

OUTBREAK OF TYPHOID FEVER IN TRINIDAD IN 1971 TRACED TO A COMMERCIAL ICE CREAM PRODUCT

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Taylor, A., Jr., A. Santiago, A. Gonzalez-Cortes and E. J. Gangarosa (CDC, Atlanta, Ga. 30333). Outbreak of typhoid fever in Trinidad in 1971 traced to a commercial ice cream product. *Am J Epidemiol* 100: 150-157, 1974.—In April 1971, a nationwide outbreak of typhoid fever involving 132 persons occurred in Trinidad; there were no deaths. Eighty per cent of cases occurred in children ages 5-14, and more than 90% of ill persons lived or went to school in the main towns or in smaller communities along their connecting roads. The epidemic curve suggested a common source, and a series of food preference questionnaires implicated a nationally distributed ice cream product. Further investigation indicated that the product was distributed on only one day, March 23. The mean incubation period was 19 days, and the attack rate for those at risk was slightly greater than 1%. Samples of the ice cream product obtained a month after the outbreak were found to contain greater than 1100 *Escherichia coli* per 100 ml. Inspection of the plant revealed frequent hand contact with the product and an absence of pasteurization facilities. Although rectal swabs and stool cultures obtained after purgation from employees failed to identify the carrier, epidemiologic evidence suggested that an employee in the plant, rather than a contaminated ingredient, was the source of the outbreak. This outbreak emphasizes the need for mandatory pasteurization of milk and ice cream products, especially when strict sanitary procedures cannot be adhered to or enforced.

disease outbreaks; *Escherichia coli*; food contamination; typhoid

INTRODUCTION

The epidemiologic and bacteriologic study of typhoid through the years has allowed the formulation and implementation of effective control measures in the United States such that reported cases of the disease decreased from 5595 in 1942 to

380 in 1972. These control measures include active surveillance of the disease, investigation of outbreaks, cases, and contacts to identify vehicles of transmission and sources of contamination, exclusion of carriers from food service activities, pasteurization of milk and milk products, improved sewage treatment and disposal, and

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chlorination of water supplies. Large urban outbreaks, however, do occur from time to time as in Zermatt, Switzerland (1), Aberdeen, Scotland (2), and Miami, Florida (3). In addition, smaller outbreaks have continued to occur in the United States and abroad (4-6). The critical role of epidemiology in elucidating the cause of an outbreak and in providing a basis for the design, implementation, and evaluation of control measures is illustrated by an outbreak in Trinidad in 1971.

THE OUTBREAK

As part of a continuing collaboration in communicable diseases surveillance between the Pan American Health Organization (PAHO) and the Center for Disease Control, an investigation was conducted of an outbreak of typhoid fever in Trinidad in May 1971. A request for assistance originating in Trinidad was received from PAHO on May 4, reporting that 70 cases had occurred in several communities on the island in the middle of April. No information was available as to the vehicle of transmission but a commercial product was strongly suspected because of the distribution of cases. Two of us (A.T. and A.G.C.) departed from Atlanta on May 6 to assist with the investigation.

The tropical island nation of Trinidad and Tobago, an independent member of the British Commonwealth, is situated in the southern Caribbean near the coast of Venezuela. Trinidad, the second largest of the former British West Indian Islands, has a total area of 4815 km² and a population of about 1,000,000 persons. Its sister island, Tobago, is 295 km² with a population of approximately 40,000.

Typhoid had been endemic in Trinidad for years prior to this investigation. Cases were reported on the basis of clinical diagnosis or positive culture. Between 1960 and 1969, there were 715 reported cases, an average of nearly one every 5 days. In the south, typhoid patients were hospitalized

in the 615-bed medical and surgical hospital in San Fernando. In the north, patients were usually hospitalized in either the 84-bed hospital at Sangre Grande in north-eastern Trinidad or in the 902-bed hospital in the capital city, Port of Spain. Prior to the epidemic only the hospital at Port of Spain had a laboratory capable of culturing *Salmonella typhi*.

METHODS OF INVESTIGATION

Laboratory and hospital records of persons hospitalized with suspect typhoid fever in Port of Spain, Sangre Grande, and San Fernando, Trinidad; and Scarborough, Tobago were reviewed. Patients with positive cultures or a clinical presentation compatible with typhoid fever associated with an O antibody titer greater than 1:100 were considered to have the disease. Clinical presentation and the O titer were used to define cases because bacteriologic confirmation was not available in southern Trinidad until late in the outbreak. Cultures identified as *S. typhi* in San Fernando and Port of Spain were sent to the U.S. Center for Disease Control for confirmation and phage typing.

Patients who met the criteria for typhoid fever were interviewed, and their date of onset, age, sex, school attended, and food histories were recorded. Maps were constructed showing the location of typhoid patients by home address and school. Six sets of questionnaires on food preferences and food histories were completed for ill individuals and control populations. Sources and distribution of water in Trinidad and Tobago were reviewed. Suspect food items and specimens obtained from individuals thought to be carriers were cultured for salmonellae. Selected food samples were also quantitatively cultured for coliforms. An investigation was subsequently conducted at the implicated establishment, and cultures were taken from food handlers.

Isolation and identification of *S. typhi*

were performed by methods described by Edwards and Ewing (7). Phage typing was performed by the method described by King and Grant (8).

RESULTS OF INVESTIGATION

In Trinidad, there were 132 culture-positive cases of typhoid fever. There were also 55 suspected cases with negative cultures; their hospital records were not reviewed, and these patients therefore could not be excluded or confirmed as having typhoid. There were no cases of typhoid in Tobago in the epidemic period. Isolates from 23 randomly selected patients were all identified as phage type A (7).

Information on dates of onset of illness was available for 130 cases (figure 1). The rapid rise, sharp peak, and rapid decline of cases shown in the curve suggested a common source outbreak with exposure occurring within a limited time period.

Epidemic curves plotted for cases in each of the major towns revealed that the outbreak occurred approximately one week

earlier in Port of Spain than in the rest of the country; thus, exposure occurred earlier in Port of Spain. This information was compatible with data showing that the contaminated product was distributed in Port of Spain before it was delivered to the rest of the country. Moreover, given the incubation period for typhoid of approximately two weeks and counting backward from the mean date of onset, April 16, exposure most likely occurred in late March or early April.

Data on age and sex were available for 127 cases (table 1). Most (80.2 per cent) of the cases were in children five to 14 years of age. Age and sex distribution was similar in all areas of the country. Based on the mid-1968 estimated population, attack rates per 100,000 persons were highest in school-age children (table 1).

With this information, water could be epidemiologically exonerated as the vehicle of infection since children under five years, who drank water and who had acquired little immunity from either vaccination or

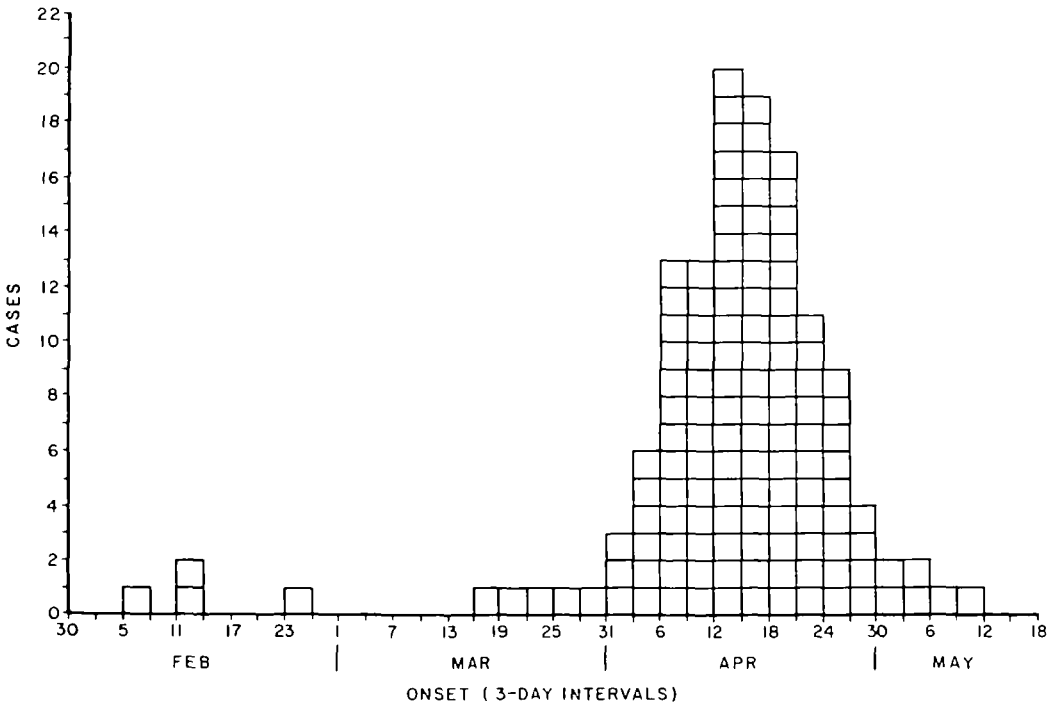


FIGURE 1. Cases of typhoid fever, by date of onset, Trinidad, 1971.

TABLE 1
Attack rates by age and sex, Trinidad, 1971

Age group (years)	Male			Female			Both sexes		
	Ill	Total	Attack rate/100,000	Ill	Total	Attack rate/100,000	Ill	Total	Attack rate/100,000
0-4	1	75,150	1.3	3	73,150	4.1	4	148,300	2.7
5-9	18	77,550	23.2	26	74,650	34.8	44	152,200	28.9
10-14	18	65,800	27.4	40	62,250	61.3	58	131,050	44.3
15-19	4	52,900	7.6	5	52,300	9.6	9	105,200	8.6
20-29	1	76,600	1.3	1	79,450	1.3	2	156,050	1.3
30-39	1	55,400	1.8	5	54,150	9.2	6	109,550	5.5
40-49	0	43,950	0	3	45,300	6.6	3	89,250	3.4
50-59	0	35,750	0	0	33,900	0.0	0	69,650	0.0
60+	0	27,060	0	1	32,250	3.1	1	59,300	1.7

chronic exposure to low numbers of typhoid organisms, had a very low attack rate. Furthermore, Trinidad had several independent water supplies, and no single supply could account for the nationwide distribution of cases.

The initial data suggested that the vehicle of infection was a food or beverage consumed primarily by school-age children. To test this hypothesis, a list of foods available to and popular with school children was prepared. From this list, a detailed food preference questionnaire containing 27 items was devised, and 70 children with typhoid fever in the Port of Spain, Sangre Grande, and San Fernando Hospitals and 43 healthy children were interviewed. Each one was asked which of the foods he had eaten since Christmas. Food-specific attack rates were then calculated for each item. No item was clearly implicated by the results of the first questionnaire, so a second questionnaire was prepared which included several new items: "pallet," a general term for all ice cream sold on a stick; fruit ice, one of several frozen nonmilk products sold on a stick; and Brand A products which included both pallets and fruit ices. The second questionnaire was administered to 71 children with typhoid and to 845 healthy controls from schools in San Fernando and Sangre Grande. These results suggested that those who ate Brand A products were

at greater risk of contracting typhoid and that those who ate competitive products (pallet, other fruit ices, and ice cream cones) were slightly protected. However, the data were not statistically significant.

At this point, a temporary impasse was reached, and we thought we might be getting inconsistent answers from the children. Some children, perhaps awed at being interviewed by an adult foreigner of a different race with a strange accent, tended to answer all questions either "yes" or "no." One child, who was answering "yes" in a rote fashion, was asked if he ate tiger meat. The child replied solemnly that he did. When asked where he got his tiger meat, he realized what he had said, laughed, and then gave what appeared to be accurate responses. Because of the question of reliability, however, responses of the ill children on the first two questionnaires were compared and inconsistent responses were noted. In addition, it was learned that some children called Brand A products "pallet" (the general term for ice cream on a stick), and other children called pallet "Brand A products." Because of the confusion regarding which ice cream and fruit ice products were eaten and in order to determine which food was responsible for the outbreak, models were made of all ice cream and fruit ice products by stuffing wrappers of the various products with toilet paper to give them a normal appear-

ance. The models were then shown to the children, who were asked to point to every item they had eaten since Christmas.

With the assistance of these models, a third questionnaire was administered to 52 children ill with typhoid in the Port of Spain and San Fernando hospitals. These patients included residents of Port of Spain, Sangre Grande, San Fernando, and those living along the main highways. There were 765 control students from three schools in San Fernando. The results implicated Brand A products with a probability value of less than 0.001 (table 2).

In the fourth survey, children in the San Fernando and Port of Spain hospitals were asked to indicate if they had eaten Brand A pallets or Brand A fruit ices since Christmas. The control population consisted of children from a girls' and boys' school in Port of Spain. The results implicated the ice cream product, Brand A pallets (table 3).

While implicating a food, these results posed a new problem. Brand A products were produced by a single factory in Port of Spain and were distributed along the main roads and to the major cities. Brand A trucks traveled to Sangre Grande and San Fernando approximately twice a week re-supplying each customer with Brand A products. Yet, of three schools in San Fernando selling Brand A products, only one school had children with typhoid. The most likely explanation for this apparent discrepancy was the distribution of the contaminated Brand A pallet to only one of the three schools, the school with the typhoid cases. More importantly, solution of this problem might indicate the date of distribution of the contaminated product.

Accordingly, the distribution records of the company and the purchase records of two of the three schools were reviewed. The records revealed that between mid-March and early April only once on March 23 were

TABLE 2
Food-specific attack rates: Questionnaire III, Trinidad, December 25, 1970-May 18, 1971*

	Ate the food				Did not eat the food			
	Well	Ill	Total	Attack rate	Well	Ill	Total	Attack rate
Brand B pallet	237	21	258	8.1	514	31	545	5.7
Brand B fruit ice	337	23	360	6.4	428	29	457	6.3
Brand B ice cream pallet	149	15	164	9.1	522	37	559	6.5
Brand A pallets & fruit ices	554	51	605	8.4	180	1	181	0.6†
Brand C pallet	133	13	146	8.9	592	39	631	6.2
Brand D pallet	161	13	174	7.5	566	39	605	6.4

* Not all children responded to each item on the questionnaire and, consequently, the total number of children responding varies slightly from one item to another.

† $p < 0.001$ by Fisher's exact test.

TABLE 3
Food-specific attack rates: Questionnaire IV, Trinidad, December 25, 1970-May 18, 1971*

	Ate the food				Did not eat the food			
	Well	Ill	Total	Attack rate	Well	Ill	Total	Attack rate
Brand A fruity pallet	94	25	119	21.0	34	17	51	33.4
Brand A ice cream pallet	94	43	137	31.4	35	2	37	5.4†

* Not all children responded to each item on the questionnaire and, consequently, the total number of children responding varies slightly from one item to another.

† $p < 0.05$.

Brand A pallets distributed to the school where cases occurred and not to the other two schools. The records also revealed that although Brand A pallets were usually sold south of San Fernando in La Brea and Point Fortin, Brand A products were not distributed to Tobago; there were no cases of typhoid in Tobago during the epidemic period. (No Brand A pallets were distributed to La Brea and Point Fortin March 23; no typhoid cases occurred in La Brea and Point Fortin during the epidemic period.) The geographic distribution of typhoid cases closely paralleled the distribution of the Brand A pallets on March 23.

Early on the morning of March 23, 97 per cent of all Brand A products distributed in Port of Spain were purchased from the factory by street vendors, who sell almost all their pallets and fruit ices the same day they obtain them. Consequently, the contaminated pallets were essentially all eaten March 23 in Port of Spain. The pallets distributed to Sangre Grande and San Fernando were sold in shops and not by vendors. The company delivers to these areas approximately twice a week, but since delivery tends to be irregular, shops must buy enough stock to last several days; remaining stock is usually sold prior to the sale of a new supply. Consequently, pallets distributed on March 23 in San Fernando and Sangre Grande were eaten approximately a week after those eaten in Port of Spain, explaining the occurrence of the epidemic a week earlier in Port of Spain.

Ill children questioned at Port of Spain Hospital ate an average of 19.5 Brand A pallets per 28 days. A sample of 363 well children from three schools in Port of Spain consumed an average of 6.5 Brand A pallets per 28 days. Information regarding consumption of Brand A pallets by 186 adults in Port of Spain was obtained by house-to-house interviews in a low-medium income area and by questioning persons returning for their second typhoid vaccination; the adults ate an average of 3.3 Brand A pallets per 28 days. The lack of

popularity of Brand A pallets among adults in addition to immunity acquired through repeated exposure or vaccination probably explains their low attack rate.

No water mains were broken near the Brand A factory within months prior to March 22, the presumed date of contamination. Food products used in the manufacture of Brand A pallets on March 22 were all used on other days or in other products. There is no evidence of contamination in more than one product or on more than one day.

Investigation of the plant revealed that the ingredients used in the products were mixed in an open drum by hand. Sticks were placed in the molds by hand, and the frozen product was wrapped by hand. Gloves were not worn, and there was no pasteurization of the product or the mix.

Samples of Brands A, B, C, and D pallets and pallets obtained in May were all found to be contaminated with *Escherichia coli*. Further samples were collected and, using the fermentation tube technique, a most probable number of greater than 1100 *E. coli* per 100 ml was calculated for Brand A pallets. *S. typhi* was not isolated from the Brand A pallets produced six weeks after the outbreak. Brand B was from a pasteurized plant and contained less than 10 *E. coli* per 100 ml. Brands C and D were from unpasteurized plants and contained between 23 and 120 *E. coli* per 100 ml.

Single rectal swab specimens and stool cultures obtained from all 17 employees of the firm producing Brand A pallets and from two employees recently discharged were negative for *S. typhi*. Unfortunately, local circumstances thwarted a more intensive search for the carrier.

DISCUSSION

The food-specific attack rates and product distribution strongly implicate Brand A pallets distributed on March 23 as the vehicle of transmission. That there were only 132 confirmed cases in several thou-

sand persons exposed suggests either heavy contamination of a few pallets or a low level of contamination of many or all pallets distributed on that date. It is unlikely that a few heavily contaminated pallets would have resulted in the scattered nationwide distribution found in the epidemic; it is more probable that all pallets distributed on March 23 had a low level of contamination. Assuming a uniformly low level of contamination, the risk of developing typhoid fever after eating Brand A pallets can be estimated. In Port of Spain, there were 31 confirmed cases of typhoid following the sale of approximately 2650 Brand A pallets on March 23, for an attack rate of 1.17 per cent. Excluding Port of Spain, there were 8075 Brand A pallets distributed on March 23 and 97 additional cases, for a comparable attack rate of 1.20 per cent.

Of the estimated 2650 Brand A pallets distributed in Port of Spain on March 23, 2600 were distributed to vendors and less than 89 were returned at the end of the day, indicating that 97 per cent of the contaminated pallets sold in Port of Spain were distributed and consumed on March 23. With the date of infection known in Port of Spain, reliable mean and median incubation periods of 19.25 and 19 days, respectively, could be calculated. Hornick et al. (9) have shown that the incubation period varies inversely with the number of ingested typhoid organisms, and Huckstep (10) states, "The incubation period in children tends to be shorter than the 10-14 days of adults . . ." The prolonged incubation period for patients in port of Spain, of whom 85 per cent were children, supports the hypothesis of a low level of contamination.

In adult volunteer experiments, Hornick et al. (11) have observed that a 50 per cent attack rate followed ingestion of 10 million typhoid organisms suspended in 30 ml of milk and that a 28 per cent attack rate followed ingestion of 100,000 organisms. No

illness occurred in 14 volunteers who ingested 1000 organisms. (Milk and milk products tend to neutralize stomach acid, thereby weakening one of the body's natural defenses against infection and possibly permitting a lower infecting dose than would otherwise be necessary to cause disease.) Although the epidemic occurred primarily in children, based on Hornick's data in a controlled environment and on the attack rates of 1.17 per cent and 1.20 per cent, a level of contamination of 1000 typhoid organisms per pallet (a pallet weighs about 49.6 gm) can be roughly estimated. Since each pallet contains about 50 ml of typhoid organisms and since approximately 10,725 Brand A pallets were produced on March 22 (distributed March 23), the initial mix must have contained slightly more than 500 liters. Using Hornick's observation that some carriers may shed as many as 10^{11} typhoid organisms per gram of feces (11), one can calculate that one ten-thousandth (1×10^{-4}) of a gram of feces from a person shedding 10^{11} organisms per gram added to the 500 liters of mix would provide, without replication of a single organism, enough typhoid bacilli to contaminate each pallet to a level of 1000 organisms.

Prior to June 3, no secondary cases had occurred. Only one additional case of typhoid had occurred, and that case was unrelated geographically and by history to the epidemic. The absence of secondary cases is explained in part by the large number of organisms required to produce clinical illness and to the prompt and effective treatment of cases.

Females outnumbered males in the epidemic approximately 2:1. This sex distribution may reflect the chance sale of contaminated pallets in or near a predominantly girls' school. Female cases outnumbered males, however, in each major town and in the group living along the main roads. In addition, results of a survey of Brand A pallet eating habits among 239

age-matched children within a coeducational school in Port of Spain indicated that boys ate Brand A pallets slightly more frequently than girls. The possibility that the female children were more susceptible to typhoid than male children cannot be excluded.

Through prompt epidemiologic investigation of this outbreak, the mode and vehicle of transmission were identified, and appropriate control measures were taken. The contamination of a nationally distributed unpasteurized milk product emphasizes that the public would be best protected by mandatory pasteurization of milk and ice cream products.

REFERENCES

1. Bernard RP: The Zermatt typhoid outbreak in 1963. *J Hyg* 63: 537-563, 1965
2. Great Britain, Scottish Home and Health Department: The Aberdeen Typhoid Outbreak, 1964, Edinburgh, Her Majesty's Stationery Office, 1964
3. Center for Disease Control: Morbidity and Mortality Weekly Rep 22(9):77, March 3, 1973; 22(10):85, March 10, 1973, & 22(13):115, March 31, 1973
4. Center for Disease Control: Morbidity and Mortality Weekly Rep 20(40):363, October 9, 1971
5. Center for Disease Control: Morbidity and Mortality Weekly Rep 20(47):428, November 27, 1971
6. Collins RN, Marine EM, Nahmias AJ: The 1964 epidemic of typhoid fever in Atlanta: Clinical and epidemiologic observations. *JAMA* 197:179-184, 1966
7. Edwards PR, Ewing WH: Identification of Enterobacteriaceae. 3rd edition. Minneapolis, Burgess Publishing Co, 1972, pp 7-12
8. King SD, Grant LS: A review of salmonella, shigella, pathogenic *Escherichia coli*, typhoid phage types, UCHWI 1957-61. *West Ind Med J* 12:90-97, 1963
9. Hornick RB, Woodward TE: Appraisal of typhoid vaccine in experimentally infected human subjects. *Trans Am Clin Climatol Assoc* 78:70-80, 1966
10. Huckstep RL: Typhoid Fever and Other Salmonella Infections. Edinburgh and London, Livingstone, Ltd, 1962, p 211
11. Hornick RB, Greisman SE, Woodward TE, et al: Typhoid fever: I. Pathogenesis and immunologic control. *N Engl J Med* 382:13, 1970