REVIEW ARTICLE

Eggs and salmonella food-poisoning: an evaluation

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Summary. Evidence on the extent of the part played by infected hens' eggs in causing salmonella food-poisoning is inconclusive. The role of freshly cooked shell eggs is currently much exaggerated. Prevention should be sought through improved catering practices and kitchen hygiene, and attempts to eradicate salmonellas from laying flocks are likely to be ineffective.

Introduction

From early this century it has been known that along with many other sources of salmonella, eggs are sometimes infected and sometimes cause food-poisoning in man.[1,2] Ducks' eggs were mainly implicated and hens' eggs only rarely, their role being thought smaller than that of meat, pork, poultry meat and unpasteurised milk.

During and after the Second World War, outbreaks were traced to imported spray-dried and bulk liquid egg, in which salmonellas from a few contaminated eggs had multiplied and disseminated during preparation and storage. Pasteurisation required by the Liquid Egg (Pasteurization) Regulations 1963 has made such products relatively safe.[3]

Most of the recent egg-associated outbreaks have been due to products such as mayonnaise, ice-cream and cold desserts, in which salmonella can multiply profusely and which are eaten without cooking after the addition of raw egg. In these nutritious products a few bacteria from an egg's shell or contents can multiply to huge numbers during storage for 1 or 2 days at warm room temperature. With a generation time of 80 min at 20°C, one bacterium can become a billion (10^9) in 40 h, and with a generation time of 40 min at 25°C, it can do so in 20 h.

In contrast, shell eggs boiled, fried, poached or scrambled in the home have until recently been thought to cause infection only rarely, despite the knowledge that light cooking which leaves some yolk or albumen liquid does not kill all contained bacteria, e.g., in duck eggs boiled for less than 8 min.[2]

Reasons for the relative safety of freshly cooked hens' eggs are seen in recent findings that very few eggs from infected flocks contain salmonellas and that the number of bacteria in these infected eggs is usually so low as to be below the human oral infective dose.

The general restriction of human (non-typhoid) salmonellosis to the food-poisoning mode of infection indicates that profuse multiplication of the bacteria in a foodstuff is needed to yield a dose sufficiently large to infect by mouth.

Recent concern

Evidence regarding the increase of food-poisoning in Britain in 1987 and 1988 has been interpreted by some epidemiologists as showing that hens' eggs have now become a common source of food-poisoning. It led the junior health minister, Mrs Currie, to state on 3 December 1988 that "most of the egg production of this country, sadly, is now infected with salmonella", which wrongly phrased pronouncement exaggerated the danger from eggs and aroused much public anxiety and political concern.

The Agriculture Committee of the House of Commons then examined evidence on the role of eggs presented by officers of the Department of Health (DOH), Ministry of Agriculture, Fisheries and Food (MAFF), Public Health Laboratory Service (PHLS) and other bodies, and published a First Report in February 1989.[4,5] Whilst admitting the evidence that eggs had become the commonest source of salmonella outbreaks was inconclusive, the Committee concluded that the Government should not wait for cast-iron evidence before taking action and that it was right to make egg production the first focus of its attention (Report, p. xii.)[4]

In this situation of doubt, the Chief Medical Officer of the DOH justifiably advised the public that foods containing raw egg should be avoided, that for the very young, very old, pregnant and sick, eggs should be thoroughly cooked (yolk hard), but for other persons (the great majority) there was very little risk from eating eggs cooked however preferred.

But there has been controversy over the Govern-

ment's hasty introduction of a policy for compulsory bacteriological testing of laying flocks (on 16 March 1989 under the Testing of Poultry Flocks Order 1989) and the slaughter of flocks found to contain any infected birds (Zoonoses Order 1989, effective from 1 March 1989).

This policy is out of line with practice in most other developed countries and is damaging to the British egg industry, particularly the small producers. It was imposed without sufficient knowledge of the epizootiology of salmonellosis in laying flocks to give reason for believing it would be practicable and effective.

The Ministry, moreover, implemented the slaughter policy almost immediately after it had advised the Agriculture Committee in a joint MAFF/DOH memorandum on 11 January 1989 (Report, p. 3[5]) that: "Salmonella organisms are present in many areas of the environment and are persistent. There is no technically sound method which is certain to eliminate them from laying houses. Even after very thorough cleansing and disinfection, salmonella may recur. Because of this, a policy of slaughtering flocks with the aim of eradicating salmonella would not have a realistic prospect of success. There would be a serious risk that even after an infected flock had been slaughtered out and the premises disinfected, subsequent flocks in the same building could become infected from the environment."

Salmonella enteritidis PT4

The basis of the belief that hens' eggs have become a major source of salmonella food-poisoning in Britain was the emergence since 1985 of one particular strain, Salmonella enteritidis phage type (PT) 4 as the cause of a large proportion of human cases. In PHLS evidence to the Agriculture Committee it was reported that in the first 10 months of 1988 in England and Wales, 36% of 384 recorded outbreaks of salmonella food-poisoning (Report, p. 17[5]) and 46% of 23 038 bacteriologically proven cases (Report, p. 6[5]) were due to the PT4 strain.

This strain has been found in flocks of laying hens and broiler (poultry meat) chickens and it has been assumed that these poultry are its major source for man. Of 108 isolates of PT4 from food animals examined by the PHLS in 1987, 81% were from poultry, the others from turkeys, ducks, cattle, pigs and sheep (Report, p. 8[5]). MAFF reports showed that overt infections with S. enteritidis were uncommon in cattle and pigs, but became common in chickens, mainly broilers, in 1987 and 1988 (Report, p. 21[5]).

The assumption that poultry are the main source of the PT4 strain may well be correct, but it is a weakness of the case against eggs and poultry meat that no thorough investigation has been made of the strain's occurrence in symptomless intestinal carriers in different food animals, pets, vermin, wild birds and kitchen staff, for it is such inapparent, "carrier"

infections that are most likely to be sources of food-poisoning.

Whilst herds and flocks may contain only a few (e.g., 1%) of symptomless carriers, cross-contamination with their intestinal contents during slaughter and processing may infect the carcases of many originally uninfected animals. Such cross-contamination presumably explains why in PHLS surveys in 1987 about 60% of retailed chicken carcases contained salmonella (Report, p. 17[5]).

Eggs versus poultry meat

If poultry are now the major source of salmonella food-poisoning, the relative importance of eggs and poultry meat remains in question and is relevant to the Government's decision to impose a slaughter policy on laying, but not broiler flocks.

Explaining this decision on 24 October 1989, the Parliamentary Secretary to the MAFF stated that the Government had targeted eggs as the main health risk because they are eaten raw and part-cooked, while it is generally accepted that thorough cooking kills salmonella in chicken. This confidence in an understanding of the need for thorough cooking of chicken seems misplaced in view of the many outbreaks of food-poisoning traced to its consumption. It also ignores the great potential for cross-contamination from raw carcases.

Until 1988 poultry meat was considered the major source of salmonella food-poisoning in Britain[6, 7] and the evidence that it has now been overtaken by eggs is inconclusive. In PHLS evidence to the Agriculture Committee (Report, pp. 17, 18[5]) it was noted that in only a minority (under 20%) of outbreaks was a food vehicle presumptively identified. Among these, in 1987 only six outbreaks were associated with egg-containing foods but 41 with poultry meat. In the first 10 months of 1988, by contrast, 46 outbreaks were associated with eggs and 28 with poultry meat.

The PHLS later cited the figures for 1988 and 1989 shown in the table.[8] They indicate an increase in the proportion of outbreaks associated with eggs in these years. This increase may partly reflect the effect of the new interest in eggs stimulating a more frequent

Table. Number of recorded outbreaks of salmonella food-poisoning in England and Wales and numbers of outbreaks associated with eggs and poultry meat

Year	Egg-associated outbreaks	Poultry meat-associated outbreaks	All outbreaks
1987	6	41	421
1988	34	27	455
1989	42	24	955
Total	82	92	1831

reporting of outbreaks in which an egg source could be suspected. There are other reasons, moreover, why the PHLS figures probably overestimate the role of eggs and underestimate that of poultry meat.

Egg-containing foods

It should not be assumed that in most or all outbreaks traced to an egg-containing food the food received its salmonella from an egg. In some or many such outbreaks the food may have received the salmonella by cross-contamination via hands or utensils from another source in the kitchen, e.g., an uncooked infected poultry carcase, human carrier, food ingredient contaminated by vermin, or vegetables contaminated by wild birds.

In some well investigated outbreaks, the role of eggs has been strongly indicated by a failure to detect any other source and a finding of the epidemic strain in the flock of hens known to have supplied the eggs. But in most supposedly egg-associated outbreaks the published information has been fragmentary and the evidence incriminating eggs as the source has been incomplete or absent.

In evidence to the Agriculture Committee (Report, pp. 256–290[5]) one of us (R.A.E.N.) analysed the findings in 13 outbreaks reported as egg-associated for which adequate information could be obtained and found that in only two of them was the evidence sufficient to demonstrate that eggs might have been the primary source.

In a MAFF/DOH memorandum to the Agriculture Committee (Report, p. 15[9]), 30 outbreaks attributed to eggs were reported for the first three-quarters of 1989, but in only 10 were the reasons implicating eggs reported to the PHLS Communicable Disease Surveillance Centre (CDSC): in three a statistical association and in four the finding of salmonella in the supplying flock.

When evidence of the association is so often incomplete or absent, conclusions drawn from the number of outbreaks reported as egg-associated must be viewed with suspicion.

Poultry meat and cross-contamination

The role of poultry meat is not confined to the many outbreaks traced to its consumption,[5–7] for infected carcases also act as potent sources of cross-contamination to other foods, which then may cause outbreaks.

Chicken carcases are almost universally present in the kitchens of hotels, restaurants, hospitals and other catering establishments and it is known that many carcases contain salmonella, e.g., 60% of retailed chickens (Report, p. 17[5]). The number of bacteria on a carcase may be very large. From work in Huddersfield Polytechnic it has been reported that the drip from a frozen carcase during defrosting may contain billions of bacteria (J. Mottishaw, personal communication). Traces of contaminated fluid on hands and utensils may seed a variety of foods in the kitchen and even introduce as many bacteria as may, without further multiplication, constitute an infective dose.

The role of cross-contamination in causing a given outbreak is difficult to detect and in most instances is likely to be unrecognised and unrecorded. That it is a significant and not merely hypothetical danger has been shown in some thoroughly investigated cases. Thus, food-poisoning with S. enteritidis PT8 affected 14 guests at a wedding lunch who ate cold roast pork, but none of 45 guests who ate hot roast turkey.[10] Pigs were not the source of the salmonella. The cold cooked pork had been contaminated in the kitchen from uncooked turkey carcases which were later found to contain the PT8 strain and to have been obtained from a flock infected with that strain.

A major factor facilitating cross-contamination is the design of commerical kitchens. Traditionally such kitchens were designed for what was called the "partie" system, with different kinds of foods prepared by chefs working in separate areas, from which the product was supplied on a raw-in, cooked-out basis. Over the years the "parties" have contracted and work with both raw and cooked meats and other foods is conducted in a "general partie", where cross-contamination is almost inevitable.

Food-poisoning is commonly attributed to the failure of food handlers to wash their hands between contacts with raw and cooked foods.[11] Our experience of food premises is that too often wash basins are soiled, obstructed or lacking in soap and drying facilities, and where adequate are frequently unused. During a food hygiene conference in Swindon in 1988 a survey in the washroom revealed that 65% of participants using the facilities did not wash their hands.[12]

Sporadic cases

Only a minority, probably less than 10%, of cases of salmonella food-poisoning occur in recognised outbreaks affecting several persons who have shared a common meal. The majority are sporadic, in single patients not known to have shared a foodstuff with other sufferers. The true number of sporadic cases is unknown, for most are unreported (Report, p. 249[5]). Whereas there were 23 038 bacteriologically proven cases of salmonella food-poisoning in England and Wales in the first 10 months of 1988 (Report, p. x[4]), the total number of cases may have been 10 times as many.

From evidence suggesting that 46 of 384 outbreaks of salmonella food-poisoning in 1988 (38 of 191 S. enteritidis outbreaks) were egg-associated (Report, p. 17[5]) it has been suggested that many or most sporadic cases are caused by eating infected shell eggs, boiled, fried, poached or scrambled, but cooked too lightly to kill salmonella. As few eggs are infected and rarely

more than one person eats from the same egg, most cases so caused would be sporadic.

It is unjustifiable thus to extrapolate to sporadic cases from uncertain conclusions about outbreaks caused by egg-containing foods. In these foods a few bacteria initially present often have the opportunity, before consumption, to multiply profusely to yield a large infective dose. But when whole eggs are eaten shortly after breaking from the shell, there is no opportunity for such multiplication.

The degree of risk in eating shell eggs depends on the proportion of retailed eggs containing salmonella, the number of bacteria usually present in the infected eggs, the extent of bacterial survival on cooking, and the number of bacteria that have to be swallowed in order to cause illness, i.e., the size of the oral infective dose.

Frequency of infection in eggs

Salmonella is found on the shell of eggs, from soiling with hen faeces, more often than in the contents. Some bacteria from the shell may enter egg-containing foods and there multiply to infective levels, but it is only the bacteria present in the egg's contents that can pose a danger to persons eating freshly cooked shell eggs.

Widely differing frequencies of internal infection in eggs have been reported. In small, recently infected flocks the proportion of eggs containing salmonella may be large, e.g., 5 out of 10 eggs from a small domestic flock,[13] 10 of 613 eggs from an artificially infected flock of 42 hens,[14] and 11 of 1119 eggs from two naturally infected flocks of 23 and 15 birds; each infected egg contained less than 10 S. enteritidis PT4 bacteria.[15]

In large flocks associated with food-poisoning the proportion of internally infected eggs has been smaller, about 0·1% or less: e.g., S. enteritidis PT4 in 4 of 2000 eggs (Report, p. 20[5]), 0 of 950[16] and 5 of 17 000,[17] and S. typhimurium in 3 of 1137 eggs[18] and 0 of 1000.[19]

As most laying flocks are uninfected, the overall frequency of infection in British eggs is likely to be much lower than 1 in 1000. A MAFF study in 1989 showed a contamination rate of about 1 in 15 000 eggs (cited by Stevens et al.) and even lower rates have been estimated.

Britons eat on average just under three eggs a week, or about 150 a year (Report, p. 11[5]). If 1 in 15 000 eggs is infected, the chance of a person eating an infected egg is very low, only 1 in 100 per year. In a population of nearly 60 million, about 600 000 persons would eat an infected egg each year, but in most such cases too few bacteria would be present in the egg to constitute an infective dose.

Number of bacteria in egg contents

Although a matter of crucial importance, few studies have been made of the number of salmonella bacteria in the contents of intact, naturally infected eggs. Those reported suggest that the number per egg is usually very small, in the order of 1–100. Each of 11 infected eggs from two flocks contained fewer than 10 S. enteritidis PT4 bacteria, though some of these eggs had been stored for 5 days at 20°C before examination.[15] After inoculation of S. pullorum into the ovary, the number of bacteria recovered from the eggs was only from 0·9 to 4·6/g.[20]

The probable reason for the presence of only small numbers of bacteria is the protective effect of the egg's complex system of membrane barriers and antibacterial components in the albumen.[21,22] These defences are overcome when eggs are subjected to artificial inoculation or broken with mixing of yolk and albumen, and they deteriorate on prolonged storage under warm conditions.

The contents of the egg may become infected in two ways: (a) through the shell, where it is laid on damp earth or litter soiled with infected droppings, and (b) transovarially, into the forming yolk or albumen from the ovary or oviduct of a systemically infected hen.

Infection through the shell

This mode of infection has been studied experimentally. In one study[23] eggs at 35°C were immersed in a cold suspension of S. montevideo, S. oranienburg or S. typhimurium bacteria and sampled after drying and storage at 29°C for periods of up to 29 days. Salmonella was absent from the yolk and albumen immediately after inoculation, penetrated in small numbers into some eggs during the first week, but penetrated into and multiplied to large numbers (10^7–10^9/ml) in most eggs during the third and fourth weeks. The egg's defences were therefore highly effective in the first fortnight of storage at 29°C, but later broke down.

The relevance of such findings to the risk from trans-shell infection under natural conditions is unclear. Eggs laid on damp, faeces-soiled earth will draw in not only salmonella, but also numerous putrefactive faecal bacteria such as pseudomonas and gram-negative anaerobes. Some of these saprophytes will multiply faster than salmonella at ambient temperatures and probably make the egg inedible before its salmonella content is large.

Transovarian infection

During the egg's formation, before it is covered by the shell, the yolk or albumen may become infected from the ovary or oviduct in a recently infected, septicaemic hen.[1,14,24] The egg's contents are thus infected with salmonella without an associated, warning infection with spoilage bacteria, and the yolk may be infected before it is protected by the anti-bacterial albumen.

The yolk may have some yet unrecognised defences, such as its relatively anaerobic state and large content of antibodies,[25] which may explain why only small

numbers of bacteria have been found in the yolks of eggs naturally infected by the transovarian route.[15] Possibly, too, it is nutritionally deficient for the non-proteolytic, non-lipolytic salmonellae which require free amino acids, of which there are no known quantities in the undisrupted yolk.[26]

Some workers have shown rapid multiplication of salmonella after artificial inoculation into the yolk. In one study[27] an inoculum of about 10 cultured bacteria multiplied to about 10^9 in 24 h at 23°C and 10^{11} in 48 h. But such inoculation introduces growth medium components and causes some mixing of yolk with albumen, changes that appear to disrupt the egg's defences. These experimental findings cannot be taken to represent what commonly happens in naturally infected, intact eggs, otherwise huge, instead of only small numbers of bacteria would be found in such eggs.

Although profuse multiplication does not take place in the generality of transovarially infected eggs, it possibly does so in some abnormally formed or mistreated ones. Such exceptional multiplication may explain those rare family outbreaks caused by a dish prepared from shell eggs only a few hours before consumption.[28] In some cases, however, an apparent absence of sufficient time for bacterial multiplication after the breaking-out of eggs may be due to wrongly remembered or reported circumstances.

Survival on cooking

Whilst salmonella bacteria in free egg fluid are killed within a few minutes when heated at a temperature as low as 60°C, light cooking does not reliably rid them from whole eggs because heat penetrates only slowly through the static mass of viscous contents.

Boiling an egg at 100°C will kill salmonella on the shell, but many studies have shown that the cooking of boiled or fried eggs to yield a firm white but partly soft yolk does not kill all bacteria in the yolk. In tests on eggs into the yolks of which S. enteritidis had been injected, viable bacteria were recovered from those boiled for only 4 min or fried on only one side (yolk soft), though not from those boiled for over 8 min or fried on both sides (yolk hard).[27] Cooking must be continued until both white and yolk are completely coagulated to ensure freedom from viable bacteria.

Size of oral infective dose

The food-poisoning (non-typhoid) types of salmonella such as S. enteritidis and S. typhimurium cause both overt and symptomless intestinal infections in a wide range of domestic and wild animals and birds. They must commonly contaminate the environment, including earth, water and vegetables, and frequently enter the human mouth in small numbers on contaminated hands, utensils, uncooked vegetables, and drinking and bathing water. Yet clinical infection in man is largely confined to the food-poisoning mode.

This limitation is generally, and probably correctly, attributed to an inability of small numbers (e.g., 1–1000) of swallowed bacteria to overcome the defences of the alimentary canal and a need for preliminary multiplication in a nutritious foodstuff to yield a large enough oral dose (e.g., over 10^5 bacteria) to overcome these defences.

The defences include the bactericidal action of the usually strongly acid (pH < 3·0) gastric secretion,[29] rapid removal by peristalsis in the small intestine[30] and the antagonistic action of the commensal flora in the large intestine.[31]

If a small oral dose of salmonella could commonly infect man, the epidemiology of the infections should resemble that of typhoid fever and bacillary dysentery, which typically spread from man to man on vehicles such as hands, towels, utensils and dilutely contaminated drinking or bathing water, likely to deliver only very small numbers of bacteria into the mouth. Thus in water-borne outbreaks of typhoid fever with low attack rates and long incubations the oral infective dose has been estimated as less than 1000 bacteria.[32, 33] A possible reason why S. typhi can commonly infect from such small doses may be that it has a specific ability to colonise the human throat and thence seed large numbers of bacteria into the stomach and intestine.

Because they have so many animal sources, the food-poisoning salmonellas must be present in water much more often than S. typhi, and if they could infect from small oral doses they should commonly cause waterborne epidemics. But such epidemics are rare. In an exceptional summer outbreak of S. typhimurium infection caused by the unchlorinated water supply of Riverside, California, the estimated number of bacteria ingested was only 17, but the estimate was based on examination of samples taken 6 days after the peak of the outbreak.[32, 34] As water is used for bathing and showering as well as drinking, small numbers of bacteria may have infected via the specially susceptible conjunctival and respiratory routes, not via the mouth.

Support for the view that oral infection normally requires the ingestion of large numbers of the salmonella bacteria is afforded by the results of experimental infections. McCullough and Eisele[35, 36] fed different-sized doses of S. anatum, S. bareilly, S. derby, S. meleagridis and S. newport in egg suspension to adult volunteers. Doses in the range of 10^5–10^8 bacteria were required to cause gastroenteritis, the attack rates being 10–60%.

Many recipients of small doses (e.g., 10^4 bacteria) became symptomless intestinal carriers, showing that when a few bacteria escape being killed by the acid in the stomach, the defences of the intestine can restrain their proliferation to a subclinical level.

Small-dose infection

Though large doses of salmonella are generally required for infection, there is evidence that in exceptional circumstances a small dose (1–1000 bacteria) may suffice. Thus in rare outbreaks caused by chocolate and cheese, only small numbers of bacteria could be recovered from the causal foodstuff.[32]

Only 2·5 S. eastbourne bacteria/g were found in chocolate balls infecting 119 persons,[37] and the infective dose was estimated to be about 50 S. napoli bacteria in chocolate bars infecting 245 persons,[38] and 1–6 S. typhimurium bacteria in cheddar cheese infecting 1500 persons.[39]

It has been suggested that dehydrated bacteria entrained in the lipids of fatty foodstuffs may be protected from killing by the gastric acid, though why a few such protected bacteria should be able to overcome the intestinal defences is unclear. Possibly components of chocolate and cheese help the bacteria colonise the throat before being swallowed. But whatever the explanation, the findings of McCullough and Eisele[35, 36] from the feeding of salmonellas in egg nog show that the lipids of egg do not have the same enhancing effect as those of chocolate and cheese.

Hospital infections

Evidence for small dose infection with food-poisoning salmonellas has been seen in certain hospital outbreaks among neonates, infants, debilitated adults and old persons not caused by infected food.[40, 41] The infection appeared to be spread from patient to patient and from nurse to patient by vehicles such as hands, clothing, towels, wash basins and ward dust, unlikely to deliver more than a few bacteria into the mouth. In a general hospital where 102 persons developed gastroenteritis and 150 became symptomless carriers, the causal strain of S. typhimurium was found in ward dust, on sheets used by excretors and in the sputum of five patients.[41]

It has been suggested that defective gastric and intestinal defences in the young, debilitated and old make such subjects specially vulnerable to small-dose infection. In neonates, for instance, gastric acidity is low in the first weeks of life[42] and the protective intestinal flora takes a few days to become properly established.

Though some small-dose infections may be due to defective defences, others may be caused by the entry of salmonella through specially susceptible non-oral routes such as the conjunctiva and respiratory tract. The bacteria may first multiply profusely in these sites before seeding in large numbers into the hostile environment of the stomach and intestine. There are many opportunities in hospital for bacteria to enter the eye or nasopharynx, as when a nurse's contaminated hand washes a baby's face, when adult patients wash their faces in contaminated basins or when patients inhale contaminated ward dust.

The great susceptibility of the conjunctival route was demonstrated in guinea-pigs by Moore;[43] infections resulted from the dropping of 100 S. enteritidis bacteria into the eye, but not from the feeding of 10^8 bacteria. And the conjunctival route has been found to be a 100-fold more susceptible than the oral route for infection of mice with S. typhimurium.[44] Comparable observations in man are lacking, but a volunteer into whose nasal antrum 25 salmonella bacteria were inoculated, developed acute sinusitis in 4 h and gastroenteritis in 37 h, showing the promoting effect of initial multiplication in the upper respiratory tract.[45]

Case-control studies of sporadic infections

The bacteriological evidence of the small proportion of eggs infected and the small numbers of bacteria present suggests that not many sporadic infections are likely to be due to the eating of undercooked shell eggs, but this conclusion needs to be confirmed by epidemiological studies. Unfortunately, only a few small studies of sporadic infections have been reported,[46, 47] insufficient for firm conclusions.

In the larger study,[47] food histories were obtained from 160 patients with sporadic S. enteritidis PT4 infections and up to three uninfected neighbours (controls) per patient. The illness was found to be significantly associated with the consumption of shop-bought sandwiches containing mayonnaise (p = 0·00004) and bought pre-cooked chicken (p = 0·006), foods liable to cross-contamination during preparation and to unrefrigerated storage before consumption.

But there was only a weak association (p = 0·02) with the eating of lightly cooked fried, poached or scrambled eggs, or omelette, and no significant association with the eating of soft-boiled eggs. It is difficult, therefore, to understand how the authors of the study concluded that "fresh shell eggs ... are vehicles of S. enteritidis phage type 4 infection in indigenous sporadic cases". Larger studies are required, but to our minds the present results suggest there is little danger from eating freshly cooked shell eggs.

Preventive measures

As salmonella and other food-poisoning bacteria have common, almost ubiquitous sources in man, animals and the environment, their absence from foods as supplied from the farm cannot be ensured by Government action. The correct sphere for governmental control is in the regulation of practices in commercial food-processing and catering to minimise opportunities for cross-contamination and bacterial multiplication.

This view accords with advice in the Code of Practice for the Control of Salmonellae in Commercial

Laying Flocks issued in 1988 by the MAFF and British Poultry Federation,[48] which stated: "Salmonella organisms are widespread and their complete elimination from the environment can never be expected. There is evidence that an increase in food poisoning has been associated with use of raw foodstuffs of animal origin in the preparation of food for human consumption. This problem could be largely overcome by applying good kitchen hygiene and practices...".

Special attention should be given to the preparation and handling of those foodstuffs that have caused a large proportion of outbreaks in recent years, namely mayonnaise, cold desserts and pre-cooked savouries.

Mayonnaise

Mayonnaise was the probable or possible vehicle of infection in 14 of the 46 "egg-associated" outbreaks of salmonella food-poisoning reported to the CDSC for the first 10 months of 1988 (Report, pp. 31–34[5]). The apparently rising incidence of food-poisoning from mayonnaise made in the home or a catering establishment has been attributed to reduction in the bactericidal vinegar content to meet consumer preference for a blander product.[49] During unrefrigerated storage the new product allows the multiplication of bacteria derived either from its content of raw egg or by cross-contamination from another source in the kitchen.

Home-made mayonnaise should be acidified with vinegar to a pH between 3·6 and 4·0.[49] Commercial products should be made with pasteurised egg and, to prevent their later contamination in catering establishments, should be supplied in sealed containers not so large as to encourage re-use on a second day after opening. In a PHLS report on a mayonnaise outbreak in the House of Lords the need for guidelines on the preparation of mayonnaise in commercial premises was suggested. Such guidelines issued in 1955 in Denmark appear to have been effective, though one outbreak in a day nursery from home-made mayonnaise was reported in 1980.[50]

Avoidance of cross-contamination

Greater emphasis should be given to the prevention of cross-contamination in catering kitchens, as egg-based foods are not made safe by excluding contamination from eggs. They may be cross-contaminated from another source in the kitchen, so that such cross-contamination and the opportunity for later multiplication of bacteria must be prevented. Raw poultry carcases are probably the commonest source of contamination and the most useful preventive measure might be a requirement that catering establishments should obtain poultry only from sources, such as irradiation plants, which could guarantee their freedom from salmonella.

Slaughter policy

The present requirement for the testing of laying flocks and the slaughter of those containing any infected birds is at variance with the advice given by the MAFF to the Agriculture Committee (Report, p. 3,[5] quoted above), and will probably not significantly reduce the amount of food-poisoning.

Periodic (12-weekly) testing of flocks by culture of faeces or cloacal swabs collected by the flock keeper will not reliably detect infection either soon enough after its entry into a flock or in flocks where only a minority of birds are carriers.

Buildings cannot be completely disinfected after a flock has been slaughtered and the replacement flocks are liable to be re-infected by residual salmonellas in the building, infected feeding stuff (Report, p. xviii[4]), the droppings of mice, which are impossible to exclude from poultry houses, human carriers, or salmonellas from the droppings of wild birds carried into the poultry house on the keeper's feet. Re-infection has already been reported in the replacement flocks on some of the farms where flocks were slaughtered in 1989, and we know of five such re-infected farms, housing a total of 385 000 birds.

In the first year of implementation of the policy, from March 1989 to February 1990, over a million hens were slaughtered, but the amount of salmonella food-poisoning continued to increase. In December 1989 the Agriculture Committee took encouragement from reports that the increase in the number of human infections with *S. enteritidis* PT4 was not as great in 1989 as in 1988 (Report, p. vii[9]). It is known that particular salmonella strains spontaneously become more and then less common over periods of years, and no doubt the PT4 strain will eventually decline, but that decline should not be taken to indicate success of the slaughter policy.

A detailed account and analysis of the data, inquiries, reports and pressures leading to imposition of the policy has recently been published.[51]

It must be questioned whether the problem of salmonella in eggs deserves the great attention it has recently received. Salmonella food-poisoning is a non-contagious disease and most cases are scarcely more serious than the common cold and similar upper respiratory viral infections, which are nearly a thousand times commoner. Only a minute proportion of food-poisoning cases are fatal and the fatalities are mainly in old debilitated persons. There are many graver health problems to be faced.

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