

ORIGINAL ARTICLE

Multidrug-Resistant *Salmonella enterica* Serotype Typhimurium Associated with Pet Rodents

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ABSTRACT

BACKGROUND

An estimated 1.4 million salmonella infections occur annually in the United States. The majority of these infections are foodborne, but many are acquired by contact with animals. In August 2004, isolates of *Salmonella enterica* serotype Typhimurium, which were indistinguishable from one another by pulsed-field gel electrophoresis (PFGE), were obtained from eight hamsters from a Minnesota pet distributor. We conducted an investigation to determine whether human cases of salmonella could be linked to this rodent-borne strain.

METHODS

To identify cases of human infection with *S. enterica* serotype Typhimurium potentially related to pet rodents, we reviewed salmonella PFGE patterns submitted to the National Molecular Subtyping Network for Foodborne Disease Surveillance. Patients with isolates matching the hamster strain were interviewed about exposure to pet rodents. Implicated rodents were traced to pet stores, distributors, and breeders.

RESULTS

We identified matching *S. enterica* serotype Typhimurium isolates from 28 patients in whom the onset of illness occurred between December 2003 and September 2004. Of 22 patients (or in the case of children, their parents) interviewed, 13 patients (59%) in 10 states reported exposure to pet hamsters, mice, or rats, and 2 (9%) had secondary infections. The median age of the 15 patients with primary or secondary rodent exposure was 16 years, and 6 patients (40%) were hospitalized. Thirteen associated pet stores supplied by seven distributors were identified in 10 states. No single source of the rodents was identified. The outbreak strain of *S. enterica* serotype Typhimurium was cultured from a patient's pet mouse and from seven hamsters from pet stores. Closely related *S. enterica* serotype Typhimurium isolates were cultured from rodent cages and reusable transport containers at a pet distributor. Human, rodent, and environmental isolates were resistant to ampicillin, chloramphenicol, streptomycin, sulfisoxazole, and tetracycline.

CONCLUSIONS

Pet rodents probably are an underrecognized source of human salmonella infection.

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EACH YEAR, AN ESTIMATED 1.4 MILLION persons in the United States acquire salmonellosis, leading to approximately 14,800 hospitalizations and 400 deaths.¹ Salmonella is found in the intestinal tract of animals, and the majority of human infections occur after ingestion of contaminated foods.² However, many infections are acquired by contact with animals. Salmonellosis outbreaks have been associated with handling reptiles and amphibians, chicks, ducklings, kittens, and hedgehogs.³⁻⁸ We report an outbreak of multidrug-resistant *Salmonella enterica* serotype Typhimurium infections associated with commercially distributed pet rodents.⁹

METHODS

RECOGNITION OF HAMSTER SALMONELLOSIS

On August 30, 2004, a veterinarian for a Minnesota pet distributor telephoned the Minnesota Department of Health about the isolation of salmonella from two ill hamsters submitted to the University of Minnesota Veterinary Diagnostic Laboratory. The hamsters were part of a shipment of 780 received from an Iowa pet distributor; of these, 243 were sent from the Minnesota distributor to 15 retail pet stores in four states. Subsequently, numerous hamster deaths occurred, and further shipments of rodents from the Minnesota distributor to pet stores were stopped.

Eight hamsters with a history of diarrhea, lethargy, and rough hair coat were ultimately submitted to the University of Minnesota Veterinary Diagnostic Laboratory for postmortem examination. To conserve resources, six hamsters were divided into three groups of two hamsters each, and pooled tissue samples from each group were cultured. Salmonella group B (4, 5) was isolated from the liver, intestine, or lungs of the two individual hamsters and all three hamster pairs. Isolates were confirmed as *S. enterica* serotype Typhimurium and subtyped by pulsed-field gel electrophoresis (PFGE) at the Minnesota Department of Health.¹⁰ All hamster *S. enterica* serotype Typhimurium isolates were indistinguishable by PFGE.

IDENTIFICATION AND CLINICAL DESCRIPTION OF INITIAL CASES IN HUMANS

A query to the National Molecular Subtyping Network for Foodborne Disease Surveillance (PulseNet) Salmonella Database in September 2004 revealed

that the PFGE pattern of the hamster isolates was uncommon, representing 23 of 17,737 *S. enterica* serotype Typhimurium isolates obtained since 1998 (0.1%). As a result of routine surveillance, isolates with this pattern from a patient in South Carolina and a patient in Minnesota had been submitted in June 2004 and August 2004, respectively. The families of these patients were reinterviewed about possible rodent exposure.

South Carolina

During June 2004, a 4-year-old boy was hospitalized for 5 days with fever (temperature, 40.6°C), watery diarrhea, and abdominal cramping. A stool culture yielded *S. enterica* serotype Typhimurium. Nine days before the onset of the boy's illness, his family had purchased a hamster from a pet store; the hamster was found dead 2 days later.

Minnesota

During August 2004, a 5-year-old boy had diarrhea (initially bloody) lasting 14 days, abdominal cramps, vomiting, and fever (temperature, 39.4°C). A stool culture yielded *S. enterica* serotype Typhimurium. Four days before the onset of the boy's illness, his family had purchased a mouse from a pet store. The mouse became lethargic and developed diarrhea immediately after purchase; nevertheless, the boy frequently handled and kissed it. One week after purchase, the mouse died; it was frozen and later submitted to the Minnesota Department of Health. Cultures of the mouse's lungs, pooled liver and spleen, and intestines yielded *S. enterica* serotype Typhimurium isolates that were indistinguishable from the boy's isolate by PFGE.

EPIDEMIOLOGIC, ENVIRONMENTAL, AND LABORATORY INVESTIGATIONS

The Minnesota and South Carolina cases prompted a national search for additional salmonella infections in humans that were potentially associated with pet rodents. The PulseNet National Salmonella Database was reviewed for isolates that matched the outbreak strain (i.e., that were indistinguishable by PFGE from the South Carolina and Minnesota human case isolates). The health departments of states where such isolates were located were asked to interview the patients or their parents about contact with pet rodents. Primary patients were defined as those from whom the outbreak strain of *S. enterica* serotype Ty-

phimurium was isolated during 2004 and who had had contact with a pet rodent, such as a hamster, mouse, rat, gerbil, or guinea pig, within 7 days before the onset of illness.

The Centers for Disease Control and Prevention (CDC), state and local health departments, and the U.S. Department of Agriculture conducted traceback investigations of rodents from patients to pet stores, distributors, and breeders. Environmental sampling and testing were performed at pet distributors in Minnesota and Georgia. Because of jurisdictional and resource constraints, other state health departments did not perform systematic environmental testing of implicated pet facilities.

Animal and environmental *S. enterica* serotype Typhimurium isolates were sent to state public health laboratories for serotyping, antimicrobial-susceptibility testing, and PFGE subtyping. Antimicrobial-susceptibility results were confirmed at the CDC laboratory for the National Antimicrobial Resistance Monitoring System — Enteric Bacteria. Three *S. enterica* serotype Typhimurium isolates from humans were phage typed at the CDC.

RESULTS

CASE INFORMATION

Twenty-eight matching isolates of *S. enterica* serotype Typhimurium from humans were identified. Of 22 patients (or their parents) who could be interviewed, 13 (59%) had had contact with rodents purchased from retail pet stores (Fig. 1). Two patients (9%) had acquired salmonellosis through secondary transmission from a primary patient who had been exposed to a rodent. Seven patients (32%) had no identified rodent exposure. Matching isolates were obtained from 1 submitted urine specimen and 27 stool specimens from patients.

The 15 patients with primary or secondary rodent exposure were from 10 states (Fig. 1). The month of onset of illness ranged from December 2003 to September 2004 (Fig. 2). The median age of the patients was 16 years (range, 0 to 43); seven patients (47%) were under 8 years of age. Symptoms included abdominal cramping (77%), fever (67%), vomiting (53%), and bloody diarrhea (20%). Six patients (40%) were hospitalized for a median of 4 days (range, 2 to 56); no patient died. Four hospitalized patients were under 8 years of age.

The 13 patients with primary rodent exposure had been exposed to pet hamsters (2 patients), pet mice or rats (4 patients), and mice or rats purchased to feed snakes (7 patients).

One of the primary patients was a 23-year-old pregnant woman who was hospitalized in September 2004 with diffuse abdominal pain, diarrhea, and fever (38.9°C). The initial white-cell count was 17,000 per cubic millimeter, with 55% neutrophils and 37% band forms. The patient underwent exploratory laparotomy, and the visualized appendix appeared normal; an incidental appendectomy was performed. A stool culture obtained subsequently yielded *S. enterica* serotype Typhimurium. After surgery, the patient went into preterm labor and on the fourth hospital day vaginally delivered a 2080-g neonate at 32.5 weeks' gestation. The mother's hospital course was further complicated by postoperative aspiration pneumonia, with progressive respiratory failure. She required mechanical ventilation for 2 days, underwent extubation, and was discharged from the hospital on day 10.

During the first week of life the neonate showed signs of gastrointestinal obstruction, with bloody stools, increasing white-cell counts (peak value, 29,000 per cubic millimeter), and thrombocytopenia (trough value, 41,000 platelets per cubic millimeter). *S. enterica* serotype Typhimurium was isolated from a stool culture obtained on the third day of life. The neonate received gentamicin and cefotaxime for 3 weeks, administered with vancomycin for the first 10 days; however, after discontinuation of antimicrobial therapy on day 21, bloody stools, abdominal distention, and evidence of inflammation redeveloped over the next 10 days. *S. enterica* serotype Typhimurium was again isolated from the stool, and ceftriaxone and gentamicin were administered. On day 46, exploratory laparotomy revealed distal small-bowel obstruction with intestinal stricture, perforation, and extensive adhesions. The gallbladder and appendix were uninvolved. A 10-cm necrotic segment of jejunum and ileum was resected. The infant was ultimately discharged home from the intensive care nursery on hospital day 56.

The *S. enterica* serotype Typhimurium isolates from both the mother and the infant were identified as the outbreak strain by PFGE subtyping. The exposure history showed that before the onset of illness, the mother had purchased live rats and mice from a local pet store to feed her ball python. No salmonella was isolated from culture of the

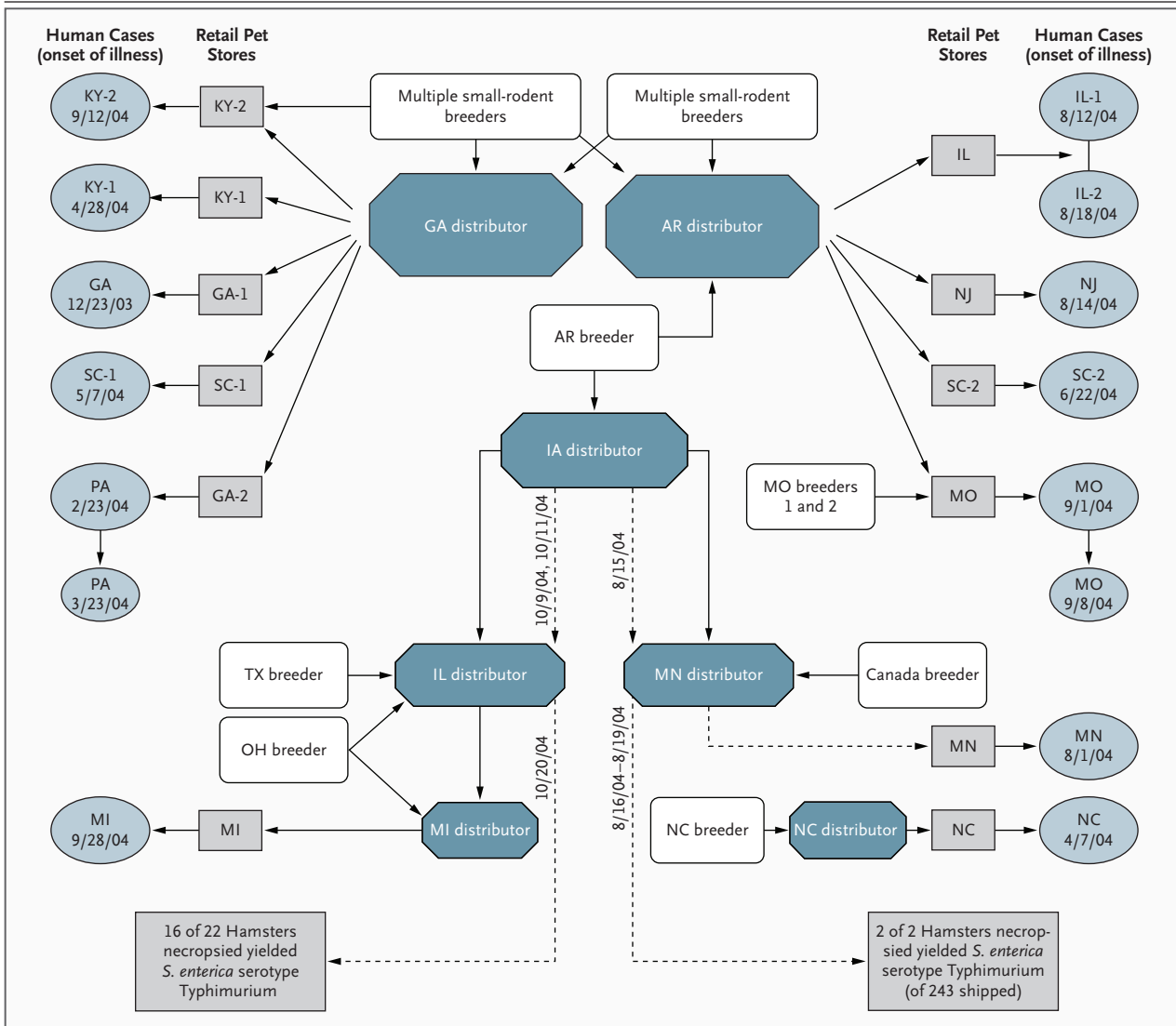
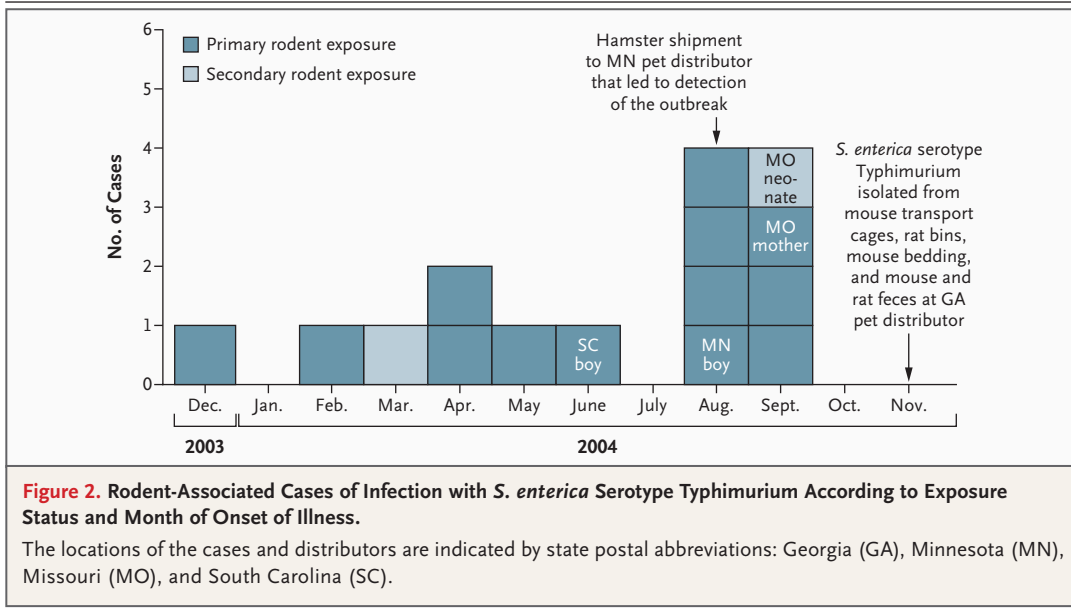


Figure 1. Traceback Results for Pet Rodents Associated with an Outbreak of Multidrug-Resistant *S. enterica* Serotype Typhimurium.

Dashed lines denote the movement of rodents with culture-confirmed multidrug-resistant *S. enterica* serotype Typhimurium. The locations of patients, pet stores, distributors, and breeders are indicated by state postal abbreviations: Arkansas (AR), Georgia (GA), Illinois (IL), Iowa (IA), Kentucky (KY), Michigan (MI), Minnesota (MN), Missouri (MO), New Jersey (NJ), North Carolina (NC), Ohio (OH), Pennsylvania (PA), South Carolina (SC), and Texas (TX); one breeder was located in Canada. Patients IL-1 and IL-2 were a mother and daughter exposed to a single purchased rodent. Cases of secondary transmission by rodent-exposed humans occurred in Pennsylvania (3/23/04) and Missouri (9/8/04). The outbreak strain of *S. enterica* serotype Typhimurium was isolated from both the Minnesota patient and a necropsied pet mouse. *S. enterica* serotype Typhimurium was isolated from pooled cultures of mouse transport cages, rat bins, mouse feces and bedding, and rat feces from the Georgia distributor; the pulsed-field gel electrophoresis (PFGE) patterns of three isolates were closely related to that of the outbreak strain, with a difference of two bands. On August 15, 2004, a total of 780 hamsters were received by the Minnesota distributor from the Iowa distributor. The outbreak strain of *S. enterica* serotype Typhimurium was isolated from the internal organs of 8 necropsied hamsters. Of these 780 hamsters, 243 were shipped by the Minnesota distributor to 15 pet stores in four Midwestern states. *S. enterica* serotype Typhimurium was isolated from 1 hamster each returned by a Minnesota and an Iowa pet store, and these hamsters were submitted for necropsy. *S. enterica* serotype Typhimurium was isolated from 16 of 22 unsold ill hamsters submitted for necropsy by an Illinois pet store supplied by the Illinois distributor; isolates from 7 hamsters were submitted for PFGE subtyping, and all were of the outbreak strain. Hamsters from the Illinois distributor were traced to shipments from the Iowa distributor received in October 2004. No human illnesses associated with these hamsters were identified.



snake feces. The original rodents and cages were unavailable for testing.

ANIMAL TRACEBACK, ENVIRONMENTAL TESTING, AND LABORATORY RESULTS

Tracing back the origin of the rodents from patients' households revealed geographically dispersed retail pet stores and distributors. Thirteen associated pet stores supplied by seven distributors were identified in 10 states. No single source of rodents was common to all cases, and each case household had purchased its rodent or rodents from a different retail pet store (Fig. 1). No common link was identified among the three primary implicated pet distributors, which were located in Arkansas, Georgia, and Iowa; a systematic review of commercial records was not possible because of inadequate record keeping by potentially involved breeders and distributors. The ultimate source of the infected rodents involved in this outbreak was not identified.

Information on antimicrobial use was obtained from five rodent breeders or distributors. Routine use of antimicrobials (e.g., spectinomycin, leptomycin, tetracycline, and nitrofurazone) for the prevention of nonspecific rodent enteritis was documented in four facilities. The rodents received antimicrobial drugs in their drinking water at weaning, before transport, or on arrival at the distributor. One distributor used tetracycline-containing feed for all rodent feedings.

In November 2004, *S. enterica* serotype Typhimurium was isolated from mouse transport cages, rat bins, mouse feces and bedding, and rat feces at the Georgia distributor. Three of the four isolates were indistinguishable by PFGE and were closely related (within two bands) to the outbreak strain. The fourth isolate, from rat feces, differed from the other three isolates and from the outbreak strain by two bands. No salmonella was isolated from environmental cultures taken from the Minnesota pet distributor; however, the cultures were obtained after the facility had undergone two thorough sanitizations with bleach in response to the initial outbreak of illness among the hamsters. Systematic environmental cultures were not obtained at the implicated Arkansas and Iowa pet distributors or at any breeding facilities.

All human, animal, and environmental *S. enterica* serotype Typhimurium isolates tested in this outbreak were uniformly resistant to ampicillin, chloramphenicol, streptomycin, sulfisoxazole, and tetracycline (R-type ACSSuT). Phage typing of three isolates from stools of patients in Kentucky, Minnesota, and New Jersey identified definitive phage type 120 (DT120).

DISCUSSION

We describe an outbreak of multidrug-resistant *S. enterica* serotype Typhimurium infections associated with handling of rats, mice, and hamsters

that were distributed through a wide network of breeders, distributors, and retail pet stores. Salmonellosis associated with rodents is rarely identified in humans.¹¹ Molecular techniques and national sharing of PFGE patterns through PulseNet were critical in identifying this outbreak, because the cases were widely dispersed temporally and geographically.

Although the majority of *S. enterica* serotype Typhimurium infections result in a self-limited gastrointestinal illness, the case of the mother–neonate pair described here illustrates a more severe clinical course. The observed hospitalization rate of 40% is consistent with previous reports that patients infected with antimicrobial-resistant *S. enterica* serotype Typhimurium strains have higher hospitalization rates than patients infected with susceptible strains.^{12,13} Additional studies have documented increased risks of bloodstream infection, treatment failure, and death associated with multidrug-resistant *S. enterica* serotype Typhimurium.^{12,14,15} Phage typing of isolates in this outbreak indicated DT120, which is often multidrug-resistant.¹⁶

S. enterica serotype Typhimurium was originally isolated from mice. As the serotype name implies, *S. enterica* serotype Typhimurium among mice can mimic *S. enterica* serotype Typhi infection among humans.¹⁷ *S. enterica* serotype Typhimurium is frequently isolated from both captive and wild rodents; morbidity and mortality are variable.^{18,19} Asymptomatic infection of rodents with salmonella does occur, with intermittent fecal shedding lasting weeks to months.^{20–23} Shedding of salmonella by mice and rats has been shown to result in infection of cage mates, with further spreading of salmonella through multiple colonies of rodents.²⁴

Transportation stress has also been associated with reactivation of *S. enterica* serotype Typhimurium among animals, including development of diarrhea and increased fecal shedding.^{25,26} *S. enterica* serotype Typhimurium can survive for more than a year in the environment, even under conditions of nutrient starvation and osmotic stress.^{27,28} Recovery of *S. enterica* serotype Typhimurium from reusable transport containers, cages, and bins illustrates how the organism may be spread geographically. Rodents transported or housed in contaminated containers may be exposed to salmonella without direct contact with

infected rodents. Transport of these rodents through a complex network of distributors before they reach their final pet-store destination could facilitate the geographic spread of *S. enterica* serotype Typhimurium. Transmission of salmonella among various rodent species may occur as a result of close proximity of rodents of different species during shipping and within the pet facilities and through the shared use of fecally contaminated containers and cages.

The dissemination of multidrug-resistant salmonella among rodents might have been further facilitated by the widespread use of prophylactic antimicrobials such as tetracycline and spectinomycin within the “pocket-pet” industry. Previous investigators have demonstrated the virtual elimination of Enterobacteriaceae flora from the intestinal tract of mice after brief administration of oral antimicrobials.²⁹ Repopulation with resistant organisms, including resistant salmonella species, was observed among mice treated with antimicrobials.^{29–32} The administration of antimicrobials to mice can also lower the infectious dose of resistant salmonella by a factor of 100,000, increase the degree of fecal shedding by a factor of 1000, and prolong the shedding of resistant organisms.^{32–34} Thus, in this outbreak, the routine delivery of nontherapeutic antimicrobials in food or water probably contributed to increased salmonella infection and shedding, facilitating increased transmission among animals and from animals to their human caretakers.

It is likely that only a small proportion of the actual number of rodent-associated cases has been detected. Surveillance for salmonellosis is limited by factors such as care seeking, stool submission, laboratory testing, culture sensitivity, and reporting. Only 1 in 38 patients with salmonella infection is believed to be ultimately identified by public health agencies.¹ In addition, not all *S. enterica* serotype Typhimurium isolates in the United States undergo subtyping by PFGE, a critical step in identifying cases that are dispersed temporally and geographically. Finally, we did not investigate other *S. enterica* serotype Typhimurium PFGE subtypes, such as those isolated at the Georgia distributor.

Medical professionals and public health practitioners should consider pet rodents a potential source of salmonellosis. Veterinarians and animal vendors should consider submitting speci-

mens to clinical laboratories for isolation of salmonella if substantial diarrhea-associated complications or death occurs among rodents intended for sale. Heightened infection-control practices by pet facilities, including routine sanitizing of animal-transport containers and cages, would most likely reduce transmission. Appropriate animal husbandry and hygiene practices can reduce the need for nontherapeutic antimicrobials to prevent disease among rodents.³⁵

Consumers and those who work with animals should be aware that rodents can shed salmonella and should expect rodent feces to be potentially infectious. Handling of pet rodents is a potential health risk, especially for children. To reduce salmonella transmissions, the hands should be thoroughly washed with soap and wa-

ter after handling rodents, their cages, or their bedding.

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