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Staphylococcal food poisoning in the United Kingdom, 1969–90

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SUMMARY

Between 1969 and 1990 strains of *Staphylococcus aureus* from 359 outbreaks and sporadic cases of staphylococcal food poisoning in the United Kingdom were examined in the PHLS Food Hygiene Laboratory for the production of enterotoxin. In a number of instances the incriminated foods were also examined for the presence of enterotoxin. Strains from 79% of incidents produced enterotoxin A alone or together with another enterotoxin. The level of *S. aureus* present in the foods ranged from no viable *S. aureus* detected to $1{\cdot}5 \times 10^{10}$ c.f.u./g with a median of $3{\cdot}0 \times 10^{7}$ c.f.u./g. Enterotoxin was detected in foods in the absence of viable *S. aureus* in only two outbreaks and in both cheese was the implicated food. Meat, poultry or their products were the vehicle in 75% of incidents with ham and chicken most frequently implicated. Other foods included fish and shellfish (7%) and milk and milk products (8%). Most contamination took place in the home followed by restaurants and shops. Seventy-one percent of the incident strains were lysed by phages of group III or I/III.

INTRODUCTION

Staphylococcal food poisoning is an intoxication caused by the consumption of foods containing enterotoxins produced by certain strains of *Staphylococcus aureus*. The principal symptom is vomiting which occurs 1–6 h after ingestion of the contaminated food and is usually followed by diarrhoea. In most instances it is the foodhandler who contaminates the food. Between 30 and 50% of the population carry *S. aureus* and one third to one half of these carry enterotoxigenic strains [1]. If foods containing enterotoxigenic *S. aureus* are stored for long enough periods at temperatures which will permit bacterial growth, enterotoxin may be produced. To date, seven antigenically distinct enterotoxins have been purified: A, B, C_1, C_2, C_3, D and E. In this study 359 incidents of staphylococcal food poisoning which occurred in the UK between 1969 and 1990 are reviewed.

MATERIALS AND METHODS

Definitions

The definitions for cases, outbreaks, sporadic cases and incidents are described by the Public Health Laboratory Service Communicable Disease Surveillance Centre (PHLS, CDSC) [2] as follows: a *case* is a person with symptoms from whom the

relevant organism has been isolated or who has been affected in an outbreak of food poisoning; an *outbreak* is defined as two or more related cases of food poisoning; outbreaks are classified as family outbreaks, when they have occurred in one household or general outbreaks if more widespread; *sporadic cases* occur when an affected patient has had no known association with another person infected with the same organism; an *incident* refers to a sporadic case or an outbreak.

Strains and incriminated foods

Strains of *S. aureus* from 357 of 359 incidents of staphylococcal food poisoning and incriminated foods from 157 were received at the Food Hygiene Laboratory (FHL) from PHLS and hospital laboratories in the UK during 1969–90. In addition, strains of *S. aureus* and foods from incidents of unknown aetiology were also received.

Enumeration of S. aureus

The number of colony-forming units (c.f.u.) of *S. aureus* per g of food was determined by the surface drop method [3] using phenolphthalein diphosphate agar containing polymyxin [4] or Baird–Parker agar (CM 275, Unipath, Basingstoke, UK). Suspect colonies were tested for coagulase production in 10% human plasma broth.

Detection of enterotoxins and toxic shock syndrome toxin-1

All strains of *S. aureus* were tested for the production of enterotoxin A (SEA), B (SEB), C (SEC), D (SED) and E (SEE) by the double gel diffusion slide method [5]. From 1980 onwards the strains were also tested for the production of toxic shock syndrome toxin-1 (TSST-1) by gel diffusion or the reversed passive latex agglutination (RPLA) test (TST-RPLA, TD940, Unipath, Basingstoke, UK). Foods were tested for the presence of enterotoxin (SE) by the double gel diffusion slide test from 1969 to 1985 [6], by the ELISA developed by Notermans and colleagues [7] (ELISA-P) from 1982 to 1986 (for SEA and SEB only), with the ELISA kit from Dr Bommeli AG, Stationsstrasse 12, CH-3097 Berne, Switzerland (ELISA-B) and/or by the RPLA test (SET-RPLA, TD900, Unipath, Basingstoke, UK), from 1985 onwards. SEC_1, SEC_2 and SEC_3 cross-react [8] and no distinction is made between them in these tests. Neither the ELISA nor the RPLA kits contain reagents for the detection of SEE. Some foods and extracts were frozen and tested in retrospect.

Phage-typing

The phage-typing patterns of the strains of *S. aureus* were determined by the Staphylococcus Reference Unit, PHLS Division of Hospital Infection, Central Public Health Laboratory, Colindale using the International Basic Set of 23 phages [9].

Detection of thermostable nuclease

Foods were tested for the presence of thermostable nuclease by the toluidine blue–DNA agar method [10] with incubation of the slides at 37 °C overnight.

RESULTS AND DISCUSSION

Incidents and cases

The number of incidents of staphylococcal food poisoning reported to the Epidemiological Research Laboratory of the PHLS from 1969 to 1975 and to the PHLS-CDSC from 1976 to 1990 [11–15] and those reported to the Communicable Diseases Scotland Unit (CDS) [16, 17] for the period 1973–90 are shown in Table 1.

For the years 1969–81 the number of cases of staphylococcal food poisoning in England and Wales comprised 1–6% of the total cases of bacterial food poisoning and salmonella infection per year. Between 1982 and 1990 this proportion fell to 0·5–1% due to a dramatic increase in salmonella infection.

Over the 22-year period 1969–90 strains, and in 44% of the episodes, foods, from incidents of staphylococcal food poisoning in the UK were examined by the FHL. Three hundred and fifty-nine incidents were investigated, 325 from England and Wales, 27 from Scotland, 6 from Northern Ireland and 1 from the Channel Isles. These could be divided into 193 general outbreaks, 86 family outbreaks and 45 sporadic cases. No information was available for 35. Half of the incidents occurred during June, July and August each year. On average 10–15 incidents were reported annually, but in years with hot summers double this number occurred. The numbers of cases were known for 333 incidents. Over the 22-year period 163 incidents, involving 2520 cases, were reported during the months June to August with 170 incidents and 2316 cases occurring during the remaining 9-month periods, giving a total of at least 4836 cases.

In at least six outbreaks *Bacillus cereus* was also isolated from the incriminated foods and may have contributed to the illness [18–23].

The number of incidents of staphylococcal food poisoning which actually occurred is likely to have been higher than reported. Symptoms usually only last for a short period of time and most people will not seek medical advice and thus their illness will not be reported. Also, when complaints are made to a shop, restaurant or food manufacturer, the information may not be passed on to official organizations. The data reported therefore must be interpreted as an indication of trends in staphylococcal food poisoning.

Incubation periods and symptoms

The incubation periods of the illness were reported in 261 incidents and time ranges were given in about half of these. For many of the remaining incidents only average incubation periods were mentioned. In 91% of the incidents the reported incubation periods were between 2 and 6 h, in 10 a time as short as 1 h was recorded and in a further 10 as long as 8 h. Time ranges of 30 min to 2 h and 30 min to 4 h were given in two incidents and 2–12 h in another.

Symptoms were reported in 271 incidents, both vomiting and diarrhoea occurred in 198, vomiting only in 66 and diarrhoea only in 7. Nausea, stomach cramps and abdominal pain were also common complaints. Collapse, prostration or fainting featured in 10 incidents, fever in 2 and dizziness in 1. Sometimes the symptoms were so severe that hospital treatment was necessary. At least 49 cases required hospitalization.

Table 1. *Staphylococcal food poisoning: number of incidents in the UK*

Place and time		Total	Number of incidents: General outbreaks	Family outbreaks	Sporadic cases
England and Wales 1969–90	Reported to CDSC*	350	201	85	64
Scotland 1973–90	Reported to CDS	27	21	6	—

* Before 1976 reported to the Epidemiological Reference Laboratory.

Staphylococcal food poisoning is rarely fatal. It was recorded as causing or being a contributory factor in five deaths over the 22-year period. All victims were elderly people.

Incubation periods and symptoms reported tend to be unreliable as often only general terms are used to describe the illness and victims do not accurately remember times and symptoms. However, our results do give an average impression of these factors.

Incriminated foods

A food vehicle was recorded for most incidents (Table 2). As the incubation period is short it is not difficult for victims to recollect what they have eaten. Meat and meat products was implicated in 53% of the incidents with ham the most common vehicle. The meat pies included six pork pies. A variety of other meat containing products were also implicated including: luncheon meat (4), pâté (4), brawn (3) and vol-au-vents (4). Poultry and poultry products accounted for 22% of the incidents, most of these were attributed to cold cooked chicken and in nine incidents turkey was the vehicle of intoxication. The meat and chicken dishes categories included casseroles and stews, meat cooked with sauces, pasta dishes and Chinese and Indian foods. Fish and shellfish, milk and milk products and eggs accounted for 7, 8 and 3·5% of the incidents respectively. The shellfish implicated were mainly prawns (5). Trifles (6) and vanilla slices were among the sweet dishes causing food poisoning. Ice cream and raw milk were the food vehicles in one outbreak each. Pasteurized milk contaminated after opening the carton resulted in one sporadic case of illness. Three sauces are included under other foods. In a few outbreaks more than one food was contaminated with *S. aureus* and any of these could have caused the illness.

Sandwiches and rolls with various fillings were associated with at least 32 incidents. When eggs were implicated they were often eaten in sandwiches.

Foods from cans or jars were implicated in 63 incidents. These included: canned corned beef (19), chopped pork (7), salmon (6), tongue (5), ham (5), fish and shellfish (6), peas (2) and rice pudding (1), and meat, chicken and fish paste in jars (12). In one additional outbreak due to corned beef the beef was pressed.

In half of these incidents the food containers were not freshly opened or the time between opening and consumption of the contents was unknown. In the remainder the cans and jars were claimed to have been freshly opened. Often only one

Table 2. *Staphylococcal food poisoning 1969–90: implicated foods*

Type of food	Number of incidents	
Ham	65	53%
Meat pies	25	
Corned beef	20	
Tongue	16	
Jars of meat, chicken or fish paste	12	
Other meats and meat containing products	43	
Meat dishes	9	
Poultry (chicken, turkey, duck)	64	22%
Poultry dishes	15	
Fish and shellfish	24	7%
Milk and desserts containing milk or cream	23	8%
Cheese	5	
Boiled eggs and egg dishes	13	3·5%
Other foods	20	5·5%
Not known	5	1%
Total	359	

Table 3. *Staphylococcal food poisoning 1969–90: place of incident*

Place	Number of incidents
Private houses	106
Restaurants	31
Shops	26
Schools	23
Hospitals	19
Wedding receptions	18
Canteens	11
Outings (packed lunches)	11
Institutions	10
Parties	9
Other centres	13
Manufacturer	4
Other	22
Not known	56
Total	359

container in a batch of a food product causes illness, probably as a result of leakage into an individual can [24, 25].

Place of incident

About one third of the incidents, for which the place was recorded, occurred in the home (Table 3). Twenty-six outbreaks were associated with shops while meals in restaurants and at receptions and parties accounted for a further 58 incidents. Wedding receptions are probably so well represented in this latter group because the food is often prepared by non-professional caterers who are unused to and often ill-equipped for preparing food for large numbers of people. Shortage of cold storage space will result in bacterial multiplication and toxin production in the

Table 4. *Staphylococcal food poisoning 1969–90: SE production and phage groups of the implicated strains of* S. aureus

Enterotoxin produced	Total number of strains (%)	Phage group							
		I	II	III	I/III	V	95	other	NT
A	203 (57)	44	—	104	38	—	—	1	16
B	6	—	—	1	—	4	—	—	1
C	5	2	—	1	—	—	1	—	1
D	15	—	—	10	4	—	1	—	—
E	3	—	—	1	—	—	—	—	2
A+B	12	1	—	5	2	—	—	—	4
A+C	9	—	—	5	2	—	—	1	1
A+D	55 (15)	1	—	34	17	—	—	1	2
A+B+D	4	—	—	4	—	—	—	—	—
B+D	1	—	—	1	—	—	—	—	—
C+D	28 (8)	2	—	4	9	—	11	—	2
None	16	2	1	4	6	—	1	—	2
Total	357	52	1	174	78	4	14	3	31

An additional two outbreaks occurred where the food contained SEA, but no viable *S. aureus*. One representative strain from each outbreak was selected.

prepared foods. Hospitals and canteens were associated with 30 outbreaks. Twenty-three outbreaks occurred in schools and in half of these the number of cases was greater than 50. Institutions and other centres reported 23 incidents and outings, mainly coach parties of elderly persons, were associated with a further 11 outbreaks. In four outbreaks it was apparent that the food producer was at fault [26–28].

Microbiological investigations

Level of S. aureus *in foods from incidents.* The level of *S. aureus* present in the incriminated foods was determined in 231 outbreaks. The c.f.u. of *S. aureus* per g of food ranged from no viable *S. aureus* found to $1{\cdot}5 \times 10^{10}$ with a median of $3{\cdot}0 \times 10^{7}$. The count of *S. aureus* was more than 10^{6} c.f.u./g of food in 77% of the incidents. *S. aureus* was not isolated from foods in two outbreaks, although both these foods contained SEA.

SE and TSST-1 production and phage typing. The SE production and phage-typing groups of strains of *S. aureus* from 357 incidents are shown in Table 4. The strains were isolated from the implicated foods in 328 of the incidents. In the remaining 29 outbreaks foods were not available for examination and the strains were only isolated from clinical specimens. SEA alone or together with other SEs caused 79% of the incidents and SED alone or together with other SEs 29%. Production of SEA and of SED was mainly associated with lysis by phages of group III or I/III and strains from 71% of incidents were lysed by phages of these groups. Production of SEB was associated with lysis by phages of group V [29]. Strains which produced both SEC and SED were often lysed by phage 95.

The production of SEE by strains of *S. aureus* is rare and three outbreaks only were caused by this enterotoxin. In the FHL 31 SEE producing strains were found among over 3000 *S. aureus* strains isolated from foods and clinical sources. Strains which produced SEB or SEC alone were not often implicated. The production of

SEB and SEC is affected by growth conditions to a greater extent than that of SEA [1].

From 1980 onwards all strains were also tested for the production of TSST-1. This toxin was first called enterotoxin F [30], but its emetic activity in monkeys could not be repeated and it was renamed TSST-1 [31]. Although TSST-1 does not cause food poisoning, testing has continued, as TSST-1 production is an additional characteristic which can be used for the differentiation of strains. Its production is associated with lysis by phages of group I and the toxin is often produced together with SEA. Since testing for TSST-1 began, 32 of 148 strains from incidents were lysed by group I phages and 21 produced SEA together with TSST-1, 1 produced TSST-1 only and 10 produced SEA only. A further 7 TSST-1 producing strains were detected: 1 of 64 group III strains, 1 of 15 group I/III strains and 5 of 25 not typable strains produced TSST-1 together with SEA.

Production of SE was not detected in *S. aureus* strains isolated from implicated foods in 16 incidents with histories compatible with staphylococcal food poisoning. There are a number of possible reasons for this. Firstly these strains may have produced an as yet unidentified enterotoxin [1]: of six strains tested for the production of SE by monkey feeding tests, four gave positive reactions (Professor M. S. Bergdoll, University of Wisconsin, Madison, USA, personal communication). A second possibility is that the enterotoxigenic strain had been overgrown by a non-enterotoxigenic strain [1, 32]. Thirdly, strains may lose their ability to produce SE on storage, a factor which could be relevant in relation to canned foods. Tests on 197 strains, from separate incidents, stored for 1–9 years on nutrient agar slopes showed that 25 no longer produced detectable SE and 20 had lost their ability to produce one of the two SEs originally formed (A. A. Wieneke, unpublished data). In the present study canned foods were incriminated in six incidents from which only SE negative *S. aureus* was isolated and in four of these the cans were thought to be freshly opened suggesting a possible loss of enterotoxin producing capability. Finally, the food containing the SE negative strain may not have been the vehicle of intoxication or the illness reported was not staphylococcal food poisoning.

Over the period covered by this study an apparent shift occurred in the type of SE produced by *S. aureus* from incidents. Between 1969 and 1977 and between 1978 and 1990, 176 and 183 incidents respectively occurred. During the first period strains which produced SEA without another SE were incriminated in 49% of incidents while in the second period this proportion was 64%. This change was mainly due to a decrease in the production of SED: strains which produced both SEA and SED were isolated from 22% of the incidents in the first period and from only 9% in the second. For strains producing SED only these percentages were 7 and 1·5% respectively. No such decrease was noticed among strains producing both SEC and SED. Holmberg and Blake [33] also noticed a decrease in SED production when comparing strains from incidents occurring in the USA between 1967 and 1971 and those occurring between 1977 and 1981. They suggested that it was due to a decrease in milk associated outbreaks, as milk is a common source of SED producing strains. However, in the period studied by the FHL, meat products were incriminated in the majority of incidents due to SED alone or together with SEA.

Table 5. *SE detection in 15 foods where the strain of* S. aureus *present produced more than one SE*

SE produced by strain	SE detected in food	Number of foods tested where SE was detected by	
		ELISA-B	RPLA
A, B	A, B	2	1
A, B	A	1	2
A, C	A, C	1	
A, C	A	2	2
A, D	A, D	4	1
A, D	A	1	
A, B, D	A, B, D	2	1
A, B, D	D		1
C, D	D	2	2
Total		15	10

Incidents in which the incriminated strain produced SEB or the three toxins SEA, SEB and SED all occurred after 1980. The outbreaks due to SEE occurred in 1971, 1973 and 1976.

Carriers of enterotoxigenic strains of *S. aureus* are frequently the source of the implicated strain. The number of incidents where foodhandlers were tested was not known, but in 58 incidents one or more foodhandlers carried a strain which produced the same SE and was lysed by the same phages as the causative strain and in ten the strain was isolated from lesions, which were often obviously infected, on the hands or face of a foodhandler.

Detection of SE in samples of food

At the start of the period studied the FHL used the gel diffusion test for the detection of SE, but the sensitivity was low and concentration of the food extracts was required, with considerable loss in the recovery of SE [6]. In the early 1980s two sandwich ELISAs (ELISA-P and ELISA-B) and an RPLA test became available. Comparisons of the four methods have been published previously [34]. The ELISA-B emerged as the most sensitive method.

Based on one representative food per incident SE was detected in 71% of foods with c.f.u. of *S. aureus* of $\geqslant 10^6$ per g with the gel-diffusion method, in 74% with the RPLA, in 87% with the ELISA-P and in 91% with the ELISA-B. SE was detected in five foods with c.f.u. of $< 10^6$ per g by at least one of the four methods. Two of these foods were canned products and in another two the incriminated foods were sheep's milk cheese [28] and Halloumi cheese in which the *S. aureus* was not detected, but SE was still present.

When the implicated strain of *S. aureus* produced more than one SE, often only one could be detected in the sample of incriminated food. Fifteen foods from separate incidents which contained such strains of *S. aureus* were tested by ELISA-B (Table 5). In 9 of these foods all the enterotoxins produced by the strain were detected, in 4 SEA was detected but not SEB, SEC or SED and in 2 SED but

not SEC. Ten of these foods were also tested by RPLA (Table 5) and in 3 all enterotoxins produced by the strain were found, in 4 SEA was found but not SEB or SEC, in 1 SED without SEA and SEB and in 2 SED without SEC. These results are in agreement with the finding that the production of SEA is affected less by growth conditions than that of SEB and SEC [1].

With the ELISA-B SE was not detected in foods from eight incidents from which non-enterotoxigenic *S. aureus* only were isolated.

Samples of foods from incidents of unknown aetiology were sent to the FHL for SE tests for a variety of reasons, including presence of *S. aureus* in the suspect food; incubation periods and/or symptoms fitting staphylococcal food poisoning; the possibility that the food contained SE although *S. aureus* could not be isolated; and elimination of staphylococcal food poisoning from diagnosis. One hundred and twenty-nine foods from 111 such incidents were tested by the gel diffusion method, 24 foods from 20 incidents by ELISA-P, 88 foods from 76 incidents by ELISA-B and 175 foods from 123 incidents by RPLA. The foods included meat and meat products, poultry, fish and shellfish and milk products. SE was not detected in any of these foods. Enterotoxigenic *S. aureus* were found in foods from 6 incidents tested by gel diffusion, from 24 tested by ELISA-B and from 31 tested by RPLA. However, except for one sample of chicken in which the level of *S. aureus* was $2{\cdot}5 \times 10^7$ c.f.u./g, all these foods contained $< 5 \times 10^6$ c.f.u. of *S. aureus*/g.

The results of tests on foods from which *S. aureus* could not be isolated confirm the finding that the presence of enterotoxin in the absence of viable *S. aureus* is rare. Over the 22-year period covered by this study this occurred in only two outbreaks [28].

Thermostable nuclease (TNase)

Two studies [35, 36] have shown that TNase is detected in most foods with *S. aureus* levels of more than 1×10^6 c.f.u./g. The absence of TNase in foods with high counts of *S. aureus* may be due to the presence of proteolytic enzymes originating from the *S. aureus* strain, from other organisms or from the food system itself [37, 38]. Both high [39] and low [40] correlations between SE and TNase presence have been reported.

In the present study 423 foods were tested for the presence of TNase; the proportion of positive foods was related to the number of *S. aureus*/g (Table 6). No relationship was detected between the type of food and the results of the TNase tests. TNase was found in 24 foods containing $< 10^6$ *S. aureus*/g. Seven of these foods were incriminated in outbreaks and large numbers of *S. aureus* had probably been present at some stage, which makes the proportion of false positive reactions less than 6%.

When considering only foods from incidents of staphylococcal food poisoning (one representative food per incident) 68 of 102 tested gave a positive TNase reaction. Fifty-four of the 102 foods were tested for SE by ELISA-B. *S. aureus* isolated from these foods all produced SE. TNase and SE were detected in 33 of the foods. These included two cheese samples in which *S. aureus* was not detected. Eight foods were negative in both tests, in 12 SE was detected without TNase and in one TNase without SE.

Table 6. *The presence of thermostable nuclease in foods in relation to the number of* S. aureus/*g*

C.f.u. of *S. aureus*/g	Number of foods*	
	Total	TNase detected
Not found	159	13 (8)†
1×10^2–$9{\cdot}9 \times 10^4$	116	6 (5)
1–$9{\cdot}9 \times 10^5$	21	5 (24)
1–$9{\cdot}9 \times 10^6$	34	14 (41)
1–$9{\cdot}9 \times 10^7$	36	18 (50)
1–$9{\cdot}9 \times 10^8$	37	25 (68)
1–$9{\cdot}9 \times 10^9$	20	19 (95)

* Duplicate foods were excluded.
† Percentage in parentheses.

Although the TNase test was positive in only 67% of foods from outbreaks and in 73% of SE positive foods, it is in certain circumstances a quick and simple test to use in the investigation of outbreaks, as demonstrated by the ewe's milk cheese outbreak [28] where a positive test indicated that at some stage large numbers of *S. aureus* had been present, although the organisms could no longer be isolated.

Examples of outbreaks

In a classic situation strains of *S. aureus* which produce the same SEs and which are indistinguishable by phage-typing, are isolated during the investigation of an outbreak from the suspected food, from patients and from a person or persons concerned in the preparation of the food. In addition the SE produced by the strain is detected in a sample of the implicated food (example 1, below). As many different strains of *S. aureus* are able to produce SE, phage typing is important for the correlation of strains from different sources; in example 2 phage typing could distinguish between SEA producing strains isolated from two foodhandlers. This outbreak also shows the usefulness of testing for TSST-1 to distinguish strains. Contamination of food occasionally occurs at the production stage as shown in examples 3 and 4. A few outbreaks were probably due to both *S. aureus* and *B. cereus* toxins, one such outbreak is described in example 5. The two outbreaks where SEA was detected in the food without viable *S. aureus* are described in examples 6 and 7. The final example is a recent outbreak where food was transported by car on a hot day.

(1) In July 1975, 71 of 147 pupils and staff became ill after a school lunch that included cooked chicken [9]. The outbreak strain produced SEA and was lysed by phages of groups I/III. It was isolated from the chicken, the patients and one foodhandler. SEA was detected in left-over chicken. The chicken had been thawed overnight and roasted the next day. It was then stored at ambient temperature for various periods of time and served cold the day after.

(2) In August 1978, 9 of 39 elderly people on a coach trip were taken ill several hours after eating pressed pork and chicken at a restaurant en route [9]. Samples of the food consumed were no longer available, but an SEA-producing strain, lysed by phages of group I, was isolated from all nine patients. A strain indistinguishable from the patients' strain was isolated from a foodhandler. A second foodhandler

also carried an SEA-producing strain, but his strain was not typable (NT) by phages. Tests carried out in 1983 showed that the NT strain produced TSST-1, but the group I strain did not.

(3) In 1979 more than 85 people in 9 incidents became ill over a 5-month period after eating corned beef from freshly opened 6 lb cans all from the same production plant in Brazil [26]. *S. aureus* was isolated from all incidents and four of the isolates produced SEA and were lysed by phage 29.

(4) Dried lasagne sheets produced in Italy were the source of an international outbreak in 1984 and in the UK 10 separate incidents were reported with 47 cases [27]. A strain of *S. aureus*, which produced SEA and was lysed by phages of group III was isolated from the lasagne.

(5) In June 1988 at least 72 businessmen in the City of London became ill after a lunch of mince, egg, rice and garnish provided by a mobile caterer [23]. The prepacked food was prepared the night before in a kitchen under unsatisfactory conditions and was left at ambient temperature for at least 9 h before consumption. An SEA-producing strain lysed by phages of groups I/III was isolated from the food. An identical strain was isolated from the patients and from the nose of one of the foodhandlers. SEA was detected in all samples of food tested. Several strains of *B. cereus* were also isolated from the food and were serotypes which are recognized to be associated with food poisoning. This outbreak was attributed to SEA, with a probable contribution from *B. cereus* toxin.

(6) In 1981 a family of four ate Halloumi cheese in brine imported from Cyprus. All four became ill with typical symptoms of staphylococcal food poisoning. SEA was detected in the cheese and brine but *S. aureus* could not be found.

(7) In December 1984 and January 1985 cheese made from raw ewe's milk by a farm dairy was implicated in at least 3 outbreaks with 27 cases [28]. SEA was found in the cheese but *S. aureus* was not detected. During subsequent examination of samples of milk from the dairy an SEA-producing strain was isolated.

(8) In August 1990, 24 delegates attending a symposium became ill after a meal on a boat during a day trip. The food had been prepared by a private caterer and was taken to the boat by car on a hot day. A strain of *S. aureus* which produced SEC and SED and was lysed by phage 95 was isolated from the implicated chicken and rice dish. SED was detected in the sample of food.

Over the period of this study there has been some evidence of a downward trend in the number of outbreaks and subsequently of cases. This may be due, particularly in latter years, to the increased public and media awareness of other forms of food poisoning, which may have overshadowed other incidents such as staphylococcal food poisoning.

However, it is of importance to be aware of the possibility of the occurrence of widespread outbreaks of staphylococcal food poisoning and continuous vigilance is essential.

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