Address for correspondence: Hiroki Kawabata, National Institute of infectious Diseases– Bacteriology, Toyama 1-23-1, Shinjuku-ku, Tokyo 162-8640, Japan; email: kbata@nih. go.jp

## Sin Nombre Virus Infection in Deer Mice, Channel Islands, California

To the Editor: Sin Nombre virus (SNV) is a highly virulent strain of hantavirus associated with rodent hosts in North America (1,2). Documenting the prevalence of SNV in wild rodent populations is an important component of determining risk for exposure and ultimately providing sound recommendations for epidemiologic management (3). Prevalence of SNV is highly variable. Deer mice (Peromyscus maniculatus) that inhabit the Channel Islands off the California coast often have rates of SNV that greatly exceed values on the mainland (2). Even though these islands have high rates of SNV prevalence and are recreational areas for humans, no surveys of the Channel Islands have been performed to document the dynamics of prevalence since 1994-1996 (2,4). We visited 4 of the Channel Islands in 2007 to document rates of SNV prevalence in P. maniculatus.

From May 3–15, 2007, we visited 4 of the Channel Islands off the California coast: East Anacapa Island, Santa Barbara Island, San Miguel Island, and Santa Rosa Island. On each island, mice were captured by using Sherman live traps from habitats characterized by giant coreopsis (*Coreopsis gigantea*), a shrub native to California, to provide a standardized habitat for comparisons across islands. The number of sampling areas depended largely upon the distribution of C. gigantea habitat and logistical considerations during each island visit (Table). Upon capture of the mice, blood samples were taken from the submandibular vein by using Medi-Point animal lancets (Medi-Point International, Inc., Mineola, NY, USA) and stored in sterile micropipette tubes. Samples were stored on ice until shipment to the California Department of Health Services' Viral and Rickettsial Disease Laboratory for processing. P. maniculatus serum samples were examined for immunoglobulin (Ig) G antibodies to the SNV nucleocapsid protein by ELISA with Centers for Disease Control and Prevention reagents (5).

Detailed information regarding SNV prevalence, sampling location, and sampling effort is presented in the Table. We compare our 2007 data with data collected in 1994 by Jay et al. (2) because 1994 was the only other year when all 4 islands used in our study were sampled. Graham and Chomel (4) also collected data from San Miguel Island and Santa Rosa Island in 1995 and 1996 (the use of the average prevalence from 1995 and 1996 for these 2 islands does not change any of our results).

There was no significant difference in prevalence of SNV antibodies between our 2007 results and the prevalence found by Jay et al. (2) in 1993–1994 (paired *t* test t = 0.13, 3; df = 3; p = 0.91). Overall, 36 male and 42 female mice were captured; the sex of captured animals was independent of SNV infection (9 males and 6 females positive for SNV; test of independence  $\chi^2 = 0.28$ , 1 df, p = 0.59). We captured only 2 subadult mice on islands where we also detected antibodies to SNV; 1 mouse tested positive, the other tested negative. Although our sample sizes precluded detecting very low rates of SNV infection with confidence on Santa Barbara and East Anacapa Islands, the consistency of our results with those of Jay et al. (2) suggests that our sampling was sufficient for comparative purposes.

Several studies now indicate the importance of long-term surveillance of SNV prevalence in wild rodent populations for understanding the factors that may contribute to outbreaks of human disease, e.g. (6). These studies often document the generally positive, though often temporally delayed, relationship between population density of P. maniculatus and seroprevalence for SNV (7). Our results suggest a high degree of temporal stability in prevalence of antibodies to SNV in P. maniculatus on the Channel Islands, despite considerable variation in host population density between earlier studies and ours (4,8). Although we cannot know the prevalence of SNV among P. maniculatus on the Channel Islands in periods between the studies by Jay et al. (2), Graham and Chomel (4), and our own, SNV prevalence on these islands is quite similar to levels previously recorded both for islands with relatively low prevalence

Table. Sin Nombre virus in <i>Peromyscus maniculatus</i> mice on 4 Channel Islands,	
California, May 3–15, 2007*	

No. trap nights	Prevalence, %	
	2007	1994
180	0	0
104	26.3	17.9
216	0	0
216	47.6	58
	180 104 216	No. trap nights 2007   180 0   104 26.3   216 0

\*The number of captured mice that were sampled for Sin Nombre virus (SNV) was 23 on East Anacapa, 19 on San Miguel Island, 15 on Santa Barbara Island, and 21 on Santa Rosa Island. The 1994 data in the table are from a study by Jay et al. (2) and are included for comparison puposes. †East Anacapa: 34°00'56"N/119°21'49"W. ±San Miguel: 34°02'18"N/120°20'54"W.

§Santa Barbara: 33º28'30"N/119º02'12"W.

¶Santa Rosa: 34º00'03"N/120º03'30"W.

### LETTERS

(i.e., East Anacapa and Santa Barbara Islands) or high prevalence (i.e., San Miguel and Santa Rosa Islands).

Future studies comparing longterm dynamics on islands and related mainlands are needed to examine the possibility that insular systems provide unique opportunities to understand the factors affecting pathogen dynamics and human risk. Given the substantial variation in mouse population density among different habitats within these islands and variation in prevalence among trapping areas in our study (Table) and others (4), we also recommend that future studies focus on the diverse array of habitats where P. maniculatus is found on the islands to more completely characterize within-island risk.

### Acknowledgments

We thank T. Rusca for outstanding assistance with data collection; the National Park Service, especially the staff at the Channel Islands National Park, for providing essential logistical support; C. Fritz, R. Davis, and B. Enge for sample processing; and C. Schwemm and W. Ryberg for helpful suggestions on drafts of the manuscript.

This work was supported by National Science Foundation grant DEB-0502069 to J.L.O. and a National Fish and Wildlife Foundation Budweiser Conservation Scholarship to B.F.A. All protocols were approved by the Institutional Animal Care and Use Committee at the University of California, Santa Barbara.

## John L. Orrock and Brian F. Allan Author affiliation: Washington University,

St. Louis, Missouri, USA

## DOI: 10.3201/eid1412.080935

#### References

 Childs JE, Ksiazek TG, Spiropoulou CF, Krebs JW, Morzunov S, Maupin GO, et al. Serologic and genetic identification of *Peromyscus maniculatus* as the primary rodent reservoir for a new hantavirus in the southwestern United States. J Infect Dis. 1994;169:1271–80.

- Jay M, Ascher MS, Chomel BB, Madon M, Sesline D, Enge BA, et al. Seroepidemiologic studies of hantavirus infection among wild rodents in California. Emerg Infect Dis. 1997;3:183–90.
- Childs JE, Mackenzie MS, Richt JA, editors. Pre-spillover prevention of emerging zoonotic diseases: what are the targets and what are the tools? Wildlife and emerging zoonotic diseases: the biology, circumstances and consequences of cross-species transmission; Berlin: Springer; 2007. p. 389–443.
- Graham TB, Chomel BB. Population dynamics of the deer mouse (*Peromyscus maniculatus*) and Sin Nombre virus, California Channel Islands. Emerg Infect Dis. 1997;3:367–70.
- Feldmann H, Sanchez A, Morzunov S, Spiropoulou CF, Rollin PE, Ksiazek TG, et al. Utilization of autopsy RNA for the synthesis of the nucleocapsid antigen of a newly recognized virus associated with hantavirus pulmonary syndrome. Virus Res. 1993;30:351–67. DOI: 10.1016/0168-1702(93)90101-R
- Mills JN, Ksiazek TG, Peters CJ, Childs JE. Long-term studies of hantavirus reservoir populations in the southwestern United States: a synthesis. Emerg Infect Dis. 1999;5:135–42.
- Madhav NK, Wagoner KD, Douglass RJ, Mills JN. Delayed density-dependent prevalence of Sin Nombre virus antibody in Montana deer mice (*Peromyscus maniculatus*) and implications for human disease risk. Vector Borne Zoonotic Dis. 2007;7:353–64. DOI: 10.1089/ vbz.2006.0605
- Schwemm CA, Coonan TJ. Status and ecology of deer mice (*Peromyscus maniculatus* subspp.) on Anacapa, Santa Barbara, and San Miguel Islands, California: summary of monitoring 1992–2001. Ventura (CA): United States Department of Interior, National Park Service, Channel Islands National Park; 2001.

Address for correspondence: John L. Orrock, Department of Biology, Washington University, 1 Brookings Dr, St. Louis, MO 63130, USA; email: orrock@wustl.edu

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.

# Parachlamydia acanthamoebae Infection and Abortion in Small Ruminants

To the Editor: Abortion in ruminants is of worldwide economic importance. Moreover, several abortigenic agents have a zoonotic potential, i.e., *Brucella abortus, Coxiella burnetii*, and *Chlamydophila abortus. C. abortus*, which causes ovine enzootic abortion, may also infect pregnant women who have had contact with *C. abortus*infected sheep and goats, and such infection can lead to miscarriage (1).

Parachlamydia acanthamoebae (2) is a Chlamydia-related organism considered as an emerging agent of pneumonia in humans. Recently, we reported its role in the setting of bovine abortion (3). Here, we investigated the prevalence of *C. abortus* and *P. acanthamoebae* infections in abortions in small ruminants.

Formalin-fixed placenta, fetal lung and liver, or both, were available from abortion products from 144 goats and 86 sheep (n = 211). These specimens had previously been investigated for several abortigenic agents (4). Placentas and fetal organs were analyzed by histopathologic examination and by specific real-time PCR and immunohistochemical protocols that detect members of the *Chlamydiaceae* family and *P. acanthamoebae*.

DNA from paraffin blocks was extracted as described (5) by using the DNeasy Tissue kit (QIAGEN, Hilden, Germany). The real-time PCR for *Chlamydiaceae* was conducted on an ABI 7500 (Applied Biosystems, Foster City, CA, USA) by using a modified version of Everett's PCR (6). Primers Ch23S-F (5'-CTGAAACCAG TAGCTTATAAGCGGT-3'), Ch23S-R (5'-ACCTCGCCGTTTAACTTA ACTCC-3'), and probe Ch23S-p (5'-FAM-CTCATCATGCAAAAGGCA CGCCG-TAMRA-3') were used to