Moreover, noise would be expected to affect f_bE_s only at the lowest observed f_bE_s value (that is, in proximity to f_oE) so that an effect would be noticeable only in summer daytime near sunspot maximum.

Broadcast interference is minor at both these South Pacific stations. A survey (*7*) of records to establish whether scaled values were affected by interference (values designated ES), based on a comparison of data at times of high and low f_bE_s, revealed that interference did not measurably influence f_bE_s estimates. The ionogram parameter f_{min}, scaled from the lowest frequency reflection from the ionosphere, is interference-limited at night at both stations. An examination of f_{min} data for morning and evening periods shows that since 1957 (when published values commence) there have been no increases in the mean f_{min} with almost constant values of 1.0 MHz for Rarotonga and 1.5 MHz for Christchurch. In addition, there have been no trends in daytime (1000 to 1400) winter values with the mean monthly f_{min} fluctuating in the range 1.18 to 2.00 MHz for Rarotonga and 1.36 to 2.20 MHz for Christchurch.

In summary, it is difficult to relate the observed marked trends in f_oE_s and f_bE_s parameters to any known changes in ionosonde or observing conditions. The identification of any such changes would have important consequences for long-term ionospheric studies based on the use of ionosondes. Alternatively, an ionospheric mechanism might be sought in terms of, for example, a temperature structure changing over many years reflected in changes in tidal wind characteristics and hence in E_s formation. It is clearly important to establish whether the trends reported here are a feature of Northern Hemisphere stations.

W. J. BAGGALEY

Department of Physics,
University of Canterbury,
Christchurch, New Zealand

References and Notes

1. J. D. Whitehead, *J. Atmos. Terr. Phys.* **36**, 861 (1961).
2. ———, *Rev. Geophys. Space Phys.* **8**, 65 (1970).
3. C. A. Reddy and S. Matsushita, *J. Geophys. Res.* **73**, 1641 (1968); W. J. Baggaley, *ibid.* **76**, 4674 (1971).
4. V. Bumba, *Bull. Astron. Inst. Czech.* **6**, 141 (1955).
5. C. D. Ellyett, *J. Geophys. Res.* **82**, 1455 (1977); B. A. Lindblad, *Nature (London)* **273**, 732 (1978).
6. *Handbook of Ionogram Interpretation and Reduction* (International Union of Radio Science, Paris, ed. 2, 1972).
7. A. Stanbury, *Survey of Ionosonde Sensitivity and Calibration* (Geophysical Observatory, Department of Scientific and Industrial Research, Christchurch, New Zealand, 1983); personal communication.

2 March 1984; accepted 7 May 1984

Animal-to-Man Transmission of Antimicrobial-Resistant *Salmonella:* Investigations of U.S. Outbreaks, 1971–1983

Abstract. *The importance and origin of antimicrobial-resistant* Salmonella *infections were examined in 52 outbreaks investigated by the Centers for Disease Control between 1971 and 1983. The case fatality rate was higher for patients infected with antimicrobial-resistant* Salmonella *(4.2 percent) than for those with antimicrobial-sensitive infections (0.2 percent). In the 38 outbreaks with identified sources, food animals were the source of 11 (69 percent) of 16 resistant and 6 (46 percent) of 13 sensitive outbreak strains.*

Antimicrobial-resistant *Salmonella* have accounted for a steadily increasing proportion of human *Salmonella* infections and now represent approximately 20 to 30 percent of salmonellae isolated from humans (*1, 2*). The importance of antimicrobial resistance in *Salmonella* has been questioned because most *Salmonella* infections do not require antimicrobial therapy. In addition, it has been suggested that antimicrobial-resistant *Salmonella* may be less virulent than antimicrobial-sensitive strains (*2–4*). The source of these resistant bacteria is also controversial. Some have argued that the administration of antimicrobials to animals—this use accounts for about half the antimicrobials produced yearly in the United States—selects for antimicrobial-resistant bacteria, has increased their prevalence in the food chain, and has been the principal cause of the increased incidence of drug-resistant strains in humans. However, others have argued that multiple-drug-resistant bacteria derive mainly from too frequent prescribing of antimicrobials for humans, particularly in hospitals, and that the human and animal pools of antimicrobial-resistant *Salmonella* overlap infrequently and transiently (*5, 6*).

The major obstacle to determining whether antimicrobial-resistant bacteria often arise from food-animal sources and present an important threat to human health has been the difficulty in tracing all the postulated steps from farm practice to human disease. Individual events in the complicated sequence have been documented, such as the selection for and persistence of resistant bacteria in food-producing animals resulting from the use of subtherapeutic doses of antimicrobials (*7–11*), the frequent presence of resistant *Salmonella* in products of animal origin (*12, 13*), the transmission of resistant microorganisms to humans (*6, 14*), and human disease resulting from multiply resistant bacteria (*1*). However, outlining all these steps in a sequence is rarely possible and does not indicate the relative frequency with which resistant bacteria arise from animal and human populations (*15*).

It may not be possible to determine exactly how often animal-to-man transmission of resistant enteric pathogens occurs (*16, 17*). However, it is possible to determine whether animal-to-man transmission of antimicrobial-resistant *Salmonella* has been demonstrated in an important fraction of *Salmonella* outbreaks investigated in the United States. We reviewed epidemiologic and laboratory data of all Centers for Disease Control (CDC) investigations of nontyphoidal *Salmonella* outbreaks between January 1971 and December 1983.

Summary reports were available and complete for 52 of 55 *Salmonella* outbreaks investigated by CDC personnel in the 12-year period. We could classify these outbreaks into three groups: (i) 34 that had occurred in the community; (ii) 12 that had occurred wholly within a hospital or institution; and (iii) 6 that had occurred both in the community and in a hospital (Table 1).

In only 5 of the 52 outbreaks was the antimicrobial susceptibility of the epidemic strain known at the time that the CDC was requested by state health departments to participate in these investigations. At the outset of CDC investigations, sources or modes of spread of outbreak strains were suspected in 11 of the 18 outbreaks traced to animals, in 9 of the 11 outbreaks originating from a common source, in 3 of the 4 outbreaks of another source, in 3 of the 5 outbreaks spread person-to-person in hospitals, and in 5 of the 13 outbreaks in which the source could not finally be determined.

Seventeen of the 52 outbreaks involved antimicrobial-resistant organisms and affected 312 persons, 13 of whom (4.2 percent) died from salmonellosis. Eight deaths in elderly persons were from community-acquired organisms in three outbreaks traced to raw milk and beef obtained from specific dairies and a beef herd (*18, 19*); the 5 deaths in 18 affected infants occurred in a single hospital nursery outbreak caused by multiply resistant *S. typhimurium* of unknown source. In contrast to fatalities caused by antimicrobial-resistant organisms, the 19 outbreaks caused by antimicrobial-sensi-

Table 1. Outbreaks of salmonellosis investigated by the CDC between 1971 and 1983. Numbers in parentheses represent the number of outbreaks involving *Salmonella* resistant to two or more antimicrobials.

Source of outbreaks	Number of outbreaks			Total
	Community	Nosocomial	Both	
Food animals or their products	12 (7)	2 (1)	4 (3)	18 (11)
Common source (food-handler, restaurant, kitchen)	10 (1)	1 (0)	0	11 (1)
Other source (food, drug, lab animal, hospital equipment)	2 (0)	2 (1)	0	4 (1)
Person-to-person spread	0	5 (2)	0	5 (2)
Unknown or unproven	10 (0)	2 (1)	2 (1)	14 (2)
Total	34 (8)	12 (5)	6 (4)	52 (17)

tive *Salmonella* resulted in only 4 (0.2 percent) fatalities in 1912 ill persons. In the 16 outbreaks caused by *Salmonella* of unspecified antimicrobial resistance, 4 (0.3 percent) of 1429 ill persons died.

In the 52 outbreaks, a specific source or mode of transmission was identified for 38 (73 percent). In those 38 outbreaks, multiply resistant salmonellae were involved in 8 (33 percent) of the 24 community-based outbreaks, in 4 (40 percent) of the 10 nosocomial outbreaks, and in 3 (75 percent) of the 4 outbreaks which occurred both in community and hospital (Table 1). Epidemiologic investigation implicated food-producing animals as the source of 18 (47 percent) of the 38 outbreaks in which a source was identified (Table 2); food animals were incriminated in 11 (69 percent) of 16 outbreaks involving antimicrobial-resistant *Salmonella*, in 6 (38 percent) of 16 outbreaks caused by antimicrobial-sensitive *Salmonella*, and in 1 (11 percent) of 9 outbreaks caused by *Salmonella* of unspecified antimicrobial susceptibility.

The antimicrobial-resistant organisms were acquired by consuming contaminated foods such as raw milk or undercooked beef (Table 2), or by complex pathways of foodborne and person-to-person transmission. An example of such complex transmission was found in the investigation of outbreak 9 (Table 2): multiply resistant *S. heidelberg* was transmitted by direct contact from ill calves to a pregnant woman, who subsequently transmitted the infection at birth to her infant, and then further transmission occurred to other infants in a hospital nursery (*20*). Similar events occurred in outbreaks 12 and 18 (Table 2), in which nosocomial infections with multiply resistant *Salmonella* were shown to have ultimately come from food animals (*18, 19*). In addition to the 18 outbreaks traced to animals, investigators suspected 5 of the 14 outbreaks of unproven origin to have originated from beef cattle, poultry, or eggs.

The data presented here have unavoidable limitations. These outbreaks were investigated by the CDC at the request of state health departments and therefore are not a random sample of all *Salmonella* outbreaks. Because in over 90 percent of the outbreaks the antimicrobial susceptibility of organisms was not known at the beginning of the CDC investigations, bias in investigating outbreaks of antimicrobial-resistant *Salmonella* is probably not a major confounding variable. However, sources of 30 of the 52 outbreaks were suspected at the start of CDC investigations (although not proven in five instances): thus, a bias to investigate *Salmonella* of animal origin may confound these data. Yet, even if this is so, of the 22 outbreaks of unsuspected and unknown origin at time of investigation, 7 (57 percent) of the 13 identified sources were traced back to animals. Further, 5 of the 14 outbreaks of unproven origin were strongly suspected to have arisen from animal populations. Finally, the frequency of animal-associated outbreaks in this study is similar to that found in a study in which sources of 651 *Salmonella* outbreaks in the United States that were reported to the CDC between 1963 and 1975 were examined (*24*).

Table 2. Outbreaks investigated by the CDC with documented transmission of *Salmonella* from food animals to man, 1971 through 1983.

Outbreak	Year	Location	Serotype	Resistence*	Animal source	Comment and technique‡	Reference
1	1971	Maine	*thompson*	S	Poultry†	Feed with antibiotics given once; As	
2	1972	Montana	*typhimurium*	R	Cattle†	Five farm families; As, Ph, Tn	
3	1972	Wisconsin, Minnesota	*typhimurium*	U	Cattle	Raw beef consumption; Ph, Tn	(*20*)
4	1972	Alaska	*agona*	S	Poultry	Peruvian fish-meal suspected	(*20*)
5	1973	Alabama	*typhimurium*	R	Calves	Same organism in feed; As	
6	1973	Maine	*typhimurium*	R	Eggs	Same organism at egg farm; As, Ph	
7	1974	Washington	*typhimurium*	R	Dairy†	? Contaminated feed; As	
8	1975	Maryland	*newport*	R	Cattle†	Beef from Colorado; As, Ph	(*14*)
9	1976	Connecticut	*heidelberg*	R	Calves†	Secondary nosocomial spread; As, Pl	(*21*)
10	1976	Missouri	*thompson*	S	Cattle	Nosocomial outbreak traced to raw beef; As	
11	1977	Kentucky	*typhimurium*	R	Dairy†	Raw milk consumption; As, Ph	(*20*)
12	1979	United States	*dublin*	R	Dairies†	Raw milk consumption; As	(*18*)
13	1980	Montana	*typhimurium*	R	Dairy†	Raw milk consumption; As, Ph	(*20*)
14	1981	Pennsylvania, New Jersey	*typhimurium* *newport*	S S	Cattle	Precooked roast beef; As, Ph, Pl	(*22*)
15	1982	District of Columbia	*typhimurium*	S	Cattle†	Beef short ribs; As	
16	1982	Wyoming	*typhimurium*	S	Eggs†	Homemade ice cream; As, Pl	
17	1983	Arizona	*typhimurium*	R	Dairy†	Raw milk consumption; As, Pl	(*19*)
18	1983	Minnesota, South Dakota	*newport*	R	Cattle	Cattle fed subtherapeutic antibiotics in feed; As, Pl	(*19*)

*R, *Salmonella* resistant to two or more antimicrobials; S, *Salmonella* susceptible to all antimicrobials tested; and U, *Salmonella* of unspecified antimicrobial susceptibility. †Animal source suspected at the outset of the CDC investigation. ‡Techniques used to characterize organisms: As, antimicrobial susceptibility; Ph, phage lysis pattern; Tn, tartrate negativity; and Pl, plasmid profile analysis (*23*).

These investigations found that in over two-thirds of U.S. outbreaks of multiple-drug-resistant *Salmonella* infections that had a defined source, such bacteria came from food animal populations. It is known that *Salmonella* are commonly transmitted in food animal products, and our analysis shows that multiply resistant *Salmonella* are also frequently transmitted from animals to man. In fact, animal origins were discovered more commonly in outbreaks involving antimicrobial-resistant *Salmonella* than in outbreaks involving antimicrobial-sensitive strains. Thus, it appears to us that animal-to-man transmission of resistant *Salmonella* is not a rare event.

The case fatality rate for patients with identified infections with multiply resistant *Salmonella* was 4.2 percent, 21 times higher than the case fatality rate associated with antimicrobial-sensitive *Salmonella* infections. The explanation for this difference in fatality rates is not known. The fatalities in these investigations were among the old and the very young. Patients at the extremes of age may be more likely to die of salmonellosis, and these patients may have underlying conditions that would lead to antimicrobial or other drug therapy which could complicate or predispose to infection with resistant bacteria. In any case, antimicrobial-resistant *Salmonella* did not appear to be less virulent than antimicrobial-sensitive strains, and the public health significance of multiply resistant *Salmonella* should not be minimized.

We conclude that antimicrobial-resistant enteric bacteria frequently arise from food animals and can cause serious infections in humans.

SCOTT D. HOLMBERG
JOY G. WELLS
MITCHELL L. COHEN

Enteric Diseases Branch,
Division of Bacterial Diseases,
Centers for Disease Control,
Atlanta, Georgia 30333

References and Notes

1. R. Ryder *et al.*, *J. Infect. Dis.* **142**, 485 (1980).
2. L. Riley, personal communication.
3. H. Smith and J. Tucker, *J. Hyg.* **83**, 305 (1979).
4. A. Linton, K. Howe, P. Bennett, M. Richmond, E. Whiteside. *J. Appl. Bacteriol.* **43**, 465 (1977).
5. C. Cherubin, *Rev. Infect. Dis.* **3**, 1105 (1981).
6. H. Neu, C. Cherubin, E. Longo, B. Flouton, J. Winter, *J. Infect. Dis.* **132**, 617 (1975).
7. R. Williams, L. Rollins, D. Pocurull, M. Selwyn, H. Mercer, *Antimicrob. Agents Chemother.* **14**, 710 (1978).
8. D. Siegel, W. Huber, F. Enloe, *ibid.* **6**, 697 (1974).
9. H. Mercer, S. Gaines, S. Wilson, J. Bennett, *Appl. Microbiol.* **22**, 700 (1971).
10. J. Garside, R. Gordon, J. Tucker, *Res. Vet. Sci.* **1**, 184 (1960).
11. S. Levy, G. Fitzgerald, A. Macone, *Nature (London)* **260**, 40 (1976).
12. K. Howe, A. Linton, A. Osborne, *J. Appl. Bacteriol.* **41**, 37 (1976).
13. M. Weissman and J. Carpenter, *Appl. Microbiol.* **17**, 899 (1967).
14. R. Fontaine *et al.*, *Am. J. Epidemiol.* **107**, 36 (1978).
15. T. O'Brien *et al.*, *N. Engl. J. Med.* **307**, 1 (1982).
16. P. Gardner, *J. Infect. Dis.* **138**, 101 (1978).
17. National Research Council, "Study of human health effects of subtherapeutic antibiotic use in animal feeds" (National Academy of Sciences, Washington, D.C., 1980).
18. D. Taylor, J. Bied, J. Munro, R. Feldman, *J. Infect. Dis.* **146**, 322 (1982).
19. C. Tacket, L. Dominguez, M. Cohen, paper presented at the 23rd Interscience Conference on Antimicrobial Agents and Chemotherapy, Las Vegas, 24 to 26 October 1983, Abstr. 356; S. Holmberg, M. Osterholm, K. Senger, paper presented at the 23rd Interscience Conference on Antimicrobial Agents and Chemotherapy, Las Vegas, 24 to 26 October 1983, Abstr. 1002.
20. R. Lyons *et al.*, *J. Am. Med. Assoc.* **243**, 546 (1980).
21. Centers for Disease Control, *Morb. Mortal. Weekly Rev.* **21**, 411 (1972); *ibid.* **22**, 29 (1973); *ibid.* **26**, 239 (1977); *ibid.* **30**, 211 (1981).
22. L. Riley, G. DiFerdinando, Jr., T. DeMelfi, M. Cohen, *J. Infect. Dis.* **148**, 12 (1983).
23. S. Holmberg, I. Wachsmuth, F. Hickman-Brenner, M. Cohen, *J. Clin. Microbiol.* **19**, 100 (1984).
24. M. Cohen and P. Blake, *J. Food Protect.* **40**, 798 (1977).

6 February 1984; accepted 12 June 1984

Favositid Tabulates: Evidence for Poriferan Affinity

Abstract. *Calcitic pseudomorphs of desma-like siliceous spicules found in the calcareous skeleton of a Devonian thamnoporid support the proposal of poriferan origin of at least some favositid Tabulata. These favositids arose from a group of Ordovician lithistid demosponges that adapted to toxic calcium excess in shallow, tropical marine environments by developing calicoblasts within the pinacoderm, supplementing their primary siliceous spicular skeletons with basally secreted calcium carbonate. They are tentatively recognized as an order of the subclass Sclerospongiae (class Demospongiae).*

Reports of peculiar modern sponges with mixed siliceous-calcareous skeletons called sclerosponges or coralline sponges (*1–4*) renewed an old discussion (*5*) on the possible poriferan affinities of such enigmatic marine fossils as chaetetids, stromatoporoids, and favositids. New observations stimulated by these discoveries have provided conclusive evidence for the sclerosponge nature of some Paleozoic (*6*) and Mesozoic (*7*) chaetetids. The poriferan origin of stromatoporoids and favositids is still a matter of considerable controversy. Stromatoporoids in a broad sense are apparently a systematic hotchpotch representing such distant groups as cyanobacteria (*8*), sponges (*9*), and coelenterates (*10*). For the favositids, however, poriferan (*11*) and zoantharian (*12*) models have been discussed, but for neither model is there satisfactorily conclusive evidence.

The finding of distinct spicules within the skeletal elements of a Devonian specimen (*13*) of *Thamnopora reticulata* (de Blainville) directly supports suggestions of the poriferan nature of some favositids. The specimen comes from Middle Devonian silty marls correlating to lithological complex XVII of the Skalska Series exposed by trenching near Skały village in the eastern part of the Holy Cross Mountains, Poland (*14*). At this locality *Th. reticulata* is associated with abundant solitary and colonial rugose corals and trilobites of Givetian age. An inner shelf environment is envisaged, characterized by a higher influx of terrigenous material.

The internal morphology of the *Th. reticulata* specimen (Fig. 1, A and B) is similar to other thamnoporids. The branchlike calcitic skeleton consists of upwardly radiating tubes, subangular to subcircular in cross section, connected with rare tunnel-like mural pores. Poorly developed septal spines project into the lumen of some tubes partitioned occasionally by tabulae. The lumen of the tubes and the thickness of tube walls (smaller in the branch center), increase considerably near the branch surface. Tube openings are situated normal to the surface; their outermost parts are obscured. The outer interskeletal spaces are filled with porous, weakly translucent, reddish, iron-rich marly sediment; the inner interskeletal spaces are occupied by sparry calcite. A similar pattern of skeletal organization characterizes other arborescent and massive favositids (*15*); the main differences being the variability of the tube shape and diameter, the number and arrangement of tabulae and mural pores, and the presence or absence of septal spines and ridges (squamulae).

Traces of pseudomorphosed (calcified) primary siliceous spicules (Fig. 1C) occur in a few places of the calcareous skeleton and are limited to its outermost zone, close to the contact with the surrounding reddish sediment. The intramural location of the spicules leaves no doubt as to their primary association with the walls of the tubes. Although examination of spicules in thin section makes a precise determination of their shape difficult, they can easily be identified as irregular megascleres generally called desmas, characteristic for assigning modern and fossil siliceous sponges to the order Lithistida (class Demospongiae) (*16*). Two types of megascleres have been recognized: (i) smooth, tetractine-like desmas with forked rays and an