extraction. DNA was extracted by using the QIAamp DNA Stool Mini Kit (QIAGEN, Hilden, Germany) according to the manufacturer's instructions.

Species-specific primers (online Technical Appendix Table, http://wwwnc.cdc.gov/EID/article/21/1/14-0136-Techapp1.pdf) were designed to amplify short sequences of mitochondrial DNA. On the basis of primer specificity and amplicon size, we determined host and parasite species (online Technical Appendix Figure 1). DNA extraction and PCR were performed in a laboratory dedicated to environmental samples (for a complete description of methods, see online Technical Appendix).

DNA was successfully extracted and amplified from 119 (86.9%) of 137 fecal samples. Of usable samples, 28 (23.5%) were from red foxes and 91 (76.5%) were from dogs. Two fox fecal samples (7.1%; 95% binomial CIs 0.9%–23.5%) were infected with *E. multilocularis* tapeworms; none of the dog samples were infected.

To verify parasite identification, we amplified DNA from the 2 *E. multilocularis*–positive samples with *E. multilocularis*–specific primers and sequenced the amplification products. To verify host species identification, we used primers that produced longer amplification products (327 and 197 bp) than the corresponding PCR primers and sequenced amplification products from 5 fox samples and 5 dog samples. Sequencing procedures were performed according to the methods of Saarma et al. (*10*).

Sequences from both *E. multilocularis*–positive samples showed 100% identity with an *E. multilocularis* tapeworm sequence (GenBank accession no. AB018440) (online Technical Appendix Figure 2). All sequenced fox and dog samples also belonged to the corresponding species.

To estimate the sensitivity of this noninvasive genetic method, we determined the number of *E. multilocularis* eggs necessary to obtain a positive PCR result (online Technical Appendix Figure 3). One egg was sufficient to give an *E. multilocularis* tapeworm–specific result.

In summary, we developed a noninvasive genetic method that identifies *E. multilocularis* tapeworms and their host species in carnivore fecal samples found in urban environments. Furthermore, these tapeworms can even be detected in fecal samples from red foxes when only 1 parasite egg is present. Thus, this method is highly sensitive and discriminatory and can be used with degraded fecal samples to monitor *E. multilocularis* tapeworms and their hosts.

This study was supported by grants from the Estonian Research Council (IUT-2032, ESF-8793, and ESF-8525), the European Union through the European Regional Development Fund (Centre of Excellence in Frontiers of Biodiversity Research), and the Estonian Doctoral School of Ecology and Environmental Sciences.

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Severe Delayed Hemolysis Associated with Regulated Parenteral Antimalarial Drug

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DOI: http://dx.doi.org/10.3201/eid2101.140147

To the Editor: Intravenous artesunate is recommended by the World Health Organization as first-line treatment for severe malaria. While artemisinin-based therapies are well tolerated, recent studies have reported cases of severe delayed hemolysis after artesunate treatment for malaria (*1*–*5*). To date, all reported cases have been associated with the use of artesunate not produced under Good Manufacturing Practice (GMP) standards. The United States and Canada are the only countries that use GMP artesunate, and a recent review concluded that delayed hemolysis may be related to differences in GMP versus non-GMP artemisinins (*5*). We report a case of severe delayed hemolysis after administration of GMP artesunate to treat a patient with severe malaria.

During October 2012, A previously healthy, 31-year-old Canadian-born man sought treatment at a hospital in Toronto, Ontario, Canada, after 3 days of fever and severe headaches. He had returned 10 days earlier from a 10-day work trip to South Sudan. He did not use malaria chemoprophylaxis while there but did sleep under an insecticide-treated net. During initial assessment, a blood smear showed *Plasmodium falciparum* malaria with parasitemia of 22% (1,100,000 parasites/mL) and the following levels: bilirubin, 88 (reference range 5–17) μmol/L; aminotransferase, 105 (reference range 10–38) U/L; creatinine, 130 (reference range 80–115) μmol/L; hemoglobin, 144 (reference range 140–160) g/L, and a platelet count of 17×10^9/L (reference range 150–450 ´ 10^9/L). In the emergency department, he was given 1 dose each of doxycycline, atovaquone/proguanil, and artemether/lumefantrine; within an hour of ingestion of these drugs, he vomited. He was transferred to a tertiary level hospital for admission to the intensive care unit and exchange transfusion. Intravenous artesunate was administered (2.4 mg/kg at 0, 12, 24, and 48 h), then a 3-day course of oral atovaquone/proguanil was ordered. On admission, his chest radiograph showed no abnormalities, and blood cultures were negative; his hemoglobin level was 125 g/L; no treatment was initiated for decreased hemoglobin. Parasitemia was undetectable within 36 hours of admission to the intensive care unit. The patient was discharged 5 days later.

Four days after discharge, the patient returned to the tertiary level hospital seeking treatment. He reported that beginning 2 days after discharge, he had fever and "merlot-colored" urine. On admission, he was noted to be jaundiced. Laboratory values included levels of bilirubin of 89 μmol/L, lactate dehydrogenase (LDH) of 1,976 (reference 120–240) U/L, hemoglobin of 81 g/L and marked hemoglobinuria. Multiple thick and thin blood smears were negative for *Plasmodium* spp.

During the course of his second admission, he required 8 blood transfusions to maintain his hemoglobin level above 75 g/L. He continued to have unexplained hemolysis and hemoglobinuria: laboratory results showed a nadir of hemoglobin at 68 g/L and an LDH peak of 3,429 U/L and a low haptoglobin level (<0.12 g/L [reference 0.3–2.0 g/L]). His glucose-6-phosphate dehydrogenase level was within reference range. Supportive therapy was continued, and hemolysis ceased spontaneously 10 days after onset. When seen during a follow-up visit 6 weeks later, he was asymptomatic and his hemoglobin level was 135 g/L. Pre- and post-transfusion and follow-up testing did not show evidence of red blood cell alloantibodies, making the possibility of a delayed hemolytic transfusion reaction unlikely. Serologic tests showed that the he was also positive for causative organisms for schistosomiasis, strongyloidiasis, and Q fever. These diagnoses were consistent with past infections and were not considered to be contributory to the current severe hemolytic event.

In all previous case reports of delayed hemolysis, patients received World Health Organization–prequalified, but not GMP-certified, artesunate (*1*–*5*). In this report, the parenteral drug used was GMP certified and produced by the US Army Medical Materiel Development Activity. A diagnosis of artesunate-associated hemolysis was made in this case based on the temporal relationship with therapy and the absence of other identified causes of intravascular hemolysis. His time course of hemolysis after treatment corresponds with recent case series in Europe (*1*–*4*): his hemoglobin level reached a nadir at approximately day 15. The outcome of this case corresponds with a proposed case definition by Rolling and colleagues to distinguish artesunate-related hemolysis from that attributable to malaria infection alone (*4*).

We suggest a case definition whereby a decrease in hemoglobin combined with an increase in LDH between week 2 and 3 is characteristic of delayed hemolysis associated with artesunate. Because treatment for severe malaria is not given as monotherapy, we cannot exclude a potential contributory role of the other antimalarial agents he received. However, severe intravascular hemolysis has rarely been reported in relationship to these agents. Additionally, we cannot exclude a potential role for drug-induced immune hemolysis. Nonetheless, given the severity of the hemolysis and the delayed onset, health care workers should be cognizant of this late, potentially life-threatening complication of artemisinin-based therapy. All patients treated with artesunate for severe malaria should be monitored for 4 weeks and evaluated for hemolytic anemia.

This work was supported in part by the Canadian Institutes of Health Research (CIHR) MOP-115160, 136813, and 13721 (K.C.K.), and a Canada Research Chair in Molecular Parasitology (K.C.K.).

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Burkholderia pseudomallei Sequence Type 562 in China and Australia

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DOI: http://dx.doi.org/10.3201/eid2101.140156

To the Editor: Melioidosis is increasingly being recognized in tropical and subtropical areas worldwide; the world's 2 major endemic foci are Thailand and northern Australia (1,2). Phylogenetic analyses of Burkholderia pseudomallei isolates, performed by using multilocus sequence typing (MLST) (3), have led to phylogeographic associations that can be used to track melioidosis epidemics (4). However, in contrast to the previous separation of B. pseudomallei into 2 phylogenetic groups (Australia and Southeast Asia/rest of the world) (5), we report an MLST sequence type (ST) that seems to be present in northern Australia, Taiwan, and southern China.

In mainland China, melioidosis was first reported in 1990 (*6*) and is now known to be endemic to several tropical provinces, including Hainan, a southern island province close to Southeast Asia. Since 2008, cases of melioidosis in Hainan have escalated; from July 2008 through July 2012, a total of 110 cases were microbiologically diagnosed at 2 general hospitals (Sanya People's Hospital and Haikou Municipal Hospital).

We characterized clinical isolates of *B. pseudomallei* from the 110 cases by using MLST, pulsed-field gel electrophoresis (PFGE), and 4-locus multilocus variable-number tandem-repeat analysis (MLVA-4) (*3*,*7*,*8*). MLST revealed 40 STs, 39 of which were consistent with STs from Southeast Asia, as evident from the global *B. pseudomallei* MLST database (http://bpseudomallei.mlst.net/). A single ST, ST562, which accounted for 3 cases in Hainan, was previously described on the global database as being from Australia; the 20 isolates from humans and 10 isolates from the environment deposited until September 1, 2014, all from Australia, had been isolated from 2005 through 2012. Although not deposited in the global MLST database, ST562 has also recently been reported from Taiwan (*7*). Among the 253 isolates of *B. pseudomallei* collected in Taiwan during 2004–2010, 1 clinical isolate and 9 environmental isolates were described as being ST562. Moreover, these 10 ST562 isolates displayed a unique PFGE pulsotype, distinct from that of other *B. pseudomallei* strains from Taiwan (*7*).

Of the 3 patients from Hainan from whom ST562 strains were isolated, 2 resided in the city of Sanya and 1 in the neighboring city of Lingshui (online Technical Appendix, http://wwwnc.cdc.gov/EID/article/21/1/14-0156-Techapp1.pdf); all denied a history of foreign travel, they shared no common risk factors, and all survived the infection. Further analysis of ST562, performed by using eBURST-based (http://eburst.mlst.net/) population analysis of the MLST dataset, showed that ST562 is a single-locus variant of ST167, which is represented on the MLST dataset by multiple human and environmental isolates from Thailand and to date by 1 human isolate from Cambodia. ST167 accounted for 1 of the 110 *B. pseudomallei* strains from Hainan. The *narK* locus of ST167contains allele 3 instead of allele 29, as seen in ST562; 3 base differences are found in allele 3: C72T (C→T position 72), C126T, and A435G. According to PFGE, the 3 ST562