2 episodes of surgical debridement during the first month. The lesions began to improve in the second month of medical therapy, when skin grafting was performed. The patient remained well and was discharged after almost 3 months. Medications were continued for a total period of 6 months. The patient has been well for >2 years.

P. insidiosum in tissues resembles agents of zygomycosis morphologically but, unlike the latter, rarely stains with hematoxylin and eosin. Various immunostainings also help identify the organism (*4*). With the exception of the facial-cranial form of the disease in the United States (*5*), most cases in Thailand occur in patients with chronic hemolytic anemia; thalassemia-hemoglobinopathy is the most common underlying disease. Major clinical manifestations include ocular and craniofacial infections in healthy children, arteritis usually originating from lower extremities, and chronic subcutaneous abscesses.

Treatment options reported to be successful include supersaturated potassium iodide for the chronic cutaneous form (*2*), a combination of terbinafine and itraconazole in a single case of an acute, severe ocular, subcutaneous infection (*6*), and therapeutic vaccination for severe infections involving major arteries (*7*,*8*) in conjunction with surgery. The first case suggested that, with chronic pythiosis involving the aorta, effective management is difficult. In the second case, the laboratory was familiar with *P. insidiosum* isolation procedures; therefore, a quick diagnosis was made and early treatment was instituted. This early form of cutaneous pythiosis is rarely diagnosed properly by most clinical and pathologic laboratories. Human cases probably occur worldwide (*9*) but are underrecognized and thus, misdiagnosed (*5*). More research into the pathogenesis, diagnosis, and new treatment modalities is urgently needed.

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References

1. Sathapatayavongs B, Leelachaikul P, Prachaktam R, Atichartakarn V, Sriphojanart S, Trairatvorakul P, et al. Human pythiosis associated with thalassemia hemoglobinopathy syndrome. J Infect Dis. 1989;159:274–80.
2. Thianprasit M, Chaiprasert A, Imwidthaya P. Human pythiosis. Curr Top Med Mycol. 1996;7:43–54.
3. Mendoza L, Mandy W, Glass R. An improved *Pythium insidiosum*-vaccine formulation with enhanced immunotherapeutic properties in horses and dogs with pythiosis. Vaccine. 2003;21:2797–804.
4. Kaufman L. *Penicilliosis marneffei* and pythiosis: emerging tropical diseases. Mycopathologia. 1998;143:3–7.
5. Mendoza L, Prasla SH, Ajello L. Orbital pythiosis: a non-fungal disease mimicking orbital mycotic infections, with a retrospective review of the literature. Mycoses. 2004;47:14–23.
6. Shenep JL, English BK, Kaufman L, Pearson TA, Thompson JW, Kaufman RA, et al. Successful medical therapy for deeply invasive facial infection due to *Pythium insidiosum* in a child. Clin Infect Dis. 1998;27:1388–93.
7. Thitithanyanont A, Mendoza L, Chuansumrit A, Pracharktam R, Laothamatas J, Sathapatayavongs B, et al. Use of an immunotherapeutic vaccine to treat a lifethreatening human arteritic infection caused by *Pythium insidiosum*. Clin Infect Dis. 1998;27:1394–400.
8. Wanachiwanawin W, Mendoza L, Visuthisakchai 5, Mutsikapan P, Sathapatayavongs B, Chaiprasert A, et al. Efficacy of immunotherapy using antigens of *Pythium insidiosum* in the treatment of vascular pythiosis in humans. Vaccine. 2004;22:3613–21.
9. Bosco Sde M, Bagagli E, Araujo JP Jr, Candeias JM, de Franco ME, Alencar Marques ME, et al. Human pythiosis, Brazil. Emerg Infect Dis. 2005;11:715–8.

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Rift Valley Fever Potential, Arabian Peninsula

To the Editor: Rift Valley fever (RVF) virus causes severe disease, abortion, and death in domestic animals (especially young sheep, cattle, and goats) in Africa and the Arabian Peninsula. Humans are infected by mosquitoes, which maintain epizootic transmission, or through exposure to infected animal tissue. Although human disease may be mild, sometimes severe retinitis, meningoencephalitis, or hemorrhagic fever syndromes may develop in patients. In Africa, epizootics and associated human epidemics usually follow heavy rainfall (*1*).

RVF was first confirmed outside Africa in September 2000. The outbreak in southwestern coastal Saudi Arabia and neighboring coastal areas of Yemen resulted in an epizootic with >120 human deaths and major losses in livestock populations from disease and slaughter (*2*,*3*). RVF virus isolated from the floodwater mosquito *Aedes vexans arabiensis* during the outbreak was closely related to strains from Madagascar (1991) and Kenya (1997), which suggests that the virus was imported through infected mosquitoes or livestock from East Africa (*3*). The Arabian outbreak followed increased rainfall in nearby highlands that flooded the coastal areas and created ideal environments for mosquito populations similar to those found in RVF-endemic regions of East Africa (*4*). Most RVF activity was associated with flooded wadi agricultural systems; no cases were reported in the mountains or in dry sandy regions, where surface water does not accumulate long enough to sustain mosquito breeding.

To provide early warning of conditions favorable for RVF epidemics, the National Aeronautics and Space Administration (NASA) and the Department of Defense Global Emerging Infections Surveillance and Response System (DoD-GEIS) monitor the satellite-derived normalized difference vegetation index (NDVI), which reflects recent rainfall and other ecologic and climatic factors (*5*–*7*). NDVI anomalies in the highlands east of affected areas during the 2000 outbreak (online Appendix Figure panel A, available from http://www.cdc.gov/ncidod/EID/vol2no03/05-0973-G.htm) showed a spatial pattern (although of lower magnitude) similar to recent anomalies in those areas (online Appendix Figure panel B). Greater than normal NDVIs (20%–60%) were seen in the Sarawat Mountains, from just northeast of Djeddah, Saudi Arabia, and southwestward beyond Jizan and into Hodeidah governorate in Yemen during May and June 2005.

Satellite-derived rainfall estimates show that widespread rainfall occurred over most of western Saudi Arabia and Yemen from mid-April to mid-June 2005 (*8*) and accounts for the high magnitude and spatial pattern of observed NDVI anomalies in May and June 2005. Rainfall was concentrated in the mountainous regions east of the Red Sea coast, and was heaviest in the areas east of Djeddah and Jizan, with rainfall totals as high as 120 mm and 60–80 mm, respectively, during April 2005, compared with the same period in 2000 (10–50 mm) (online Appendix Figure panels C and D) and in southwestern Yemen, with totals as high as 120 mm during May. In the area east of Djeddah, total rainfall in April 2005 was 150 mm above the long-term average for that month. Flooding was reported in Hodeidah Governorate, Yemen during May (*9*) and could be expected in other Red Sea coastal areas following such heavy rainfall. This created habitats appropriate for breeding of mosquitoes capable of transmitting RVF, as occurred in 2000.

No human cases of RVF have been reported in Saudi Arabia and Yemen since the 2000 outbreak, but in September 2004 the Saudi Ministry of Agriculture reported that 5 RVF-seropositive sheep had been identified during routine surveillance in Jizan where most infected persons were exposed during the outbreak in 2000 (*2*). The primary infection was estimated to have occurred in April 2004 (*10*). The NDVIs and rainfall patterns alerted the Yemen and Saudi Arabia Ministries of Health and Ministries of Agriculture to conduct field investigations with the Food and Agriculture Organization and the World Health Organization.

Since RVF virus can be maintained in mosquito eggs for extended periods and transmitted under favorable conditions (*6*), the high magnitude of NDVI and rainfall patterns reported should prompt heightened veterinary and human surveillance for RVF in coastal Arabia and mass vaccination of susceptible animals. The current RVF model (*7*) is indicative of conditions that would promote vector breeding and could result in an outbreak of mosquitoborne diseases.

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References

1. Davies FG, Linthicum KJ, James AD. Rainfall and epizootic Rift Valley fever. Bull World Health Organ. 1985;63:941–3.
2. Centers for Disease Control and Prevention. Outbreak of Rift Valley fever—Saudi Arabia, August–October, 2000. MMWR Morb Mortal Wkly Rep. 2000;49:905–8.
3. Miller BR, Godsey MS, Crabtee MB, Savage HM, Al-Mazrao Y, Al-Jeffri MH, et al. Isolation and genetic characterization of Rift Valley fever virus from *Aedes vexans arabiensis*, Kingdom of Saudi Arabia. Emerg Infect Dis. 2002;8:1492–4.
4. Food and Agriculture Organization. Update on RVF outbreaks in Saudi Arabia and Yemen. EMPRES Transboundary Animal Diseases Bulletin. 2000 Jul–Dec [cited 2005 Jul 11]. Available from http://www.fao.org/documents/show_cdr.asp?url_file=/DOCREP/003/X9550E/X9550E03.HTM
5. Linthicum KJ, Anyamba A, Tucker CJ, Kelley PW, Myers MF, Peter CJ. Southern oscillation index, sea surface temperature and satellite vegetation index indicator to forecast Rift Valley fever epizootics/epidemics in Kenya. Science. 1999;285:397–400.

6. Anyamba A, Linthicum KJ, Mahoney R, Tucker CJ, Kelley PW. Mapping potential risk of Rift Valley fever outbreaks in African savannas using vegetation index time series data. Photogrammetric Engineering and Remote Sensing. 2002;68:137–45.
7. Department of Defense Global Emerging Infections Surveillance and Response System. Climate and disease connections: Rift Valley fever monitor; 2000–2005. [cited 2005 Jul 11]. Available from http://www.geis.fhp.osd.mil/GEIS/SurveillanceActivities/RVFWeb/indexRVF.asp
8. International Research Institute for Climate Prediction. Malaria early warning system [cited 2005 Jul 11]. Available from http://iridl.ldeo.columbia.edu/maproom/.Regional/.Africa/.MEWS/
9. ReliefWeb. Yemen: floods – May 2005. [cited 2005 Jul 11]. Available from http://www.reliefweb.int/rw/dbc.nsf/doc108?OpenForm&rc=3&emid=FL-2005-000070-YEM
10. World Organization for Animal Health. Rift Valley fever in Saudi Arabia – serological findings. ProMed. 2004 Oct 3 [cited 2005 Dec 30]. Available from http://www.promedmail.org (archive no. 20041003.2723)

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Screening and Toxigenic Corynebacteria Spread

To the Editor: Diphtheria is rare in countries with high vaccination coverage, but as seen in Europe in recent decades, control can disintegrate rapidly. When diphtheria is rare, surveillance is challenging because clinicians have no experience with the infection, and disease may be mild or atypical in vaccinated persons (*1*). Clinicians may give inadequate information to laboratories, and appropriate investigations may not be performed. Identifying cases is facilitated if all throat swabs from patients with pharyngitis are screened by laboratories for corynebacteria, but this procedure is expensive and time consuming. To help balance priorities in diphtheria surveillance, we evaluated the potential benefits of microbiologic screening in preventing secondary spread of toxigenic corynebacteria in England and Wales and estimated the possible consequences of not detecting a case.

The mean number of secondary cases that might occur per index case if screening is not undertaken depends on the mean number of contacts and attack rates, vaccine coverage and efficacy, and duration of protection. Some of these factors are not known precisely, so we estimated them within plausible ranges of values. We varied the number of contacts per case-patient from 2 to 20. Secondary attack rates in susceptible persons are difficult to estimate and distinguish from carriage rates (*2*), and we varied these from 5% to 50%. Vaccine efficacy in children was varied from 50% to 95%. We estimated the susceptibility of UK adults at 40% (*3*), vaccination coverage in children at 95% (*4*), and case-fatality ratio at 6% to 10% (*5*). For simplicity, the ratio of adults to children among contacts was assumed to be 1:1. We assumed that without specific microbiologic identification of cases, no intervention would take place and that intervention to protect contacts is 100% effective. Such intervention includes early treatment and isolation of cases, chemoprophylaxis, and booster vaccination of contacts. The number of cases that need to be detected to prevent 1 secondary case for different numbers of contacts and attack rates was calculated as the inverse of the number of secondary cases that would result from each case not detected by screening.

The number of cases that must be detected by microbiologic screening to prevent 1 secondary case was most affected by varying the number of contacts per patient and the secondary attack rate (Figure). If one assumes vaccine efficacy of 95%, an attack rate in susceptible contacts of 5%, and 4 contacts per patient, 1 secondary case is prevented for every 18 cases detected; if attack rates are 30%, then 1 secondary case is prevented for <5 index cases detected. If vaccine efficacy was 50%, the number of cases that would need to be detected to prevent 1 secondary case would fall from 18 to <10 cases for a mean of 4 contacts per case and secondary attack rates of 5%.

For the 53 toxigenic strains of corynebacteria detected in England

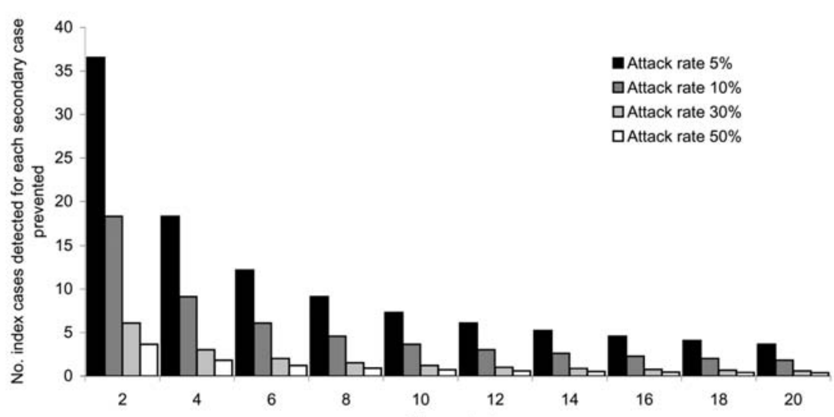

Figure. Number of cases needed to detect to prevent 1 secondary case.