Staphylococcus and Streptococcus Carriers*

Sources of Food-borne Outbreaks in War Industry

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WHEREAS enteric infections form the principal type [1] of food-borne disease prevalent in Continental Europe, in the United States staphylococcal food poisoning [2] is probably the most common of all food-borne diseases. Since this disease is not reportable, we have no accurate knowledge as to the actual number of cases occurring annually. The control of food-borne disease is not only the concern of the epidemiologist [3] and of the public health officials, but also of every physician, food handler, and citizen, especially now that we are engaged in total war.

Gastrointestinal diseases are a major cause of absenteeism in industry. McGee and Creger [4] determined that 18.6 per cent of 40,942 days lost by employees of a Hercules Powder Company plant in New Jersey in 1941 were due to gastrointestinal disturbances. Of 5,402 absences from work, 24 per cent were due to this group of diseases. Of the 7,605 days lost from diseases of the digestive tract, 30.6 per cent were from gastrointestinal upsets and colon dysfunction, and 4.1 per cent from enteritis and dysentery. Most of the absenteeism from these causes occurred after week-ends and after holidays. We, ourselves, have repeatedly observed food poisoning disrupt a large war plant.

The health officer, the industrial physician, the industrial manager, the food handler, and the consumer himself play important rôles in controlling food-borne disease. Health officers are anticipating an increase in food-borne disease from public eating places and industrial cafeterias as a direct result of the war. More people are eating in restaurants because of the rationing of food and because of the placing of women in industry. There are increasing difficulties encountered in these restaurants. The lack of responsible personnel, trained in the sanitary and proper handling of food, the overburdening of the kitchen facilities, the unavailability of proper equipment, and, in some instances, the short cuts and improper processing of foods are the principal causes for the increase in food-borne disease. Horwood and Pesare [5] made sanitary surveys of public eating places in Rhode Island and concluded: "Ignorance of accepted

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sanitary requirements and procedures as manifested by the managers and employees of public eating and drinking establishments appeared to be the most important single cause of insanitary practices in such places."

In the investigation of any suspected food-borne outbreak, an analysis of the symptoms will often give the epidemiologist a clue as to the etiological agent. The enterotoxin of *Staphylococcus aureus* produces symptoms in 2 to 6 hours. Whereas fever is a common symptom of food-borne infections it is not a frequent finding in staphylococcus enterotoxin poison. Nausea, vomiting, abdominal cramps or pain and diarrhea leading to prostration are the usual rapid sequence in severe staphylococcal poisoning; chills, sweats, and toxemia are other frequent findings. The onset is more acute than in Salmonella infections, the duration is usually a matter of hours and recovery is rapid. In Salmonella infections, on the other hand, recovery is a matter of days.

In several instances, green-producing streptococci have been incriminated as the source of food-borne outbreaks. Linden, Turner, and Thom [6] reported two such outbreaks in both of which cheese was found to be the vehicle. *Streptococcus lactis* [7] when isolated from this food and fed to kittens produced symptoms similar to those caused by staphylococcus enterotoxin. Carey, Dack, and Meyers [8] isolated alpha-streptococci from canned sausages in an outbreak involving 75 persons. Living cultures of this organism fed to volunteers produced a syndrome clinically indistinguishable from staphylococcal food poisoning. Bacteria-free filtrates were non-toxic. Similar results were obtained by Carey, Dack, and Davison [9] in another outbreak caused by this organism. There is, therefore, some evidence that green-producing streptococci may cause gastroenteritis in man. However, unlike staphylococcal food poisoning, the toxic material is not contained in bacteria-free filtrates of this organism.

The clinical picture presented by this type of food infection resembles that of staphylococcal poisoning very closely. A differentiating feature is the longer incubation period found in outbreaks caused by green-producing streptococci. This usually varies from 5 to 18 hours, with an average of about 12 hours.

DEMONSTRATION OF THE ETIOLOGICAL AGENTS

The application of bacteriological procedures to the study of food poisoning must take account of the diverse etiological agents encountered in these outbreaks. Epidemiological and clinical data may suggest the identity of the causative agent as in botulism but are of less value in differentiating between the more common agents of food poisoning. Assuming the special technics necessary for the isolation and identification of the Clostridiae to be indicated or excluded clinically, the bacteriologist must provide technics adequate for the isolation and identification of: (1) staphylococci, (2) streptococci, (3) the enteric group. Staphylococci and streptococci were encountered in the outbreaks reported here. Members of the enteric disease group were excluded as possible causes by appropriate clinical and bacteriological examinations.

Nose, throat, and stool cultures were taken on all suspected food handlers. Swabs were cultured on 5 per cent horse blood agar and in beef infusion broth for 24 and 48 hours. Stool specimens were cultured on differential media for members of the enteric disease group.

Adequate epidemiological investigation usually reduces the number of foods suspected in a given outbreak. The foods thought to be responsible were brought to the laboratory im-

mediately or packed in dry ice for shipment.* Representative samples of each food were cultured in beef infusion broth on 5 per cent horse blood agar and differential media for the enteric disease group. Since chromogenesis varies with different media and cultural conditions, the differentiation of all staphylococci was made on the basis of pigment production on beef infusion agar incubated 24 hours at 37.5° C. No detailed study of colony variants was attempted.

The streptococci were classified according to the group-precipitation test described by Lancefield.[10] The serological type of Group A strains was determined by the slide-agglutination technic reported by Griffith.[11]

Casein hydrolysate filtrates were prepared according to the method of Favorite and Hammon.† [12] Filtrates were tested for enterotoxin by feeding to kittens and by the intravenous kitten test described by Hammon.‡ [13] Diarrhea and vomiting in 1 to 3 hours following administration of the filtrate was interpreted as a positive reaction.

The results of these toxigenic studies are summarized in Table 1. Unlike *Staphylococcus aureus,* the streptococci studied failed to produce an enterotoxic substance in casein hydrolysate but did produce a toxic substance in sterile tissue media which when administered intravenously to kittens induced an enterotoxic reaction. These substances could be readily distinguished from staphylococcus enterotoxin in that they were non-toxic by mouth. A detailed study of these tissue media filtrates has been reported by Foley, *et al.*[14] As has been the experience of others,[8, 15] whole cultures of streptococci produced enterotoxic reactions when fed to kittens.

There are conflicting reports as to the value of the various biochemical technics devised to distinguish enterotoxic strains.[16, 17] The "potential" enterotoxicity as judged by procedures other than the kitten test does not appear to be sufficient evidence to incriminate either a food or food handler as the source of illness.

Certain biochemical characteristics of the enterotoxic staphylococci isolated from foods and food handlers involved in these outbreaks are summarized in Table 1. Such tests are of some value in establishing the probable identity of strains but do not indicate enterotoxicity. There appears to be a rough correlation between alpha-hemotoxin titer and enterotoxicity as observed by Woolpert and Dack [18] but exceptions were noted.

ANALYSIS OF OUTBREAKS

It is well known that the staphylococcus and the streptococcus are ubiquitous in their distribution. They are found in the nose and throat of healthy persons where they become quite abundant during upper respiratory infections. They are readily isolated from superficial skin lesions and from routine air samples. In spite of modern aseptic surgical technics, they are often incriminated as the cause of postoperative stitch abscesses. It is, therefore not surprising that foods are often contaminated by them.

Our experience with staphylococcal food poisoning has demonstrated that it is by no means infrequent. The few outbreaks coming to the attention of public health authorities represent but a fraction of the actual number. Very often only the most dramatic incidents

* After bacteriological examination, samples were homogenized in a Waring Blender, mixed with milk or fish, and fed to kittens (4–6 months old) which had been starved the previous 24 hours. Typical "enterotoxic" reactions could be produced with contaminated foods by this method.

† In our experience, rotation was not essential to the production of an enterotoxin satisfactory for routine tests.

‡ Results of feeding experiments with original foods and casein hydrolysate filtrates agreed with those of the intravenous test with filtrates of the same strain.

TABLE 1

Summary of Epidemiological and Bacteriological Data, 18 Outbreaks, Food Poisoning

Outbreak	Microörganism	Source	Fermentation of: Trehalose	Sorbite	Lactose	Mannite	Salicin	Enterotoxin	Alpha Hemotoxin Titer	No. Exposed	No. Ill	Attack Rate
1	*Staph. aureus*	Food Handler	+	−	+	+	+	+	0.008	200	58	29.0
	"	"	+	−	+	+	+	+	0.008			
	"	"	+	+	+	+	+	+	0.008			
	"	Potato Salad	+	−	+	+	+	+	0.008			
	"	Ice Cream	+	+	+	+	+	+	0.008			
2	*Staph. aureus*	Food Handler	+	−	+	+	+	+	0.031 [1]	211	139	66.0
	"	Egg Salad	+	−	+	+	+	+	0.016 [1]			
3	*Staph. aureus*	Food Handler	+	+	+	±	+	+	0.125	Unknown	22+	Unknown
	"	Cream Filling	+	+	+	±	+	+	0.125			
4	*Staph. aureus*	Food Handler	+	+	+	±	+	+	0.125 [2]	Unknown	9+	Unknown
	"	Cream Filling	+	+	+	±	+	+	0.125 [2]			
5	*Staph. aureus*	Food Handler	+	+	+	+	+	+	0.125	2	2	100.0
	"	"Scotch" Ham	+	+	+	+	+	+	0.125			
6	*Staph. aureus*	Food Handler	+	+	+	+	+	+	0.004	800	180	22.2
	"	Boiled Ham	+	+	+	+	+	+	0.004			
7	*Staph. aureus*	Food Handler	+	+	+	±	+	+	0.008	Unknown	30	Unknown
	"	Corned Beef	+	+	+	±	+	+	0.008			
8	*Staph. aureus*	Food Handler	+	+	+	−	+	+	0.031	52	48	92.3
	"	Salad Dressing	+	+	+	−	+	+	0.031			
9	*Staph. aureus*	Food Handler	+	+	+	+	+	+	0.008	40	28	70.0
	"	Boiled Ham	+	+	+	+	+	+	0.008			
10	*Staph. aureus*	Food Handler	+	+	+	+	+	+	0.031 [1]	Unknown	60	Unknown
	"	Custard Pie	+	+	+	+	+	+	0.063 [1]			
11	*Staph. aureus*	Noodle Soup †	+	+	+	+	+	+	0.016	2	2	100.0
12	*Staph. aureus*	Boiled Ham Sand.†	+	+	+	−	+	+	0.008	70	17	24.3
13	*Staph. aureus*	Roast Turkey †	+	+	+	+	+	+	0.031	17	17	100.0
14	*Staph. aureus*	Boiled Ham †	+	+	+	+	+	+	0.008	3	3	100.0
15	* Beta Hemolytic Strep., Group A, Griffith Type 2	Food Handler	+	−	+	+	+	−− [3]	.....	192	102	56.0
		Boiled Ham	+	−	+	+	+	− [3]	.....			
16	* Alpha Strep. Lancefield, Group B	Food Handler	−	+	−	−	−	−− [3]	.....	Unknown	18	Unknown
		Cream Chicken	−	+	−	−	−	− [3]	.....			
17	* Alpha Strep. Lancefield, Group H	Food Handler	+	+	+	+	+	−− [3]	.....	6	6	100.0
		Baked Beans	+	+	+	+	+	− [3]	.....			
18	* Alpha Strep. Lancefield, Group B	Chop Suey †	−	+	+	−	+	− [3]		3	3	100.0

[1] Difference of 1 tube in titration

[2] Weak Enterotoxin in casein hydrolysate media. (Checked through kindness of Dr. G. M. Dack, University of Chicago.) Toxicity enhanced by culture in cream filling

[3] Whole cultures and tissue media filtrates enterotoxic for kittens

* Present in pure culture

† Food handlers not cultured

are reported. Sometimes newspaper accounts of such epidemics serve as the first notice to the health officer that something has happened. About half of the outbreaks listed in this paper took place in a single community. The interest of the health officer in this problem was a major factor in bringing most of these to light. We must assume that although rarely reported, similar episodes are a constant occurrence.

Certain of these outbreaks are more apt to become manifest than others. Institutional outbreaks, or those coming after a large gathering where a single meal common to a considerable number of persons has been incriminated, are readily detected. However, in other instances in spite of large numbers of cases, relatively few are reported. Outbreak 10 (Table 1) illustrates this point. The epidemiological investigation revealed that an infected pastry product had been shipped to numerous restaurants and diners in many different communities. For the most part it was sold in individual helpings. Although hundreds of potentially infected pies were distributed, only 60 cases came to our attention. For the most part these consisted of multiple cases occurring among small groups of persons who had eaten together at a single restaurant. In such groups the common source could be established without too much difficulty. " Car sickness " was considered the cause of one patient's illness with symptoms so severe that hospitalization became necessary. A newspaper account of the outbreak eventually led to the association of this case with the epidemic. In individual cases it is often very difficult to pick out any single food as the cause of symptoms. This factor alone tends to obscure the source of many outbreaks and is a definite asset to dirty and poorly managed food dispensing establishments.

The storage of food is an important factor in the morbidity rate. The longer the infected food is kept in a warm place, the greater the attack rates. In each of the outbreaks reviewed here, the epidemiological investigation revealed that the food was kept warm for a period of several hours. Thus certain procedures of food preparation may be the means of incubating pathogenic organisms accidentally introduced.

It has been assumed at times that bacteria harbored in the nose and throat of food handlers may be responsible for staphylococcal and streptococcal food-borne disease. Roberts and Wilson [19] in the study of one outbreak attempted to show that a strain of staphylococcus cultured from infected pastry was the same as that recovered from a food handler. Repeated cultures of this person revealed the same strain for a period of 4 months. Identical organisms were recovered from other food handlers in the bakery.

Our study demonstrates that staphylococcal food poisoning outbreaks may be traced to specific food handlers as sources of infection. In each instance where it was possible to culture the nose and throat of persons responsible for the preparation of an incriminated product, an apparently identical strain of *Staphylococcus aureus* was recovered from at least one of the food handlers and the infected food (Table 1). Although staphylococci are a universal contaminant of the environment, those strains harbored in the nose and throat of the food handler are invariably associated with outbreaks.

Figures collected from several sources [19] showed that the incidence of staphylococcus in the nose and throat of the general population was quite high, ranging from 76.3 per cent to 86.1 per cent in several different samples. Apparently all of these

strains were not chromogenic and only a portion of them were hemolytic.

Utilizing the kitten tests, we compared the incidence of enterotoxin-producing strains of *Staphylococcus aureus* in the nose and throat of food handlers involved in food poisoning outbreaks with that of other food handlers. There was a significant statistical difference between the two groups. Eighteen per cent of a total of 122 in the incriminated group harbored enterotoxin-producing staphylococci, while only 3.4 per cent of the control group of 146 carried such organisms.

Our study suggests that staphylococcal food-borne outbreaks are associated with a high enterotoxin-producing staphylococcus carrier rate among the incriminated food handlers. In four outbreaks, the incriminated strain was recovered from more than one food handler of a particular group. In one outbreak 3 members of the cafeteria personnel in an industrial establishment were found harboring the same organism as that isolated from the incriminated food. Moreover, this appeared to be the identical strain which had been recovered in a previous outbreak 7 months before at this very plant. Another group of food handlers was cultured 5 months after the occurrence of a staphylococcal food poisoning outbreak. Enterotoxin-producing staphylococci were isolated from 3 of the 21 members of this group.

Three of the reported outbreaks were attributed to green-producing streptococci. In two of these, apparently identical strains were recovered both from the incriminated food and the nose and throat of food handlers. Alpha-streptococci of Group H (Lancefield) were involved in one, and of Group B (Lancefield) in two. In each instance, kittens were made ill by whole cultures of the respective streptococcus, but not by its filtrate. The incubation period both in patients and kittens was prolonged as compared with staphylococcal food poisoning.

Green-producing streptococci belonging to Group H are a common inhabitant of the nose and throat.[20] The distribution of this organism should be borne in mind in the diagnosis of obscure food-borne outbreaks where the incubation period is somewhat longer than that of the staphylococcal outbreak and where no other bacterial or toxic agent is demonstrable. Alpha-streptococci of Group B are less frequently encountered [21] in routine nose and throat cultures. To our knowledge, these three outbreaks are the first reported food-borne epidemics produced by alpha-streptococci classified according to group-precipitation tests (Lancefield).

Outbreak 15 is of unusual interest. A beta-hemolytic streptococcus (Group A, Griffith Type 2) was established as the causative organism. The outbreak consisted of 24 cases of scarlet fever, 56 cases of septic sore throat, 7 cases of diarrhea, 7 cases of nausea and vomiting, and 8 cases of miscellaneous complaints. Gastrointestinal symptoms were unusually prevalent in all the patients. The identical strain of streptococcus was isolated from the throat of the responsible food handler, from the ground ham, and from the throats of several patients. Animal experiments and other laboratory procedures suggest that an enterotoxic substance may have been responsible for the unusually large incidence of nausea, vomiting, and diarrhea. Detailed descriptions of this outbreak and laboratory procedures are contained in other published reports.[14, 22]

Although three different groups of bacteria were involved in these eighteen food-borne outbreaks, carriers of a specific organism were the apparent source of infection in thirteen. In the five remaining outbreaks, food handling personnel were not cultured.

PREVENTION

One of the most effective methods of reducing food-borne diseases is the enforcement of proper personal hygiene practices by all food handlers. Keeping the hands away from the mouth and nose, covering the mouth with a handkerchief while coughing or sneezing, followed by washing the hands, covering foods whenever possible, refrigerating those that are perishable, reducing the interval between cooking and eating, eliminating food handlers with purulent wounds, boils, or infections of the hand, preventing food handlers with sore throats from preparing food—all these will reduce most of the instances whereby staphylococci and streptococci may contaminate food. One of the methods for controlling the ubiquitous staphylococcus in cream or cream-filled pastries is the rebaking of these delicacies as described by Stritar, Jungewaelter, and Dack.[23]

SUMMARY

Food-borne outbreaks in war plants may become an ever increasing cause of absenteeism among workers. Health officers, industrial physicians, and cafeteria managers working coöperatively may accomplish a great deal toward the prevention of food poisoning.

It must be borne in mind that although staphylococci and streptococci are found everywhere in the environment, food handlers carrying such organisms in the nose and throat are almost invariably responsible for food-borne outbreaks. In thirteen out of eighteen outbreaks reviewed in this paper, apparently identical organisms were recovered from incriminated food and the nose and throat of food handlers.

It is suggested that staphylococcal food-borne outbreaks are associated with a high enterotoxin-producing staphylococcus carrier rate among the incriminated food handlers.

Three of the outbreaks were produced by alpha-streptococci, the responsible organism in each instance having been classified according to group precipitation tests (Lancefield). One outbreak had as its etiological agent beta-hemolytic streptococci (Group A—Griffith Type 2).

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California Acts on Cheese-borne Typhoid Fever

According to a report recently released by Wilton L. Halverson, M.D., Dr.P.H., Director of Public Health of the State of California, approximately 76 cases of typhoid fever, all of Type C, have occurred in California during the period between April 9 and May 26. Seventy of these cases occurred in four counties, namely, Kern, Tulare, San Benito, and Monterey. Four additional cases have been reported from Nevada.

In view of the fact that all cases were of the same type and since all occurred during the same approximate time interval, it was assumed that the outbreak was from one source rather than from multiple sources.

Epidemiological investigation elicited the information that in various areas cases tended to trade at certain grocery stores. This was especially true in Bakersfield, where practically all of the 20 cases traded at one store.

The investigation elicited the information that the common factor was the ingestion of unripened, unpasteurized cheese of the Romano Dolce type, a type of cheese recently developed in California. When it became evident that well over 90 per cent of all the cases had ingested this type of cheese, a conference was called with representatives of the dairy industry, the University of California, the State Department of Agriculture, and the State Department of Public Health.

According to Dr. Halverson, after the data had been canvassed all concerned agreed that the evidence at hand called for immediate action, namely, the impounding of all cheese of this type as well as companion types manufactured in the same factories. To this end, action was immediately taken by the State Department of Agriculture, the regulatory agency for dairy products. An intensive investigation of the cheese factories producing cheese distributed in the areas concerned began. Because of complex and overlapping distribution systems, it was not possible to incriminate any single factory.

The previous call for an extraordinary session of the State Legislature for another purpose gave an opportunity to secure legislation to prevent a repetition. The Director of Health requested Governor Earl Warren to request in a supplementary call legislation which would correct this situation. The Governor immediately saw the importance of setting up safeguards to prevent a recurrence and as a result legislation was prepared, has been passed by both houses, and signed by the Governor, which provides that all cheese sold in the State of California must be pasteurized, must be made of pasteurized milk, or must be ripened or cured for a period of not less than 60 days. The bill also provides for proper labeling of the product whether manufactured in or out of the state, and contains an urgency clause which makes it effective immediately.

The short interval of time between the discovery of the cases and the cooperative action on the part of the State Departments of Agriculture and Public Health and the immediate action of the Legislature are notable.