Humans and Pets as Sources of Salmonellae[1]

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Abstract

Reported non-typhoid salmonella infections in man have increased in the United States from 723 in 1946 to 20,040 in 1966, and this may represent only 1% of the salmonella infections actually occurring. The temporary carrier state following infections in man represents an important source for the spread of infection to other persons. Pets including dogs, cats, Easter chicks and ducklings, and turtles can also be a source of human salmonella infections. Approximately 15–20% of normal household dogs may be infected with salmonellae, although documented human infections traced to dogs are rare. Pet Easter chicks and ducklings have been incriminated frequently as a source of infection in young children. More than 100 cases of salmonellosis have been traced to pet turtles. Only by the implementation of appropriate control measures attacking each one of these potential sources of infection can we hope to reduce the increasing incidence of salmonellosis in the United States.

Introduction

Among the more significant advances in the study of salmonellosis during the past two decades have been the numerous contributions to our knowledge of the ecology of the genus *Salmonella,* improved reporting of the occurrence of salmonellosis, and more thorough epidemiologic investigation of outbreaks of the disease. Unfortunately, the majority of the infections diagnosed in man still are classified as sporadic cases. This situation is due largely to lack of personnel and facilities to investigate each case thoroughly. This conclusion is supported by reports from states appearing throughout the Communicable Disease Center Salmonella Surveillance Reports. From these, it is quite evident that the number of cases in which associated familial infections are found vary widely from one State to another. Some States consistently report much higher percentages of family-associated infections than do others. Epidemiologic investigation has also been hindered by lack of adequate facilities or methods for exact identification of strains within those serotypes which are widely distributed and isolated frequently.

While methodology in the detection of salmonellosis has improved within the past 20 years the knowledge that has been gained concerning the occurrence and distribution of salmonellae within that period stems not so much from improved methodology as from wider application of known methods and from early isolated observations regarding the presence of salmonellae in previously unsuspected locations and materials which stimulated a systematic search for the organism in man, animals, foods and environment.

The fact that the primary host-adapted salmonelloses of man and fowls rapidly were being brought under control directed the attention of investigators to other salmonelloses which were being reported in increasing numbers. For the period 1946–1964, the reported annual incidence of typhoid fever in the United States fell steadily, whereas reports of other salmonelloses increased almost 30-fold. In fowls, the incidence of *Salmonella pullorum* and *S. gallinarum* among salmonella serotypes isolated from fowls decreased from 70.4% in 1956 to 24.0% in 1963. The figures well illustrate the degree of control which can be exercised in dealing with strictly host-adapted salmonellae. Similar measures are not effective in dealing with non-host-adapted types because of the widespread distribution and serologic diversity of the organisms and the multiple pathways by which they are transmitted.

What part of the marked increase in reported human salmonellosis is due to actual increased incidence and what effect improved reporting has played are questions that are impossible to answer. An indication of the effect of the salmonella surveillance program of the National Communicable Disease Center is the rapid rise in reported infections since the program began in 1962. Nevertheless, there undoubtedly has been a real increase as well, as evidenced by reports such as that of MacCready et al. (2) in which it was noted that salmonella isolations in Massachusetts increased nearly sevenfold between 1950 and 1955; whereas the number of specimens from which the recoveries were made increased less than twofold.

While the situation in regard to reporting undoubtedly has improved, infections still are grossly under-reported. If the reported rates of general outbreaks of salmonellosis in the United States are compared with those of England and Wales, the gradual improvement in reporting in this country is apparent, yet United States rates fall far short of the rates reported in Britain. Nevertheless, the British workers are convinced that the notified cases of salmonellosis in the United Kingdom are only a fraction of those that actually occur. It is of interest, too, that under an identical system of reporting employed over a 10-year period, the British rates are falling, perhaps due to intensive efforts to eliminate salmonellae from foods. During the same period, rates in the United States have doubled, but this is due, in part, to increased interest in the problem and improved reporting mechanisms.

In the past, it was generally assumed that persons affected with acute gastroenteritis due to salmonella infections excreted the organisms in large numbers while symptomatic and that the causative agents persisted in the intestinal tract only for a short period after disappearance of symptoms. Likewise, little was known of the symptom-less excreter without history of preceding intestinal infection. Today, it is known that a certain percentage of convalescents, as well as persons without history of overt infection, may excrete salmonellae for long periods. I do not mean to imply that such persons are permanent carriers, such as those who excrete *S. typhi* due to chronic infection of the biliary or urinary systems. Permanent carriers of non-host-adapted serotypes do exist but these are very exceptional. Salmonella serotypes have been isolated from gallstones following cho-

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lecystectomy, just as *S. typhi* often is recovered under similar circumstances. There is no reason to believe that the persons from whom such organisms were isolated are not permanent carriers. It should be emphasized, however, that such circumstances are exceptional and probably result from only a small percentage of patients who develop salmonella bacteremia.

On the contrary, long-term temporary carriage occurs frequently. Our knowledge of carriers has evolved with the widespread use of adequate enrichment media and closer surveillance of convalescents. One need cite only the experiences of Leeder (3) and Szanton (4) to illustrate the frequency of long-term carriers in children and familial spread of infection through contact with convalescents. McCuigan et al. (5) noted the persistence of the carrier state in young adults, together with intermittency of positive stool cultures. Similar observations on elderly patients were reported by McCall et al. (1960) who described an extreme case in which an adult excreted *S. bovis-morbificans* for 6 years. Many asymptomatic carriers have been recognized through fecal examinations of food handlers. Galton and Hardy (6) noted that 63% of the salmonellae isolated in Florida came from asymptomatic persons, the majority of whom were food handlers. The carrier rate in the population of Greater Manhattan was estimated by Saphra and Winter (7) to be 2/1000, and a similar figure (2.4/1000) was given by Savage (8) for the United Kingdom. On the contrary, Schaeffer (9) estimated the carrier rate in West Germany to be 50/1000 and Sharma et al. (10) and Bokkenheuser and Richardson (11) arrived at similar figures for the population of Mathura, India, and for Bantu food handlers, respectively. Bokkenheuser and Richardson (12), in the examination of Bantu school children, found 6.5% positive on single examinations, but as the result of seven examinations during a 12-month period, a cumulative total of 35.5% of the children were positive on at least one examination. This well illustrates that in the average carrier, the organisms persist for only a short period and that rates estimated from single examinations by no means reflect the percentage of the asymptomatic population which excretes salmonellae at one time or another during a period of one year. Also, it illustrates very well the difficulties encountered in attempting to screen food handlers through fecal examinations. The detection of salmonella carriers among food handlers by the methods used to detect typhoid carriers is a rather futile process. It would be necessary to perform repeated examinations at short intervals, and even with such precautions it would be most unlikely that sporadic excretion of salmonellae would be detected before an opportunity for contamination of food occurred. Although longitudinal studies of salmonella excretion by professional food handlers are not available, it seems most likely that excreters among persons habitually handling meats and carcasses would be more numerous than among the general population. The statement of Felsenfeld and Young (13) that 55.7% of the salmonellosis outbreaks which they studied were caused by human carriers, indicates the role of that source of contamination, but one must remember that it is not always easy to distinguish culprits from victims in retrospective investigations. Following an epidemic, the excretion of salmonellae by apparently normal food handlers may be the result of the outbreak and not the cause, since many persons who contract the infections remain asymptomatic.

Dogs

During the past two decades, numerous reports have appeared regarding the prevalence of salmonellae in domestic pets (14,15). Wolff et al. (15) found 16 salmonella serotypes in 18% of 100 dogs in Michigan. The source of infection was believed to be rejected eggs being fed to these animals. In Florida (16), 15% of 1,626 normal household dogs and 12% of 73 normal cats were harboring salmonellae. Community surveys of the owners and families of these normal dogs provided no secure evidence on which to judge the epidemiological significance to man of salmonella infections in the normal family dog. The prevalence of infection among greyhounds in kennels was much higher. During a 9-month observation period, salmonellae were recovered from 67.7% of 572 dogs cultured. The annual minimal attack rates, based on monthly cultural findings, were in excess of 600 infections per 100 dogs per annum or an infection every 2 months (17). Kennel sickness reportedly occurred among the human handlers of these dogs.

In spite of the rather high prevalence of salmonella infection in dogs, only a relatively few reports have incriminated these animals as the source of human infections. Magnusson (18), in Sweden, isolated *S. abortus-canis* from three ill patients and from his dog. Kauffmann and Henningsen (19) isolated *S. glostrup* from members of one family during an outbreak of gastroenteritis and from their dog ill at the same time. Nevertheless, dogs are still a potentially important source of salmonella infection in man. Evidence has been reported to indicate that dogs may be victims rather than culprits and acquire their infection from their human contacts. In Florida, *S. saint-paul* was isolated from a 3-month-old infant; subsequently, there was an outbreak in the family due to this type. Tests on the infant remained positive for 5 months. Two dogs, one with severe diarrhea, both acquired by the family after the child became ill, yielded *S. saint-paul* by rectal swab culture.

Pet Birds

Usually, one does not consider poultry in the category of domestic pets, but during the past 5 years, chicks and ducklings given to young children at Easter have been incriminated as the source of salmonella infections in many of these children. In attempting to find the source of more than 20 *S. enteritidis* infections in children after Easter 1960, McCroan found that each of them had received an Easter chick. Unfortunately, none of the chicks were still available for examination. In 1962, a similar increase in *S. tennesee* infections in children after Easter prompted immediate search for chicks. Six clinical cases and two asymptomatic infections were traced to Easter chicks from one hatchery. Seven of the birds were infected with *S. tennessee* and one with *S. typhi-murium*. Later, Hines in North Carolina reported the isolation of *S. typhi-murium* from a severely ill 13-month-old child and from a pet duckling the child had played with one week prior to onset of illness. Some state health departments now issue warnings in the press just prior to Easter regarding the potential hazard of such pets.

Many household and pet parakeets, canaries and other exotic species of birds have been implicated. Although their role in the transmission of salmo-

nellosis to man is not clearly defined, several *S. typhi-murium* infections in infants and young children have been traced to parakeets in the home (21,22). These reports illustrate the need for epidemiological investigation of all reported cases of salmonellosis. They further point out a hazard that may occur in many homes with pet birds. This potential source of infection should also be considered where pet birds are kept in public eating places and appropriate steps should be taken to prevent human exposure.

Pet Turtles

Cold-blooded animals long have been known to harbor salmonellae. Among the first to call attention to their presence in these animals were Hinshaw and McNeil (23,24) and McNeil and Hinshaw (25) who isolated a number of serotypes from snakes, turtles and lizards. Recently, attention has been directed toward turtles because of the close association of pet turtles and man. Boycott has studied excretion of salmonellae by the genus *Testudo* over a period of years. Together with Taylor and Douglas (27), he reported the isolation of one or more serotypes from 10 of 11 animals examined. Later, Boycott (26) found salmonellae in very high percentages of the coprophagic species *Testudo graeca* and *Testudo hermanni*. The organisms occurred both in animals kept in captivity and in their native habitat. Periods of excretion in some animals ranged from 3 to 9 years. The number of infected individuals and the number of organisms excreted by the individual animal tended to decrease during the period of observation. Hirsch and Hirsch (28) also noted the prevalence of salmonellae in *T. graeca* in their native habitat. Bovre and Sandbu (29) isolated salmonellae from 80% of tortoises imported into Norway.

Thomas (30) described an apparent case of transmission of a paracolon bacillus from a tortoise to a child. The organism involved was the same as a salmonella-like strain described by Boycott, Taylor, and Douglas (27) and is identical with *S. sofia* of Wesselinoff and Dimow (31). Over the past few years, a number of documented cases of turtle-associated salmonellosis have been reported in the United States. Over 60 instances have been recorded in which the same salmonella serotype was recovered from the patient and the pet turtle involved. An additional group of cases numbering over 50 have been reported in which the turtle was implicated, but definite proof was lacking for one reason or another. Of particular interest is the report of Williams and Helsden (32), who investigated a case of *S. panama* infection which apparently was contracted from an infected turtle kept in the home. Examination of a number of additional turtles in the vicinity resulted in the isolation of 16 salmonella serotypes. Investigation of human infections due to these turtle associated serotypes revealed three other instances in which turtles carrying serotypes corresponding to those found in individual infections and familial outbreaks were present in the homes. In an additional nine instances, turtle contact was established, but the turtles were not available for examination. Like Boycott, Williams and Helsdon noted that the longer turtles had been kept in captivity, the less likely they were to excrete salmonellae. They related the infections to the conditions under which breeder turtles were kept and the environments in which eggs were hatched and to which the young turtles were exposed. In most instances, the turtle ponds in which breeders are kept are extremely insanitary and the animals are fed refuse, carcasses of dead animals, and commercial animal by-products, all of which are likely to contain a variety of salmonella serotypes. Williams and Helsdon advocate that breeder turtles be fed on a diet free of salmonellae and that eggs be hatched in a sanitary manner and the young protected from contamination. Their recommendation of holding turtles for a specified time before marketing might improve the situation but should be viewed in light of Boycott's experiences on long-term excretion of the microorganisms in other turtle species. Undoubtedly, turtles recently have played a significant role in transmission of salmonellosis, and measures should be taken to control this method of spread.

REFERENCES

1. U.S. Department of Health, Education, and Welfare, Public Health Service, National Communicable Disease Center: Salmonella Surveillance Report Annual Summary, Atlanta, Georgia, 1964.
2. MacCready, R. A., J. P. Reardon and I. Saphra, New Engl. J. Med. *256*, 1121–1128 (1957).
3. Leeder, F. S., Ann. N.Y. Acad. Sci. *66*, 54–60 (1956).
4. Szanton, V. L., Pediatrics *20*, 794–808 (1957).
5. McGuigan, J. E., W. C. Berry and P. R. Carlquist, U.S. Armed Forces Med. J. *11*, 1288–1293 (1960).
6. Galton, M. M., and A. V. Hardy, Public Health Lab. *11*, 88–93 (1953).
7. Saphra, I., and J. W. Winter, New Engl. J. Med. *256*, 1128–1134 (1957).
8. Savage, W., Brit. Med. J. *11*, 317–321 (1956).
9. Schaeffer, W., Zbl. Bakt. (Orig.) *172*, 272–281 (1956).
10. Sharma, S. P., V. K. Sharma and C. M. Singh, Indian J. Med. Res. *51*, 404–406, (1963).
11. Bokkenheuser, V., and N. J. Richardson, S. African Med. J. *33*, 784–786 (1959).
12. Bokkenheuser, V., and N. J. Richardson, J. Hyg. *58*, 109–117 (1960).
13. Felsenfeld, O., and V. M. Young, Am. J. Trop. Med. Hyg. *29*, 483–491 (1949).
14. Galton, M. M., J. E. Scatterday and A. V. Hardy, J. Infect. Dis. *91*, 1–5 (1952).
15. Wolff, A. H., N. D. Henderson and G. L. McCallum, Amer. J. Public Health *38*, 403–408 (1948).
16. Mackel, D. C., M. M. Galton, H. Gray and A. V. Hardy, J. Infect. Dis. *91*, 15–18 (1952).
17. Stucker, C. L., M. M. Galton, J. Cowdery and A. V. Hardy, Ibid. *91*, 6–18 (1952).
18. Magnusson, K. E., Ztschr. f. Hyg. u. Infektionskr. *121*, 136–138 (1938).
19. Kauffmann, F., and E. J. Henningsen, Acta Path. Microbiol. Scand. *16*, 99–102 (1939).
20. McCroan, J. E., T. W. McKinley, A. Brim and C. H. Ramsey, Public Health Rep. *78*, 1073–1080 (1963).
21. Kaye, D., H. R. Shinefield and E. W. Hook, New Engl. J. Med. *264*, 868–869 (1961).
22. U.S. Department of Health, Education, and Welfare, Public Health Service, National Communicable Disease Center: Salmonella Surveillance Report Numbers 16 and 17, Atlanta, Georgia, 1963.
23. Hinshaw, W. R., and E. McNeil, Cornell Vet. *34*, 248–254 (1944).
24. Hinshaw, W. R., and E. McNeil, Amer. J. Vet. Res. *6*, 62–63 (1945).
25. McNeil, E., and W. R. Hinshaw, Ibid. *7*, 62–63 (1946).
26. Boycott, J. A., Science *137*, 761–762 (1962).
27. Boycott, J. A., J. Taylor and S. H. Douglas, J. Path. Bact. *65*, 401–411 (1953).
28. Hirsch, W., and R. Shapiro-Hirsch, Harefuah *46*, 237–238 (1954).
29. Bovre, K., and P. Sandbu, Acta Path. Microbiol. Scand. *46*, 339–342 (1959).
30. Thomas, M. E. M., Monthly Bull. Minist. Health (Lond.,) *16*, 29–31 (1957).
31. Wesselinoff, W., and I. Dimow, Zbl. Bakt. (Orig.) *187*, 263–265 (1962).
32. Williams, L. P., Jr., and H. L. Helsdon, J. Am. Med. Assoc. *192*, 347–351 (1965).

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