

## The bacteriology of food poisoning

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A good indication of the bacterial causes of food poisoning in this country is given in Table 1 taken from the reports of the Public Health Laboratory Service (Public Health Laboratory Service, 1956). In 1955, for example, the P.H.L.S. investigated 612 general outbreaks, 723 family outbreaks, and 7626 sporadic cases (Public Health Laboratory Service, 1956).

Table 1. *Causal agents of food poisoning in Britain in 1955*  
(Public Health Laboratory Service, 1956)

Presumed cause	Outbreaks	Family outbreaks	Sporadic cases	All incidents
<i>Salmonella typhi-murium</i>	82	375	3742	4199
Other salmonellas	41	129	900	1070
Staphylococci	64	40	34	138
<i>Clostridium welchii</i>	76	9	5	90
<i>Clostridium botulinum</i>	0	1	0	1
Other organisms	5	3	4	12
Chemical	3	1	0	4
Not discovered	341	165	2941	3447
All agents	612	723	7626	8961

From the figures it would appear right to concentrate most attention upon salmonellas, staphylococci, and *Clostridium welchii*. It is necessary also to consider briefly other organisms known to cause food poisoning. Of these by far the most interesting, as well as the most deadly, is *Clostridium botulinum*, the organism that caused the tragic disaster at Loch Maree in August 1922, when all of eight patients affected died within a week of eating sandwiches containing a 'wild duck' paste. Bacteria of the genus *Shigella*, responsible for one form of dysentery, are sometimes spread by food; and there are other organisms whose status as agents of food poisoning is a matter of further investigations and discussion—for example members of the genera *Proteus* and *Bacillus* as well as other bacteria, of which some are normal inhabitants of the intestine. It may well be that any organism that grows abundantly enough in a food may do some harm to it and may be a potential danger. For a discussion of these and many other problems of food poisoning the reader is referred to a valuable symposium held by the Society for Applied Bacteriology (1955).

### *Salmonellas*

Savage (1956), in an admirable and authoritative review of salmonella food poisoning, emphasized the dominance of these organisms as causal agents. Their

elimination or material reduction would bring food poisoning down to manageable limits. Salmonellas are Gram-negative rods which, although they do not form spores, are fairly hardy and can survive for quite long periods outside the body. They grow rapidly in a great many human foods. By refined methods of antigenic analysis, over 300 different types have now been identified. Determination of these types, especially if, phage typing is used as well as serological typing, is a specialized laboratory exercise, whose organization and the labour involved are justified because typing is of the greatest value to those seeking to trace the spread of infection. Of salmonellas isolated from cases of food poisoning in this country *Salmonella typhi-murium* is by far the commonest. Of other salmonellas known to bacteriologists *Salm. typhi* and *Salm. paratyphi* may be spread by food, milk, or water, and they give rise to an illness whose main clinical features are those of a continued febrile illness, rather than of typical acute food poisoning, which is the subject under discussion. Salmonellas commonly produce intestinal disease in birds, pigs, cattle, rats, mice, cats and dogs. Horses and sheep are less often affected. Infected animals that recover may become carriers, and there is thus abundant opportunity for man to become infected. Birds, pigs, and cattle, and their products eggs, meat and milk, are commonly used as human food, which may thus be infected from its source. Rats and mice are frequent contaminators of human food; and dogs and cats often live far too close to their masters' tables. It is not surprising, therefore, that salmonellas are such a frequent cause of human food poisoning. The part played by human carriers in spreading infection is slight compared to that attributable to animal carriers, being greatest and most certainly significant for *Salm. typhi* and *Salm. paratyphi*. On the whole, human carriers of salmonellas are more often the victims than the causes of a food-poisoning incident. In salmonella outbreaks in this country, meat, milk, artificial cream, and eggs are the commonest vehicles of infection. In particular, imported egg products—dried-egg powders, dried egg albumen, frozen whole egg, and frozen egg albumen—are common sources of infection. These products are widely distributed to bakers and confectioners and are often used to make up articles of food that are little heated in preparation. Made-up meats, including meat pies, are common, well-recognized sources of infection (Public Health Laboratory Service, 1956). There is no mystery or difficulty in explaining the bacteriological issues involved. As I have emphasized, the foods that we distribute and eat are often contaminated with salmonellas. The bodies of salmonellas contain thermostable lipopolysaccharides which are toxic to man, giving rise to gastro-intestinal irritation (vomiting, diarrhoea) and general symptoms of poisoning (headache, giddiness, raised temperature and collapse). The only question is whether a particular article of food contains enough salmonellas at the time when it is eaten to set up symptoms and permit the organisms to establish themselves for a time within the human intestine. Compared to other bacteria, relatively large numbers of most salmonellas are required to initiate infection. The mere presence of food-poisoning bacteria in a food is not the whole matter; often it is their opportunity to grow freely within the food that causes trouble. Too much should not be made of actual figures purporting to show the numbers of salmonellas

required to produce infection in man, because the range is from 125,000 to about 50 million (McCullough & Eisele, 1951*a-c*) depending on the species, the strain, the food vehicle, and the human subject; but it is clear that many human beings can ingest from 2000 million to 4000 million salmonellas and suffer only very mild diarrhoea or nothing at all. A great many meat pies, custards and trifles will have salmonellas in them but in numbers too few to produce symptoms. If the whole of one of these dishes is not consumed, however, and the food is set aside in a mild or warm room temperature, the salmonellas will increase logarithmically and in a few hours the food will become dangerous to anyone who eats it. The bacteriology of this type of food poisoning dictates, therefore, that its prevention involves the following issues: (1) control over the distribution of foodstuffs likely to be contaminated with salmonellas; (2) higher cooking temperatures and lower storage temperatures wherever possible (more use of pressure-cookers and refrigerators) for foods likely to contain salmonellas; and (3) more combined operations by public-health officers and bacteriologists to discover much more precisely which food ingredients carry the greatest risks.

### *Staphylococci*

Staphylococci are hardy but not extremely resistant Gram-positive organisms carried in the nose of many normal people. According to Dack (1956) staphylococcus food poisoning is the type most frequently diagnosed in the U.S.A. Not surprisingly, therefore, Dack's account of the condition is both full and illuminating. As he points out, the ability of some strains of *Staphylococcus aureus* (= *Micrococcus pyogenes* var. *aureus*) to produce a toxic substance (enterotoxin) capable of irritating the gastro-intestinal tract has been 'discovered' four times—in 1894, 1907, 1914 and 1930. A notable feature of staphylococcus food poisoning is the shortness of the interval that often elapses between ingestion of the food and the first symptoms—often a matter of 2–5 h. This is because the enterotoxin responsible for the trouble has already been formed and liberated during growth of the staphylococci within the food. This may be compared with salmonella food poisoning, in which the poisonous substances are retained within the bacterial bodies to be released only on their disintegration. In a patient this happens only some hours after an infection has been established. One great obstacle to the investigation of staphylococcus food poisoning is that there is no better method of identifying the toxin. Tests with kittens, puppies, monkeys and other animals have not given consistent results, and bacteriologists have so far been unable to discover a better method of identifying the toxin than to isolate the suspected staphylococcus, grow it in culture, and offer 2–10 ml. of the culture filtrate to human volunteers, in some of whom it reproduces the typical symptoms: nausea, vomiting, abdominal pain, diarrhoea, cramps, and faintness. Isolation of the responsible staphylococcus from the food is not always possible for it may be killed by cooking temperatures which do not destroy the toxin. In uncooked foods or foods contaminated after cooking, enormous numbers of staphylococci may be present and are easily isolated. Their typing by 'phage, which is a complicated and troublesome procedure not within the resources

of most laboratories, may be helpful in indicating whether the suspect organism belongs to 'phage group III, the one that contains the food-poisoning strains. 'Phage typing is an essential part of any serious attempt to discover the source from which the food was contaminated.

In staphylococcus food poisoning, unlike salmonella food poisoning, the human carrier is extremely important. Staphylococci capable of producing enterotoxin are often carried in the human nose and on the human skin. They are the usual cause of septic fingers and superficial abscesses and thus are frequent contaminants of food and milk. Such staphylococci may also be found in cow's milk, although it is important to emphasize that harmless staphylococci and non-food poisoning strains of *Staph. aureus* are common, normal inhabitants of milk. Fortunately it is only when conditions favour growth and toxin production in the food that food poisoning results. A few hours' growth at summer and autumn temperatures, however, is enough (Anderson & Stone, 1955). In most outbreaks of staphylococcal food poisoning the foods concerned have been milk products, particularly cakes and éclairs filled with cream or custard, or made-up, processed, or canned meat (Wilson & Miles, 1955, p. 1809). Boiled ham and salted meats seem to be particularly favourable to the growth of staphylococci. In view of the importance of synthetic cream as a vehicle for food-poisoning bacteria, salmonellas as well as staphylococci, Hobbs & Smith (1954) presented good evidence and arguments in favour of adding hydrogen peroxide, from 0.005 to 0.02%, as a preservative. Notwithstanding the many protests likely to be advanced against reintroducing the principle of adding preservatives to cream, Hobbs & Smith cogently argued in favour of their proposal by citing the numerous sources of contamination in bakeries from which synthetic cream cannot be protected. The whole question of bacterial contamination of foods has been comprehensively reviewed by Hobbs (1954). So far as staphylococci are concerned, apart from raising again the question of the possible use of preservatives, the bacteriologist must emphasize how difficult it may be to establish the precise identity of this infection and to prove its source. A notably difficult bit of detection, for example, was required to incriminate staphylococcus toxin as the cause of food poisoning spread by spray-dried milk (Anderson & Stone, 1955). People who are heavy nasal carriers of food-poisoning types of *Staph. aureus* are a source of danger; so are food handlers with septic lesions about their persons. Cooked food that has been sneezed upon or touched by a hand with *Staph. aureus* upon it may be very heavily contaminated with staphylococci. Their presence in the food will become highly significant if they are enterotoxin producers and if they are allowed a few hours at warm room temperature in which to grow and produce their toxin. A comfortable kitchen and the sunny window of a baker's shop are excellent substitutes for a bacteriological incubator.

#### *Clostridium welchii*

*Clostridium welchii* is a Gram-positive, sporing, anaerobic bacillus. Because it forms spores it has a higher grade of resistance to heat than salmonellas or staphylococci. The form of *Cl. welchii* responsible for food poisoning differs only a little,

but differs in significant points, from the modal type-A *Cl. welchii* found as a natural inhabitant of the intestine of man and many animals. In particular, the spores of the modal type-A *Cl. welchii* are destroyed by boiling in water (100°) in 5 min, whereas those of the food-poisoning variant resist 100° for an hour or more. The food-poisoning variant also differs from the modal type-A *Cl. welchii* in producing non-haemolytic colonies on blood-agar plates and rather small amounts of lecithinase ( $\alpha$ -toxin) in fluid cultures. Whereas the modal type-A *Cl. welchii* may be grown from almost any sample of human faeces, the food-poisoning (heat-resistant) variant is carried by only about 2–5% of normal healthy people (Hobbs, 1954). After an outbreak of this type of food poisoning the heat-resistant form may be grown from the stools of about 90% of those who have eaten the contaminated food. There is some uncertainty about how this organism acts: like the salmonellas by liberating endotoxin within the intestine, like staphylococci by releasing a specific enterotoxin (unidentified), or by producing a non-specific toxic breakdown product from the food. The usual incubation period of 8–18 h suggests that it must actually establish an infection within the intestine. There is no mystery, however, about how the organisms gain access to food or under what conditions they multiply sufficiently to cause trouble. *Cl. welchii* is a normal inhabitant of the soil and consequently also of human and animal intestines. From these two sources it is very widely distributed in carcass and poultry meat, water, milk, dust, and sewage. It is also commonly found on blowflies. The foods responsible are contaminated meats that have been heated on one day and not eaten until the next day. The heat-resistant spores of *Cl. welchii* survive cooking and thereafter germinate and grow rapidly while the cooling food passes through the temperature range from about 40 to 20°. Growth of the organisms is greatly assisted by the presence of gravy or other sources of moisture, so the infection is commonest in meats that have been boiled, stewed, or steamed (see, for example, Knox & Macdonald, 1943; McClung, 1945; Hobbs, Smith, Oakley, Warrack & Cruickshank, 1953; Collee, 1954; Smith & Wallace, 1956). Large steak-pies and shepherd's pies provide excellent conditions for the growth of *Cl. welchii*. Survival of spores is favoured when the food is cooked in large portions because meat is a poor conductor of heat. Large bulks of meat—as in canteen stews, for example—are also favourable for growth of the organism because they effectively prevent penetration of oxygen from the air and thus provide the anaerobic conditions required for germination and growth. Cooking, of course, has the effect of driving off oxygen and of removing competition for growth requirements by killing non-sporing organisms, most of which are soon destroyed at or above 60°. Meat is rich in the nutrients required by *Cl. welchii* and in reducing substances which favour growth by lowering oxygen tension. The bacteriology of *Cl. welchii* food poisoning thus clearly shows how the trouble may be prevented. Meat should be cut into small enough portions to ensure penetration of heat to the centre; short periods of pressure-cooking at 105–120° are better able to destroy *Cl. welchii* than longer periods of steaming, boiling, or stewing; and these shortened periods of cooking may allow more meat dishes to be eaten immediately after they have been cooked. If the meat must be cooked the day before it is eaten, it ought

to be cut in small pieces and cooked in a pressure cooker; it should not be stored in a moist state or covered with gravy; and it must be cooled as rapidly as possible. Warming-up temperatures are not sufficient to make the food safe. The organism is easily isolated from the victims of this form of food-poisoning if a small portion of faeces is heated at 100° for 1 h in Robertson's meat broth—a medium prepared in much the same manner as some canteen stews. The organism is not isolated by the same method from the food itself because there the spores have germinated and the organism is in the non-heat-resistant vegetative form; resporulation is accomplished during passage through the intestinal tract. However, the heat resistance of *Cl. welchii* cultures isolated from suspected food may be established by methods such as those used by Smith & Wallace (1956), and their serological identity with the strains isolated from the faeces of victims may be examined by agglutination tests and by toxigenic analysis (Hobbs *et al.* 1953; Oakley & Warrack, 1953).

Food poisoning due to *Cl. welchii* type F (Oakley, 1949) may evidently be a serious and rapidly fatal event (Zeissler & Rassfeld-Sternberg, 1949). Happily it appears to be extremely rare.

#### Clostridium botulinum

*Cl. botulinum*, also a Gram-positive sporing anaerobe, differs as a food-poisoning agent from *Cl. welchii* in being a much stricter anaerobe, in having a much more heat-resistant spore, and in forming the most potent exotoxin known to bacteriologists. The lethal dose of purified toxin for man is estimated at 0.01 mg or less. The status of botulism as a world-health problem has been fully reviewed (ninety-five references) by Meyer (1956) whose long and devoted work in this field has given his views a unique standing.

Botulism is food poisoning in a true sense: a powerful toxin is produced by growth of the causal organism in the food. The organism does not establish an infection in the human body. When the poisoned food is eaten, the powerful toxin is absorbed from the stomach and characteristic symptoms follow in from 24 to 72 h. The leading symptoms and signs are the result of the action of the toxin upon peripheral nerve endings: they are vomiting; ataxia; constipation; paralysis of eye movements, swallowing, and speech; and secretion of thick saliva. The patient remains conscious and aware of events until near the time of death, which is generally within a week of the onset of symptoms. Not all sufferers die, although the case fatality rate is high as a rule: 63.7% in U.S.A. from 1899 to 1954, but in Europe as low as 1.5–8% in France and 10–19% in Germany. Meyer (1956) was able to trace records of 5635 cases with 1714 deaths over the past 50 years. Our experience of the disease in this country is very slight: only seven episodes with fifteen cases and thirteen deaths from 1922 to 1954 (Meyer, 1956). In 1955 there were two non-fatal cases in London due to pickled fish prepared in Mauritius and brought to London by a family friend as a special delicacy (Public Health Laboratory Service, 1956). The dramatic and tragic events of the Loch Maree incident in 1922 are fully and movingly recorded by Leighton (1923). Invariably botulism is caused by

avoidable faults in the preparation and preservation of foods. Emphasis in prevention is laid upon adequate heat treatment of commercially canned meats; adequate sealing of tins; sufficient heating of home-preserved vegetables and fruits, especially the non-acid variety; and the discouragement of local customs which favour the eating of preserved foods in an uncooked state in the form of salads, watery conserves, poorly cured or inadequately smoked pork, and salted-fish products. It is worth mentioning that horses, cattle, sheep, chickens, and ducks may suffer botulism through eating badly preserved fodder or silage, or carrion. (For a brief but useful review see Wilson & Miles, 1955, p. 1824).

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### Inspection of food

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In this age of progress it would indeed be surprising if food-inspection technique had not also undergone change. Yet, it is true to say that many of the basic principles of food inspection have only required modification to keep pace with some new food treatments. Other instances, however, demand such highly specialized and detailed knowledge that fair controlling legislation can only be made after consultations with scientists, doctors, bacteriologists, dietitians, analysts, veterinarians and inspectors.

I will refer first to three definitions in my paper.

(1) 'Food' means food as defined in the Food and Drugs Act 1955 and Food and Drugs (Scotland) Act 1956, i.e. 'food' includes drink, chewing gum and other