zoonoses is increasing. This increase indicates that Cryptosporidium spp. host specificity is not as stringent as previously thought.

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Liette S. Waldron, Cristel Cheung-Kwok-Sang, and Michelle L. Power
Author affiliation: Macquarie University, Sydney, New South Wales, Australia
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References
1. Fayer R. Taxonomy and species delimitation in Cryptosporidium. Exp Parasitol. 2010;124:90–7. DOI: 10.1016/j.exppara.2009.03.005
2. Morgan-Ryan UM, Fall A, Ward LA, Hijawi N, Sulaiman I, Fayer R, et al. Cryptosporidium hominis n. sp. (Apicomplexa: Cryptosporididae) from Homo sapiens. J Eukaryot Microbiol. 2002;49:433–40. DOI: 10.1111/j.1550-7408.2002.tb00224.x
3. Xiao L, Ryan U. Cryptosporidiosis: an update in molecular epidemiology. Curr Opin Infect Dis. 2004;17:483–90. DOI: 10.1097/00001432-200410000-00014
4. Chalmers RM, Davies AP. Minireview: clinical cryptosporidiosis. Exp Parasitol. 2010;124:138–46. DOI: 10.1016/j.exppara.2009.02.003
5. Waldron LS, Ferrari BC, Power ML. Glycoprotein 60 diversity in C. hominis and C. parvum causing human cryptosporidiosis in NSW Australia. Exp Parasitol. 2009;122:124–7. DOI: 10.1016/j.exppara.2009.02.006
6. Xiao L, Morgan UM, Limor J, Escalante A, Arrowood M, Shulaw W, et al. Genetic diversity within Cryptosporidium parvum and related Cryptosporidium species. Appl Environ Microbiol. 1999;65:3386–91.
7. Xiao L, Limor JR, Li L, Morgan U, Thompson RC, Lal AA. Presence of heterogeneous copies of the small subunit rRNA gene in Cryptosporidium parvum human and marsupial genotypes and Cryptosporidium felis. J Eukaryot Microbiol. 1999;46:445–55.
8. Sulaiman IM, Lal AA, Xiao L. Molecular phylogeny and evolutionary relationships of Cryptosporidium parasites at the actin locus. J Parasitol. 2002;88:388–94.
9. Power ML, Cheung-Kwok-Sang C, Slade M, Williamson S. Cryptosporidium fayeri: diversity within the gp60 locus of isolates from different marsupial hosts. Exp Parasitol. 2009;121:219–23. DOI: 10.1016/j.exppara.2008.10.016
10. Power ML, Sangster NC, Slade MB, Veal DA. Patterns of Cryptosporidium oocyst shedding by eastern grey kangaroos inhabiting an Australian watershed. Appl Environ Microbiol. 2005;71:6159–64. DOI: 10.1128/AEM.71.10.6159-6164.2005

Canine Distemper
Epizootic among Red Foxes, Italy, 2009

To the Editor: Canine distemper virus (CDV) is an enveloped, single-stranded, negative RNA virus of the family Paramyxoviridae, genus Morbillivirus (1). The host range for CDV is broad, and infection has been found in several mammalian species of the families Canidae, Mustelidae, Procyonidae, Ursidae, and Viverridae (2).

Stelvio National Park (SNP) encompasses 1,333 km² of protected land in Italy and covers 2 regions (Lombardia and Trentino Alto Adige); the Lombardia section of the park covers the northern part of Sondrio Province (Valsellina). SNP is surrounded by other parks (Schweitzer National Park, Adamello, and Adamello–Brenta) to form a large protected area (2,500 km²) in the heart of the Alps mountains. Within SNP, the terrestrial mammals that are susceptible to CDV include red foxes (Vulpes vulpes), stoats (Mustela erminea), weasels (Mustela nivalis), pine martens (Martes martes), beech martens (Martes foina), badgers (Meles meles), and bears (Ursus arctos).

In August 2009, three young red foxes were captured in Valtellina (Sondrio), Lombardia, Italy, within the southwestern borders of SNP. The animals showed canine distemper–like signs (e.g., prostration, altered behavior, and conjunctivitis), and CDV infection was confirmed by quantitative reverse transcription–PCR of pooled organs (3). In September and October 2009, another 2 young foxes were captured and found to be positive for CDV. From September on, at least 30 foxes with altered behavior were seen near human habitations and facilities in SNP; 10 were captured. In the same period, infected foxes were also reported from Engadina, Switzerland, at the northern and western borders of SNP. In February 2010, two symptomatic foxes were euthanized in Grosotto, 50 km south of where the initial cases were identified. The epizootic appeared to have originated from the eastern regions of Italy (Trentino Alto Adige, and Veneto), where CDV infection had been reported in red foxes and badgers since August 2006 (4) (Figure). A large CDV epidemic in foxes in southern Bavaria in 2008 has also been described, thus suggesting spread of the virus throughout the Alps area (5).

Reverse transcription–PCR genotyping of the hemagglutinin (H) gene (6) identified 15 CDV strains, which were analyzed and characterized as European genotypes. The full-length H gene of the CDV strains was determined (GenBank accession no. HM120874). Sequence analysis of the H gene indicated that the fox CDV...
strains were highly related to each other (>99.9% nt and 100% aa identity), to the CDV strains identified in foxes in southern Bavaria 2008 (>99.7% nt and 99.3% aa identity; accession nos. FI416336–FI416338), and to a canine strain identified in Hungary during 2005–2006 (99.4% nt and 99.2% aa identity; accession no. DQ889177).

During the CDV epizootic in SNP in 2009, cases of CDV in 3 domestic dogs living within the borders of the park were also reported. Because vaccination against CDV is a core recommendation for dogs, most dogs are expected to be vaccinated and protected; population immunity is high enough to keep CDV infection under control, and only sporadic cases occur (7). Accordingly, the reported CDV cases in dogs were more likely a spillover event caused by the high pressure of CDV infection in SNP wildlife. In addition to foxes, badgers in the same area were also reported to have canine distemper–like disease. These findings are consistent with spread of a multihost epizootic, in which foxes likely played a major role in CDV amplification and diffusion because of their social behavior during reproductive season and because of the wide geographic range over which juveniles migrate during the dispersion period.

Serologic investigations for CDV in some fox populations in Europe have identified antibody prevalence rates of 4%–26.4% (8), suggesting that CDV circulates in foxes in Europe, but these investigations did not examine spatial and temporal variations in CDV activity. Clues for understanding the pattern of CDV disease in wildlife have been provided by structured surveillance of wild canids living in Yellowstone National Park, USA. Yearly fluctuations in CDV seroprevalence with evidence of multihost outbreaks in distinct years, contemporaneously affecting different animal species, have been noted. Cycles of CDV epizootics that swept through the animals in the park were associated with low pup survival rates (9).

In SNP, most foxes captured during the epizootic were juveniles. We have no information on the prevalence rates of CDV-specific antibodies in SNP foxes before the epizootic. However, CDV disease had not been reported in the SNP for at least 10 years, and no animal with CDV infection had been identified in a 2004–2005 survey of red foxes in SNP (10). Similarly, no evidence for CDV infection had been found in carnivores in Trentino Alto Adige during 2001–2002 (10). Accordingly, one can assume that the population immunity in SNP foxes (and in other susceptible hosts) was low.

Adequately controlling CDV infection in wildlife in Europe is difficult. It requires concerted transnational actions, including effective surveillance and prompt gathering and dissemination of information.

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Ribavirin for Lassa Fever Postexposure Prophylaxis

To the Editor: Lassa fever is an acute, viral, hemorrhagic illness endemic to West Africa. Intravenous ribavirin drastically reduces deaths from Lassa fever (1). During outbreaks, oral ribavirin is often considered for postexposure prophylaxis (PEP), but no systematically collected data exist for this indication of drug use (1–5). We therefore conducted a retrospective follow-up study to examine adherence and adverse effects associated with oral ribavirin given as PEP during an outbreak of Lassa fever in Sierra Leone in 2004 (6). During this outbreak, family members and some healthcare workers who had direct contact with patients did not use personal protective equipment and were subsequently prescribed oral ribavirin as PEP (200 mg capsules; Schering-Plough Corporation, Kenilworth, NJ, USA).

Approximately 3 months after the possible exposures, we surveyed 23 (92%) of 25 persons known to have been prescribed ribavirin PEP. Respondents were asked about demographics, medical history, details of possible exposure to Lassa virus (LASV), dosage and duration of ribavirin prescribed and taken, and use of concomitant medications. When possible, serum was obtained and tested by ELISA for LASV-specific immunoglobulin (Ig) M and IgG (7).

The mean age of the 23 respondents was 38 years (range 23–73 years); 14 (61%) were male, 17 (74%) had been exposed at home (during bathing, cleaning, and feeding of family members with Lassa fever), and 6 (26%) had had in-hospital contact with blood and bodily fluids. No needle-stick injuries were reported.

All respondents had begun taking oral ribavirin within 2 days after exposure. The drug was prescribed at a mean dose of 800 mg 1/d (most often as 400 mg 2/d) for 10 days; however, respondents reported actually taking 400–1,200 mg/d. Only 10 (43%) completed the full 10 days of therapy; mean duration of therapy was 8 days (range 1–14 days). No correlation was found between the prescribed daily dose of ribavirin and the likelihood of completing therapy (p = 0.60).

Therapy was completed by 6 (38%) of the 16 (70%) respondents who reported having experienced minor adverse effects and by 4 (57%) of the 7 who reported not having experienced adverse effects (Figure). Many respondents reported having had symptoms even before beginning ribavirin, suggesting at least a partial psychosomatic or other etiology. No correlation was found between likelihood of adverse effects and age (p = 0.18), sex (p = 0.16), or place of exposure (p = 0.63). Six (26%) respondents reported having premorbid health conditions (gastric ulcers, n = 3; gastrointestinal reflux disease, n = 2; hypertension, n = 1), and 15 (65%) took medications in addition to ribavirin during the postexposure period.

Address for correspondence: Vito Martella, Department of Veterinary Public Health. S.p. per Casamassima km 3, 70010, Valenzano, Bari, Italy: email: v.martella@veterinaria.uniba.it