SALMONELLA JAVIANA FOOD INFECTION*

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Organisms of the Salmonella group have been incriminated in a variety of infections,[2] including food poisoning. This latter condition, due to organisms of this genus, may become manifest clinically in conjunction with such processes of bacterial invasion as gastroenteritis, enteritis, septicemia, pyelitis, and meningitis. In general, these infections are marked by a high morbidity but a low mortality. The diagnosis of food poisoning depends primarily on the isolation of the responsible micro-organism, either from the feces or, in rare instances, from the blood stream.

The purpose of this communication is to describe a series of patients exhibiting, in some measure, an atypical symptom complex of food poisoning. The etiological agent in this series was a Salmonella which failed to fall into any of the well-known Salmonella groups. The organism in question proved to be a *Salmonella javiana* strain, and it was responsible for a series of clinical aspects not hitherto attributed to infection by this bacterium. The detailed studies of this bacterium, presented in part in this communication, are further elaborated by Edwards,[1] who first discovered the organism in cultures obtained from the Eijkman Institute in Batavia.

The patients here discussed were from a group of Navajo Indians living in Puertocito, about 90 miles southwest of Albuquerque, New Mexico. The source of the organisms was a cottage cheese made by a local cheesemonger, for it was only in those homes where this cheese made its appearance that the disease occurred. Furthermore, other than from the patients themselves, it was only from this cheese and from the ferment used in making it that the organisms could be recovered.

In general, the symptoms developed by the patients were alarming, being characterized by a sudden intense diarrhea, intermittent vomiting, dehydration, and profound collapse. In most cases of Salmonella food poisoning there is a remission of the disease following the vomiting and diarrhea. In these cases, however, the sequelae were atypical, since instead of improvement, the majority

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of the patients became moribund. Even when fluid and electrolyte balance was re-established, recovery was equivocal and in some instances where a complete recovery was established, sudden exacerbations of the disease appeared, manifesting themselves in collapse, and, on occasion, in sudden death. It was obvious, therefore, that the organism was highly virulent and that the somewhat uniform behavior of the patients indicated an uncommon variety of food poisoning.

The patients and their clinical course

Three groups of patients were admitted to the Albuquerque Indian School Hospital on July 16, 19, and 20, 1942. In general, it can be said that the dietary background of these patients was poor, inasmuch as their diet consisted largely of carbohydrate cereals (in the main refined), with a low protein intake, a moderate fat and an inadequate vitamin intake. Constitutionally, their body economy was poor, and it is conceivable that their resistance to disease and their recuperative vitality were below an acceptable threshold.

Patient No. R-82-43—B. G. T. 94°; P. 130; R. 24; B. P. 70/20-10. This 33-year-old married Indian Navajo male was admitted to the hospital, on July 16, in a state of exhaustion. The main complaint was vomiting and diarrhea of five days' duration. The family history was non-contributory and the review of past illnesses unreliable. The present illness came as an acute manifestation six hours after ingesting a cottage cheese manufactured by a local Mexican cheesemonger. On admission the patient exhibited all the clinical signs of dehydration; his face was pinched and his eyes sunken. He was unable to walk. There was no dyspnea or orthopnea.

The positive physical findings were as follows: abdomen moderately tense and tender to deep palpation; pellagroid icthyotic dermatitis of the elbows, wrists, and ankles. The hemoglobin was 88 per cent (Sahli); white blood cell count, 15,350; differential, polymorphonuclear neutrophils 94, lymphocytes 2, monocytes 4. Urinalysis negative. Stools were obtained at once and within 24 hours gram-negative non-lactose fermenting organisms were obtained. Wassermann and Eagle serological tests for syphilis were negative. Roentgenograms of the chest showed no active pulmonary lesions.

The patient was given on admission 1000 cc. of 10 per cent dextrose in saline and improved considerably. The blood pressure rose to 130/70 and the pulse dropped in rate to 80 per minute and improved in volume. Clinically, the patient felt much better. On the following day he received additional dextrose in saline by vein, as well as vitamin concentrates. He was given small sips of fluid, such as ginger ale, and also isotonic saline solutions. Two grams of sulfaguanidine were given every six hours. The diarrhea

subsided and the patient was able to walk about. He was afebrile. On the 18th day of July he was placed on a soft diet and continued to improve. On July 19 the white blood cell count was 21,250, the temperature 97.2° F., and he appeared suddenly in the morning to be in a state of collapse. Blood cultures taken on admission revealed gram-negative bacilli of the non-lactose fermenting group. Sulfanilamide therapy was instituted even though the action of this drug on Salmonella is of questionable efficacy. Sulfanilamide was not only given orally but also by clysis. The patient failed to recuperate from his sudden collapse and died that same day at 7:20 P. M.

A complete autopsy was performed, with the following findings. The body was that of a well-developed but rather poorly nourished 33-year-old Indian male. The body orifices were negative except for the anus which showed considerable blood, both free and clotted. The peritoneal cavity contained no free fluid and the peritoneum showed no signs of inflammation. The intestinal tract from duodenum to anus was filled with frank blood. This was removed and the small bowel from the duodenum to the ileocecal valve was found to be markedly injected but without ulceration. No source for the bleeding was located. There was no injection of the large bowel. Except for a small milk-patch on the anterior surface of the right ventricle of the heart and some old pleuritic adhesions of the left lung, the rest of the autopsy findings were negative.

Patient No. R-85-43—M. G. T. 98.8°; P. 108; R. 26; B.P. 125/90. This 26-year-old married housewife, the wife of the patient B. G., gave precisely the same history as the first patient. The complaint on admission was the same. Although this patient did not appear so profoundly exhausted as her husband, she was obviously acutely ill. In contrast to her husband, she was able to walk. There was no cyanosis, dyspnea, or jaundice. The physical findings were singularly negative, except that her breasts were engorged and milk could be obtained. Milk cultures as well as blood and stool cultures were taken. The white blood cell counts taken at intervals were as follows: 7/16/42, 4450; 7/22/42, 21,750; 7/24/42, 11,050. The blood cultures on this patient were negative. The hemoglobin content was 75 per cent (Sahli) and the red blood cell count 4,800,000. The differential was as follows: polymorphonuclear neutrophils 86, lymphocytes 9, monocytes 5. The urine on admission was negative for albumin and sugar, specific gravity 1.020, and 100 pus cells per low-power field. Wassermann and Eagle serological tests for syphilis were negative. Stool cultures revealed gram-negative, non-lactose fermenting organisms. Roentgenograms of the chest revealed no active pulmonary lesions. This patient did not have any obvious pellagroid dermatitis. The clinical course was one of definite improvement following fluid balance. The patient was placed on sulfaguanidine, grams 2 every 6 hours. This was kept up until July 19, 1942. A soft diet was instituted. The patient was discharged improved on July 27.

Patient No. R-84-43—G. G. T. 104°; P. 140; R. 42. This patient, a baby boy of 2½ months, was admitted at the same time as his mother (patient M. G.). This is the only patient who did not consume the cheese made by the local cheesemonger. However, the baby exhibited symptoms similar to those of his parents on the day following the initial occurrence of the disease. Vomiting and blood-streaked diarrhea prevailed. The baby was breast-fed up to two days before admission, when the mother felt that she might be "poisoning" the baby and transferred him to raw goat's milk. On admission the child appeared acutely and desperately ill; he had a glassy stare and was breathing rapidly. He appeared moribund and was not easily aroused. No cyanosis, orthopnea, or jaundice. The skin was cold and clammy. There was no upper respiratory infection and the pharynx was negative. No disease of the middle ear. The chest was clear and the heart not enlarged, the rate being rapid with the sounds snapping; no murmurs. The abdomen was relaxed and there were no pediatric signs of pain. No generalized glandular enlargement. The white count on admission was 14,000 with a differential of 54 polymorphonuclear neutrophils, 35 lymphocytes, and 11 monocytes. The red blood cell count was 5,300,000 and the hemoglobin 85 per cent (Sahli). The urinary findings were a 2-plus albumin with occasional pus cells, but no sugar. The quantity was insufficient for specific gravity determination. The watery stools obtained revealed gram-negative non-lactose fermenting bacilli. The blood cultures taken on admission revealed the same organism. On admission the child received 1000 cc., slowly by the intravenous route, of 10 per cent dextrose in saline. In spite of this the child craved water ravenously. He was placed on sulfaguanidine, grams 0.5 every 4 hours, and ginger ale and isotonic saline by mouth. On July 17, 1942, there was some improvement and he was placed on fluids, vitamins, and calcium lactate. Sulfaguanidine was continued and sulfathiazole was introduced when the blood culture was found positive. The dosage was 0.25 gm. every 6 hours following an initial dose of 0.5 gm. On July 18 the child had a sudden turn for the worse. Roentgenograms of the chest were negative for pulmonary lesions. Convulsions appeared intermittently and were non-Jacksonian in character. However, there was no rigidity of the neck and no contributing neurological signs. Lumbar puncture yielded 0.5 cc. of spinal fluid, which was normal. On the evening of July 18 the patient appeared more comfortable; tetany and convulsions had ceased. The white cell count was 29,750 and the prognosis questionable. Although the stools were no longer watery but soft in character, the abdomen became distended and enemata were successful in reducing tympanites. On the morning of July 20, 1942, the child died suddenly.

A complete autopsy was performed but no anatomic diagnosis could be established. The body was that of a well-developed and well-nourished Indian infant male. The external orifices were all free of blood or other

TABLE 1

TABULATION OF HOSPITALIZED PATIENTS AND CERTAIN PERTINENT FINDINGS

Name	*Age*	*Case No.*	*Time between cheese ingestion and symptoms*	*First symptoms*	*Duration of symptoms*	*Stay in hospital*	*Exhaustion*	*W. B. C. on admission*	*S. javiana in*		*Clinical features*	*Treatment*	*Result*
									Stool culture	*Blood culture*			
B. G.	33 yrs.	R82-43	6 hours	V & D*	6 days	8 days	+++	15,350	+	+	dehydration, pellagra	Fluid balance Sulfaguanidine Sulfanilamide Vitamin	Died
M. G.	26 yrs.	R85-43	6 hours	V & D	7 days	11 days	++	4,450	+	neg.	dehydration	Fluid balance Sulfaguanidine Vitamin	Recovered
G. G.	2½ mos.	R84-43	No cheese ingestion; 12 hours after parents	V & D	8 days	8 days	+++	14,000	+	+	dehydration	Fluid balance Sulfaguanidine Vitamin	Died
B. G.	7 yrs.	R87-43	6 hours	V & D	7 days	11 days	++	5,250	+	+	dehydration, pellagra	Fluid balance Sulfaguanidine Vitamin	Recovered
D. G.	5 yrs.	R86-43	6 hours	V & D	8 days	11 days	++	6,850	+	neg.	dehydration	Fluid balance Sulfaguanidine Sulfanilamide Vitamin	Recovered
A. G.	9 yrs.	R83-43	6 hours	V & D	8 days	11 days	++	8,500	+	neg.	dehydration	Fluid balance Sulfaguanidine Vitamin	Recovered
J. G.	1 yr.	R112-43	6 hours	V & D	8 days	9 days	++	13,950	neg.	neg.	dehydration, x-ray shows thymus shadow in chest	Fluid balance Sulfaguanidine Sulfanilamide Vitamin	Recovered
R. V.	8 yrs.	R111-43	8 hours	V & D	6 days	9 days	+	14,800	+	neg.	Active pulm. TBC, dehydration (minimal)	Fluid balance Sulfanilamide Vitamin	Recovered
L. G.	8 yrs.	R113-43	4 hours	V & D	6 days	9 days	+	13,400	not taken	neg.	Dehydration (minimal)	Fluid balance Sulfaguanidine	Recovered
K. M.	12 yrs.	R110-43	5-6 hours	V & D	5 days	9 days	+	14,400	+	neg.	Dehydration (minimal)	Fluid balance Sulfaguanidine	Recovered
L. S.	13 yrs.	R109-43	5-6 hours	V & D	5 days	9 days	+	5,500	not taken	neg.	Dehydration (minimal)	Fluid balance Sulfaguanidine Sulfanilamide Vitamin	Recovered
D. A.	21 yrs.	R100-43	6-7 hours	V & D	19 days	12 days	++	11,900	not taken	neg.	Active pulm. TBC, dehydration	Fluid balance Sulfaguanidine Vitamin	Recovered
J. A.	35 yrs.	R121-43	8-9 hours	V & D	16 days	5 days	++	9,600	neg.	neg.	Dehydration (minimal)	Sulfaguanidine Vitamin	Recovered
J. G.	26 yrs.	R106-43	6-7 hours	V & D	7 days	5 days	+	9,250	+	neg.	Dehydration (minimal)	Sulfaguanidine Vitamin	Recovered
M. A.	13 yrs.	R107-43	7 hours	V & D	7 days	5 days	+	12,500	+	neg.	Dehydration (minimal)	Sulfaguanidine Vitamin	Recovered

*V & D = vomiting and diarrhea.

discharge. The peritoneal cavity was free of fluid and the peritoneum showed no signs of inflammation. The bowel contained semi-fluid fecal material without evidence of bleeding and showed no inflammation. The rest of the autopsy also revealed no gross evidences of pathology.

Patient No. R-87-43—B. G. T. 99.8°; P. 122; R. 28; B. P. 98/75. This 7-year-old school girl (daughter of patient M. G.) was admitted to the hospital on July 16, 1942, with complaints similar to those of the preceding case. The history of the present illness was the same as that of her parents. The physical findings were entirely negative except for dental caries and pellagroid icthyotic dermatitis. On admission she was obviously acutely ill, although not in a state of exhaustion or collapse. There was no cyanosis, orthopnea, dyspnea, or jaundice. Blood studies revealed a white cell count of 5250, with a differential of 63 polymorphonuclear neutrophils, 35 lymphocytes, and 2 monocytes. The red cell count was 5,050,000, with a hemoglobin content of 81 per cent (Sahli). Stool cultures yielded a gram-negative non-lactose-fermenting bacillus and a blood culture on admission was likewise positive for the same organism. The patient was placed on liquids consisting of ginger ale and isotonic saline; sulfaguanidine was given at once and continued in 1.0 gram doses. On July 17, 1942, a soft diet was introduced and the patient improved. The sulfaguanidine was continued until July 19, at which time sulfanilamide was introduced on the basis of the positive blood culture. The patient continued to improve on a bland diet. On July 24 her white cell count was 8000. She was discharged from the hospital on July 27, 1942.

Since the rest of the patients admitted followed somewhat similar courses as those recorded above, they can best be summarized as a group including these already mentioned.

Discussion of hospitalized patients

In our series of hospitalized patients it seemed obvious that the main problems clinically were those of overcoming the dehydration and the use of some chemotherapeutic agent against *Salmonella javiana*. The first problem of dehydration was not a difficult one, since a balance with intravenous dextrose and saline solution could be effected. The second problem was more baffling. The action of sulfaguanidine on intestinal bacteria is well known, but its action on a Salmonella hitherto obscure was not known. In fact, repeated stool cultures following sulfaguanidine therapy were still positive in some instances. Thus the use of sulfaguanidine was empirical, and proof of its efficacy on this infection is lacking. The *Salmonella javiana* bacteremia likewise received no treatment other than the empirical use of sulfanilamide, and even though one of the three

cases with bacteremia recovered, recovery cannot be attributed to sulfanilamide therapy. Further investigation of the chemotherapy of *Salmonella javiana* infection is indicated.

The Puertocito field problem and epidemiology

One of us (R.D.A.) made several trips to Puertocito, New Mexico. We had been informed of the seriousness of the enteritis on July 19th by patients admitted to the hospital and, as a consequence, on July 20th a trip was made to Puertocito to investigate the situation. There were 40 patients ill with enteritis, some desperately ill and in a state of collapse. Since no physician had ever visited them before, they were suspicious of our motives and remained in their hogans. Thus, although very little cooperation could be secured with these Navajo Indians, eight patients agreed to hospitalization.

Since cheese implicated in the outbreak had been sold by a Mexican family, the home was visited and blood samples were taken from each member of the household. Specimens of cheese and of the ferment (autolysed pancreas) were secured.

The Indians were advised not to consume any more cheese from this cheesemonger, and a resolution to this effect was passed by the tribal council.

A second trip made two days later revealed that the situation was well in hand and, although we were able to secure blood samples from the cows belonging to the cheesemonger, no further studies could be carried out either on the family or in their dwelling. By advising the Indians not to procure any further cheese from the cheesemonger, we alienated ourselves with the Mexican vendor. It can be said that when consumption of this cheese ceased the epidemic ceased, and by July 20th no further cases of enteritis occurred.

It is of interest to note that the first case of food poisoning occurred in the Mexican family where the cheese was made. The elder cheesemonger, M. G. Sr., was desperately ill on July 3, 1942, and his servant, V. G., died following a severe enteritis at that time.

The cheesemonger responsible for the sale of the cheese gave only limited cooperation in the epidemiological investigation. Since we were interested in gaining an insight into the resistance of the Mexican family toward the infection and since they may have acted as carriers, agglutinin titrations of their sera with the appropriate antigen were indicated. Furthermore, the family owned five cows,

whose milk was used in the manufacture of the cheese. Their sera were likewise obtained for agglutination tests. The results are expressed in the following table.

TABLE 2

AGGLUTINATION TESTS WITH THE SERA OF MEMBERS OF THE MEXICAN FAMILY AND OF THEIR COWS

	Titer of sera obtained from											
Source of organism	*E.G.*	*M.G.*	*R.G.*	*M.G. Sr.*	*C.G.*	*A.G.*	*M.G. Jr.*	*Cow 949*	*Cow 950*	*Cow 951*	*Cow 952*	*Cow 953*
Cheese	1:80	1:40	1:160	1:80	1:80	1:40	0	1:20	0	1:40	1:40	1:40
Blood culture (Case R84-43)	1:80	1:40	1:320	1:320	1:160	1:40	1:20	1:20	1:20	1:40	1:40	1:20

A titer below 1:80 can be regarded, in general, as being without significance; a titer of 1:80, however, is interpreted as indicative of positive antibody formation. It can be seen from the foregoing table that three persons had agglutination titers of less than 1:80 (M. G., A. G., and M. G. Jr.). Of these persons two denied having eaten any of the cheese (M. G. Jr. and A. G.), whereas an equivocal history was obtained from the third (M. G.). The highest titer was obtained in M. G. Sr., who had the history of having had a severe enteritis. The only conclusion that can be drawn is that generally the family had a high agglutination titer and their cows gave essentially negative findings.

It was of further interest to compare these results with those obtained with the sera from our hospital patients. The contrast in titer reveals a difference presumably based on the active infection in our patients. The results are as follows:

TABLE 3

AGGLUTINATION TESTS WITH THE SERA OF HOSPITALIZED PATIENTS

Titer of sera obtained from	*Source of organism used as antigen*	
	Cheese	*Blood culture (Case R84-43)*
B.G. (R82-43)	1:640	1:640
M.G. (R85-43)	1:640	1:640
D.G. (R86-43)	1:640	1:640
A.G. (R83-43)	1:640	1:640
R.V. (R111-43)	1:320	1:640
K.M. (R110-43)	1:320	1:640
J.G. (R112-43)	1:320	1:320
M.A. (R107-43)	1:320	1:640
L.G. (R113-43)	1:320	1:640
J.G. (R106-43)	1:160	1:640
L.S. (R109-43)	1:320	1:320
D.A. (R100-43)	1:40	1:20

In these cases the titer is high for the organisms obtained from the cheese and from the blood culture. There is no explanation for the low titer in one case (D. A.), where the values were 1:40 and 1:20, respectively. This patient had an active pulmonary tuberculosis. The blood culture was negative and a stool culture was not taken. In the light of inadequate data no interpretation can be attempted.

A gram-negative, motile, non-lactose-fermenting bacillus was obtained from the following sources in the Puertocito epidemic:

Blood cultures from patients R-84-43, R-82-43, R-86-43, and R-87-43.

Stool cultures from all but five patients were positive, as were repeat cultures from R-87-43, R-85-43, and R-86-43, who had positive stools on admission.

Autopsy specimens from the large intestine, small intestine, and lung section of G. G., R-84-43, yielded the organism.

The mother-ferment used in making the cheese gave positive cultures. The ferment was made at some time during the winter of 1941-42 from beef pancreas which was permitted to autolyze at room temperature.

A sample of cheese, made the day of purchase, was contaminated with the organism. Some of the same mother-ferment was used in making this cheese as was used in making the cheese which poisoned the Navajos.

The organism was not found in M. G.'s milk—a point of interest inasmuch as her nursling experienced the same illness a day later than the rest of the family and is said not to have eaten the cheese.

Characteristics of the organism obtained: The organism when grown on a blood agar or desoxycholate agar plates gives rise to smooth-edged, elevated, translucent, yellowish-gray colonies from 2 to 3 mm. in diameter. The organism has the following fermentation characteristics:

On Russell's double sugar medium there is fermentation of dextrose with the production of acid and gas. Arabinose, dextrose, xylose, mannose, dulcite, maltose, and trehalose are fermented with the production of acid and gas. Lactose and saccharose are not fermented. H_2S is produced in lead acetate agar. There is a negative indole reaction in tryptone broth.

Antigens were prepared from the organisms derived from the

blood cultures of B. G., G. G., and the cheese by killing 24-hour broth cultures with 0.3 per cent tricresol. Agglutination tests, using these antigens and antisera specific for the common human Salmonella pathogens gave the following results:

TABLE 4
AGGLUTINATION OF ORGANISMS BY SPECIFIC ANTISERA

	Specific antisera for:*			
Antigens	*S. paratyphi*	*S. schottmuelleri*	*S. aertrycke*	*S. enteritidis*
Blood culture B. G. (R-82-43)	0	1:80	0	1:640
Blood culture G. G. (R-84-43)	0	1:640	1:640	1:640
Blood culture from guinea-pig inoculation†	0	1:160	0	1:640

*Obtained from Professor John F. Kessel.
†Guinea-pig inoculated with ferment, died within 24 hours, and yielded an organism from which the antigen was prepared.

From this table it is evident that agglutination at high titers occurred with *S. schottmuelleri* and *S. enteritidis*, and that of these the highest was with *S. enteritidis*. This would lead one to suspect the etiological agent as being an enteritidis strain. However, with the organism obtained from G. G. the titer of 1:640 was the same with the antisera for *S. schottmuelleri*, *S. aertrycke*, and *S. enteritidis*.

Agglutinin absorption tests, employing *S. enteritidis*, *S. aertrycke*, and *S. schottmuelleri* antisera which exhibited no cross agglutination in dilutions of 1:5000, were performed by absorbing with the antigen made from the organism obtained from the G. G. blood culture. The absorbed antisera were then tested against their homologous antigens with the following results: *S. enteritidis*, agglutination 1:160; *S. schottmuelleri*, agglutination 1:640; *S. aertrycke*, agglutination 1:2360. Since agglutinin absorption was greatest with *S. enteritidis* it suggested that this organism might be responsible for the infection. However, the absorption of antisera and agglutinin reactions are dependent on somatic, and to a greater degree, on flagellar antigens which are shared by other Salmonella. It might thus be possible that the organism responsible for this disease might have some of the flagellar and somatic antigens of *S. enteritidis*, *S. aertrycke*, or *S. schottmuelleri*, with a dominance of certain *S. enteritidis* antigens. Specific antigens prepared from these organ-

isms were obtained, and when these were tested with the sera from our patients the results were as follows:

TABLE 5

AGGLUTINATION OF SPECIFIC ANTIGEN* BY VARIOUS SERA

	Sera from:					
Antigen	*B.G. (R-82-43)*	*D.G. (R-86-43)*	*A.G. (R-83-43)*	*R.V. (R-111-43)*	*G.M.*	*Cow 952*
S. enteritidis	1:160	1:160	1:320	1:160	1:640	1:160
S. aertrycke	1:160	1:80	1:160	1:160	1:160	1:80
S. schottmuelleri	1:40	1:40	1:80	1:40	1:160	1:80

*Obtained from Professor John F. Kessel.

The only conclusion that could be derived from these data is that the organism in question has several components of its antigenic formula in common with *S. enteritidis.*

Since no monophasic antisera were available for this investigation, cultures were sent to Edwards[1] for identification. He reported *Salmonella javiana* with the antigenic formula [I], IX, XII . . .: 1, z_{28} . . .-1, 5 . . . . This organism, *Salmonella javiana,* has in common with *S. enteritidis* 0 antigens IX and XII, but differs in its flagellar antigen constituency and in addition by having the 0 antigen [I].

Summary

An epidemic of enteritis, in some cases followed by septicemia, due to *Salmonella javiana* infection is described. The source of the infection was traced to contaminated cheese which had been ingested by the patients. The mother ferment used in the preparation of the cheese also contained the organism. When restrictions were placed upon the distribution and use of the cheese the outbreak ceased.

REFERENCES

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We wish to thank Professor John F. Kessel of the Department of Bacteriology, University of Southern California Medical School, Dr. P. R. Edwards of Louisville, Kentucky, and Marion Pierce of the L. F. Pierce Laboratories for their cooperation and advice in this study of *Salmonella javiana* infection.