of the prevalence and distribution of the infection in wildlife and livestock populations on the Zimbabwe side of the Great Limpopo Transfrontier Conservation Area is urgently needed. Control options in wildlife are limited (2,10), but chances of success are greater if control measures are initiated at the early stage of disease spread into a new area. Adequate risk-mitigation strategies should be developed and implemented to reduce the risk for bovine TB transmission to livestock and humans living at the periphery of the unfenced Gonarezhou National Park. Failure to promptly assess the situation and adopt appropriate measures would have far-reaching conservation, economic, and public health consequences, not only for Zimbabwe but also for the political and social acceptance of the transfrontier conservation areas in southern Africa.

Acknowledgments

We thank the Department of National Parks and Wildlife Management Authority of Zimbabwe for permission to operate in Gonarezhou National Park and for efficient field support; Stuart Hargreaves for permission to publish this letter; Mike Lagrange, the Africa Wildlife Management and Conservation team, John Mc Taggart, and Hugo Van der Westhuizen for their assistance in wildlife capture operations; and Dewald Keet for provision of unpublished data.

This work was conducted within the framework of the Research Platform “Production and Conservation in Partnership” (RP-PCP) and the Animal and Human Health for the Environment and Development project. It was funded by the European Union Partnership in the South Development project. It was funded by the Ministère Français des Affaires Etrangères through the French Embassy in Zimbabwe (RP-PCP grant 2008). The processing of samples by the National Zoological Gardens of South Africa was funded by the United States Fish and Wildlife Service, Wildlife without Borders, Africa Program.

Michel de Garine-Wichatitsky, Alexandre Caron, Calvin Gomo, Chris Foggin, Keith Dutlow, Davies Pfukenyi, Emily Lane, Sébastien Le Bel, Markus Hofmeyr, Tiny Hlokwe, and Anita Michel

Author affiliations: Centre de Coopération Internationale en Recherche Agronomique pour le Développement, Harare, Zimbabwe (M. de Garine-Wichatitsky, A. Caron, S. Le Bel); Centre de Coopération Internationale en Recherche Agronomique pour le Développement, Montpellier, France (M. de Garine-Wichatitsky, S. Le Bel); University of Pretoria, Pretoria, South Africa (A. Caron, A. Michel); University of Zimbabwe, Harare (C. Gomo, D. Pfukenyi); Department of Veterinary Services, Harare (C. Foggin, K. Dutlow); National Zoological Gardens of South Africa, Pretoria (E. Lane); South Africa National Parks, Kruger National Park, South Africa (M. Hofmeyr); and Agricultural Research Council–Onderstepoort Veterinary Institute, Pretoria (T. Hlokwe, A. Michel)

DOI: 10.3201/eid1605.090710

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Address for correspondence: Michel de Garine-Wichatitsky, Centre de Coopération Internationale en Recherche Agronomique pour le Développement, Département Environnements et Sociétés, UR AGIRs, 37 Arcturus Rd, Highlands, Harare, Zimbabwe; email: degarine@cirad.fr

No Resistance Plasmid in Yersinia pestis, North America

To the Editor: Plague, caused by Yersinia pestis, is now largely controlled by improved sanitation and the use of antimicrobial drugs. However, before the widespread availability of antimicrobial drugs, an estimated >200 million persons died during pandemics (1). Today, if Y. pestis were to acquire antimicrobial drug resistance determinants, plague could again be a deadly disease.

Antimicrobial drug resistance in Y. pestis has been documented for
only a few strains. The best available information is for 2 strains isolated in Madagascar in 1995 (2), in which resistance was conferred by plasmids not typically found in Y. pestis. Strain 16/95 was resistant to streptomycin only; this resistance was mediated by plasmid pIP1203 (3). Strain 17/95 was resistant to 8 antimicrobial drugs, including some commonly used to treat plague, such as streptomycin, tetracyclines, and sulfonamides (2). Multidrug resistance in 17/95 was mediated by plasmid pIP1202 (4). Both plasmids could be transferred by conjugation from the source Y. pestis strains to other Y. pestis strains and Escherichia coli (3,4). pIP1203 could be transferred from E. coli to Y. pestis in the midgut of co-infected rat fleas (Xenopsylla cheopis), common vectors of plague (5).

Comparative sequence analysis has indicated that pIP1202 shares an almost identical IncA/C backbone with multidrug-resistant (MDR) plasmids from Salmonella enterica serotype Newport SL254 and Yersinia ruckeri YR71, suggesting recent acquisition from a common ancestor (6). In this study, this backbone was detected in numerous MDR enterobacterial pathogens (e.g., E. coli, Klebsiella spp., and multiple Salmonella serotypes) isolated from retail meat products. Many of these plasmids transferred at high rates to a plasmid-free Y. ruckeri strain, indicating the ability to efficiently transfer among species. Meat products examined in that study originated from 9 US states, including western plague-endemic states such as California, Colorado, New Mexico, and Oregon.

To determine whether the IncA/C plasmid backbone previously found in MDR Y. pestis and other species exists in Y. pestis isolates from western U.S. states, we screened Y. pestis DNA. The 713 isolates were collected from humans, small mammals, and fleas in 14 of the 17 western plague-endemic states (Table), including all states that reported human cases during 1970–2002 (7). We used Primer Express software (Applied Biosystems, Foster City, CA, USA) to design a TaqMan-MGB single-probe assay to detect repA, a plasmid replication gene present in the IncA/C plasmid backbone. We based this assay on the repA assay described by Carrattoli et al. (8) and used the same forward primer but a different reverse primer and an additional probe to facilitate screening on a real-time PCR platform.

Real-time PCRs were conducted in 10-μL reaction mixtures that contained 900 nmol/L of forward (5′-GA GAACCAAAAGACAAAGACCTGG A-3′) and reverse (5′-TGCCGGAG ATTCAATGATC-3′) primers, 200 nmol/L of the repA-specific probe (5′-6FAM-AGACTCACCGCA TGCCGGAG ATTCAATGATC-3′), 10 min, and 40 cycles of 95°C for 15 s and 60°C for 1 min. DNA extracts from Y. pestis strain 17/95 (4) and Salmonella enterica serotype Newport strain SL254 (6) were used as positive controls.

Of the 713 Y. pestis isolates screened, none was positive for the IncA/C plasmid backbone, indicating that MDR as mediated by pIP1202-like MDR plasmids described by Welch et al. (6) was not in these samples. This finding is encouraging with regard to public health. However, we screened only for the plasmid backbone; MDR genes may have been in some of these samples but not carried by pIP1202-like MDR plasmids, especially considering that plasmids can be readily integrated into the Y. pestis chromosome (7).

Could MDR Y. pestis arise in North America by acquisition of an MDR plasmid, such as pIP1202, from food-animal production activities in plague-endemic regions? If so, Salmonella spp. would be a likely MDR plasmid donor for several reasons. First, Y. pestis has several plasmids that are highly similar to those in Salmonella spp., indicating active transfer of plasmids between these 2 bacterial groups (6). Second, fleas that are common vectors of plague have been shown to be naturally co-infected with Salmonella spp. and Y. pestis and capable of transmitting both organisms to rodent hosts (9). Third, MDR plasmids are readily transferred to Y. pestis in the flea gut (5). Fourth, transferable MDR plasmids are common among Salmonella spp.

### Table: Yersinia pestis isolates collected from humans, small mammals, and fleas, United States

<table>
<thead>
<tr>
<th>State</th>
<th>No. isolates</th>
<th>Years collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idaho</td>
<td>2</td>
<td>1987, 1997</td>
</tr>
<tr>
<td>Kansas</td>
<td>17</td>
<td>1997, 1999</td>
</tr>
<tr>
<td>North Dakota</td>
<td>2</td>
<td>1986, 1993</td>
</tr>
<tr>
<td>Texas</td>
<td>5</td>
<td>Unknown</td>
</tr>
<tr>
<td>Washington</td>
<td>2</td>
<td>1984</td>
</tr>
</tbody>
</table>
spp. isolates in US food animals (10). Given these linkages, the transfer of an MDR plasmid from *Salmonella* spp. to *Y. pestis* seems possible. However, we emphasize that to date no evidence supports this type of event.

The Centers for Disease Control and Prevention provided many of the DNA samples.

This work was funded by National Institutes of Health, National Institute of Allergy and Infectious Diseases (grant nos. AI070183 and AI30071); the Pacific Southwest Regional Center of Excellence (grant no. AI065359); the Department of Homeland Security Science and Technology Directorate (grant nos. NBCH2070001 and HSHQDC-08-C-00158); and the Cowden Endowment in Microbiology at Northern Arizona University.


Author affiliations: Northern Arizona University, Flagstaff, Arizona, USA (D.M. Wagner, J. Runberg, A.J. Vogler, J. Lee, P. Keim); Translational Genomics Research Institute, Flagstaff (E. Driebe, L.B. Price, D.M. Engelthaler, P. Keim); and University of Maryland School of Medicine, Baltimore, Maryland, USA (W.F. Fricke, J. Ravel)

DOI: 10.3201/eid1605.090892

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Address for correspondence: David M. Wagner, Center for Microbial Genetics and Genomics, Northern Arizona University, Flagstaff, AZ 86011-4073, USA; email: dave.wagner@nau.edu

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**Triatoma infestans**

*bugs in Southern Patagonia, Argentina*

To the Editor: *Triatoma infestans* bugs, the main vector of Chagas disease, historically occupied a large area from northeastern Brazil to Chubut Province in Patagonia, Argentina (1). Large-scale insecticide spraying during the 1980s and 1990s reduced its geographic range and abundance and interrupted transmission of *Trypanosoma cruzi*, mainly in Uruguay, Chile, and Brazil (2). However, *T. infestans* and transmission of *T. cruzi* persist in the Gran Chaco, a large ecoregion in Argentina, Bolivia, and Paraguay (3).

Chubut Province has historically been an area with no risk for vector-mediated transmission of *T. cruzi*, only its extreme northern region was categorized as having a low transmission risk (4,5). However, increased immigration from disease-endemic rural areas in Argentina and Bolivia into Chubut has raised concerns about accidental introduction of *T. infestans* in travelers’ luggage (1) and establishment of a transmission cycle.

In January 2007, we conducted a province-wide survey of 21 villages in Chubut Province previously infested with *T. infestans* bugs by using 0.2% tetramethrin as a dislodgent agent (1 person-hour/house); no *T. infestans* bugs were detected (online Appendix Figure, www.cdc.gov/EID/content/16/4/887-appF.htm). Only *T. patagonica* bugs were found in 11% of peridomestic structures, and none were infected with *T. cruzi*. In June 2007, a *T. infestans*-like bug was found in a primary healthcare center in Comodoro Rivadavia (45°51’S, 67°28’W), a city in southern Chubut Province (online Appendix Figure). Healthcare center staff reported visits by immigrants from Bolivia a few days before this finding and suspected them to be the source. The bug was identi-