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A milk-borne outbreak of food poisoning due to *Salmonella heidelberg*

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INTRODUCTION

An explosive milk-borne outbreak of *Salmonella heidelberg* infection due to unpasteurized tuberculin-tested milk occurred in Cirencester in November 1961 and was traced to a cow with a symptomless salmonella mastitis. The origin of the infection was thought to be contaminated animal feeding stuffs.

Salm. heidelberg infection is rare in cattle and the organism has not been previously reported as a cause of milk-borne salmonellosis. For this reason and because animal feeding stuffs were implicated as the origin of the infection it is considered that the outbreak is of sufficient interest to be recorded.

The veterinary aspects of the outbreak have been discussed by Davies & Venn (1962).

THE OUTBREAK

Between 3 November and 3 December 1961 there were 56 reported incidents* of *Salm. heidelberg* infection, comprising 77 cases and 46 symptomless excreters, in Cirencester and the surrounding rural district. The dates of onset of symptoms of the first or only case in 51 of these are shown in Fig. 1. In three incidents the dates of onset were not recorded and two incidents comprised only symptomless excreters.

During the period of the outbreak 53 other incidents of 'food poisoning' were reported in the area, but salmonellae were not isolated from 131 patients examined. These incidents were unrelated to the *Salm. heidelberg* outbreak and the disease was probably 'epidemic' or non-specific diarrhoea and vomiting which had been prevalent in the district during the late summer.

* Incident means either a single case or a group of 2 or more cases in a household.

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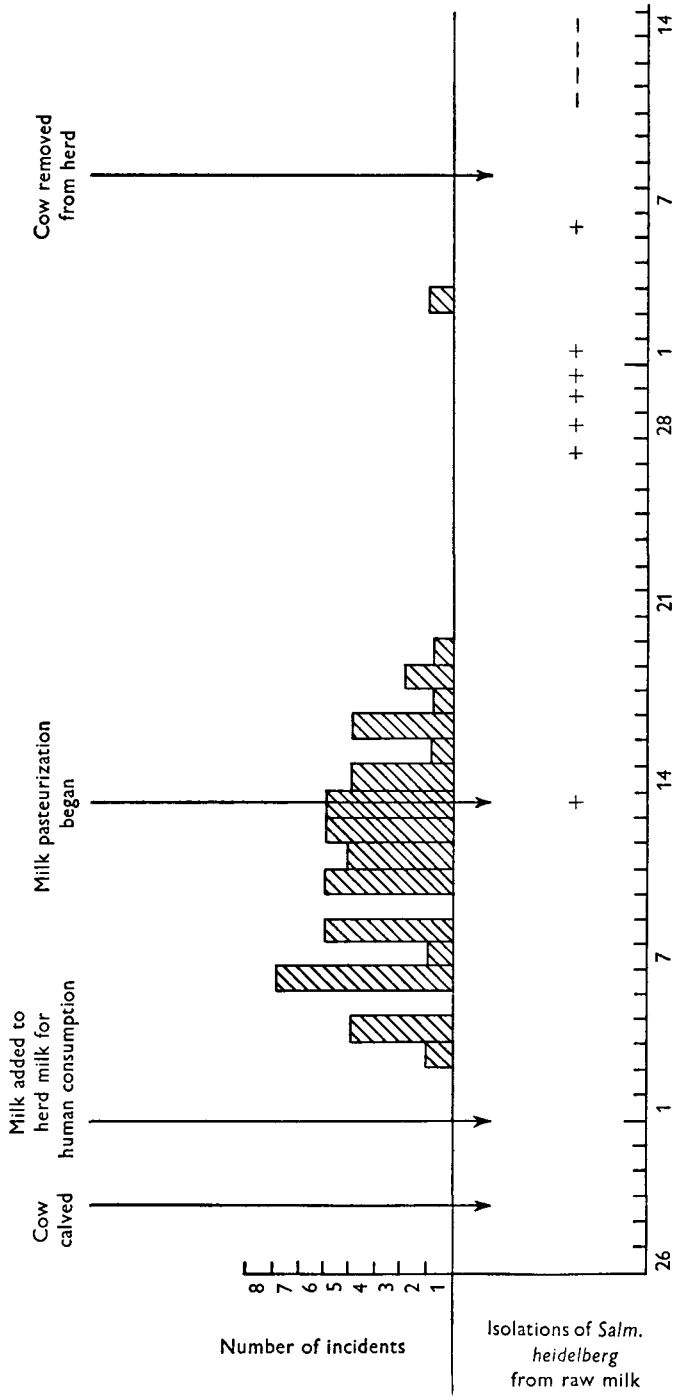

Fig. 1. Dates of onset of symptoms of first or only case in each incident. History of infected cow and isolations of *Salmonella heidelberg* from raw milk. +, *Salm. heidelberg* isolated; —, salmonellae not isolated.

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Clinical features

Seventy-two of the 77 patients from whom *Salm. heidelberg* was isolated had diarrhoea and in 36 vomiting was recorded. Abdominal pain was prominent in 16 patients, one of whom, a girl aged 8 years, was admitted to hospital because of suspected appendicitis. A girl aged 7 months had convulsions at the onset of the illness. There were no deaths.

The ages of the 77 cases and 46 symptomless excreters are given in Table 1. Fifty-nine of the 123 cases and excreters were males and 64 females. Thirty-eight of the 77 cases were males and 39 females.

Table 1. *Ages of cases and symptomless excreters*

Age (years)	Numbers of cases and symptomless excreters: Total	Cases	Symptomless excreters
0– 4	41 (33)	36 (47)	5 (11)
5– 9	27 (22)	18 (23)	9 (19)
10–14	19 (16)	8(10)	11 (24)
15–19	3	2	1
20 and over	32 } 36 (29)	12 } 15 (20)	20 } 21 (46)
Not known	1	1	0
Total all ages	123 (100)	77 (100)	46 (100)

Percentages are given in parentheses.

Milk—the vehicle of infection

The first two notifications of cases in the outbreak were on 10 November; further cases were reported 3 days later. Raw tuberculin-tested milk from a local dairy A had been supplied to all the households concerned. Commencing on the evening of 13 November all milk sold by the dairy was pasteurized and after 19 November only one further incident occurred (see Fig. 1).

Subsequent investigations revealed that in 53 of the 56 incidents milk had been obtained from dairy A. In one incident, a boy aged 2 years whose family obtained milk from a different dairy had consumed milk from dairy A on several occasions when he had visited his grandmother. In the remaining two incidents there was no apparent association with milk from dairy A.

The dairy

Dairy A was supplied daily from four farms with about 300 gal. of milk, which was bottled and distributed to 1260 families in and around Cirencester. *Salm. heidelberg* was isolated from milk sampled on 13 November from three churns from farm B but not from samples from 17 churns from the three other farms.

Farm B supplied daily about 80 gal. of Guernsey milk, which on any one day was distributed to about 350 families. The milk was probably contaminated on several occasions between 1 and 13 November and in this period it was sold to about

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600 families. The number of families at risk may have been greater than 600 because occasionally there was more Guernsey milk than was required and the excess milk from farm B was added to the milk from the other farms. In addition cross-contamination of the milk from the other farms might have occurred in the bottling machine. Fifty-eight families were known to have been affected in the outbreak but others may not have been reported. Taking into account these factors it is estimated that about 10 % of the families at risk were affected.

There were six people employed at dairy A, one of whom developed diarrhoea on 6 November and two of whom were found to be symptomless excreters of *Salm. heidelberg*. There were three other cases and seven symptomless excreters in their families. These cases occurred in the outbreak and the excreters were probably infected at the same time.

The farm

The source of infection on farm B was found to be a cow with mastitis. *Salm. heidelberg* was isolated from the udder post mortem (Davies & Venn, 1962). This cow was born on the farm in February 1956 and had never been ill.

The cows were milked by machine and the milk was poured into churns through a strainer containing a cellulose filter pad. *Salm. heidelberg* was isolated from milk from 9 of 20 churns sampled at the farm and from two cellulose filter pads after they had been used. The churn containing milk from the infected cow and the churn next filled were found to be contaminated on four occasions, but when a new filter pad was used for each churn only the first churn was positive. It seems therefore that spread of the organism to the second and possibly to subsequent churns was due to contamination of the strainer and filter pad. The infected cow was slaughtered on 8 December and *Salm. heidelberg* was not subsequently isolated from 64 milk samples collected over a period of three months.

Davies & Venn (1962) have discussed how *Salm. heidelberg* may have reached the udder of the cow, either by infection through the teat canal or by ingestion and septicaemic spread from the intestine. How the organism reached the cow remains to be considered.

It seems unlikely that the infection was brought into the farm by a carrier animal because the only recent addition to the cattle herd was a bull purchased in 1959, and the herd had had no contact with other animals. Salmonellae were not isolated from faecal specimens of cows, sheep and chickens on the farm. Pigs were not kept and there was no possibility of contact with pigs on neighbouring farms. The only other animal infected was a calf, born after the outbreak, which was fed on the contaminated milk (Davies & Venn, 1962).

There was no evidence that any of the four farm workers or their families were excreting the organism before the outbreak. One child in each family developed symptoms during the outbreak and subsequently three of the workers and three other family members were found to be infected. These ten persons had all consumed the contaminated milk, whereas two of four family members not infected did not consume the milk. The cases and symptomless excreters all ceased to excrete the organism within 3 months of the outbreak and it seems

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reasonable to conclude that none of them was a chronic carrier and that they were all infected in the outbreak at the same time.

Before August 1961, the cattle were daily driven past a cesspit which drained the farm workers' cottages, and as this occasionally overflowed the cows may have had access to the sewage. It is unlikely that the sewage was contaminated with *Salm. heidelberg* before the outbreak, because none of the occupants of the cottages could be shown to be a chronic carrier; moreover, it is improbable that anyone in the cottages was infected twice, once before the outbreak and again in the outbreak.

In 1961 there were 289 reported incidents of *Salm. heidelberg* infection in England and Wales (Report, 1962), the nearest case being in Bristol about 30 miles from Cirencester. None of the farm workers or their families could recall having had symptoms before the outbreak which could be attributed to infection with *Salm. heidelberg* nor had they visited Bristol or other districts at the time the reported cases occurred. Pork and pork products have been suspected as the source of several outbreaks in the past but the farm workers and their families were not in the habit of eating such meat nor had they eaten foods processed by producers previously implicated in any outbreaks.

A sewer from a neighbouring hutted camp for Polish refugees traversed the farm but it had not been blocked nor was there evidence of leakage of sewage and it could not therefore have been the origin of the infection.

The cattle herd was supplied with chlorinated water direct from the mains and had no access to ponds or streams. A sample of mains water from the cowshed on 12 December contained no salmonellae and 12 routine samples collected during the year from the source of supply to the area had presumptive coliform counts of nil per 100 ml.

Animal feeding stuffs

Animal feeding stuffs manufactured by the firm which supplied cattle cake to farm B were found to be contaminated with several salmonella serotypes; *Salm. heidelberg* was isolated from English meat and bone meal at the supplier's factory. This serotype is uncommon in animal feeding stuffs (Walker, 1957; Reports, 1959*a*, 1961) and the isolation of the organism from the cow and from material at the factory suggested that the two might be associated.

The dairy cows were fed on cattle cake which contained vegetable and inorganic mineral ingredients—it did not contain animal protein. Two of 23 samples of cattle cake and one of 62 samples of various vegetable ingredients, sampled at the factory, were contaminated with salmonellae (Table 2). These samples were obtained from batches made after the outbreak. Twenty-one samples of inorganic minerals and 26 samples of calf and poultry foods were negative. Salmonellae were isolated also from sacking and sack dust from bags which had contained cattle cake.

At the factory where the cattle cake was made, English meat and bone meal and a protein concentrate were used as ingredients of other feeding stuffs. Salmonellae were isolated from both these materials—*Salm. heidelberg* from

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English meat and bone meal. The same machine was sometimes used to mix these materials as was used to mix the ingredients of the cattle cake, so that cross-contamination could easily have occurred in the machine.

Table 2. *Isolations of salmonellae from animal feeding stuffs, sacking and sack dust November* 1961–*May* 1962

Type of material	Number of samples examined	Number of samples positive	Salmonella serotypes
Cattle cake	23	2	cubana, kiambu
Calf and poultry foods	26	0	—
Cattle cake ingredients			
(1) Vegetable materials	62	1	taksony
(2) Inorganic minerals	21	0	—
Sacking and sack dust	24	2	cubana, taksony
Protein concentrate	18	2	oranienburg, seftenberg
English meat and bone meal	15	1	heidelberg
Total all materials	189	8	

The materials were sampled at an animal feeding-stuffs mill and a factory where used sacks were cleaned.

The English meat and bone meal was made in Bristol from butchers' scraps, offal, and meat and bone from abbattoirs and knackers' yards in the west of England.

A sample of knacker meat obtained from a pet food shop in Bristol in August 1961 was found to be contaminated with *Salm. heidelberg*. This suggests that animal infection was present in the area and, although it was not possible to determine the farm of origin of the infected meat, the raw materials of the meat and bone meal might well have been contaminated with *Salm. heidelberg* before the Cirencester outbreak.

It is not clear how the animals from which the meat and bone meal was made came to be infected in the first place, but the organism may have been introduced in some material such as an imported animal feeding stuff. The cycle of: infected animals—contaminated meat and animal products—contaminated animal feeding stuff—infected animals, would then have led to the perpetuation of the infection in the area.

LABORATORY METHODS

Faeces specimens from patients were cultured on deoxycholate-citrate-agar (D.C.) plates (Hynes, 1942) and in selenite F broth (Hobbs & Allison, 1945), and non-lactose-fermenting organisms obtained from these media were tested biochemically. Organisms giving the reactions of the salmonella group were then typed by serological methods.

Samples of milk were examined by an enrichment method. Fifty ml. of milk was added to an equal volume of double-strength Leifson's selenite F medium and incubated for 18 hr. The culture was then plated on deoxycholate-citrate-agar and non-lactose-fermenting colonies were picked off for biochemical and serological

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tests. Water samples were examined by the same enrichment method and presumptive coliform counts were also performed.

Animal feeding stuffs were collected from previously unopened containers using sterile spoons and the specimens forwarded to the laboratory in polythene bags. Twenty g. samples—four from each bag—were added to 100 ml. amounts of Ringer's solution and incubated at 37° C. for 2 hr. To each of these 100 ml. of double-strength selenite F broth was then added and incubation continued overnight. Subcultures were made on to D.C. plates and incubated for 24 hr., after which non-lactose-fermenting colonies were picked off and examined serologically and biochemically.

DISCUSSION

Milk-borne outbreaks of salmonellosis are not common in this country (Taylor, 1960), but nevertheless over the past 20 years the number of known cases in the recorded outbreaks was nearly 3000 (Table 3) and the actual number of cases was certainly much greater. Most of the outbreaks were due to contaminated raw milk from sick or carrier cows and could have been prevented by pasteurization. The outbreak described in this paper emphasizes again the need for the pasteurization of all milk.

It is considered that the origin of this outbreak was contaminated animal feeding stuffs. Wright, Norval & Orr (1957) suggested also this source of infection in a milk-borne outbreak of *Salm. thompson* in Edinburgh in 1956. In an outbreak of *Salm. dublin* in Somerset in 1952, surface water contaminated by cattle on a neighbouring farm was a possible source (McCall, 1953). In no other reported outbreak of salmonellosis was there evidence to suggest how the cattle became infected.

It is of interest to consider the salmonella serotypes isolated in milk-borne outbreaks in the United Kingdom in the past 20 years (Table 3). *Salm. dublin* was the most common organism until 1950, after which it was replaced by *Salm. typhimurium*. More recently other less common serotypes have appeared.

The most likely origin of *Salm. dublin* is the cattle themselves because the organism is primarily a bovine pathogen and is uncommon in other species and in animal feeding stuffs (Taylor, 1960). However, *Salm. typhimurium* and the other serotypes are much less host specific and are common in other species and in animal feeding stuffs (Walker, 1957; Reports 1959*a*, 1961); cattle are therefore less likely to be the origin of the infection. It is possible that the change in the salmonella serotypes causing milk-borne outbreaks may be related to the increased use of imported animal feeding stuffs in this country in recent years.

There has also been a change in the seasonal incidence of the reported milk-borne outbreaks (Table 3). Most have been between April and September but, since 1953, six outbreaks have occurred in the colder weather between October and February—three of them in January and February. The reason for this is not apparent but it might be related to seasonal variations in the use of animal feeding stuffs.

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ıble 3. *Reported milk-borne outbreaks of salmonellosis in the United Kingdom*, 1942–61

Date	S	nella ype	Number of persons infected	*Type of milk	Probable source of infection	Authors
Aug. 1943	du		162	Raw	Symptomless cow excreting organism in faeces	Sutherland & Berger (1944)
Sept. 1947	du		97	Raw	Sick cow which died	Henderson *et al.* (1948)
Sept. 1947	du		2 large outbreaks	—	—	Report (1948)
May 1949	du		50	Raw	Sick cow which died	Cromb & Murdock (1949)
May 1949	ne	;	165	Raw T.T.	Sick cow with enteritis	Unpublished P.H.L.S. records
July 1949	ty	ırium	337	Raw T.T.	Possibly sick farm worker	Report (1949)
July 1950	du		—	Raw	—	Report (1951)
July 1950	ty	ırium	—	Raw	—	
July 1951	du		2	Fresh	—	Report (1954)
July 1951	ty	ırium	2	Fresh	Evidence that milk was responsible not complete	
July 1951	or	burg	2	Fresh		
Sept. 1952	du		610	Raw T.T.	Sick cow with mastitis, possibly infected by contaminated water	McCall (1953)
Feb. 1953	ty	ırium	121	Raw T.T.	Symptomless farm worker or cattle	Lennox *et al.* (1954)
June 1953	ty	ırium	252	Raw T.T.	Symptomless cow excreting organism in faeces and probably also in milk	Norton & Armstrong (1954)
June 1953	ty	ırium	211	Raw T.T. and pasteurized	Sick cattle or farm worker. The pasteurized milk was contaminated by raw milk in the bottling machine	Riddell (1954)
Aug. 1953	ty	ırium	13	Raw	Cow	Report (1955)
Aug. 1955	ty	ırium	17	Pasteurized	Contaminated after pasteurization by bottle tops soiled with mouse faeces	Report (1956)

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Table 3 (*cont.*)

Date	Salmonella serotype	Number of persons infected	*Type of milk	Probable source of infection	Authors
Jan. 1956	typhimurium	6	Raw T.T.	Cow excreting organism in faeces	Report (1957)
Aug. 1956	thompson	11	Raw	Sick cow excreting organism in milk and faeces	Wright *et al.* (1957)
1950–1957	typhimurium	3 outbreaks	—	—	Chalmers & Sampson (1958)
1950–1957	dublin	—	Raw T.T.	—	
1950–1957	enteritidis	320	Pasteurized	Milk contaminated at centre where it was consumed	
June 1957	newport	21	Raw	Sick cow excreting organism in milk	Boyd (1958)
Jan 1958	typhimurium	125	Raw	Sick cattle	Riddell *et al.* (1959)
Aug. 1958	typhimurium	44	Raw T.T.	Symptomless dairy worker	Report (1959*b*)
Sept. 1958	typhimurium	20	Raw T.T.	Sick cow with enteritis	
Sept. 1959	typhimurium	26	Raw	—	Report (1960)
Oct. 1959	typhimurium	3	Raw	Sick calves. Milk probably contaminated by dairy workers who handled the calves	
Oct. 1960	typhimurium	58	Raw T.T.	Sick cow	Whitehead (1961)
May 1961	typhimurium	60	Raw	Cow excreting organism in milk	Report (1962)
July 1961	typhimurium	21	Raw T.T.	Cow with enteritis	Parry (1962)
Sept. 1961	enteritidis var. jena	Several families	Raw	Sick cow	Report (1962)
Nov. 1961	heidelberg	123	Raw T.T.	Symptomless cow excreting organism in milk	This paper

* Outbreaks due to canned or dried milk are not included.

SUMMARY

A milk-borne outbreak of *Salm. heidelberg* infection due to unpasteurized milk from tuberculin-tested cows is described. There were 77 cases and 46 symptomless excreters of the organism. There were no deaths. The infection was traced to a cow with a symptomless salmonella mastitis and the origin of the organism was thought to be contaminated animal feeding stuffs.

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