BRITISH MEDICAL JOURNAL

LONDON SATURDAY AUGUST 11 1956

PROBLEMS OF SALMONELLA FOOD-POISONING

BY

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Tables I, II, and III are compiled from the annual reports on food-poisoning contained in the *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service*. Large as are the figures, they undoubtedly are not a complete record of all cases of food-poisoning. Three important facts stand out from these tables: (1) the steady increase in the number of recorded cases of food-poisoning; (2) the overwhelming proportion of cases which are ascertained to be due to salmonellae; and (3) the very high proportion of "sporadic" cases. It is obvious that the most important problem in food-poisoning is salmonella outbreaks, and their elimination, or at least material reduction, would reduce food-poisoning to manageable limits. For this reason this paper is limited to this group.

TABLE I.—*Food-poisoning Outbreaks*

Type	1949	1950	1951	1952	1953	1954
Outbreaks ..	410	539	343	372	492	506
Family outbreaks	265	453	287	340	422	630
Sporadic cases ..	1,753	2,987	2,717	2,807	4,363	4,880
Total incidents ..	2,428	3,979	3,347	3,519	5,277	6,016

Note.—All cases, single or multiple, are called *incidents*, and these are classified into three groups: "Outbreak," two or more related cases in persons in different families; "Family outbreak," two or more related cases in members of the same family; and "Sporadic case," a case which was not, so far as could be ascertained, related to other cases.

TABLE II.—*Total Incidents*

Causation	1949	1950	1951	1952	1953	1954
Salmonellae ..	1,369	2,021	1,668	2,098	3,114	3,508
Staphylococci ..	97	82	65	82	132	127
Cl. welchii ..	—	24	20	20	25	48
Other organisms	88	43	5	17	6	9
Chemical ..	3	2	2	1	0	2
Not discovered ..	874	1,807	1,587	1,301	2,000	2,322

TABLE III.—*Incidents with Causation Determined*

	1949	1950	1951	1952	1953	1954
Outbreaks and family outbreaks:						
No. of salmonella origin ..	307	467	301	242	532	647
Percentage of ,, ,, ..	64·5	77·1	73·4	69·8	81·9	81·5
Sporadic cases:						
No. of salmonella origin ..	1,062	1,554	1,367	1,766	2,582	2,861
Percentage of ,, ,, ..	98·5	98·5	99·5	99·2	98·2	98·3

Figures for earlier years are given in the *Monthly Bulletin* for June, 1950. The number of incidents reported from 1941 to 1948 are: 1941, 119; 1942, 73; 1943, 244; 1944, 291; 1945, 433; 1946, 685; 1947, 846; and 1948, 804. For these eight years the total was 3,495 incidents, and 3,193 of these were due to salmonellae (91.4%). The total for the whole eight years is conspicuously less than for a single year to-day.

Still further back, during the period 1921–4, by arrangement with the Ministry of Health and the Medical Research Council, local health authorities throughout the country were offered free examination and investigation in my laboratory for all food-poisoning outbreaks. Although the number so investigated was large, it was quite small compared with the number of present-day outbreaks. The enormous increase is striking. On the other hand, three factors have influenced the *recognition* of outbreaks which in earlier years might not have come to light. One is the compulsory notification of food-poisoning which undoubtedly has supplied information of more outbreaks. Secondly, the extensive laboratory facilities now available all over the country have greatly increased the utility of notification and of scientific investigation. Thirdly, these laboratory facilities have also encouraged medical officers of health to take more interest in food-poisoning. These factors have played an important part, but I do not think they have been responsible for any large share in closing the enormous gap between, say, 20 years ago and to-day.

The most striking features of Table I are the enormous number of "sporadic" cases and their regular increase year by year. The laboratory returns give very little information on these incidents. Steps have been initiated asking for samples of faeces from the home contacts, but I am informed that the results are not very encouraging. It is unrealistic to suggest that these are isolated cases without any connexion with other cases, and this is borne out by the only serious effort I have come across to investigate them.

Dr. G. T. Cook, formerly of the Public Health Laboratory Service, at Oxford, now at Guildford, carried out some investigations on sporadic salmonella cases and is kind enough to allow me to quote his findings.

He visited 27 sporadic cases, 22 due to *Salmonella typhi-murium*, and made detailed inquiries into possible sources of infection. Faecal specimens from contacts and, when indicated, samples of food were examined bacteriologically. In two of the seven men affected the attacks were associated with the consumption of duck eggs the day before onset. Another was linked with previously unsuspected gastro-enteritis in his daughter two days before, who was found to be excreting *Salm. typhi-murium*. Two further cases, apparently unrelated, were probably infected from sandwiches shared at work and prepared by the mother-in-law of one of the patients, a symptomless excreter of *Salm. typhi-murium*. The six women included three infected with *Salm. typhi-murium* who shortly before the attack had tasted, while

cooking, a " mix " containing raw duck eggs, which may have been the source of infection. Nine out of 19 family contacts of these six women were found to be excreting *Salm. typhi-murium*, though none had had symptoms. Among eight cases of children under 5, no evidence of source was obtainable, though two households had positive family contacts. In another group of six older children, one had been associated with duck eggs and another had probably been infected by his mother, a symptomless excreter of salmonellae who had recently returned home from nursing a relative with gastro-enteritis.

In all, the households of 40 sporadic cases were investigated, and in 23 of these one or more positive salmonella contacts were found. Of the 105 home contacts examined, 36 were positive for salmonellae, though only 6 of these had symptoms of food-poisoning.

In view of the fact that these were one-man investigations by a bacteriologist without the full resources of a health department, and that it was usually possible to collect only a single sample from each person, the findings suggest how well worth while really detailed investigations would be, both in the homes and into possible outside sources of infection.

A feature brought out in Table II is the large proportion of incidents for which the cause was not ascertained. Remedies for this unsatisfactory feature are discussed below.

The Salmonella Group

When I started working on this group around 1905 only three distinct serotypes (excluding *Salm. typhi*) were recognized. More were separated over the next few years, and slowly at first and then more rapidly the number has increased to just over 300 different serotypes. The group is united by cultural and chemical characteristics with some variations with special selected agents, but the main distinctions are founded upon antigenic differences. Still further improvements of classification are being obtained by bacteriophage typing. The types most commonly met with in food-poisoning cases in this country are shown in Table IV. The two striking features are the outstanding prevalence

TABLE IV.—*Main Salmonella Types Isolated from Food-poisoning Incidents*

Salmonella Strains	1949	1950	1951	1952	1953	1954	6-year Total
Endogenous:							
Typhi-murium ..	1,053	1,593	1,236	1,604	2,438	3,038	10,962
Enteritidis (including substrains) ..	51	91	99	145	126	70	582
Thompson	82	67	97	90	84	83	503
Newport	53	77	71	40	50	35	324
Dublin	24	40	23	17	20	15	139
Cholerae-suis ..	2	2	3	6	12	11	36
Bovis-morbificans ..	13	23	14	6	81	26	163
Derby	1	7	5	6	11	10	40
Stanley	0	1	4	19	4	3	31
Exogenous:							
Anatum	8	20	11	10	30	20	99
Montevideo ..	9	18	23	12	18	13	93
Oranienburg ..	9	11	6	10	29	6	71
Bareilly	1	14	5	15	25	3	63
Minnesota	0	4	5	13	17	11	50
Give	1	2	10	17	8	2	40
Tennessee	2	13	16	5	15	8	59

Endogenous types = those always common in England and Wales.
Exogenous types = mainly isolated only after 1940.

of *Salm. typhi-murium*, constituting 83% of all the strains isolated, and the complete absence of *Salm. paratyphi B* as a cause of acute food-poisoning. Both features are also shown by the reports for earlier years.

None of the group is a natural saprophytic inhabitant of the intestines of men or animals. I wrote in 1920 (Savage, 1920) : " These bacilli are not *natural* inhabitants of the animals used for food and not found more frequently than can be accounted for on the supposition that their presence is due to an actual case of disease or the carrier state after infection." (This was by no means accepted by all in 1920.) They are found causing disease in a very wide variety of animals and with some evidence of host selection. The literature on their distribution is so extensive that only a broad sketch of their animal distribution can be attempted.

Animal Sources

Birds.—Poultry seem to be the commonest reservoir of salmonellae. Ducks are especially significant in relation to food-poisoning because salmonella infections may be widespread on duck farms and the organisms can invade the blood stream and oviduct, with infection of the individual egg, while from dirty infected egg-shells the organism may penetrate the interior of the egg. Smith and Buxton (1951) examined faeces from 500 ducks and found 1.2% positive. Salmonella infections in fowls are even more common, but are much less a source of human food-poisoning, because the two commonest strains *Salm. pullorum* (cause of chick diarrhoea) and *Salm. gallinarum* (cause of fowl typhoid) are practically non-pathogenic to man. A whole range of other salmonellae have also been isolated from eggs, particularly in the U.S.A. *Salm. typhi-murium, Salm. thompson*, and *Salm. enteritidis* are important strains isolated from hens or their eggs. Turkeys also suffer from salmonella infections and have been a cause of food-poisoning. Many other birds, such as pigeons, geese, and pheasants, may be infected.

Pigs.—Pigs seem to have a considerable liability to salmonella infections. Scott (1940), in Great Britain, examined batches of mesenteric glands and spleens from apparently normal pigs, and in one factory found salmonellae in 23 of 500 pigs and in 18 out of 500 pigs of another factory. While nine different strains were isolated, the commonest were *Salm. cholerae-suis* (10), *Salm. typhi-murium* (9), and *Salm. thompson* (7). In a more recent investigation in Great Britain (M.R.C., 1947) salmonellae were present in 133 of 1,058 batches of lymph nodes representing 5,285 pigs (2.5%). Smith and Buxton (1951) isolated salmonellae from faeces from 4 out of 600 apparently healthy pigs.

Cattle.—Although probably less common as a reservoir than pigs and birds, cattle are important and illustrate some interesting points. *Salm. dublin* is the main type found, but *Salm. typhi-murium* also occurs, especially in older cattle. Full surveys have not been made, but our present information suggests large pockets of infection in areas geographically far apart. One pocket is in Mid- and West Wales, where Field (1948) has carried out extensive studies. Salmonellosis was diagnosed on 70 farms during 1946–7, in every instance due to *Salm. dublin*, except on one farm with *Salm. typhi-murium*. The cattle mortality rate was about 70%. Another pocket seems to extend over parts of Ireland. Ritchie and Clayton (1951), investigating in 1949–50 presumed healthy cattle slaughtered at Birkenhead abattoirs, found *Salm. dublin* in 10% of faeces and in 3.4% of bile samples from Irish cattle. The corresponding figures for non-Irish cattle were 3.7 and 0.5%. Liver swabs from 25 out of 202 Irish cattle yielded *Salm. dublin*, but swabs from 171 non-Irish cattle yielded none. Smith and Buxton (1951), found only three salmonella-positives among 750 samples of faeces from cattle in Great Britain; all were *Salm. dublin*. In 1950–2 Murdock and Gordon (1953) isolated *Salm. dublin* from 86 (8.6%) North Ireland cattle. In Somerset there were 14 outbreaks of *Salm. dublin* infection in 1947–53, and McCall (1953) reported on three human *Salm. dublin* outbreaks in that county, the largest involving 598 children infected from school milk.

Rats and Mice.—Salmonella infections are common in both species and many survivors continue as excreters of salmonellae. In rats the prevalence varies with the sources of these animals. In places where animals are killed, such as slaughterhouses, the prevalence may be over 6%, and in

Liverpool docks with much food storage Khalil (1938) found 7.3% harbouring salmonellae, all but four strains being *Salm. enteritidis* or *Salm. typhi-murium*.

Cats and Dogs.—Cruickshank and Smith (1949) examined 500 samples of faeces from apparently healthy dogs and found 5 positives, and from 500 apparently healthy cats 7 were salmonella-positive—3 *Salm. typhi-murium*, 2 *Salm. anatum*, and one each of *Salm. montevideo* and *Salm. paratyphi B*. They give particulars of many other investigations on cats and dogs with positive findings. Both animals not only can carry salmonellae but can suffer from definite infections. (This is in accord with findings in a few food-poisoning outbreaks.) The authors make the interesting calculation, based on dog licences issued, that their findings, if a fair sample, represent 20,000 to 30,000 dogs in Great Britain excreting salmonellae at any one time.

Commercially Prepared Egg Mixtures

Egg mixtures raise problems quite distinct from findings from flocks or from single eggs, so are discussed separately. The bacteriology of imported U.S.A. spray-dried egg has been described in detail (M.R.C., 1947). The powder is either prepared from shell eggs or from frozen egg pulp. Two facts stand out. One is that the examination of 7,584 samples revealed salmonella strains in 9.9%. The other is that the use of these mixtures in Great Britain resulted in the introduction of a large number of salmonella types new to this country but present in the U.S.A. and later found to be responsible for food-poisoning outbreaks in this country. In that M.R.C. report four such outbreaks are described, due to the introduced strains *Salm. oranienburg*, *Salm. montevideo*, *Salm. tennessee*, and *Salm. sundsvall*.

Murdock (1954) during 1951–3 examined liquid egg mixtures made up of North Ireland eggs sampled at the stage from homogenizer to the receptacle cans. Salmonellae were isolated 21 times from batches representing 1,610 hen's eggs (2.1%) and 120 times from 249 batches of duck eggs (48.2%). The majority of strains were *Salm. typhi-murium*.

A more comprehensive survey (Report, 1955) undertaken by members of the Public Health Laboratory Service dealt with English egg mixture collected at egg factory stations, classified into three groups—fresh untreated mixtures, chilled mixtures, and frozen mixtures in cans. When hen eggs were used, the first two groups gave 0.3% positive for salmonellae; frozen egg mixtures gave 2.0% positive. With duck egg mixtures the percentages were respectively 6.1, 0, and 1.7.

Chinese frozen egg mixtures have been examined by Smith and Hobbs (1955). Salmonella strains were isolated from 27% of 128 cans examined. The findings varied according to types of can : tall 22-lb. (10-kg.) cans, 5.9% ; 22-lb. (10-kg.) flat cans, 23% ; and 44-lb. (20-kg.), 3% positive. The strains isolated were *Salm. pullorum*, *Salm. thompson*, *Salm. aberdeen*, and *Salm. typhi-murium*, but the last was found only twice.

Newell (1955) describes a number of outbreaks and single cases associated with Chinese egg mixtures used in bakeries for cream cakes. Newell *et al.* (1955) reported two outbreaks, in Worthing and Weymouth areas, of paratyphoid fever due to *Salm. paratyphi B* type 3a in cakes filled with imitation cream prepared in two bakeries in which there were unopened cases of Chinese frozen whole egg contaminated with the same type 3a *Salm. paratyphi B*. It is suggested that the imitation cream was contaminated in the bakeries from the frozen egg.

This brief summary of the distribution of salmonellae in the animal world of Great Britain shows how widespread are these reservoirs of salmonellae, and suggests that it is here we must look for the origins of the salmonellae in most food-poisoning outbreaks.

In other countries the distribution of salmonellae types shows considerable differences, as indeed we find from the many foreign types introduced by packaged eggs from the U.S.A. Edwards and Bruner (1943) made an analysis of 3,090 salmonella strains from outbreaks in man and animals up to 1943 in the U.S.A. and distinguished 59 types. Pigs, for example, yielded 27 different types with *Salm. choleraesuis* (*kunzendorf*) from 305 outbreaks, 329 cultures, and 28 human outbreaks. *Salm. dublin* was only twice isolated from cattle, but foxes had a high incidence. *Salm. newport* was often found in fowls and human outbreaks, but rarely in swine, while *Salm. thompson* was almost confined to human outbreaks. *Salm. bovis-morbificans* is rare in the U.S.A. and until recently was uncommon in Great Britain, but it seems to be fairly widely distributed in Australia.

Evolution of Salmonella Strains

The enormous number of distinguishable salmonella types, the wide range of animals invaded, and the varying types of infections caused form a complex picture. In my opinion the problems raised can best be linked, and in part explained, by considering them from an evolutionary standpoint. As long ago as 1932 I expressed the view that the salmonella group is in an evolutionary stage as regards the separation of types, selection of animal hosts, and varieties of parasitic functions, and later I elaborated this conception (Savage, 1940). *Salm. typhi-murium* is probably nearest to the common ancestor, catholic in its distribution and undifferentiated in its host selections. Specialism has enabled some strains to attain a high measure of host specificity. *Salm. paratyphi B* has become for all practical purposes a human invasive parasite causing long-continued infection and rarely gastro-intestinal symptoms. It is no longer invasive to the lower animal by natural infection. Other strains with a high degree of host specialization are *Salm. abortus-ovis* for sheep, *Salm. gallinarum* and *Salm. pullorum* for fowls, and *Salm. abortus-equi* for horses. *Salm. dublin* is particularly interesting, for it tends to limit itself to calves and cattle but so far very incompletely, and this characteristic is accompanied by varied patterns of pathological changes which emphasize its instability.

These statements are probably in no single instance absolute, which is exactly what we should anticipate in evolutionary strains. For example, *Salm. paratyphi B* in a very few instances does cause gastro-intestinal symptoms, while there is reliable evidence that dogs and possibly pigs may occasionally be invaded by this otherwise purely human parasite. The general picture of paratyphoid fever is, however, very definite, and not a single outbreak of food-poisoning in the long records of the Public Health Laboratory Service has been ascribed to *Salm. paratyphi B*.

Given suitable conditions for growth, bacteria produce in less than a week as many generations as man takes from the Bronze Age to the present time, so evolutionary changes are to be anticipated with the production of new antigenic types better adapted to extend their distribution in animals. This extension may very well be taking place, but so far the evidence is not conclusive. Evidence concerning the evolutionary character of the salmonella group is reinforced by comparatively recent work demonstrating that type-transformation is possible. Three methods have been successful. One is by growing the organism in antiserum : Bruner, for instance, in 1949, succeeded in transforming *Salm. oranienburg* into *Salm. montevideo*, but was unable to reverse the process.

Factors Necessary for Full Elucidation of Each Outbreak of Food-poisoning

No outbreak can be said to be fully explained unless four factors are solved. These are the bacterial or other cause of the symptoms, the vehicle responsible for conveying the causal agent to man, the reservoir from which the infecting organism originated, and the path from the reservoir to the infected vehicles.

Salmonella Infections

Only salmonellae are under consideration, and their distribution has been discussed. It is of interest to study the relative presence of the different salmonella types in actual

cases of food-poisoning. Those for the last six years available are shown in Table IV. The most striking feature is the overwhelming prevalence of *Salm. typhi-murium,* and we may associate this with its ubiquity of distribution in the animal kingdom. Another interesting point is the fairly regular way *Salm. enteritidis* and *Salm. thompson* take the second and third place, with *Salm. newport* usually fourth. This, I think, is evidence of an association of food-poisoning with animal reservoirs. *Salm. typhi-murium* is of wide animal distribution largely impartial in respect of animal hosts. *Salm. enteritidis* is persistently found connected with diseased cattle, and is perhaps the most frequently isolated strain from emergency-slaughtered cattle in Germany causing food-poisoning. It is especially associated with meat as a food-poisoning vehicle.

The primary and prevalent sources of *Salm. thompson* and *Salm. newport* are still somewhat of a puzzle. Both have been found in lymph nodes of pigs; *Salm. thompson* is a common cause of chicken disease in Great Britain (Buxton and Gordon, 1947) and is not uncommon in imported eggs. It is possible that birds are the primary sources of both strains. The origins of the exogenous strains have been considered under imported eggs. Naturally some strains may be more prominent in particular years owing to special outbreaks due to a particular type—such as the rise in frequency of *Salm. bovis-morbificans* in 1953 associated with a large outbreak in Lancashire due to meat pies.

Vehicles

The main groups were fairly comprehensively worked out many years ago, but it is of value to discover if there have been material changes over the last 20 years or so. This is done in Table V, but it includes outbreaks due to causes

TABLE V.—*Vehicles in Food-poisoning Outbreaks*

Vehicle	Outbreaks 1919–31*	Outbreaks 1951–3†
Meat, fresh	8	4
,, somewhat manipulated	10 } 47	92
,, heavily processed	37 }	
Canned foods	8	14
Milk and milk products	12	11
Eggs, duck	6	45
,, other	1	12
Shellfish	2	1
Sweetmeats (trifle, ice-cream, custard, etc.)	Not separately recorded	36

* From Savage (1932).
† From *Monthly Bull. Minist. Hlth,* 1954, **13**, 12; and 1955, **14**, 34.

other than salmonellae, as the bacterial groups are not separated. While only a rough guide, the figures show that the vehicles to-day are essentially the same and in very much the same proportion as 30 years or so ago, with one striking exception—that is, the great increase in eggs as a vehicle (partly because of failure to recognize their importance). This is particularly so for duck eggs. The overwhelming importance of manipulated meat food is about the same for both periods. There are, however, other differences between the two age periods not brought out in the table. One is the much greater safety of canned food to-day; many of the outbreaks now recorded as associated with canned foods are due to the infection of such products after removal from the can. The present ice-cream regulations have greatly increased the safety of this food. Changing food habits and commercial practices have resulted in a far more extensive consumption of meat foods prepared in quantity in works outside the places of consumption, and, if specific infection occurs in places where such food is prepared, resultant outbreaks are usually much more extensive than those from the old-time home or shop-made brawn, pressed beef, and the like. A good example, described by Miller *et al.* (1955) is the very extensive outbreak of salmonella food-poisoning (*Salm. bovis-morbificans*) in 1953, extending over a large part of West Lancashire and involving over 1,149 persons, with 5 deaths. The vehicle was meat pies, all made in one bakery but using meat supplied by a number of butchers.

Another example of widespread salmonella infection from meat is the very extensive outbreak in 1953, extending over many parts of Sweden (Lundbeck *et al.,* 1955) due to *Salm. typhi-murium* and involving 8,845 known cases with at least 90 deaths. The meat was all distributed from one large abattoir, the infection probably being introduced by infected animals and spreading widely through the abattoir by direct transfer, infected faeces playing a large part. Examination of carcasses showed *Salm. typhi-murium* of the same phage type in 11.2% of glands and 9.6% of muscles, while the pork carcasses yielded even higher percentages. Of 360 personnel examined 53 (15%) showed the same strain of salmonella.

An important factor to be considered in relation to the vehicle is its suitability for bacterial multiplication. Large numbers of salmonellae appear to be necessary to cause food-poisoning, and this usually involves multiplication outside the human body. Hormaeche and Salsamendi (1936, 1939) found that human volunteers might ingest as many as 2,000–4,000 million salmonellae (*Salm. typhi-murium*) without any ill effects.

McCullough and Eisele (1951) carried out a very large number of feeding tests with human volunteers, and the following findings illustrate the influence both of dosage and of strain variations within the same type.

Salm. meleagridis.—Of the three strains used strain III was the most virulent, and illness was caused in a minority of cases with about 750,000 bacteria; with a 20-million dose of strain I. only one out of five was ill, but with a 30-million dose four out of six were ill.

Salm. anatum.—With the two most toxic strains doses of 3,500,000 and 1,250,000 were the lowest to cause symptoms, but with the third strain 45,500,000 bacilli merely caused mild ill effects and then only in one out of six fed.

Salm. newport.—More toxic, but of 16 fed with doses of 152,000 to 385,000 bacteria only two became ill; with 1,350,000 three out of six were ill.

Salm. derby.—No illness until 15,000,000 fed, and then only three out of six affected.

Salm. bareilly.—Dosage 125,000: out of six fed, one ill. Dosage 695,000: out of six fed, two ill. Dosage 1,170,000: out of six fed, four ill.

Salm. pullorum.—A large number of strains used, but only when the dose ranged from 1.3 billion to 10 billion did any illness result, and that was slight and brief.

These findings emphasize the significance of the nutrient vehicle and explain a common experience that articles such as trifle, part consumed at time of preparation, are usually harmless, while next day, if kept under conditions favouring multiplication of bacteria, they cause widespread illness.

This numerical factor explains how frequently salmonella carriers can work on food premises for long periods without causing outbreaks if the conditions deny opportunities of inoculation of nutritive foods. Thomson (1954) has shown that salmonella carriers of typhoid and paratyphoid bacilli may excrete enormous numbers of these bacilli in their faeces, but I have not found any information on whether this applies to carriers of food-poisoning salmonella.

Paths of Infection

Frequently the most unrewarding part of an investigation is tracing the path from the reservoir to the vehicle. The following points have to be considered.

The consumption of meat from a salmonella-infected animal, either obviously ill or without noticeable changes even to a trained meat inspector. Outbreaks from emergency-slaughtered animals used to be fairly common in Germany. Food-poisoning bacilli may be transferred by animal products, usually milk, the bacilli being either derived from cows suffering from a salmonella infection (usually *Salm. dublin*) or gaining access from infected dung. Infection of the food by salmonella-carrying intermediaries such as rats, mice, cats, and dogs. The risk is materially increased when rats are prevalent in places where

food is prepared. Savage and White (1922–3) isolated *Salm. enteritidis* from 6 out of 96 rats from slaughterhouses, and Ludlam (1954) found salmonellae in only 4.4% of rats from mixed types of premises, but in 6.4% from 94 rats from a butcher's by-products factory.

Originally sound food in catering premises, shops, or homes may be specifically contaminated either directly by the incorporation of infected egg mixtures or indirectly by such egg mixtures contaminating various food-preparing machines or utensils difficult to clean and when unsterilized allowing bacterial multiplication.

Infection from Human Carriers of Salmonella

This path requires special consideration in view of the undue prominence accorded to it by many doctors and particularly medical officers of health. An ascertained human salmonella carrier amongst the food preparation staff is often accepted as the proved source of infection without any real evidence. Salmonellae are excreted by man under three conditions : (1) during an actual attack of food-poisoning, (2) persistence of the bacilli after recovery either for a limited time (temporary carriers) or for a prolonged period (chronic carriers), or (3) from a symptomless but infected person.

After an attack of salmonella food-poisoning the bacilli can readily be isolated from the faeces, but they diminish in a fairly orderly sequence for some weeks, after which they usually cannot be isolated apart from a minority which may persist longer or even become chronic carriers. A few examples illustrate this sequence.

Kwantes (1952), reporting on a *Salm. typhi-murium* outbreak in Llanelly in 1949, was able to follow up 112 cases. Those faeces-positive in each week were 112 (end of one week), 60, 34, 16, 9, 9, 3 (end of nine weeks). The 61 symptomless excreters were also followed up, and their rate of elimination was roughly comparable to that of actual cases, with two cases (5.5%) still excreting at the end of seven weeks. Lennox *et al.* (1954), investigating a milk-spread *Salm. typhi-murium* outbreak, found a slow rate of clearance from the faeces for three weeks and then a rapid and fairly uniform decline each week, leaving only 2 positives out of 60 cases at the end of seven weeks, with persistence of 1 or 2 positives until 18 weeks.

Miller *et al.* (1955) investigated the clearance rate of 563 cases infected with *Salm. bovis-morbificans*. Only 14 were still excreting after four to five weeks ; these were reduced to four after 13 to 14 weeks, and several were still positive after 15 weeks.

A few infected persons undoubtedly become chronic carriers, for scattered through the literature are records of individuals found positive after one or two years. A percentage figure cannot be given for such chronic carriers, but probably it is low and below that for paratyphoid fever carriers. Investigations carried out by the Public Health Laboratory Service (personal communication) yielded the following figures :

Of 3,303 children under 15 years admitted to hospitals	7 positive
,, 2,968 mothers at antenatal clinics	11 ,,
,, 2,467 persons waiting admission to mental hospitals	5 ,,
,, 1,091 adults waiting admission to general hospitals (none gastro-intestinal cases)	1 ,,

In all, out of 9,829 persons examined 24 (0.24%) were positive for salmonellae, 14 strains being *Salm. typhi-murium* and 6 other strains being represented. Bearing on this point, Armstrong and Allison (1955) examined 200 gall-bladders removed after death and 50 cholecystectomy specimens, and only one was positive (*Salm. typhi-murium*). Salmonella strains probably may persist in the intestine for some weeks without systemic invasion, but more permanent continuance must be associated with lodgement in internal organs such as the spleen or gall-bladder. In this connexion the experiments of Ørskov *et al.* (1928), and Ørskov and Moltke (1928) on oral feeding of mice with salmonellae are of great interest and support this view.

Consumption of left-over foods, etc., by staff is common in places where food is prepared, and may result in staff members being victims in many food-poisoning outbreaks. Distinction between a salmonella carrier and an infected victim is usually difficult, but it should always be attempted. Lines of investigation are the following : (1) Is there any history of a previous gastro-intestinal attack ? (2) If the person is a real chronic carrier there is a possibility of a history of food-poisoning outbreaks at previous places of employment. (3) Critical consideration of the connexions of the alleged food-handlers with the infected food and with opportunities of infecting it. (4) Detailed studies on agglutination titres, both H and O, and particularly their fluctuations.

The fourth line of investigation is of considerable value for typhoid and paratyphoid infections, but seems to be of limited utility for the food-poisoning salmonella cases. Not much recent work appears to have been done on this point, but available facts suggest irregular findings for both H and O agglutinins. Miller *et al.* (1955) studied this factor, but owing to shortage of time no agglutination observations were made until after two months, and then rather less than 70% of actual cases showed evidence of antibody production.

In their studies of experimental salmonellosis produced by feeding different salmonellae to human persons McCullough and Eisele (1951) also studied agglutination development reached by those who developed illness of food-poisoning type ; their findings are shown in Table VI. Of their 47 cases, only 16 (34.5%) showed any significant rise in titre. Apparently separate O and H reactions were not used. Also a number of the infections were quite mild. These estimations are important enough for them to be regularly made, and more detailed study may show that they are more helpful than the above figures suggest.

TABLE VI

Strain	No. of Cases	No Titre Change	Non-significant Rise	Moderate Increase	Considerable Increase
Salm. meleagridis	16	6	5	2	3
,, *anatum* ..	16	8	2	3	3
,, *newport*	5	3	1	—	1
,, *derby* ..	3	1	—	2	—
,, *bareilly* ..	7	4	1	2	—
	47	22	9	9	7

Measures to Reduce the Incidence of Food-poisoning

We appear to be amassing a great deal of highly useful information which pin-points the increase of food-poisoning, throws much light upon the organisms responsible, the reservoirs of infection, and the vehicles infected, but is unaccompanied by adequate measures of prevention. A good deal is being done by health education, by inspection of food-producing and catering premises under inadequate powers of control, and from technical improvements in appliances used in food kitchens and eating-places. All this is valuable, but the plain fact is that food-poisoning continues to increase, and there is evidence of an expanding distribution of salmonella infection in both man and animals.

(1) Detailed Investigation of all Outbreaks to Ascertain More Accurately the Extent of the Problem

Table II shows that for 1953 and 1954 the outbreaks for which no cause was found were respectively 2,000 and 2,322—that is, about 39% of the total reported. This unsatisfactory position is commented upon in the *Monthly Bulletin* of December, 1955, and the authors are probably correct in their supposition that " most of the outbreaks for which no cause was found were small outbreaks reported so late to the medical officer of health that no investigation was worth carrying out."

Another feature, brought out in Table I, is the enormous number of sporadic cases which remain unconnected with other cases, in large part probably from faulty co-operation on their detailed investigation. For these two failures medical practitioners and medical officers of health must both share part of the responsibility. Food-poisoning is at present notifiable by medical practitioners under the Food and Drugs Act, 1938, due to be repealed when the Food and Drugs Act, 1955, comes into operation. Food-poisoning is difficult to define in legal enactments, but the change-over will give the Minister of Health an opportunity of revision in the new memorandum which will be necessary. One suggestion is the introduction of "acute" before food-poisoning (the enteric infections are notifiable under another section) and with an implication on promptitude. Stress should be laid upon the *urgency* to communicate at once with the medical officer of health, which is hardly sufficiently expressed by the "forthwith" in the Act.

The investigation of a food-poisoning outbreak requires experience, and, although inquiries by the practitioner concerned can be most helpful, wider exploration is indicated, not only within the family, but into such matters as possible infection from food eaten in a catering establishment or intimate association with animals. The medical officer of health should regard these isolated sporadic cases as probably linked to a more extensive outbreak only to be discovered by *personal* inquiries on his part, linked with the full co-operation of the Public Health Laboratory Service. From wide experience I do not consider that routine inquiries by a sanitary inspector alone are adequate, and direct medical oversight is needed.

(2) More Complete Ascertainment of the Animal Reservoir of Salmonellae

We know much about salmonellosis in animals, but are still very incompletely informed about their quantitative distribution. To extend our knowledge of reservoirs of salmonella strains we need more extensive surveys of animals that may possibly be associated with food-poisoning. A good illustration is *Salm. dublin* infections in cattle. As set out above, we know of a number of scattered pockets of infection, but we do not know its extent in cattle generally or if these infections are on the increase. Recent evidence that salmonella-infected animal foods may spread the disease to healthy animals is a new light on a possible method of spread. Combined veterinary and bacteriological studies could contribute much to our knowledge of salmonella reservoirs.

(3) Reduction of the Risk of Food Infection from Human Carriers of Salmonella

Chronic human carriers are rare, but with the present large number of outbreaks annually there must be at any one time a considerable number of people in the community who are for limited periods temporary salmonella carriers. There is little risk of direct infection to other persons, but a real risk that they may infect foods which foster rapid bacterial multiplication and so become vehicles to spread food-poisoning. These risks occur in places where food is manipulated and where food is prepared for consumption, such as in catering kitchens.

The use of administrative means to ascertain carriers of food infections in catering or food-preparation places is admittedly a difficult problem. It is tackled in Section II (1) of the Food Hygiene Regulations, 1955; but this takes us only a little way towards ascertainment and is far inferior in potential effectiveness to the corresponding recommendations in the standard code of the Catering Trade Working Party.

(4) Improvement of Standards of Hygiene in Commercial Feeding-places

The Food Hygiene Regulations, 1955, go a long way towards this aim and are a decided improvement on previous enactments. At the same time they have a number of serious defects which limit their effectiveness in controlling food-poisoning. The whole regulations cannot be discussed, but three adverse features may be mentioned. They are charged with indefinite phrases such as "reasonably practical," "sufficient," "suitable and sufficient," "adequate," etc., which cannot fail to be interpreted differently by individual local authorities, so that anything like uniformity of administration is most unlikely. Many local authorities will fear to press for necessary requirements lest, if tested in the courts, these vague phrases may be interpreted by the court against them. The risks of specific infection from inadequately cleaned utensils cannot be ignored, but this seems to have been done in the Regulations. These do not enforce either effective cleaning or sterilization, for "suitable and sufficient sinks" is not enough, and sterilization is not ensured by "an adequate supply *either* of hot and cold water *or* hot water at a suitably controlled temperature." The definite risks of carrying infection from day to day from objects so difficult to clean as sausage machines or egg mixers are not covered. Particularly unsatisfactory is the absence of any requirement for the registration of catering establishments of all kinds with the local authority, as recommended by the Catering Trade Working Party. Experience in connexion with another business (ice-cream making) has shown registration to be a most valuable instrument for the hygienic control of such establishments. There is not even a suggestion that every medical officer of health should keep a complete register of all catering premises in his district, and deal in his annual report with the actions taken and results achieved.

Within the last few years many practical surveys of catering establishments have been published, and invariably they disclose numerous hygienic defects, many of which facilitate the transference of food-poisoning organisms to food on the premises. Many of these defects cannot be put right under the new regulations except voluntarily.

(5) Hygienic Control of Bulk Food Preparation

It is essential to ensure hygienic control in premises used for the bulk preparation of meat foods and those distributed in package form. Risks of infection from such foods and appropriate measures of control have been considered in detail by the Manufacturers Meat Products Working Party. They made a number of valuable recommendations, some of which are incorporated in the new Food Hygiene Regulations. Among the Working Party recommendations was the registration of such premises. This is incorporated in the new Food and Drugs Act, 1955, Section 16, requiring the registration with the local authority of premises for "the preparation or manufacture of potted, pressed, pickled, or preserved food intended for sale." Registration will be very useful, but time is necessary to see to what extent the regulations will be tightened to ensure adequate hygienic premises and practices. It would be helpful to work out better bacteriological standards for these premises to assist in this control.

(6) Steps to Reduce Risk of Salmonella Infections from Imported Egg Products

The danger does not arise from unopened shell eggs (except from duck eggs from infected farms) but from pooled mixtures of shell eggs involving the distribution through the mass of any salmonellae present and usually also some multiplication of the bacilli. An important feature is the unknown (to health authorities) and insidious way these infected egg mixtures are distributed all over the country and used by bakers and confectioners in cakes and confectionery products which subsequently do not receive effective heat treatment. A considerable list of salmonella outbreaks so caused could be compiled. This risk is apt to be extended in duration because of the considerable difficulty in effectively cleansing egg-mixing machines in the absence of any regulations requiring their sterilization. The spread of salmonella infection may also occur indirectly,

for dried-egg mixtures that are unfit are sometimes distributed to manufacturers of animal foods. The M.R.C. (1947) report on spray-dried egg includes an examination in 1944–5 of the mesenteric lymph nodes of 5,285 pigs from a number of abattoirs. As stated earlier, from 133 of these nodes salmonella strains were isolated—an incidence of 2.5%. At least 10 of the different types isolated were exogenous types not previously found in this country, and comprised more than half of all the salmonellae isolated. These mixed-egg products are a valuable addition to our food supply, but this is inadequate to justify their use when infected with large numbers of living salmonella organisms. The destruction of pathogenic organisms in egg mixtures is difficult but by no means insuperable. Presumably these products require import licences, which would give power to impose reasonable conditions of safety sufficient to remove or at least largely reduce these risks.

Conclusions

1. After making full allowance for the effects of notification of food-poisoning by medical practitioners and the greater facilities for bacteriological investigation there is clear evidence of a marked increase in food-poisoning in Great Britain over the last decade or so, an increase which shows no signs of diminution but rather of still further increase. Undoubtedly, also, a material number of outbreaks remain unreported and therefore not investigated.

2. Owing to the increased laboratory facilities and the improved technical procedures now available for investigation it is difficult to compare present and past experience, but the evidence points to an extension of salmonella infections in animals. It is not merely that we know more about them ; they appear to be more extensive.

3. The increases mentioned in the above two paragraphs have in recent years led to a rise in the number of persons who, either as symptomless cases or transitory carriers, are excreting salmonella strains in faeces.

4. It is suggested that the above three conclusions may be associated with evolutionary changes in the make-up of the salmonella group.

5. Five factors seem to be operating in the increase in food-poisoning. (*a*) Changing food habits, taking the form of a considerable increase in the number of meals eaten outside the home in professional catering-places, where defects in hygienic control may affect large numbers of customers. (*b*) The extension of bulk preparation of packaged or other foods under factory conditions which inherently increase the risk of contamination with salmonellae. (*c*) The extensive and extending use of bulked eggs (consigned to this country frozen or in other forms) in confectionery, etc. A large number of hitherto unknown salmonella types have been introduced in this way into this country. The association of liquid egg foods with food-poisoning in Great Britain is clear-cut. (*d*) The extension of the reservoirs of salmonella in animals indicated in paragraph 2. (*e*) The increase in human carriers of salmonella organisms (mentioned under paragraph 3) supplies opportunities for specific infection of foods, particularly under the conditions set out in *a* and *b*.

6. The number of single-case salmonella incidents yearly is very large and in a material proportion is likely to be associated with other cases at present unrecognized. It would be helpful if official steps were made to define more precisely the cases to be notified as food-poisoning and to point out that *prompt* notification is essential as an aid for investigation. These should be supplemented by efforts by medical officers of health to enlist the help of family doctors in giving prompt notification and co-operating in their elucidation.

7. The problem of the greatly increased and yearly increasing toll of food-poisoning may be summed up by pointing out that public health action has not kept step with the modern revolution in food habits. More and more food preparation is escaping from the domestic kitchen into the hands of the bulk commercial manufacturer, whose practices often fall below essential hygienic standards. Prepared foods such as meat pies are passing from production by the few to production on an extensive scale, with an absolute need of much higher sanitary standards of safety, which investigation of food-poisoning outbreaks shows are often not reached. The very machinery necessary in large-scale production carries and spreads the risks because of the difficulty of effective cleaning and sterilization. Methods comparatively harmless in the domestic kitchen are fraught with risk when practised in the communal kitchen. All bulk-feeding habits add to the risk, since the few infected persons may contaminate inefficiently cleaned food utensils and so spread infection.

We smile at Mrs. Beeton's direction to "take 12 eggs," but the point to us should be that they are single eggs and (except for some duck eggs) free from risk, but the mixed dry or frozen egg mixtures of to-day are far more liable not only to bacterial infection in the mixing stages but to distribution of the bacteria through the mass, probably with multiplication before usage.

These revolutionary changes in habits, with their resultant changes, are not adequately guarded against even in the latest control orders. A pathetic faith in the value of voluntary education is no adequate substitute for control based on education and exhortation with adequate reserve powers of enforcement should the will be lacking to attain to proper hygienic standards.

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