Trends in salmonella food poisoning in England and Wales 1941–72

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SUMMARY

Cattle and pig herds and flocks of domestic fowl have formed the main reservoir of human salmonella food poisoning in England and Wales from 1941 to 1972.

Changes in the incidence of human salmonella food poisoning and in the serotypes of salmonellas isolated from human infections are shown to have been associated with the introduction of new foods, with changes in animal husbandry, and with changes in the relative proportions of flesh food from different species consumed. New foods, dried powdered egg, liquid egg and frozen liquid egg were introduced during the period of food rationing which extended from 1940 to 1953. Changes in animal husbandry, in particular the intensive production of pigs, poultry and eggs, followed the re-establishment of pig herds and fowl flocks after the derationing of animal feed in 1953. The changes in the proportions of flesh foods consumed followed the introduction of frozen oven-ready fowl in the late 1950s and early 1960s which by 1964 became cheaper than traditional flesh foods.

INTRODUCTION

The series of annual reports on salmonella food poisoning in England and Wales (Report, 1950a, b, 1951, 1954, 1955a, b, 1956, 1957, 1958a, 1959b, 1960, 1961b, 1962, 1963, 1964; Vernon, 1965, 1966, 1967, 1969, 1970; Vernon & Tillett, 1974) covering the years 1941 to 1972, have been analysed to determine the trends in human salmonella food poisoning.

Although all serotypes of salmonellas when given in sufficient numbers will produce clinical disease in man or animals or in both, one group of serotypes can be distinguished whose ingestion regularly produces septicaemic disease with high morbidity and mortality.

These serotypes are designated 'host-adapted' as the characteristic disease is confined usually to the host species. Other species are largely immune or become infected only under specific conditions, e.g. massive infecting doses, or as terminal infections in chronic disease. The host-adapted serotypes are, in bovines S. dublin: in pigs S. choleraesuis: in sheep S. abortusovis: in fowl S. gallinarum, S. pullorum. In man they are S. typhi and S. paratyphi B.

The host-adapted serotypes are responsible for serious economic loss in animals. One striking characteristic of the host-adapted infections is that a proportion

					% of all		% of	
$\mathbf{Serotype}$	Cattle	$\mathbf{Poultry}$	Pigs	Sheep	incidents	Human	incide	ents
Dublin	6359	49	35	203	34.5)	278	0.8	}
Gallinarum/pullorum	9	5780		2	29.9 71	4	0.01	
Choleraesuis	1	2	745	1	3.9 (11	109	0.3	1.1
Abortusovis	—			481	2.5	<u> </u>	—	J
Typhimurium	2391	1523	73	57	$21 \cdot 4$	22480	64.0	
Thompson	9	495	8	1	2.7	658	1.8	
Menston	8	221	3	4	1.2	183	0.5	
Enteritidis	42	138	3		1.0	1183	3.3	
Newport	14	18	1)		817	$2 \cdot 3$	
Bredeney	4	3	3	-1		403	1.1	
Brandenburg	3		1	1		830	$2 \cdot 4$	
Anatum	5	49			6.0	463	1.3	22
Bovis morbificans	13	6			1	206	0.6	
Heidelberg	9	8		— 〉 1·0		1475	$4 \cdot 2$	
Indiana	3	8				114	0.3	
Saintpaul	10	23	—	1		448	1.3	
Panama	—	1	2			476	$1 \cdot 3$	
Stanley		1	2	_	,	658	1.9/	
Paratyphi B				/		2830		
Typhi			_	—		1344		
					98 ·1		87.4	

Table 1. Numbers of incidents of salmonella infection in animals and man, England and Wales, 1958–67

Total animal incidents, 19,371; human incidents, 35,127. These totals are slightly higher than those given by the figures above, as they include a number of additional serotypes, each of which produced a very small number of incidents, and also a few incidents in several other animals.

Sources: Annual Reports on Food Poisoning, England & Wales, 1958-67; Health Trends (1969); Sojka & Field (1970).

of those infected, whilst recovering clinically continue to excrete the infecting organism indefinitely. The perpetuation of the infection is thus ensured in human and animal populations.

The remaining serotypes in general produce a transient infection characterized by acute intestinal infections of short duration, rarely complicated by septicaemia. Excretion of the serotype continues for some weeks after recovery from clinical illness, but chronic carriers are rare. Certain of these serotypes, S. typhimurium in cattle and fowl, S. thompson and S. menston in fowl, have, through methods of husbandry, become associated with particular species for longer or shorter periods.

INFECTIONS OF HUMANS AND ANIMALS

Table 1 shows the incidence of clinical salmonellosis in animals and man during a defined period for which figures are available. Animal host-adapted serotypes were isolated from 71% of food-animal infections but from only 1% of human infections. S. typhimurium, an animal host-adapted serotype, was isolated from 21% of animal infections but from 64% of human infections.

	Incidents				Incidents		
	All serotypes	S. typhi- murium	%		All serotypes	S. typhi- murium	%
1941	120	65	54	1958	4841	3329	69
1942	104	35	34	1959	5038	3198	63
1943	262	108	41	1960	3954	2907	74
1944	454	185	41	1961	3771	2503	66
1945	506	319	63	1962	2846	1864	65
1946	748	573	77	1963	2969	1820	61
1947	689	473	69	1964	3093	1725	56
1948	908	663	73	1965	2945	1721	58
1949	1393	1053	76	1966	2496	1407	56
1950	2021	1564	77	1967	3259	1810	56
1951	1367	1001	73	1968	3796	1654	44
1952	1766	1346	76	1969	4577	1346	29
1953	3114	2405	77	1970	5055	1723	34
1954	3508	2988	85	1971	5053	1955	39
1955	5269	4199	80	1972	3560	1451	41
1956	4323	3176	73	1973	5312	1708	32
1957	4218	2931	69				

 Table 2. Annual number of human incidents: S. typhimurium

 and other serotypes

Other serotypes were isolated from 6% of animal infections but from 22% of human infections.

It will be appreciated that the veterinary control of clinical salmonellosis in animals can have little effect on the incidence of human salmonellosis, as the host-adapted serotypes of animals play little part in human infection.

Feeding experiments with known numbers of salmonella of many species in pigs (Report, 1947; Smith, 1960), humans (McCullough & Eisele, 1951*a*, *b*), and poultry (Gordon & Tucker, 1965), have shown that subjects receiving numbers of organisms insufficient to cause clinical disease nevertheless excrete salmonellas. In the human experiments no significant difference was shown in the duration of excretion by those subjects who became ill and those who remained symptomless. In the pig experiments, clinical illness was not produced, but animals excreted salmonellas whilst receiving contaminated food and for some weeks after cessation of the trial. In the poultry trial, salmonellas were isolated from eggs laid within 24 hr. of the feeding of food containing on average one salmonella per g. Clinical illness was again not produced.

As clinically ill animals are rarely slaughtered for human food, and as meat inspection eliminates fevered or septicaemic carcasses, human food poisoning is ultimately attributable to contamination of carcass meats at slaughter from salmonellas present in the intestines of healthy animals.

INCIDENTS OF HUMAN INFECTION

The number of human incidents of infection rose steadily from 120 in 1941 to a peak in 1955 of 5269 incidents, followed by a steady fall to 2496 incidents

in 1966 (Table 2). Thereafter the number of incidents rose steadily to 5312 incidents in 1973 (provisional).

In 1941, 54 % of incidents were caused by *S. typhimurium*. The proportion of incidents caused by this serotype rose steadily to a maximum of 85 % in 1954, and thereafter declined to the lowest recorded proportion, 29 % in 1969.

The proportion of incidents caused by other serotypes first exceed those caused by S. typhimurium in 1968 when 56% of incidents were attributed to other serotypes.

The numbers of incidents of infection were probably under-reported up to 1948. From 1949, however, all hospital and other laboratories were reporting human incidents weekly in standard form and the trends in numbers and serotypes can be considered representative. In very broad terms the trends shown can be correlated with changes in the numbers of abattoirs, with changes in animal husbandry, and with changes in the nature of foods available for consumption.

Food rationing both human and animal extended from the outbreak of World War II to 1953. To control meat supplies, slaughter was centralized and the pre-war number of 12,500 abattoirs reduced to 600. The consequent holding of animals at abattoirs resulted in transfer of infection amongst animals awaiting slaughter, with subsequent contamination of carcass meats during slaughter. The inclusion in the meat ration of raw meats and corned beef facilitated transfer of contamination to the latter which was usually consumed without further cooking.

The derationing of food in 1953 aggravated the problem of abattoir contamination of carcass meats, as the number of abattoirs increased rapidly to roughly 5000, for which adequate numbers of trained slaughtermen and meat inspection staff were unavailable. The major animal reservoir of S. typhimurium in Great Britain is the cattle population, and until the re-establishment of the national pig and poultry populations after derationing in 1953, beef and veal formed the largest part of meat consumption.

In the mid 1950s, liquid egg both imported and home-produced was shown to be a source of human salmonella infection. The increasing numbers of outbreaks of human paratyphoid infection attributed to cream confectionery and bakers' ware was shown epidemiologically to be attributed to the use of imported Chinese liquid egg (Newell, 1955). In several surveys of liquid egg from all sources, the contamination of Chinese liquid egg and egg products with paratyphoid bacilli was confirmed and English liquid egg was shown to be heavily contaminated with S. typhimurium (Report, 1958b).

The continuation of incidents of human paratyphoid infection led eventually to the pasteurization of all liquid egg, the Regulation taking effect from 1 January 1964. By that time, however, English liquid egg and egg products had largely ceased to be a source of human salmonellosis as a result of the control of production of liquid egg by the British Egg Marketing Board which from its inception in 1957 had firmly advocated pasteurization of liquid egg and had gradually introduced pasteurization into plants under its control.

	1941/48	1949/55	1956/63	1964/68	1969/72
\mathbf{Meat}	20 (40)	245 (44)	102 (56)	85 (74)	100 (79)
Sweetmeats	10 (20)	87 (15)	32 (17)	6 (5)	
Milk	4 (8)	20 (4)	17 (9)	20 (17)	25(20)
Cheese		2	2	<u> </u>	
Eggs: duck	8 (16)	134 (24)	19 (10)	1	
other	4 (8)	19 (3)	8 (4)	4 (3)	2
\mathbf{Fish}	3	24 (4)	3		
Fruit	1	10	<u> </u>		
Vegetables		9			<u> </u>
Other foods		7	_	_	
Total	50	557	183	115*	127

Table 3. Outbreaks: vehicles of infection (percentages in parentheses)

* 1 outbreak attributed to cooked meats and sweetmeats.

The broiler industry

With the derationing of animal feeding stuffs and the resumption of imports, the broiler industry was reconstituted on modern intensive lines and by the early 1960s with the introduction of freezing for carcasses assumed essentially its present form. By 1964 chicken was the cheapest animal protein available and consumption since then has risen steadily. Coincident with this rise in the consumption of poultry meats, the number of human incidents due to serotypes other than *S. typhimurium* has risen steadily.

The pig industry

On re-establishment after de-rationing in 1953 the pig industry assumed its modern intensive form with a gradual increase in the size of units. In all intensive husbandry, one element favouring the transfer of infection is the common pattern of concentration of young animals susceptible to infection, with dispersion after slaughter of surface-contaminated meats for processing and consumption. Husbandry practices in the pig industry intended to reduce human labour requirements, such as floor feeding, and skim milk systems, have contributed to the spread of infection within herds.

Vehicles of infection

The vehicles responsible for outbreaks of human infection are shown in Table 3. Flesh meats, responsible for 40% of outbreaks in 1941/48, were incriminated in 79% of outbreaks in 1969/72. The change in the types of meat involved will be considered later.

Sweetmeats

The majority of infections caused by sweetmeats is attributed to the use of raw, liquid or dried whole egg or other egg product in bakers' wares to which no heat was applied either during preparation or after. Examples are artificial or natural creams containing egg or egg products, and the use of egg products

	1954/55	1956/63	1964/68	1969/72
Beef, veal, 'meat'	8 (28)	17 (19)	27 (36)	17 (17)
Pork	12 (41)	31 (36)	12 (16)	17 (17)
Poultry	1 (3)	12 (14)	33 (45)	66 (66)
Made up meats	8 (28)	27 (31)	2 (3)	
All	29	87	74	100

Table 4. Outbreaks: vehicles of infection, meat (percentages in parentheses)

 Table 5. Outbreaks: vehicles of infection, poultry meat

 (percentages in parentheses)

	1954/55	1956/63	1964/68	1969/72
Fresh roast/cold/reheated	1	11 (92)	25 (72)	53 (80)
Sandwiches		1		—
Spit roasted			8 (24)	7 (11)
Other and unspecified	<u> </u>	—		6 (9) .

for the final glazing of pies after cooking. Though many artificial creams have been shown to be incapable of supporting bacterial growth and multiplication, and although salmonellas in egg incorporated in cake mixtures have been shown to be destroyed on baking, the moisture from creams applied to cakes leached from the cakes sufficient nutrients to support bacterial growth and multiplication (Thomson, 1953). A further source of contamination of bakers' confectionery was found in desiccated coconut, particularly that imported from Ceylon, where the use of polluted surface waters in the manufacture of the product resulted in heavy contamination with salmonellas including paratyphoid bacilli (Anderson, 1960). Since the introduction of pasteurization for liquid egg in 1964, and the use of hygienic methods of preparation of desiccated coconut, sweetmeats have ceased to be a source of human infection.

Eggs

Duck eggs, largely unrationed during the period of rationing, proved a potent source of human infection. Most of the outbreaks were caused by individual duck eggs in distinction to other egg outbreaks which were caused by liquid, dried or other egg products.

After the introduction of pasteurization, egg outbreaks fell to negligible numbers. The two outbreaks in 1969/72 attributed to eggs were caused by salad creams based on raw shell eggs.

Milk outbreaks

Most milk outbreaks result from the consumption of raw milk, mainly on farms producing milk, but extensive outbreaks also occur amongst consumers supplied by producer/retailers of unpasteurized (raw) milk. Despite the fall in the number of producer/retailers from 53,480 in 1945 to 6246 in 1972 (Dairy Facts and Figures, 1972), the number of milk-borne outbreaks shows no decline.

$\mathbf{276}$

	1941/48	1949/55	1956/63	1964/68	1969/72
S. meleagridis	28 (10)	31	110	15	N.A.
oranienburg	125 (5)	87	70	139	N.A.
montevideo	108 (6)	102 (10)	114	139	289
thom pson*	210 (3)	667 (3)	757 (5)	89	244
newport*	207 (4)	392 (4)	863 (4)	182	270
bovismorbificans	43 (8)	195 (5)	229 (10)	60	N.A.
brandenburg	1	19	215	684 (4)	285
dublin*	62 (7)	168 (6)	192	244 (9)	317
anatum	37 (9)	149 (7)	346 (8)	309 (8)	422
typhimurium*	2421 (1)	15238 (1)	22009 (1)	8316 (1)	10360 (1)
enteritidis*	261 (2)	708 (2)	973 (3)	830 (2)	3528 (2)
$stanley^*$	5	140 (8)	306 (9)	585 (5)	585 (10)
heidelberg		104 (9)	1585 (2)	330 (6)	1088 (5)
bredeney	4	42	351 (7)	218 (10)	739 (9)
saintpaul	6	69	403 (6)	124	800 (7)
panama	4	4	58	808 (3)	2302 (3)
virchow	2	29	43	321 (7)	932 (6)
indiana			1	200	797 (8)
agona					1778 (4)
infantis	1	11	142	126	514
4, 12:d:-					379
derby*	7	82	202	117	307
muenchen	2	40	127	74	265
menston		24	145	46	N.A.
choleraesuis*	16	64	102	44	N.A.
senftenberg	20	60	83	122	N.A.

Table 6. Human salmonellosis: serotypes (rank in parentheses)

* 'native' serotypes present, Great Britain 1923-39. N.A. = figures not yet available.

Fish, fruit, vegetables

Fish, fruit, vegetables and other foods appear as vehicles of infection only during the period of food rationing. Most of these outbreaks were attributed to canned, dried or dehydrated materials.

Types of meat

Table 4 analyses in more detail the types of meat incriminated in outbreaks where the vehicle was identified exactly.

The most marked features are the steady increase in poultry and the fall in 'made-up' meats as vehicles. Poultry has been more attractive in terms of value for money since about 1964 and the annual consumption in lb. per head has risen steadily from about 5 lb. during food rationing to 18 lb. in 1968. The decline in the proportion of outbreaks attributed to 'made-up' meats may tentatively be ascribed to the growing consumption of 'convenience' foods which have replaced many traditional 'made-up' meats.

Table 5 analyses in more detail the types of poultry meat involved in outbreaks. Spit-roasted chicken appears first in 1968 as the source of extensive outbreaks in north-east England. Turkey is mentioned first in the period 1969/ 72, a reflexion of the slower growth of the turkey 'broiler' industry. Most of

18

нүс 74

these outbreaks can be attributed to either insufficient thawing of frozen birds before cooking: to inadequate cooking particularly of spit-roasted birds and large 'banqueting' turkeys, to contamination after cooking, or to a combination of all these faults.

Distribution of serotypes

Table 6 shows the change in the distribution of serotypes isolated from human infections from 1941 to 1972.

In general this change can be linked with the introduction of new foods, the cessation of rationing, the pasteurization of liquid whole egg, and the use of contaminated animal feed.

New foods

1941/48. Dried egg

Two new foods were introduced, dried egg and dried milk, both rich in protein and for which contracts had been prepared and orders placed during contingency planning in the mid 1930s for the coming war and food rationing. Dried milk provided no problems. Dried egg introduced a range of salmonella serotypes common in fowl in the U.S.A. but not isolated from any source in Great Britain before the introduction of dried egg. A subsidiary source of human infection resulted from the use for animal feed of dried egg imports considered unfit for human consumption. The serotypes mainly involved were *S. meleagridis*, *S. oranienburg*, *S. montevideo* and *S. anatum* (Report, 1947).

1948/63. Frozen liquid egg

Imports of dried egg and other products from the U.S.A. ceased abruptly in 1948 with the end of Lease-Lend, and supplies, particularly liquid frozen egg, were obtained from Commonwealth (soft currency) areas to supplement home production. Frozen liquid egg proved to be no less contaminated with salmonellas than dried egg.

Extensive surveys in 1955/56 showed Chinese liquid egg and egg products to be heavily contaminated with S. paratyphi B, a purely human pathogen, and S. thompson (Report, 1958b). English liquid egg was more heavily contaminated with S. typhimurium than egg from any other source. In general during this period the pattern of serotypes had almost reverted to the pre-war pattern. No source was however found for S. heidelberg which first appeared in 1953.

,1964/72. Broiler fowl, spit-roasted fowl

Broiler fowl were produced in increasing numbers annually after the introduction of freezing in the late 1950s, which extended the shelf life of fowl after production by many months in comparison to the shelf life measured in days of fresh chilled uneviscerated fowl. The expansion of the industry depended largely on the acquisition of desirable genetic characters which became available through the import of day-old chicks and fertile eggs with the revocation in 1963 of regulations directed against the import of Newcastle disease in day-old chicks. Fowl salmonellosis is, however, egg-transmitted and several serotypes imported

278

with day-old chicks rapidly became established in breeding flocks. S. enteritidis, S. heidelberg, S. bredeney, S. virchow, are now recognized as established in breeding flocks of fowl.

Spit-roasted broiler chicken as a vehicle first came to attention in 1967 (Vernon, 1969), and 1968 extensive outbreaks of *S. virchow* (Semple, Turner & Lowry, 1968; Pennington, Brooksbank, Poole & Seymour, 1968). Spit-roasting on a large scale involves difficulties in the proper time of cooking, in cooling, reheating and the prevention of contamination of cooked chicken.

The cessation of rationing

With the cessation of rationing of the raw materials of animal feed in 1953, imports from traditional and new sources were resumed. The re-establishment of the national pig and poultry stocks from the breeding stock at which they had been maintained during the War and post-war years began on intensive lines. In fowl, the first increase was in fowl for egg production and egg production increased rapidly. Most of the increase in infection with *S. thompson*, *S. menston* and *S. typhimurium* in the period 1956/63 can be attributed to the use of homeproduced liquid egg. The increases in infection due to *S. newport*, *S. bredeney*, *S. infantis*, *S. derby* and *S. muenchen* can be attributed to meat products. These serotypes were amongst the most common isolated from imported and homeproduced animal and fish protein used in animal feed. These proteins had been shown in the late 1950s and early 1960s to be heavily contaminated with salmonellas (Report 1959a, 1961a).

The pasteurization of liquid egg

The pasteurization of liquid whole egg was introduced on 1 January 1964 mainly as the result of outbreaks of human paratyphoid fever from bakers' confectionery incorporating Chinese liquid frozen egg, known since 1955/56 to be heavily contaminated. The immediate effect was a reduction in human outbreaks of infection by serotypes known to be egg-transmitted, S. thompson, S. typhimurium and S. menston.

Infections with S. thompson and S. menston have continued since 1964, as the main reservoir in breeding stocks of fowl remains, though one vehicle, liquid egg, has been eliminated.

The magnitude of the fall in human infections with S. typhimurium which can be attributed to the pasteurization of liquid egg cannot at the moment be assessed exactly. The fall in the annual number of incidents of human infection with S. typhimurium in the two years after the introduction of pasteurization is about 25%, but this figure is thought to underestimate the effect of pasteurization as the fall in human infections with S. thompson and S. menston in the same period is much greater. The animal reservoirs of S. typhimurium are cattle in addition to fowl, whilst S. thompson and S. menston have their reservoirs almost entirely in poultry.

Contaminated imported fish meal was responsible for the introduction of two serotypes, S. 4, 12:d:- in 1969, and S. agona in 1970.

18-2

The first feed containing imported fish meal contaminated with S. agona was in March 1970. By early June 1970 human infections had occurred from spitroasted chicken, roast chicken, and contact with day-old chicks (PHLS records, unpublished). In the following month a milk-borne outbreak was recorded, along with deaths in several areas of turkey poults from the same hatchery. Since its introduction, S. agona has been isolated in addition from liquid egg before pasteurization, from another extensive milk-borne outbreak, from broiler chickens, turkeys, cattle, and pigs.

S. 4, 12:d:- has since its introduction been isolated from the same sources, with the exception that no milk-borne outbreak has yet been recorded. S. agona has been isolated from only one cargo of imported fish meal since its first introduction. However it is now established in breeding flocks of fowl where it may be expected to maintain itself indefinitely.

DISCUSSION

The control of human typhoid and paratyphoid infections in Great Britain has been effected, largely by the elimination of sewage pollution in water supplies for drinking and for the preparation of human foods.

The elimination of salmonella food poisoning is more difficult as the reservoirs of infection are food animals and fowl; the vehicles of infection their flesh and products.

Except in special circumstances, the detection and eradication of healthy animals and fowl excreting salmonellas has not been attempted and for economic reasons is unlikely to be attempted.

The main lines of approach to control are therefore indirect and may be summarized briefly as the prevention of the introduction of infection to flocks and herds by either the introduction of excreting animals or the use of contaminated feed: limitation of the spread of infection once introduced; reduction of contamination by intestinal contents during slaughter; refrigeration to prevent growth and multiplication of organisms; the use of heat to destroy organisms by pasteurization or sterilization after processing; proper cooking and the prevention of contamination after cooking.

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280

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Note added in proof. 28 January 1975. The enteric fevers of man (typhoid and the paratyphoid fevers) have not been considered as these infections, though food and waterborne, are not classified as food poisoning. The statistics of these infections are published in the Annual Reports of the Chief Medical Officer.

During the years reviewed the following changes in the reporting of food poisoning have been made.

- 1941-61 Incidents of food poisoning including symptomless excretors.
- 1962–68 Incidents and cases of food poisoning including symptomless excretors.
- 1969-72 Cases of food poisoning. Symptomless excretors not included but shown separately.

To ensure continuity, the average number of cases per incident was calculated from the 1962–68 statistics, and this factor used to convert the 1969–72 numbers of cases and symptomless excretors to incidents.