coln Marsh (38/81, 46%). The number of latrines per backyard ranged from 1 to 6 ($\chi = 2.15$). *B. procyonis* eggs were found at 14/61 latrines sampled (23%; 95% CI 12%–34%), and no significant difference in prevalence was found between the Ned Brown (6/23, 26%; 95% CI 8%–44%) and Lincoln Marsh areas (8/38, 21%; 95% CI 8%–34%).

Evaluation of the main effect model identified a decreasing probability of latrine occurrence with increasing distance from the nearest forested area and the presence of an outdoor pet, although these relationships were only marginally significant ($p = 0.07$ and 0.08, respectively). No other variables were closely associated with the presence of raccoon latrines ($p>0.20$). When evaluated alone, distance from the forest preserve was significantly related to latrine occurrence ($p = 0.03$); probability decreased with increasing distance. Evaluation of the simplified model identified a weakly positive association with the presence of a food source ($p = 0.09$) and no association with the presence of latrine substrate ($p = 0.35$). Although the findings were not statistically significant, raccoon latrines did appear to be associated with the availability of a food source such as bird feed (odds ratio [OR] 1.9, 95% CI 0.9–4.1); the presence of an outdoor pet (OR 0.27, 95% CI 0.06–1.2) and increasing distance from the nearest forested area reduced the likelihood of latrines. No other variables were associated with the presence of raccoon latrines; however, low statistical power may have precluded adequate assessment.

Our results suggest that when humans live close to protected forests or natural areas, they are more likely to attract raccoons into their yards. In addition, anthropogenic food sources such as pet food, garbage, and bird feed may increase the likelihood that a raccoon will create a latrine, and the presence of outdoor pets appears to be a deterrent. In areas of high raccoon density, these attractants should be removed. Homeowners with small children should remove latrines as quickly as they are discovered (*2*). The risk of children acquiring potentially fatal baylisascariasis can be reduced if parents understand how to reduce the likelihood that children will come into contact with raccoon latrines.

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References

1. Kazacos KR. Protecting children from helminthic zoonoses. Contemp Pediatr. 2000;17:1–24.
2. Kazacos KR. *Baylisascaris procyonis* and related species. In: Samuel WM, Pybus MJ, Kocan AA, editors. Parasitic diseases of wild mammals. Ames (IA): Iowa State University Press; 2001.
3. Sorvillo F, Ash LR, Berlin OGW, Yatabe J, Degiorgio C, Morse SA. *Baylisascaris procyonis:* an emerging helminthic zoonosis. Emerg Infect Dis. 2002;8:355–9.
4. Pai PJ, Blackburn BG, Kazacos KR, Warrier RP, Begue RE. Full recovery from *Baylisascaris procyonis* eosinophilic meningitis. Emerg Infect Dis. 2007;13: 928–30.
5. Gavin PJ, Kazacos KR, Shulman ST. Baylisascariasis. Clin Microbiol Rev. 2005;18:703–18. DOI: 10.1128/CMR.18.4.703-718.2005
6. Page LK, Swihart RK, Kazacos KR. Raccoon latrine structure and its potential role in transmission of *Baylisascaris procyonis* to vertebrates. Am Midl Nat. 1998;140:180–5. DOI: 10.1674/0003-0031(1998)140[0180:RLSAIP]2.0.CO;2
7. Sloss MW, Kemp RL, Zajac AM. Veterinary clinical parasitology. Ames (IA): Iowa State University Press; 1994.
8. Page LK, Swihart RK, Kazacos KR. Implications of raccoon latrines in the epizootiology of baylisascariasis. J Wildl Dis. 1999;35:474–80.

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Reemergence of Strongyloidiasis, Northern Italy

To the Editor: Strongyloidiasis is a helminth infection caused by *Strongyloides stercoralis,* a nematode ubiquitous in tropical and subtropical countries and occasionally reported in temperate countries, including Italy (*1*). Sources of infection are filariform strongyloid larvae present in soil contaminated by infected feces; the larvae penetrate through the skin of a human host. After the first life cycle, a process of autoinfection begins, which persists indefinitely in the host if the infection is not effectively treated. The infection can remain totally asymptomatic for many years or forever or cause cutaneous (itching and rash), abdominal (epigastric pain, pseudoappendicitis, diarrhea), respiratory (cough, recurrent asthma), and systemic (weight loss, cachexia) symptoms that can be enervating. More importantly, when host immunity is impaired because of

a concurrent disease or immunosuppressive therapy (including corticosteroids, sometimes used to treat symptoms of the unrecognized infection or the concurrent eosinophilia), disseminated strongyloidiasis may occur (*2*–*4*), causing a massive and almost invariably fatal invasion of virtually all organs and tissues by filariform larvae and even adult worms (Figure), often combined with bacterial superinfection. This complication is believed to be rare but is probably underestimated because of the extreme variability of the clinical presentation.

Although strongyloidiasis can be suspected in the presence of symptoms or eosinophilia (which is frequent but not mandatory), the low sensitivity of direct diagnostic methods often lets the disease go unrecognized (*5*–*7*). By far the most sensitive diagnostic tools are serologic tests: sensitivity and specificity of indirect fluorescent antibody test (IFAT) (in-house produced IFAT) are 97.4% and 97.9%, respectively, at a dilution ≥1/20, and 70.5% and 99.8% at a dilution ≥1/80 (*6*). A suspected case is defined by a positive antibody titer ≥20 (IFAT); a case is confirmed by a positive direct test result (culture in agar being the most sensitive direct technique) or by a positive antibody titer ≥80 (*6*). Despite some anecdotal reports on the presence of strongyloidiasis in Italy (*1*,*6*), reliable information about the real prevalence of the infection is lacking. After seeing several patients affected by the disease, 1 of whom died because of dissemination (Z. Bisoffi, unpub. data), we decided to carry out a preliminary rapid assessment of the extent of the problem in elderly patients with eosinophilia.

During a 4-month period, from February through May 2008, every patient born in 1940 or earlier who came to the clinical laboratories of 2 contiguous health districts in northern Italy (Mantova, Lombardy Region, and Legnago, Veneto Region) for a diagnostic blood test (hematocrit and leukocyte count/formula) for whatever reason and having a eosinophil count >500 cells/μL was asked to join the study. This study was the pilot phase of a larger, multicentered study, which obtained formal approval from the Ethical Committee of Sacro Cuore Hospital of Negrar, Verona. Informed consent was required of each patient. Of the 132 patients eligible for inclusion (mean age 76.4 years, range 68–90 years, male:female ratio 1.6), none refused to give informed consent. Serum specimens were subjected to the IFAT for *S. stercoralis* at the Sacro Cuore Hospital Centre for Tropical Diseases.

Unexpectedly, we found that 37 (28%) of 132 patients were positive, with titers ranging between 20 and ≥320 (and ≥80 in most cases). However, caution should be exercised in interpreting the results because the patients may not be representative of the general population. Moreover, our results are based on an indirect (although highly sensitive and specific) test. Because the reported cases involve only a few patients every year (of whom some are anecdotally reported as dying from the infection, usually unpublished), we suspect that most strongyloidiasis cases remain undetected.

If relevant transmission still exists in the area, it is unknown but is unlikely because of the improvement of hygienic conditions in the past 5 decades. Reports of the infection in children or young adults with no travel history outside Italy are lacking. Strongyloidiasis in the elderly is therefore most likely to result from an infection that occurred much earlier in life, either in infancy or at a young age, while walking or working barefoot in agricultural fields. The long persistence is the consequence of the autoinfection cycle typical of this parasite as described above. The result is an important and unrecognized public health problem affecting the geriatric population of northern Italy. These preliminary results confirm the need for the already planned, multicentered study involving a larger sample and a wider geographic area.

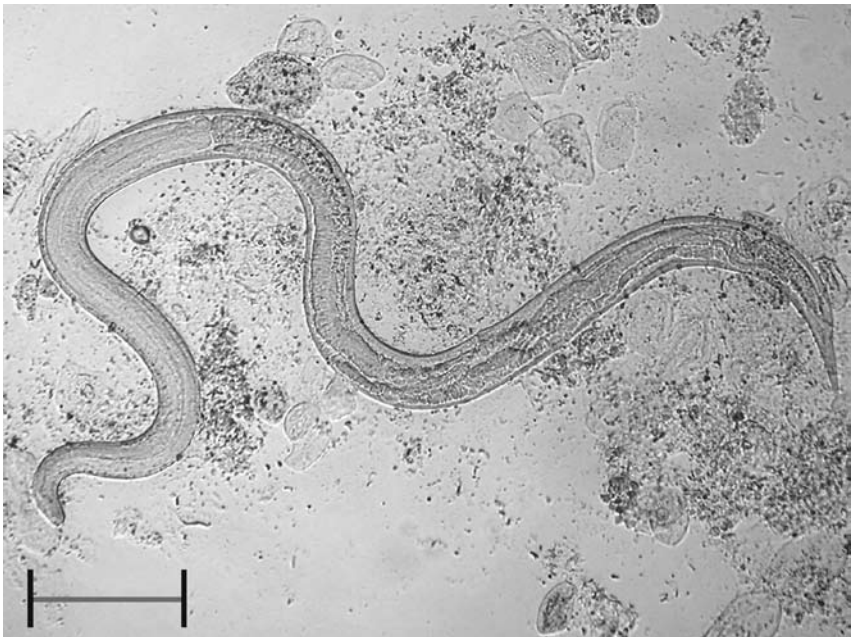

Figure. Adult female of *Strongyloides stercoralis* collected in bronchial fluid of a patient with disseminated disease. Scale bar = 400 μm. A color version of this figure is available online (www.cdc.gov/EID/content/15/9/1531-F.htm).

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References

1. Pirisi M, Salvador E, Bisoffi Z, Gobbo M, Smirne C, Gigli C, et al. Unsuspected strongyloidiasis in hospitalised elderly patients with and without eosinophilia. Clin Microbiol Infect. 2006;12:787–92.
2. Scowden EB, Schaffner W, Stone WJ. Overwhelming strongyloidiasis: an unappreciated opportunistic infection. Medicine. 1978;57:527–44. DOI: 10.1097/00005792-197811000-00004
3. Fardet L, Genereau T, Poirot JL, Guidet B, Kettaneh A, Cabane J. Severe strongyloidiasis in corticosteroid-treated patients: case series and literature review. J Infect. 2007;54:18–27. DOI: 10.1016/j.jinf.2006.01.016
4. Boscolo M, Bisoffi Z. Dissemination: the fatal risk for a missed diagnosis of *Strongyloides stercoralis* infection. J Infect. 2007;55:284–5. DOI: 10.1016/j.jinf.2007.01.009
5. Siddiqui AA, Berk SL. Diagnosis of *Strongyloides stercoralis* infection. Clin Infect Dis. 2001;33:1040–7. DOI: 10.1086/322707
6. Boscolo M, Gobbo M, Mantovani W, Degani M, Anselmi M, Badona Monteiro G, et al. Evaluation of an indirect immunofluorescence assay for strongyloidiasis as a tool for diagnosis and follow-up. Clin Vaccine Immunol. 2007;14:129–33. DOI: 10.1128/CVI.00278-06
7. Loutfy MR, Wilson M, Keystone JS, Kain KC. Serology and eosinophil count in the diagnosis and management of strongyloidiasis in a non-endemic area. Am J Trop Med Hyg. 2002;66:749–52.

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Salmonella enterica Serovar Typhi with CTX-M β-Lactamase, Germany

To the Editor: Infection with *Salmonella enterica* serovar Typhi, the causative agent of typhoid fever, is an acute systemic illness with a high proportion of illness and deaths, especially in developing countries. In Europe, *S. enterica* ser. Typhi infections occur among travelers returning from disease-endemic areas. After emergence of multidrug-resistant *S. enterica* ser. Typhi strains that confer resistance to chloramphenicol, trimethoprim, and ampicillin, quinolones have become the primary drugs for treatment (*1*). Here we report the isolation of CTX-M–producing *S. enterica* ser. Typhi in Germany.

We isolated *S. enterica* ser. Typhi from blood and feces specimens from a 30-year-old Iraqi woman who was admitted to the hospital in Cologne in August 2008. The patient was febrile, dizzy, and had epigastric pain and headache. The symptoms began 2 weeks earlier, after she had returned from a month-long visit to her relatives in Sulaymaniya, the capital of As Sulaymaniyah Governorate in the northeastern Iraqi Kurdistan region. The interview indicated that the same symptoms had developed in other family members in Iraq. The patient was treated successfully with meropenem (1 g 3×/day) for 2 weeks, and no relapse was observed in a follow-up period of 6 months.

The isolated strain was identified as *S. enterica* ser. Typhi with the VITEK2 system (VITEK2 GN-card; bioMérieux, Brussels, Belgium) and by slide agglutination with *Salmonella* antisera (SIFIN, Berlin, Germany) in accordance with the Kauffmann-White scheme. By using Vi-phage typing according to the International Federation for Enteric Phage Typing (L.R. Ward, pers. comm.), the strain was classified as *S. enterica* ser. Typhi Vi-phage type E9. Antimicrobial drug susceptibilities were determined according to the guidelines of the Clinical Laboratory Standards Institute with the VITEK2 AST-N021 card and Etest (bioMérieux). The extended-spectrum β-lactamase (ESBL) phenotype was confirmed with a combined disk diffusion test (MASTDISCS ID, Mast Diagnostica GmbH, Germany). PCR and sequence analyses were performed with universal primers for the ESBL genes $bla_{\text{CTX-M}}$, bla_{TEM}, and bla_{SHV} as described previously (*2*). Primer CTX-M-F 5′-GTTCGTCTCTTCCAGAATAAGG-3′ and primer CTX-M-R 5’-CAGCACTTTTGCCGTCTAAG-3′ were used for sequencing the entire $bla_{\text{CTX-M}}$ gene. Investigation of the CTX-M environment was performed with primers IS*26*-F (5′-GCCCTGGTAAGCAGAGTTTTTG-3′) and IS26-CTX-R (5′-ACAGCGGCACACTTCCTAAC-3′). The presence of plasmid-mediated quinolone resistance genes (*qnr*) was determined by PCR and sequencing of *qnrB* (*3*), *qnrS* (primer F, 5′-CGGCACCACAACTTTTCAC-3′; primer R, 5′-CAACAATACCCAGTGCTTCG-3′), and *qnrA* (primer F, 5′-ATTTCTCACGCCAGGATTTG-3′; primer R, 5′-CGGCAAAGGTTAGGTCACAG-3′). In addition, the nucleotide sequences of the quinolone resistance-determining regions of the *gyrA*, *gyrB*, *parC,* and *parE* genes were determined as previously described (*4*). Transfer of β-lactam resistance was tested by broth mating assays with a sodium azide–