

Central Washington University
ScholarWorks@CWU

All Faculty Scholarship for the College of the
Sciences

College of the Sciences

8-2002

Human Exposure to Herpesvirus B–Seropositive Macaques, Bali, Indonesia

Gregory A. Engel

Lisa Jones-Engel


Michael A. Schillaci

Komang Gde Suaryana

Artha Putra

See next page for additional authors

Follow this and additional works at: <https://digitalcommons.cwu.edu/cotsfac>

 Part of the [Animal Diseases Commons](#), [Immunology and Infectious Disease Commons](#), [Virology Commons](#), and the [Virus Diseases Commons](#)

Authors

Gregory A. Engel, Lisa Jones-Engel, Michael A. Schillaci, Komang Gde Suaryana, Artha Putra, Agustin Fuentes, and Richard Henkel

Human Exposure to Herpesvirus B–Seropositive Macaques, Bali, Indonesia

Gregory A. Engel,* Lisa Jones-Engel,* Michael A. Schillaci,* Komang Gde Suaryana,† Artha Putra,† Agustin Fuentes,‡ and Richard Henkel,§

Herpesvirus B (*Cercopithecine herpesvirus 1*) has been implicated as the cause of approximately 40 cases of meningoencephalitis affecting persons in direct or indirect contact with laboratory macaques. However, the threat of herpesvirus B in nonlaboratory settings worldwide remains to be addressed. We investigated the potential for exposure to herpesvirus B in workers at a “monkey forest” (a temple that has become a tourist attraction because of its monkeys) in Bali, Indonesia. In July 2000, 105 workers at the Sangeh Monkey Forest in Central Bali were surveyed about contact with macaques (*Macaca fascicularis*). Nearly half of those interviewed had either been bitten or scratched by a macaque. Prevalence of injury was higher in those who fed macaques. Serum from 31 of 38 Sangeh macaques contained antibodies to herpesvirus B. We conclude that workers coming into contact with macaques at the Sangeh Monkey Forest are at risk for exposure to herpesvirus B.

Herpesvirus B (*Cercopithecine herpesvirus 1* [CeHV-1]) is an alphaherpesvirus endemic to macaques of South and Southeast Asia (1). In macaques, the usual host, CeHV-1 causes mild symptoms, similar to the effects of herpes simplex virus 1 in humans (1). Clinical findings in macaques usually consist of oral or perioral vesicular lesions. After initial infection, CeHV-1 remains latent in the dorsal root or trigeminal ganglia of an infected macaque and can be shed periodically through herpetic lesions.

In contrast to its benign course in macaques, in humans CeHV-1 produces a fulminating meningoencephalitis with a mortality rate approaching 70% (2). Since first reported in the 1930s, a total of 43 cases of CeHV-1 have been diagnosed worldwide, all reported from the United States, Great Britain, or Canada, exclusively in people who had direct or indirect contact with laboratory macaques (2–6).

Several modes of primate-to-human transmission have been implicated, most involving direct exposure of tissue or fluid from an infected macaque. Weigler’s 1992 review of human CeHV-1 cases (1) found that most were infected through direct bite and scratch wounds: one case resulted from direct contamination of a preexisting wound with monkey saliva, two cases occurred after lacerations from culture bottles containing macaque cells, and two occurred in persons punctured by needles previously used in macaques. One case of human-to-human transmission has been documented, when infection developed in the wife of a man who subsequently died of a CeHV-1 infection. She had a rash on her finger that came into contact with a vesicular lesion on her husband’s arm, at the site of a monkey bite. The most recent documented

case occurred in 1997 at the U.S. Yerkes Regional Primate Center, where a young worker who received an ocular exposure with contaminated body fluids from a CeHV-1–positive macaque became ill and subsequently died (6).

Two published case series have studied transmission of CeHV-1 from primates to humans. Friefeld et al. (7) examined prevalence of antibodies to CeHV-1 in primate handlers exposed to bites, scratches, needle-sticks, and mucosal splashes from laboratory macaques. None of the 166 exposed persons had antibodies to the virus. Similarly, in a small study of eight persons bitten by pet macaques, none seroconverted (8). Nevertheless, the threat of herpesvirus B has led the Centers for Disease Control and Prevention to recommend strict precautions for persons who come into contact with monkeys in occupational settings (2,6,9,10).

The threat of CeHV-1 to humans in nonlaboratory contexts worldwide has yet to be studied, despite the fact that the laboratory macaques that harbor the virus originated in Asia or are descendants of macaques originating there. Macaque species range throughout South and Southeast Asia and have adapted well to human-altered environments. In turn, macaques have become incorporated into religious mythology and local culture. Hindus in Indonesia, Nepal, and India, for example, regard macaques as sacred (11), and in many areas protected macaque populations have thrived alongside dense human settlements for centuries. On the Indonesian island of Bali, more than 44 Balinese Hindu temples have, over the centuries, become refuges for populations of free-ranging macaques (11). These monkeys subsist at least in part on the food and flower offerings left by Balinese Hindu worshippers. Over time, some of these temples have become tourist destinations known as “monkey forests,” where macaques are the premier attraction. At the Sangeh Monkey Forest in Central Bali, dozens of local photographers make a living by enticing macaques to

*University of New Mexico, Albuquerque, New Mexico, USA; †Udayana University, Denpasar, Bali, Indonesia; ‡Central Washington University, Ellensburg, Washington, USA; and §Georgia State University, Atlanta, Georgia, USA

climb onto visitors and selling the photos of visitors posing with macaques. However, photographers and tourists are by no means the only humans who come into contact with these macaques. At Sangeh, three troops of macaques, a total of >200 monkeys, range throughout the monkey forest, along a road lined with merchants' shops, and into the adjacent town. Their daily travels afford ample opportunity for contact with shop owners and others who pass near the monkey forest.

This study investigated human-macaque contact in people who work in and around the Sangeh Monkey Forest. Our aim was to examine the risk of exposure to CeHV-1 from Sangeh's macaques. We addressed two principal questions: 1) does human-primate contact capable of transmitting CeHV-1 occur in this context? and 2) are the Sangeh macaques seropositive for antibodies to this virus?

Methods

Study Site

The Sangeh Monkey Forest is located in the Abiensem District, central Bali, approximately 20 km north of Denpasar, Bali's most populous city. Built in the 17th century by the royal family of Mengwi, it is now maintained by the people of the village of Sangeh. The macaque (*Macaca fascicularis*) population at Sangeh totals approximately 200 animals. Their range extends through the monkey forest proper and across a main road that abuts the area. The monkey forest, a 6-hectare stand of *Dipterocarpus hasseltii* and other climax forest trees with heights up to 40 m, actually provides little food for the macaques. Most of their caloric intake is from daily provisions provided by temple workers and food given to them by visitors.

Visitors interact with macaques mainly in an open area adjacent to the principal temple structures. The main entrance to the monkey forest is reached by a promenade lined by shops offering clothing and souvenirs, in addition to peanuts and bananas for the macaques. Groups of macaques also make their way down this thoroughfare or along the shop roofs during their daily ranging.

Ethnographic Surveys

The local temple committee provided researchers with a list of 250 persons whose work in and around the monkey forest brought them into regular proximity with the macaques. Most persons were either photographers or merchants whose shops lined the road leading to the monkey forest. Of this group, 105 persons (42%) volunteered to participate in the study. In July 2000, a questionnaire that focused on human-primate contact, written in Bahasa Indonesia, the national language of Indonesia, was administered by Balinese team members. Information requested included the type and number of contacts with Sangeh's macaques, injuries or sequelae resulting from macaque bites and scratches, and treatments of those injuries.

Field Protocol

Universal precautions were observed during animal handling and specimen collection to minimize the risk of pathogen transmission between researchers and nonhuman primate subjects. All methods were reviewed and approved by the University of New Mexico's Institutional Animal Care and Use Committee.

Macaques were opportunistically darted within the monkey temple area and surrounding forest by using a Pneu-Dart air-powered pistol (Pneu-Dart Inc., Williamsport, PA). Darts were loaded with 15 mg of Telazol (Fort Dodge Laboratories, Fort Dodge, IA; tiletamine HCl/zolazepam HCl) to ensure initial sedation. Immediately after darting, the macaque was moved to a secluded area and <5 mg/kg of supplemental Telazol was administered for sedation. Six milliliters of blood was withdrawn from the femoral vein, placed in a serum separator tube, and centrifuged in the field to extract the serum. Sera were frozen and stored at -20°C. Dental eruption sequence was recorded and used as a proxy measure of chronologic age. Macaques were observed and allowed to recover from anesthesia in a quiet area before being released. No macaques were injured as a result of this protocol.

Laboratory Techniques and Data Analysis

Enzyme-linked immunosorbent assays (ELISA) to detect antibodies to CeHV-1 in macaque sera were performed at the B-Virus Reference Laboratory at Georgia State University (12). Questionnaire and serologic data were entered into a spreadsheet, and univariate analysis was performed with the JUMP-IN 4 statistical software package (SAS Institute, Inc., Cary, NC, version 4). The association between macaque CeHV-1 seropositivity and age was determined by chi-square test. Prevalence ratios, regarded by some as the most appropriate tool for analyzing cross-sectional studies, were calculated to describe associations between demographic variable and feeding behavior and prevalence of bite and scratch exposure (13,14). For all variables the category with the lowest prevalence was used as the referent. Calculation of prevalence ratios and 95% confidence intervals [CI] was performed with the NCSS Statistical Software package (Kaysville, UT).

Results

Seroprevalence of Antibodies to CeHV-1 in Macaques

Demographic and serologic data on the macaques sampled are shown (Table 1).

Thirty-one (81.6%) of the 38 sampled macaques tested positive for antibodies to CeHV-1. One (25%) of the four juveniles, two (33.3%) of the six subadults, and all 28 adults (100%) were seropositive. The association of increased seroprevalence with increasing age was statistically significant ($p < 0.0001$, chi square). These figures are consistent with those of other seroprevalence studies performed on captive and non-captive macaques (15-18).

Table 1. Seroprevalence of antibodies to *Cercopithecine herpesvirus 1* (herpesvirus B) in Sangeh macaques (*Macaca fascicularis*), Bali

Age group ^a /sex	No. (%) ELISA positive/total
Juvenile	
Male	1/4 (25)
Female	—
Total	1/4 (25)
Subadult	
Male	1/4 (25)
Female	1/2 (50)
Total	2/6 (33.3)
Adult	
Male	21/21 (100)
Female	7/7 (100)
Total	28/28 (100)
All ages	
Male	23/29 (79.3)
Female	8/9 (88.8)
Total	31/38 (81.6)

^aJuveniles are defined as 1–3 years of age; subadults as 3–5 years of age; adults as >5 years of age.

—, no data; ELISA, enzyme-linked immunosorbent assay.

Demographics of the Human Study Population

Demographic data for the human study participants are summarized in Table 2. The mean age of the study population was 35.6 years (standard deviation 12.0). The median age was 35 years (range 18–75); 63.8% of respondents were male. The three most common occupations were merchants (34.3%), photographers (24.8%), and farmers (18.1%). Consistent with cultural norms, 35 of the 36 merchants were women who owned shops along the main promenade leading to the temple area. Of the 38 female respondents, all but 3 were merchants. All respondents were Balinese Hindus residing in the village of Sangeh.

Bite and Scratch Results

Prevalences of bite and scratch injuries are summarized in Table 3. Results from the survey showed that 76.9% of persons had touched or been touched by a macaque. Almost a third (29.5%) reported that they had been bitten on at least one occasion by a macaque; 39% had been scratched at least once; some persons reported being both bitten and scratched. Of the 51 injured, 94.1% were holding food at the time of the injury.

Anatomic Distribution of Injuries, Sequelae, and Wound Treatment

By anatomic distribution, 64.7% of the 51 injuries were to the hands, 7.8% to the arms, 7.8% to the legs, 11.8% to the head, and 7.8% to the back or buttocks. Of the 51 persons bitten or scratched, 51% reported that the wound had bled, 9.8%

reported a rash at the bite site, 11.8% noted fever after their injury, 3.9% had headache, and 5.9% had generalized weakness. None of those interviewed reported symptoms consistent with previously described cases of CeHV-1 infection.

In terms of wound treatment, 54.9% of those injured did not use any kind of treatment for their scratch or bite injuries, 21.6% washed the wound with an antiseptic solution, and 19.6% washed with soap. Of the injured, 11.7% were treated at a medical clinic, and 2% were treated by an herbalist. Five of the persons treated at the medical clinic received antibiotics; none received antiviral prophylaxis.

Prevalence Ratios for Exposures to Monkey Bites and Scratches

Table 4 presents prevalence and prevalence ratios for exposure to monkey bites or scratches, by respondent's age

Table 2. Demographic characteristics of human study participants, Sangeh, Bali

Demographic characteristic	No. (% of total)
All persons	105 (100)
Age group	
<20	11 (10.5)
20–29	27 (25.7)
30–39	26 (24.8)
40–49	31 (29.5)
>49	10 (9.5)
Sex	
Male	67 (63.8)
Female	38 (36.2)
Marital status	
Single	29 (27.6)
Married	75 (71.4)
Widowed	1 (1)
Education	
≤6th grade	38 (36.2)
7th–9th grade	23 (21.9)
10th–12th grade	38 (36.2)
University	6 (3.8)
Occupation	
Merchant	36 (34.3)
Photographer	26 (24.8)
Farmer	19 (18.1)
Security	7 (6.7)
Government employee	5 (4.8)
Traditional guard	5 (4.8)
Laborer	4 (3.8)
Other	3 (2.9)

Table 3. Prevalence of bite and scratch injuries from monkeys to humans, Sangeh, Bali

Descriptor	No. (%) of persons (n = 105)
Bitten	31 (29.5)
Bitten more than once	7 (6.7)
Scratched	41 (39.0)
Scratched more than once	15 (14.3)
Bitten or scratched	51 (48.6)
Possessed food at time of injury	48 (94.1)
All persons	105 (100)

group, sex, occupation, level of education, and whether the respondent fed macaques. Persons in their twenties had the highest prevalence of exposure (63%) while those <20 years of age had the lowest (9%). The exposure rate for male participants (62.7%) was higher than that for females. The prevalence ratio for males was 2.6, indicating that their exposure was more than two and a half times as common as that of female study participants. Of occupations represented by more than five persons, farmers (94.7%) had the highest exposure prevalence, followed by photographers (57.7%), merchants (25%), and security guards (14.3%). For farmers, the prevalence ratio was 6.6, with a 95% CI (1.61, 78.46). Exposure rates were higher in persons with grade school or less education (57.9%) and middle school education (56.5%) than those who had reached high school (39.5%) and university (16.7%).

Most men (89.4%) and women (79%) reported that they had offered food to macaques. Injury was more prevalent in persons who reported feeding macaques (55.6%) than in those who denied feeding them (7.1%). The prevalence ratio in those who fed monkeys was 8.3, indicating that exposure in those who fed monkeys was nearly eight times more common than in those who did not feed monkeys.

Discussion

Exposure to Macaque Bites and Scratches at the Sangeh Monkey Forest

The survey data presented in this study suggest that many workers in and around the Sangeh Monkey Forest have been bitten or scratched by a macaque. Serologic data show that >80% of these macaques have been exposed to CeHV-1. Current understanding of the pathophysiology of this virus predicts that seropositive animals periodically shed it through mucosal lesions (1,17–19). Therefore, these workers report injuries that put them at risk for exposure to the virus.

Wound Care and the Risk for Pathogen Transmission

In contrast with laboratory settings, where protocols regarding care of nonhuman primate-inflicted wounds specify immediate and thorough decontamination, awareness of the risk of zoonotic disease in workers at Sangeh is low. Data on care of macaque bite and scratch wounds reflected this. Lack

of prompt and rigorous wound care may thus pose added risk for transmission of CeHV-1 and other nonhuman primate-borne pathogens for workers and visitors at Sangeh.

CeHV-1 a Cause of Human Disease on Bali?

The above data suggest that human-macaque contact capable of transmitting CeHV-1 is relatively common at Sangeh. Indeed, these data may represent only a small fraction of the human-macaque contact occurring there. Wheatley (11) reported that up to 40% of visitors to Sangeh are bitten by a macaque. Given that thousands of tourists visit Sangeh during a typical month, a reasonable estimate of the annual number of injuries inflicted by macaques is in the thousands, and Sangeh is but one of a handful of monkey forests on Bali that draw large numbers of visitors. Yet no case of human CeHV-1 infection has been reported in Bali, either in association with monkey forests or in any other nonlaboratory context (K. Suaryana, pers. comm.). Several explanations for this observation can be offered.

Recent work supports the existence of three genotypes of CeHV-1, each associated with a distinct species of macaque (20). It has been suggested that only the strain associated with rhesus macaques (*M. mulatta*), the most commonly used laboratory macaque, causes virulent disease in humans (20). This hypothesis is based on the observation that, when the identity of the source animal was known, human CeHV-1 infection was associated with exposure to rhesus macaques but never solely with exposure to other macaque species, including *M. fascicularis*, the species found in Bali, and the second-most commonly used macaque in laboratory research.

The hypothesis that only certain species of macaques may carry a pathogenic strain of CeHV-1 has not been tested. Almost all such infections have occurred in laboratory settings where rhesus macaques constitute most of laboratory nonhuman primates. In addition, rhesus macaques tend to be the more aggressive species. Thus, rhesus macaques may simply cause more injuries and hence be associated with more viral exposures than other macaque species. Furthermore, no case of CeHV-1 infection in humans has been reported in India or Nepal, countries where human-macaque contact is known to occur outside the laboratory and where the predominant macaque species is the rhesus monkey. However, no active surveillance for CeHV-1 is carried out in Nepal and India, and persons diagnosed with encephalitis in these countries are highly unlikely to be tested for this virus.

Another possible explanation for the lack of reported human CeHV-1 cases is that opportunities for exposure to actual virus may be rare. Previous research suggests that, in the laboratory setting, macaques seropositive for CeHV-1 antibodies, even under certain kinds of stress, infrequently shed the virus (17,19). If the macaques at Sangeh behave similarly and shed the virus infrequently, the opportunity for exposure may be rare. One must also take into account the probability that the macaques that bite or scratch carry CeHV-1. Specifically, infant and juvenile macaques are less likely to test posi-

Table 4. Prevalence and prevalence ratios for exposure to monkey bites or scratches by different variables, Sangeh, Bali

Variable	Total no. (%) persons exposed	Prevalence ratio	95% CI	p value
Age group (yrs)				
<20	11 (9.0)	1.0	—	—
20–29	27 (63.0)	6.9	1.75, 117.30	0.045
30–39	26 (50.0)	5.5	1.34, 93.86	0.080
40–49	31 (45.2)	5.0	1.21, 84.81	0.100
>49	10 (60.0)	6.0	1.47, 113.93	0.056
Sex				
Female	38 (23.7)	1.0	—	—
Male	67 (62.7)	2.6	1.55, 5.26	0.002
Occupation				
Security	7 (14.3)	1.0	—	—
Merchant	36 (25.0)	1.7	0.41, 29.67	0.564
Photographer	26 (57.7)	4.0	1.08, 66.80	0.138
Farmer	19 (94.7)	6.6	1.90, 108.43	0.041
Other	17 (47.1)	2.2	0.80, 55.40	0.215
Education				
University	6 (16.7)	1.0	—	—
High school	38 (39.5)	2.4	0.64, 38.91	0.356
Middle school	23 (56.5)	3.4	0.93, 55.53	0.190
Grade school or none	38 (57.9)	3.5	0.99, 56.51	0.177
Fed monkeys				
No	15 (6.7)	1.0	—	—
Yes	90 (55.6)	8.3	2.08, 142.05	0.029

CI, confidence intervals.

tive for anti-CeHV-1 antibodies than older macaques and are thus, as a group, probably less likely to shed virus (1,15). Data from the Ubud Monkey Forest (A. Fuentes, pers. comm.) suggest that adult male and female macaques accounted for approximately half of all bites, with the remainder attributed to juveniles. At least half the bites, therefore, are caused by macaques that are less likely to harbor the virus. Unfortunately, no large-scale studies of CeHV-1 shedding in wild macaques have been performed to date, so the rate at which these animals shed the virus is unknown.

CeHV-1 infection in humans might also be underreported if the disease is rare, especially since awareness of this virus among health-care providers in Bali is low. The symptoms of infection might be mistaken for those of other neurologic diseases, such as polio or Guillain-Barré syndrome. Thus it is theoretically possible that humans reporting mild sequelae following monkey bites and scratches might be describing a mild variant of B virus syndrome. We are aware of no research examining the prevalence of antibodies to CeHV-1 in persons with neurologic syndromes in Asia.

Finally, resistance to CeHV-1 in the exposed human population may explain the lack of reported cases. Human populations living in proximity to the monkey forests in Bali have been living commensally with macaques for centuries. These populations could conceivably acquire immunologic resistance to the virus as a result of frequent exposure over time.

More data are needed to assess whether CeHV-1 poses a substantial public health threat to workers at Sangeh. Serologic data from humans who have been scratched or bitten by macaques could help to determine whether these persons have been exposed to the virus. In addition, a thorough search for cases of human CeHV-1 infection, in Southeast Asia as well as in South Asia, could yield further insight into the epidemiology of this virus in the human population. However, the virus has not surfaced as a recognized infectious threat for humans in areas where the two species have lived commensally for centuries.

Public Health Significance of Nonhuman Primate Zoonoses

Data such as those presented in this study can play an important role in preventing the emergence of primate zoonoses. By understanding where and how interspecies contact occurs we may take informed steps toward reducing the likelihood of interspecies pathogen transmission. Specifically, information about interspecies pathogen transmission may help to identify priority areas for intervention to reduce the emergence of nonhuman primate-borne zoonoses.

Implications and Recommendations for Public Health Practice

Over the past decades, the widespread use of laboratory nonhuman primates as models for the study of human diseases has led to the exposure of laboratory workers to infectious agents endemic in nonhuman primates. Relatively little is known about the epizootology of infectious agents harbored by nonlaboratory macaques and even less is known about the transmission of these agents from macaques to humans with whom they come into contact. CeHV-1 is one of a few infectious agents known to be transmitted from macaques to humans. Serologic, virologic, and molecular studies have demonstrated that a handful of laboratory workers have acquired infection with *Simian foamy virus* as well as *Simian immunodeficiency virus* and simian retrovirus, though no known adverse health effects were associated with these infections (21–29). Very little is known about the effects of these viruses on humans, since the number of seroconverters is low, <10 cases for each virus. No serologic studies outside laboratory settings have been conducted. Given the paucity of data on the effects of CeHV-1 and other endemic macaque pathogens on humans, especially in nonlaboratory settings, reducing the kinds of interspecies contact most likely to lead to pathogen transmission would be prudent. Our data suggest some possible interventions. Because workers who fed macaques were far more likely to be bitten or scratched than their other col-

leagues, an intervention aimed at reducing injuries in workers might logically focus on feeding practices. Restricting feeding to specially trained personnel who distribute food to macaques in such a manner as to avoid physical contact with them is one strategy that has worked effectively at other monkey forests (A. Fuentes, pers. comm.). Also, since approximately two thirds of those who are injured report injuries to the hands, the use of protective gloves should be advocated for personnel coming into frequent contact with monkeys. Of course, implementing these kinds of changes would require a commitment from the community to change the way the monkey forests operate.

Another incentive for enacting such changes is that monkey forests and the macaques that live in them are valuable cultural and economic resources to the communities in which they are located. Disease transmission in the opposite direction, namely human to nonhuman primate, may threaten these macaques. If so, regulating interspecies contact could help to preserve the monkey forests as an economic resource for the community. Perhaps a long-term strategy to preserve monkey forests will recognize the importance of minimizing infectious risks to both humans and macaques.

Acknowledgments

The authors thank P. Sukrama, D. Cohn, J. Heidrich J. Froehlich, R. Kyes, and A. Stone, as well as Pak Budi, Pak Nyoman, the Sangeh Temple Committee, G. Emel, Y. Singeo, G. Kelemen, H. Engel, L. Engel, and Singapore Airlines for facilitating this research; J. Supriatna and the Indonesian Directorate of Nature Conservation and Wildlife Management; and E. Bedrick, J. Cheek, D. Goade, and K. Tollestrup for their comments on earlier drafts.

This research was supported in part by grants from the Chicago Zoological Society, the University of New Mexico's Student Research and Allocations Committee and the University of New Mexico School of Graduate Studies. This research presented here was approved by the University of New Mexico's Human research and Review Committee and Institutional Animal Care and Use Committee.

Dr. Engel is on the faculty of the Swedish/Providence Family Practice Residency and on the clinical faculty of the Department of Family Medicine at the University of Washington. His current research focus is bidirectional pathogen transmission between humans and nonhuman primates in Asia.

References

1. Weigler BJ. Biology of B virus in macaque and human hosts: a review. *Clin Infect Dis* 1992;14:555-67.
2. Holmes GP, Chapman LE, Stewart JA, Straus SE, Hilliard JK, Davenport DS, et al. Guidelines for the prevention and treatment of B-virus infections in exposed persons. *Clin Infect Dis* 1995;20:421-39.
3. Hummeler K, Davidson WL, Henle W, LaBocchetta AC, Ruch HG. Encephalomyelitis due to infection with *Herpesvirus simiae* (Herpesvirus B): report of two fatal laboratory cases. *N Engl J Med* 1959;261:64-8.
4. Centers for Disease Control. B-virus infection in humans—Michigan. *MMWR Morb Mortal Wkly Rep* 1989;38:453-4.
5. Holmes GP, Hilliard JK, Klontz KC, Rupert AH, Schindler CM, Parrish E, et al. B-virus (*Herpesvirus simiae*) infection in humans: epidemiological investigations of a cluster. *Ann Intern Med* 1990;112:833-9.
6. Centers for Disease Control and Prevention. Fatal *Cercopithecine herpesvirus 1* (B Virus) infection following a mucocutaneous exposure and interim recommendations for worker protection. *MMWR Morb Mortal Wkly Rep* 1998;47:1073-6.
7. Freifeld AG, Hilliard J, Southers J, Murray M, Savarese B, Schmitt JM, et al. A controlled seroprevalence survey of primate handlers for evidence of asymptomatic herpesvirus B infection. *J Infect Dis* 1995;171:1031-4.
8. Ostrowski SR, Leslie MJ, Parrott T, Abelt S, Piercy PE. B-virus from pet monkeys: an emerging threat in the United States? *Emerg Infect Dis* 1998;4:117-21.
9. Wells DL, Lipper SL, Hilliard JK, Stewart JA, Holmes GP, Herrmann KL, et al. *Herpesvirus simiae* contamination of primary rhesus monkey kidney cell cultures. CDC recommendations to minimize risks to laboratory personnel. *Diagn Microbiol Infect Dis* 1989;12:333-5.
10. Centers for Disease Control. Guidelines for the prevention of *Herpesvirus simiae* (B virus) infection in monkey handlers. *MMWR Morb Mortal Wkly Rep* 1987;36:680-2, 687-9.
11. Wheatley BP. The sacred monkeys of Bali. Prospect Heights (IL): Waveland Press, Inc.; 1999.
12. Katz D, Hilliard JK, Eberle R, Lipper SL. ELISA for detection of group-specific antibodies in human and simian sera induced by herpes simplex and related simian viruses. *J Virol Methods* 1986;14:99-109.
13. Lee J. Odds ratio or relative risk for cross-sectional data? *Int J Epidemiol* 1994; 23:201-2.
14. Zocchetti C, Consonni D, Bertazzi PA. Estimation of prevalence rate ratios from cross-sectional data. *Int J Epidemiol* 1995;24:1064-5.
15. Kessler MJ, Hilliard JK. Seroprevalence of B virus (*Herpesvirus simiae*) antibodies in a naturally formed group of rhesus macaques. *J Med Primatol* 1990;19:155-60.
16. Keeble SA. B virus infection in monkeys. *Ann NY Acad Sci* 1960;85:960-9.
17. Weigler BJ, Hird DW, Hilliard JK, Lerche NW, Roberts JA, Scott LM. Epidemiology of *Cercopithecine herpesvirus 1* (B virus) infection and shedding in a large breeding cohort of rhesus macaques. *J Infect Dis* 1993;167:257-63.
18. Weigler BJ, Roberts JA, Hird DW, Lerche NW, Hilliard JK. A cross-sectional survey for B virus antibody in a colony of group housed rhesus macaques. *Lab Anim Sci* 1990;40:257-61.
19. Weier EC, Bhatt PN, Jacoby RO, Hilliard JK, Morgenstern S. Infrequent shedding and transmission of *Herpesvirus simiae* from seropositive macaques. *Lab Anim Sci* 1993;43:541-4.
20. Smith LS, Black DH, Eberle R. Molecular evidence for distinct genotypes of monkey B virus (*Herpesvirus simiae*) which are related to the macaque host species. *J Virol* 1998;72:9224-32.
21. Schweizer MA, Turek R, Hahn H, Schliephake A, Netzer KO, Eder G, et al. Markers of foamy virus infection in monkeys, apes and accidentally infected humans: appropriate testing fails to confirm suspected foamy virus prevalence in humans. *AIDS Res Hum Retroviruses* 1995; 11:161-70.
22. Schweizer M, Falcone V, Gange J, Turek R, Neumann-Haefelin D. Simian foamy virus isolated from an accidentally infected human individual. *J Virol* 1997;71:4821-4.
23. Callahan ME, Switzer WM, Mathews AL, Roberts BD, Heneine W, Folks TM, et al. Persistent zoonotic infection of a human with simian foamy virus in the absence of an intact orf-2 accessory gene. *J Virol* 1999;73:9619-24.
24. Sandstrom PA, Phan KO, Switzer WM, Fredeking T, Chapman L, Heneine W, et al. Simian foamy virus infection among zoo keepers. *Lancet* 2000;355:551-2.
25. Centers for Disease Control and Prevention. Nonhuman primate spumavirus infections among persons with occupational exposure—United States. *MMWR Morb Mortal Wkly Rep* 1996;46:129-31.

26. Centers for Disease Control and Prevention. Seroconversion to simian immunodeficiency virus in two laboratory workers. *MMWR Morb Mortal Wkly Rep* 1992;41:36.
27. Centers for Disease Control and Prevention. Anonymous survey for simian immunodeficiency virus (SIV) seropositivity in SIV-laboratory researchers-United States. *MMWR Morb Mortal Wkly Rep* 1992;41:814-5.
28. Khabbaz RF, Heneine W, George JR, Parekh B, Rowe T, Woods T, et al. Brief report: infection of a laboratory worker with simian immunodeficiency virus. *N Engl J Med* 1994;330:172-7.
29. Lerche NW, Switzer WM, Yee JL, Shanmugam V, Rosenthal AN, Chapman LE, et al. Evidence of infection with simian type-D retrovirus in persons occupationally exposed to nonhuman primates. *J Virol* 2001;75:1783-9.

All material published in *Emerging Infectious Diseases* is in the public domain and may be used and reprinted without special permission; proper citation, however, is appreciated.

Address for correspondence: Lisa Jones-Engel, Department of Psychology and National Primate Research Center, University of Washington, Seattle, WA 98195, USA; fax: 206-543-7959; e-mail: ga_engel@yahoo.com

Research Studies: Articles should be 2,000 to 3,500 words and should include references, not to exceed 40. Use of subheadings in the main body of the text is recommended. Photographs and illustrations are encouraged. Provide a short abstract (150 words) and a brief biographical sketch of first author—both authors if only two.

These articles report laboratory and epidemiologic results within a public health perspective. Although these reports may be written in the style of traditional research articles, they should explain the value of the research in public health terms and place the findings in a larger perspective (e.g., "Here is what we found, and here is what the findings mean").

OPPORTUNITIES FOR PEER REVIEWERS

The editors of *Emerging Infectious Diseases* seek to increase the roster of reviewers for manuscripts submitted by authors all over the world for publication in the journal. If you are interested in reviewing articles on emerging infectious disease topics, please e-mail your name, address, qualifications or curriculum vitae, and areas of expertise to eideditor@cdc.gov

At *Emerging Infectious Diseases*, we always request reviewers' consent before sending manuscripts, limit review requests to three or four per year, and allow 2-4 weeks for completion of reviews. We consider reviewers invaluable in the process of selecting and publishing high-quality scientific articles and acknowledge their contributions in the journal once a year.

Even though it brings no financial compensation, participation in the peer-review process is not without rewards. Manuscript review provides scientists at all stages of their career opportunities for professional growth by familiarizing them with research trends and the latest work in the field of infectious diseases and by improving their own skills for presenting scientific information through constructive criticism of those of their peers. To view the spectrum of articles we publish, information for authors, and our extensive style guide, visit the journal web site at www.cdc.gov/eid.

For more information on participating in the peer-review process of *Emerging Infectious Diseases*, e-mail eideditor@cdc.gov or call the journal office at 404-371-5329.

Upcoming in

EMERGING INFECTIOUS DISEASES

A Peer-Reviewed Journal Tracking and Analyzing Disease Trends

Research

Spatial Analysis of Human Granulocytic Ehrlichiosis in the 12-Town Area around Lyme, Connecticut

Molecular Epidemiology of Measles Viruses, United States, 1997-2001

Demographic Factors Associated with Hantavirus Infection in Bank Voles (*Clethrionomys glareolus*)

Public Health Impact of Reemergence of Rabies, New York

First Characterization of a Cluster of VanA-Type Clycopeptide-Resistant *Enterococcus faecium* in a Colombian Hospital

A Case-Case Comparison of *Campylobacter coli* and *Campylobacter jejuni* Infection: A tool for Hypothesis Generation

The Impact of the El Niño/Southern Oscillation on Visceral Leishmaniasis, Brazil

Characterization of Flagella Produced by Clinical Strains of *Stenotrophomonas maltophilia*

For a complete list of articles included in the September issue, and for articles published online ahead of print publication, see <http://www.cdc.gov/ncidod/eid/upcoming.htm>