Molecular typing analysis showed that both case-patients had a monoclonal infection caused by *C. difficile* ribotype 027. Both isolates had *tcdA*, *tcdB*, and *cdtB* and had a deletion in *tcdC* (data not shown).

In summary, the described severe cases of *C. difficile* infection in Chile were caused by epidemic *C. difficile* ribotype 027. One of these case-patients required urgent colectomy. These results demonstrate that epidemic *C. difficile* 027 strains are present in South America, highlighting the need for enhanced screening for this ribotype in other regions of the continent.

Acknowledgments

We thank Nigel Minton (University of Nottingham) for kindly providing *C. difficile* strain R20291.

This work was supported by grants from the Comisión Nacional de Investigación en Ciencia y Tecnología (FONDECYT REGULAR 1100971) to M.A.-L. and S.B. and by grants from MECESUP UAB0802, Comisión Nacional de Investigación en Ciencia y Tecnología (FONDECYT REGULAR 1110569) and from the Research Office of Universidad Andres Bello (DI-35-11/R) (to D.P.-S.); and from the Medical Research Center, Facultad de Medicina of the Pontificia Universidad Católica de Chile, Resident Research Project (PG-20/11) to C. H.-R.

Cristian Hernández-Rocha, Jonathan Barra-Carrasco, Marjorie Pizarro-Guajardo, Patricio Ibáñez, Susan M. Bueno, Mahfuzur R. Sarker, Ana Maria Guzman, Manuel Álvarez-Lobos, and Daniel Paredes-Sabja

Author affiliations: Pontificia Universidad Católica de Chile, Santiago, Chile (C. Hernández-Rocha, P. Ibáñez, S.M. Bueno, A.M. Guzman, M. Álvarez-Lobos,); Universidad Andrés Bello, Santiago, Chile (J. Barra-Carrasco, M. Pizarro-Guajardo, D. Pareses-Sabja); and Oregon State University, Corvallis, Oregon, USA (M.R. Sarker, D. Paredes-Sabja)

DOI: http://dx.doi.org/10.3201/eid1808.120211

References

1. McDonald LC, Killgore GE, Thompson A, Owens RC Jr, Kazakova SV, Sambol SP, et al. An epidemic, toxin gene-variant strain of *Clostridium difficile*. N Engl J Med. 2005;353:2433–41. http://dx.doi.org/10.1056/NEJMoa051590
2. Quesada-Gómez C, Rodríguez C, Gamboa-Coronado Mdel M, Rodríguez-Cavalini E, Du T, Mulvey MR, et al. Emergence of *Clostridium difficile* NAP1 in Latin America. J Clin Microbiol. 2010;48:669–70. http://dx.doi.org/10.1128/JCM.01919-09
3. Ballassiano IT, Yates EA, Domingues RM, Ferreira EO. *Clostridium difficile*: a problem of concern in developed countries and still a mystery in Latin America. J Med Microbiol. 2012;61:169–79. http://dx.doi.org/10.1099/jmm.0.037077-0
4. Wilson KH, Kennedy MJ, Fekety FR. Use of sodium taurocholate to enhance spore recovery on a medium selective for *Clostridium difficile*. J Clin Microbiol. 1982;15:443–6.
5. Dawson LF, Donahue EH, Cartman ST, Barton RH, Bundy J, McNerney R, et al. The analysis of para-cresol production and tolerance in *Clostridium difficile* 027 and 012 strains. BMC Microbiol. 2011;11:86. http://dx.doi.org/10.1186/1471-2180-11-86
6. Fedorko DP, Williams EC. Use of cyclerin-cefoxitin-fructose agar and L-proline-aminoepipetidase (PRO Discs) in the rapid identification of *Clostridium difficile*. J Clin Microbiol. 1997;35:1258–9.
7. Rupnik M. *Clostridium difficile* toxinootyping. Methods Mol Biol. 2010;646:67–76. http://dx.doi.org/10.1007/978-1-60327-365-7_5
8. Bidet P, Barbut F, Lalande V, Burg-hoffner B, Petit JC. Development of a new PCR-ribotyping method for *Clostridium difficile* based on ribosomal RNA gene sequencing. FEMS Microbiol Lett. 1999;175:261–6. http://dx.doi.org/10.1111/j.1574-6968.1999.tb13629.x

Address for correspondence: Daniel Paredes-Sabja, Laboratorio de Mecanismos de Patogénesis Bacteriana, Departamento de Ciencias Biológicas, Facultad de Ciencias Biológicas, Universidad Andrés Bello, Santiago, Chile; email: daniel.paredes.sabja@gmail.com

Zoonotic Pathogens among White-Tailed Deer, Northern Mexico, 2004–2009

To the Editor: Intense wildlife management for hunting affects risks associated with zoonotic pathogens (1). White-tailed deer (*Odocoileus virginianus*) are increasingly managed by fencing, feeding, watering, and translocation to increase incomes from hunting in northern Mexico (2). These deer also play a major role in dissemination and reintroduction of pathogens and vectors from Mexico into the United States (3,4). White-tailed deer are suitable reservoir hosts for *Mycoplasma bovis* (1), and an *M. bovis*-positive white-tailed deer was recently found in Tamaulipas in northeastern Mexico (2). Brucellosis is widespread in many animal hosts in Latin America (5) and thus of interest in white-tailed deer. Another major zoonosis, sometimes linked to raw deer meat consumption, is hepatitis E, which is caused by genotypes of hepatitis E virus (HEV) (6). HEV is increasingly prevalent in red deer (*Cervus elaphus*) (7), but its prevalence in white-tailed deer is unknown.

The objective of this study was to determine the prevalence of zoonotic pathogens in white-tailed deer in northern Mexico. This study was conducted under a scientific collecting permit issued by the Mexican Division of Animal and Wildlife Health and on 8 ranches in 3 states in northern Mexico (−26–28°N, 99–100°W).

Serum samples (n = 347) were collected during 2004–2009 in a cross-sectional survey for antibodies against HEV, *Brucella* spp., and mycobacteria. Deer were opportunistically sampled during live-capture operations as described by Cantú et al. (8). Bleeding was performed by using jugular venipuncture and vacuum tubes.
from at 6 of 8 sampling sites; in deer
PPA3 (Table). Antibody responses
against bovine PPD, and 2.6% (95%
CI 6%–13%) for antibodies against
antibodies against
HEV, 0.4% (95% CI 0%–2%) for antibodies
antibodies against all pathogens
samples prevented testing for
cross-reacting with 2 widely used
mycobacterial antigens, bovine
purified protein derivative (PPD)
and paratuberculosis protoplasmatic
antigen 3 (PPA3), was conducted as
described (9). The sensitivity and
specificity of this assay have not been
established for white-tailed deer, but
it has been used in seroprevalence
studies of wild boar and fallow deer
(9,10).

Insufficient volumes of serum
samples prevented testing for
antibodies against all pathogens
(Table). Limited serum volume and
lack of other (organ) samples also
precluded additional analyses to verify
presence of pathogens.

Prevalence was 62.7% (95% CI
54%–70%) for antibodies against
HEV, 0.4% (95% CI 0%–2%) for antibodies
against Brucella spp., 8.9%
(95% CI 6%–13%) for antibodies
against bovine PPD, and 2.6% (95% CI
1%–5%) for antibodies against
PPA3 (Table). Antibody responses
to bovine PPD were detected in deer
from 6 of 8 sampling sites; in deer
from 3 of these sites, antibodies were
also detected against PPA3 antigen.
Seroprevalence against bovine PPD
was higher than that against PPA3 ($\chi^2$
10.9, df 1, p<0.01).

This cross-sectional survey of
white-tailed deer in northern Mexico
detected antibodies to several pathogens relevant to public
and animal health. High prevalence
of antibodies against HEV and
frequent detection of antibodies
against mycobacterial antigens are
public health concerns. Prevalence
of antibodies against HEV were 3×
higher than that reported for red deer in Europe (7). This result suggests
wide circulation of HEV in the study
region and warrants further research,
including detection and sequencing
of virus RNA. Low Brucella spp.
antibody prevalence confirms results
of a study in this region (8).

Antibody responses to bovine PPD
were detected in serum samples
from deer from most sampling
sites, occasionally in the absence
of antibodies against PPA3. These
results, and a recent report of an M.
bovis–positive white-tailed deer from
this region (2), suggest that these
deer may be contracting M. bovis in
northern Mexico. If one considers
that white-tailed deer are M. bovis
reservoirs in other parts of North
America and that risk factors such
as supplemental feeding are present
in northern Mexico, there is a high
risk for pathogen transmission to
animals and humans (1). White-tailed
deer are probably exposed to several
pathogens that are relevant to animal
health in northern Mexico.

Although this cross-sectional
survey provided only an indication
of pathogen prevalence in the study
populations, high antibody prevalence
to HEV and mycobacterial antigens
requires antigen-targeted surveillance.
Risks associated with pathogen
translocation by white-tailed deer are
also relevant to neighboring states in
Mexico and the United States.

Acknowledgments
We thank M. Villar and B. Peralta for
providing ELISA antigen and A. Cantú
and co-workers for providing deer serum
samples.

This study was supported by the
Caesar Kleberg Wildlife Research Institute,
Texas A&M University–Kingsville, and
European Union Framework Program
grant 212414 TB-STEP.

Citlaly Medrano,
Mariana Boadella,
Hugo Barrios, Antonio Cantú,
Zeferino García,
José de la Fuente,
and Christian Gortazar

Author affiliations: Instituto de Investigación en Recursos Cinegéticos, Ciudad Real, Spain (C. Medrano, M. Boadella, J. de la Fuente, C. Gortazar); Universidad Autónoma de Tamaulipas, Ciudad Victoria, Mexico (C. Medrano, H. Barrios); Instituto Nacional de Investigaciones Forestales, Agrícolas, y Pecuarias, Tamaulipas, Mexico (A. Cantú); Instituto Nacional de Investigaciones Forestales, Agrícolas, y

Table. Prevalence of serum antibodies against zoonotic pathogen antigens among white-tailed deer on 8 ranches, northern Mexico,
2004–2009*

| Ranch no. | State     | Municipality | Sample size | HEV        | Brucella spp. | Bovine PPD | PPA3       |
|-----------|-----------|--------------|-------------|------------|---------------|------------|-------------|
| 1         | Tamaulipas| Guerrero     | 1           | 0/1 (0)    | 0/1 (0)       | 0/1 (0)    | 0/1 (0)    |
| 2         | Tamaulipas| Guerrero     | 106         | 41/71 (57.7)| 0/102 (0)    | 9/102 (8.8)| 0/102 (0)  |
| 3         | Tamaulipas| Nuevo Laredo | 37          | 21/27 (77.7)| 0            | 0/37 (0)   | 0/37 (0)   |
| 4         | Nuevo León| Unknown      | 35          | 0          | 0            | 3/35 (8.5) | 2/35 (5.7) |
| 5         | Coahuila  | Guerrero     | 35          | 1/1 (100)  | 0/33 (0)     | 2/33 (6.0) | 1/33 (3.0) |
| 6         | Coahuila  | Hidalgo      | 17          | 4/6 (66.6) | 1/16 (6.2)   | 3/16 (18.7)| 5/16 (31.2)|
| 7         | Coahuila  | Guerrero     | 66          | 16/19 (84.2)| 0/39 (0)    | 7/39 (18.0)| 0/39 (0)   |
| NA        | Total     | NA           | 347         | 89/142 (62.7)| 1/231 (0.4) | 27/303 (8.9)| 8/303 (2.6)|

*HEV, hepatitis E virus; PPD, purified protein derivative; PPA3, paratuberculosis protoplasmatic antigen 3; NA, not applicable.
KIs Virus and Blood Donors, France

To the Editor: KIs-V is a new putative virus identified recently in the blood of persons in Japan (1). First partial sequence of KIs-V was characterized unexpectedly, when PCR primers were used that were directed primarily to the consensus domain of helicase of positive-stranded RNA viruses. Extensive physicochemical and molecular analysis suggested that KIs-V is an enveloped virus with a circ器ular, double-stranded RNA genome of ≈9,500 bp (prototype isolate: GenBank accession no. AB550431); its genetic diversity is presumed to be extremely low because the 4 complete genomes already characterized in Japan harbor strict identical sequences. The 13 potential genes identified by silico analysis exhibit an overall low sequence homology to other known viral proteins (1). Until now, KIs-V epidemiologic data have been related only to the original study in which the authors analyzed plasma samples from 516 blood donors categorized into 4 groups by alanine aminotransferase (ALT) level (either <60 IU/L or >60 IU/L) and the presence or absence of hepatitis E virus (HEV) antibodies. As a result, KIs-V DNA was detectable at elevated prevalence in the high ALT level/HEV antibody–positive group (36%, n = 100); viral loads, checked for a few samples, ranged from 10^6 to 10^9 copies/mL. KIs-V DNA also was identified in HEV antibody–negative samples, with low or high ALT level (<0.8%, n = 120, and 1%, n = 100, respectively) (1).

To gain insights about the potential presence of this virus in the blood of persons in France, we investigated KIs-V DNA in the plasma of 576 healthy blood donors (mean age 40 years; 306 men; men:women 1:1.13). Blood samples were collected in vacuum tubes (Vacutainer, SST, Becton Dickinson, Meylan, France) and centrifuged, and plasma aliquots were stored at −80°C until use. Nucleic acids were extracted from 1-mL plasma volumes (MagNA Pure LC, Roche Diagnostics, Meylan, France) (2) and tested for KIs-V DNA by using the same nested PCR system for screening Japanese blood donors (1). Briefly, one tenth (5 μL) of extracted nucleic acids were first amplified by using primers 101-C (5′-CTCGTCCTCGTGTCATC-3′) and N101-B (5′-AACATTTGAAACGTCATGTCC-3′) (0.8 μM each) in a 50-μL mix containing deoxynucleotide triphosphates (0.2 mM each) (Roche) and 2 U Taq DNA polymerase (Invitrogen, Cergy Pontoise, France) with its corresponding buffer. One microliter of the amplification mixture was subsequently used in a second-round PCR with primers KS-2 (5′-CTCGTCCTCGTGTCATC-3′) and N101-D (5′-CA TTTGCCTCCGCTGGAGATG-3′) under the same conditions as above. The amplification conditions for first-