Salmonellosis Associated With Chicks and Ducklings—Michigan and Missouri, Spring 1999

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During the spring of 1999, outbreaks of salmonellosis associated with handling chicks and ducklings occurred in Michigan and Missouri. This report summarizes the epidemiologic information for the outbreaks and provides an overview of legislative efforts to control the distribution of chicks and ducklings. These outbreaks demonstrate that handling chicks and ducklings is a health risk, especially for children, and highlight the need for thorough handwashing after contact with chicks, ducklings, and other young fowl.

**Michigan**

In May 1999, the Michigan Department of Community Health (MDCH) was notified of an increase in *Salmonella* serotype Infantis infections with closely related pulsed-field gel electrophoresis (PFGE) patterns; 21 case-patients were reported with onset of illness during April 1-July 31, 1999. Ages of infected persons ranged from 8 days to 82 years (mean: 25 years); eight (38%) were aged less than 10 years. Twelve (57%) were female. Symptoms reported during patient interviews included diarrhea (81%), fever (57%), bloody diarrhea (24%), and vomiting (14%). Three patients were hospitalized. Overall, 17 (82%) patients reported direct and/or indirect contact with young fowl: eight (38%) with chicks, two (10%) with ducklings, one (5%) with pheasant, and six (29%) with multiple species, including chicks and ducklings. Of the young fowl that were traceable, 88% were shipped from a single hatchery.

MDCH conducted a case-control study to identify exposures associated with illness. Nineteen patients were enrolled and were matched by age and place of residence to 37 healthy controls using sequential-digit dialing. During the 5 days before illness onset, 14 (74%) of 19 patients had direct contact with young fowl or resided in a household that raised fowl (chicks, ducklings, goslings, pheasants, and/or turkeys) compared with six (16%) of 37 controls (matched odds ratio [MOR] = 20; 95% confidence interval [CI] = 3-378). In several households, young birds were kept inside the home. One child kept young birds in his bedroom and another carried chicks inside his jacket.

MDCH, with assistance from the Michigan Department of Agriculture (MDA), visited the implicated hatchery in September 1999. During the spring, the hatchery shipped approximately 100,000 birds per week by mail order directly to customers and to several feed and farm supply retail outlets across the state. Fowl were shipped in lots of 25 to 100 birds, and usually were raised for backyard use (i.e., meat and egg production for the family). *S. Infantis* with the outbreak PFGE pattern was recovered from three of 47 environmental samples and five of 33 bird samples taken at the hatchery. Other *Salmonella* serotypes also were isolated from the environmental samples, including serotypes Montevideo (seven), Chester (one), and Mbandaka (one).

**Missouri**

In April 1999, the Missouri Department of Health (MDOH) noted a cluster of *Salmonella* serotype Typhimurium infections with an identical PFGE pattern; 40 case-patients were identified with onset of illness during April 4—May 30, 1999. The ages of infected persons ranged from 8 months to 46 years (mean: 13 years); 28 (70%) were age less than 20 years; 23 (58%) were male. Symptoms reported by the 33 patients interviewed included fever (42%), bloody diarrhea (27%), stomach cramps (27%), and vomiting (21%). Three patients were hospitalized. Overall, 32 (97%) persons reported exposure to young fowl: 18 (56%) were exposed to chicks, 10 (31%) to ducklings, three (9%) to both chicks and ducklings, and one (3%) to a young turkey.

MDOH conducted a case-control study of persons exposed to chicks or ducklings to identify whether specific behaviors were associated with illness. Twenty case-patients were enrolled; 40 controls who had been exposed to chicks and ducklings during the same time were identified through media advertisements and word-of-mouth. During the 4 weeks before onset of patient illness, chicks or ducklings that were identified as ill by the patient or handler were associated with human illness (odds ratio [OR] = 21; 95% CI = 2—508). Handwashing after handling fowl was protective against illness (OR=0.0; 95% CI=0.0—0.2).

**Legislative Efforts**

During February 2000, CDC contacted 51 state and territorial public health departments to ascertain laws on the sale of baby fowl to noncommercial distributors and private persons; 28 (55%) responded. Ten (36%) states have laws restricting the sale of baby fowl for noncommercial purposes, including the sale of fowl aged less than 3 weeks (Indiana and Maryland), less than 4 weeks (Ohio and Pennsylvania), less than 8 weeks (Massachusetts and Virginia), and less than 12 weeks (Connecticut). In addition, Connecticut, Ohio, and Virginia require fowl to be sold in groups of greater than five birds. Illinois prohibits the sale of chicks during the Easter season, and Kansas requires persons to have a temporary or permanent license to sell chicks.
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CDC Editorial Note: Although most of the 1.4 million human salmonellosis cases that occur annually in the United States are caused by foodborne sources, direct contact with animals, particularly reptiles and occasionally birds, also may be a source of infection. Most reptiles and many birds shed Salmonella in their feces. Humans become infected when contaminated food, hands, or other objects are placed in the mouth; therefore, handwashing is critical to prevent Salmonella infections following direct or indirect contact with animals. The Missouri outbreak described in this report and previous outbreaks demonstrate that handling young fowl can be a risk for Salmonella infections, particularly in children who receive fowl as gifts during Easter; children have more frequent hand-to-mouth contact and are less likely to practice handwashing after handling fowl. The Michigan outbreak describes the risk for infection associated with the backyard production of fowl.

Prevention efforts, such as sales restrictions and consumer education, may be difficult because selling pet fowl and raising backyard fowl are largely unregulated. Several states responding to the survey reported laws that restrict the sale of chicks, ducklings, and other young fowl. Some of these restrictions are based on previous reports of chick-associated and duckling-associated salmonellosis during Easter. Enforcement may also be difficult because young fowl can be purchased by mail and Internet orders from out-of-state hatcheries. State-mandated point-of-sale educational material may be effective in educating consumers about the risk for salmonellosis. States may wish to join Michigan and Missouri in issuing a press release during the spring of 2000 to raise public awareness about the risk for Salmonella infections posed by young fowl. MDCH, MDA, and MDOH have developed safety instructions to be distributed with young fowl that emphasize the importance of handwashing and supervision of young children interacting with young fowl.

To prevent the transmission of Salmonella from chicks, ducklings, and other young fowl to humans, persons should avoid contact with feces and carefully wash their hands with soap and water after handling young fowl or anything that has come in contact with them. Chicks, ducklings, and other young fowl may not be appropriate pets for children and should not be kept in households with infants, children aged less than 5 years, or immunocompromised persons. During investigations of Salmonella infections, especially during spring and Easter, health-care workers and public health personnel should consider contact with young fowl as a potential source and obtain cultures from these animals if they are suspected as the source of infection.

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Fatal Yellow Fever in a Traveler Returning From Venezuela, 1999

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On September 28, 1999, a previously healthy 48-year-old man from California sought care at a local emergency department (ED) and was hospitalized with a 2-day history of fever (102°F [38.9°C]), chills, headache, photophobia, diffuse myalgias, joint pains, nausea, vomiting, constipation, upper abdominal discomfort, and general weakness. On September 26, he had returned from a 10-day trip to Venezuela. On September 29, an infectious disease physician from the ED contacted the Marin County Health Department (MCHD) about the patient’s symptoms; MCHD reported his illness to the California Department of Health Services (CDHS) as a suspected case of viral hemorrhagic fever. This report describes the investigation of the case.

On admission to the hospital, physical examination revealed icteric sclerae and tenderness in the upper abdomen. Multiple red papular lesions with excoriations consistent with recent mosquito bites were seen on his lower legs and feet. No hepatosplenomegaly or lymphadenopathy was noted. Laboratory results indicated markedly elevated serum bilirubin (5.9 mg/dL) and liver enzymes (alanine aminotransferase: greater than 5000 U/L; aspartate aminotransferase: greater than 3750 U/L; and alkaline phosphatase: 194 U/L), leukopenia (white cell count: 3.4x10³/mm³ with 82% segmented, 2% bands, and 2% atypical lymphocytes), thrombocytopenia (platelet count: 77,000/mm³), and evidence of acute renal failure (creatinine: 5.9 mg/dL; potassium: 6.4 mmol/L; and bicarbonate: 16 mmol/L).

A preliminary diagnosis of hemorrhagic fever syndrome was made, and the patient was placed on doxycycline and ceftiraxone. Cultures of blood and urine were negative for bacterial pathogens. Blood smears for malaria were negative. On October 1, the patient developed general seizures and upper respiratory obstruction. He was placed on mechanical ventilation and transferred to the intensive care unit. His condition deteriorated rapidly, with severe coagulopathy and cardiac arrhythmias. He died on October 4.

On October 7, an autopsy of the chest and abdomen was performed at the University of California San Francisco.
Medical Center. Histopathologic examination of the liver showed extensive necrosis, steatosis, and numerous Councilman bodies compatible with fulminant yellow fever (YF) hepatitis. Evidence of disseminated angioinvasive aspergillosis involving the lungs, heart, kidneys, adrenal glands, small and large bowel, stomach, and disseminated intravascular coagulation also was seen. Specimens of the liver were examined at CDC; YF viral antigens were found by immunohistochemistry (IHC) and YF virus-specific nucleic acids by polymerase chain reaction. Other IHC tests were negative for dengue virus, leptospira, New World arenaviruses, spotted fever group rickettsiae, and hantavirus. The patient’s serum was tested by CDHS; no antibody to YF virus (17D) was detected by immunofluorescence in serum drawn September 28, but an IgG titer of 1:128 and an IgM titer of greater than 1:80 were detected in serum drawn October 1.

During September 16-25, the patient had traveled with six companions to rainforests in southern Venezuela (Amazonas State). He experienced multiple mosquito bites during his visit despite using DEET-based repellents. Before his trip, the patient had received tetrus toxinoid, typhoid vaccine, hepatitis A vaccine, and malaria prophylaxis, but not YF vaccine. The six travel companions were contacted by CDHS about their health and vaccination status; none had become ill during or following the trip. Five had received YF vaccine before travel. The unvaccinated traveler’s serum was negative for YF virus antibody tested at CDC by enzyme-linked immunosorbent assay.

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CDC Editorial Note: This report describes the second case of imported fatal YF in a U.S. resident returning from South America since 1996, and the first such cases since 1924. Neither patient had received YF vaccine before travel. In the case described in this report, viral hemorrhagic fever was suspected and reported to the local health department. Histopathology, IHC studies, nucleic acid testing, and serology all demonstrated that the traveler died of YF complicated by angioinvasive aspergillosis. In 1996, a Tennessee resident returned from a 9-day trip to Brazil with fever, headache, and myalgias. He died 10 days after onset of symptoms, and YF virus was identified from tissue culture.

YF occurs in at least seven tropical South American countries (Bolivia, Brazil, Colombia, Ecuador, French Guiana, Peru, and Venezuela) and much of sub-Saharan Africa. The sylvatic cycle involves nonhuman primates and mosquitoes that breed in tree holes. Persons living or working in proximity to such jungle or forest habitats who are bitten by infected mosquitoes can develop “jungle YF.” Another cycle exists between humans and Aedes aegypti mosquitoes. Aedes aegypti mosquitoes are present in most urban centers of South and Central America, the Caribbean, and parts of the southern United States; persons in these areas are at risk for urban YF infection. YF has not been reported from India or other parts of Asia despite the presence of Aedes aegypti.

World Health Organization (WHO) data suggest that YF transmission is increasing. After adjustments for underreporting, WHO estimates that approximately 200,000 YF cases occur each year, most in sub-Saharan Africa. Concomitant with increased YF transmission, the number of travelers from the United States to South America and Africa has more than doubled since 1988. These travelers may be at risk for YF unless precautions are taken, including receipt of YF vaccine.

YF is one of three diseases (the others are plague and cholera) subject to international quarantine regulations. CDC is required to notify WHO of all YF cases in the United States within 24 hours. Accordingly, all suspected and confirmed cases should be reported immediately through local and state health departments to CDC’s National Center for Infectious Diseases, Division of Quarantine (DQ), telephone (404) 639-8100; acute and convalescent-phase serum should be collected and sent for viral isolation and diagnosis to CDC’s National Center for Infectious Diseases, Division of Vector-borne Infectious Diseases, telephone (970) 221-6400. CDC’s DQ also is responsible for certifying YF vaccination centers in the United States. Since September 1, 1977, CDC has delegated to state and territorial health departments the responsibility to designate and supervise nonfederal YF vaccination centers within their jurisdictions. The location of certified U.S. YF vaccination centers is available from local and state health departments. If YF vaccine is medically contraindicated, health-care providers should supply persons with a letter listing reasons for not vaccinating, and persons should carry this with them when traveling. Details of vaccine recommendations and requirements of individual countries are available from the CDC World-Wide Web site, http://www.cdc.gov/travel.

CDC recommends YF vaccination for travelers to countries reporting YF. Vaccination also is recommended for travel outside urban areas of countries that officially do not report the disease but are in the YF-enzootic zone. Travelers should also take protective measures to reduce contact with mosquitoes; these include wearing clothes that cover most of the body, staying in well-screened areas, using insect repellent (containing DEET at a concentration of less than 35% are recommended) on exposed skin and clothing, and sleeping under bed nets treated with permethrin or deltamethrin insecticides.

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Hantavirus Pulmonary Syndrome—Panama, 1999-2000

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HANTAVIRUS PULMONARY SYNDROME (HPS) is an acute viral rodentborne zoonosis characterized by severe cardio-pulmonary illness with a 40%-60% case-fatality rate. Since its identification in the United States in 1993, the recognized clinical spectrum of illnesses associated with human hantavirus infection has expanded to include mild illness, and case-patients have been identified in Canada and South America.1,2 This report describes the first confirmed HPS cases from Central America and summarizes preliminary results of clinical, epidemiologic, and ecologic investigations. Investigators identified 12 suspected cases with typical disease and captured four common species of rodents near case households.

In mid-January 1999, reports of a cluster of acute febrile respiratory illnesses associated with three deaths in Las Tablas and Guarare districts, Los Santos province, Panama (population: 55,000), led to an investigation by the Gorgas Memorial Institute for Health Studies (GMIHS) and the Panamanian Ministry of Health (MOH). Human illness was characterized by fever, myalgia, headache, and cough with rapid progression to respiratory failure and bilateral pulmonary infiltrates, hypotension, and thrombocytopenia consistent with HPS. Initial laboratory testing of specimens at CDC from three case-patients confirmed the presence of IgM and IgG antibodies using antigens of Sin Nombre virus and the presence of detectable hantavirus RNA by reverse-transcription polymerase chain reaction (RT-PCR).

As of March 6, 2000, 12 patients with suspected HPS have been identified; three died. The mean age of patients was 42 years (range: 26-58 years); 58% were women. Serum specimens taken from eight case-patients who met the CDC epidemiologic case definition1 had hantavirus antibodies, including two specimens that also were positive by RT-PCR. Serologic testing is pending for another suspected case-patient. Although the three patients who died had signs and symptoms compatible with HPS, epidemiologic data are limited and no specimens from these patients are available for diagnostic testing. The first case-patient was identified retrospectively as having onset of illness in August 1999; the latest case-patient had onset of illness on February 28. All 12 case-patients had clinical disease typical of HPS; however, seven case-patients had atypical extrapolmonary symptoms of hepatic, renal, and cerebral dysfunction. No cases have been reported among health-care workers or from person-to-person transmission. Studies to define the prevalence of hantavirus infection among community members and health-care workers, the extent of mild or asymptomatic infection, and the risk factors for developing HPS are in progress.

Rodent trapping at 10 homes, six occupied by confirmed case-patients, yielded 54 rodents; four common species accounted for 98% of captured rodents and were identified tentatively as Zygodontomys brevicauda (25 [46%]), Sigmodon hispidus (14 [26%]), Mus musculus (eight [15%]), and Oligoryzomys sp (six [11%]). Although only 5% of traps captured rodents, reports from GMIHS indicated a recent increase in peridomestic rodents associated with increased rainfall and flooding in surrounding areas.

To prevent additional hantavirus transmission to humans, MOH disseminated information to the Los Santos community about risk-reduction measures and to physicians about how to recognize patients and manage HPS. A public awareness and risk-reduction campaign was implemented nationwide and included televised public service announcements and distribution of educational posters and pamphlets. In addition, an outbreak communication center was established and staffed 24 hours a day by specially trained physicians, public health officials, and health educators to collect reports of suspected HPS cases and to answer questions from the public and health-care providers about hantaviruses. MOH physicians are contacting hospitals nationwide to promote HPS awareness, to evaluate suspected cases, and to provide treatment guidance on the basis of standard HPS criteria and guidelines.2

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CDC Editorial Note: HPS is a pan-American zoonosis identified in 1993 with confirmed cases ranging from Canada and the United States to southern South America.2,3 HPS is attributed to infection with New World hantaviruses maintained by natural hosts in the rodent family Muridae, subfamily Sigmodontinae. Infection in humans occurs after inhalation of aerosolized virus or direct contact with infected rodents or their excreta. Per-
Study of which are related New York viruses, the reser-

virus. An increasing number of hantaviruses is be-

ing identified by genetic sequencing, including from the two RT-PCR-positive cases described in this re-

port. On the basis of antigenic simi-

larities, CDC has identified all cases of hanta-

virus. Reports of HPS and requests for diag-

nostic testing and epidemic assistance can be directed to CDC's National Center for Infectious Diseases, Division of Viral and Rickettsial Diseases, Special Pathogens Branch, telephone (404) 639-1511. Information about HPS can be found on the CDC World-Wide Web site, http://www.cdc.gov/ncidod/diseases/hanta/hps/index.htm.

The epidemiology of HPS is closely related to the ecology of the rodent reservoir populations. Increased US incidence of HPS has been linked to peri-

ods of above average rainfall in normally dry areas of the southwestern United States resulting in improved habitat quality and increased numbers of infected reservoir rodents. Blood and tissue samples from the captured species of sigmodontine rodents in Panama will be tested for hantavirus antibody and RNA to identify the specific reservoir. Preliminary data indicate that rainfall in parts of Los Santos was unusually high during the fall and winter of 1999.

A possible association between increased precipitation and increased numbers of reservoir rodents in the affected and surrounding areas is being explored. Sigmodontine rodents occur throughout the Americas, but HPS never has been found between Texas and Brazil. However, the discovery of HPS in Panama was expected and should lead to increased vigilance for the syndrome in Mexico, Central America, and northern South America.

Because no specific therapy exists for HPS, prevention measures should be taken to avoid contact with wild rodents, including preventing entry of wild rodents into human dwellings; eliminating food and shelter for rodents in the peridomestic environment; safe trapping (using kill-traps) and disposal of wild rodents that enter homes; and careful cleaning and disinfecting of areas in and around homes that have been contaminated by rodents. Reports of HPS and requests for diagnostic testing and epidemic assistance can be directed to CDC's National Center for Infectious Diseases, Division of Viral and Rickettsial Diseases, Special Pathogens Branch, telephone (404) 639-1511. Information about HPS can be found on the CDC World-Wide Web site, http://www.cdc.gov/ncidod/diseases/hanta/hps/index.htm.

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Motor-Vehicle Occupant Fatalities and Restraint Use Among Children Aged 4-8 Years—United States, 1994-1998

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IN THE UNITED STATES, MORE CHILDREN AGED 4-8 YEARS DIE AS OCCUPANTS IN MOTOR-VEHICLE-RELATED CRASHES THAN FROM ANY OTHER FORM OF UNINTENTIONAL INJURY. To reduce the number of deaths and injuries caused by motor-vehicle-related trauma, child passengers in this age group should be restrained properly in a vehicle's back seat. To characterize fatalities, restraint use, and seating position among occupants aged 4-8 years involved in fatal crashes, CDC analyzed 1994-1998 data from the Fatality Analysis Reporting System (FARS), which is maintained by the National Highway Traffic Safety Administration (NHTSA). This report summarizes the results of that analysis, which indicate that during 1994-1998, little change occurred in the death rate, restraint use, and seating position among children aged 4-8 years killed in crashes.

Motor-vehicle occupants who died in crashes during 1994-1998 were included in the analysis of FARS data. FARS is a census of traffic crashes in which at least one occupant or non-motorist (e.g., pedestrian) died within 30 days of a crash on a public road within the 50 states, District of Columbia, and Puerto Rico. FARS includes information about restraint use and seating position derived from police crash reports. Restraint use (e.g., seat belts, child-safety seats [CSSs], and belt-positioning booster seats) was reported as used or not used. Seating position was designated as front, back, other, or unknown. Injury death rates per 100,000 population were calculated using annual estimates from the Bureau of the Census.

During 1994-1998, 14,411 child occupants aged 4-8 years were involved in crashes where one or more fatalities occurred; of these, 2549 (17.7%) died. Approximately 500 child occupants died each year during the study period; the average annual age-specific death rate was 2.6 per 100,000 population. In 1994, restraint use among fatally injured children was 35.2% (177 of 503); in 1998, restraint use was 38.1% (201 of 527). The proportion of fatally injured children seated in the back seat of a vehicle involved in a crash was 50.1% (252 of
possible to reduce risk for injury in a crash, approximately one fourth of child passengers ride in the front seat.7 Riding in the back virtually eliminates injury risk from deployed front-seat passenger air bags and places the child in the safest part of the vehicle in the event of a crash. As of January 1, 2000, 35 children aged 4-8 years have died while seated in front of air bags. Of these children, 31 (89%) were either unrestrained or improperly restrained.8 Riding in the back seat is associated with at least a 30% reduction in the risk for fatal injury.9 Approximately half of those children in this study who were fatally injured were sitting in the back seat.

The 50 states, District of Columbia, and Puerto Rico have child-passenger safety laws; however, substantial gaps in coverage exist for child passengers aged 4-8 years. For example, in 19 states, children this age can ride unrestrained or improperly restrained. In 2002, children aged 4-8 years may use an adult seat belt. No state requires the use of booster seats for children who have outgrown their CSSs.10 Three states have laws requiring that children be seated in the back seat of passenger vehicles. The ages of the children covered by these laws vary by state.

The findings in this study are subject to at least three limitations. First, police crash reports overestimate restraint use; therefore, restraint use may be lower for children in this age group. Second, vehicle miles traveled have increased during 1994-1998; consequently, improvements in fatality rates may be masked by increased exposure to travel. Finally, increases in restraint use and resulting changes in occupant fatalities may require many years of investigation before they become apparent.

Reducing fatalities among motor vehicle occupants aged 4-8 years will require finding effective strategies to promote booster seat use and placement of children in the back seat. Public health and traffic safety efforts should be accelerated to increase appropriate occupant protection among children aged 4-8 years as a primary means to reduce fatal motor-vehicle-related injuries. Efforts are under way by CDC and others to determine the best ways to encourage booster seat use and to increase the prevalence of properly restrained children riding in the back seat.

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