

2. Tabachnick WJ, Powell JR. A worldwide survey of genetic variation in the yellow fever mosquito, *Aedes aegypti*. *Genet Res.* 1979;34:215–29. <http://dx.doi.org/10.1017/S0016672300019467>
3. Kim H, Lee C, Kim M. A case of imported dengue hemorrhagic fever [in Korean]. *Korean Journal of Infectious Diseases.* 1995;27:403–6.
4. Kwon S, Cho B, Yoon S, Cho Y, Kim I, Chung M, et al. A case of dengue hemorrhagic fever imported from Africa [in Korean]. *Korean Journal of Infectious Diseases.* 2000;32:467–9.
5. Korea Tourism Organization. 2008 Survey for international travel in Koreans [in Korean]. Korea Tourism Organization, 2009 [cited 2012 May 15]. http://korean.visitkorea.or.kr/kor/t2Main.kto?func_name=t2main
6. Wu JY, Lun ZR, James AA, Chen XG. Dengue fever in mainland China. *Am J Trop Med Hyg.* 2010;83:664–71. <http://dx.doi.org/10.4269/ajtmh.2010.09-0755>
7. Gjenero-Margan I, Aleraj B, Krajcar D, Lesnikar V, Klobučar A, Pem-Novosel I, et al. Autochthonous dengue fever in Croatia, August–September 2010. *Euro Surveill.* 2011;16: pii: 19805.
8. La Ruche G, Souarès Y, Armengaud A, Peloux-Petiot F, Delaunay P, Desprès P. First two autochthonous dengue virus infections in metropolitan France, September 2010. *Euro Surveill.* 2010;15: pii: 19676.
9. Effler PV, Pang L, Kitsutani P, Vorndam V, Nakata M, Ayers T, et al. Dengue fever, Hawaii, 2001–2002. *Emerg Infect Dis.* 2005;11:742–9.

Address for correspondence: Dong-Woo Lee, Epidemic Intelligence Service, Division of Public Health Crisis Responses, Korea Centers for Disease Control and Prevention, 187 Osongsaengmyeong 2(i)-ro, Cheongwon-gun, Chungcheongbuk-do 363-951, South Korea; email: aryumput2@gmail.com

EID
online
www.cdc.gov/eid

Brucellosis in Takins, China

To the Editor: Brucellosis is a highly contagious bacterial disease and one of the world's major zoonoses. It is responsible for enormous economic losses in livestock, and it threatens human health and wildlife populations (1). In most host species, brucellosis primarily affects the reproductive system, leading to concomitant loss in productivity of affected animals (1). Brucellae have been found in wildlife, such as bison, elk, and wild boar, potentially posing a threat for zoonosis (2). Currently, the genus *Brucella* comprises 10 species, which are divided according to host specificity and ability to cause chronic infections in human and animals (3,4). Most *Brucella* species are associated primarily with certain hosts, presumably the result of evolutionary adaptation to a successful host. *Brucella melitensis* is the species most pathogenic in humans and the species most commonly involved in ovine and caprine brucellosis.

In January 2009, in the nature reserve in Qinling Mountains, China, hygromas were found on the knees, stifles, hocks, haunches, and bursae between the nuchal ligament and the primary thoracic spines of 10 free-ranging takins (*Budorcas taxicolor*). The hygroma contents and tissue samples were collected by using aseptic technique, packed separately, cooled immediately, and stored frozen at -20°C until cultured. The samples were streaked onto blood agar and MacConkey agar and incubated aerobically or anaerobically with 5% CO₂ at 37°C for 4 days.

Tiny gram-negative coccobacilli were isolated. The organism was nonmotile at 20°C and 37°C, and it stained red with the Stamp modification of the Ziehl-Neelsen method. The organism was identified as *B. melitensis* by the Vitek 2 GN identification system (bioMérieux,

Marcy l'Étoile, France). The isolate was urease positive, catalase positive, and oxidase positive. It did not require carbon dioxide for growth and did not produce hydrogen sulfide. The isolate could be agglutinated by A-monospecific antiserum but not by M-monospecific antiserum or rough *Brucella*-specific antiserum. It was sensitive to Berkeley and Iz phages at routine test dilution but not sensitive to Tbilisi, Weybridge, Firenze, and R/C phages. According to classical biotyping methods, the isolate was identified as *B. melitensis* biotype 2 (5).

Molecular identification by 16S rRNA gene sequencing was used in this study (6). According to nucleotide–nucleotide GenBank search by using BLAST (<http://blast.ncbi.nlm.nih.gov/>), the sequence was 100% identical to the sequences of 16S rDNA of brucellae, especially reference strains including *B. melitensis* 16M (GenBank accession no. NC_003317), *B. abortus* biovar 1 str. 9–941 (NC_006932), *B. suis* 1330 (NC_004310), *B. canis* American Type Culture Collection 23365 (NC_010103), and *B. ovis* American Type Culture Collection 25840 (NC_009505). The isolate was further confirmed as *B. melitensis* according to the 731-bp product by using AMOS-PCR, which discriminates among species by the unique locations of the IS711 element (7,8). The restriction pattern of the *omp2b* gene by *Hinf* I was accordant with pattern 3 reported by Cloeckaert et al. (9); this finding further indicated that the isolate was *B. melitensis* (9).

The takin (*Budorcas taxicolor*) is a ruminant belonging to the family Bovidae, subfamily Caprinae, genus *Budorcas* (Figure). Takins are found in eastern Asia and Southeast Asia and are listed as “vulnerable A2cd” by the International Union for Conservation of Nature (10). Brucellosis might pose a major direct or indirect threat to the conservation of endangered species,



Figure. Takin (*Budorcas taxicolor*).

such as takins, and can be a source of conflicts among stakeholders in conservation efforts.

Several antelopes, such as takins, serows (*Capricornis sumatraensis*), and gorals (*Naemorhedus goral*), occur sympatrically in the Qinling Mountains of China. Because brucellae are often transmitted by direct contact or exposure to a contaminated environment, it is possible that rather than being a natural reservoir for the bacteria, takins are infected horizontally by contact with birth exudates from other infected animals (2). However, information on brucellosis prevalence in those sympatric ruminants in China is insufficient. Therefore, further investigation and research are needed to test this hypothesis. Also, brucellosis is endemic among livestock and human populations in western China. Because domestic sheep and goats are grazed in the mountains, infections in livestock can spill over into wildlife, such as takins. Brucellosis in humans might

also be caused by exposure to infected animals during activities like the handling, skinning, and eviscerating of the carcasses of infected animals.

Whether takins are the reservoir host or an accidental host for *B. melitensis* is still unclear. To further understand the interaction of brucellae among wildlife, domestic animals, and humans, and for purposes of brucellosis management and control, systematic investigations of brucellosis prevalence among wildlife should be conducted.

This study was supported by grants to the Wildlife-borne Diseases Surveillance Project from the State Forestry Administration of China, the joint project of the National Wildlife Research Center of the US Department of Agriculture and the Institute of Zoology of the Chinese Academy of Sciences (O760621234); the Science and Technology Support Project of the Eleventh Five-Year Plan of China (2009BAI83B01); and the Special Major Project of the National Department of Science and Technology (2009ZX10004-109).

**Jing Luo, Zhigao Zeng,
Yanling Song,
and Hongxuan He**

Author affiliation: Chinese Academy of Sciences, Beijing, People's Republic of China

DOI: <http://dx.doi.org/10.3201/eid1809.120069>

References

1. Cutler SJ, Whatmore AM, Commander NJ. Brucellosis—new aspects of an old disease. *J Appl Microbiol*. 2005;98:1270–81. <http://dx.doi.org/10.1111/j.1365-2672.2005.02622.x>
2. Bienen L, Tabor G. Applying an ecosystem approach to brucellosis control: can an old conflict between wildlife and agriculture be successfully managed? *Frontiers in Ecology and the Environment*. 2006;4:319–27. [http://dx.doi.org/10.1890/1540-9295\(2006\)4\[319:AAEATB\]2.0.CO;2](http://dx.doi.org/10.1890/1540-9295(2006)4[319:AAEATB]2.0.CO;2)
3. Corbel MJ. Brucellosis: an overview. *Emerg Infect Dis*. 1997;3:213–21. <http://dx.doi.org/10.3201/eid0302.970219>
4. Scholz HC, Nockler K, Gollner C, Bahn P, Vergnaud G, Tomaso H, et al. *Brucella inopinata* sp. nov., isolated from a breast implant infection. *Int J Syst Evol Microbiol*. 2010;60:801–8. <http://dx.doi.org/10.1099/ijs.0.011148-0>
5. Alton GG, Jones LM, Angus RD, Verger JM. Techniques for the brucellosis laboratory. Paris: Institut National de la Recherche Agronomique; 1988. p. 42–60.
6. Gee JE, De BK, Levett PN, Whitney AM, Novak RT, Popovic T. Use of 16S rRNA gene sequencing for rapid confirmatory identification of *Brucella* isolates. *J Clin Microbiol*. 2004;42:3649–54. <http://dx.doi.org/10.1128/JCM.42.8.3649-3654.2004>
7. Bricker BJ, Halling SM. Differentiation of *Brucella abortus* bv. 1, 2, and 4, *Brucella melitensis*, *Brucella ovis*, and *Brucella suis* bv. 1 by PCR. *J Clin Microbiol*. 1994;32:2660–6.
8. Ewalt DR, Bricker BJ. Validation of the abbreviated *Brucella* AMOS PCR as a rapid screening method for differentiation of *Brucella abortus* field strain isolates and the vaccine strains, 19 and RB51. *J Clin Microbiol*. 2000;38:3085–6.
9. Cloeckaert A, Verger JM, Grayon M, Grepinet O. Restriction site polymorphism of the genes encoding the major 25 kDa and 36 kDa outer-membrane proteins of *Brucella*. *Microbiology*. 1995;141:2111–21. <http://dx.doi.org/10.1099/13500872-141-9-2111>

10. International Union for Conservation of Nature. IUCN red list of threatened species. Version 2011.2. *Budorcas taxicolor*. 2008 [cited 2012 May 29]. <http://www.iucnredlist.org/apps/redlist/details/3160/0http://www.iucnredlist.org>

Address for correspondence: Hongxuan He, Institute of Zoology, Chinese Academy of Sciences, No. 1-5 Beichenxi Rd, Chaoyang District, Beijing, People's Republic of China; email: hehx@ioz.ac.cn

Measles and Secondary Hemophagocytic Lymphohistiocytosis

To the Editor: We found interesting the article by Lupo et al. about a case of fatal measles in an immunocompetent 29-year-old woman (Fatal measles without rash in immunocompetent adult, France; <http://dx.doi.org/10.3201/eid1803.111300>). Perhaps, however, the possible diagnosis of secondary hemophagocytic lymphohistiocytosis (HLH) should also have been considered in that setting.

HLH is a potentially fatal hyperinflammatory syndrome characterized by histiocyte proliferation and hemophagocytosis. HLH may be inherited (i.e., primary, familial, generally occurring in infants) or may occur at any age secondary to infection, malignancy, or rheumatologic disease. Secondary HLH is determined according to clinical criteria from the HLH Study Group of the Histiocyte Society, which require >5 of the following for a diagnosis: fever; splenomegaly; cytopenia (affecting >2 cell lineages); hypertriglyceridemia or hypofibrinogenemia; hemophagocytosis in the bone marrow, spleen, or lymph

nodes; low or absent natural killer cell cytotoxicity; hyperferritinemia; and elevated levels of soluble CD25.

We conducted a PubMed search and found 5 articles that described 6 cases of HLH in patients with measles (1–5). Pneumonia was described in all of them (1–5), and central nervous system involvement was described in 3 (1,4). Four cases occurred in children, 3 of them immunocompetent (1,3–5). The 2 adults were an immunocompetent 18-year-old man who had acute respiratory distress (2) and a 19-year-old man with acute lymphocytic leukemia who had measles pneumonia and acute hemorrhagic leukoencephalitis (1). The only fatal case occurred in an immunocompromised 8-year-old boy with giant-cell pneumonia (3).

The identification of hemophagocytosis in bone marrow aspirate represents only 1 of the 5–8 criteria needed for a diagnosis of HLH; conversely, a bone marrow aspirate lacking hemophagocytosis does not rule out the diagnosis of HLH. Still, we believe HLH should be considered for any patient with fever and pancytopenia, especially in the presence of respiratory distress or multiorgan dysfunction. An appropriate therapy could save the patient (Secondary hemophagocytic syndrome in adults: a case series of 18 patients in a single institution and a review of literature; <http://dx.doi.org/10.1002/hon.960>).

**Chiara Iaria,
Maria Silvana Leonardi,
Agata Buda, Maria Luisa Toro,
and Antonio Cascio**

Author affiliations: Azienda Ospedaliera Piemonte-Papardo, Messina, Italy (C. Iaria); and Policlinico “G. Martino” University Hospital, Messina (M.S. Leonardi, A. Buda, M.L. Toro, A. Cascio)

DOI: <http://dx.doi.org/10.3201/eid1809.120235>

References

- Pearl PL, Abu-Farsakh H, Starke JR, Dreyer Z, Louis PT, Kirkpatrick JB. Neuropathology of two fatal cases of measles in the 1988–1989 Houston epidemic. *Pediatr Neurol*. 1990;6:126–30. [http://dx.doi.org/10.1016/0887-8994\(90\)90046-4](http://dx.doi.org/10.1016/0887-8994(90)90046-4)
- Komatsuda A, Chubachi A, Miura AB. Virus-associated hemophagocytic syndrome due to measles accompanied by acute respiratory failure. *Intern Med*. 1995;34:203–6. <http://dx.doi.org/10.2169/internalmedicine.34.203>
- Nakano T, Shimono Y, Sugiyama K, Nishihara H, Higashigawa M, Komada Y, et al. Clinical features of measles in immunocompromised children. *Acta Paediatr Jpn*. 1996;38:212–7. <http://dx.doi.org/10.1111/j.1442-200X.1996.tb03472.x>
- Yamamoto K, Otake M, Takayanagi M. Therapeutic effect of cyclosporin A combined with methylprednisolone pulse therapy on hemophagocytic syndrome with the central nervous system involvement [in Japanese]. *No To Hattatsu*. 2002;34:66–71.
- Joshi R, Phatarpekar A, Currimbhoy Z, Desai M. Haemophagocytic lymphohistiocytosis: a case series from Mumbai. *Ann Trop Paediatr*. 2011;31:135–40. <http://dx.doi.org/10.1179/1465328111Y.0000000009>

Address for correspondence: Antonio Cascio, Programma di Infettivologia Speciale, Medicina Tropicale e delle Migrazioni e Parassitologia, Policlinico “G. Martino”, Via Consolare Valeria n. 1, 98125 Messina, Italy; email: acascio@unime.it

In Response: We thank Iaria et al. (1) for their comments on our letter reporting an unusual case of fatal measles without rash in an immunocompetent woman who manifested cytopenias and an intractable acute respiratory distress syndrome (2). The authors suggest that secondary hemophagocytic lymphohistiocytosis (HLH) could have been considered in this patient.