To monitor Sin Nombre virus (SNV) dynamics in natural rodent communities, we established longitudinal studies at two sites in western Colorado, each near a location where human hantavirus infections occurred in 1993. This article provides a summary of the data collected during the first 3 years of the studies. The results indicate that rodent populations in western Colorado have decreased since 1993; SNV or an SNV-like hantavirus persists at these sites; and prevalence of immunoglobulin G (IgG) antibody to SNV fluctuates with time and perhaps with weather patterns that modify the ecosystem.

The Study

Selected Sites

Each study area was selected on the basis of its proximity to residences of hantavirus pulmonary syndrome (HPS) case-patients, convenience for field work, and guaranteed cooperation by land managers. Sites at Fort Lewis (La Plata County, southwest Colorado) (N 37° 13' 30.9" latitude, W 108° 10' 51.1" longitude, altitude 2,438 m) and Molina (Mesa County, west central Colorado) (N 39° 09' 45.8" latitude, W 108° 03' 18.4" longitude, altitude 1,951 m) were within a few kilometers of case-patient residences.

Fort Lewis (approximately 22 km west of Durango, 8 km south of Hesperus) is 10 km north of Red Mesa, Southern Ute Indian Reservation, Colorado, where rodent trapping in 1993 showed that deer mice had an antibody prevalence rate of 50% to SNV and near where one of the persons who later died of HPS had been infected with SNV (1). Seroprevalence in Peromyscus maniculatus, the principal rodent reservoir of SNV, is approximately 50% to 19%, respectively, near study sites in La Plata and Mesa Counties (1).

We established trapping webs (Mills et al., this issue, pp. 95-101) in two protected areas.
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(Fort Lewis A and Fort Lewis B) of the 2,550 ha-
Colorado State University San Juan Basin
Research Center, which serves as a model for
cattle breeders and livestock geneticists. The
natural characteristics of these sites have been
preserved.

Fort Lewis is in the drainage of the La Plata
River, south of Mount Hesperus in the La Plata
Mountains. The general ecosystem of the area is
montane shrubland (2) superimposed on intru-
sive igneous rocks forming laccoliths (3). The
overstory vegetation at Fort Lewis A is
predominately ponderosa pine (Pinus ponderosa)
and Gambel’s oak (Quercus gambeli); understory
vegetation is primarily blue grama (Bouteloua
gracilis), black grama (B. eriopoda), and floral
components also seen at Fort Lewis B. At Fort
Lewis B, 500 m from Fort Lewis A, overstory is
essentially all Gambel’s oak; understory is
composed of blue and black grama or there is no
overstory, with the microcommunity composed
primarily of blue and black grama, small soapweed
(Yucca glauca), tree cholla (Opuntia imbricata),
asnd pasture sagebrush (Artemesia frigida).

The trapping sites near Molina (approxi-
mately 60 km east of Grand Junction) are within
2 km of the home of a 1993 case-patient. In
1993, deer mice had an antibody prevalence
rate of 19% to SNV (4).

At Molina we established webs in two areas
(Molina A and Molina B, 500 m apart) that are
privately owned and have not been grazed by
cattle for many years. The sites have no standing
water sources, but an irrigation ditch, containing
rapidly running water, flows during the summer
at the west and north edges of Molina A.

The general ecosystem of the area is
semidesert shrubland (2) superimposed on
Mancos shale (3). At Molina A, we found
principally Rocky Mountain juniper (Juniperus
scopulorum), pinyon pine, small soapweed, and
pasture sagebrush. Molina B is characterized by
pasture sagebrush, Rocky Mountain juniper,
Parry’s rabbitbrush (Chrysothamnus paryii),
asnd pinyon pine at the periphery.

All field data were recorded on hard copy and
entered into EPI-5, a database and statistical
program available from the Centers for Disease
Control and Prevention (CDC) (5).

Sampling Methods

All materials were transported to the study
sites or were available in towns near the sites.

Under license of the State of Colorado’s
Department of Natural Resources, sampling was
done every 6 weeks, weather permitting.

Trapping webs were established according to
methods agreed upon by collaborating groups
(Mills et al., this issue, pp. 95-101). In brief, each
web comprised 12 rows of 12 Sherman traps (7.6
cm x 8.9 cm x 22.9 cm; H.B. Sherman Traps, Inc.,
Tallahassee, FL) each, the first four traps in each
row being placed 5 m apart, the next eight placed
10 m apart; rows were 30 degrees from each
other. The location of each trap was marked with
a construction flag. Rodents were anesthetized
with Metaphane (methoxyflurane, Pitman-
Moore, Mundelein, IL) during processing,
marked with sequentially numbered stainless
steel ear tags, and released at the capture site.

Wes A and B at each location were sampled
for 2 or 3 consecutive nights, but rodents were
neither bled nor swabbed at webs B until October
1996, when animals from both sites were
sampled. The original intent had been to not take
blood or oropharyngeal swab samples at either
web B to determine, by comparison with data
from the corresponding web A, the impact of
these invasive procedures on the rodent
populations. Because the death rates at webs A
and B were essentially the same after 2 years (6;
C.H. Calisher and B.J. Beaty, unpub. data), in
October 1996, we began to take blood samples
from rodents at both webs and to no longer collect
oropharyngeal swabs. Rodents, principally deer
mice, were processed and samples were placed on
dry ice (-70°C), returned to the laboratory in Fort
Collins, and placed in a mechanical freezer
(-80°C) until they were tested for IgG antibody.

Sampling was conducted according to
standardized protocols (Mills et al., this issue,
pp. 95-101). To compare age categories, in the
field we empirically classified captured animals as
juvenile, subadult, or adult, according to
Fitzgerald, Meaney, and Armstrong (2). For final
determination, we separated animals into
weight classes (10% to 40% of adult mean weight
= juvenile, 41% to 80% = subadult, and 81% to
100% = adult).

After being tested at Colorado State
University, blood samples and oropharyngeal
swabs were shipped to Atlanta, Georgia, where
confirmatory testing for IgG antibody to SNV
was conducted with blood samples, and
oropharyngeal swabs were stored for possible
future testing.
Enzyme-Linked Immunosorbent Assays (ELISA) for IgG Antibody to SNV

ELISA was performed at Colorado State University as described (Mills et al., this issue, pp. 95-101). Results presented here were obtained at Colorado State University; testing at CDC provided confirmation. We initially screened whole blood samples at 1:100; antibody-positive samples were titrated to determine end points.

Population Densities

We estimated the population size at each sampling period by calculating the minimum number of rodents alive (7). The minimum number of rodents alive for a given trapping session was calculated by taking the total number of rodents captured during that session and adding to that sum all rodents that had been captured on at least one previous and one subsequent occasion. The minimum number of antibody-positive rodents was calculated similarly, and the estimated standing prevalence was calculated as minimum number of antibody-positive rodents/minimum number of rodents alive.

Findings

Over the 41-month trapping period at Fort Lewis and the 37-month trapping period at Molina, antibody reactive with SNV was detected in 29 (9.6%) of 302 deer mice at Fort Lewis and 36 (9.4%) of 385 at Molina; 4 (2.6%) of 155 of pinyon mice at Molina also had antibody (Table 1). For comparison, in 1993, prevalence of antibody to SNV in P. maniculatus was approximately 50% near Fort Lewis (La Plata County) and 19% near Grand Junction (Mesa County) (1). Of 112 least chipmunks (Tamias minimus), two Colorado chipmunks (T. quadrivittatus), and two western harvest mice (Reithrodontomys megalotis), none had antibody to SNV.

At Fort Lewis, trapping success (number of animals per total number of trap nights) was 0.3% to 7.6%, depending on the season (lowest rates, April–June; highest, August–October). Antibody-positive deer mice were found in 13 of 21 trapping intervals. Antibody prevalence was 0% to 33% in deer mice (mean 9.4%) and 0% to 18.2% in pinyon mice (mean 2.6%). Antibody to SNV was detected in adult (11.3%), subadult (1.7%), and juvenile (4.4%) deer mice; the stages represented 73.1%, 15.3%, and 11.7%, respectively, of the mice captured. Males represented 45.5% of the deer mouse population, 46.3% of the recaptured deer mice, and 60% of the antibody-positive mice. Antibody was detected in four adult (three male, one female) pinyon mice (P. truei). Of 118 pinyon mice collected, 62 (50.8%) were female and 56 (49.4%) were male. We detected seropositive pinyon mice only during May and June 1995 and April 1996.

Wounds and Antibody

Because we were working with a large number of anesthetized rodents, we did not closely examine each animal for wounds, as had been done by Glass et al. (8). However, we noted the most obvious wounds (ear nicks, torn ears, scarred tail) and those likely not to have been caused by trapping, tagging, or processing, and

| Location   | Species                  | No. positive/ No. tested | Antibody-positive |
|------------|--------------------------|--------------------------|-------------------|
| Fort Lewis | Peromyscus maniculatus   | 29/302                   | 9.6               |
|            | Tamias minimus            | 0/48                     | 0                 |
| Molina     | P. truei                 | 1/3                      | 33                |
| P. maniculatus | 36/385 | 9.4               |
| P. truei   | 4/155                    | 2.6                      |
| P. leucopus | 1/2                     | 50                       |
| Reithrodontomys megalotis | 0/2     | 0                      |
| T. minimus | 0/64                     | 0                       |
| T. quadrivittatus | 0/2        | 0                      |
we evaluated the data for deer mice at webs A for Fort Lewis and Molina.

Of 233 adult deer mice at Fort Lewis, 20 had both antibody and wounds, 76 had no antibody but had wounds, 4 had antibody and no wounds, and 133 had neither antibody nor wounds; thus, wounds were associated with antibody to SNV among adult deer mice (Yates-corrected chi-square 17.71, p = <0.001). At Molina, of 339 adult deer mice, 8 had antibody and wounds, 23 had no antibody but had wounds, 21 had antibody and no wounds, and 287 had neither antibody nor wounds; again wounds were associated with antibody to SNV (Yates-corrected chi-square 10.67, p = <0.001).

**Seroconversion**

Fifteen deer mice and one pinyon mouse seroconverted (i.e., seronegative to seropositive or a fourfold or greater increase in titer) between captures (Figure 1). At Fort Lewis, 302 deer mice (150 female and 152 male) were captured. Of these, 37 female and 37 male mice were recaptured at least once. Five male and three female deer mice at Fort Lewis seroconverted. One deer mouse had antibody for the first time 14 months after it was initially captured. At Molina, 385 deer mice (212 female, 173 male) and 155 pinyon mice (85 female, 70 male) were captured. Of these, 33 female and 30 male deer mice and 12 female and 10 male pinyon mice were recaptured at least once. Five male and two female deer mice and one male pinyon mouse seroconverted. An additional three deer mice (two male, one female) at Molina were recaptured and had significant (3,200 to 25,600) but stable IgG antibody titers; we did not consider these as having seroconverted. The five male mice seroconverted at Fort Lewis during the summer (one between July and September 1994, two between July and September 1995, one at [estimated] midsummer 1995, and one between June and September 1997); two female mice seroconverted between October 1994 and May 1995, and one female mouse seroconverted during late summer (September to October) 1997. At Molina, one male deer mouse seroconverted in late spring (estimated May) 1995, one in late fall 1995, two male deer mice and a male pinyon mouse during the winter or early spring of 1995 to 1996, and one male deer mouse during late spring 1996; one female deer mouse seroconverted in late summer 1995 and one during the winter 1995 to 1996. Seropositive samples were titrated by IgG ELISA with fourfold dilutions. Titers were 100 to 102,400, with most of them at 6,400 to 25,600.

**Incidence Rates**

We calculated incidence rates of IgG antibody to SNV in deer mice recaptured and sampled at least twice at Fort Lewis and Molina (Table 2). At Fort Lewis A, the overall incidence was 4.6 new infections per 100 mice per month (4.8 for male, 4.4 for female); at Fort Lewis B, the overall incidence was 10.91 (9.1 for male, 18.2 for female); and at the two sites combined, the overall incidence was 6.1 (6.3 for male, 5.8 for female). At Molina A, the overall incidence rate in deer mice was 2.8 new infections per 100 mice per month (3.2 for male, 2.3 for female); at Molina B, no new infections were detected during the observation period; the incidence at the two sites combined was 2.6 (3.0 for male, 2.2 for female). Because sufficient numbers of pinyon mice were captured and a seroconversion was detected at Molina A, we were able to calculate the incidence of seroconversion: 0.6 overall (1.4 for male, 0 for female); the incidence at the two sites combined was 0.5 (1.3 for male, 0 for female).

**Longevity**

By recapturing animals, we were able to estimate the longevity of infected and uninfected mice at the sites. Most *Peromyscus* spp. (75.7% at Fort Lewis, 66.2% at Molina) were not recaptured after they were first caught. At Fort Lewis, of 118 female and 117 male deer mice, 79

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**Figure 1.** Approximate month of seroconversion in *Peromyscus* species at Fort Lewis and Molina, Colorado, June 1994–October 1997, by sex.
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Table 2. Incidence of immunoglobulin G antibody reactive with Sin Nombre virus in *Peromyscus maniculatus* (deer mice) recaptured and sampled at least twice at Fort Lewis (June 1994-October 1997) and *P. maniculatus* and *P. truei* (pinyon mice) recaptured and sampled at least twice at Molina, western Colorado (October 1994-October 1997)

| Species/location | Sex | No. at | No. of new infections | Cum. % antibody-pos. | Mouse mos. of observation | Incidencec |
|------------------|-----|--------|------------------------|----------------------|--------------------------|------------|
| Deer mice/       |     | No. at risk⁴ | | | | |
| Ft. Lewis        |     | Grid A | 38 | 4 | 10.5 | 88 | 4.6 |
|                  | male | 17 | 2 | 5.9 | 42 | 4.8 |
|                  | female | 21 | 2 | 9.5 | 46 | 4.4 |
|                  | Grid B | 11 | 3 | 27.3 | 27.5 | 10.9 |
|                  | male | 6 | 2 | 33.3 | 22 | 9.1 |
|                  | female | 5 | 1 | 20.0 | 5.5 | 18.2 |
|                  | Grids A + B | 49 | 7 | 14.3 | 115.5 | 6.1 |
|                  | male | 23 | 4 | 17.4 | 64 | 6.3 |
|                  | female | 26 | 3 | 11.5 | 51.5 | 5.8 |
| Deer mice/       |     | Grid A | 59 | 5 | 8.5 | 179.5 | 2.8 |
| Molina           | male | 33 | 3 | 11.0 | 94 | 3.2 |
|                  | female | 26 | 2 | 7.7 | 85.5 | 2.3 |
|                  | Grid B | 7 | 0 | 0 | 12.0 | 0 |
|                  | male | 4 | 0 | 0 | 7.5 | 0 |
|                  | female | 3 | 0 | 0 | 4.5 | 0 |
|                  | Grids A + B | 66 | 5 | 7.6 | 191.5 | 2.6 |
|                  | male | 37 | 3 | 8.1 | 101.5 | 3.0 |
|                  | female | 29 | 2 | 6.9 | 90 | 2.2 |
| Pinyon mice/     |     | Grid A | 22 | 1 | 4.5 | 157 | 0.6 |
| Molina           | male | 10 | 1 | 10.0 | 71 | 1.4 |
|                  | female | 12 | 0 | 0 | 86 | 0 |
|                  | Grid B | 15 | 0 | 0 | 33.5 | 0 |
|                  | male | 7 | 0 | 0 | 9 | 0 |
|                  | female | 8 | 0 | 0 | 24.5 | 0 |
|                  | Grids A + B | 37 | 1 | 2.7 | 190.5 | 0.5 |
|                  | male | 17 | 1 | 5.9 | 80 | 1.3 |
|                  | female | 20 | 0 | 0 | 110.5 | 0 |

²No. of mice antibody-negative at first capture.
³Total time intervals between successive captures when mice were antibody-negative, plus half the interval when mice changed from antibody-negative to antibody-positive.
⁴New infections per 100 mice per month.

and 83, respectively, were caught only once; 61 were recaptured only within a 5-month period, eight within 6 to 9 months, and four 11 to 14 months after they were first captured. At Molina, of 164 female and 150 male deer mice, 115 and 93, respectively, were caught only once; 89 were recaptured only within a 4-month period, 15 only within 5 to 9 months, and 2 as long as 10 months after they were first captured. Of 63 female and 59 male pinyon mice, 49 and 43, respectively, were caught only once; 21 were recaptured only within a 4-month period, 5 only within 5 to 9 months, and 1 each for 10, 11, 19, and 20 months after they were first captured.

Longevity data of seropositive and seronegative deer mice at Fort Lewis and Molina are summarized in Table 3. Eighteen deer mice had antibody at two or three bleeding intervals from 1 to 7 months after first capture (mean = 2.4 months). Totals do not match the text above because blood samples were not collected from captured rodents at sites B until October 1996 and because we included separately periods of seronegativity and seropositivity for deer mice that seroconverted. Differences between mean longevities by sex, site, or antibody status were not statistically significant (Yates-corrected chi-square, p = >0.2).

**Population Densities**

Deer mouse populations and prevalence of antibody to SNV at Fort Lewis (Figure 2) were relatively low throughout this study, except in May and June 1995 when samples included only four deer mice and one deer mouse, respectively. Mean minimum number of rodents alive was 28 in 1994 but lower from 1995 to 1997 (10.8, 13.4, and 16.4, respectively). At Molina, populations were relatively stable between 1995 and 1997 (only one collection made in 1994), with mean minimum number of rodents alive values of 31.2, 20.4, and 25.4, respectively. As at Fort Lewis, estimated standing prevalence values were commensurately low (Figure 3).
Conclusions

On the basis of the high antibody titers of these seropositive samples, our findings elsewhere in Colorado (Calisher, Beaty, and Mills, unpub. data), and the findings of others studying hantaviruses in the Southwest (9), we presumed that IgG antibody to SNV in deer mice indicated infection with SNV and not with El Moro Canyon or another hantavirus. Although we did not attempt to isolate or detect hantaviral RNA in blood or other tissues from mice with antibody, the only hantavirus specifically identified in deer mice in western Colorado has been SNV (10).

The presence of IgG antibody to hantaviruses in rodents is presumed to indicate past infection and present infection, at least in the primary vertebrate hosts of hantaviruses (Mills et al., this issue, pp. 135-142). That is, rodents infected with hantaviruses with which they appear to be closely associated coevolutionarily (e.g., deer mice and SNV, Western harvest mice and El Moro Canyon virus, rice rats [Oryzomys palustris] and Bayou virus, Black Creek Canal virus and cotton rats [Sigmodon hispidus]) do not appear ill or otherwise affected by hantaviruses specific to them. In host-virus associations that have been studied, the specific hosts become infected early or later in life, are viremic for a short period, and excrete virus in their saliva, urine, and feces, perhaps for life (11-14).

Fighting (including exchange of blood and saliva) between infected and uninfected adult rodents has been suggested as the primary mechanism by which hantaviruses are amplified epizootically (8). Infected rodents become viremic and viruric and serve as subsequent sources of infection for others in the population. Earlier studies using Seoul virus and laboratory rats as a model system had indicated that while in newborn rats infection became persistent, in older rats it was transient (15). However, evidence using Black Creek Canal virus and

| Site     | Sex | Sero-status | Total No. | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | Mean |
|----------|-----|------------|-----------|---|---|---|---|---|---|---|---|---|---|---|---|-----|
| Fort Lewis | F   | +          | 3 2       |   |   |   |   | 1 |   |   |   |   |   | 3  |
|           | M   | +          | 6 3 2     |   |   |   |   | 1 |   |   |   |   |   | 2.7|
| Molina   | F   | +          | 3 2       |   |   |   |   |   | 1 |   |   |   |   |   | 1.7|
|           | M   | +          | 6 4 1     |   |   |   |   |   |   |   |   |   |   |   | 2.3|
| Fort Lewis | F   | -          | 27 15 5 2 | 2 | 1 | 1 | 1 | 1 | 1 |   |   |   | 2.5|
|           | M   | -          | 22 7 4 2 2 | 1 | 1 | 2 | 2 | 1 |   |   |   | 3.7|
| Molina   | F   | -          | 37 24 2 4 2 | 1 | 2 | 1 | 1 | 1 |   |   |   |   | 2.4|
|           | M   | -          | 27 13 1 4 2 | 3 | 1 | 1 | 2 |   |   |   |   | 3.2|

Table 3. Longevity of hantavirus-infected and -uninfected male and female Peromyscus maniculatus at Fort Lewis and Molina, Colorado, June 1994–October 1997 expressed as number of months between first and last capture.

Figure 2. Minimum number of deer mice alive (MNA) (the number of individual mice captured in a month plus those mice captured on at least one previous and one subsequent occasion) and estimated standing prevalence (ESP) (minimum number infected divided by MNA), Fort Lewis, June 1994–October 1997.

Figure 3. Minimum number of deer mice alive (MNA) (the number of individual mice captured in a month plus those mice captured on at least one previous and one subsequent occasion) and estimated standing prevalence (ESP) (minimum number infected divided by MNA), Molina, June 1994–October 1997.
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adult hispid cotton rats, Hantaan virus and Apodemus agrarius, and Puumala virus and Clethrionomys glareolus indicates that whereas viremia may diminish over time, virus can still be detected in various organs, including the salivary gland, for several months after infection (11-14). Given the relatively brief life span of rodents, infection and concomitant infectivity for a few weeks or months would provide a mechanism for seasonal, albeit not transseasonal, persistence of hantaviruses. Passive acquisition of maternal antibody may protect the offspring of infected dams early in their lives, but when antibody wanes, they enter the adult population as susceptibles. Infected later in life, they can become persistent shedders of virus and sources of infection for others in the population.

Deer mice infected with SNV when very young likely are able to serve as reservoirs of the virus for the remainder of their lives. Although our studies do not distinguish between death and dispersal, the life span of many deer mice at these sites may not be much more than a month. However, because some deer mice live for 1 or 2 years, longevity of even a small proportion of the deer mouse population may provide a transseasonal mechanism for virus persistence.

A second mechanism of virus transmission, an epizootic one, depends on short-term infections of deer mice infected as subadults or as adults. At periods of deer mouse population peaks (e.g., at the end of the breeding season, in late summer and fall, and during period of decreased availability of food), male mice fight one another for breeding partners, food, and territory. This premise is supported by results of serologic tests of recaptured deer mice at Fort Lewis and at Molina. At Fort Lewis, 48.8% of the deer mice and 47.8% of the recaptured deer mice were male, but 58.3% of the seropositive deer mice were male. At Molina, 45% of the deer mice and 46.3% of the recaptured deer mice were male, but 60% of the seropositive deer mice were male. These data support the hypothesis that male deer mice contribute more to the epizootic cycle of SNV than female deer mice. However, the lack of association between sex, wounds, and antibody at either Fort Lewis or Molina indicates that individual mice of either sex may fight and, through this mechanism or another, become infected with a hantavirus. That most mice with antibody to SNV are male supports the suggestion that fighting among mice, biting, and scratching can lead to hantavirus transmission from an infected to an uninfected, wounded mouse (4). The limited time these mice may be able to transmit virus might be sufficient to maintain virus infection in the population.

When deer mouse populations decrease precipitously because of decreased availability of food and water, the likelihood that SNV will disappear from the population increases. However, a few long-lived, persistently infected deer mice can serve as reservoirs until conditions are suitable for the populations to recover.

Our data appear to support such a unified hypothesis. Fluctuations in IgG antibody prevalence in deer mice at Fort Lewis and at Molina have lagged somewhat behind but have been similar to fluctuations in deer mouse population. In male deer mice at Fort Lewis and at Molina, most seroconversions (recent infections) occurred during the summer or fall, whereas in female deer mice, most occurred between fall and spring. During winter, Colorado deer mice reduce their home range, aggregate in nests, and enter short-term torpor—strategies that together temper reduced food availability and energy loss due to cold (2). Although we did not find deer mice that had been infected for more than 3 months, we recovered a few more than 1 year (some nearly 2 years) after they first were trapped; thus, under natural conditions and despite the usual declines caused by predation, cold, heat, and decreases in food, deer mice that reach adulthood can live as long as 2 years (2), a period sufficient to allow SNV to survive adverse conditions of low populations and the resulting decreased number of susceptibles. Furthermore, whereas the overall seroprevalence of IgG antibody to SNV in deer mice at Fort Lewis was 6.8% (12 of 165) and in deer mice and pinyon mice, respectively, at Molina 7.2% (15 of 193) and 5.5% (3 of 52), the rate of seroconversion among deer mice at Fort Lewis was 16.3% (8 of 41) recaptures, and among deer mice and pinyon mice, respectively, at Molina 9.9% (7 of 64) and 3.3% (1 of 29). These results suggest that the longer deer mice live, the greater the cumulative probability they will become infected with SNV.

The deer mouse, the most numerous mammal in North America, often described as a “quintessential generalist,” can survive on any dry land habitat in its range and invade and exploit areas disturbed by flood, fire, avalanche,
landslides, mining, construction, extreme grazing, or land development. In ecologically stable areas, deer mice may be limited by the presence of more specialized rodent species (2), but they are found from forests to grasslands, canyons to deserts, farmlands to farm houses and suburban homes, moving into the latter more often in fall but able to take up residence whenever an opportunity presents itself. Thus, the movement of SNV-infected deer mice into human residences itself creates a risk factor for HPS. The Fort Lewis and Molina sites have not been affected by obvious ecosystem perturbations in recent years, and deer mouse populations at these sites are not high, yet are considerably decreased from the apparently inordinately high levels of 1993 (J. Mills, pers. comm. 1997). Each site seems ecologically stable, but subtle changes may have gone unnoticed.

Deer mice are omnivores, storing food for winter consumption but known to feed on acorns, nuts, insects, other small invertebrates, carrion, fungi, bone, and various plant parts, including seeds, leaves, and bark, roots, and tubers (2,16). In one study, seeds accounted for 69% to 76% of stomach contents of deer mice in Colorado, insects for 14% to 25% (2). This proportion depends on the season (i.e., availability of food supply); deer mice are more likely to feed on insects and insect larvae in spring, seeds and berries in fall.

Notwithstanding the nature of deer mice to consume a variety of foods, they rely heavily on acorns, when oaks (Quercus sp.) comprise a significant proportion of their habitat (16). Further, in the northeastern United States, the quantity of mast seems directly related to population size of white-footed mice (P. leucopus) and eastern chipmunks (T. striatus) (17). Although Gambel’s oak is abundant at the Fort Lewis trapping sites and copious numbers of acorns were attached to the trees and on the ground in 1994, we did not observe acorns on trees there between spring 1995 and fall 1997. The chipmunk population at this site was, with the exception of a transient, moderate increase in June 1996, never high during our study period (June 1994 to October 1997). At the Molina sites, which do not have oaks, the chipmunk population declined considerably after June 1996 and did not return to its previous level. Chipmunks may serve as an indicator for the ready availability of acorns and other nuts or food in general.

The continued low population densities of deer mice at Fort Lewis and Molina are puzzling. If, for example, deer mouse population densities in surrounding areas are higher than at the study sites, one might expect deer mice from those areas to move into the area with the low population. However, at Fort Lewis 59% of adult and 5% of subadult deer mice were recaptured; at Molina 86% of adults and no subadult or juvenile deer mice were recaptured as adults. These data indicate that few, if any, young deer mice are immigrating to these sites or if they are, they did not survive long enough to be captured, and the survival rate of this species’ young is not high.

Temperature fluctuations that affect habitat characteristics can influence rodent breeding seasons (e.g., rate of plant growth, total available nutritional biomass). However, analyses of available data (not presented) did not provide obvious evidence for such direct relationships. In contrast, a paucity of precipitation at Fort Lewis, between March 1995 and October 1996, and at Molina between May 1995 and April 1997, coincided with the usual breeding season of deer mice, least chipmunks, and other rodents at these sites, and with lower rodent population densities between the end of 1995 and the end of 1997; antibody prevalence fluctuated in parallel. Whether the two consecutive relatively wet years 1996 and 1997 will bring about conditions suitable to increase rodent populations near Fort Lewis and Molina and lead to an increase in HPS in the near term has not been determined. Availability of water may be the sine qua non of plant food availability, reproductive preparedness, gravidity rates, and attendant intraspecific fighting among individual mice within an increased population. From data (not shown) collected at these sites since October 1997, indications are that both rodent population densities and antibody prevalence are increasing at both sites. If precipitation excess correlates with rodent population, density increases, and the prevalence of hantaviruses, we will be able to predict increases in risk for hantavirus infection in the human population.

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