J. Hyg., Camb. (1974), **72**, 185
Printed in Great Britain

Recent trends in human salmonellosis in England and Wales: the epidemiology of prevalent serotypes other than *Salmonella typhimurium*

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(*Received* 2 *July* 1973)

SUMMARY

In the period 1960–70 meat and poultry products caused over 70% of successfully investigated outbreaks of human salmonellosis. The number of human incidents of salmonellosis declined from 1960 to 1966, but then more than doubled between 1966 and 1971. This increase was mainly due to a threefold increase of incidents of salmonella serotypes other than *Salmonella typhimurium*. The serotypes which increased most and contributed significantly to this trend were *S. enteritidis*, *S. panama*, *S. stanley*, *S. virchow*, *S. agona*, *S.* 4,12:d:- and *S. indiana*.

Strong evidence exists that these serotypes have a path of infection from animal feedingstuffs to the pig and poultry animal reservoirs to pork and poultry foods to man. Cattle appear to be a less important source than pigs and poultry and this may be because the nature and content of their feed is different.

The importance of the control of the pig and poultry reservoirs of salmonella infections is stressed and a significant role of animal feedingstuffs in the maintenance of these reservoirs strongly suggested.

INTRODUCTION

Since 1960 two major trends in the epidemiology of human salmonellosis have become evident. These are an increase in the proportion of outbreaks caused by meat and meat products, and an increase in the number of human incidents caused by salmonella serotypes other than *S. typhimurium*.

From 1949 to 1959 meat and meat products were the commonest vehicle of infection, accounting for 47% of the outbreaks; 27% were attributed to eggs and egg products, 15% to sweetmeats and 11% to other foodstuffs (Galbraith, 1961). In the period 1960–70, however, the proportion of outbreaks associated with meat products increased to 71%, while eggs and egg products were responsible for only 4% (Reports, 1961–4; Vernon, 1965–7; Vernon, 1969–70; Vernon, unpublished). The decline in the importance of egg products was due to the introduction of legislation in January 1964 requiring the compulsory pasteurization of domestic and imported liquid egg used in the manufacture of egg products (Statutory Instrument, 1963). Some producers carried out voluntary pasteurization for a few years before legislation was introduced.

Table 1. *Number of human incidents of* Salmonella *infection reported to the Epidemiological Research Laboratory*, 1960–71

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971
S. typhimurium	2907	2503	1864	1820	1725	1721	1407	1810	1654	1512	1865	2124
Other salmonellas	1047	1268	982	1149	1368	1224	1089	1449	2142	3305	3360	3540
Total	3954	3771	2846	2969	3093	2945	2496	3259	3796	4817	5225	5664

Table 2. *Number of human incidents of certain prevalent serotypes reported to the Epidemiological Research Laboratory*, 1960–71

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971
S. panama	9	7	8	12	39	86	95	209	379	307	517	247
S. stanley	68	42	50	65	19	50	85	262	169	195	144	28
S. virchow	7	5	2	25	25	10	2	31	253	326	123	93
S. enteritidis	145	90	93	64	122	180	114	143	271	649	650	511
S. 4,12:d:-	—	—	—	—	—	—	—	—	38	156	50	30
S. agona	—	—	—	—	—	—	—	—	—	2	232	620
S. indiana	—	—	—	1	2	10	11	90	87	164	143	180

Table 1 shows the number of human incidents of salmonella infection reported to the Epidemiological Research Laboratory for the years 1960–71. The number of incidents due to *S. typhimurium* fell from 2907 in 1960 to 1407 in 1966 and fluctuated between that figure and 2124 in 1971. In contrast incidents due to other salmonellas remained fairly constant until 1966 and then increased each year from 1089 in 1966 to 3540 in 1971 – more than a triple increase. Total salmonella infections, therefore, declined from 3954 in 1960 to 2496 in 1966 and then more than doubled to 5664 in 1971.

The second change was thus a very large increase in the number of incidents of salmonella serotypes other than *S. typhimurium* after 1966. The serotypes which increased most in incidence and contributed significantly to this trend were – *S. enteritidis*, *S. panama*, *S. stanley*, *S. virchow*, *S. agona*, *S.* 4,12:d:- and *S. indiana*. This report examines the epidemiological features associated with the prevalence of these serotypes.

SOURCES OF INFORMATION

Reports of isolations have been taken from the annual reports of food poisoning of the Public Health Laboratory Service (Reports, 1950–64; Vernon, 1965–7; Vernon, 1969–70) from Taylor *et al.* (1965) and from information abstracted from the Communicable Disease Reports of the Public Health Laboratory Service for the years 1964–71.

THE SEROTYPES

S. panama

S. panama was not isolated from humans in the United Kingdom before the war. In 1940 Scott examined the mesenteric lymph glands of pigs, but *S. panama* was not one of the serotypes found. During the war dried egg contaminated with salmonellas was imported into the United Kingdom from the U.S.A., Canada and the Argentine and any found unfit for human consumption was distributed to animal food manufacturers. Some of it was fed to pigs, without sterilization, and in 1944 pig mesenteric glands were examined to see whether salmonellas had been introduced into the pig population. *S. panama* was isolated from four such samples in 1944 (Medical Research Council, 1947) and in the same year it was isolated from a human case. From 1944 to 1963, however, *S. panama* caused only 68 human incidents, but after 1963 the number increased considerably (Table 2) reaching a peak in 1970.

From the mid-1960's *S. panama* was isolated on several occasions from animal, including pig, feed ingredients, from pigs, abattoirs and bacon factories; and from pork, sausages and made-up meat.

Investigation of human incidents

In August and September 1967 there were 154 human isolations of *S. panama*, a notable seasonal increase over the three previous years. Cases were widely distributed throughout the country. People of all age groups were affected, several elderly people severely enough to require hospital admission, and two patients

died. Most cases were sporadic in occurrence, but a few family and hospital outbreaks were reported.

In an investigation of sporadic cases in London (unpublished) it was not possible to find a common vehicle and source of infection. In one case, however, cold roast pork appeared to be the vehicle of infection and it was possible to trace its source via a butcher and wholesaler to an abattoir where *S. panama* was isolated from three of several swabs taken from pigs.

In a family outbreak in 1969 firm bacteriological evidence was secured that cooked ham was the vehicle of infection and in 1970 that roast pork was the vehicle in a large general outbreak affecting over 300 persons.

Firm epidemiological evidence was thus obtained that pork products were responsible for transmitting *S. panama* to man. More recently *S. panama* has been found to have contaminated processed poultry. Between 1969 and 1971 a total of 29 isolations from poultry were reported. The chief source of human infection remains, however, pigs.

S. stanley

S. stanley was isolated from humans in the United Kingdom before the last war. It caused very few human cases until 1955 when there were 101 human incidents, most of these being attributable to pork (Report, 1956). There were 56 incidents in 1956, but few in the following years until 1960 when there were 68 incidents. The number of incidents remained fairly steady until 1965. There was an increase in 1966 and a peak was reached in 1967 when *S. stanley* was the commonest human serotype other than *S. typhimurium* (Table 2). After 1967 the number of incidents declined, but *S. stanley* remained among the commonest human serotypes until 1970.

S. stanley was isolated a number of times from coconut in the early 1960's. Thereafter it was isolated most commonly from animal, including pig, feed ingredients, pigs, abattoirs, bacon factories and sausages and made-up meat. There were very few isolations from poultry.

Investigation of outbreaks

During the period 1964 to 1969 there were five outbreaks due to *S. stanley* in which pig products were implicated bacteriologically or epidemiologically. The foods responsible were boiled ham (twice), pork products, pork sausage and pork pies. In a sixth outbreak sausage (unspecified) was implicated.

In June and July 1968 an outbreak affecting 13 persons, with one fatal case, occurred in the West Riding of Yorkshire. The cases were associated with pork products and isolations of *S. stanley* were made from an abattoir supplying the suspected pork.

At the end of August 1968, an outbreak affecting 46 persons occurred in a West Midlands Hospital and a further 49 cases were reported from West Midlands counties. Investigation of 15 cases in two districts strongly suggested that infection was distributed in cooked meat, pork pies and sausages prepared by one local manufacturer. This manufacturer and six of the retail outlets were investigated.

One excreter of *S. stanley* was found at the factory and nine excreters at four of the six retail outlets.

As with *S. panama*, there was strong evidence that *S. stanley* infection originated from pigs and was transmitted via abattoirs and pork products to man.

S. virchow

S. virchow was very rarely isolated in the United Kingdom until 1968. The first single human case occurred in this country in 1942. From 1941 to 1953 there were only four human incidents. Twenty-one incidents occurred in 1955 and then only a few until 1963 when there were 25, the same number being recorded in 1964 (Table 2). From 1965 to 1967 there were 43 incidents. In 1968, 253 incidents occurred and a further increase occurred in 1969, when there were 326 incidents.

Since 1967 chickens have been the main non-human source of *S. virchow*. It has been isolated on numerous occasions from poultry for human consumption and also from poultry processing plants, chickens on farms and feed ingredients, particularly products of poultry origin such as feather meal and poultry offal meal. It has been found on only a few occasions in pigs and other animal sources.

Investigation of outbreaks

Most cases of *S. virchow* in 1968 and 1969 occurred in North West England. Cases first started to occur in this region towards the end of 1967 when there was an outbreak in a county borough and the surrounding districts. The vehicle of infection was chicken and *S. virchow* was isolated from faecal specimens of staff and chicken samples at three chicken retailers, and from staff members and a sewer swab at two processing plants supplying them. Cloacal swabs of birds arriving at one of the plants yielded *S. virchow* on culture.

In the summer of 1968 a second outbreak due to chickens occurred in the North West (Semple, Turner & Lowry, 1968). The source was traced to a packing station where *S. virchow* was isolated from chicken carcasses. The organism was also found in chickens on farms supplying the packing station (Pennington, Brooksbank, Poole & Seymour, 1968).

Undoubtedly, *S. virchow* was introduced into chicken flocks in the North West and gave rise to contaminated processed chicken which resulted in outbreaks and sporadic cases of human infection.

S. enteritidis

S. enteritidis was prevalent in England and Wales when records started in 1923. In the 1940's it caused between 10 and 50 human incidents annually. Between 1950 and 1967 the number of incidents varied between 70 and 200. Table 2 shows that the number of incidents almost doubled in 1968 when 271 were recorded and more than doubled again in 1969 when over 600 incidents occurred, the same number being reported in 1970. The number of incidents declined slightly in 1971.

In the early 1960's *S. enteritidis* was isolated on a few occasions from egg products but since 1967 poultry, particularly chickens, has been the only human food in which it has been found in substantial numbers. The organism has been isolated

from abattoirs and poultry factories, from the main food animal sources, particularly poultry, and from feed ingredients.

Investigation of outbreaks

Thirty-five outbreaks due to *S. enteritidis* were reported from 1966 to 1969. One outbreak occurred amongst persons who attended two wedding receptions in September 1969; of 250 guests, 55 were taken ill. *S. enteritidis* was cultured from the faeces of 45 patients. The incriminated food appeared to be cooked chicken. At the first reception the chicken was cooked, allowed to cool and served cold. At the second reception the chicken had been cooked, cooled and then re-heated. One firm of caterers cooked the chickens for both wedding receptions. *S. enteritidis* was cultured from one of two unconsumed whole cooked chickens from the caterers.

This outbreak was the only one in which bacteriological proof of a food vehicle was obtained. In the other outbreaks no food was available for examination, or no food samples had been taken. But in nine of the outbreaks a food was implicated on epidemiological grounds. Chicken was the vehicle in six; turkey, scotch egg and liver pâté in one each.

It is clear that the cause of the increase in *S. enteritidis* infections was contaminated poultry from infected flocks.

S. 4,12:d:-

S. 4,12:d:-, an un-named monophasic serotype, was isolated for the first time in England and Wales in 1968.

There is circumstantial evidence that this serotype may have been introduced into England and Wales by an imported feed ingredient. *S.* 4,12:d:- was isolated from three sewer swabs at a poultry processing plant in September 1968. Subsequently it was isolated at the plant from chicken carcasses and giblets, factory water and sewer swabs on a number of occasions until the end of January 1969. It was isolated from cloacal swabs taken from chickens which arrived at the processing plant from three chicken broiler farms and subsequently from the faecal droppings on one of these farms, but not from the feed. Cultures made from infertile eggs at the hatchery where the chickens were hatched were negative. Samples of feed and droppings from two breeding farms supplying the eggs to the hatchery were also negative. However, feed supplied to one of the breeding farms contained South African fish pellets from a consignment that arrived in England and Wales in August 1968. *S.* 4,12:d:- was isolated from a sample taken at the manufacturer in January 1969. It was also found in a sample of meat and bone meal from the same feed compounder in December 1969. Thus, a contaminated imported consignment of a feed ingredient appears to have introduced the serotype into a chicken breeding flock, whence it was transmitted via eggs to the chicken broilers and so to processed chickens and equipment in the processing plant.

Table 2 shows the number of human incidents of *S.* 4,12:d:- from 1968 to 1971. In 1969, the year after its introduction, it was amongst the ten commonest serotypes isolated from humans. The number of incidents fell in 1970 and 1971.

Chicken has been the commonest food vehicle of *S.* 4,12:d:- but there have also

been several isolations from pork, sausages and made-up meat. The organism has often been found in chicken factories and abattoirs, in chickens and pigs and in feed ingredients.

S. 4,12:d:- was probably introduced into England and Wales in 1968 by an imported South African feedstuff. It rapidly established itself in the domestic pig and poultry population, contaminated pork and poultry foods and caused human outbreaks and sporadic cases throughout the country.

S. agona

S. agona had only rarely been isolated in England and Wales before 1970. In 1969 two cases were reported in patients who had returned from Spain (Public Health Laboratory Service Report, 1972). In May 1970 there were a number of human incidents in the North of England and Wales (Public Health Laboratory Service Report, 1970). Poultry was implicated in three incidents and pork in one. *S. agona* was isolated from a mincing machine and chopping block in a food shop which supplied one affected family with chickens. It was also isolated from a wooden stacking tray on a farm where the chickens had been killed. In June 1970 *S. agona* was isolated from chicken carcasses from a farm in the West Midlands. Both the farmer and his wife had had gastro-enteritis in May and *S. agona* was isolated from the farmer and his two children, who were symptomless. *S. agona* was also isolated from 12 of 19 sets of broilers' giblets from a batch killed in June 1970 in the North East and from imported fish meal used as raw material for animal food in the same area.

In 1970 and 1971 *S. agona* was frequently isolated from processed poultry, pork, sausages and made-up meat; and from poultry processing plants, abattoirs, chickens, pigs and feed ingredients.

Table 2 shows that in 1970 there were 232 human incidents of *S. agona* whereas in 1971 there were 620 and it was the second commonest serotype isolated from man.

How did *S. agona* first get into England and Wales? The most likely explanation for the appearance of a new serotype seems to be importation in an animal feed ingredient. *S. agona* was isolated from imported fish meal in May 1970 and it is likely that this introduced the organism into domestic livestock in England and Wales. Since then it has been isolated from feed ingredients including meat and bone meal, feather meal and poultry offal meal. All these are treated animal by-products. It is likely that it is now being maintained through re-cycling of these treated animal wastes which are fed back to the animals. It is clear that pork and poultry have become contaminated causing numerous cases of human infection.

S. indiana

The first isolation of *S. indiana* in England and Wales was from a sample of fish meal in 1958. In 1959 and 1960 isolations were made from American meat meal. The first recorded human incident of *S. indiana* occurred in 1963, but there were only a few in the following years until 1967 when there were 90 incidents (Table 2).

The number of incidents increased from 1969 to 1971 during which time *S. indiana* has been among the 10 commonest salmonellas causing human infection.

The increase in human incidents was accompanied by numerous isolations from food sources, particularly chicken, from pigs and poultry and from feed ingredients.

Investigation of outbreaks

In 1969, *S. indiana* was isolated from eight of ten patients with food poisoning in the North of England (Public Health Laboratory Service Report, 1969). The patients had all eaten spit-roasted poultry from a local shop. The shop received eviscerated unfrozen chickens from the company's own processing plant. They were kept in a refrigerator until required and then cooked on a rotary spit for 70 min. at a temperature of 350° F. After cooking, the chickens were removed from the spits and placed whole or quartered in the shop window where, even with a fan operating, the temperature was found to be as high as 84° F. A number of samples of chickens and swabs from various articles of equipment used in the preparation and handling of the chickens were examined for salmonellas with negative results, though subsequently *S. senftenberg* was isolated from two uncooked carcasses. However, at the processing plant, *S. indiana* and other serotypes were isolated from carcasses and rinse water.

Pork has also been shown to be the vehicle of infection in outbreaks due to *S. indiana*.

It is clear that *S. indiana* has gained access to the domestic poultry and pig population, contaminated poultry and pork after slaughtering and processing, and caused cases of human infection.

DISCUSSION

The salmonella serotypes which have been prevalent during recent years are closely associated mainly with pigs and poultry, but cattle do not appear to be an important source. There are probably two main reasons for this. First, there has been a greater increase in the amount of pork and poultry produced and consumed than in that of beef and veal. Between 1964 and 1971 the annual production of beef and veal increased from 862 to 938 thousand tons, an increase of 9%, while consumption did not increase, but remained at about 1170 thousand tons annually. In the same period the annual pig meat production increased from 776 to 938 thousand tons and the number of pig slaughterings increased from 12,804 to 15,957 thousands, increases of 20%. The estimated consumption of poultry meat increased from 15·8 lb. per head of population in 1964 to 23·6 lb. in 1971, an increase of 49% (*Meat and Dairy Produce Bulletins*). During this period imports of beef and veal did not increase, but remained at about 250 thousand tons annually, a quarter of total consumption; imports of pig meat (mostly bacon), remained at about 400 thousand tons annually, about half the quantity home-produced; and imports of poultry meat, only a small fraction of home production, remained at about 10,000 tons annually (*Meat and Dairy Produce Bulletins*).

The second reason concerns feeding practice. There are two important differences between the feeding of cattle on the one hand and pigs and poultry on the other. First, 90–100% of compounded cattle feeds are in the form of pellets and cubes in which the salmonella content is reduced by heating during manufacture. In contrast, only 50–60% of pig feed and 55% of poultry breeder feed is pelleted (Riley, 1967). Secondly, pigs and poultry farmed under intensive conditions are dependent on prepared feeds for all their protein requirements and it is necessary to include in their rations sources of animal proteins such as meat and bone meal and fish meal, known to be frequently contaminated with salmonellas (PHLS Working Group, Skovgaard & Nielsen, 1972). Cattle, on the other hand, graze on pastures and do not require the same proportion of concentrated animal proteins in their compounded feeds which contain mainly vegetable proteins such as groundnut. The fact that the animal by-products known to be contaminated are fed mainly to pigs and poultry and not to cattle, and that it is pigs and poultry that have been implicated in the rise in the number of human infections by serotypes other than *S. typhimurium*, strongly suggests that these animal proteins have contributed significantly to this increase.

There is strong circumstantial evidence that imported feed ingredients introduced three serotypes, *S.* 4,12:d:-, *S. agona* and *S. indiana*, into England and Wales. These types were subsequently isolated from other feed ingredients such as meat and bone meal, feather meal and poultry offal meal, which are treated animal by-products. They continued to be isolated from the animals so that it is likely that they are maintained in their animal hosts by re-cycling through the feed ingredients.

The fluctuation in incidence of some of the serotypes is difficult to explain. It is possible that serotypes whose incidence has not varied so greatly such as *S. typhimurium* and *S. enteritidis* are able to establish a host–parasite relationship that allows for their intestinal carriage and transmission from animal to animal indefinitely. Those serotypes whose prevalence has fluctuated may be more dependent on introduction in feedingstuffs for their maintenance in animals than are *S. typhimurium* and *S. enteriditis*. In a longitudinal study of salmonellas in feed, on farms and in abattoirs (Lee, Ghosh, Mann & Tee, 1972) it was shown that feed introduced a number of serotypes into the pigs, but the longest they were found in pig faeces was one month.

Investigation of incidents and outbreaks showed a path of infection from the animal reservoir to the food vehicle to man. It is difficult to break the chain of infection once carcass contamination has taken place. There may be cross-contamination between raw and cooked meats and, by handling, to other foods and equipment. Salmonellas may survive high cooking temperatures if frozen foods are not adequately thawed throughout their depth. A subinfective dose of salmonellas may multiply to an infective dose if foods are left to stand at room temperatures. The importance of the study and control of the primary animal reservoir is thus obvious.

The role of feedingstuffs in introducing serotypes into these reservoirs and their maintenance in them has recently been investigated (PHLS Working Group *et al.*

1972; Lee *et al.* 1972). In the first of these studies a comparison was made between serotypes occurring in pigs in Denmark where there is legislation requiring the sterilization of feed ingredients of animal origin whether imported or home produced, and in England where there is no such legislation. In the second study infection in pigs at abattoirs was related to contamination of feeds on the farm. It was concluded from these studies that feedingstuffs played a significant role in infecting pigs in England and Wales with serotypes other than *S. typhimurium*. The same conclusion is reached if the serotypes in poultry in Denmark (Hansen & Marthedal, 1970) are compared with those in England and Wales.

I am indebted to the Medical Officers of Health and Public Health Inspectors under whose authority investigations were carried out and to the Public Health Laboratory Directors and Microbiologists responsible for examination of the samples. These are:

Dr A. I. Ross, Medical Officer of Health, Bolton
Dr J. Kerr-Brown, Medical Officer of Health, London Borough of Greenwich
Mr F. G. Britcher, Chief Public Health Inspector, Egham Urban District Council
Mr J. G. Jenkins, Chief Public Health Inspector, Borough of Lymington, Hants.
Dr L. Robertson, Public Health Laboratory, Preston
Dr R. J. Henderson, Public Health Laboratory, Worcester
Dr L. A. Little, Public Health Laboratory, Wakefield
Dr W. J. Ryan, Public Health Laboratory, Exeter
Dr R. W. S. Harvey, Public Health Laboratory, Cardiff

I thank Dr T. M. Pollock, Dr J. W. G. Smith and Dr Mair E. M. Thomas, Epidemiological Research Laboratory, for helpful comments and assistance with the report.

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