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DELAYED DENSITY-DEPENDENT PREVALENCE OF *SIN NOMBRE VIRUS* INFECTION IN DEER MICE (*PEROMYSCUS MANICULATUS*) IN CENTRAL AND WESTERN MONTANA

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Abstract

Understanding how transmission of zoonoses takes place within reservoir populations, such as *Sin Nombre virus* (SNV) among deer mice (*Peromyscus maniculatus*), is important in determining the risk of exposure to other hosts, including humans. In this study, we examined the relationship between deer mouse populations and the prevalence of antibodies to SNV, a system where the effect of host population abundance on transmission is debated. We examined the relationship between abundance of deer mice in late summer–early autumn and SNV antibody prevalence the following spring–early summer (termed *delayed density-dependent* [DDD] prevalence of infection) at both regional and local scales, using 12 live-trapping grids for 11–14 yr, across central and western Montana. When all trapping grids were combined (regional scale), there was a significant DDD relationship for individual months and when months within seasons were averaged. However, within individual grids (local scale), evidence of DDD prevalence of infection was observed consistently at only one location. These findings suggest that, although there is evidence of DDD prevalence of infection at regional scales, it is not always apparent at local scales, possibly because the regional pattern of DDD infection prevalence is driven by differences in abundance and prevalence among sites, rather than in autumn–spring delays. Transmission of SNV may be more complex than the original hypothesis of autumn–spring delayed density dependence suggests. This complexity is also supported by recent modeling studies. Empirical investigations are needed to determine the duration and determinants of time-lagged abundance and antibody prevalence. Our study suggests predicting local, human exposure risk to SNV in spring, based on deer mouse abundance in autumn, is unlikely to be a reliable public health tool, particularly at local scales.

Keywords

Antibody prevalence; deer mouse; delayed density-dependent transmission; *Peromyscus maniculatus*; *Sin Nombre virus*

INTRODUCTION

Clarifying mechanisms underpinning transmission of zoonoses are critical to determining the risk of human exposure. Relationships between host or vector abundance and pathogen prevalence are particularly important components of understanding transmission. For

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example, much research has focused on understanding whether transmission of zoonotic pathogens among a reservoir population is either a linear or a saturating function of host density (termed *density-dependent transmission* and *frequency-dependent transmission*, respectively; Keeling and Rohani, 2008). In addition to direct relationships between host density and infection prevalence, some systems may have a delayed function of density; termed *delayed density-dependent* (DDD) prevalence of infection (Mills et al., 1999a). For example, Burthe et al. (2006) found the probability of *Microtus agrestis* voles acquiring antibodies to *Cowpox virus* infection was positively related to vole density three months prior. Because reports of DDD prevalence of infection are rare in wildlife populations, investigations evaluating the existence of such patterns are valuable. We evaluate evidence for DDD prevalence of infection in a system in which this type of relationship has been suggested to occur (Mills et al., 1999a): *Sin Nombre virus* (SNV; *Bunya-viridae: Hantavirus*) infection in its natural host, the deer mouse (*Peromyscus maniculatus*). *Sin Nombre virus* is an important zoonotic pathogen that causes hantavirus pulmonary syndrome (HPS) when the infection spills over into humans (Nichol et al., 1993).

Sin Nombre virus causes a chronic infection in its natural host, so antibody-positive deer mice are generally considered actively infected (Mills et al., 1999a). Transmission of SNV in deer mouse populations occurs primarily through horizontal routes, including fighting, mutual grooming, or contact with virus-contaminated materials (Mills et al., 1997; Botten et al., 2002). Increases in host population abundance should, therefore, increase interactions among deer mice, resulting in increased numbers of antibody-positive individuals and also increased antibody (infection) prevalence, consistent with density-dependent transmission (Dobson and Hudson, 1995; Begon et al., 2002). The expected positive relationship between deer mouse abundance and the number of antibody-positive individuals has been observed (Douglass et al., 2001). However, studies reporting a simultaneous relationship between abundance and antibody prevalence have been mixed, showing positive, negative, or no relationship (e.g., Boone et al., 1998; Douglass et al., 2001; Yates et al., 2002). The underlying relationship between deer mouse abundance and SNV antibody prevalence remains a subject of debate.

Several *Hantavirus* studies have shown that there is a time-lagged relationship between host population abundance in autumn and antibody prevalence the following spring (Niklasson et al., 1995; Mills et al., 1999a; Madhav et al., 2007). Evidence of autumn-spring DDD prevalence for deer mice and SNV in the southwestern United States led to an explanatory conceptual model for strongly seasonal climates (Mills et al., 1999a). There are three steps to this conceptual model. First, the increase in the numbers of uninfected juveniles following the spring/summer breeding period results in high population densities but low antibody prevalence in the autumn because newly recruited juveniles have not yet been exposed to the horizontally transmitted virus. Second, juvenile recruitment ceases after the breeding season, cumulative virus transmission events (particularly in late autumn and early spring) and overwinter mortality results in lower population density and higher antibody prevalence the following spring. Third, if increased population density in the autumn results in increased interactions between mice, the antibody prevalence in the spring should be proportional to the population density the previous fall.

Madhav et al. (2007) found broadscale evidence of DDD prevalence of infection with SNV among deer mice in Montana by combining data across multiple live-trapping grids. Our preliminary investigations of individual trapping grids in western Montana suggest DDD prevalence may occur at some locations, but not others. Here, we reanalyze data examined by Madhav et al. (2007) and investigate whether DDD infection prevalence occurs at local as well as regional scales.

MATERIALS AND METHODS

This investigation was undertaken on 18 live-trapping grids located at six sites across central and western Montana (Douglass et al., 1996; Douglass et al., 2001). These trapping grids were established as part of a long-term effort to investigate the ecology of *Hantavirus* hosts (Douglass et al., 1996; Mills et al., 1999b). Elevations at these grids ranged from 738 to 1,957 m. Live-trapping grids were located in a range of habitat types, including grassland, sagebrush, meadowland, and forest (Douglass et al., 2001). Deer mice were live-trapped monthly for three consecutive nights between June and October on all grids for 12 yr (1994–2005). Three grids (Polson 5, Cascade 11, and Cascade 12) were trapped for a longer time (1994–2008). All trapping grids were 1 ha in area and consisted of 100 equally spaced Sherman live-capture traps (H.B. Sherman Traps Inc., Tallahassee, Florida, USA), baited with rolled oats and peanut butter and provisioned with polyester Fiberfil bedding. Upon capture, each rodent was given a uniquely numbered ear-tag (model 1005-1; National Band & Tag Co, Newport, Kentucky, USA), and its species, sex, body mass, reproductive condition, and presence of scars or wounds were recorded.

Blood samples were collected monthly from rodents trapped on two grids at each site ($n=12$). We analyzed blood samples for immunoglobulin G antibodies to SNV using an enzyme immunoassay (Feldmann et al., 1993). The assay detects antibodies to all known North American hantaviruses but does not distinguish among those viruses. We followed animal handling, blood collection, and safety precautions described by Mills et al. (1995) and approved by the University of Montana Institutional Animal Care and Use Committee.

Similar to Madhav et al. (2007), we used the minimum number of deer mouse individuals known to be alive (MNA) during 3-day monthly trapping sessions as an index of population abundance (Krebs, 1996). The minimum number of SNV antibody-positive (infected) deer mice (MNI) during each trapping session was calculated in the same way. Estimated standing antibody prevalence (ESAP) of deer mice for a given month was calculated by dividing the MNI by the MNA (Mills et al., 1999a). Because hantaviruses cause chronic (likely lifelong) infection in their hosts, antibodies are often considered a marker of infection (Mills et al., 1999a). We considered antibody-positive deer mice to be actively infected.

Analyses

We used the Spearman correlation to examine evidence for DDD prevalence at each live-trapping grid and across all grids combined. We correlated MNA in late summer–early autumn (August, September, and October) and ESAP in the following spring–early summer (May, June, and July). We evaluated evidence for DDD prevalence for each of these months individually and for MNA and ESAP for all months combined. Analyses were performed using SPSS 15.0 (SPSS Inc., Chicago, Illinois, USA). To avoid Type II statistical errors with inherently variable ecological data, analyses were not corrected for multiple comparisons (Nakagawa, 2004).

RESULTS

Over the period of this study, 30,417 captures of 14,398 individual deer mice were made, spanning 459,000 trap-nights. Both deer-mouse abundance (MNA) and antibody prevalence (ESAP) varied among live-trapping grids and among months within the grids (Table 1). Between August and October, the MNA was greatest on average at Polson and Cascade, which are sagebrush and grassland habitats, respectively (Table 1). Deer mice were least abundant at Wisdom, which was the highest elevation site (Table 1). On average, ESAP was greater in May than June or July (Table 1). The ESAP was generally greater at Cutbank,

Gold Creek, and Polson relative to other sites (Table 1). The ESAP was lowest at Wisdom, where deer mice were least abundant (Table 1).

When all live-trapping grids were combined, there was a significant DDD prevalence relationship between MNA (abundance) and estimated antibody prevalence for individual and averaged months (Tables 2 and 3). In all cases, although correlations were highly significant, the relationships were not strong (Tables 2 and 3). However, within individual trapping grids, evidence of DDD prevalence was observed only at Cascade grid 11 consistently and less consistently at Charles M. Russell (CMR) grid 18 between August MNA and June–July ESAP, September MNA and June ESAP, and October MNA and July ESAP (Tables 2 and 3). We found no evidence for DDD prevalence of infection at the other live-trapping grids.

DISCUSSION

Transmission of SNV is believed to be DDD in nature (spring antibody prevalence is proportional to autumn deer mouse abundance; Mills et al., 1999a), and supporting evidence for this has been found across western and central Montana (Madhav et al., 2007). We also found evidence that DDD prevalence of infection occurred regionally (across western and central Montana). However, at local scales, evidence in support of DDD prevalence of infection was only found consistently on one of 12 grids, and less consistently on one other grid (CMR 18).

Our results suggest there is a regional pattern of DDD infection prevalence for SNV among deer mice. Although this relationship between deer mouse abundance in autumn and ESAP in spring was significant, the amount of variation explained in our analysis was relatively low, suggesting the regional DDD pattern we observed is not strongly predictive. Furthermore, the regional positive relationship between deer mouse abundance and SNV prevalence appears to be related to a general trend for sites with higher average abundance to also have higher average SNV prevalence, which may suggest DDD prevalence of infection is a pattern, but not a cause, at the regional scale. Similar to our study, evidence of DDD prevalence for hantaviruses was found by Madhav et al. (2007) in Montana, Escutenaire et al. (1997) in Belgium, Calisher et al. (1999) in Colorado, and by Yates et al. (2002) in the Four Corners region of the United States.

Our study provides evidence that delayed density-dependence (as proposed by Mills et al., 1999a) in Montana is scale dependent, observed at the regional scale, but rarely observed at local scales. There was no obvious determinant (habitat or deer mouse abundance factors) as to why DDD prevalence was found at these individual trapping grids but not at others. Possibly some combination of factors resulted in DDD prevalence being found at Cascade 11, in particular, which we were unable to identify. Perhaps survival and behavioral patterns among deer mice differ at Cascade 11 from other live-trapping grids, but examining this hypothesis is beyond the scope of this investigation.

It may be that a DDD pattern emerges when using regional and long-term averages, but they may not be observed at a given trapping grid in a given year because of stochastic factors. For example, the number of deer mice trapped or the prevalence of SNV calculated on a given trapping occasion could be influenced by local environmental conditions, such as precipitation and temperature (Luis et al., 2010), meaning analysis of relationships between abundance and prevalence of SNV may be more accurate when averaging consecutive trapping sessions within a season. Additionally, the relationship between autumn population density and spring prevalence depends on many highly variable factors, including autumn food supply and environmental conditions that would influence frequency of interactions,

conditions affecting overwinter survival, and the timing of breeding and recruitment in the spring (Luis et al., 2010). For example, a May sampling may include juveniles if winter and early spring environmental conditions were favorable but may not, if conditions were harsh. Early spring breeding and recruitment of juveniles into the population would result in lower prevalence of SNV antibodies for the population than in spring seasons with low juvenile recruitment because juveniles with SNV antibodies are rare (Douglass et al., 2001).

In this study, we specifically investigated the autumn-spring DDD relationship proposed by Mills et al. (1999a) and evaluated data used by Madhav et al. (2007) at regional and local scales. It is possible that transmission of SNV may be more complex than the original hypothesis of delayed density-dependence proposed by Mills et al. (1999a) suggests, at least at the local scale, and that other time lags should be examined in future studies. For example, using epidemic models, Adler et al. (2008) concluded time lags in SNV transmission were influenced by fluctuations in deer mouse abundance, mean prevalence, and whether transmission was frequency-dependent or density-dependent. Further, empirically based research is needed to delineate the complexities that determine how deer mouse abundance translates to time-lagged infection prevalence. For example, the age structure of deer mouse populations may also be important because antibodies to SNV are most frequently detected in adult deer mice, particularly males, and rarely detected in subadults and juveniles (e.g., Douglass et al., 2001). This pattern of infection in older, particularly male, deer mice may be due to age-specific behavior and contact heterogeneity among deer mice (Clay et al., 2009; Laverty and Adler, 2009). Regardless of the cause, our results show that predicting local, human exposure risk to SNV in spring, based solely on deer mouse abundance in autumn, is unlikely to be a reliable public health tool, particularly at local scales.

Understanding patterns associated with the transmission of infectious disease are fundamental to evaluating human exposure risk to these pathogens. Human exposure to SNV predominates in peridomestic environments during spring-summer, when infection prevalence peaks among deer mouse populations. Therefore, a capacity to predict years where prevalence of SNV among deer mouse populations is high in spring-summer would be valuable to understanding human exposure and to implementing appropriate public health interventions. We examined whether spring prevalence of SNV antibody among deer mouse populations could be predicted by deer mouse abundance in autumn, based on the relationship proposed by Mills et al. (1999a). We examined evidence for DDD prevalence of SNV infection in deer mice at two scales (local and regional) in western Montana. We found regional evidence of DDD prevalence across our 12 live-trapping grids, but there was little evidence of DDD prevalence at individual trapping grids (except Cascade 11), and this may be a common pattern among studies of SNV infection prevalence. We suggest that the duration of time lags and the determinants of time lags are more complex than originally proposed, and more empirical investigations are warranted.

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Live-trapping grids, their habitat characteristics, and elevations where deer mice were trapped and tested for antibodies to *Sin Nombre virus* (SNV), 1994–2005, in western Montana. Average deer mouse abundance expressed as the minimum number alive (MNA $\pm$ SE) August–October, and estimated standing antibody prevalence (ESAP $\pm$ SE) May–July. Polson 5, Cascade 11, and Cascade 12 span 1994–2008.

Table 1

Site/grid	Habitat	Elevation, m	Average MNA (SE)				Average ESAP (SE)		
			August	September	October	May	June	July	
Cascade 11	Grassland	1,402	28.93 (4.83)	28.79 (5.39)	27.21 (3.91)	0.11 (0.025)	0.08 (0.022)	0.06 (0.014)	
Cascade 12	Grassland	1,415	14.00 (4.67)	18.71 (6.29)	19.07 (3.72)	0.08 (0.036)	0.09 (0.039)	0.07 (0.039)	
CMR ^a 17	Forest	738	14.27 (2.63)	22.18 (4.09)	17.00 (2.82)	0.02 (0.013)	0.05 (0.020)	0.06 (0.026)	
CMR ^a 18	Sagebrush	927	10.18 (2.11)	11.27 (2.18)	7.09 (2.57)	0.09 (0.072)	0.09 (0.042)	0.02 (0.014)	
Cutbank 14	Grassland	1,256	11.55 (2.14)	11.45 (1.88)	9.91 (2.19)	0.06 (0.031)	0.01 (0.009)	0.02 (0.015)	
Cutbank 15	Grassland	1,299	11.09 (2.33)	9.64 (1.36)	9.55 (1.82)	0.27 (0.065)	0.17 (0.044)	0.11 (0.033)	
Gold Creek 8	Meadow	1,591	16.27 (5.06)	19.82 (5.12)	13.18 (3.58)	0.25 (0.080)	0.12 (0.048)	0.15 (0.052)	
Gold Creek 9	Forest	1,598	12.09 (2.77)	13.64 (3.05)	10.91 (2.81)	0.22 (0.116)	0.10 (0.062)	0.07 (0.038)	
Polson 5	Sagebrush	820	65.64 (10.72)	69.21 (9.60)	63.36 (10.29)	0.32 (0.046)	0.30 (0.045)	0.20 (0.032)	
Polson 6	Forest	915	18.55 (2.79)	16.55 (2.56)	13.73 (3.01)	0.17 (0.048)	0.12 (0.038)	0.13 (0.047)	
Wisdom 2	Forest	2,050	1.27 (0.33)	3.00 (0.66)	1.27 (0.47)	0 (0)	0 (0)	0 (0)	
Wisdom 3	Forest	2,146	0.36 (0.28)	0.45 (0.21)	0.45 (0.25)	0 (0)	0 (0)	0.09 (0.09)	

^aCMR = Charles M. Russell Wildlife Refuge.

Table 2

Spearman correlation of monthly relationships between deer mouse abundance expressed as the minimum number alive (MNA) and estimated standing antibody prevalence (ESAP), 1994–2005. Analysis of Polson 5, Cascade 11, and Cascade 12 span 1994–2008. No antibody-positive deer mice were detected at Wisdom 2. Significant correlations are in bold.

	ESAP site/grid	May		June		July	
		ρ	<i>P</i>	ρ	<i>P</i>	ρ	<i>P</i>
MNA August	Cascade 11	0.530	0.051	0.520	0.056	0.568	0.034
	Cascade 12	-0.131	0.654	-0.231	0.428	-0.169	0.564
	CMR ^a 17	-0.341	0.305	0.104	0.761	0.408	0.213
	CMR ^a 18	0.125	0.715	0.847	0.001	0.541	0.086
	Cutbank 14	0.070	0.839	0.301	0.369	0.301	0.369
	Cutbank 15	0.132	0.699	0.161	0.636	0.033	0.924
	Gold Creek 8	0.078	0.821	0.358	0.280	0.184	0.588
	Gold Creek 9	-0.145	0.670	-0.216	0.524	0.186	0.584
	Polson 5	-0.346	0.226	-0.372	0.190	-0.320	0.264
	Polson 6	0.317	0.342	0.275	0.413	0.303	0.365
MNA September	Wisdom 2	—	—	—	—	—	—
	Wisdom 3	—	—	—	—	-0.148	0.663
	All	0.384	<0.001	0.419	<0.001	0.408	<0.001
	Cascade 11	0.531	0.051	0.550	0.041	0.641	0.014
	Cascade 12	-0.178	0.544	-0.267	0.357	-0.288	0.319
	CMR ^a 17	-0.061	0.859	0.196	0.563	0.102	0.765
	CMR ^a 18	-0.246	0.467	0.545	0.083	0.273	0.417
	Cutbank 14	-0.067	0.845	0.201	0.554	0.201	0.554
	Cutbank 15	0.267	0.428	0.125	0.715	0.269	0.423
	Gold Creek 8	-0.043	0.900	0.205	0.546	0.048	0.889
MNA September	Gold Creek 9	-0.116	0.734	-0.244	0.469	0.290	0.387
	Polson 5	-0.331	0.247	-0.348	0.223	-0.233	0.422
	Polson 6	0.345	0.299	0.254	0.451	0.338	0.309
	Wisdom 2	—	—	—	—	—	—
	Wisdom 3	—	—	—	—	-0.235	0.488

	ESAP site/grid	May		June		July	
		ρ	P	ρ	P	ρ	P
MNA October	All	0.309	<0.001	0.392	<0.001	0.366	<0.001
	Cascade 11	0.484	0.079	0.583	0.029	0.641	0.014
	Cascade 12	-0.198	0.498	-0.308	0.285	-0.257	0.375
	CMR ^a 17	0.058	0.866	-0.203	0.549	-0.502	0.116
	CMR ^a 18	0.314	0.348	0.467	0.148	0.542	0.085
	Cutbank 14	-0.231	0.494	0.100	0.770	0.100	0.770
	Cutbank 15	0.409	0.212	0.501	0.116	0.256	0.447
	Gold Creek 8	-0.483	0.133	-0.357	0.281	-0.483	0.133
	Gold Creek 9	0.191	0.573	-0.087	0.799	0.397	0.227
	Polson 5	-0.239	0.410	-0.168	0.567	0.280	0.332
	Polson 6	-0.147	0.666	0.093	0.785	0.177	0.602
	Wisdom 2	—	—	—	—	—	—
	Wisdom 3	—	—	—	—	-0.191	0.573
	All	0.316	<0.001	0.341	<0.001	0.357	<0.001

^aCMR = Charles M. Russell Wildlife Refuge.

Table 3

Spearman correlation of the relationships between average deer mouse abundance expressed as the minimum number alive (MNA) in late summer–autumn (August–October) and the estimated standing antibody prevalence (ESAP) the following spring–early summer (May–July), 1994–2005. Analysis of Polson 5, Cascade 11, and Cascade 12 span 1994–2008. No antibody-positive deer mice were detected at Wisdom 2. Significant correlations are in bold.

Site/grid	ρ	P
Cascade 11	0.620	0.018
Cascade 12	−0.253	0.383
CMR ^a 17	0.077	0.821
CMR ^a 18	0.383	0.246
Cutbank 14	−0.107	0.754
Cutbank 15	0.253	0.452
Gold Creek 8	−0.105	0.758
Gold Creek 9	0.047	0.890
Polson 5	−0.319	0.267
Polson 6	0.308	0.357
Wisdom 2	—	—
Wisdom 3	−0.324	0.332
All	0.385	<0.001

^aCMR = Charles M. Russell Wildlife Refuge.