanatory variables referring to preceding trapping sessions were not comparable between seasons (electronic supplementary material, table S2), we analysed the four trapping seasons separately.

For each of the four trapping seasons, the probability of being genuinely infected was analysed in a similar way. First we fitted a full model using a generalized linear model (see above) that included population-level predictors—phase of the population cycle, current bank vole abundance (N_t), bank vole abundance during preceding 12 months ($N_{t-1}, \ldots, N_{t-12}$) (natural log-transformed data) and PUUV infection prevalence during the three preceding trapping sessions (electronic supplementary material, table S2)—and the individual-level predictors weight and sex. Preceding MatAb prevalences were not included at this stage (but see below) because of the functional linkage between these and the infection prevalences that precede them. Second, we restricted the full model, including two-way interactions, using the stepAIC procedure (see above). The selected model was then applied to a GLMM (see above) where the best model (the simplest model within two units of the lowest AIC value; electronic supplementary material, table S1) was selected manually. Then, the effect of maturity status was tested using the subset of data where maturity status had been determined, where it was applied as a predictor to the best model and additional model selection carried out (results provided only if this improved the final model).

Finally, we carried out separate analyses to clarify the effect of MatAb prevalence in the population (see above) during the breeding season on the probability of being infected in late autumn using GLMM as explained above. As the effects of MatAbs on infection prevalence are likely to be observable only after a delay (since their effect is an absence of infection seropositives that would otherwise have appeared), this effect is likely to be seen only in autumn. Hence, the MatAb prevalence in summer and late summer was used as covariates to predict the probability of being infected in late autumn. Also, individual-level variables (sex and weight) were included in the model selection procedure (electronic supplementary material, table S1).

3. RESULTS

Bank vole abundance varied both seasonally and multi-annually (figure 1). Of 2576 animals captured, 2476 were examined for the presence of anti-PUUV antibodies, and of these, 818 (33.3%; 95% CI: 31.2–34.5) were PUUV seropositive. Seroprevalence varied between trapping sessions from 10 to 80 per cent, being highest in spring (mean prevalence $\pm$ s.e.: 48.8 $\pm$ 6.1%), decreasing through the breeding season (summer: 40.9 $\pm$ 4.0%; late summer: 26.3 $\pm$ 2.0%) and being lowest in late autumn (22.9 $\pm$ 3.8%) (figure 1).

(a) *Maternal antibodies versus genuine infection*

PUUV seropositivity was studied in relation to weight to distinguish individuals that were carrying MatAbs from those that were genuinely infected (§2). In spring, most individuals were over-wintered and therefore too old to carry MatAbs. In support of this, all seropositive individuals weighed 18.4 g or more and the model describing the probability of being PUUV seropositive in relation to weight did not show any indication of the presence of MatAb-positive individuals (figure 2*a*). Hence, in spring, all seropositive individuals were classified as infected. In summer, the model was markedly nonlinear and indicated that the lowest likelihood of being seropositive was at a weight of 16.4 g (figure 2*b*). Hence, only seropositive individuals that weighed more than this cut-off were considered to be infected: 40 out of 262 seropositive individuals in summer were considered to be carrying MatAbs. In late summer, the model again indicated the presence of MatAb-positive individuals, and the lowest probability of being seropositive was at a weight of 15.9 g (figure 2*c*), and consequently, 70 out of 218 seropositives were considered to be carrying MatAbs. In late autumn (*ca* eight weeks after the end of the breeding season), there were no indications of the presence of MatAb-positive individuals (figure 2*d*) and therefore all positive records were considered to be a result of genuine infection.

(b) *Probability of carrying maternal antibodies*

During the breeding season, the probability that an individual was carrying MatAbs was positively related to the infection prevalence in spring (figure 3*a*) and was higher in summer (July) than in late summer (August) (table 1).

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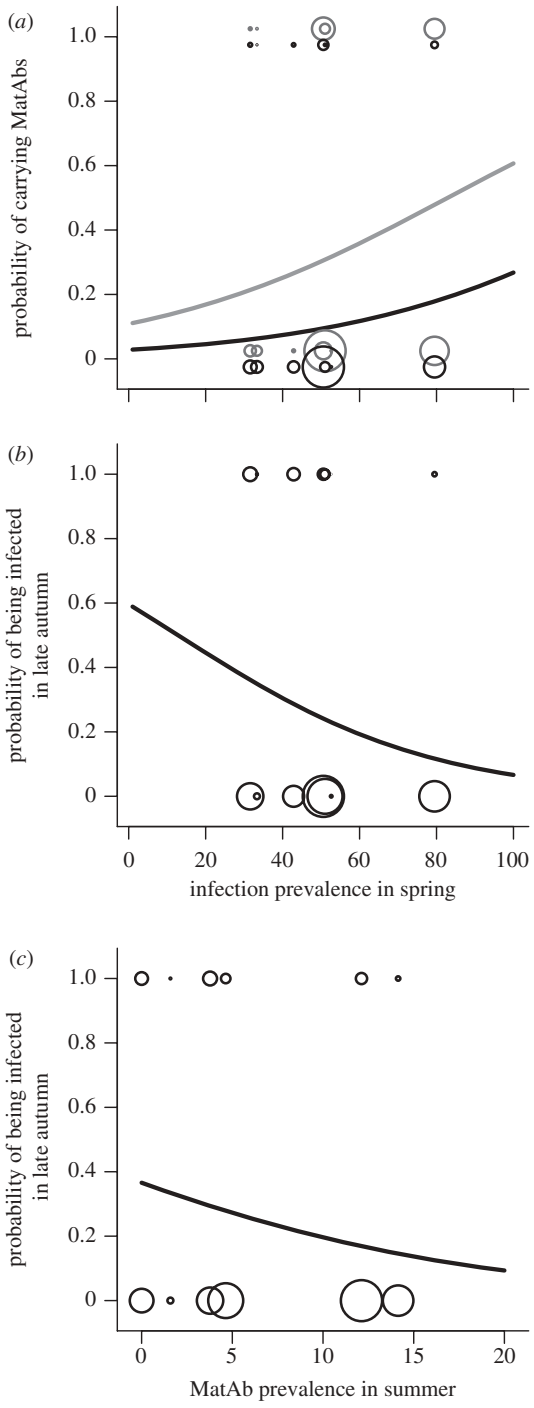

Figure 3. Predicted probability, at the individual level, of (*a*) carrying maternal antibodies in summer (grey) and late summer (black) in relation to infection prevalence in the host population in the preceding spring; and being PUUV-infected in late autumn in relation to (*b*) infection prevalence in the host population in spring, and (*c*) MatAb prevalence in summer. Corresponding observed values are shown (circles; (*a*) 0 = MatAb negative or (*b*,*c*) uninfected, (*a*) 1 = MatAb positive or (*b*,*c*) infected). The size of the circles is determined by the numbers of overlaying observations. Predictions are based on the best selected GLMM and are made in (*a*) for an individual with a body mass of 10 g and at fixed past and current bank vole abundances (both approximately 55 individuals, natural log-transformed value = 4), and in (*b*) and (*c*) for an individual with a body mass of 20 g. GLMMs do not allow reliable confidence intervals to be applied to these curves, but standard errors for the model parameter estimates for (*a*–*c*) are given in table 1, table 3 and in the text, respectively.

Also, current bank vole abundance was positively related, and bank vole abundance six months before negatively related, to the probability of carrying MatAbs (table 1).

(c) *Probability of being PUUV-infected*

Overall, the probability of PUUV infection was highest in spring and lowest in late summer (table 2), depending on different variables in different trapping seasons (best selected models and parameter estimates in table 3). Of the individual characteristics, weight was included in all models, and individuals' probability of being infected increased with weight. Sex was selected in the summer and late summer models, and there were interactions with weight, indicating that prevalence tended to be higher in males, especially heavier males (table 3). Breeding status was selected only in the late summer model, where the probability of being infected was higher in the sexually mature voles (table 3).

Of the population-level variables, few were included in the best models (table 3). In late autumn, the infection probability was negatively related to the PUUV infection prevalence in the populations in the previous spring, approximately five and a half months earlier (table 3 and figure 3*b*).

The effect of earlier MatAb prevalence on the infection status in late autumn was studied separately. The probability of being infected was negatively related to the MatAb prevalence in summer, approximately three and a half months earlier (predictors in the selected GLMM were *MatAb prevalence in summer and weight*; parameter estimate for *MatAb prevalance in summer* (logit scale) $\beta = -0.086$, s.e. 0.034, $z = -2.571$, $p = 0.010$; figure 3*c*).

4. DISCUSSION

Previously, we have shown the protective effect of MatAbs against PUUV in bank voles at the individual level (Kallio *et al.* 2006*a*). Here, we hypothesised that young host individuals may have a significant effect on infection dynamics in wildlife host populations through MatAb protection, and this has been supported.

PUUV infection prevalence was highest in spring and decreased during the breeding season. In spring, the population consisted mostly of individuals born during the previous breeding season (which ended eight to nine months earlier) that are most likely to be infected with PUUV (Bernshtein *et al.* 1999; Olsson *et al.* 2002). During the breeding season, there was naturally an influx of young uninfected individuals. The decrease in hantavirus prevalence over a breeding season has generally been attributed to this influx (Mills *et al.* 1999; Davis *et al.* 2005; Adler *et al.* 2008), and the seasonal variation in PUUV prevalence observed here and previously (Verhagen *et al.* 1986; Escutenaire *et al.* 2000; Sauvage *et al.* 2002) may in part be caused by such a juvenile dilution effect (Mills *et al.* 1999). But it may often not be the whole story. Ultimately, increasing the host (especially susceptible host) density should facilitate transmission and increase infection prevalence (Mills *et al.* 1999; Adler *et al.* 2008). However, this increase may be substantially delayed if maternally protected individuals are prevalent in the host population, since this will delay the influx of *susceptible* individuals into

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Table 1. Best selected GLMM model and parameter estimates (logit scale) for the probability of carrying maternal antibodies during the breeding season (summer and late summer). (Bank vole abundances indicate natural log-transformed data. σ^2, the variance attributable to random effect; s.d., standard deviation of σ^2.)

source of variation	coefficient (s.e.)	*z*-value	*p*-value
intercept	−2.101 (3.648)	−0.794	0.427
weight	−0.276 (0.078)	−3.556	<0.001
infection prevalence in preceding May	0.025 (0.013)	1.954	0.051
breeding season (late summer)	−1.439 (0.464)	−3.099	0.002
current bank vole abundance	1.536 (0.803)	1.912	0.056
bank vole abundance six months before	−0.849 (0.456)	−1.869	0.062
random effect: site ∗ year	$\sigma^2 = 1.212$; s.d. = 1.101		

Table 2. Parameter estimates (logit scale) on the influence of trapping seasons on the probability of an individual bank vole being PUUV-infected. (Intercept represents season spring (May). σ^2, the variance attributable to random effect; s.d., standard deviation of σ^2.)

source of variation	estimate (s.e.)	*z*-value	*p*-value
intercept	0.086 (0.132)	0.651	0.515
season			
summer (July)	−0.572 (0.159)	−3.847	<0.001
late summer (August)	−1.757 (0.152)	−11.549	<0.001
late autumn (October)	−1.342 (0.151)	−8.903	<0.001
random effect: site ∗ session	$\sigma^2 = 0.323$; s.d. = 0.569		

Table 3. Parameter estimates (logit scale) for the best selected GLMMs on bank vole PUUV infection probability separately for spring, summer, late summer and late autumn using the full datasets. (σ^2, the variance attributable to random effect; s.d., standard deviation of σ^2.)

source of variation	coefficient (s.e.)	*z*-value	*p*-value
spring (May)			
intercept	−2.584 (0.986)	−2.621	0.009
weight	0.103 (0.041)	2.527	0.012
random effect: site ∗ year	$\sigma^2 = 0.202$; s.d. = 0.449		
summer (July)			
intercept	−5.830 (1.181)	−4.938	<0.001
cycle			
increase phase	0.008 (0.430)	0.019	0.985
peak phase	2.156 (0.868)	2.485	0.013
infection prevalence in preceding October	−0.068 (0.037)	−1.862	0.063
sex			
male	−2.475 (1.476)	−1.676	0.094
weight	0.248 (0.042)	5.923	<0.001
sex ∗ weight	0.140 (0.063)	2.232	0.026
random effect: site ∗ year	$\sigma^2 = 0.483$; s.d. = 0.697		
late summer (August)			
intercept	−7.611 (1.002)	−7.596	<0.001
sex			
male	−5.980 (1.735)	−3.446	<0.001
weight	0.270 (0.052)	5.272	<0.001
maturity			
mature	1.016 (0.367)	2.768	0.005
sex ∗ weight	0.351 (0.090)	3.981	<0.001
random effect: site ∗ year	$\sigma^2 = 1.129$; s.d. = 1.062		
late autumn (October)			
intercept	−3.616 (1.011)	−3.578	<0.001
weight	0.200 (0.044)	4.605	<0.001
infection prevalence in preceding May	−0.030 (0.012)	−2.514	0.012
random effect: site ∗ year	$\sigma^2 = 1.788$; s.d. = 1.337		

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the population until the MatAb effect has disappeared (Gavrilovskaya *et al.* 1990; Kallio *et al.* 2006*a*).

The proportion of MatAb-protected individuals during the breeding season was positively correlated with the infection prevalence at the beginning of the breeding season. Further, in late autumn, the probability of being infected was negatively related to the MatAb prevalence in summer. In addition, infection prevalence earlier in the breeding season, the key element in MatAb effect, showed a negative relationship with infection probability in late autumn. All these results support the proposition that MatAbs may reduce PUUV transmission: a high PUUV infection prevalence in the early breeding season can lead to a high proportion of maternally protected young individuals during the summer, which, in turn, decreases later infection likelihoods.

In spring, infection prevalence varied substantially (between 32% and 80%). Our analyses support the idea that high spring infection prevalence may cause delays in the spread of PUUV via a MatAb effect. When the spring prevalence was very high in our study (peak phases 2002, 2005 and 2008), the prevalence of infection declined substantially over the year (and numbers of infected individuals did not increase) despite the increasing bank vole density (figure 1). By contrast, low infection prevalence in spring should result in only a small proportion of summer-born individuals being maternally protected, allowing transmission to proceed without any substantial time lags. This was indeed observed in the increase phase years 2004 and 2007, where the numbers of infected individuals increased together with host abundance over the breeding season (though prevalence remained stable; figure 1).

Factors influencing the likelihood of being infected varied with season. Individual-level characteristics were the most important predictors in spring and late summer, whereas population-level predictors were also selected in the best models for summer and late autumn. Interestingly, none of the host population abundances was selected in any of the best models, and of the cycle phases, only the peak phase had a significant positive effect on the infection likelihood and only in summer. The individual-level characteristics sex and weight (and their interaction) were often important: increasing body mass increased the probability of infection and males were more often infected than females. These observations are in line with other studies (e.g. Gavrilovskaya *et al.* 1990; Bernshtein *et al.* 1999; Escutenaire *et al.* 2000; Olsson *et al.* 2002).

Hence, we have, for the first time, evaluated data on seropositivity from a natural population and partitioned those data into separate dynamic patterns for MatAb and infection. Our main aim was to test whether MatAbs have a detectable influence on pathogen transmission dynamics in wildlife populations. This study supports that idea. Such effects have been neglected in the past. We suggest that more detailed investigations on the role of MatAb-protected individuals in transmission dynamics in a range of systems are highly desirable.

This research adhered to the Association for the Study of Animal Behaviour/Animal Behaviour Society Guidelines for the Use of Animals in Research, the legal requirements in Finland and all institutional guidelines.

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Electronic supplementary material

Table S1. Model selection for analyses. For each analysis the selected model is shown in bold and all models with AIC within 2 of the best model are shown. Parameter estimates for the best selected models for analysis 1 is provided in the main text in Table 1, for analysis 2 in Table 3 and for analysis 3 in the text. Analyses are based on GLMM approach.

Analysis	Model	AIC
1) Probability of carrying MatAbs	**w + inf_pre in spring + breeding season + current bv + bv 6 months before**	**467.4**
	w + inf_pre in spring + breeding season + current bv + bv 4 months before	467.7
	w + inf_pre in spring + inf_pre in summer + breeding season + current bv + bv 4 months before	469.2
	w + inf_pre in spring + inf_pre in summer + breeding season + current bv + bv 4 months before	469.2
	w + inf_pre in spring + inf_pre in summer+ breeding season + current bv + bv 6 months before	469.3
2) Probability of being infected in		
a) Spring	s + w + inf_pre in late autumn + (s * inf_pre in late autumn)	364.2
	s + w + inf_pre in late autumn	365.2
	s + w	364.4
	w	**365.4**
b) Summer	s + w + c+ inf_pre in late autumn + (s * c) + (s * w)	469.8
	s + w + c + inf_pre in late autumn + (s * w)	**471.7**
c) Late summer	s + w + m + inf_pre in spring + (s * w) + (s * inf_pre in spring)	442.7
	s + w + m + (s * w) + inf_pre in spring	444.5
	s + w + m + (s * w)	**444.2**
d) Late autumn	w + inf_pre in spring + inf_pre in summer	716.5
	w + s + inf_pre in spring + inf_pre in summer	717.9
	w + inf_pre in spring	**718.5**
3) Probability of being infected in late autumn in relation to preceding MatAb prevalences	**MatAb_pre in summer + w**	**719.8**
	MatAb_pre in summer + MatAb_pre in late summer + w	717.9

w = weight; s = sex; m = maturity; inf_pre = infection prevalence in preceding (in the given season); bv = bank vole abundance (in the given time); c = phase of the vole cycle; m = maturity; MatAb_pre = MatAb prevalence (in the given season)

Electronic supplementary material

Table S2.Trapping sessions in relation to the previous trapping sessions. Three previous trapping sessions (PREV_1, PREV_2 and PREV_3) are located under the month the trapping has been carried out. Also the time (t-X) indicates how many months (X) earlier the preceding trappings took place. The rows show the trapping seasons that were studied separately.

Trapping season	**OCT**	**AUG**	**JUL**	**MAY**	**OCT_1**	**AUG_1**	**JUL_1**
Spring					t-7	t-9	t-10
(May)					PREV_1	PREV_2	PREV_3
Summer				t-2	t-9	t-11	
(July)				PREV_1	PREV_2	PREV_3	
Late Summer			t-1	t-3	t-10		
(Aug)			PREV_1	PREV_2	PREV_3		
Late Autumn		t-2	t-3	t-5			
(Oct)		PREV_1	PREV_2	PREV_3			