and Wales from 1993 to 2000, an estimated 2–10 secondary cases would have been prevented if attack rates were 5% and each patient had 4 contacts. The number of index cases needed to be detected to prevent 1 death (assuming 6%–10% case-fatality ratio) would have been 150–180 with attack rates of 5% and 50–83 with attack rates of 30%. Thus, deaths were not likely to have been prevented during this period by screening.

Are the parameter estimates valid? We focused on secondary cases, but spread in outbreaks may be exponential, so the effect of missing cases may be greater once tertiary cases and further spread are taken into account. Vaccination coverage may be higher or lower in different risk groups. Secondary attack rates in the literature are reported from outbreaks and regions with vulnerable populations during periods of high incidence and may not apply in affluent countries with high coverage and may be <5%. Adult protection may be better than indicated by serosurveys and may have improved in the United Kingdom with use since 1994 of combined tetanus-diphtheria toxoid vaccine instead of tetanus toxoid for injuries (5).

Outbreaks are not reported from countries without routine screening (1), which indicates that some of our assumptions and estimates may be incorrect. Alternatively, this fact may indicate defective surveillance; countries that do not detect primary cases may not detect secondary cases.

Surveillance for diphtheria in European Union member states varies widely (1). Only 5 of 19 reporting countries screen throat swabs routinely for corynebacteria, raising doubts about the quality of surveillance. The absence of reports of diphtheria may not reflect the absence of disease or of circulating toxigenic corynebacteria. Our results show the possible consequences of not detecting such infections and help demonstrate the public health priority of diphtheria surveillance.

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Rickettsia slovaca Infection, France

To the Editor: *Rickettsia slovaca* was first isolated in 1968 in a *Dermacentor marginatus* tick collected in Slovakia, and serologic evidence of infection with this bacteria was reported in patients with enlarged lymph nodes and a scalp eschar after being bitten by a tick (1). However, the first proven case of *R. slovaca* infection was reported only in 1997 in France (2). This rickettsiosis is called tickborne lymphadenopathy (TIBOLA) because the most pronounced sign is lymph node enlargement. In Spain the same condition is called *Dermacentor*-borne-necrosis-erythema lymphadenopathy (3,4).

In this study, we describe 14 new patients with TIBOLA from southern France who sought treatment from January 2004 to May 2005 and compare the features of these patients with those in whom Mediterranean spotted fever (MSF) was diagnosed during the same period. All the patients were referred to our center with a suspected rickettsial infection characterized by a tick bite located on the scalp, an inoculation eschar, and enlarged lymph nodes (see online Appendix Figure, available at http://www.cdc.gov/ncidod/EID/vol12no03/05-0911-appG.htm). For each patient, an acute-phase and a convalescent-phase serum sample were obtained for serologic analysis. Culture and polymerase chain reaction (PCR) were performed on tick, skin biopsy, or blood specimens. A multiple-antigen immunofluorescence assay (IFA) was performed by using 5 spotted fever group (SFG) rickettsial antigens: *R. conorii conorii*, *R. slovaca*, *R. helvetica*, *R. sibirica mongolitimonae*, and *R. felis*. Titers of at least 64 for immunoglobulin G (IgG) and 32 for IgM in acute-phase serum samples, evidence of seroconversion with 4-fold increases in IgG titers, or both, were considered as evidence of recent

infections with a *Rickettsia* sp. (*5*). For serum specimens confirmed by IFA at the species level, Western immunoblotting and cross-adsorption assays procedures were performed as described elsewhere (*6*) by using *R. conorii conorii* and *R. slovaca* antigens. Patients with a definite serologic diagnosis at the species level were analyzed for their epidemiologic and clinical information.

Culture from skin biopsy specimen and ticks were injected into human embryonic lung cells and cultivated into shell-vial culture as previously described (*7*). DNA was extracted from skin biopsy specimens, acute-phase serum samples, and ticks by using the QIAamp DNA Mini Kit (Qiagen, Hilden, Germany) (*8*). Standard PCR was performed with primers suitable for hybridization within the conserved region of genes coding for outer membrane protein A (*ompA*) and citrate synthase (*gltA*) (*8*).

Among the 14 patients in a scalp lesion and cervical or occipital (1 case) lymph node enlargement developed after they were bitten by a tick, 9 were female (1 was pregnant) and 5 were male. The median (range) age was 34.9 (5–85) years with half of the patients <10 years of age. The incubation period ranged from 5 to 15 days (median 10.5 days; n = 7). Only 3 patients had fever. All patients fully recovered with doxycycline or, for the pregnant patient, josamycin therapy. Serology confirmed the diagnosis of *R. slovaca* infection for 10 patients by microimmunofluorescence and Western blot analysis after cross-adsorption studies (online Appendix Table, available from http://www.cdc.gov/ncidod/EID/vol12no03/05-0911.htm#apptable). *R. slovaca* was amplified by PCR for 7 cases, including 3 skin biopsy specimens, 3 *Dermacentor marginatus* ticks, and 1 acute-phase serum sample (Appendix Table). Three isolates (2 from skin biopsy specimens and 1 from a tick) were obtained by using the shell-vial

culture assay. During the period of our study, in the same French region, 40 patients with MSF were clinically and laboratory diagnosed using the same procedures. The median (range) age was 54.2 (5–85) years with only 3 children <10 years of age (compared to 7/7 children with *R. slovaca* infection, p = 0.0015). MSF occurred mainly during the summer, whereas *R. slovaca* infection was seen during the colder months with 6 cases from October to January and 8 cases from February to May (Figure).

In France, *R. conorii* has long been considered to be the only SFG rickettsiosis but *R. slovaca* may also be prevalent (*9*), contributing 25% of the cases in the present study. This organism is also a common cause of disease in Hungary and in La Rioja, Spain (*3*). These data suggest that TIBOLA mainly occurs in young children, affects women predominately, and occurs primarily during the colder months (*9,10*). As previously reported (*9*), we found that standard microimmunofluorescence serologic testing was insensitive and that Western blot is more useful and allows identification to the species level after cross-adsorption studies. Finally, DNA amplification by PCR from skin biop-

sy tissue, serum samples, or in ticks allowed confirmation of the diagnosis in only 50% of the cases, which suggests that other rickettsial species may be responsible for TIBOLA. Epidemiologic and clinical presentations are so characteristic that the clinical diagnosis should be considered in patients who have been bitten on the scalp during the colder months. In Europe, *R. slovaca* infection is likely to be a significant cause of cervical lymph node enlargement, and microbiologic investigation and tick analysis will underline the relative importance of this disease.

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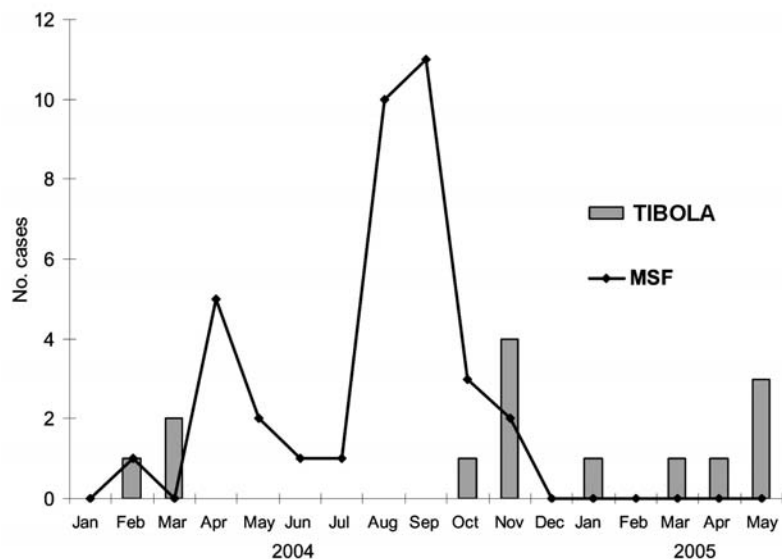

Figure. Seasonal distribution of Mediterranean spotted fever (MSF) and tickborne lymphadenopathy (TIBOLA) in southern France from January 2004 to May 2005.

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Cutaneous Anthrax, Belgian Traveler

To the Editor: Anthrax is a rare zoonotic disease among travelers. The clinical spectrum includes cutaneous lesions, respiratory anthrax, pharyngeal inflammation, gastrointestinal infection, septicemia, and meningitis. Interest in anthrax increased after the bioterrorist attacks in the United States in 2001. The following case history describes a cutaneous infection suspected to be anthrax in a tourist who had indirect contact with dead mammals in a disease-endemic area.

After indirect contact with dead antelopes and a hippopotamus in Botswana, an acute necrotic lesion developed on a finger of a 31-year-old, healthy, female Belgian woman. The lesion became covered with a black crust, followed by massive swelling of the hand and arm. The clinical aspect and history strongly suggested cutaneous anthrax. This diagnosis was supported by seroconversion to protective antigen of *Bacillus anthracis* and the presence of antibodies against lethal factor. The bacterium itself could not be cultured or identified by polymerase chain reaction (PCR). Other members of the group with which she traveled were contacted, but no other cases were reported.

The Belgian woman traveled with friends to Namibia, Botswana, and South Africa from December 12, 2004, until January 22, 2005. She visited Chobe National Park in Botswana early January 2005. On January 8, a small, painless, vesicular lesion developed on the dorsal side of her fourth left finger. This lesion increased in size quickly and developed a black aspect with a red elevated border. Small vesicles appeared in the immediate vicinity of the primary lesion. No pus was noted. Her general

condition was good. She treated herself with amoxicillin-clavulanic acid 2 gm/day for 3 days. The next day, massive edema of the finger, hand, and left arm developed. When admitted to a hospital in Johannesburg, her left arm and hand were massively swollen with painful left axillary lymphadenopathy. Her temperature never exceeded 37.8°C. Wound cultures showed only the presence of viridans streptococci, bacteria that are not implicated in wound infections. The patient was treated with intravenous ciprofloxacin, gentamicin, tetracycline, flucloxacillin, and topical mupirocin. She was discharged after 6 days with oral flucloxacillin and returned to Belgium on January 22. On February 4, her general condition was excellent; the edema had diminished. A painless necrotic lesion on the left fourth finger measured 3 cm^2 (Figure). She mentioned minor discomfort of her left underarm and loss of sensation at the distal radial side of the left underarm. She could not extend the terminal phalanx of the fourth left finger because the underlying tendon had been destroyed. The left axillary lymph nodes were still slightly swollen. No evidence indicated parapox viral infection or necrotic arachnidism. Upon questioning, she mentioned that in Chobe National Park, some fellow travelers had manipulated the legs of dead antelopes. One person had climbed on a dead hippo for a picture and sank into the putrefying carcass. He soon afterwards cleaned a small abrasion on the patient's finger. Some hours later, all group members washed their hands in a common small plastic basin containing water and chloroxylenol.

Full blood count, erythrocyte sedimentation rate, and biochemistry were normal. Antistreptolysin O levels were within normal limits. Serologic test results for rickettsiae, orthopoxviruses, and *Bartonella henselae* were negative. The patient was not