The roles played in the spread of hepatitis in the Bristol outbreak by housing type, population density, sanitary provision, social class, play situation, locality itself, and the possible roles of dental shedding and virus transfer to water reservoirs by migrating wildfowl have been referred to elsewhere (Bothwell (1963a, 1963b).

Summary

An outbreak of 2,395 cases of infectious hepatitis in Bristol is recorded, and investigations of the 2,107 cases in 1960 and 1961 are described. The outbreak reached its maximum between October, 1960, and January, 1961, and had subsided by the end of 1962. It appeared to be part of a world-wide increase in the incidence of the infection.

The age and sex incidence was similar to that in previous British studies—that is, predominance in 5–10-year-olds—but differed from that reported from Canada. The incubation period was usually about a month, so that the outbreak can be attributed to virus A.

The spread of cases from central wards to peripheral wards may simply be an indication of concentration of susceptible sections of the population in these areas. Definite or strong presumptive contact history was available in 76% and 87% respectively of two groups checked.

There was no distinct evidence that poor hygiene was a factor in the spread of the disease. School meals and water supplies were not incriminated as spreading infection, nor were prophylactic injections. The implications of such an outbreak for blood-transfusion departments are considerable.

Three patients died, but it appears that permanent liver damage is seldom a sequel in survivors, so far as current follow-up studies extend.

Supplies of gamma-globulin would have to be more extensive than at present to cope with this type of outbreak. Statutory notification for Britain as a whole would help in the detection of early cases and would enhance the chances of control by gamma-globulin.

We are glad to acknowledge the assistance of senior administrative education, medical, and nursing colleagues, district health visitors, head teachers, and school welfare officers. Among hospital staff, who readily gave us access to records, we are particularly grateful to Dr. J. Macrae and Dr. A. E. Read. The success of the inquiry was made possible by the co-operation of the general medical practitioners in Bristol.

REFERENCES

- Bothwell, P. W. (1963a). *Brit. med. J.*, **2**, 241.
 — (1963b). W.H.O. Working Paper (October, 1963).
 Boggs, J. D., Capps, R. B., Weiss, C. F., and McLean, I. W. (1961). *J. Amer. med. Ass.*, **177**, 678.
Brit. med. J., 1963, **1**, 343, 1494.
 Capps, R. B., Bennett, A. M., and Stokes, J., jun. (1952). *Arch. intern. Med.*, **89**, 6.
 Cullinan, E. R., King, R. C., and Rivers, J. S. (1958). *Brit. med. J.*, **1**, 1315.
 Findlay, G. M., Martin, N. H., and Mitchell, J. B. (1944). *Lancet*, **2**, 301, 365.
 Flanagan, K. T., and Lister, J. (1962). *Brit. med. J.*, **2**, 376.
 Krugman, S., Ward, R., and Giles, J. P. (1962). *Amer. J. Med.*, **32**, 717.
Lancet, 1962, **2**, 491.
 Raska, K., and Radkovsky, J. (1959). *J. Hyg. Epidem. (Praha)*, **3**, 365.
 Rightsel, W. A., Keltsch, R. A., Taylor, A. R., Boggs, J. D., and McLean, I. W. (1961). *J. Amer. med. Ass.*, **177**, 671.
 Smith, C. M. (1961). *Lancet*, **2**, 818.
 Tilley, J. H. M. (1960). *Brit. med. J.*, **2**, 1354.
 World Health Organization (1953). *Techn. Rep. Ser.*, No. 62.
 U.S. Department of Health Education and Welfare. Expert Committee on Hepatitis (First Report).

OUTBREAK OF CLOSTRIDIUM WELCHII FOOD-POISONING

BY

WILFRID H. PARRY, M.D., D.P.H.

Medical Officer of Health, Altrincham M.B., Bowdon, Hale, and Knutsford U.D.s, Bucklow R.D.; Divisional Medical Officer, Cheshire County Council

Clostridium welchii as a cause of food-poisoning was first described by McClung (1945) when he reported a series of four outbreaks due to chicken dishes cooked the day before they were served. Since then many outbreaks have been reported, and the recorded cases of food-poisoning attributed to this organism in England and Wales have gradually increased from 20 in 1952 to 110 in 1959, then falling to 82 in 1961. In the period 1954–61, 257 deaths from food-poisoning of all types occurred, only 16 (6.2%) of which were attributed to *Cl. welchii* (*Mth. Bull. Minist. Hlth Lab. Serv.*, 1954–62).

Cockburn (1962) recorded that between 1949 and 1960 *Cl. welchii* was responsible for 1% of all incidents, 11% of general outbreaks, 1% of family outbreaks, and less than 1% of sporadic cases of food-poisoning in England and Wales. More recently the *Monthly Bulletin of the Ministry of Health* (1962), in recording the incidence of food-poisoning in England and Wales for 1961, reported the incidence of *Cl. welchii* for that year as 2% of all incidents, 27% of general outbreaks, 1% of family outbreaks, and less than 1% of sporadic cases. An analysis of these figures to show the percentage distribution by causal agents (excluding the incidents where there was no definite knowledge of the agent) showed that *Cl. welchii*

was still responsible for 2% of all incidents, but 37% of general outbreaks,* 2% of family outbreaks, and less than 1% of sporadic cases. Incidents due to *Cl. welchii* were said to have fallen by 14% compared with 1960 and 25% compared with 1959.

Throughout, the common vehicle of infection has been meat or meat dishes, and reheated meats were responsible for more than half the investigated outbreaks.

In the U.S.A. *Cl. welchii* has generally been ignored as a cause of food-poisoning, until Kemp *et al.* (1962) showed that there was a fourfold increase in reported cases of *Cl. welchii* in the State of California between 1959 and 1961. Twenty-six persons were affected in 1959, 108 in two outbreaks in 1960, and 464 in five outbreaks in 1961. In 1961 *Cl. welchii* accounted for 5.5% of the total reported outbreaks in California and 17.5% of the total persons affected.

In December, 1962, 41 cases of gastroenteritis (three fatal) occurred in a geriatric hospital welfare home in Knutsford, Cheshire. The causal organism was *Cl. welchii*; ham was thought to have been the vehicle of infection, being the only food common to all the victims.

*This change is probably due to increased awareness of *Cl. welchii* food-poisoning and to improved techniques.

As ham is an unusual vehicle for *Cl. welchii* the following details of the outbreak are reported.

Description of Outbreak

During the evening of Saturday and the early morning of Sunday, December 29–30, 1962, 41 elderly persons suffered severe gastroenteritis: 25 (60.9%) were patients in the hospital and 16 (39.1%) were inmates of the welfare home. There were three fatal cases among the hospital victims. No cases occurred among the staff.

The hospital victims were from three wards:

Ward	Persons Affected	Persons at Risk
Egerton	13 (2 fatal)	30
Toft	8 (1 ..)	30
Tatton	4	17
Total ..	25 (32.5%)	77

The welfare victims were also from three wards:

Ward	Persons Affected	Persons at Risk
C1	4	51
B1	11	47
B2	1	27
Total ..	16 (12.8%)	125

There were 28 female and 13 male patients affected; the average age for both groups was 79. The youngest was 48 and the eldest 95. Their clinical illness consisted of severe diarrhoea in all cases, abdominal pain in 13 cases, and vomiting in only 4. The onset started some 12–24 hours after the Saturday midday meal. The duration of illness averaged between 24 hours and 7 days, the average being 3 days.

Treatment

At the first symptom of enteritis on the Sunday all cases received $\frac{1}{2}$ fl. oz. (14 ml.) of kaolin and morphine mixture B.P.C. either two- or four-hourly for 24 hours: dosage depended on severity of symptoms. Next day the treatment was changed to oral guaninycin (Allen and Hanburys) in doses of 4 fl. oz. (114 ml.) (streptomycin sulphate 1 g., sulphaguanidine 8 g.) daily for five days. Exceptions to this therapy were the first two fatal cases, in which death occurred before the change to guaninycin.

Fatal Cases

Two of the hospital victims died on Sunday, December 30; the third died on Thursday, January 3. Post-mortem examinations were performed on the first two victims on January 1, 40 hours after death. Details are as follows.

Case 1.—Man aged 81. Gastroenteritis started at 4 a.m. on Sunday. Death occurred at 5.55 p.m. the same day. Necropsy showed distension of large bowel; congestion and injection of wall of small bowel; fluid faecal contents in small and large bowels, with some residual solid faecal material in lower part of large bowel; marked frothiness of contents of large bowel. *Cl. welchii* isolated from wall of large bowel, terminal ileum, small bowel, and contents of large bowel. No organisms of the staphylococcal, dysentery, or salmonella groups isolated. The immediate cause of death was certified as acute gastroenteritis. There was also evidence of cerebrovascular arteriosclerosis. His normal expectation of life would have been 12 months.

Case 2.—Woman aged 48. Gastroenteritis began at about 4 a.m. on Sunday. Death occurred at 8 p.m. the same day. Necropsy showed distension of large bowel; marked conges-

tion and injection of the wall of the jejunum and ileum; fluid faecal contents in small and large bowel. *Cl. welchii* isolated from small bowel, terminal ileum, and large bowel. A light growth of *Staph. aureus* isolated from large bowel. No organisms of the dysentery or salmonella groups isolated. Immediate cause of death was certified as acute gastroenteritis. There was also evidence of post-encephalitic parkinsonism. This condition was so advanced that her expectation of life was only a further 6 months.

Case 3.—The third fatality was a man aged 85 who suffered from severe gastroenteritis during the night of December 29. He recovered from the immediate effects of the gastroenteritis, but was so weakened that he died of heart failure at 8 a.m. on January 3. *Cl. welchii* together with a light growth of *Staph. aureus* were isolated from his faeces. No organisms of the dysentery or salmonella groups were found. His expectation of life was approximately the same as Case 1: 12 months.

Meal Consumed

Examination of the menu for the 48 hours preceding the onset of the outbreak showed that the Saturday midday meal was probably responsible for the food-poisoning. This meal consisted of cold sliced or minced ham, carrots, creamed potatoes, rice pudding, and jam. There was a choice of roast chicken as an alternative to the ham. Of the 202 patients at risk, 72 (35.6%) had eaten ham and 130 (64.3%) had eaten chicken. Those who ate chicken had no illness, while 41 (56.9%) of the 72 who ate ham developed gastroenteritis. Twelve of the more seriously ill hospital victims, including the 3 fatal cases, ate minced ham covered with hot onion sauce.

Preparation of Ham

The ham had been prepared as follows: Six sides of mild-cured Danish greenback bacon were delivered to the kitchen from a local grocer in Knutsford on December 22, 1962. It was refrigerated for six days. On Friday, December 28, the bacon was removed from the refrigerator. Three hams were cut off from three separate sides of bacon; boned, rolled, and boiled for three hours; then cooled off for one hour in the kitchen, skimmed, bread-crumbed, and returned to the refrigerator at 4 p.m. the same day. They were taken out of the refrigerator at 11.30 a.m. the following day, sliced or minced, and served for lunch from midday onwards until 1.30 p.m. Minced ham covered with hot onion sauce was served to those experiencing difficulty in masticating their food. This onion sauce had been prepared at 9 a.m. on Saturday from dehydrated onions, fresh milk, and cornflour. It was heated for 1½ to 2 hours, and allowed to come to the boil. The sauce was added to the minced ham before being placed in the food trolley.

No remnants of the suspected meal were available as at the end of each day it was the practice to destroy leftovers from meals, but slices from another ham in the kitchen were sent for examination.

Cooling Temperatures of the Ham

If ham was the vehicle of infection, then the cooling period immediately after cooking, and until refrigeration occurred, was probably the vital period during which *Cl. welchii* could have multiplied. An experiment was devised whereby the time taken for the temperature within the ham to cool to 50° F. (10° C.) could be ascertained.

A ham of similar size was cooked in the same way as those used for the suspected meal. A recording thermometer was inserted into the ham on its removal from the

boiler, so that the bulb was at the centre of the ham. The cooling temperatures over a four-hour period were recorded as follows.

Temperature at Centre of Ham			
When removed from boiler	..	..	162° F. (72.2° C.)
After ½ hour's cooling in kitchen	..	..	146° F. (63.3° C.)
" 1 " " " refrigerator	..	..	138° F. (58.8° C.)
" 2 " " " " "	..	..	128° F. (53.3° C.)
" 2 hours' " " " " "	..	..	64° F. (17.7° C.)
" 3 " " " " "	..	..	50° F. (10° C.)

The air temperatures recorded in the kitchen and the refrigerator during this experiment were 59° F. (15° C.) and 38° F. (3.3° C.) respectively.

Food Trolleys

There is one central kitchen for both hospital and welfare home. To take food from the kitchen to the various wards insulated food trolleys are used. These trolleys consist of a warm upper and cold lower section. It was the practice to connect them to the electricity mains about one to two hours before use. On the day in question the power was switched on at about 10.15 a.m.; so that the trolleys were heated for 1½ hours before the food was placed in them at approximately 11.30 a.m. The food was served from the trolleys from about 12 noon onwards until 1.30 p.m. The ham was placed in the cold lower section.

Examination of the trolleys after the food-poisoning incident showed that in three of them the lower sections were warmer than in the others. Two of these trolleys had been used in the hospital wards affected by the gastro-enteritis. They were tested. In both, the temperature in the lower cold section recorded at the start of the test was 40° F. (4.4° C.). After one hour the temperature in one trolley rose to 65° F. (18.3° C.), and in the other to 50° F. (10° C.). After two hours the temperature rose to 84° F. (28.8° C.) and 83° F. (28.3° C.) respectively.

Examination of the hospital records showed that both trolleys were 20 years old. They were dismantled, and it was found that the cork packing used for insulation had crumbled away and was in the bottom third of the compartments. This may account for the unusual temperatures recorded.

There seemed to be a relation between persons affected and the time of serving the meal in that 35 (85.3%) of the victims were served from 1 p.m. to 1.30 p.m.—that is, between 1½ and 2 hours after the food was placed in the trolleys.

Bacteriological Results

The slices of ham were negative for *Cl. welchii*, but, in any event, the actual ham suspected to be the vehicle of infection was not available for examination.

Samples of stools from the victims and eight kitchen staff were negative for organisms of the dysentery or salmonella group. Heat-resistant *Cl. welchii* were isolated from 26 of the victims (stools of 23 living victims and one fatal case, and large-bowel specimens at necropsy of two of the fatal cases). These cultures were sent to the Central Public Health Laboratory, Colindale, for typing. Examination showed that 24 were non-haemolytic heat-resistant strains of Hobbs's serological type 3/4 *Cl. welchii*. The other two strains differed: one was a non-haemolytic type 7, the other a β -haemolytic strain not in the serological group 1-13.

In addition *Staph. aureus* of phage types 3A/3B/3C/55+, 29+, 29/52/52A/80, and 29/52 were isolated from the bowels of the three fatal cases and one other victim.

Stool specimens and hand and nasal swabs from the kitchen staff were negative for *Cl. welchii*. *Staph. aureus* was isolated from nasal and hand swabs of three of the kitchen staff, the phage types being 29+, 81/6/7/47/53/54/75/42E+, and 29/52/80/77. Strain 81/6/7/47/53/54/75/42E+ is a group III strain and could be considered a potential food-poisoning strain. It was isolated also from the nasal and hand swabs taken from the assistant chef. As it was not isolated from the faeces of the victims it can surely be disregarded. None of the other *Staph. aureus* strains can be regarded as typical food-poisoning strains.

Discussion

Cl. welchii is widely distributed in the stools of human, animal, and fly origin, and the spores are likely to survive for indefinite periods in soil and dust, on clothes, and in the environment generally. Furthermore, the organism has often been isolated from samples of raw meat, so that its presence in the kitchen is almost inevitable (Hobbs, 1962).

Hobbs and Greenwood Wilson (1959), investigating the contamination of wholesale meat supplies at the Port of London and at Smithfield Market, reported that heat-resistant *Cl. welchii* were to be found in 76 of 722 (10.5%) samples of boneless meat and 3 of 195 (1.5%) samples of meat carcasses examined.

Silvester and Green (1961) assessed the presence of *Cl. welchii* in 713 samples of raw meat taken from 405 carcasses in Reading. They found that the average proportion from which *Cl. welchii* was isolated was 28%. English pork showed a higher percentage of positive results than other types of meat. They concluded that the contamination may have spread within the individual carcass itself by the increased degree of handling to which the boned meat carcass is subjected.

Hobbs (1955) has stated that, as with staphylococci and salmonellae, it seems that large numbers of *Cl. welchii* must accumulate in the food before symptoms are produced.

In this outbreak of *Cl. welchii* food-poisoning ham was the food common to all victims and was considered to be the vehicle of infection. If this hypothesis is a correct one then there are two factors which may have encouraged growth of *Cl. welchii* in the ham. (1) The four-hour period taken by the ham to cool to 50° F. (10° C.) after removal from the boiler (one hour in the warm kitchen and three hours in the refrigerator), as shown in the experiment. (2) The additional re-warming of some of the ham in the defective food trolleys pending and during transport to the wards.

The cooling experiment showed that during the first hour in the kitchen the temperature within the centre of the ham fell 24° F. (13.4° C.) from 162° F. (72.2° C.) to 138° F. (58.8° C.) before the ham was placed in the refrigerator. Growth of *Cl. welchii* would be encouraged during this period and probably continued in the refrigerator.

The following day ham was taken from the refrigerator and sliced or minced. Hot onion sauce was added to the minced ham in the kitchen. These foods were placed in the food trolleys at 11.30 a.m. in the kitchen, being served in the wards from 12 noon until 1.30 p.m. Examination of the food trolleys showed that those sent to two of the three hospital wards involved had defective lower cold sections when tested: temperatures from 50° to 65° F.

(10° to 18.3° C.) after one hour, and 83° to 84° F. (28.3° to 28.8° C.) after two hours were recorded. It seems likely that further multiplication of *Cl. welchii* present in the ham may have occurred during this period. It is interesting to note that 35 (85.3%) of the victims were served 1½ to 2 hours after the food was placed in the trolleys.

Jensen (1944) regarded food held for four to eight hours in the range 50–120° F. (10–48.9° C.) as potentially dangerous owing to the bacterial multiplication which may occur, depending upon the type of food and species of organism concerned.

Hobbs (1957) carried out experiments in which 200,000 *Cl. welchii* vegetative cells and spores were inoculated on to slices of freshly boiled lamb. When the meat was stored in gravy on top of a hot plate at 104–122° F. (45–50° C.) for three hours the numbers increased to 46 million per slice. Without gravy and stored at the same temperature the organisms increased to approximately 20 million in three hours.

The majority of cases (60.9%), including the three fatal cases, occurred in the hospital wards, and 32.4% of those at risk were affected. In comparison, only 39.1% of the victims were from welfare wards, and they comprised but 12.8% at risk. That the hospital victims were more seriously affected is probably due to two factors: (1) They were in a lower state of health, and as such would be a more susceptible group. (2) The defective hospital food trolleys may have been a factor in encouraging growth in the minced ham and onion sauce. (Twelve of the more seriously ill victims, including the three fatal cases, ate minced ham covered with onion sauce).

Even though the suspected food may not be available for examination, Hobbs *et al.* (1953) have stated that if a high percentage of stools examined showed a heat-resistant *Cl. welchii* of the same serological type throughout, then this would be a strong indication that the outbreak was in fact due to *Cl. welchii*.

Heat-resistant non-haemolytic *Cl. welchii* of serological type 3/4 were identified from 24 of the 26 positive *Cl. welchii* isolations from victims in this outbreak. These are regarded as food-poisoning strains (Hobbs, personal communication, 1963), and it would appear that they were the type that could cause an outbreak. Two different strains of *Cl. welchii* were also found in two of the victims. It seems probable that these were carriers and the finding was incidental to the main results.

Recently, Collee *et al.* (1961), in an investigation into the carriage of *Cl. welchii* with special reference to food-poisoning strains, demonstrated the carriage of heat-resistant *Cl. welchii* by 6–8% of a student population. Food-poisoning strains were non-haemolytic and resisted 212° F. (100° C.) for one hour.

Previous reports had given 2.2% as the average carrier rate of non-haemolytic heat-resistant strains in normal people (Hobbs *et al.*, 1953); hospital personnel and their families have shown 20% of persons to be symptomless excretors (Disch and Elek, 1957). Leeming *et al.* (1961) have indicated that carrier rates may now be above 30% in partly closed communities—for example, hospitals.

Although a fairly high carrier rate of *Cl. welchii* has been shown to exist in communities, it is far more likely that the ham concerned in the present outbreak arrived at the kitchen already contaminated. The original source

of the *Cl. welchii* may have been the slaughterhouse, curing-tank, or some other place *en route*.

Despite the observance of a high standard of personal hygiene, *Cl. welchii* can often enter the kitchen on raw meat contaminated from many sources. It has been shown that spores will survive boiling, stewing, and light roasting, particularly on the inner side of rolled joints, and germinate into actively growing bacilli (Norval and Collee, 1957; Collee *et al.*, 1961; Silvester and Green, 1961).

Hobbs (1962) has summarized preventive measures concerned with the cooking and storage of meat dishes. She states that in general they should be either cooked and immediately eaten hot or cooled rapidly and refrigerated within one and a half hours until required. When there must be unavoidable delay in the distribution of cooked sliced meat, the portion should be kept at a temperature either above 140° F. (60° C.) or below 59° F. (15° C.).

Summary

Clostridium welchii food-poisoning affected 41 elderly persons in a geriatric hospital welfare home in Knutsford, Cheshire. Twenty-five (60.9%) were hospital patients (three fatal cases) and 16 (39.1%) were inmates of the welfare home. Both sliced and minced ham were thought to be the vehicle of infection. Heat-resistant *Cl. welchii* was isolated from 26 victims. A non-haemolytic serological type 3/4 strain of *Cl. welchii* was isolated from 24 of these cultures (including those from three fatal cases). It is thought that *Cl. welchii* spores survived cooking and multiplied in the ham immediately after cooking, in the cooling period in the kitchen, and for a time in the refrigerator. The possible part played by food trolleys in the spread of infection is discussed.

I am grateful to Dr. Betty C. Hobbs for helpful criticism and advice, and to Dr. Anne White, of the Central Public Health Laboratory, Colindale, London, for the serological typing. My thanks are also due to Dr. J. D. Abbott, of Manchester Public Health Laboratory, for the bacteriological examination of stools and swabs; to Dr. R. C. Jennings, Department of Pathology, Altrincham General Hospital, for the post-mortem results; and to Dr. J. M. Greenwood for assistance in collating clinical details of the outbreak.

REFERENCES

- Cockburn, W. C. (1962). *Food-poisoning*, p. 7. Royal Society of Health, London.
- Collee, J. G., Knowlden, J. A., and Hobbs, B. C. (1961). *J. appl. Bact.*, **24**, 326.
- Dische, F. E., and Elek, S. D. (1957). *Lancet*, **2**, 71.
- Hobbs, B. C. (1955). *Ann. Inst. Pasteur Lille*, **7**, 190.
- (1957). *Bact. Proc.*, **14**.
- (1962). *Food-poisoning*, p. 52. Royal Society of Health, London.
- Smith, M. E., Oakley, C. L., Warrack, G. H., and Cruickshank, J. C. (1953). *J. Hyg. Camb.*, **51**, 75.
- and Wilson, G. J. (1959). *Mon. Bull. Minist. Hlth Lab. Serv.*, **18**, 198.
- Jensen, L. B. (1944). *J. Amer. vet. med. Ass.*, **104**, 63.
- Kemp, G. E., Proctor, R., and Browne, A. S. (1962). *Pub. Hlth Rep. (Wash.)*, **77**, 910.
- Leeming, R. L., Pryce, J. D., and Meynell, M. J. (1961). *Brit. med. J.*, **1**, 501.
- McClung, L. S. (1945). *J. Bact.*, **50**, 229.
- Norval, J., and Collee, J. G. (1957). *Scot. med. J.*, **2**, 427.
- Mon. Bull. Minist. Hlth Lab. Serv.*, 1954, **13**, 12.
- 1955, **14**, 34, 203.
- 1956, **15**, 263.
- 1957, **16**, 233.
- 1958, **17**, 252.
- 1959, **18**, 169.
- 1960, **19**, 224.
- 1961, **20**, 160.
- 1962, **21**, 180.
- Silvester, P. K., and Green, J. (1961). *Med. Offr*, **105**, 231, 289.