

AN OUTBREAK OF FOOD POISONING DUE TO *SALMONELLA* *TYPHI-MURIUM* IN A GENERAL HOSPITAL

I. Epidemiological features

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SUMMARY

An explosive outbreak of food-poisoning caused by *Salmonella typhi-murium* which occurred in a large teaching hospital in Salvador, Bahia, is described. A total of 213 cases were recorded between December 11 and 13, 1961, among 253 hospital employees who were exposed to the risk of infection. The fact that no primary cases were observed among approximately 290 ward-patients was an important hint to establishing the vehicle of infection. Seventeen secondary cases, presumably representing person-to-person spread, were found to have occurred, five of them among patients. Although the clinical manifestations were of moderate or high severity, no deaths were recorded.

Strong epidemiologic evidences is presented that infected beef was the vehicle. It was not possible to determine with certainty if it was an original infection of the animals or a secondary contamination of the meat during slaughtering or preparation. A possible important role of salmonella organisms in underdeveloped areas is suggested and the need of carrying out surveys to determine the distribution of the organism in man and in livestock is emphasized. The importance of rigid enforcement of administrative measures aimed at controlling an explosive hospital outbreak such as this was clearly demonstrated.

INTRODUCTION

There is unpublished clinical and epidemiological evidence that infection by *Salmonella* organism has occurred in Bahia both sporadically and in outbreaks. At the end of 1961 a sharp outbreak of food poisoning due to *Salmonella typhi-murium* took place in a 300 — bed teaching hospital in Salvador, Bahia, which permitted a rather intensive epidemiological investigation.

The epidemic provided an opportunity to demonstrate that even in areas with underdeveloped public health services and devoided of specialized laboratory facilities, efficient control measures can be applied promptly if supported by carefully collected clinical and epidemiological data. For this reason and because a large number of health hospital personnel were involved it was

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considered that the outbreak is of sufficient interest to be recorded.

THE OUTBREAK

On the morning of December 12, 1961 a number of cases of acute gastroenteritis began to be reported by the medical staff among the hospital employees. When the epidemic began the hospital was housing 290 patients distributed in seventeen wards and cared for by a staff of approximately 830 persons. A total of 213 primary cases were found to have occurred among 253 hospital employees and staff who were exposed to the risk of infection. No primary cases were observed among the patients. Most of the cases were of moderate or high degree of severity and 72.8 per cent of them required hospitalization but there were no fatalities.

The number of reported cases piled up so rapidly that it soon became apparent that we were dealing with a sharp outbreak. The possibility of a common source and a single essentially simultaneous exposure was considered from the outset. A rapid spot investigation indicated that all the sick individuals had eaten at the hospital dining room the previous day. It was further observed that no cases had occurred among the hospital patient and no apparent increase in diarrheal disease could be observed among them. Suspicion was focused on food as a probable vehicle, though the diets consumed by the patients had come from the single central kitchen where the meals for the hospital employees had been prepared and only the latter became ill. The hospital water supply was considered as an unlikely source, since the water used by both the hospitalized patients and the hospital employees came from a single reservoir through a common piping system.

Methods

A systematic epidemiological investigation was then immediately initiated. Interview forms were prepared which included a check list of food consumed and informations on the time of onset, the clinical manifestations,

their character, intensity and course as well as data on treatment received by the patients. Complete records were obtained on all the 253 hospital employees who ate one or more meals in the hospital on December 11 and on 84 additional employees who did not eat in the hospital but had close contact with the epidemic cases that were hospitalized. The former are hereafter referred to as primarily exposed persons.

One or more stool cultures were secured from many of these exposed persons. Bacteriological cultures of blood, urine and vomitus as well as a white blood cell count and an agglutination test were performed in a number of non-purposefully selected cases. Methods and results of these laboratory tests are described in more details in a forthcoming paper¹⁸.

Samples for bacteriological examination were taken from left-over prepared and raw food-stuffs. The specimens were ground in a sterile mortar and then inoculated into Kauffmann's medium and streaked on to blood agar plates. From the Kauffmann's medium, subcultures were carried out on to SS agar and Teague's plates which were incubated overnight. Next morning suspected colonies were subcultured on to Costavernin medium³.

Epidemiological features

The epidemic curve is shown in Figure 1. A total of 213 cases occurred within a span of 56 hours, between 2:00 p.m. of December 11 and 8:00 p.m. of December 13 (Fig. 1A). These cases are hereafter referred to as primary cases. It is apparent that 72 percent of these patients became ill on the first 24-hour period and 99 percent within 48-hours. Assuming a single simultaneous exposure to a common source of infection the incubation period could easily be calculated giving a mean value of 19.9 with standard deviation of 9.7 and a median value of 18:0 hours. The average duration of the illness was 6.4 ± 3.8 days and the median 6.2 days. The average durations of the diarrhea and the fever were 4.5 ± 3.5 and 2.6 ± 1.7 days, respectively¹⁴.

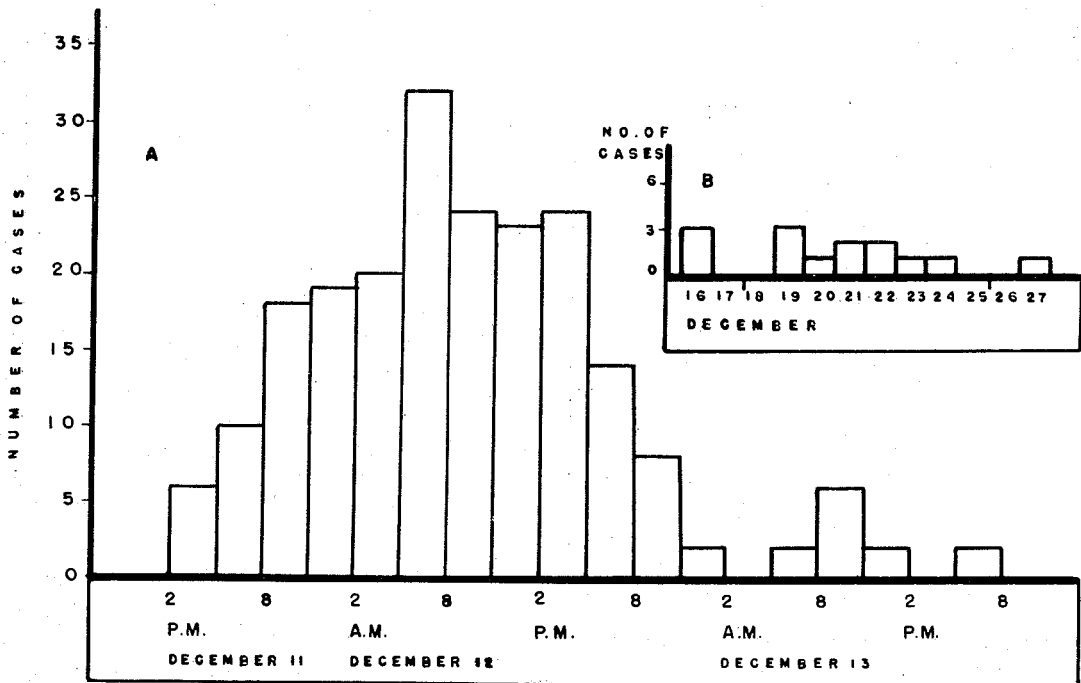


Fig. 1 — Time distribution (in hours) of (A) 252 primary cases and (B) thirteen secondary cases of gastroenteritis due to *Salmonella typhi-murium*.

TABLE I

Attack rates among 253 exposed hospital employees according to the type of meal eaten in the hospital

Time of meal	No. of persons		Attack rate %
	Exposed	Ill	
Noon	196	177	90.3
Evening	29	18	62.1
Noon and evening	17	17	100.0
Other*	10	1	10.0
Total	253	213	84.2

* Other includes snacks of various types in the afternoon and evening periods.

The attack rate among the 253 primarily exposed employees was 84.2 per cent (Table I). Table I demonstrates that the risk of infection as expressed by attack rates varied widely according to the type of meal eaten. The highest attack rate (100.0 per cent) was observed among those employees who ate at noon and the evening in the hospital dining room while the rate among those who ate only at noon (90.3 per cent) was considerably higher than that among the persons who had only the evening meal (62.1 per cent). The attack rate among the employees who had only snacks in the afternoon and the evening periods (10.0 per cent) was the lowest.

The distribution of cases by age and sex is presented in Table II as age and sex-specific attack rates. It can be observed that males tended to exhibit a higher rate than females and this was particularly true among persons in the age groups with the highest rates, namely the youngest and the oldest age groups.

TABLE II

Age and sex-specific primary attack rates among persons exposed to infection

	Sex	Age in years				Total
		17-24	25-34	35-44	45-59	
Exposed persons *	Male	23	44	23	10	100
	Female	28	74	39	12	153
	Total	51	118	62	22	253
Cases	Male	22	35	19	9	85
	Female	25	60	33	10	128
	Total	47	95	52	19	213
Attack rate %	Male	95.6	79.5	82.6	90.0	85.0
	Female	89.3	81.1	84.6	83.3	83.7
	Total	92.2	80.5	83.9	86.4	84.2

* Persons who ate one or more meals at the hospital on December 11.

Primary attack rates by occupational groups were calculated. The nursing staff, the largest exposed hospital group, exhibited an attack rate (81.4 percent) which tended to be lower than the general rate (84.2 percent). The rate among members of the Nutrition Department (95.6 percent) on the other hand, was remarkably higher than the average attack rate while the attack rate among the technical service personnel (90.9 percent) also tended to be high. The remaining occupational groups which included the clerk, administration, laundry and medical staffs exhibited rates which were close to the average primary attack rate.

Thirteen cases that occurred between December 16 and 27 were considered as secondary cases representing person-to-person spread (Fig. 1B), and so were four additional cases with inapparent infection. The later were observed among employees that did not eat in the hospital either on December 11 or on subsequent days. The former included five patients, one staff-physician, one nurse, five auxiliary nurses and one clerk. A secondary attack rate was calculated by considering as exposed to the risk of secondary infection all those individuals who had close contact with the patients or with convalescent cases exclusive of those who had become ill as the result of primary exposure on December 11. A ge-

neral secondary attack rate of 15.9 percent was observed.

A *Salmonella* organism was isolated from stool specimens in a large number of cases (Table III) and was serologically identified

TABLE III

Results of stool cultures in clinical cases resulting from primary exposure to the risk of infection on December 11, 1961

Time after onset of illness	Cases with cultures		
	No.	Pos.	Percent pos.
First day	1	1	—
Second day	30	24	80.0
Third day	15	7	46.7
Fourth day	33	8	24.2
Fifth day	55	12	21.8
Sixt and seven days .	39	8	20.5
Second week	49	11	22.4
Third week	32	2	6.2
Fourth and fifth weeks	39	2	5.1

as *S. typhi-murium* of the fermentation type I of KRISTENSEN et al.⁷. Table III shows that the percentages of cases with positive stool cultures decreased from 80 percent on the third and 24.2 percent on the fourth. Then the relative frequency of cases with positive stool cultures tend to remain at the 20 percent level up to the end of the second week after the onset to the illness. By the third week, however, only 6.2 percent of the cases were still excreting bacteria in their stools and during the fourth and fifth weeks the proportion of cases with positive cultures had decreased to 5.1 percent. These percentages correspond to two cases which were considered as temporary carriers. No bacteria could be recovered from the stools of the two temporary carriers after six weeks from the onset of the illness.

One or more stool cultures were also obtained from most of the individuals who were exposed primarily or secondarily to the risk of infection but did not become ill. However, only seven of these employees submitted specimens within seven days from the start of the outbreak and these cultures yielded negative results. During the second week, cultures from four out of 34 individuals gave positive results. These four employees with inapparent infection were considered as secondary cases since they did not eat in the hospital on December 11 or on subsequent days. All of them had close contact with cases or laboratory specimens therefrom. By the third and fourth weeks stool specimens from these case were again cultured with negative results.

The source and vehicle of infection

The particular characteristics of this outbreak suggested from the outset the possibility of a single simultaneous exposure to infection from a common source. Food was considered the probable vehicle. The fact that no primary cases had occurred among the 290 hospitalized patients in contrast with the high attack rate among the employees, though intriguing, in fact strengthened suspicion against food. To establish the vehicle of infection, a necessary initial step was clearly to find out in what ways the patient's diets has differed in composition from

the meals eaten by the employees, since both had been prepared in a single central kitchen.

It was found that the same persons were concerned with the preparation and handling of food for both the patients and the employees. None of those gave a history of recent diarrheal disease. At the time of this investigation a good standard of general cleanliness was being maintained in the kitchen which was equipped with modern stainless-steel appliances, good dishwashing facilities and working cold rooms. No break in food-handling procedures could be detected.

Diet lists including all food served in the hospital at noon and in the evening on December 11 were compiled. An intensive investigation revealed that most of the patients had received a standard diet consisting of cooked beans, rice, mashed potatoes and ground meat. The meals served to the employees had the same composition, with a single exception — the meat which was used had been of a different batch of beef and was processed and prepared independently.

Thus the evidences which were gathered focused suspicions on the role of the meat as a possible vehicle. The delivery of beef to the hospital was too late on that morning of Monday, December 11. For this reason the dietician on duty decided to use a left-over batch to prepare the patient's diets. The meat was ground and cooked in a steam-pressure pan. Approximately thirty kilograms of beef from a single carcass was delivered at 10 a.m. Part of that meat was used for the preparation of meals for the hospital personnel. As time was short, it is doubtful whether or not the beef was adequately cleaned. The handling of it was not otherwise different from the established procedures. After the meat was cut it was prepared as follows: one large steam-pressure pan was pre-warmed after a certain amount of lard and seasoning were added to it. The cuts of meat were then distributed over the bottom and along the lower third of the walls. The steam valve was half opened and the pan was left unclosed throughout the whole cooking period. When the edges of the meat cuts began to detach,

usually after ten to fifteen minutes, the cuts were mixed with a large wooden spoon and immediately removed from the pan and placed in a stainless-steel container ready to serve.

To determine the specific food that was responsible for the outbreak an analysis was made of attack rates among persons according to the food eaten. In order to have an homogeneous group it was decided to include in that analysis only the employees who had a single meal in the hospital at noon December 11 and hence had presumably a single exposure to the infected food-stuff. Table IV shows that the highest attack rate was observed among the employees who ate either the beef steak, the mashed potatoes or both. The attack rates among those who ate beans, rice and dessert were also high. However, since these items were eaten by most of the employees, the attack rates according to food eaten does not necessarily reflect risk or infection. Table IV also indicates that the attack rates among individuals that did not eat the beef steak was remarkably low as compared with the ge-

neral attack rate. The attack rates for persons who did not eat beans, dessert and other food were as high as those for persons who did eat these food. The differences in attack rates among persons who ate a specified food and those who did not are also indicated in Table IV. A remarkably high positive value for the beef steak is further indicative of a possible role in the transmission. The differences in rates for mashed potatoes and rice are also relatively high although much less so than for the meat. The attack rates were roughly of the same magnitude among persons who ate and among those who did not eat either beans, dessert and other food.

The foregoing evidences strongly point to the beef as the most probable vehicle and suggest that either beans, dessert and the other food had no effect on the risk of infection. However, they do not exclude a possible role of the mashed potatoes and the rice in the transmission of infection.

To evaluate further the influence of each of these items a cross analysis was made

TABLE IV

Influence of food eaten on the rates of illness for 196 persons who ate only at noon in the hospital on December 11

Specified food	Persons who ate specified food				Persons who did not eat specified food				Difference in attack rate %
	Total	Ill	Not ill	Attack rate %	Total	Ill	Not ill	Attack rate %	
One or more items ..	196	177	19	90.3	—	—	—	—	—
Steak (Beef)	185	176	9	95.1	11	1	10	9.1	86.1
Mashed potatoes	167	159	8	95.2	29	18	11	62.1	33.1
Beans	118	110	8	93.2	78	64	11	87.3	5.9
Rice	179	166	13	92.7	17	11	6	64.7	28.0
Dessert	131	124	7	94.7	65	53	12	81.5	13.2
Other *	59	45	14	76.3	137	132	5	96.4	—20.1

* Other food includes bread, chicken, macarroni and ground meat prepared from a different batch of beef in a steam-pressure pan.

and results are shown in Tables V and VI. Table V indicates that those who had eaten both the steak and the mashed potatoes experienced the highest attack rate while the rate for those who ate the meat but did

not eat the mashed potatoes though still high, was lower than the former rate. Eating each of the other items in addition to the meat had no apparent influence on the risk of infection.

TABLE V

Attack rate among persons who ate the beef steak and specified food at noon on December 11

Specified food	No. of persons	No. of cases	Attack rate %
Total	185	176	95.1
Mashed potatoes Yes	161	158	98.1
No	24	18	75.0
Beans	117	109	93.2
No	68	67	99.4
Rice	173	165	95.4
No	12	11	91.7
Dessert	128	123	96.1
No	57	53	93.0
Other *	51	44	86.3
No	134	132	98.5

* For definition see foot-note of table IV.

TABLE VI

Influence of eating mashed potatoes and/or specified food on the rate of illness for persons who had only the noon meal in the Hospital on December 11

Specified food	Ate mashed potatoes		Did not eat mashed potatoes	
	No. of persons	Attack rate %	No. of persons	Attack rate %
Total	167	95.2	29	69.1
Beef	161	98.1	24	75.0
Yes	6	16.7	5	0.0
No				
Beans	107	97.2	11	54.5
Yes	60	91.7	18	66.7
No				
Rice	159	95.0	20	75.0
Yes	8	100.0	9	33.3
No				
Dessert	120	95.8	11	81.8
Yes	47	93.6	18	50.0
No				
Other *	45	88.9	14	35.7
Yes	122	97.5	15	86.7
No				

* For definition see foot-note of Table IV.

Only five individuals had no contact with either the beef steak or the mashed potatoes and none became ill (Table VI). The number of persons that did not eat the beef steak was too small as to allow a satisfactory analysis. Only eleven persons had no contact with beef and did not eat the steak. Among them only one became ill. This one ate mashed potatoes, beans, rice, dessert and the other food.

As pointed out above, the highest rate was observed among the employees who ate both the meat and the mashed potatoes. The relatively low attack rate for those who ate the mashed potatoes but did not eat the meat (Table VI) seems to indicate that the highest risk of infection was associated with the ingestion of the latter.

The attack rates for persons that ate rice or dessert but did not eat mashed potatoes are higher than the rates among those who did not eat either the mashed potatoes and rice or dessert (Table VI). This is probably due to the fact, not brought out in table VI, that most of the people who ate rice or dessert also ate the beef steak while the few persons who did not eat rice or dessert or both did not tend to eat the steak.

The foregoing data seem to definitely point to the beef as the main vehicle of infection in the present outbreak of food poisoning. A possible role of the mashed potatoes cannot be definitely excluded, in spite of the fact, which was pointed out above, that about 290 wardpatients also ate this food and none became ill. It is possible that the mashed potatoes was contaminated at the time of serving by means of a common ladle which might have been used to serve both the meat gravy and the mashed potatoes to some of the employees. Unfortunately no *Salmonella* organisms could be recovered from either the beef or any other food-stuff. Large number of *Proteus*, coliform organisms and *Staphylococcus* colonies were isolated from samples of the refrigerated raw meat which were collected on December 12, the second day of the outbreak.

Control measures

Administrative procedures to control this outbreak included: (a) prompt discharge of all patients but those with severe diseases. After the second day of the outbreak, only 15 patients (5.2 percent) had remained in the hospital; (b) selective assignment of nurses and clerks, chosen among the employees who were not exposed to infection, particularly in the pediatric service and to care for old and debilitated patients; (c) rigid enforcement of rigorous personal hygienic procedures among staff and employees and (d) prompt discontinuance of service in the kitchen. Though most of the clinical cases were promptly hospitalized no strict isolation of patients was attempted.

DISCUSSION

Because it was not possible to determine the path of infection from the reservoir to the vehicle, the precise source of infection in this outbreak must remain a matter of conjecture. The epidemiological data clearly incriminated the beef as the main vehicle. The steak eaten by virtually all the employees who became ill was clearly undercooked and, as experience has established, meats subjected to cooking may not be heated to a point which renders them free of viable organisms. The failure to isolate salmonellae from the food-stuff was probably related to the massive contamination of the meat with *Proteus*, *Staphylococcus* and coliform organisms.

Fresh meat may carry salmonellae as the result of infection in the slaughtered animal or may be contaminated at any stage of its processing and preparation. In this outbreak the latter possibility seems less likely since no carrier could be demonstrated among the hospital's food-handlers and out of numerous batches of beef coming from the same abattoir only one appeared to be contaminated.

In technically developed countries, for which statistics are available most of the food-poisoning outbreaks have been traced to processed and "made-up" meat. Fresh meat has been rarely implicated. In En-

gland and Wales, for instances, in 3100 food-borne outbreaks recorded from 1949 to 1960, 73 percent were associated with meat of which only 1.5 percent were traced to fresh meat^{2, 13, 17}. In the underdeveloped countries, the reverse appears to be case²¹. In these areas, because of the low standard of sanitation, raw meat is expected to be frequently contaminated with pathogenic organisms. However, statistical data for these countries are either not available or very scanty.

Salmonella typhi-murium, the organism responsible for this outbreak, probably due to the ubiquity of its distribution among animals and man^{5, 11}, is the serotype most commonly found in outbreaks of gastroenteritis^{6, 10, 11, 17}. In England and Wales it is responsible for approximately two thirds of the recorded outbreaks^{11, 13, 17}. It has been shown that its prevalence in livestock parallels its frequency in man⁷. Its main natural reservoir is undetermined, however. Fowl, poultry and pork have been considered the major sources^{11, 17} but cattle, though probably less common as a reservoir than pigs and birds, are nonetheless an important source of infection; the meat and milk thereof being important vehicles^{1, 2, 12, 13}. *Salmonella dublin* has been the most common organisms in cattle's infection^{11, 17}. In recent years, however, evidences have been presented in the literature that, in some areas, *S. typhi-murium* is replacing *S. dublin* as the main species isolated from cattle⁸. Medical and veterinary surveys, to define the distribution of salmonellae are needed in order to establish a clear picture of the ecology of the organisms both in the human host and the animal population. That this is essential to arriving at more efficient control measures than those presently available cannot be over-emphasized.

The sharpness and explosive character of the present outbreak, reflected in the large number of cases, their short time distribution and the exceedingly high attack rate are evidences suggesting the ingestion of a large number of viable organisms by the employees who later became infected. Fortunately, the employees and staff rather than the patients were the hospital group actually exposed to the risk of infection

inasmuch as salmonella gastroenteritis may become a major problem among patients in hospitals¹⁵ particularly among those with severe and debilitating diseases for whom the case fatality rate may reach high levels^{15, 16}.

In view of the large number of secondary infections that occur in institutional outbreaks and the low secondary attack rate presently recorded, the value of prompt enforcement of control measures becomes apparent. Control measures were aimed at reducing to the lowest possible point the chance of secondary spread through food while strict isolation procedures were not attempted. The low secondary attack rate seems to indicate that spread from person-to-person is a minor problem in outbreak of salmonellosis; the infection being usually food borne.

The relative importance of the carrier as source of salmonella infection has been emphasized by some workers^{11, 16, 20}, although the carrier state after clinical or inapparent infection with salmonella that cause gastroenteritis is of low incidence and usually transient^{16, 17, 19, 20}. Our findings show that the high proportion of persons with positive stool cultures on the second day of illness is followed by a rapid and uniform decline during the few subsequent days, then the rate of positive cultures tended to level of up to the end of the second week when again a rapid rate of clearance is initiated. By the fifth week, only 5.1 percent of persons were found with positive cultures while after six weeks no carrier could be detected.

RESUMO

Surto de intoxicação alimentar devido à Salmonella typhi-murium ocorrido em hospital geral. I. Aspectos epidemiológicos.

Descreve-se explosiva epidemia de gastroenterite por *Salmonella typhi-murium* observada em hospital de ensino de Salvador, Bahia. Entre 11 e 13 de Dezembro de 1961, registraram-se 213 casos entre os 253 funcionários do hospital, expostos à infecção. A não ocorrência de casos primários entre 290 doentes hospitalizados constituiu-se em elemento importante na detecção do veículo da infecção. Observaram-se apenas 17

casos secundários entre funcionários e doentes. Embora a intensidade das manifestações clínicas variasse de moderada a severa, não se registraram óbitos.

Apresentam-se evidências epidemiológicas que indicam ter sido a carne bovina o veículo responsável pela propagação da infecção. Não foi possível, porém, descobrir a fonte donde a mesma se originou. Chama-se a atenção para a possível importância de produtos animais não industrializados, particularmente da carne bovina, na propagação das salmoneloses em áreas subdesenvolvidas e demonstra-se a importância da aplicação de rígidas medidas administrativas no combate às epidemias de gastroenterite de tipo explosivo, como a descrita acima.

REFERENCES

1. ANDERSON, E. S.; GALBRAITH, N. S. & TAYLOR, C. E. D. — An outbreak of human infection due to *Salmonella typhi-murium* phage-type 20 associated with infection in calves. *Lancet* 1:854-858, 1961.
2. COCKBURN, W. G. — Reporting and incidence of food-poisoning. In COCKBURN, W. G.; TAYLOR, J.; ANDERSON, E. S. & HOBBS, B. C. — Food poisoning. *Royal Society of Health*, London, 1962, pg. 3.
3. COSTA, G. A. & VERNIN, G. S. — Sobre uma modificação do meio de Monteverde. *Mem. Inst. Oswaldo Cruz* 53:105-114, 1955.
4. DARK, G. M. — Food poisoning. *Chicago, University of Chicago Press*, 1956.
5. EDWARDS, P. R.; BRUNER, D. W. & MORAN, A. B. — Further studies on occurrence and distribution of *Salmonella* types in the United States. *J. Infect. Dis.* 83:220-231, 1948.
6. FESENFELD, O. & YOUNG, V. M. — Study of human Salmonellosis in North and South America. *Am. J. Trop. Med.* 29:438-491, 1949.
7. HARHOFF, N. — Bakteriologische Erfahrungen über Infektionen mit Gastroenteritisbazillen der Paratyphusgruppe. *Zentralb. f. Bakt. (Abt 1)*, 147:194-206, 1941.
8. KNOX, W. A.; GALBRAITH, N. S.; LEWIS, M. J.; HICKIE, G. C. & JOHNSTON, H. H. — A milk-borne outbreak of food poisoning due to *Salmonella heidelberg*. *J. Hyg.* 61: 175-185, 1963.
9. KWANTES, W. — Explosive outbreak of *Salmonella typhi-murium* food poisoning in Lianelly. *Month. Bull. Ministry Health & Pub. Health Lab. Services* 11:239-248, 1952.
10. MAC CREADY, R. A.; REARDON, J. P. & SAPHRA, I. — Salmonellosis in Massachusetts. A sixteen-year experience. *New England J. Med.* 256:1121-1128, 1957.
11. MEYER, K. F. — Food poisoning. *New England J. Med.* 249:804-812, 1953.
12. PARRY, W. H. — A milk-borne outbreak due to *Salmonella typhi-murium*. *Lancet* 1:475-477, 1962.
13. PUBL. HEALTH LAB. SERVICE — Food poisoning in England and Wales, 1960. A report. *Bull. Ministry Health & Publ. Health Lab. Services* 20:160-171, 1961.
14. RODRIGUES DA SILVA, G.; ROCHA, H. P. da & PRATA, A. R. — An outbreak of food-poisoning due to *Salmonella typhi-murium* in a general hospital. II. Clinical aspects. *To be published*.
15. SANDERS, E.; AWEENEY, F. J., Jr.; FRIEDMAN, E. A.; BORING, J. R.; RANDALL, E. L. & POLK, L. W. — An outbreak of hospital — associated infections due to *Salmonella derby*. *J. A. M. A.* 186: 984-986, 1963.
16. SAPHRA, I. & WINTER, J. W. — Clinical manifestations identified at the New York *Salmonella* Center. *New England J. Med.* 256:1128-1134, 1957.
17. SAVAGE, W. — Problems of *Salmonella* food-poisoning. *Brit. Med. J.* 2:317-323, 1956.
18. SILVA, I.; RODRIGUES DA SILVA, G. & PRATA, A. R. — An outbreak of food-poisoning due to *Salmonella typhi-murium* in a general hospital. III. Bacteriological findings. *To be published*.
19. SILVERSTOLPE, L. & WRANNE, N. — Salmonellaepidemierna i Östergötlands län ar 1953. *Nord hyg. tidskr.* 36:213-229, 1955.
20. TAYLOR, J. — Salmonella and salmonellosis. In COCKBURN, W. G.; TAYLOR, J.; ANDERSON, E. S. & HOBBS, B. C. — Food Poisoning. *Royal Society of Health*, London, 1962, pg. 15.
21. WORLD HEALTH ORG. — Food poisoning. Fourth report of the Expert Committee on Environmental Sanitation. *Tech. Rep. Ser.* 104, 1956.