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## Changes in Sin Nombre Virus Antibody Prevalence in Deer Mice Across Seasons: The Interaction Between Habitat, Sex, and Infection in Deer Mice

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**ABSTRACT:** We examined the impact of season and habitat on Sin Nombre virus (SNV) seroprevalence in deer mice (*Peromyscus maniculatus*) in Utah's Great Basin Desert from May 2002 through summer 2003. Low mouse captures in 2002 limited analysis for that year. In two seasons during 2003, mouse density and sagebrush cover were positively linked (spring:  $r=0.8$ ,  $P=0.01$ ; summer:  $r=0.8$ ,  $P=0.04$ ). In the spring, seroprevalence was negatively correlated with density ( $r=-0.9$ ,  $P<0.01$ ); male and female antibody prevalence did not differ; and scarring was unrelated to antibody status. In the summer, density and antibody prevalence were unrelated; male seroprevalence was higher ( $\chi^2=3.6$ ,  $P=0.05$ ); and seropositive mice had more scars ( $t=2.5$ ,  $P=0.02$ ). We speculate nesting behavior could maintain SNV over the winter, whereas summer territoriality could be responsible for transmission.

**Key words:** Deer mouse, frequency-dependent transmission, habitat quality, *Peromyscus maniculatus*, seasonality, Sin Nombre virus.

The ecology of Sin Nombre virus (SNV) transmission in deer mice (*Peromyscus maniculatus*) is still not well understood; in particular, the effect of local habitat quality on SNV seroprevalence is unknown. Infection is asymptomatic (Childs et al., 1994); virus is shed in mouse urine, feces, and saliva (Netski et al., 1999) and adults are more likely to be infected than juveniles (Mills et al., 1999). Theoretically, habitat quality can increase mouse density and it is hypothesized that SNV seroprevalence is high when density is high (density-dependent) (Mills et al., 1999). However, past studies do not consistently support this model (Douglass et al., 1996; Graham and Chomel, 1997; Boone et al., 1998; Biggs et al., 2000) and studies in

other disease systems suggest frequency-dependent transmission might be a better descriptor (McCallum et al., 2001). Whereas density-dependent transmission assumes the number of contacts made by a mouse increases with increasing population size, frequency dependence assumes a constant contact rate (McCallum et al., 2001) and can result in a pattern of similar seroprevalence across a range of densities. Here we present preliminary results regarding habitat, density, host sex, and seroprevalence in deer mouse populations in central Utah's Great Basin Desert. Because trapping across habitat types (common in other SNV studies) can limit understanding of local patterns (Douglass et al., 2001), we hoped to discern local-level interactions by trapping at numerous sites within the same habitat type.

Sampling was performed from May 2002 through September 2003. Given the ecological importance of sagebrush (*Artemisia tridentata*) to deer mice in this region (Parmenter and MacMahon, 1983; Zou et al., 1989), we utilized variation in sagebrush cover as a proxy for habitat quality in examining patterns of mouse density and seroprevalence. Eight sites, each 3.14 ha, spanned a range of sage cover. Site latitude ranged from 39°47'30.4"N to 39°50'51.9"N and longitude from 112°8'25.7"W to 112°24'1"W; elevation was approximately 1,750 m for all sites. To estimate sagebrush cover, transects of 100 m were extended along each spoke of the trapping web (described below). Plant cover was measured for the 2.5 m preceding and following trapping stations and the percentage of total

TABLE 1. Density, prevalence of antibodies to Sin Nombre virus, and scarring patterns observed in deer mice. Mean density and range are in units of mice/hectare and include all 8 study sites. Density in the spring and summer of 2002 were calculated using number mice captured/3.14 hectares because sample sizes were too low for accurate density calculations in DISTANCE. Individual site data has been summed in the seroprevalence and % scarring categories.

		Spring 2002	Summer 2002	Fall 2002	Spring 2003	Summer 2003
<b>Mean density</b>		2.0 (0.3–5.7)	2.0 (0.3–3.2)	4.2 (1.2–10.5)	11.5 (3.2–27.4)	9.2 (1.6–17.5)
<b>(Range)</b>						
<b>Seroprevalence</b>	<b>Total</b>	14% (7/51)	12% (6/50)	6% (5/81)	13% (41/324)	7% (19/258)
<b>(seropositive/total)</b>	<b>Male</b>	21% (6/29)	18% (6/34)	8% (4/51)	13% (20/157)	9% (13/146)
	<b>Female</b>	n/a (1/21)	n/a (0/16)	3% (1/30)	13% (21/167)	5% (6/112)
<b>% Scarring</b>	<b>Total</b>	16% (8/50)	33% (17/52)	42% (34/81)	24% (108/449)	23% (90/398)
<b>(scarred/total)</b>	<b>Male</b>	69% (20/29)	34% (12/35)	49% (25/51)	18% (41/229)	16% (37/232)
	<b>Female</b>	n/a (8/21)	n/a (5/17)	30% (9/30)	9% (19/219)	9% (15/167)

distance attributable to sage was determined by dividing sage cover by the total area sampled.

Rodents were trapped in May 2002 (spring), August 2002 (summer), October 2002 (fall), May–June 2003 (spring), and August–September 2003 (summer). Winter trapping was not possible because of impassable conditions at the sites. Rodents were captured using Sherman traps (LFA, H. B. Sherman Traps, Inc., Tallahassee, Florida, USA) arranged in a trapping web of twelve 100 m spokes (Mills et al., 1999). Trapping lasted from three to five nights, until 90% of mice were recaptured. Species, mass, sex, reproductive status, presence of cutaneous scars, and number of scars were recorded for all deer mice. Blood samples were obtained via retro-orbital puncture and analyzed at the University of Nevada, Reno using ELISA (Otteson et al., 1996). Deer mouse densities were calculated from deer mouse captures using the program DISTANCE (Thomas et al., 2004). The minimum mass of captured reproductive animals was established as the age threshold during each year (scrotal males and perforate females); animals above that mass were classified as adults and animals below were juveniles (2002: adults >15.9 g; 2003: adults >14 g). Statistical analyses were performed in SPSS 12.0.

A total of 702 deer mice were captured

over 20,754 trap nights in 2002 and 2003 (Table 1). Because only 50 mice were captured in the spring and summer of 2002 ( $\leq 10$  mice on 7 of 8 sites each season), we excluded these data from subsequent analyses; there are large error rates associated with antibody prevalence estimates using less than 25 individuals. Captures were higher in fall 2002 but were still less than 25 mice/site; these also were excluded from regression analyses. In 2003, mice were captured in sufficient numbers to estimate seroprevalence on all but one site; this single site was excluded from seroprevalence regression analyses.

In the spring of 2003, sagebrush cover and deer mouse density were positively associated ( $r=0.82$ ;  $F_{1,6}=12.7$ ;  $P=0.01$ ; Figure 1a) but deer mouse density and SNV seroprevalence were negatively associated ( $r=-0.93$ ;  $F_{1,5}=34.3$ ;  $P=0.002$ ; Figure 1b). Mean body mass at a site was not related to sage cover ( $r=0.01$ ;  $F_{1,255}=0.02$ ;  $P>0.05$ ). In the summer of 2003, sage cover and mouse density were still positively associated ( $r=0.85$ ;  $F_{1,6}=15.4$ ;  $P=0.008$ ; Figure 1c) but there was no relationship between deer mouse density and SNV seroprevalence ( $r=0.15$ ;  $F_{1,5}=0.11$ ;  $P>0.05$ ; Figure 1d). Because there was a negative relationship between cover and mean body mass ( $r=0.24$ ;  $F_{1,261}=16.3$ ;  $P<0.001$ ), the density–seroprevalence analysis was rerun using only

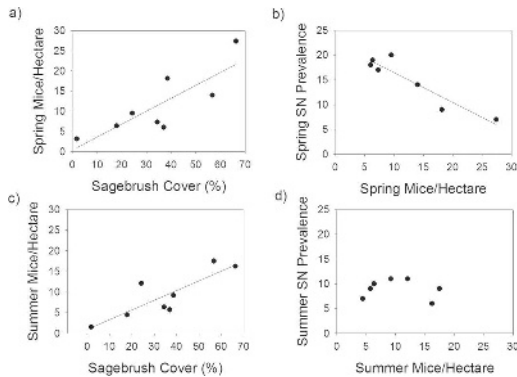


FIGURE 1. (a) Linear regression between percent sagebrush cover and deer mouse density (mice/hectare) at all sites in spring 2003 ( $P=0.01$ ). (b) Linear regression of density and SNV seroprevalence across seven sites in spring 2003 ( $P=0.008$ ). (c) Linear regression of percent sagebrush cover and deer mouse density across all sites in summer 2003 ( $P=0.002$ ). (d) Deer mouse density and SNV seroprevalence across seven sites in summer 2003. No correlation was found.

adult seroprevalence; there was still no detectable association ( $r=0.14$ ;  $F_{1,5}=0.1$ ;  $P>0.05$ ).

Data from all sites were combined to investigate season, scarring, and infection status in male and female mice across the seasons (Table 1). There was an interaction between season and scarring on infection status (Multinomial regression analysis:  $\chi^2_2=13.7$ ;  $P<0.01$ ). In spring 2003, there was no difference in the number of scars on seropositive or seronegative mice ( $t_{60}=0.56$ ;  $P>0.05$ ). However, in fall 2003, the number of scars on seropositive mice was greater than on seronegative mice ( $t_{25}=2.49$ ;  $P=0.02$ ). Across seasons, males were more likely to be scarred than females ( $\chi^2_1=10.1$ ;  $P<0.005$ ; Figure 2a). In fall 2002, there was a trend for higher male seroprevalence (Figure 2b), but it was not supported statistically, possibly because there were too few mice sampled to detect a pattern. According to the a posteriori power analysis that we performed, at least 166 mice would be needed to detect a pattern at our statistical effect size of 0.28. In spring 2003, male seroprevalence

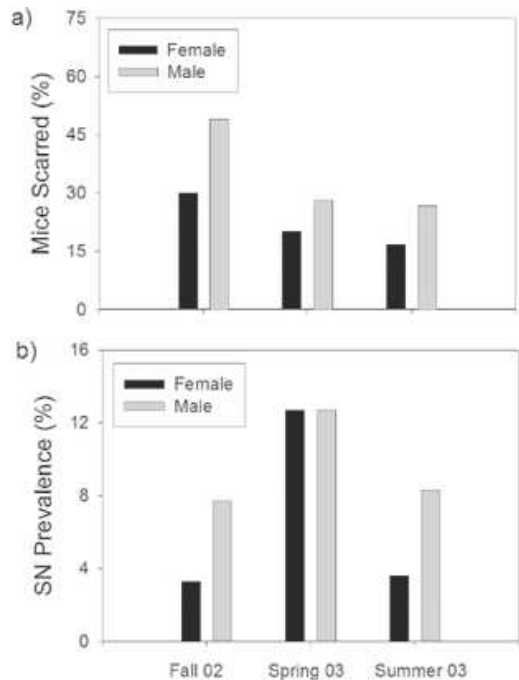


FIGURE 2. (a) The percent mice scarred across seasons grouped by sex. Males were significantly more scarred than females ( $P<0.005$ ). (b) SNV seroprevalence across seasons grouped by sex. There is a trend towards different male and female seroprevalence in fall 2002 but it was not significant, probably due to low power. In 2003, male and female seroprevalence was not different in spring but was in the summer ( $P=0.05$ ).

and female seroprevalence were equal ( $\chi^2_1=0.002$ ;  $P>0.05$ ). Male seroprevalence was higher than female seroprevalence in summer 2003 ( $\chi^2_1=3.6$ ;  $P=0.05$ ) (Figure 2b).

In the spring, mouse density increased with sagebrush cover across the study sites but SNV seroprevalence decreased with increasing density. In the Great Basin, sagebrush cover is a proxy for nesting sites and food resources, such as herbaceous plants and insects (Parmenter and MacMahon, 1983). Thus sites with greater sagebrush cover might be higher quality sites for mice and support larger mouse populations. A possible explanation for the seroprevalence results is that more juveniles occur in higher quality habitat (Calisher et al., 2001), resulting in ser-

oprevalence dilution (Calisher et al., 1999). However, we did not find evidence for dilution as mouse mass was similar across sites. We hypothesize instead that the negative relationship results from an interaction between habitat quality and winter nesting patterns. In higher cover areas, even though populations are larger, the number of nest sites per capita might be greater, reducing the number of interactions between individuals. We were unable to test this speculation because of the challenge of live trapping during the subzero (C) temperatures at the sites in winter. Continuous sampling in Montana resulted in only two winter seroconversions but trapping occurred in lower elevation grassland (Douglass et al., 2001). Winter nesting dynamics might be more important in areas in which trapping is difficult, precisely because of cold climatic conditions. We speculate also that the absence of a relationship between scars and SNV antibodies in both males and females might be due to winter behaviors. In colder climates, winter transmission has been hypothesized to result from female nest sharing and grooming (Calisher et al., 1999), which predicts higher female than male spring seroprevalence, with males reflecting the pattern of the preceding fall. We found that males and females had equal seroprevalence, suggesting both sexes participate in transmission. Male and female mice nest communally and switch nests and partners (Wolff and Hurlbutt, 1982; Wolff and Durr, 1986). Another possible explanation is that spring transmission was driven by mating interactions (Douglass et al., 2001); equal aggression in the two sexes could explain sex-specific seroprevalence patterns (Douglass et al., 2001). However, it is not clear how this explanation fits with the spring sagebrush-density-prevalence results; given the lack of territoriality and equal opportunity aggression during the mating season (Sadleir, 1965; Healey, 1967), conditions would presumably result in either a positive or nonexistent relation-

ship between mouse density and seroprevalence, but not a negative one.

In the summer, sagebrush cover and mouse density were still positively related, but seroprevalence was similar across different population densities; a similar relationship was also observed by Graham and Chomel (1997). Frequency-dependent transmission predicts this type of pattern (McCallum et al., 2001) and could be caused by deer mouse territoriality. Deer mice establish territories in the summer and fall (Sadleir, 1965; Healey, 1967). At higher population densities, territory sizes might be smaller, allowing more mice to exist within the same area of habitat (Healey, 1967; Taitt, 1981; Wolff, 1985). Territory structure could be such that mice maintain the same number of neighbors and, hence, interactions. In the summer, body mass was negatively associated with sagebrush cover, suggesting a possible dilution effect of juveniles on seroprevalence. However, because mouse density and seroprevalence remained unassociated in the adult-only analysis, the pattern could not be explained by dilution only.

Because male mice are both more likely to be scarred and more likely to be infected, it has been postulated that male aggression is the primary driver for SNV transmission between mice (Mills et al., 1999). Our finding that scarring and the presence of SNV antibodies are associated during the summer and fall, and that males are more often scarred and have more scars per individual than females, supports the role of male aggression in transmission and is consistent with numerous other studies (Boone et al., 1998; Calisher et al., 1999; Biggs et al., 2000; Douglass et al., 2001).

Our conclusions are preliminary in nature given the limited number of seasons and the single habitat type in which sampling took place. Our interpretation of the spring results remains speculative, given that we did not sample throughout the winter. Because we did



not sample at regular, frequent intervals throughout the year, we might also have missed irregular, nonseasonal drivers of SNV seroprevalence. However, our work highlights the potential importance of habitat quality in affecting behavior, and therefore SNV transmission, in a temporally variable way and generates testable hypotheses for future research in this system. Such hypotheses should be tested in long-term studies.

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