Hantavirus pulmonary syndrome is a newly recognized rodent-borne zoonosis. We report a case of hantavirus pulmonary syndrome in an employee of a California utility company who was probably occupationally exposed to Sin Nombre virus. Environmental assessment and genetic comparison of the patient’s hantavirus isolates to hantavirus isolates from rodents trapped at possible sites of exposure suggested that the patient contracted his infection at the work site. The study revealed a close correspondence between the patient’s viral genotype and that from a rodent trapped at the work site. This report alerts the public health and medical community to the fact that employees of utility companies and similar industries may be an important risk group in areas where hantavirus is endemic and emphasizes the need to incorporate strategies for preventing exposure to hantavirus and other emerging infections into occupational safety protocols.

**Case Report**

A 56-year-old male resident of Mono County, California, presented to an emergency department on 29 August 1994 with progressive dyspnea following 5 days of fever and myalgias. He was admitted to the intensive care unit. Because of a combination of dyspnea, hypoxia, bilateral interstitial lung infiltrates, thrombocytopenia, and an elevated hemoglobin level, the possibility of hantavirus pulmonary syndrome (HPS) was considered early. Acute sera obtained on days 5 and 7 of his illness showed high titers of IgM and IgG antibodies to SNV with use of an ELISA [1]. In addition, both serum samples contained IgM and IgG antibodies to N protein and IgG antibodies to SNV G1 glycoprotein by western blot assay [4] as well as SNV RNA in the peripheral blood by reverse transcription–polymerase chain reaction (RT-PCR) analysis [5–7]. Although his clinical course was similar to that of previously described patients with HPS [8, 9], his illness was relatively mild during 4 days of hospitalization and he never required intubation or therapy with pressors.

**Human and Rodent Surveys**

The patient lived in a frame house on 5 acres of utility company property located in the eastern Sierra Nevada at an elevation of 7,800 feet. He was employed as an operator for a utility company and spent most of his work week in the control room of a hydroelectric substation (Substation A) that abutted a steep canyon. His wife was a forestry worker at a ranger station located in the canyon between their residence and Substation A. The patient occasionally worked in three smaller substations (Substations B, C, and D) and several outbuildings, all of which were within 15 km of his residence and primary work site. A serosurvey with use of ELISA showed that the patient’s wife and 13 co-workers lacked IgM or IgG antibodies to SNV.

Environmental investigations of the patient’s home, four work sites, and the nearby ranger station were conducted. The inside of the patient’s residence appeared free of rodent infestation, and efforts had been made to ensure that there were no rodents in his home. In contrast, many entry points for rodents were identified at the substations and ranger station; moderate to heavy rodent infestation was observed at these sites. The patient and several co-workers reported
seeing rodents and rodent droppings or nests in and around the substations throughout the year. Two weeks before the patient’s illness, the ceiling panels in Substation A were replaced, causing dust and rodent droppings to fall on his desk. The patient denied handling rodents, but he admitted cleaning rodent droppings on his desk without wearing gloves and entering small, enclosed work areas without respiratory protection.

A systematic rodent survey was conducted at the patient’s residence, the four substations, and the forest service ranger station. Routine methods were used to livetrap and to obtain sera from wild rodents [10]. Sera were examined for IgG antibodies to the SNV nucleocapsid protein by ELISA and western blot studies [1, 11]. RT-PCR, DNA sequencing, and phylogenetic analysis were performed as reported previously [11]. In all, 73 rodents of 8 species were captured in 320 trap-nights (a trap success rate [number of rodents captured/number of traps set at location] of 23%). The trap success rates were 23% at the patient’s residence, 7% at Substation A, 20% at Substation B, 15% at Substation C, 50% at Substation D, and 33% at the ranger station. Rodents were captured in the patient’s yard, garage, and storage shed; none was trapped inside the home. In contrast, rodents were caught inside and outside buildings at the work sites and ranger station.

The deer mouse *Peromyscus maniculatus* was the dominant species captured at all sites and the only rodent with antibodies to SNV. Species that were seronegative for SNV included meadow voles, wood rats, chipmunks, ground squirrels, pocket mice, and pinyon mice. Seven (17%) of 42 deer mice were seropositive for SNV. Of these 7, 5 were also positive for antibodies to SNV by RT-PCR. The number of rodent samples from each individual site was too small to adequately compare seroprevalences of SNV. Amplification of viral cDNA was performed with lung tissue from all seropositive rodents as well as with the patient’s blood sample. The sequences of all amplification products were determined and were compared with one another by phylogenetic tree analysis. The maximum parsimony tree (figure 1) is based upon a 274-nucleotide portion of the SNV GI glycoprotein gene that shows a high degree of synonymous variation [11]. This tree shows that the patient (Case B-Mono) and a deer mouse from a woodpile inside Substation A (PM-Mono-129) were infected with viruses with identical nucleotide sequences.

The deer mice whose SNV was not identical to that of the patient (as determined by RT-PCR) were trapped at the patient’s residence (PM-Mono-167), Substation D (PM-Mono-148), and the ranger station (PM-Mono-179 and PM-Mono-186); these sites were located 10 km, 15 km, and 4 km from Substation A, respectively. The deer mice who were positive for SNV by RT-PCR appeared to form two clusters on the parsimony tree (figure 1); the grouping partially correlates with the location of the traps. The two deer mice from the ranger station were trapped within 10 m of each other and examination of SNV from these mice revealed that it differed by only a single nucleotide. In contrast, the other three deer mice who were positive for SNV by RT-PCR had closely related sequences but were trapped 10–15 km apart.

![Figure 1. Unweighted parsimony tree depicting relationships among the GI glycoprotein genes of the virus isolated from the case-patient (Case B-Mono) and those from the viruses isolated from rodents trapped at various candidate exposure sites (PM-Mono-129, PM-Mono-148, PM-Mono-167, PM-Mono-179, PM-Mono-186). The human case and five rodents are indicated in bold and are underlined. The length of the horizontal branch is proportional to the number of nucleotide substitutions. Vertical distances are for clarity only; the vertical line between Case B-Mono and PM-Mono-129 indicates that those sequences are identical. The number of nucleotide substitutions between two sequences can be determined by adding all of the individual branch lengths (numbers above horizontal lines) between the two sequences. Human-derived sequences are indicated by “Case,” and rodent-derived sequences are indicated by “PM” (Peromyscus maniculatus) or “PL” (Peromyscus leucopus); a specimen number and state or county of origin follows the rodent species. PM-Mono-C107 and PM-Mono-CC74 are SNV isolates obtained from another investigation of human cases at a nearby site in Mono County, California [12]. Other SNV sequences included for comparative purposes came from case-patients from Arizona (Case E-AZ), New Mexico (Case P-NM, Case 3H226-NM), and Idaho (Case W-ID) as well as from rodents from New Mexico (PL-NM-1153), Arizona (PM-AZ-92), Santa Barbara County, California (PM-Santa Barbara-CA-41), and Kern County, California (PM-Kern-CA-150).](https://academic.oup.com/cid/article-abstract/22/5/841/361062)
Risk Management

State and local public health officials worked with the utility company management to develop a comprehensive plan to reduce the risk of their employees being occupationally exposed to hantavirus in the future. The utility company incorporated hantavirus into their injury and illness prevention program. The issues specifically addressed included protective clothing and respiratory protection for employees who were responsible for cleaning up rodent-infested areas; consultation with industrial hygienists, engineers, and vector-control agencies regarding long-term rodent management and habitat modification at substations and other problem work areas; and employee training on avoidance of rodents and rodent droppings.

Discussion

As newly emerging infectious diseases are recognized, there is a need to develop risk assessment and management strategies for persons who may be exposed to agents that cause these illnesses while performing job duties. Occupational exposures to hantaviruses found in Europe and Asia are well documented [3, 13, 14]. For example, factors that increase the risk of exposure to hantaviruses that cause hemorrhagic fever with renal syndrome (HFRS) have been described for personnel working in laboratory animal facilities and agriculture [15]. In the United States, antibodies to hantaviruses have been documented in several occupational groups, including longshoremen, granary workers, and forestry employees [14].

Case-control studies of HFRS and HPS have implicated several specific risk factors, including increased rodent density and increased cleaning, for persons who live in private residences [16–18]. It is notable that the patient described in our report had worked in rodent-infested buildings for substantial periods and that he had cleaned rodent droppings at his primary work site during the incubation period for hantavirus. Studies of more patients with potential occupational exposures are needed to better define work conditions and activities associated with HPS.

The genetic match between viruses from the patient and PM-Mono-129 (figure 1) provides evidence that these viruses originated from the same site and that the patient was exposed while on the job. To be certain that the patient contracted his infection at Substation A, it would be necessary to have a more complete representation of SNV genotypes from rodents found at each candidate site of exposure; this proof could be obtained by performing genetic studies of many more seropositive rodents. Examination of SNV genotypes from rodents trapped in nearby ‘‘nonexposure’’ locations during the period in which the patient was exposed to the rodent droppings would also be useful in determining how variable the genotype of the G1 glycoprotein is in this geographic region. However, the finding of genetic identity between G1 glycoprotein genes from a rodent from Substation A and from the patient supports a higher exposure risk at Substation A, where the patient reported direct contact with rodent droppings.

Employees who are engaged in activities that bring them into contact with rodents or their excretions should consider taking special precautions (e.g., respiratory protection) to avoid exposure to hantavirus. This report emphasizes the need for health care providers to be aware of occupational groups within the utility company industry and similar industries who may be at an increased risk of exposure to hantavirus, and it also underscores the importance of incorporating strategies for preventing exposure to hantavirus and other emerging infectious diseases into occupational safety programs.

Acknowledgments

The authors are indebted to Jack Bertman, Dennis Lampson, Jim Goodlow, Lewis Melina, and Robin Erickson (Mono County Health Department, Mammoth Lakes, California) and Jim Clover, Jim Hitchcock, and Chuck Myers (Vector-Borne Disease Section, California Department of Health Services, Sacramento and Ontario), for their assistance with field investigations. They also thank Barryett Enge and David Cottam (Viral and Rickettsial Disease Laboratory, California Department of Health Services, Berkeley), Norah Torrez-Martinez and Wannin Song (University of New Mexico School of Medicine, Albuquerque), and the staff at Northern Inyo Hospital (Bishop, California) for technical assistance. Finally, they thank the employees of the utility company, and the staff of the forest service ranger station as well as the patient and his family, who kindly allowed them to conduct this survey at their work site and residence.

References


