transmission after natural disasters is low but real and that it is not directly related to the disasters and dead bodies, but primarily associated with the characteristics of the displaced population within the local disease ecology. This belief supports the need for rapid but accurate assessment of health status, risk, and needs, the results of which greatly influence the nature of relief activities (*4*). Key functions of relief teams are communicable diseases surveillance, early warning, and rapid response to epidemic-prone situations or outbreaks.

As an example, on October 26, 2005, after an earthquake in Pakistan, the World Health Organization asked the French military epidemiologic assessment team (1 epidemiologist and 1 veterinarian) to perform a sanitary assessment after cases of acute bloody diarrhea were reported in the camp of Tariqabad (estimated population ≈2,000), near Muzaffarabad. The assessment highlighted a lack of safe water and sanitation facilities, low routine immunization coverage, and disruption of healthcare services.

To prevent further diarrhea, we recommended improving the overall water and sanitation conditions. A medical team from a French nongovernment organization was also provided to help the 1 physician at the camp. Concurrently, we recommended a vaccination campaign as preventive strategy against diseases likely to occur in such conditions: tetanus, diphtheria, and measles. These measures were quickly implemented to reduce the overall risk, and no further unusual increases in disease incidence were noted during the following weeks. As in another outbreak documented in a camp in the Muzaffarabad area (*5*), rapid detection, response, and implementation of control measures are critical for minimizing the illness and death associated with outbreaks in these high-risk populations.

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# Recombinant Sapovirus Gastroenteritis, Japan

**To the Editor:** Sapovirus and norovirus are causative agents of gastroenteritis in children and adults. Norovirus is the most important cause of outbreaks of gastroenteritis, whereas only a few outbreaks of sapovirus have been reported (*1*,*2*). On the basis of complete capsid gene sequences, sapovirus can be divided into 5 genogroups, among which GI, GII, GIV, and GV infect humans, whereas sapovirus GIII infects porcine species.

We report 2 outbreaks of gastroenteritis in Hokkaido, Japan. The first outbreak (A) occurred at a college from May 29 to June 2, 2000. A total of 12 persons (11 students and 1 teacher) reported symptoms of gastroenteritis (nausea, vomiting, stomachache, diarrhea, and fever); 11 stool specimens were collected from days 1 to 7 after onset of illness (Table). These specimens were negative for norovirus (data not shown), but 5 were positive for sapoviruslike viruses by electron microscopy (Table).

The 11 specimens were then examined for sapovirus by using nested reverse transcription–PCR (RT-PCR) as described (*3*). A total of 9 (82%) of 11 specimens were positive for sapovirus. Sequence analysis showed that these 9 viruses had 100% nucleotide identity and likely represented the same sapovirus strain (termed Yak2 strain, GenBank accession no. AB046353). To determine the number of cDNA copies per gram of stool, we performed real-time RT-PCR as described (*4*). The number of sapovirus cDNA copies ranged from $5.36 \times 10^5$ to $7.47 \times 10^9$/g stool (median $5.49 \times 10^9$ copies/g stool) (Table).

The second outbreak (B) occurred at a kindergarten from February 1 to 22, 2005. A total of 23 persons (15 children and 8 adults) reported symp-

Table. Analysis of 18 stool specimens for sapovirus during 2 outbreaks of gastroenteritis, Japan*

| Outbreak, specimen | Date of illness onset | EM | Nested RT-PCR | Real-time PCR† |
|---|---|---|---|---|
| A | | | | |
| Yak1 | Jun 2, 2000 | – | + | $7.47 \times 10^9$ |
| Yak2 | May 29, 2000 | – | – | – |
| Yak3 | May 30, 2000 | – | – | – |
| Yak4 | May 31, 2000 | + | + | $6.55 \times 10^9$ |
| Yak5 | Jun 1, 2000 | – | + | $9.38 \times 10^8$ |
| Yak6 | Jun 1, 2000 | – | + | $1.30 \times 10^8$ |
| Yak7 | May 29, 2000 | – | + | $1.46 \times 10^9$ |
| Yak8 | May 29, 2000 | + | + | $2.78 \times 10^{10}$ |
| Yak9 | Jun 1, 2000 | + | + | $3.00 \times 10^9$ |
| Yak10 | Jun 1, 2000 | + | + | $2.05 \times 10^9$ |
| Yak11 | Jun 1, 2000 | + | + | $5.36 \times 10^5$ |
| B | | | | |
| Nay1 | Feb 17, 2005 | NT | + | $1.65 \times 10^{10}$ |
| Nay2 | Feb 14, 2005 | NT | + | $1.82 \times 10^9$ |
| Nay3 | Feb 18, 2005 | NT | + | $1.14 \times 10^9$ |
| Nay4 | Feb 17, 2005 | NT | + | $5.41 \times 10^{10}$ |
| Nay5 | Feb 16, 2005 | NT | + | $5.26 \times 10^{10}$ |
| Nay6 | Feb 18, 2005 | NT | + | $2.50 \times 10^{10}$ |
| Nay7 | Feb 17, 2005 | NT | + | $2.38 \times 10^{10}$ |

*EM, electron microscopy; RT-PCR, reverse transcription–PCR; NT, not tested.
†cDNA copies/g stool.

toms of gastroenteritis (nausea, vomiting, stomachache, diarrhea, and fever); 7 stool specimens were collected (Table). These specimens were negative for norovirus (data not shown), but all were positive for sapovirus by nested RT-PCR. The 7 sequences from this outbreak had 100% nucleotide identity and likely represented the same sapovirus strain (termed Nay1 strain, GenBank accession no. EF213768). The number of sapovirus cDNA copies ranged from $1.14 \times 10^9$ to $5.41 \times 10^{10}$/g stool (median $2.50 \times 10^{10}$ copies/g stool) (Table).

One positive sapovirus specimen from each outbreak was subjected to further sequence analysis in which a single overlapping PCR fragment covering the partial polymerase gene and capsid gene was amplified. The Yak2 and Nay1 sequences shared ≈71% nucleotide identity for this fragment and likely represented different sapovirus strains. The Yak2 sequence closely matched sapovirus GIV Ehime1107 and SW278 sequences (GenBank accession nos. DQ058829 and AY237420, respectively) and had 98% and 97% nucleotide identity for the entire fragment, respectively (*5*). The Nay1 sequence closely matched the sapovirus GII C12 sequence (AY603425) and had 91% nucleotide identity for the entire fragment.

The Nay1 sequence closely matched the C12 sequence, which was detected in Osaka, Japan, in 2001 (*6*), whereas the Yak2 sequence closely matched the Ehime1107 sequence, which was detected in Matsuyama, Japan, in 2002 (*5*), and the SW278 sequence, which was detected in Sweden in 2003 (*1*). We recently described the C12 strain as intragenogroup recombinant sapovirus strain (*6*), whereas the Ehime1107 and SW278 strains were described as intergenogroup recombinant sapovirus strains (*5*). Our results indicate that recombination sites in intragenogroup and intergenogroup recombinant sapovirus strains were at the polymerase and capsid junction (*5*,*6*). Sapovirus Sydney53 (DQ104360) and Sydney3 strains (DQ104357), which were detected in Australia from August 2001 to August 2004 (*7*), closely matched C12 and Ehime1107/SW278 sequences, respectively. These results showed that recombinant sapovirus strains are stable in the environment and may be globally distributed. Our findings also suggest a changing distribution of sapovirus-associated gastroenteritis in Hokkaido because different sapovirus GI strains were predominant in outbreaks of gastroenteritis in Hokkaido (*8*,*9*).

In a recent study, the number of norovirus cDNA copies per gram of stool specimen was analyzed and a discrepancy was found between the different norovirus genogroups (*10*). Chan et al. found that noroviruses GI and GII showed medians of $8.4 \times 10^5$ and $3.0 \times 10^8$ copies/g of stool specimen, respectively, and speculated that increased viral loads were caused by higher transmissibility of norovirus GII strains (*10*). Our results showed that sapovirus GII Nay1 and GIV Yak2 strains showed higher viral loads than norovirus GII strains. These results suggest that a high degree of shedding of sapovirus GII Nay1 and GIV Yak2 strains may have caused the outbreak of gastroenteritis. However, to elucidate this suggestion, further studies are needed with other sapovirus strains.

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## *Salmonella* Typhimurium in Hihi, New Zealand

**To the Editor:** The recent finding of a previously unrecorded *Salmonella* strain in an endangered New Zealand passerine (the hihi, *Notiomystis cincta*; [*1*]) offers the rare opportunity to observe the initial arrival and pathology of an epizootic and to determine its population-level effect. Over 8 days in February 2006, 6 freshly dead hihi were discovered in a free-living island population. Pathologic findings were similar: birds were in good body condition with substantial subcutaneous fat reserves and no gross lesions in the crop, indicating death from a highly pathogenic disease. Histopathologic examination showed septicemia and inflammatory necrosis of organs, particularly the liver and spleen, typical of salmonellosis in birds (*2*). Microbiologic examination of liver samples isolated heavy growths of the bacterium *Salmonella enterica* serotype Typhimurium DT195. During the same period, 3 more dead hihi were found, but they were too decomposed for postmortem examination.

Hihi are nectar-feeders that declined to near extinction after European colonization of New Zealand and survived on a single island refuge (Hauturu). Since 1980, 14 attempts have been made to reintroduce the species to 6 other sites, resulting in 3 new populations that persist with management. The *S.* Typhimurium DT195 outbreak occurred within a reintroduced population on Tiritiri Matangi Island. Management includes providing supplementary food (sugar water) diluted with local rain water; feeders are sterilized before each use.

Because disease in hihi is closely monitored, the outbreak indicates that *S.* Typhimurium DT195 is a novel serotype for this species. During December 2005, fecal screening of 18 broods (37 nestlings) from Tiritiri Matangi Island found no evidence of enteric pathogens; screenings in February and May 2005 (40 adult and juvenile birds) from Tiritiri Matangi Island similarly returned negative results. Screening in all hihi populations during 2004 also found no evidence of *Salmonella* infection (32 adults and juveniles at Tiritiri Matangi, 29 at Hauturu, and 27 at Kapiti), and a 15-year pathology database from 230 dead hihi collected across these populations and a captive breeding facility lists no salmonellosis cases (J.G. Ewen and M.R. Alley, unpub. data).

Documentation of the emergent stages of infectious disease in endangered species is rare (*3*,*4*). This bacterium strain is absent from New Zealand's livestock and wildlife (www.surv.esr.cri.nz/enteric_reference/non human_salmonella.php). Nontyphoid *Salmonella* spp. are a major health concern worldwide (*5*), and New Zealand conducts intensive surveillance to maintain food safety. The New Zealand Wildlife Health Centre has not reported *S.* Typhimurium DT195 despite necropsies of >3,000 wild birds during 1996–2006, which suggests this strain is rare in New Zealand, despite its presence in other countries (*6*).

*S.* Typhimurium DT195 has been detected in 3 human patients in New Zealand (1 each in 2002, 2003, and 2006). The *S.* Typhimurium DT195 isolated from hihi in the February 2006 outbreak were indistinguishable from those isolated from the human case-patient in 2006 (see [*2*] for methods). Tiritiri Matangi is an isolated island nature reserve 3 km off the New Zealand coast, which prevents movement of hihi to other areas. How this strain appeared in a human patient and as an epizootic in an isolated island nature reserve is intriguing. The most recent human case was diagnosed on the North Island of New Zealand, but the person was not living in close proximity to the birds. Tiritiri Matangi receives ≈30,000 human visitors per year, but whether the person with *S.*