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Coccidioidomycosis Outbreak

ABSTRACT & COMMENTARY

Synopsis: *Eighteen of 19 physicians failed to diagnose coccidioidomycosis in members of a church group from Washington returning from Mexico.*

Source: Cairns L, et al. Outbreak of coccidioidomycosis in Washington State residents returning from Mexico. *Clin Infect Dis* 2000;30:61-64.

A cluster of flulike illnesses in a church group was reported to the Washington State Department of Health in July 1996. The 126-member group had recently returned from Tecate, Mexico, a town in the Sonoran Desert adjacent to the United States-Mexico border, where members had stayed in an orphanage for six days and assisted in construction projects. One person was eventually diagnosed with coccidioidomycosis. As a result, a retrospective cohort study was conducted to assess the extent of the outbreak and risk factors for acquisition of this disease.

Investigators used a questionnaire to collect demographic data, travel and medical histories, information on activities while in Tecate, and symptoms of illness since the trip. Spherulin skin tests were done on all consenting group members. Serum specimens were collected from persons with any flulike symptoms or those with a positive skin test. These specimens were tested for antibodies to *Coccidioides immitis* by 1) quantitative complement fixation (CF) tests; 2) immunodiffusion tube precipitin (IDTP) for IgM antibody; and 3) immunodiffusion complement fixation (IDCF) for IgG antibody. A case of acute coccidioidomycosis was defined by a positive serological test for *C. immitis*. In addition, soil specimens were collected from various sites at the orphanage in Tecate and inoculated into mice for growth of *C. immitis*.

Of the 126 members, 59 (47%) completed questionnaires and underwent skin tests. Thirty-five (59%) were female and 51 (86%) were 14-18 years of age. None reported being immunocompromised or pregnant. Twenty-seven (46%) members had a positive skin test. Forty members underwent serological testing. Twenty-one of these members met the case definition of coccid-

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ioidomycosis with positive serology. The majority of cases were adolescents (95%) and female (86%). The attack rate for the 59 members who responded to the questionnaire was 36%, and the minimum attack rate for the entire group was 17%. A total of 95% of the cases were symptomatic, with an average incubation period of 12 days (range, 7-20 days). The symptoms included fever (85%), headache (81%), chest pain (76%), body aches (71%), cough (66%), fatigue (66%), rash (62%), muscle pain (52%), nausea (43%), and joint pain (33%). Four patients reported lesions consistent with erythema nodosum.

A total of 16 symptomatic patients saw 19 health care providers, who appeared to be aware of the patients' travel history. The patients were diagnosed with bacterial bronchitis, contact dermatitis, and viral infection. Only an infectious disease specialist trained in California made the diagnosis of coccidioidomycosis after seeing a patient with erythema nodosum.

Analysis of reported activities found that digging a swimming pool was associated with an increased risk of acute coccidioidomycosis. Furthermore, soil inoculation studies yielded illness in mice as well as lesions that contained spherules characteristic of *C. immitis*.

COMMENT BY LIN H. CHEN, MD

Coccidioidomycosis is caused by *C. immitis*, a dimorphic fungus. The mycelial form is found in soil, which produces arthroconidia that become airborne. When inhaled by a host, the arthroconidia develop into spherules and cause infection.[1,2]

C. immitis is endemic in the southwestern United States—primarily California, Arizona, Texas, and New Mexico.[3] *C. immitis* is also highly endemic in parts of Mexico, including Sonora, Chihuahua, Coahuila, Sinaloa, Nayarit, Jalisco, Colima, Coshuila, Nuevo Leon, Durango, San Luis Potosi, and Guanajuato. Venezuela has endemic areas in the northwestern states of Falcon, Lara, and Zulia. Other endemic areas include Comayagua Valley in Honduras, the Motagua River Valley in Guatemala, and Patagonia and Rio Hondo in Argentina. Cases have also been reported in Bolivia, Paraguay, and Colombia.[4]

Sixty percent of those infected with *C. immitis* are asymptomatic or have symptoms resembling an upper respiratory infection.[1,5] They are only diagnosed because of positive coccidioidal skin tests. The other 40% develop a pulmonary infection after an incubation period of 1-3 weeks. The clinical presentation may include cough, sputum production, fever, chills, anorexia, weakness, arthralgias, chest pain, and rashes such as erythema nodosum or erythema multiforme. Chest radiography may show infiltrates, pleural effusions, and hilar adenopathy. The acute infection usually resolves without specific treatment, although the symptoms may last several weeks. Five percent of those infected may have remaining pulmonary nodule or cavitary lesions, and a progressive pneumonia or chronic lung infection can occasionally develop. Disseminated infections may develop in 0.5-1% of infected people, more commonly in immunocompromised hosts, pregnant women, diabetics, and nonwhites.[3,7] The usual sites involved in disseminated infections are bone and joints, meninges, skin, and soft tissues.

A diagnosis of coccidioidomycosis can be made by culture, staining of tissue specimen, skin testing to coccidioidal antigens, and serology. The latter include the tube precipitin test for IgM, immunodiffusion test (for IgM and IgG), enzyme immunoassay (for IgM and IgG), and complement fixation test for IgG. Although most acute infections resolve spontaneously, progressive primary disease or disseminated disease

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requires treatment with amphotericin B and/or itraconazole or fluconazole.[8]

The study by Cairns and associates made some interesting observations. First of all, the majority (85%) of patients reported a rash, which was more frequent when compared to the incidence of rash (20%) in other reports.[6] Cairns et al suggested that children and young adults possibly have a different clinical presentation during acute coccidioidomycosis. Second, the incidence of headache was also high (81%), yet the headaches did not appear to indicate meningitis. Next, excavation was a definite risk factor, similar to the outbreaks associated with archaeological digging.[8] Finally, the study demonstrated the difficulty in recognizing and diagnosing coccidioidomycosis in nonendemic areas, emphasizing the need to improve health care providers' awareness of the epidemiology and presentations of coccidioidomycosis. *C. immitis* is clearly endemic in areas of Central and South America as well as the southwestern United States. Physicians and travelers to these areas should understand the specific epidemiology, risk groups, and manifestations of coccidioidomycosis. Immunocompromised individuals should especially be aware of this disease. (*Dr. Chen is Clinical Instructor, Harvard Medical School and Travel/Tropical Medicine Clinic, Lahey Clinic Medical Center.*) ❖

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Typhoid Fever: Vaccination Failures

ABSTRACT & COMMENTARY

Synopsis: *Three commercial typhoid fever vaccines are available, yet data on efficacy in travelers have been lacking. The occurrence of typhoid fever in a group of travelers to Indonesia who had been vaccinated against typhoid fever suggests incomplete efficacy.*

Source: Cobelens FG, et al. Typhoid fever in group travelers: Opportunity for studying vaccine efficacy. *J Travel Med* 2000; 7:19-24.

In 1994, 91 cases of typhoid fever were reported to the Inspectorate of Healthcare of the Netherlands Ministry of Health. Eight patients had recently traveled to Indonesia, linked to package tours conducted by the same tour operator. One hundred ten travelers participated in a questionnaire-based study designed to describe demographics, history of typhoid fever vaccine use, pretravel medical conditions, medications, diagnosed or suspected typhoid fever, and details of their journey, including meals taken.

Six patients were defined as typhoid fever cases in which *Salmonella typhi* was isolated from one or more blood cultures. Fifteen others reported an illness compatible with typhoid fever but did not have positive blood cultures. Many were diagnosed with diarrheal illness or received empiric treatment with ciprofloxacin. Vaccination status was ascertained by asking the respondents to indicate the most recent entry for typhoid fever in their International Certificate for Yellow Fever vaccination or Military Passport of the Netherlands Armed Forces. Vaccination status was considered "documented" if either written proof was available or if the provider had confirmed the vaccination from records. Seven respondents were excluded because of reduced gastric acidity or use of an antibiotic. Of the remaining 103 respondents, 96 (93.2%) claimed to have been vaccinated against typhoid fever within the years preceding the journey. Immunization status was well documented for 85 (82.5%); however, 11 respondents who claimed to have received the oral typhoid vaccine did not have written documentation.

There were no differences in age, sex, or travel group. All cases of typhoid fever occurred in documented recipients of the oral Ty 21a vaccine with an attack rate of 8.6% (95% CI 3.2-17.7%) or 10.2% (95% CI 3.8-20.8%) if restricted to respondents with documented vaccination status.

COMMENT BY MOLLY STENZEL, MD, AND MARIA D. MILENO, MD

This important publication clearly indicates that the risk of typhoid fever can be considerable, even for an immunized population of travelers. Other additional cases may well have been masked by antibiotic prophylaxis or by antibiotic treatment of diarrhea. The following case history illustrates the degree of morbidity that may accompany typhoid fever.

A 20-year-old, previously healthy, male college student from Rhode Island returned from a one-month stay in West Africa. He had traveled to Argentina 10 months prior to admission, to Senegal and the Ivory Coast five years ago, and had lived in Africa for several years as a young child. While recently abroad in Mauritania and Senegal he developed watery diarrhea without noting any blood in his stool, fevers, chills, or abdominal pain. He took several doses of loperamide, but he did not self-administer either ciprofloxacin or other antibiotics, and his diarrhea resolved within three days, and just prior to his return to the United States.

Four days prior to admission, he experienced headache, malaise, and generalized fatigue. The following evening he noted fever and rigors every 4-6 hours. Diarrhea symptoms returned, consisting of one or two watery, nonbloody stools per day with mild abdominal cramping. He denied photophobia, neck stiffness, or change in mentation, but stated that he felt "kind of slow," particularly when febrile. On the day of admission he experienced nausea and vomited once.

During his trip to West Africa, he first stayed in the coastal city of Nouakchott, then traveled along the southwest border of Mauritania. He resided in river villages and spent several days in the desert of Senegal. When traveling in rural areas, he stayed with families in tent homes or grass huts. He sustained mosquito bites, despite sleeping under mosquito netting. He was not aware of being bitten by flies or ticks. He drank primarily bottled or filtered water or hot tea, but did occasionally drink fruit juice or milk diluted with water obtained from uncertain sources. He ate well-cooked fish or meat, often from a communal bowl using his hands, without utensils as dictated by local custom. He sampled a drink consisting of sweetened camel's milk. He had no sexual contact during his trip and received no blood transfusions.

The patient had received all of his routine childhood immunizations and yellow fever vaccines in 1995. He had been immunized with BCG vaccine as a small child. His most recent PPD test, during August 1999, was negative. He received a meningococcal vaccine just prior to his trip, but did not receive typhoid fever vaccine. However, he did obtain and use mefloquine prophylaxis before and during his stay in Africa.

Physical examination revealed a thin, flushed young man with a temperature of 39.4°C, a pulse of 96/min, and a respiratory rate of 16. There were no skin lesions. Aside from mild epigastric tenderness, his physical findings were otherwise normal.

Results of admission laboratory studies showed a white blood cell count of 5.9; 47% segmented neutrophils; 32% band forms; 13% lymphocytes; 8% monocytes; hemoglobin 14.6 g/dL with normal red cell morphology; platelets 148,000, creatinine 0.9 mg/dL; AST 64 IU/L (normal range 10-42): ALT, alkaline phosphatase, and total bilirubin were within normal limits. Urinalysis was normal except for mild proteinuria. Cerebrospinal fluid analysis revealed no abnormalities and the chest radiograph was normal.

Blood smears revealed no malaria parasites. Two sets of blood cultures drawn on admission, and another set drawn on the first hospital day, grew *Salmonella typhi*. The blood isolate was sensitive to all antibiotics tested, including trimethoprim/sulfamethoxazole, ampicillin, ceftriaxone, and ciprofloxacin. Stool examination revealed many cysts and trophozoites of *Giardia lamblia*.

Treatment with ciprofloxacin and metronidazole was initiated. Over the subsequent hospital days, the diarrhea resolved and he finally defervesced by day 8. A transient rise in hepatic transaminases (peak AST 488 IU/L) occurred on hospital day 6. Two weeks later he continued to have abdominal tenderness on palpation of the right upper quadrant, but was otherwise doing well. Follow-up stool cultures and examination were negative. This young man was relatively fortunate in that he did not experience an episode of severe gastrointestinal bleeding, intestinal perforation, bacterial peritonitis, or pneumonia. A review of the recent literature revealed no cases of typhoid fever reported from Mauritania. Surveillance of group travelers may reveal typhoid activity in regions where reporting is not adequate. (*Dr. Mileno is Director, Travel Medicine, The Miriam Hospital, Assistant Professor of Medicine, Brown University, Providence, RI. Dr. Stenzel is an Infectious Disease Fellow at Brown University, Providence, RI.*) ❖

Special Feature

Linezolid (Zyvox)—A New Antimicrobial in the Fight Against Antimicrobial Resistance

By Thomas G. Schleis, MS, RPh

Linezolid is the first in a class of synthetic antibiotics known as oxazolidinones. It has been shown to be effective against vancomycin-resistant strains of *Enterococcus faecium*, *Staphylococcus aureus* (including methicillin-resistant strains), *Streptococcus agalactiae*, penicillin-resistant strains of *Streptococcus pneumoniae*, and *Streptococcus pyogenes* in in vitro and clinical infections. Linezolid binds to a site on the bacterial 23S ribosomal RNA of the 50S subunit and prevents the formation of a functional 70S initiation complex, which is an essential component of the bacterial translation process. Linezolid is bacteriostatic against enterococci and staphylococci and bactericidal for the majority of strains of streptococci. Because it inhibits bacterial protein synthesis by a mechanism different from other antibacterial agents, cross-resistance between linezolid and other agents is felt to be unlikely.

When administered either intravenously or orally every 12 hours, linezolid exhibits a half-life of approximately five hours with maximum plasma concentrations reached approximately 1-2 hours after dosing. Bioavailability from the oral formulations is approximately 100%, resulting in the same dosing either orally or intravenously. Linezolid can be administered without regard to meals.

Linezolid is rapidly distributed to well-perfused tissues. The ratio of linezolid in saliva relative to plasma is 1.2:1 and for sweat relative to plasma was 0.55:1. The concentration-independent plasma protein binding of linezolid is approximately 31% and it exhibits a volume of distribution of 40-50 liters in healthy adults.

Linezolid is primarily metabolized by oxidation of the morpholine ring, resulting in two inactive metabolites. Nonrenal clearance accounts for approximately 65% of the total clearance of linezolid, with approximately 30% of the dose appearing in the urine as linezolid, 40% as metabolite B, and 10% as metabolite A. While there is accumulation of metabolites of linezolid in patients with renal impairment, the clinical significance of this is unknown. As a result of this and other information, no dosage adjustments are suggested in patients with renal or hepatic insufficiency, or for elderly patients. There are limited data on treatment of pediatric patients with linezolid and it does not have FDA approval for that patient population.

Linezolid is indicated for adult patients with the following:

- Vancomycin-resistant *E. faecium*;
- Nosocomial pneumonia caused by *S. aureus* or *S. pneumoniae* (penicillin-susceptible strains);
- Complicated skin infections caused by *S. aureus*, *S. pyogenes*, or *S. agalactiae*;
- Uncomplicated skin infections caused by *S. aureus* (methicillin-susceptible strains) or *S. pyogenes*;
- Community-acquired pneumonia caused by *S. pneumoniae* (penicillin-susceptible strains) or *S. aureus* (methicillin-susceptible strains).

The most common adverse reactions in patients treated with linezolid were diarrhea, headache, and nausea. Other adverse events reported were oral and vaginal moniliasis, hypertension, dyspepsia, localized abdominal pain, pruritus, and tongue discoloration.

Pseudomembranous colitis and thrombocytopenia have been reported in patients receiving linezolid. While pseudomembranous colitis can occur with most antimicrobials, thrombocytopenia is more specific to linezolid and platelet counts should be monitored in patients who are at increased risk for bleeding.

Even more specific to linezolid is its ability to inhibit monoamine oxidase. This can potentially result in a number of drug-drug and drug-food interactions. Medications and foods with which interactions may occur include: adrenergic agents; sympathomimetic agents; vasopressors; dopaminergic agents; serotonergic agents; monoamine oxidase inhibitors (e.g., Nardil and Parnate); selective serotonin reuptake inhibitors (e.g., Prozac, Paxil, Zoloft, Celexa, Luvox, and Effexor); and foods high in tyramine (e.g., aged cheeses, smoked meats, sauerkraut, tap beers, red wines, soy sauce, etc.).

Dosages of linezolid for various indications are:

- Vancomycin-resistant *E. faecium* infections—600 mg IV or po q 12 hours for 14-28 days;
- MRSA—600 mg IV or po q 12 hours;
- Nosocomial or community-acquired pneumonia—600 mg IV or po q 12 hours for 10-14 days;
- Complicated skin infections—600 mg IV or po q 12 hours for 10-14 days;
- Uncomplicated skin infections—400 mg po q 12 hours for 10-14 days.

Linezolid is available as ready-to-use injections of 200, 400, and 600 mg; oral tablets of 400 and 600 mg;

and an oral suspension of 100 mg/5 mL in 240-mL bottles. The average wholesale pricing (AWP) for each dose of the 600-mg injectable is $71.88 and for the 600-mg tablet is $53.12.

Like the recently released Synercid (dalfopristin/quinupristin), the main interest in linezolid will be in the treatment of infections due to vancomycin-resistant *E. faecium* and resistant organisms when other antimicrobials are contraindicated. Most clinicians will limit its use in other areas due to the high cost of therapy and to avoid the potential of resistance development.

Most notable for linezolid is the potential for drug-food interactions due to its inhibition of monoamine oxidase. The true significance and incidence of interactions is not known and the manufacturer of linezolid, Pharmacia & Upjohn, can only suggest the likelihood of occurrences at this time. In our practice we had one incident of hypertension with a patient on Zoloft receiving linezolid, but have not completed our investigation into what role, if any, linezolid may have played. Nevertheless, it is an area that requires caution when prescribing linezolid, as many patients may not provide complete and factual information as to medications they are taking.

Fortunately, we now have two antibiotics that are potentially effective against vancomycin-resistant organisms, allowing us to maintain a fragile control over the problem of bacterial resistance. ❖

Oral Therapy for Kala-azar

ABSTRACT & COMMENTARY

Synopsis: *There are approximately 500,000 cases of visceral leishmaniasis per year in Asia, South America, and East Africa, with India, Brazil, and Sudan accounting for the most cases in these regions. An orally administered inexpensive therapy for visceral leishmaniasis would be a godsend.*

Source: Jha TK, et al. Miltefosine, an oral agent, for the treatment of Indian visceral leishmaniasis. *N Engl J Med* 1999;341:1795-1800.

There is currently no effective orally administered medication for any leishmania infection. Jha and colleagues describe a phosphocholine analogue that affects cell-signaling pathways and membrane synthesis and that can be administered orally to patients for the treatment of Indian visceral leishmaniasis (kala-azar). The study used an escalating dose, open-label phase 2 trial of four regimens that were tested at three clinical centers in India in 120 patients with a diagnosis of visceral leishmaniasis (VL). Eligible patients had signs and symptoms of VL (fever, loss of appetite, enlarged spleen) and at least "2+ leishmania" on splenic aspiration performed one week before the first dose of drug. Severe cases of VL with peripheral white cell count (WBC) less than 2000 cells per mm^3 and platelet count less than 75,000 per mm^3, Hgb less than 6.5 g/dL, or patients with HIV infection were excluded. A second splenic aspirate, obtained two weeks after completion of therapy, determined either parasitologic cure or failure of therapy. Cure was defined as parasitologic cure and clinical cure (loss of fever, 33% drop in spleen size, improvement of cytopenias and albumin level). Four cohorts, containing 30 persons in each, received 50, 100, or 150 mg of miltefosine per day for 4-6 weeks and in all 120 patients there was an initial parasitologic cure. Only six patients had clinical and parasitologic relapse six months after treatment. With the 100-mg dose for four weeks, 97% of patients were cured. Adverse effects noted were mild nausea and occasional vomiting soon after medication ingestion. Asymptomatic reversible hepatotoxicity and nephrotoxicity were observed in a few patients.

COMMENT BY MICHELE BARRY, MD, AND WENDY THANASSI, MD

This trial looks quite promising. Current treatment modalities for VL are largely impractical for the developing world. Liposomal amphotericin B, while 95% effective, is only available parenterally and costs a prohibitive $5000. Standard amphotericin is also 95% effective but frequently causes renal dysfunction and requires IV injections for 2-3 weeks. Other pentavalent antimonials have a clinical failure rate of 40% in some parts of India and are associated with a high incidence of pancreatitis and other adverse effects.

Miltefosine was originally developed as a treatment for cancer, and its exact mechanism of cytotoxicity with respect to the parasite is unknown. The recommended regimen from this trial was 100 mg/d orally (approximately 2.5 mg/kg) for four weeks, with a maximum dose of 4 mg/kg/d. The main side effect was a relatively minor gastroenteritis. Miltefosine's cost is unknown, but it is predicted to be "acceptable," as it is easily synthesized. This phase 2 trial is an encouraging step toward the establishment of an effective oral agent that could reduce mortality from this severe disease. We await more information on a phase 3 trial in VL and its

efficacy in children and in persons with HIV. How broadly applicable miltefosine therapy is for the other leishmanial clinical syndromes (caused by 21 species in 88 countries) is unknown at this time. (*Dr. Barry is Professor of Medicine, Co-Director, Tropical Medicine and International Travelers' Clinic, Yale University School of Medicine. Dr. Thanassi is a Resident, Yale University School of Medicine.*) ❖

CME Questions

38. Which of the following are true regarding coccidioidomycosis?

a. Culture, serology, skin test, or tissue staining can diagnose coccidioidomycosis.
b. *C. immitis* is endemic in the southwestern United States as well as northwestern Mexico, Venezuela, parts of Honduras, Guatemala, Argentina, Bolivia, Paraguay, and Colombia.
c. The majority of acute infections with *C. immitis* are asymptomatic or mild upper respiratory infections.
d. Clinical presentations of coccidioidomycosis include cough, fever, chills, arthralgias, erythema nodosum, and weakness.
e. All of the above

39. Which of the following is true about typhoid fever vaccination?

a. The oral formulation is as effective as the injectable.
b. It can free the traveler of strict food and water scrutiny.
c. It was shown to ameliorate but not eliminate typhoid.
d. Travelers have acquired typhoid fever despite vaccination.
e. a and d

40. Linezolid is indicated for adult patients with which of the following?

a. Vancomycin-resistant *E. faecium*
b. Complicated skin infections caused by *S. aureus*, *S. pyogenes*, or *S. agalactiae*
c. Uncomplicated skin infections caused by *S. aureus* (methicillin-susceptible strains) or *S. pyogenes*
d. Community-acquired pneumonia caused by *S. pneumoniae* (penicillin-susceptible strains) or *S. aureus* (methicillin-susceptible strains)
e. All of the above

41. Miltefosine is a new, potentially useful antiparasitic agent found to:

a. be effective in treatment of cutaneous and visceral leishmaniasis.
b. have irreversible and fatal hepatotoxicity in a few cases.
c. cause both parasitologic cure and clinical cure of visceral leishmaniasis.
d. interfere with mitochondrial metabolic pathways of leishmania.
e. be useful in consolidation therapy for chronic trypanosomal disease in Africa.

In Future Issues:

Rabies Exposures Overseas

UPDATES

By Carol A. Kemper, MD, FACP

Are Seals a Reservoir for Influenza B?

Source: Osterhaus AD, et al. *Science* 2000;288:1051-1053.

While birds are a well-recognized reservoir for influenza A, and influenza C has been isolated from pigs, no known animal host or non-human reservoir of infection has heretofore been identified for influenza B virus. While investigating the causes of respiratory illness in 12 juvenile harbor seals stranded on the Dutch Coast in 1999, the Seal Rehabilitation and Research Center in the Netherlands found evidence that at least two of the animals were infected with influenza B virus (B/Seal/Netherlands/1/99). Although both animals were suffering from respiratory illness at the time, a number of other pulmonary infections (e.g., lungworm) were also identified in the seals, obscuring the relationship of influenza B infection to illness. However, researchers demonstrated that the virus could be successfully propagated in seal kidney cell cultures.

Studies demonstrated that B/Seal/Netherlands/1/99 was similar to the strain of influenza B virus that had circulated in the human population in 1995—about 4-5 years earlier. Studies on stored sera demonstrated that eight of 391 seals (2%) cared for at the center after 1995 but none of 580 seals cared for before 1995 had evidence of influenza B infection. Thus, these data suggest the transmission of this or a related virus from the human population to seals in or around 1995, with subsequent persistence of low-level infection.

Harbor seals may therefore represent a previously unrecognized reservoir of influenza B infection for humans. These are the same seals tourists in San Francisco love to watch cavorting off of Pier 39, although presumably closer contact would be necessary for transmission of influenza. Such reservoirs are important for two reasons: they have the potential to allow for mutation of virus in a non-human host, and could serve as a point source for later introduction of virus into the human population. ■

Michigan White-tailed Deer Spreading TB

Source: ProMED-mail postings, April 26 and May 15, 2000; www.promedmail.org.

An outbreak of bovine tuberculosis (TB) due to *Mycobacterium bovis* has recently escalated in the white-tailed deer population in the northeastern section of Michigan's lower peninsula, potentially threatening domestic cattle in the area and in nearby Indiana. TB first appeared in the deer population in the mid-1990s, with reports of bagged deer with pulmonary and thoracic lesions. While 58 infected deer were identified in 1999, screening thus far this year has identified 282 positive deer of 27,175 tested (~1%). Most of the infected deer appear to be limited to Osceola, Antrim, and Mecosta counties, although three deer tested positive outside these areas. TB has also been identified in at least five herds of cattle in the lower peninsula—and authorities in both Michigan and Indiana are concerned about the potential for migrating deer to affect the vital cattle industry in both states. Indiana officials have even threatened to "fence in" their state to prevent deer migration across the state border.

Although pasteurization sterilizes milk products sold to the public, persons in contact with infected cattle, especially their respiratory secretions and mucous, would be at risk for infection. This reminds me of the outbreak of TB in circus elephants in 1996—the elephants with runny noses (runny trunks?) were highly contagious, especially if the animals sneezed (Kemper CA. *Infect Dis Alert* 1998;17:133-134). We often forget that other warm-blooded animals—domestic or wild—are potential TB carriers. Dogs in contact with infected humans have been known to "catch" TB, and their distant cousins, coyotes, are another increasing source of infection in the wild. ■

Rabies Devastating Cattle Herds in Chiapas

Source: ProMED-mail post, April 7, 2000; www.promedmail.org.

An outbreak of rabies transmitted by vampire bats preying on cattle in Chiapas, Mexico, is threatening the local population and economic base. And at least 2300 cattle have died from rabies infection in the first three months of this year—90% of which have been confirmed on necropsy. At least 92 ranches, most of which are small in size, have been affected. One rancher alone has lost 50 bulls, cows, and calves thus far this year. In addition, 27 people have been bitten by bats, all of whom are receiving therapy.

Rabies was first detected in Chiapas in 1987, but bat migration has resulted in spread of disease throughout the region. Ranchers are faulting the local government which they claim has been slow to respond to the endemic. Government officials claim that ranchers ignored the problem for too long, failing to adequately vaccinate their herds. Rabies vaccine is being made available to ranchers for an approximate cost of $0.11 per dose, but too few vaccines are being administered. Whichever group has been less responsive to date is irrelevant. Government officials would do better by initiating an area-wide vaccine program for all cattle at no charge to ranchers, less they face a public health disaster. Human rabies immune globulin is both costly and in short supply. ■