Journal of Mammalogy, 91(2):467–481, 2010

Spatiotemporal variation in *Akodon montensis* (Cricetidae: Sigmodontinae) and hantaviral seroprevalence in a subtropical forest ecosystem

ROBERT D. OWEN*, DOUGLAS G. GOODIN, DAVID E. KOCH, YONG-KYU CHU, AND COLLEEN B. JONSSON

Department of Biological Sciences, Texas Tech University, Lubbock, TX 79409-3131, USA (RDO)
Martín Barrios 2230 c/ Pizarro, Barrio Republicano, Asunción, Paraguay (RDO)
Department of Geography, Kansas State University, Manhattan, KS 66506, USA (DGG, DEK)
Southern Research Institute, Department of Biochemistry and Molecular Biology, 2000 9th Avenue South, Birmingham, AL 35205-5305, USA (Y-KC, CBJ)
Department of Microbiology and Immunology, University of Louisville, Louisville, KY 40202, USA (CBJ)

* *Correspondent: rowen@tigo.com.py*

Relatively little information is available concerning the natural history or population ecology of the montane akodont, *Akodon montensis*, a sigmodontine species that harbors Jaborá and Ape Aime hantaviruses. On the basis of mark-recapture sampling of 3 locales during 2 years, this report provides comparative data on populational and other characteristics of the species near its distributional limit, in the Upper Paraná Atlantic Forest in Paraguay. We found *A. montensis* to be almost exclusively terrestrial (nonarboreal) and to vary in population density among locales and between years, findings consistent with earlier studies. Population density was not related either to neotropical seasonality or to most precipitation variables, but sex ratio varied seasonally, which has not been reported previously. We evaluated the seroprevalence of hantavirus in *A. montensis*, in association with external, populational, and individual variables. As in most previous studies of rodent–hantavirus systems, seroprevalence varied among sampling locales and was higher among males. However, unlike previous reports, especially those from North America, we did not find a correspondence between seroprevalence and population density, seasonal variation, or most precipitation variables. Simple and direct associations between seroprevalence levels and either seasonality or precipitation may be less characteristic of humid neotropical or subtropical systems. Our analyses revealed that seropositive animals exhibit several noteworthy behavioral and life-history modifications, in particular that they have larger home ranges than do seronegatives; exhibit greater maximum distance moved; have greater home range displacement between sampling sessions, particularly those individuals that are seroconverting during the same period; and have greater longevity, especially among males. In general, these characteristics could be postulated as favorable to the capacity of the virus to be transmitted horizontally to other hosts. DOI: 10.1644/09-MAMM-A-152.1.

Key words: *Akodon montensis*, hantavirus, land conversion, Mbaracayú Biosphere Reserve, Paraguay, Rodentia, Sigmodontinae, Upper Paraná Atlantic Forest, viral ecology

The sigmodontine rodent genus *Akodon* is a large, diverse, and geographically widespread group distributed throughout South America from Colombia to southern Argentina and Chile (Musser and Carleton 2005). As currently understood, the montane akodont, *Akodon montensis*, is limited to an area in southern Brazil (Geise et al. 2001; Rieger et al. 1995), northeastern Argentina (Massoia and Fornes 1962; Pardiñas et al. 2003), and eastern Paraguay (Gamarra de Fox and Martin 1996), generally corresponding to the historical distribution of the Upper Paraná Atlantic Forest (UPAF) ecoregion (Di Bitetti et al. 2003). In Paraguay it occurs in most of the eastern portion of the country (east of the Paraguay River; Fig. 1).

Because the specific status of *A. montensis* has been recognized only recently, relatively little information is available concerning the natural history or population ecology of the species. It is considered to be a habitat generalist

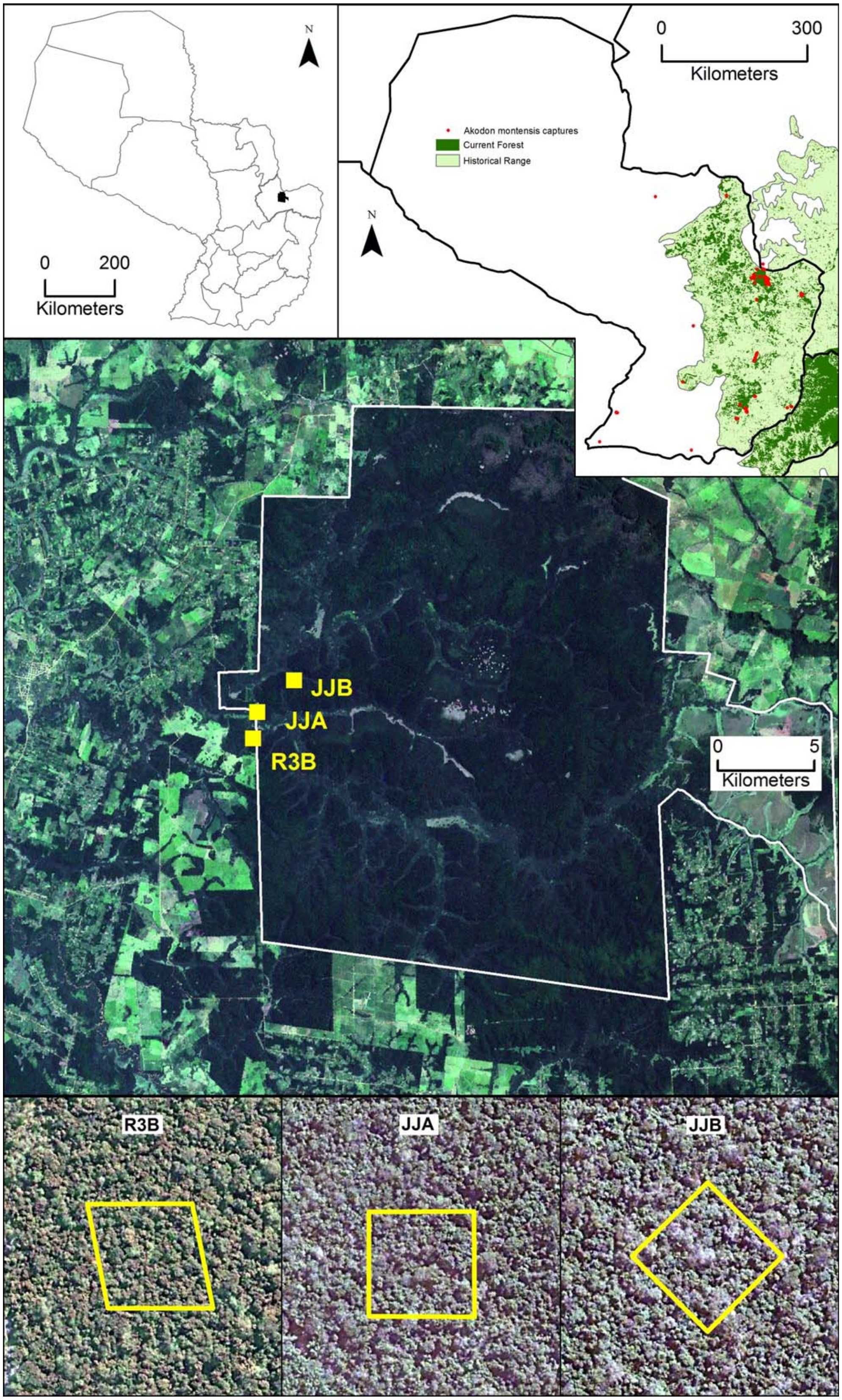

FIG. 1.—Maps showing (upper left) location of study site; (upper right) original and remaining extent of the Upper Paraná Atlantic Forest (Eva et al. 2003; Olson et al. 2001) and known distribution of *Akodon montensis*, within Paraguay (R. D. Owen, pers. obs.); (center) satellite images showing locations of 3 sampling locales (R3B, JJA, JJB) in and near the Reserva Natural del Bosque Mbaracayú (white outline); and (lower images) forest in and around each of the sampling locales.

(Dalmagro and Vieira 2005; Puttker 2007), favoring sites with increased litter and dense herbaceous cover (Gentile and Fernandez 1999), both of which are characteristic of forest edges and disturbed areas (Malcolm 1994) where it is often found abundantly (Bergallo and Magnusson 1999). It can occur abundantly in second-growth areas and often near human habitation and/or agricultural edges (Nitikman and Mares 1987).

Akodon montensis is a species that hosts 2 distinct strains of hantavirus (Jaborá and Ape Aime—Chu et al. 2006, 2009). Moreover, it extensively inhabits an ecoregion that is home to approximately 30 million human inhabitants (Di Bitetti et al. 2003) and undergoing rapid land-cover change that may promote hantavirus prevalence in the host species (Goodin et al. 2006, 2009). Therefore, we undertook this study to investigate some of the factors that may affect both population and individual characteristics, and hantavirus occurrence and prevalence levels, within *A. montensis*.

In North America outbreaks of hantavirus pulmonary syndrome (HPS) have been correlated with precipitation patterns (Eisen et al. 2007; Engelthaler et al. 1999; Gubler et al. 2001; Yates et al. 2002), and remotely sensed indices of vegetation greenness (a surface property closely related to interannual climatic variation) have been correlated with HPS cases, as have topography (elevation and aspect) and host macrohabitat (Calisher et al. 2002; Eisen et al. 2007; Glass et al. 2000, 2002; Mills et al. 1997). However, a growing awareness has emerged that increases in host–rodent population density driven by climate-induced resource availability do not necessarily translate into an increase in viral infection rates within the host–rodent population, or to increased occurrence of HPS (Davis et al. 2005; Stapp 2007).

Habitat or landscape characteristics also might promote or constrain the prevalence of hantaviruses in their host populations (Goodin et al. 2006, 2009; Langlois et al. 2001; McIntyre et al. 2005, 2009). Anthropogenic landscape disturbances have been linked to presence of hantavirus (Goodin et al. 2006), especially in tropical or subtropical forest (Suzán et al. 2006). In contrast, human disturbance is negatively correlated with hantavirus presence in a North American desert environment (Lehmer et al. 2008). In general, occurrence and prevalence of hantaviruses within areas of reservoir endemism may be related more complexly to a combination of environmental and landscape factors in South America, a region with a complex biogeographic history, particularly for sigmodontine rodents (D'Elía 2003).

Ecological disturbances, especially those of anthropogenic origin, might play a role in the emergence of hantaviruses as human pathogens (Mills 2006; Ruedas et al. 2004; Sauvage et al. 2007; Suzán et al. 2008, 2009). Given the accelerating pace of anthropogenic changes to the environment, especially in the UPAF of Paraguay (Huang et al. 2007), it is imperative that we identify ecological variables associated with such risk.

Effects of the virus on host individuals and populations are poorly understood. Although most studies have failed to reveal effects on the host of hantaviral infection of such variables as respiratory function (O'Connor et al. 1997), survivorship (Douglass et al. 2001), or dispersal (Lonner et al. 2008) in adult *Peromyscus maniculatus*, physical condition in *Myodes glareolus* (Yanagihara et al. 1985), and maturation rates, survival, and fecundity in adult *Rattus norvegicus* (Childs et al. 1989), a body of evidence is emerging that hantaviruses have subtle but significant effects on their hosts. For example, infected animals have lower survival rates for adult *M. glareolus* (Kallio et al. 2007) and neonatal *Mus musculus* (Kim and McKee 1985; Tsai et al. 1982), lower body mass in juvenile *P. maniculatus* (Borucki et al. 2000), slower weight gain in juvenile *R. norvegicus* (Childs et al. 1989), elevated testosterone levels in *R. norvegicus* (Easterbrook et al. 2007), and less weight gained after seroconversion in *P. maniculatus* (Douglass et al. 2007).

A variety of behavioral and ecological traits in rodent hosts, such as aggression or ranging, may be factors that alter risk of infection (Escutenaire et al. 2002; Hinson et al. 2004). Thus, identification of both ecological and behavioral traits associated with viral presence and prevalence in a host population is necessary in determining why certain hosts within a population are more susceptible to infection than are others and which factors facilitate or inhibit viral transmission and influence other aspects of infectious disease ecology and epidemiology (Abbott et al. 1999; Giuggioli et al. 2005).

Previous studies examining ecological variables (e.g., Armién et al. 2004; Biggs et al. 2000; Boone et al. 2000; Calisher et al. 1999; Loehman 2006; Mills et al. 1999, 2007; Olsson et al. 2005; Root et al. 1999; Suzán et al. 2006; Yates et al. 2002) typically have focused on a macroscopic scale, identifying regional variables associated with hantavirus presence and prevalence in host populations. The likelihood of detecting hantaviral antibodies in hosts has been correlated with rodent density, biodiversity, topography, and vegetation at a macroscopic scale (Biggs et al. 2000; Boone et al. 2000; Glass et al. 2000; Olsson et al. 2002, 2005; Peixoto and Abramson 2006; Root et al. 1999; Suzán et al. 2006, 2008, 2009; Yates et al. 2002), but relatively few published studies have examined possible ecological associations of host or hantavirus presence or prevalence at a finer scale of resolution, such as the scale of an individual host's territory (Abbott et al. 1999; Gottesman et al. 2004; Lozada et al. 2000) or an individual animal. A consideration of scale is crucial, however, to our understanding of the environmental variables associated with disease risk (Giuggioli et al. 2005). Differences may exist in the relative influences of landscape versus local variables (Orrock et al. 2000; Suzán et al. 2006) in explaining seasonal or other patterns in human cases (Davis et al. 2005).

Several studies have emphasized the importance of examining host and viral associations at multiple geographic scales (Goodin et al. 2006, 2009; McIntyre et al. 2005, 2009). We suspected that both the abundance of the hantavirus reservoir species and seroprevalence levels within the reservoir population are associated complexly with a suite of biotic and abiotic environmental variables, and that these

Table 1.—Summary of the vegetation characteristics and sampling dates of the 3 mark-recapture sampling sites. See Fig. 1 for location of grids.

Site	Dominant vegetation	General description	Dates (2005)	Dates (2006)
Jejui Mi A (JJA)	*Chusquea ramosissima* *Sorocea bonplandii* *Merostachys* sp.	Native forest. Low levels of human disturbance. Evidence of selective logging visible, few large trees.	15 Mar.–22 Mar. 7 Aug.–14 Aug. 1 Oct.–8 Oct.	15 Feb.–22 Feb. 9 Jul.–16 Jul. 22 Nov.–29 Nov.
Jejui Mi B (JJB)	*Sorocea bonplandii* *Merostachys* sp. *Heliconia* sp.	Native forest. Lowest levels of human disturbance. Minimal evidence of selective logging visible, large trees remaining.	15 Mar.–22 Mar. 7 Aug.–14 Aug. 1 Oct.–8 Oct.	15 Feb.–22 Feb. 9 Jul.–16 Jul. 22 Nov.–29 Nov.
Rama III B (R3B)	*Sorocea bonplandii* *Bromelia* sp. *Merostachys* sp.	Native forest cover, but evidence of more recent human disturbance by selective logging and nearby road building. Characterized by numerous internal edges and canopy openings resulting in a dense understory.	12 Feb.–18 Feb. 14 Jun.–21 Jun. 12 Sep.–19 Sep. 10 Nov.–17 Nov.	27 Feb.–6 Mar. 19 May–26 May 27 Jul.–3 Aug. 3 Dec.–10 Dec.

associations might be differentially detectable at the population and individual levels. This study, therefore, examines characteristic associations at the scales of the host population (as sampled by mark-recapture grid) and the host individual.

We evaluated patterns of population abundance, density, sex ratio, individual home-range size, home-range displacement over time, and longevity of *A. montensis*, the primary reservoir for Jaborá virus in eastern Paraguay. Comparisons were made between sexes, among habitat types, between years, and among precipitation seasons, and we then compared these patterns between seronegative and seropositive host rodents.

Materials and Methods

Study area.—This research was conducted within the Reserva Natural del Bosque Mbaracayú (RNBM), a World Biosphere Reserve located within Departamento Canindeyú in northeastern Paraguay. Although no confirmed HPS cases have been reported in Canindeyú, the region is significant for research because of the presence of rodent vectors for hantavirus (Chu et al. 2003) and anecdotal information on possible cases in indigenous populations (M. Hurtado, Arizona State Univ., pers. comm.).

RNBM is located within the UPAF ecoregion (Fig. 1) and is one of several privately managed protected areas in Paraguay. RNBM covers about 590 km^2 and is divided into an inner conservation core, dominated by 1 large forest fragment, the largest extant UPAF forest fragment in Paraguay, and an outer buffer area. Access to the inner core area is limited, and forest conversion does not occur in this area due to its protected status. Within the buffer zone of the reserve are a number of inhabited areas, including indigenous (Ache, Guarani) and rural Paraguayan communities, minifundias (small, subsistence farms), latifundias (larger commercial farms), and estancias (ranches). Although prohibited by law since 2004, some land-use and land-cover conversion continues in the outer buffer zone of the reserve, typically near the inhabited areas.

Management of the area as a biosphere reserve provides some relative control to compare areas surrounding the Reserve that are undergoing rapid land-cover conversion. This conversion is primarily of forested land to agriculture (crop or pasture), interspersed by smaller forest remnants (Koch et al. 2007). The intensive mosaic of surface cover characteristic of this area provides a variety of potential rodent habitat, thus providing an excellent opportunity to investigate the habitat characteristics of a predominant reservoir and vector for hantavirus, *A. montensis*.

Our study was conducted as part of a larger research program to investigate the effect of anthropogenic disturbance on hantaviral dynamics in the UPAF of Paraguay. Data collection consisted of 2 components: mark-recapture sampling of rodents for ecological (individual, population, and community) and seroprevalence analysis; and vegetation sampling to determine the composition and structural characteristics of habitat. We selected 3 sites on which to establish rodent trapping grids (Table 1; Fig. 1). These locales were chosen to represent a cross-section of potential microhabitat types representing a continuum of human disturbance. Two of the sites, Jejui Mi A (JJA) and Jejui Mi B (JJB), were native forest sites chosen for their relatively low levels of human disturbance. Both of these sites show some evidence of selective logging, although indicators of human disturbance (stumps, etc.) are much more evident on JJA than JJB. On both sites the observable human disturbance typically is more than 15 years old, and the general history of land management in this area would suggest that this selective logging probably occurred between 1960 and 1990 (Cartes 2003). Rama III B (R3B), another native forest site along the edge of the reserve, was included due to its more recent disturbance by human forest conversion. Detailed information on these 3 sites is provided in Goodin et al. (2009).

Mark-recapture sampling.—A mark-recapture grid was established at each of the 3 field locales to collect demographic and ecological information and physical samples from rodents. These mark-recapture grids were monitored on a rotating basis between February 2005 and December 2006 (Table 1). To account for the sensitivity of rodent populations to seasonal variability, the trapping sessions were spaced throughout the year. On the basis of the 30-year climatic mean for precipitation in the RNBM area, we defined 3 precipitation seasons (Fig. 2) of equal length as potentially important to both rodent and hantavirus annual fluctuations: dry (June–September), wet (October–January), and transitional (February–May).

Each of the 3 mark-recapture grids consisted of an 11 × 11 array of trap stations spaced 10 m apart over an area of ≈1 ha. Except for the initial session (locale R3B, February 2005, 7 nights sampling), each trapping campaign consisted of 8 consecutive nights to enable robust estimates of population sizes. Although our primary focus in this paper is on *A. montensis*, our trapping protocol was designed to sample the entire nonvolant small-mammal community in each locale. Therefore, each trapping station had 1 standard Sherman live trap (7.5 × 9.0 × 23.0 cm; H. B. Sherman Traps, Tallahassee, Florida) placed on the ground, and another placed in branches or vines 2–3 m above ground, to sample species that are primarily arboreal. Traps were baited each evening with a mixture of rolled oats and peanut butter.

Traps were checked each morning and animals processed and released at the point of capture. For each capture, the species, date, grid, trap station number, and trap placement (ground or aboveground) were noted. Upon first capture, a passive integrated transponder (PIT) tag (12.5 × 2.0 mm; Biomark Inc., Boise, Idaho) was implanted subdermally, and a small (1–2 mm) snip was taken from the tip of the tail to provide material for DNA sequencing for confirmation of specific identity, if needed. Upon each recapture, the PIT tag number was recorded along with the specific identity, sex, age class, reproductive condition, weight, and general health of the animal, and the presence of any lesions or scars indicative of agonistic encounters possibly pertaining to hantavirus transmission (Hinson et al. 2004). If the capture was the first for the animal during the 8-night sampling session, a blood sample was obtained by retro-orbital bleeding and preserved in a 2-ml cryotube in liquid nitrogen. All field protocols followed American Society of Mammalogists guidelines for the use of wild mammals in research (Gannon et al. 2007) and were approved by the Texas Tech University Animal Care and Use Committee.

Analyses of serostatus for hantaviral antibodies.—Blood samples were tested for the presence of hantaviral antibodies using an immunofluorescence assay (IFA) that screens for hantaviral antibodies in radiation-inactivated Andes virus-infected Vero E6 cells, as described in Chu et al. (2003). Throughout the study presence of antibodies to Andes virus by IFA was used as an indicator of reservoir serostatus.

Estimation of Akodon montensis *population and individual parameters.*—Population estimates were calculated using the program MARK (White and Burnham 1999) for each species encountered during each sampling session on each grid. We used the Huggins closed-capture model, allowing variable capture probabilities among sampling events (trapping nights) and constraining the probability of capture and recapture to be equal. Although this model appears appropriate for our data and concordant with our understanding of the biological characteristics of the populations under study, it did not provide robust or reliable estimates for populations in which 4 or fewer individuals were encountered. In these instances, the minimum number known alive (MNKA) was used as the population estimate.

Population density was calculated for *A. montensis* as the estimated population divided by the effective grid size. The effective grid size assumes that the trap-station configuration is effectively sampling an area that extends beyond the borders of the grid by a distance equal to one-half of the mean maximum distance moved (MMDM), calculated using only those individuals with 3 or more captures (Schnell et al. 2008). This maximum distance moved is calculated as the hypotenuse of a right triangle, the other 2 sides of which are the differences between the greatest and smallest values of row and column where an animal was encountered. Preliminary analyses of MMDM indicated variation among sampling sessions in 1 locale (R3B), so effective grid size was calculated separately for each session in each locale, and population densities were calculated accordingly.

Home range was estimated as the 75% utilization distribution (UD) for each individual that was encountered at 3 or more trap stations during a particular session. We used program CALHOME (http://nhsbig.inhs.uiuc.edu/wes/home_range.html) for this, and used the lowest least-squares cross-validation (LSCV) score as the criterion for selecting the bandwidth, or smoothing parameter. Generally, the default values in CALHOME resulted in the lowest LSCV score.

Home-range displacement was calculated as the straight-line distance between the UD centroid of an individual during 1 trapping session and the centroid during a subsequent session. UD centroids were defined as the arithmetic centroid of the 10% UD, also calculated using program CALHOME and the LSCV score criterion.

Longevity (in months) was estimated for each individual at the time of each trapping session as the minimum time that the animal was known to have been alive. For the first capture, an animal was assessed to have been alive for a minimum of 1 month if it was a juvenile, 2 months if subadult, and 3 months if adult. For captures in subsequent sessions the number of months between sessions was added to the age estimated at 1st capture.

The sex ratio was calculated for each *A. montensis* population on the basis of the numbers of individuals encountered (MNKA) during the trapping session. Sex ratio was expressed as the proportion of males in the population.

Seroprevalence was calculated as the ratio of seropositive individuals to all individuals screened for serostatus. See Chu et al. (2003, 2006) for description of analytic techniques and criteria for determining presence of antibodies to hantaviral antigens.

Precipitation history was expressed as several variables for each trapping session. These included the total accumulated rainfall of the current month, most recent 2 months (including current month), most recent 3 months, most recent 6 months, most recent 12 months, and of the same 4-month precipitation season (T, D, or W) of the previous year. Precipitation values

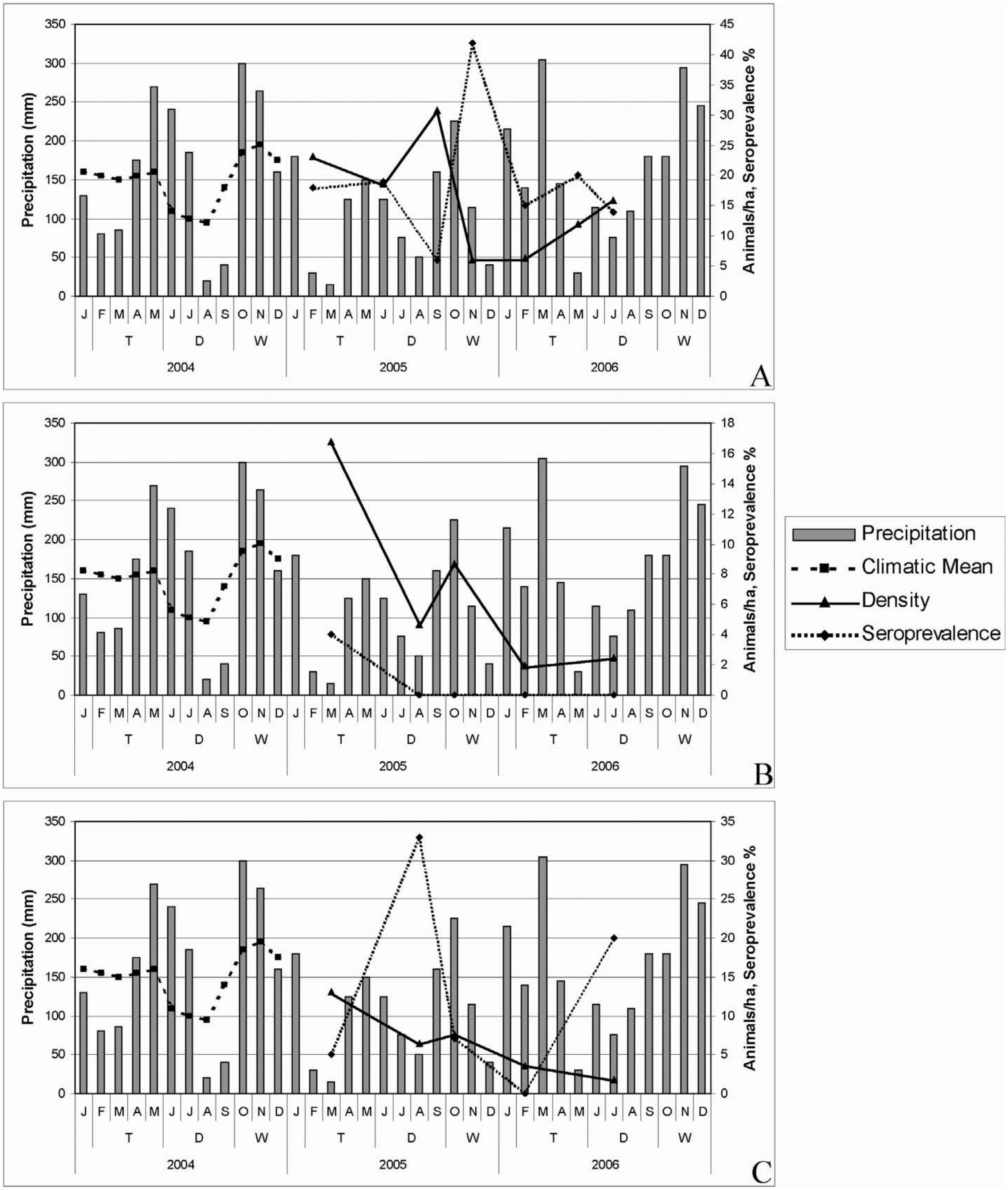

Fig. 2.—Monthly precipitation for the Mbaracayú study area for 2004–2006; climatic (30-year) mean precipitation (overlaid in year 2004); and estimated population densities of *Akodon montensis*, and percent hantaviral seroprevalence levels in *A. montensis* for 2005–2006 for 3 sampling locales: (A) Rama III B (R3B), (B) Jejui mi A (JJA), and (C) Jejui mi B (JJB). Annual precipitation values are derived from the TRMM 3B43 data set. Climatological averages are from the 1961–1990 CRU 0.5-degree mean monthly climate data set (New et al. 1999). Note that the right-hand vertical scale (Animals/ha, Seroprevalence %) is different in each of the 3 graphs.

for 2004, 2005, and 2006 were obtained from the TRMM 3B43 data set, and climatological averages are from the 1961–1990 CRU 0.5-degree mean monthly climate data set (New et al. 1999).

All distributions of quantitative variables characterizing populations and individuals were skewed and/or kurtotic. These variables (population abundance and density, sex ratio, and seroprevalence, and individual home range, home-range displacement, and longevity) were transformed to normalized rank variables using the formula of Blom (1958), and these transformed variables were used in the analyses described below. Because most analyses of variance (ANOVAs) described below were of unbalanced design, tests for significant differences among group means were based on comparisons of least-squared means, which is appropriate for unbalanced design (Sokal and Rohlf 1981). All statistical analyses were done using SAS 9.1.3 (SAS Institute, Cary, North Carolina).

Analyses of population-level characteristics.—Two-way ANOVAs were used to investigate the variation of *Akodon* population abundance and density across years and locales, and sex ratio and seroprevalence across seasons and locales. One-way ANOVAs were used to compare seroprevalence across seasons for each locale separately. The ANOVA model can be applied to data without replications in certain circumstances (Sokal and Rohlf 1981). In our analyses the error term was derived from multiple random samplings of the population, in that each capture of a rodent is a sample of the rodent population, and each viral antibody screening is a sampling of the antibody prevalence in the host population. This derivation of the error sum of squares enables testing of both main effects and interaction in the ANOVAs. We accepted main or interaction effects to be significant only when the overall model was significant. Where ANOVA results were statistically significant for a main or interaction effect, least-squares means comparisons were used to identify significantly different groups. Stepwise regression analysis was used to evaluate the effects of seroprevalence and precipitation history on population abundance, density, and sex ratio.

Analyses of individual-level characteristics.—Two-way ANOVAs were used to assess the effects of sex and serostatus on *A. montensis* home-range size, across grids, seasons, and years, and for all data combined. Differences between means of seropositive and seronegative individuals, and between males and females, were compared using the least-squared means.

One-way ANOVAs were used to test for differences of MMDM between serostatus classes, sexes, and years, and among seasons and locales. Additionally, two-way ANOVAs were conducted to evaluate interaction effects between serostatus and other variables, and between sex and other variables.

Two-way ANOVAs were conducted to assess the relationship between longevity and the serostatus and sex of individuals for all data combined, and for each season, locale, and year separately. Because significant interaction was found between the effects of sex and serostatus on longevity in several of these tests, additional one-way ANOVAs of the effect of serostatus on longevity were run for each sex separately for each of the above comparisons.

Home-range displacement between sampling sessions (HRD) was evaluated by 1-way ANOVAs due to small available samples for this variable, which required that an animal be encountered and captured several times to calculate a 10% UD in 2 or more sampling sessions. Analyses were conducted of all individuals, and of males only, to determine whether seropositive and seronegative animals showed differences in the displacement of their home-range centroids from the previous sampling session. Additionally, we tested seropositive animals to determine whether those animals that seroconverted (from negative to positive) during the same period exhibited differential HRD relative to those that already were seropositive in the previous session.

RESULTS

General results.—In 38,720 trap nights of effort we recorded 1,018 captures (2.6% overall trap success) of 361 individuals, representing 7 species (6 sigmodontine and 1 didelphid) and 5 genera (Table 2). Of these, 862 captures (84.7%) and 289 individuals (80.1%) were *A. montensis*. Of the 862 captures of *A. montensis*, 842 (97.7%) were captured in traps placed on the ground and 20 (2.3%) in traps placed above the ground in branches or vines. The 862 captures of *A. montensis* included 289 individuals (80.1% of the total individuals captured) and 573 recaptures.

Akodon montensis *population characteristics.*—*Akodon* population abundance and density varied significantly between years and among locales, but not seasonally, nor were there significant interaction effects between the independent variables (Table 3). The best regression model for population abundance ($F_{1,15} = 7.02$; $P = 0.018$; $R^2 = 0.319$) included only 1 of the 8 independent variables available, Precip12, the total accumulated rainfall over the previous 12 months. Similarly, stepwise regression of population density indicated that the best model included only Precip12 ($F_{1,15} = 7.06$; $P = 0.018$; $R^2 = 0.320$).

In contrast to population abundance, the sex ratio in *Akodon* populations did not vary between years or among locales but did vary seasonally, with males comprising around 39% of the population in the transitional season compared with 52–54% in the dry and wet seasons (Table 3). Although the overall model was not significant for this analysis, the least-squares means test confirmed the transitional-season sex ratio as significantly different from the other seasons. Stepwise regression indicated that none of the tested variables was a significant predictor of sex ratio.

Population characteristics in relation to seroprevalence of Hantavirus.—*Akodon* population seroprevalence varies significantly among locales, with each of the 3 locales differing significantly from the others. Additionally, an interaction effect between locale and season on seroprevalence

Table 2.—Minimum number known alive (MNKA, number of animals encountered) and estimated population ($\hat{N}$) for all rodent species for each sampling session on 3 mark-recapture grids in the Reserva Natural del Bosque Mbaracayú, Canindeyú, eastern Paraguay. See Table 1 and Fig. 1 for description and location of grids and text for description of population estimation methods. For sessions, 1st digit identifies the year (2005 or 2006) and subsequent 2 digits the month of the sample. Seasons are T, transitional (February–May); D, dry (June–September); W, wet (October–January). Nomenclature follows Carleton and Musser (2005).

			Akodon montensis		*Oligoryzomys fornesi*		*Oligoryzomys nigripes*		*Calomys callosus*		*Oryzomys angouya*		*Oryzomys megacephalus*		
Locality	Session	Season	MNKA	$\hat{N}$	MNKA	$\hat{N}$	MNKA	$\hat{N}$	MNKA	$\hat{N}$	MNKA	$\hat{N}$	MNKA	$\hat{N}$	Total
Rama III B	502	T	34	34.2	1	1.0	1	1.0	0	0.0	0	0.0	0	0.0	36.2
Rama III B	506	D	30	30.4	5	5.3	0	0.0	0	0.0	0	0.0	3	3.0	38.7
Rama III B	509	D	43	68.2	9	12.6	0	0.0	0	0.0	0	0.0	2	2.0	82.8
Rama III B	511	W	12	14.8	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	14.8
Rama III B	602	T	11	11.1	0	0.0	0	0.0	0	0.0	0	0.0	1	1.0	12.1
Rama III B	605	T	22	23.8	1	1.0	0	0.0	0	0.0	0	0.0	1	1.0	25.8
Rama III B	607	D	26	27.0	1	1.0	1	1.0	3	3.0	0	0.0	0	0.0	32.0
Rama III B	612	W	31	35.9	2	2.0	0	0.0	0	0.0	0	0.0	1	1.0	38.9
Jejui mi A	503	T	23	26.8	1	1.0	2	2.0	0	0.0	1	1.0	4	4.0	34.8
Jejui mi A	508	D	6	7.2	1	1.0	0	0.0	0	0.0	0	0.0	5	5.0	13.2
Jejui mi A	510	W	16	17.1	7	7.9	0	0.0	0	0.0	0	0.0	2	2.0	27.0
Jejui mi A	602	T	4	4.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1.0	5.0
Jejui mi A	607	D	4	4.5	0	0.0	0	0.0	0	0.0	0	0.0	1	1.0	5.5
Jejui mi A	611	W	2	2.0	0	0.0	0	0.0	4	4.0	0	0.0	3	3.0	9.0
Jejui mi B	503	T	20	20.1	1	1.0	0	0.0	0	0.0	0	0.0	1	1.0	22.1
Jejui mi B	508	D	12	12.1	3	3.0	0	0.0	0	0.0	0	0.0	2	2.0	17.1
Jejui mi B	510	W	15	16.2	1	1.0	0	0.0	1	1.0	1	1.0	4	4.0	23.2
Jejui mi B	602	T	7	8.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1.0	9.0
Jejui mi B	607	D	5	5.7	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	5.7
Jejui mi B	611	W	3	3.0	0	0.0	0	0.0	0	0.0	1	1.0	0	0.0	4.0

of *Akodon* populations was significant (Table 3). To understand better the nature of this interaction, a one-way ANOVA was run to test for the effect of locale on seroprevalence for each season separately. Least-squared means tests showed significant differences among locales in the transitional and dry seasons but were not estimable due to insufficient samples in the wet season. Regression analysis failed to produce a significantly supported model of seroprevalence on the basis of precipitation history, or on the basis of *Akodon* abundance, sex ratio, and precipitation history.

Individual characteristics in relation to Hantavirus *infection.*—For all comparisons, seropositive animals had larger home ranges than seronegative ones (Table 4). For all data combined, seropositives had home ranges averaging twice as large as seronegatives, although the difference was marginally nonsignificant (overall model: $F_{3,57} = 1.44$; $P = 0.2410$; main effect: $F_{1,57} = 3.89$; $P = 0.0535$). Evaluation of the two-way comparisons by season, by locale, and by year demonstrated both seasonal and locational differences in serostatus relative to home-range size, with seropositive animals having significantly larger home ranges than seronegatives in the transitional season and in the population found in locale JJB.

Mean maximum distance moved varied significantly between serostatus classes (positive = 45.2 m, negative = 30.7 m; $F_{1,124} = 4.85$; $P = 0.0295$). MMDM differed between sexes (male = 39.5 m, female = 26.3 m; $F_{1,124} = 10.79$; $P = 0.0013$) and among seasons (wet = 47.6 m, dry = 32.2 m, transitional = 29.9 m; $F_{1,123} = 3.74$; $P = 0.0264$), and a significant interaction between these effects was found (overall model, $F_{1,120} = 5.75$; $P < 0.0001$; interaction effect, $F_{2,120} = 4.59$; $P = 0.0120$). One-way analyses of sex in each season subsequently indicated a significant difference between sexes only in the dry season (male = 42.7 m, female = 18.9 m; $F_{1,39} = 12.50$; $P = 0.0011$).

An interaction effect on individual longevity between sex and serostatus was highly significant (Table 5). Subsequent 1-way ANOVAs of the effect of serostatus on longevity for each sex separately indicated that serostatus significantly affected longevity for males but not for females.

Analyses of each season separately showed an interaction between the effects of serostatus and sex on longevity in the dry and wet seasons. Longevity differed significantly according to serostatus in the dry season (sexes combined), in males in the dry season, and in females in the wet season (Table 5). An interaction effect between serostatus and sex on longevity was significant in 1 locale (R3B) and 1 year (2005). Longevity differed significantly according to serostatus in males in 2 locales (R3B, JJB) and in both years.

With sexes combined, seropositive animals had a significantly greater mean HRD (63.1 m) than seronegatives (22.1 m) ($F_{1,7} = 9.56$; $P = 0.0175$). Seropositive males also showed significantly greater mean HRD (63.1 m) than seronegatives (14.1 m; $F_{1,5} = 8.01$; $P = 0.0366$). The difference in HRD for seroconverted animals (78.0 m) and those that were already positive (40.8 m) was marginally nonsignificant ($F_{1,3} = 7.52$, $P = 0.0712$).

Table 3.—Results of selected 2-way ANOVAs for population abundance and density of *Akodon montensis* across years and sampling locales and sex ratio and seroprevalence across seasons and locales, and 1-way ANOVAs of seroprevalence across seasons for each locale separately. *F*-values (with *d.f.*) are shown for overall model and main and interaction effects. Significance levels are indicated by asterisks: * $0.05 \geq P > 0.01$; ** $0.01 > P > 0.001$. Means of each group are shown. Means within the same row containing the same letter have nonsignficant differences between least-squared means ($\alpha = 0.05$); means with no letters (wet season in seroprevalence results) have differences between least-squared means that were not estimable.

Test	Model	Effects			Means and nonsignificant differences				
		Year	Locale	Interaction	2005	2006	JJA	JJB	R3B
Abundance	6.62** (5, 14)	10.53** (1)	10.67** (2)	1.11 (2)	24.7 a	12.5 b	10.3 c	10.8 c	30.7 d
Density	4.29* (5, 11)	11.34** (1)	5.64* (2)	0.44 (2)	13.6 a	6.3 b	6.9 c	6.5 c	16.1 d
		Season	Locale	Interaction	Transitional	Dry	Wet		
Sex ratio	1.61 (8, 11)	4.41* (2)	0.45 (2)	0.92 (4)	0.39 a	0.52 b	0.54 b		
					JJA	JJB	R3B		
Seroprevalence	11.14** (8, 8)	2.76 (2)	29.80** (2)	8.37** (4)	0.008 a	0.130 b	0.191 c		
				Transitional	0.020 a	0.025 ab	0.177 b		
				Dry	0.000 a	0.265 b	0.130 c		
				Wet	0.000	0.070	0.420		

Discussion

Ecology of Akodon montensis *in western UPAF.*—Little is known of the ecology or natural history of *A. montensis* because few studies have been conducted and the species has been recognized only recently as distinct from *A. cursor* (Christoff 1997; Geise 1995; Geise et al. 1998, 2001). Limited reports exist regarding habitat (Figueiredo and Fernandez 2004; Gentile and Fernandez 1999), abundance (Bergallo and Magnusson 1999), food habits (Carvalho et al. 1999; Emmons and Feer 1990; Talamoni et al. 2008; Vieira et al. 2006), and reproduction (Bergallo and Magnusson 1999; Gentile and D'Andrea 2000). Notably, none of these reports is based on studies in Paraguay or other localities near the western edge of the species' distribution (Cartes 2003; Di Bitetti et al. 2003). Thus, this report provides comparative data on populational and other characteristics of the species near its distributional limit. This may be of particular importance as the UPAF becomes increasingly deforested and fragmented, as these changes generally result in increased presence of forest edges and second-growth areas (Malcolm 1994) that could favor *A. montensis* populations (Gentile and Fernandez 1999; Umetsu et al. 2008), especially if the fragments are connected by corridors of suitable habitat (Pardini et al. 2005).

Our data confirm that *A. montensis* is a consistently terrestrial species, consistent with the findings of Vieira and Monteiro-Filho (2003) and Oliveira et al. (2007), only rarely venturing into low branches or vines. Pardini et al. (2005) found *A. montensis* to be most abundant in small, connected forest fragments with dense understory and sparse overstory. In our study population density varied considerably among our

Table 4.—Two-way (sex × serostatus) ANOVAs of home-range size in *Akodon montensis*. *F*-values are shown for overall model and main and interaction effects, with *d.f.* in parentheses for overall models and treatment; *d.f.* = 1 for all main and interaction effects. For each test, *F*-values (on the basis of ranked home-range values) are shown for sex, serostatus, and interaction effect. Significance levels are indicated by asterisks: * $0.05 \geq P > 0.01$; ** $0.01 \geq P > 0.001$. Mean home-range values (m^2) are shown for males and females and for seropositive and seronegative animals. Means within the same row followed by the same letter have nonsignificant difference (on the basis of least-squares means of ranked values, $\alpha = 0.05$). Means within the same row followed by different letters have significantly different means of ranked values. Means with no letters indicate that 1 or both least-squared means were not estimable. Line indicates that *F*-value was not estimable or no seropositive individuals were encountered.

Test	Model	Serostatus	Sex	Sero × sex	Positive	Negative	Male	Female
Combined data	1.44 (3, 57)	3.89	0.86	1.34	954 a	484 a	630 b	444 b
By season								
Transitional	3.45* (3, 34)	7.77**	3.74	0.94	1,128 a	315 b	355 c	441 c
Dry	4.26* (2, 12)	0.37	8.41*	—	643	529	857	284
Wet	1.07 (2, 5)	0.00	1.84	—	1,111	1,016	1,358	697
By locale								
R3B	0.48 (2, 29)	0.11	0.55	—	705	558	746	339
JJA	0.06 (1, 11)	—	0.06	—	—	534	512 a	553 a
JJB	3.02 (3, 12)	8.57*	0.50	0.01	1,578 a	297 b	380 c	504 c
By year								
2005	0.81 (2, 41)	1.18	0.14	—	723	467	563	391
2006	0.91 (3, 13)	0.76	0.60	2.46	1,128 a	539 a	860 b	549 b

Table 5.—Two-way (sex × serostatus) and 1-way (serostatus) ANOVAs of longevity in *Akodon montensis*. Significant differences are indicated by asterisks: * $0.05 \geq P > 0.01$; ** $0.01 \geq P > 0.001$; *** $0.001 \geq P$. F-values are shown for overall model and main and interaction effects, with *d.f.* in parentheses for overall models and treatment; *d.f.* = 1 for all main and interaction effects. For each 2-way test, F-values (on the basis of rank-normal transformed longevity values) are shown for overall model, sex, serostatus, and interaction effect, and mean longevity values (in months) shown for seropositive and seronegative animals, and for males and females. For 1-way tests, F-value is shown for serostatus, and mean longevity is shown for seropositive and seronegative animals. Means within the same row followed by the same letter have nonsignificant differences (on the basis of least-squares means of ranked values); means within the same row followed by different letters have significantly different means of ranked values. Line indicates F-value not estimable or no seropositive individuals were encountered.

Test	Model	Serostatus	Sex	Sero × Sex	Positive	Negative	Male	Female
Combined data	12.43*** (3, 269)	6.00*	3.23	13.16***	4.31 a	3.04 b	3.18 c	3.23 c
Males	35.26*** (1, 129)	35.26***			4.64 a	2.78 b		
Females	0.52 (1, 140)	0.52			3.12 a	3.24 a		
By season								
Transitional	2.03 (3, 114)	1.06	0.70	0.85	3.79 a	3.10 a	3.33 b	3.07 b
Males	(1, 47)	3.72			3.92 a	3.14 a		
Females	(1, 67)	0.00			3.00 a	3.07 a		
Dry	9.57*** (3, 109)	10.70**	0.15	4.68*	5.31 a	3.08 b	3.21 c	3.62 c
Males	(1, 59)	33.76***			5.75 a	2.59 b		
Females	(1, 50)	0.33			4.00 a	3.58 b		
Wet	4.28* (3, 38)	0.77	5.88*	12.72***	2.83 a	2.75 a	2.71c	2.81 d
Males	(1, 19)	4.18			3.50 a	2.53 a		
Females	(1, 19)	11.32**			1.50 a	2.95 b		
By locale								
R3B	7.57*** (3, 160)	2.46	2.92	8.12**	4.32 a	3.07 a	3.35 b	3.22 b
Males	(1, 80)	21.33***			4.57 a	2.88 b		
Females	(1, 80)	0.52			3.20 a	3.22 a		
JJA	0.80 (2, 48)	0.00	1.57	—	3.00 a	3.25 a	2.82	3.24
Males	(0, 21)	—			—	2.82		
Females	(1, 27)	0.00			3.00 a	3.25 a		
JJB	3.65* (3, 54)	2.87	0.03	2.70	4.43 a	2.92 a	2.93 b	3.26 b
Males	(1, 25)	8.55**			5.00 a	2.45 b		
Females	(1, 29)	0.01			3.00 a	3.28 a		
By year								
2005	5.96*** (3, 195)	2.53	2.05	8.37**	3.92 a	3.03 a	3.06 b	2.23 b
Males	(1, 92)	20.23***			4.21 a	2.77 b		
Females	(1, 103)	0.61			3.14 a	3.23 a		
2006	6.50*** (3, 70)	2.33	0.50	2.53	5.30 a	3.05 a	3.46 b	3.24 b
Males	(1, 35)	14.62***			5.56 a	2.79 b		
Females	(1, 35)	0.00			3.00 a	3.25 a		

3 sampling locales and between our 2 sampling years. These results indicate the importance of vegetational structure (Goodin et al. 2009; Püttker et al. 2008).

Although increased precipitation has been linked to rodent population irruptions in southern South America (Jaksic and Lima 2003), no published reports of such irruptions in Paraguay exist, and we are unaware of any reported relationship between precipitation and population density in *A. montensis*. In our study 12-month cumulative total precipitation was a significant predictor of population density. Precipitation levels were considerably lower in 2005 than in 2004 (Fig. 2), and population densities of *A. montensis* were significantly lower in 2006 than in 2005 (Table 3). Population density did not vary significantly by season (defined by climatic precipitation values), perhaps reflecting the high level of interannual variation in monthly or seasonal precipitation.

In contrast to general populational trends, we found that sex ratio varied seasonally, with females constituting around 60% of the population during the transitional season compared with about 50% during the rest of the year. Nevertheless, none of the precipitation variables predicted sex ratio, suggesting that the rise in female predominance in the population is an annual phenomenon not related proximally to precipitation. Although the annual fluctuation in sex ratio could be expected to relate to an annual fluctuation in population abundance, we did not find this to be the case as neither population abundance nor density was found to vary significantly by season. One possible explanation of a sex ratio favoring females is the presence of XY* females in the population. These females produce approximately a 2:1 ratio of viable female to viable male embryos (Bianchi 2002) and thus could affect populational sex ratio in the manner we observed, although we are unaware of any reports of temporal (including seasonal) variability in either the relative abundance of XY* females in *A. montensis* populations or in their relative reproductive activity. Alternative explanations of these results are reduced trappability of males or increased trappability of females (or both) during the transitional season, rather than fluctuation in the actual sex ratio. We cannot definitively exclude either of these possibilities on the basis of our data, and our provisional conclusion that fewer males than females are present in the population during the transitional season needs further investigation.

Püttker et al. (2006) found that *A. montensis* had the lowest MMDM (26.3 m) of 5 rodent species in an Atlantic Forest setting but did not find differences between sexes or among seasons. In contrast, we found differences in MMDM between sexes and among seasons and some interaction between these 2 effects. With seasons combined, males moved farther than females. Evaluated seasonally, this trend held true in the dry season but not in the other 2 seasons. Overall (sexes combined), however, individuals tended to move farther in the wet season than in the other two seasons.

Ecological and behavioral aspects of Hantavirus *infection*.—In contrast to previous studies (e.g., Engelthaler et al. 1999; Gubler et al. 2001; Suzán et al. 2009; Yates et al. 2002), we did not find that population abundance or density was significantly associated with seroprevalence levels in *A. montensis*. This finding eliminates hypotheses of density-dependent seroprevalence levels as applicable in the rodent–virus system we are investigating. We hypothesize that such associations are less generally applicable than had been proposed by the earlier investigators, at least in humid subtropical ecosystems that harbor communities of high species richness (Redford et al. 1990), especially within the sigmodontine radiations (D'Elía 2003).

Seroprevalence levels did vary consistently among our sampled locales, although the locales were separated by <5 km and were connected by similarly forested lands undoubtedly occupied by *A. montensis*. This indicates an important effect of habitat characteristics on seroprevalence, as was found by Goodin et al. (2009), Heyman et al. (2009), McIntyre et al. (2009), and Pearce-Duvet et al. (2006). Although we are unable from our data to discern specific habitat parameters involved, the highest average seroprevalence levels were found in the locale with the most recent and extensive disturbance (R3B), consistent with conclusions reached in Goodin et al. (2006, 2009).

Mackelprang et al. (2001) reported higher seroprevalence levels to be associated with anthropogenic habitat disturbance, whereas Lehmer et al. (2008) found a negative relationship between seroprevalence levels and disturbance. Our results suggest that the relationship may be more complex than reported by either of these previous studies. In our study seroprevalence levels differed by locale and this difference varied seasonally. Seroprevalence levels always were lowest in the locale of intermediate habitat disturbance (JJA). Seroprevalence was highest in the locale with the highest level of disturbance (R3B) in the wet and transitional seasons and in the locale with the lowest level of disturbance in the dry season. Pearce-Duvet et al. (2006) reported similar interaction between habitat and season in hantaviral seroprevalence in *P. maniculatus* in a desert sagebrush community in Utah.

Seasonal variation in seroprevalence levels in *Oligoryzomys longicaudatus* was reported by Cantoni et al. (2001). In contrast, we did not find significant seasonal variation in seroprevalence, although seasonal variation in seroprevalence among locales did exist. To our knowledge, our study is the first to report both a lack of consistent seasonal variation together with a strong seasonal interaction with habitat in seroprevalence levels.

Several previous studies have reported correlations between increased precipitation and actual or potential outbreaks of HPS (Engelthaler et al. 1999; Gubler et al. 2001; Yates et al. 2002). In contrast to the studies of Anyamba et al. (2006), Conley (2006), and Parmenter et al. (1999), our results do not demonstrate a predictive relationship between seroprevalence and precipitation, other than 12-month cumulative totals. Our finding of an absence of response of either population levels or seroprevalence to seasonality or to short-term precipitation patterns may be characteristic of humid neotropical or subtropical systems with less pronounced seasonality and systems (such as ours) with low precipitation predictability (Fig. 2; Brown and Ernest 2002). Nevertheless, our finding of a predictive relationship between 12-month cumulative precipitation totals and seroprevalence agrees with Glass et al. (2000, 2002), who found concordance between seroprevalence levels and remotely sensed indices of vegetation greenness, a surface property closely related to longer-term (annual) precipitation levels.

Very few studies have directly addressed behavioral characteristics of hantavirus hosts that might affect horizontal transmission of the virus. Although Escutenaire et al. (2002) found differences in the mobility of seropositive and seronegative bank voles (*Myodes glareolus*), Lonner et al. (2008) found no differences in seroprevalence levels of resident and dispersing *P. maniculatus*. We found that seropositive animals had greater MMDM; larger home ranges in the transitional season and in locale JJB (the least disturbed habitat); and greater HRD between sampling sessions. These represent observable behavioral effects of *Hantavirus* infection in a natural host, all of which are modifications that would facilitate horizontal transmission of the virus.

Seropositive animals exhibited greater longevity than seronegatives. We note that longevity, as measured in mark-recapture sampling such as this, can confound actual longevity with site fidelity. However, given our finding that seropositive animals have higher rather than lower HRD between sampling periods, we believe that our results strongly indicate a higher longevity among seropositive animals, especially seropositive males.

Other studies of hantaviral seroprevalence in host rodent populations generally have not reported the behavioral and ecological associations with infection status that were revealed in our study. Nevertheless, we expect that additional studies designed to incorporate habitat diversity and seasonal and interannual variation will reveal that infection patterns for hantaviruses and other pathogens are associated with demographic and behavioral phenomena.

Along with additional well-designed field-sampling protocols, future investigations will require mathematical modeling that incorporates multiple patches (habitat fragments—Allen et al. 2006a, 2006b; McCormack and Allen 2007a), land-use conversion (Allen et al. 2009; Goodin et al. 2006, 2009), and other host demographic and biogeographic characteristics (Wesley et al. 2009). Additionally, evidence is emerging that land-use conversion and subsequent edge effect may bring multiple potential hosts into contact, thus facilitating ''host-jumping'' and/or genetic reassortment (Chu et al. 2009; McCormack and Allen 2006, 2007b). As demonstrated by our study, of particular importance will be mathematical models that explicitly incorporate spatial and habitat variability and are capable of investigating these phenomena at multiple spatial scales (Goodin et al. 2006, 2009).

RESUMEN

Relativamente poca información se tiene disponible sobre la historia natural o ecología poblacional de *Akodon montensis*, una especie sigmodontina que alberga los hantavirus de Jaborá y Ape Aime. Basado en muestras de marca-recaptura de 3 localidades durante 2 años, este informe provee de datos comparativos sobre población y otras características de la especie cerca de los límites de su distribución, en el Bosque Atlántico del Alto Paraná en Paraguay. Nuestros resultados, en lo general, apoyan y abarcan estudios previos, demostrando que *A. montensis* es casi exclusivamente terrestre (no arborícola), y que varía en densidad poblacional entre localidades y años. Aunque no encontramos que las densidades poblacionales estén relacionadas ya sea por estaciones Neotropicales del año o en la variabilidad de las precipitaciones, sí encontramos variaciones estacionales en las proporciones de los sexos, la cual no ha sido reportado previamente. Evaluamos la seroprevalencia de hantavirus en *A. montensis*, en asociación con variables externas, poblacional e individual. Como en la mayoría de estudios previos del sistema de roedores-hantavirus, encontramos que la predominancia de individuos seropositivos fue más alta en machos, y que los niveles de seroprevalencia variarían entre las diferentes localidades. A diferencia de otros reportes anteriores, especialmente aquellos de América del Norte, no encontramos una correspondencia entre seroprevalencia y densidad poblacional, variaciones estacionales, o entre la mayoría de las variables de precipitación. Asociaciones simple y directa entre niveles de seroprevalencia, y estaciones del año o precipitación, como fueran reportadas en estudios en América del Norte, serían tal vez menos características de sistemas húmedos Neotropicales o sub tropicales. Nuestro análisis revela que animales seropositivos exhiben varios comportamientos notables y modificaciones en su historia natural, en particular: tienen un rango del hogar mayor que los seronegativos; exhiben mayores valores de distancia de movimiento máximo; tienen mayor desplazamiento del rango de hogar entre sesiones de muestreo, especialmente aquellos individuos que se seroconvierten durante el mismo período; y tienen mayor longevidad, especialmente los machos. En general, estas características podrían ser consideradas como favorables a la capacidad del virus para ser trasmitido horizontalmente a otros huéspedes.

ACKNOWLEDGMENTS

This research was supported by a grant from the Fogarty International Center #R01TW006986-02 under the NIH/NSF Ecology of Infectious Diseases initiative. We thank the Fundación Moises Bertoni (especially Myriam Velazquez, René Palacios, and the guardabosques at RNBM) for facilitating access to the field sites and patient logistical assistance, and the Vendramini family for allowing us to work in Estancia Rama III. The Secretaría del Ambiente provided necessary permits for working with wildlife. We thank Ismael Mora, Crisaldo Mora, Sergio Villanueva, César Manchini, César Benítez, Flavia Neto, María Luisa Ortiz, Guillermo Santacruz, Hector Pintos, Reinaldo Chaparro, Sixto Fernández, and Freddy Ramírez for hard and diligent work in the field. Special thanks go also to Dr. Fatima Mereles and Gloria Raquel Céspedes Domínguez for assistance with vegetation mapping and identification. Thanks to Dr. Robert Baker and Heath Garner for authorizing and facilitating tissues loans from the Natural Science Research Lab, Texas Tech University; to Aida Luz Aquino for translating the abstract; to Rhett Mohler for developing and constructing Fig. 2; and to Associate Editor Rodrigo Medellin, 2 anonymous reviewers, and Edgar Owen for their comments concerning an earlier version of the manuscript.

LITERATURE CITED

ABBOTT, K. D., T. G. KSIAZEK, AND J. N. MILLS. 1999. Long-term hantavirus persistence in rodent populations in central Arizona. Emerging Infectious Diseases 5:102–112.

ALLEN, L. J. S., E. J. ALLEN, AND C. B. JONSSON. 2006a. The impact of environmental variation on hantavirus infection in rodents. Pp. 1–15 in Contemporary mathematics series, 410, Proceedings of the Joint Summer Research Conference on Modeling the Dynamics of Human Diseases: Emerging Paradigms and Challenges (A. B. Gumel, C. Castillo-Chavez, R. E. Mickens, and D. P. Clemence, eds.).

ALLEN, L. J. S., R. K. MCCORMACK, AND C. B. JONSSON. 2006b. Mathematical models for hantavirus infection in rodents. Bulletin of Mathematical Biology 68:511–524.

ALLEN, L. J. S., ET AL. 2009. A habitat-based model for the spread of hantavirus between reservoir and spillover species. Journal of Theoretical Biology 260:510–522.

ANYAMBA, A., J.-P. CHRETIEN, J. SMALL, C. J. TUCKER, AND K. J. LINTHICUM. 2006. Developing global climate anomalies suggest potential disease risks for 2006–2007. International Journal of Health Geographics 5:60–67.

ARMIÉN, B., ET AL. 2004. High seroprevalence of hantavirus infection on the Azuero Peninsula of Panama. American Journal of Tropical Medicine and Hygiene 70:682–687.

BERGALLO, H. G. AND W. E. MAGNUSSON. 1999. Effects of climate and food availability on four rodent species in southeastern Brazil. Journal of Mammalogy 80:472–486.

BIANCHI, N. O. 2002. *Akodon* sex reversed females: the never ending story. Cytogenetic and Genome Research 96:60–65.

BIGGS, J. R., ET AL. 2000. Relationship of ecological variables to Sin Nombre virus antibody seroprevalence in populations of deer mice. Journal of Mammalogy 81:676–682.

Blom, G. 1958. Statistical estimates and transformed beta variables. John Wiley & Sons, Inc., New York.

Boone, J. D., et al. 2000. Remote sensing and geographic information systems: charting Sin Nombre virus infections in deer mice. Emerging Infectious Diseases 6:248–258.

Borucki, M. K., et al. 2000. Role of maternal antibody in natural infection of *Peromyscus maniculatus* with Sin Nombre virus. Journal of Virology 74:2426–2429.

Brown, J. H., and S. K. Morgan Ernest. 2002. Rain and rodents: complex dynamics of desert consumers. BioScience 52:979–987.

Calisher, C. H., J. J. Root, J. N. Mills and B. J. Beaty. 2002. Assessment of ecologic and biologic factors leading to hantavirus pulmonary syndrome, Colorado, U.S.A. Croation Medical Journal 43:330–337.

Calisher, C. H., W. Sweeney, J. N. Mills, and B. J. Beaty. 1999. Natural history of Sin Nombre virus in western Colorado. Emerging Infectious Diseases 5:126–134.

Cantoni, B., et al. 2001. Seasonal variation in prevalence of antibody to hantaviruses in rodents from southern Argentina. Tropical Medicine and International Health 6:811–816.

Carleton, M. D., and G. G. Musser. 2005. Order Rodentia. Pp. 745–1600 in Mammal species of the world: a taxonomic and geographic reference (D.E. Wilson and D.M. Reeder, eds.). Johns Hopkins University Press, Baltimore, Maryland.

Cartes, J. L. 2003. Brief history of conservation in the Interior Atlantic Forest. Pp. 269–287 in The Atlantic Forest of South America. (G. Galindo-Leal and I. Camara, eds.). Island Press, Washington, D.C.

Carvalho, F. M. V., P. S. Pinheiro, F. A. S. Fernandez, and J. L. Nessimian. 1999. Diet of small mammals in Atlantic Forest fragments in southeastern Brazil. Revista Brasileira de Zoociencias 1:91–101.

Childs, J. E., G. E. Glass, G. W. Korch, and J. W. Leduc. 1989. Effects of hantaviral infection on survival, growth and fertility in wild rat (*Rattus norvegicus*) populations of Baltimore, Maryland. Journal of Wildlife Diseases 25:469–476.

Christoff, A. U. 1997. Contribução a sistemática das espécies do gênero *Akodon* (Rodentia: Sigmodontinae) do leste do Brasil: estudos anatômicos, citogenéticos e de distribução geográfica. Ph.D. dissertation, Universidade do São Paulo, Brazil.

Chu, Y.-K., D. Goodin, R. D. Owen, and C. B. Jonsson. 2009. Sympatry of two hantaviral strains in the interior Atlantic forest of Paraguay. Emerging Infectious Diseases 15:1977–1980.

Chu, Y.-K., B. Milligan, R. D. Owen, D. Goodin, and C. B. Jonsson. 2006. Phylogenetic and geographical relationships of hantavirus strains in eastern and western Paraguay. American Journal of Tropical Medicine and Hygiene 75:1127–1134.

Chu, Y.-K., R. D. Owen, L. González, and C. B. Jonsson. 2003. The complex ecology of hantavirus in Paraguay. American Journal of Tropical Medicine and Hygiene 69:263–268.

Conley, H. K. 2006. Understanding the relationship between climate variability and hantavirus pulmonary syndrome. Ph.D. dissertation, The University of Iowa, Ames.

Dalmagro, A. D., and E. M. Vieira. 2005. Patterns of habitat utilization of small rodents in an area of Araucaria forest in southern Brazil. Austral Ecology 30:353–362.

Davis, S. M., E. Calvet and H. Leirs. 2005. Fluctuating rodent populations and the risk to humans from rodent-borne disease. Vector-Borne and Zoonotic Diseases 5:305–314.

D'Elía, G. 2003. Phylogenetics of Sigmodontinae (Rodentia, Muroidea, Cricetidae), with special reference to the akodont group, and with additional comments on historical biogeography. Cladistics 19:307–323.

Di Bitetti, M. S., G. Placci, and L. A. Dietz. 2003. A biodiversity vision for the Upper Paraná Atlantic Forest ecoregion: designing a biodiversity conservation landscape and setting priorities for conservation action. World Wildlife Fund, Washington, D.C.

Douglass, R. J., C. H. Calisher, K. D. Wagoner, and J. N. Mills. 2007. Sin Nombre virus infection of deer mice in Montana: characteristics of newly infected mice, incidence, and temporal pattern of infection. Journal of Wildlife Diseases 43:12–22.

Douglass, R. J., et al. 2001. Longitudinal studies of Sin Nombre virus in deer mouse-dominated ecosystems of Montana. American Journal of Tropical Medicine and Hygiene 65:33–41.

Easterbrook, J. D., J. B. Kaplan, G. E. Glass, M. V. Pletnikov, and S. L. Klein. 2007. Elevated testosterone and reduced 5-HIAA concentrations are associated with wounding and hantavirus infection in male Norway rats. Hormones and Behavior 52:474–481.

Eisen, R. J., et al. 2007. A spatial model of shared risk for plague and hantavirus pulmonary syndrome in the southwestern United States. American Journal of Tropical Medicine and Hygiene 77:999–1004.

Emmons, L. H., and F. Feer. 1990. Neotropical rainforest mammals: a field guide. The University of Chicago Press, Chicago, Illinois.

Engelthaler, D. M., et al. 1999. Climatic and environmental patterns associated with hantavirus pulmonary syndrome, Four Corners region, United States. Emerging Infectious Disease 5:87–94.

Escutenaire, S., et al. 2002. Behavioral, physiologic, and habitat influences on the dynamics of Puumala virus infection in bank voles (*Clethrionomys glareolus*). Emerging Infectious Diseases 8:930–936.

Eva, H. D., et al. 2003. GLC2000 database, European Commission Joint Research Centre. http://www.gvm.jrc.it/glc2000. Accessed 07 January 2005.

Figueiredo, L. T., and F. A. Fernandez. 2004. Contrasting effects of fire on populations of two small rodent species in fragments of Atlantic forest in Brazil. Journal of Tropical Ecology 20:225–228.

Gamarra de Fox, I., and A. J. Martin. 1996. Lista de mamíferos del Paraguay. Pp. 469–573 in Colecciones de fauna y flora del Museio Nacional de Historia Natural del Paraguay (O. Romero, ed.). Dirección de Parques Nacionales y Vida Silvestre, Asunción, Paraguay.

Gannon, W. L., R. S. Sikes, and the Animal Care and Use Committee of the American Society of Mammalogists. 2007. Guidelines of the American Society of Mammalogists for the use of wild mammals in research. Journal of Mammalogy 88:809–823.

Geise, L. 1995. Os roedores Sigmodontinae do Estado do Rio de Janeiro (Rodentia, Muridae). Sistemática, citogenética, distribução e variação geográfica. Ph.D. dissertation, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil.

Geise, L., F. C. Canavez, and H. N. Seuánez. 1998. Comparative karyology in *Akodon* (Rodentia, Sigmodontinae) from southeastern Brazil. Journal of Heredity 89:158–163.

Geise, L., M. F. Smith, and J. L. Patton. 2001. Diversification in the genus *Akodon* (Rodentia: Sigmodontinae) in southesastern South America: mitochondrial DNA sequence analysis. Journal of Mammalogy 82:92–101.

Gentile, R., and P. S. D'Andrea. 2000. Population dynamics and reproduction of marsupials and rodents in a Brazilian rural area: a five-year study. Studies on Neotropical Fauna and Environment 35:1–9.

Gentile, R., and F. A. S. Fernandez. 1999. Influence of habitat structure on a streamside small mammal community in a Brazilian rurual area. Mammalia 63:29–40.

Giuggioli, L., G. Abramson, V. M. Kenkre, G. Suzán, E. Marcé, and T. L. Yates. 2005. Diffusion and home range parameters from rodent population measurements in Panama. Bulletin of Mathematical Biology 67:1135–1149.

Glass, G. E., et al. 2000. Using remotely sensed data to identify areas at risk for hantavirus pulmonary syndrome. Emerging Infectious Disease 6:238–247.

Glass, G. E., et al. 2002. Satellite imagery characterizes local animal reservoir populations of Sin Nombre virus in the southwestern United States. Procedings of the National Academy of Sciences 99:16817–16822.

Goodin, D. G., D. E. Koch, R. D. Owen, Y.-K. Chu, J. M. S. Hutchinson, and C. B. Jonsson. 2006. Land cover associated with hantavirus in Paraguay. Global Ecology and Biogeography 15:519–527.

Goodin, D. G., et al. 2009. Microhabitat characteristics of *Akodon montensis*, a vector for hantavirus, and hantaviral seroprevalence in an Atlantic Forest site, in eastern Paraguay. Journal of Vector Ecology 34(1):104–113.

Gottesman, A. B., P. R. Krausman, M. L. Morrison, and Y. Petryszyn. 2004. Movements and home range of brush mice. Southwestern Naturalist 49:289–294.

Gubler, D. J., P. Reiter, K. L. Ebi, W. Yap, R. Nasci, and J. A. Patz. 2001. Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. Environmental Health Perspectives 109 Suppl. 2::223–233.

Heyman, P., R. Van Mele, L. Smajlovic, A. Dobly, C. Cochez, and C. Vandervelde. 2009. Association between habitat and prevalence of *Hantavirus* infections in bank voles (*Myodes glareolus*) and wood mice (*Apodemus sylvaticus*). Vector-Borne and Zoonotic Diseases 9:141–146.

Hinson, E. R., S. M. Shone, M. C. Zink, G. E. Glass, and S. L. Klein. 2004. Wounding: the primary mode of Seoul virus transmission among male Norway rats. American Journal of Tropical Medicine and Hygiene 70:310–317.

Huang, C., et al. 2007. Rapid loss of Paraguay's Atlantic forest and the status of protected areas—a Landsat assessment. Remote Sensing of Environment 106:460–466.

Jaksic, F. M., and M. Lima. 2003. Myths and facts on ratadas: bamboo blooms, rainfall peaks and rodent outbreaks in South America. Austral Ecology 28:237–251.

Kallio, E. R., et al. 2007. Endemic hantavirus infection impairs the winter survival of its rodent host. Ecology 88:1911–1916.

Kim, G. R., and K. T. McKee. 1985. Pathogenesis of Hantaan virus infection in suckling mice: clinical, virologic, and serologic observation. American Journal of Tropical Medicine and Hygiene 34:388–395.

Koch, D. E., R. M. Mohler, and D. G. Goodin. 2007. Stratifying land use/land cover for spatial analysis of disease ecology and risks: an example using object-based classification techniques. Geospatial Health 2:15–28.

Langlois, J. P., L. Fahrig, G. Merriam and H. Artsob. 2001. Landscape structure influences continental distribution of hantavirus in deer mice. Landscape Ecology 16:255–266.

Lehmer, E. M., C. A. Clay, J. Pearce-Duvet, S. St. Jeor, and M. D. Dearing. 2008. Differential regulation of pathogens: the role of habitat disturbance in predicting prevalence of Sin Nombre virus. Oecologia 155:429–439.

Loehman, R. A. 2006. Modeling interactions among climate, landscape, and emerging diseases: a hantavirus case study. Ph.D. dissertation, The University of Montana, Missoula.

Lonner, B. N., R. J. Douglass, A. J. Juenzi, and K. Hughes. 2008. Seroprevalence against Sin Nombre virus in resident and dispersing deer mice. Vector-Borne and Zoonotic Diseases 8:433–441.

Lozada, M., N. Guthmann, and N. Baccala. 2000. Microhabitat selection of five sigmodontine rodents in a forest–steppe transition zone in northwestern Patagonia. Studies on Neotropical Fauna and Environment 35:85–90.

Mackelprang, R., M. D. Dearing, and S. St. Jeor. 2001. High prevalence of Sin Nombre virus in rodent populations, central Utah: a consequence of human disturbance? Emerging Infectious Diseases 7:480–481.

Malcolm, J. R. 1994. Edge effects in Central Amazonian forest fragments. Ecology 75:2438–2445.

Massoia, E., and A. Fornes. 1962. Un cricétido nuevo para la Argentina: *Akodon arviculoides montensis* Thomas (Rodentia). Physis 23:185–194.

McCormack, R. K., and L. J. S. Allen. 2006. Stochastic SIS and SIR multihost epidemic models. Pp. 775–786 in Proceedings of the conference on differential & difference equations and applications (R. P. Agarwal and K. Perera, eds.). Hindawi Publishing Company, New York.

McCormack, R. K., and L. J. S. Allen. 2007a. Multi-patch deterministic and stochastic models for wildlife diseases. Journal of Biological Dynamics 1:63–85.

McCormack, R. K., and L. J. S. Allen. 2007b. Disease emergence in multi-host epidemic models. Mathematical Medicine and Biology 24:17–34.

McIntyre, N. E., et al. 2005. A longitudinal study of Bayou virus, hosts, and habitat. American Journal of Tropical Medicine and Hygiene 73:1043–1049.

McIntyre, N. E., et al. 2009. Ecological correlates of serological status for Bayou virus in *Oryzomys palustris*. Mastozoología Neotropical 16:83–93.

Mills, J. N. 2006. Biodiversity loss and emerging infectious disease: an example from the rodent-borne hemorrhagic fevers. Biodiversity 7:9–17.

Mills, J. N., et al. 1997. Patterns of association with host and habitat: antibody reactive with Sin Nombre virus in small mammals in the major biotic communities of the southwestern United States. American Journal of Tropical Medicine and Hygiene 56:273–284.

Mills, J. N., T. G. Ksiazek, C. J. Peters, and J. E. Childs. 1999. Long-term studies of hantavirus reservoir populations in the southwestern United States: a synthesis. Emerging Infectious Diseases 5:135–142.

Mills, J. N., K. Schmidt, B. A. Ellis, G. Calderón, D. A. Enría, and T. G. Ksiazek. 2007. A longitudinal study of hantavirus infection in three sympatric reservoir species in agroecosystems on the Argentine Pampa. Vector-Borne and Zoonotic Diseases 7:229–240.

Musser, G. G., and M. D. Carleton. 2005. Rodentia. Pp. 745–1600 in Mammal species of the world: a taxonomic and geographic reference (D. E. Wilson and D. M. Reeder, eds.). Johns Hopkins University Press, Baltimore, Maryland.

New, M., M. Hulme, and P. Jones. 1999. Representing twentieth-century space time climate variability. Part I: development of a 1961–1990 mean monthly terrestrial climatology. Journal of Climate 12:829–856.

Nitikman, L. Z., and M. A. Mares. 1987. Ecology of small mammals in a gallery forest of central Brazil. Annals of the Carnegie Museum 56:75–95.

O'Connor, C. S., J. P. Hayes, and S. C. St. Jeor. 1997. Sin Nombre virus does not impair respiratory function of wild deer mice. Journal of Mammalogy 78:661–668.

Oliveira, F. F. R., R. Nessim, L. P. Costa, and Y. L. R. Leite. 2007. Small mammal ecology in an urban Atlantic forest fragment in southeastern Brazil. Lundiana 8:27–34.

Olson, D. M., et al. 2001. Terrestrial ecoregions of the world: a new map of life on earth. BioScience 51:933–938.

Olsson, G. E., et al. 2002. Demographic factors associated with hantavirus infection in bank voles (*Clethrionomys glareolus*). Emerging Infectious Diseases 8:924–929.

Olsson, G. E., N. White, J. Hjalten, and C. Ahlm. 2005. Habitat factors associated with bank voles (*Clethrionomys glareolus*) and concomitant hantavirus in northern Sweden. Vector-Borne and Zoonotic Diseases 5:315–323.

Orrock, J. L., J. F. Pagels, W. J. McShea, and E. K. Harper. 2000. Predicting presence and abundance of a small mammal species: the effect of scale and resolution. Ecological Applications 10:1356–1366.

Pardiñas, U. F. J., G. D'Elía, and S. Cirignoli. 2003. The genus *Akodon* (Muroidea: Sigmodontinae) in Misiones, Argentina. Zeitschrift für Säugetierkunde 68:129–143.

Pardini, R., S. M. de Souza, R. Braga-Neto, and J. P. Metzger. 2005. The role of forest structure, fragment size and corridors in maintaining small mammal abundance and diversity in an Atlantic forest landscape. Biological Conservation 124:253–266.

Parmenter, R. R., E. P. Yadav, C. A. Parmenter, P. Ettestad, and K. L. Gage. 1999. Incidence of plague associated with increased winter–spring precipitation in New Mexico. American Journal of Tropical Medicine and Hygiene 61:814–821.

Pearce-Duvet, J. M. C., S. C. St. Jeor, J. D. Boone, and M. D. Dearing. 2006. Changes in Sin Nombre virus antibody prevalence in deer mice across seasons: the interaction between habitat, sex, and infection in deer mice. Journal of Wildlife Diseases 42:819–824.

Peixoto, I. D., and G. Abramson. 2006. The effect of biodiversity on the hantavirus epizootic. Ecology 87:873–879.

Püttker, T. 2007. Effects of fragmentation on use of vegetation structures, density, movement patterns and parasite load of selected small mammal species in secondary forest fragments of the coastal Atlantic forest, Brazil. Ph.D. dissertation, University of Hamburg, Hamburg, Germany.

Püttker T., Y. Meyer-Lucht, and S. Sommer. 2006. Movement distances of five rodent and two marsupial species in forest fragments of the coastal Atlantic rainforest, Brazil. Ecotropica 12:131–139.

Püttker T., Y. Meyer-Lucht, and S. Sommer 2008. Fragmentation effects on population density of three rodent species in secondary Atlantic rainforest, Brazil. Studies on Neotropical Fauna and Environment 43:11–18.

Redford, K. H., A. Taber, and J. A. Simonetti. 1990. There is more to biodiversity than the tropical rain forests. Conservation Biology 4:328–330.

Rieger, T., A. Langguth, and T. Weimer. 1995. Allozymic characterization and evolutionary relationships in the Brazilian *Akodon cursor* species group (Rodentia-Cricetidae). Biochemical Genetics 33:283–295.

Root, J. J., C. H. Calisher, and B. J. Beaty. 1999. Relationships of deer mouse movement, vegetative structure, and prevalence of infection with Sin Nombre virus. Journal of Wildlife Diseases 35:311–318.

Ruedas, L. A., et al. 2004. Community ecology of small mammal populations in Panama following an outbreak of Hantavirus Pulmonary Syndrome. Journal of Vector Ecology 29:177–191.

Sauvage, F., M. Langlais, and D. Pontier. 2007. Predicting the emergence of human hantavirus disease using a combination of viral dynamics and rodent demographic patterns. Epidemiology and Infection 135:46–56.

Schnell, G. D., et al. 2008. Conservation status, demographic features, and habitat preference of the endemic tawny deer mouse (*Peromyscus perfulvus*). Southwestern Naturalist 53:9–20.

Sokal, R. R., and F. J. Rohlf. 1981. Biometry. 2nd ed. W. H. Freeman and Company, New York.

Stapp, P. 2007. Trophic cascades and disease ecology. EcoHealth. 4:121–124.

Suzán, G., et al. 2006. Modeling hantavirus reservoir species dominance in high seroprevalence areas on the Azuero Peninsula of Panama. American Journal of Tropical Medicine and Hygiene 74:1103–1110.

Suzán, G., et al. 2008. The effect of habitat fragmentation and species diversity loss on hantavirus prevalence in Panama. Annals of the New York Academy of Sciences 1149:80–83.

Suzán, G., 2009. Experimental evidence for reduced rodent diversity causing increased hantavirus prevalence. PLoS ONE 4:e5461.

Talamoni, S. A., D. Couto, D. A. Cordeiro Júnior, and F. M. Diniz. 2008. Diet of some species of Neotropical small mammals. Mammalian Biology 73:337–341.

Tsai, T. F., S. Bauer, J. B. McCormick, and T. Kurata. 1982. Intracerebral inoculation of suckling mice with Hantaan virus. Lancet 2:503–504.

Umetsu, F., J. P. Metzger, and R. Pardini. 2008. Importance of estimating matrix quality for modeling species distribution in complex tropical landscapes: a test with Atlantic forest small mammals. Ecography 31:359–370.

Vieira, E. M., and E. L. A. Monteiro-Filho. 2003. Vertical stratification of small mammals in the Atlantic rain forest of southeastern Brazil. Journal of Tropical Ecology 19:501–507.

Vieira, E. M., G. Paise, and P. H. D. Machado. 2006. Feeding of small rodents on seeds and fruits: a comparative analysis of three species of rodents of the *Araucaria* forest, southern Brazil. Acta Theriologica 51:311–318.

Wesley, C. L., L. J. S. Allen, C. B. Jonsson, Y.-K. Chu, and R. D. Owen. 2009. A discrete-time rodent–hantavirus model structured by infection and developmental stages. Advanced Studies in Pure Mathematics 53:387–398.

White, G. C., and K. P. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study 46 Supplement 120–138.

Yanagihara, R., H. L. Amyx, and D. C. Gajdusek. 1985. Experimental infection with Puumala virus, the etiologic agent of nephropathia epidemica, in bank voles (*Clethrionomys glareolus*). Journal of Virology 55:34–38.

Yates, T. L., J. N. Mills, C. A. Parmenter, T. G. Ksiazek, R. R. Parmenter, and J. R. Vandecastle. 2002. The ecology and evolutionary history of an emergent disease: hantavirus pulmonary syndrome. BioScience 52:989–998.

Submitted 23 April 2009. Accepted 23 August 2009.

Associate Editor was Rodrigo A. Medellin.