Us and Them: Bacterial Invasion and Colonisation in the Twentieth Century

Anne Hardy

Histories of infectious disease are a staple item in the history of medicine, and tracking the passage of epidemics and pandemics has attracted both historians and epidemiologists. The global spread of bubonic plague, syphilis, cholera, yellow fever and influenza on the tides of trade, military activity, and exploration is a familiar text; their local origins are sometimes – as with syphilis – a matter of historical debate; all are well represented in the historiography. [1] These diseases are the big hitters of history, whose stories are both a public drama and a confrontational challenge to human societies. But there are many other infections, more humdrum in public manifestation, more insidious in their progression, more pervasive in effect, which have none the less brought personal suffering and even death to millions across the world. Such diseases also have a story to tell, although they rarely attract attention. They too begin from local origins and may or may not achieve a global reach, depending on circumstance. There can be little more ordinary in the annals of human experience than a bout of food poisoning; other than influenza and the common cold, it is probably the infection that most of us will suffer most frequently in our lifetimes, and which, on a day to day basis, causes more human misery, loss of productivity and even loss of life, if not than influenza, then certainly than the common cold.

[1] Amid a vast literature see, for example, Ole J. Bendictow, *The Black Death, 1346-1353: The complete history*, Woodbridge, Suffolk: Boydell and Brewer, 2004; Claude Quetel, *A history of syphilis*, translated Judith Braddock and Brian Pike, London: Polity Press, 1992; Norman Longmate, *King cholera: The biography of a disease*, London: Hamish Hamilton, 1966; Donald R. Hopkins, *The greatest killer: smallpox in history with a new introduction*, Chicago, IL: University of Chicago Press, 2003; William Coleman, *Yellow fever in the north: The methods of early epidemiology* , Madison, WI: University of Wisconsin Press, 1987; William H. McNeill, *Plagues and peoples*, Oxford: Basil Blackwell, 1977.

Not all disease conditions have histories that end in resolution, or the achievement of a degree of equilibrium between host and parasite. The history of food poisoning – or rather, of one of its principal causative organisms – is a history of anarchy. In this bacterial world, historical tropes of centre and periphery, connectivity and power take on a kaleidoscopic quality; here the centres are shadowy and shifting, the peripheries unexpected, connectivities and power in a continual process of translation.

For most of the twentieth century, bacteria belonging to the salmonella family were probably the main source of food poisoning incidents in the Western world.[2] There are some 2,500 different types of salmonella, around 150 of which are pathogenic for humans.[3] The most virulent of these is s. typhi – the causal organism of typhoid, a disease which seems to be exclusive to humans – but others can cause serious illness in certain birds and animals, and are variously carried by a great many creatures, from mice, geckoes and tortoises to domestic poultry, pigs and cattle. They are named for Daniel Salmon, the American microbiologist who is credited with isolating the first member of the wider family to be identified, s. cholerae-suis, the organism of hog-cholera, in 1884.[4] From the beginnings of bacteriology in the 1880s, the salmonella family emerged as commonly occurring bacteria with a wide geographical distribution. To begin with, in the years to 1914, this caused considerable confusion, with the same organism being identified and differently named in several places. Thus the serotype later formally known as s. typhimurium, or

---

[2] The situation is not fully clear but see Public Health Laboratory Service report, "Food poisoning in England and Wales" *Monthly Bulletin of the Ministry of Health* ,18, 1959, pp. 169-178: "It becomes clearer each year that food poisoning in England and Wales is largely a matter of salmonellosis" (p.177); Anon., "Epidemiology of salmonellosis", *Medical Officer*, 111, 1964. pp. 236-7.

[3] Michael P. Doyle, and Dean O. Cliver, "Salmonella", in Dean O Cliver ed, *Foodborne diseases* , San Diego, CA: Academic Press, 1990, p. 187; Kare Molbak, John E. Olsen, and Henrik C. Wegner, "Salmonella infections" in Hans P. Reimann and Dean O. Cliver eds, *Foodborne infections and intoxications*, Amsterdam: Elsevier, 2006, p. 57.

[4] For the complex story of this attribution, see, Claude E. Dolman and Richard J. Wolfe, *Suppressing the diseases of animals and man. Theobald Smith, microbiologist*, Boston, MA: Boston Medical Library, 2003, chapter 5.

mouse typhoid, was variously known in the 1890s as bacillus aertryke (named in Belgium), the 'Mutton' strain, and 'Breslau type'. [5] Between 1920 and 1939, however, increasing international co-operation among microbiologists led to the convention that each salmonella serotype would be named after the locality from which the first isolate was obtained.[6] Thus, for example, the family came to contain s. bareilly (India), s. montevideo (Uruguy), s. agona (Ghana), s. arizona (USA), s. kentucky (USA), s. ballerup (Denmark), s. derby (UK), and our own very local version here, s. heidelberg (Germany), among many others. Various generic names established before this convention persisted: typhi and typhimurium, s. gallinarum and s. pullorum (chickens), s. cholerae-suis (pigs), s. bovis morbificans (cattle), and s. abortus (cows, sheep and horses). The naming of most salmonellas, therefore, enables us to establish something of a geography of spread for these organisms. The local place of naming may not be the original birth-place of a particular serotype, but the global distribution of that particular organism bears witness to the extent of its travels across the world.

As this history of naming suggests, the story of the salmonella family is intimately connected to that of the development of laboratory techniques. The increasing refinement of serological methods in the interwar period permitted the differentiation, identification, and naming of a growing number of salmonella types, mostly named for place of origin. On the eve of World War II, Canadian researchers introduced the new technique of phage-typing, by which the bacteria could be further differentiated according to the phages (pathogenic bacterial viruses) to which they are susceptible. Initially applicable to typhoid (s. typhi), phage typing was soon extended to include paratyphoid B and s. typhimurium. One result of this development was the

[5] Philip Bruce White, "The salmonella group", in Medical Research Council, *A system of bacteriology in relation to medicine*, London: HMSO, 1929, vol 14, pp. 86-7.
[6] This convention still holds; see Molbak et al, 2006, p. 59.

realisation that these individual subspecies could be further broken down into multiple units according to phage-type – there are currently recorded at least 170 different salmonella typhimurium phage types. Even s. typhi itself, hitherto thought to be one distinct entity, was revealed to contain more than half a dozen phage types. The complexities of identification which this new process introduced excited epidemiologists but appalled others: the *British Medical Journal*, for example, noted despairingly that the typhoid organism, which it described as hitherto 'an oasis of simplicity in the desert of complexity' that constituted the salmonella group in general, had now acquired seven different varieties.[7] But for the epidemiologists, the technique was swiftly recognised as offering an "extremely delicate and accurate" means of studying various aspects of the epidemiology of the disease. [8]

The relevance of phage-typing for elucidating epidemiological mysteries was soon made apparent, and its delicacy and accuracy confirmed. For the historian too, the advent of phage-typing resulted in epidemiological findings that cast light on the local and global aspects of disease ecology. The seven phage-types of typhoid, for example, were instrumental in solving a wartime outbreak, which was at the time a source of considerable consternation to the military establishment and might, in other circumstances have had wider ramifications for the final stages of Britain's war effort. This outbreak occurred in the spring of 1945, in the fraught months leading up to D-Day. Planning for D-Day had been in train for four years and involved top secret administrative preparations conducted by selected and high-powered Forces personnel. Suddenly, that spring, a number of important military officers developed typhoid. With the spectres of espionage and bacteriological warfare in mind, the War

[7] Annotation, "Serological diagnosis of typhoid", *British Medical Journal* 1938, 2, pp.1211-1212, p. 1212.
[8] J S K Boyd, "Laboratory methods in the diagnosis and control of fevers of the enteric group", *BMJ* 1939, 2, pp. 902-04, p. 904; see also Epidemiological Notes, "Paratyphoid fever in Liverpool", *Monthly Bulletin of the Emergency Public Health Laboratory Service*, 1, 1941-42, p. 7.

Office contacted the Ministry of Health requesting an immediate investigation. The Ministry of Health knew that quite a number of well-to-do London residents in several of the city's boroughs were suffering from typhoid at the time – a somewhat unusual occurrence, as the investigating epidemiologist wryly noted. But they were by no means the only typhoid victims in the city, and the epidemiological picture was by no means clear.

It was a case tailor-made to demonstrate the power of phage-typing. The epidemiologists went to the well-to-do residents and "made sure that the organisms causing their disease reached the central laboratory".[9] In other words, they persuaded them to provide the necessary samples of urine and faeces, and personally conducted the samples to the laboratory. The laboratory results showed the victims all to be suffering infections caused by the same phage-type, one not previously identified in Britain. Inquiry revealed that all these people, five of whom died, had recently returned from holiday at a "pleasant and fashionable hotel" in Cornwall, which investigation revealed to have a history of associated typhoid cases. The source of infection was traced to an intermittent carrier on the hotel staff, who had contracted typhoid while serving in the Boer Wars in South Africa forty-three years earlier. Since he was able to name the stream from which he had drunk the water that infected him, the investigators contacted South Africa: cases of the same phage-type were still occurring in the vicinity of that African stream. [10]

The translation of an African strain of typhoid to a Cornish well in this instance not only illustrated the obvious significance of human agency in spreading this disease but also opened up a more complex natural history for the infection. What had appeared as a universal disease with global distribution was now fragmented into

[9] WH Bradley, "The control of typhoid fever", *Public Health*, 62, 1949, pp. 159-163, p. 89.
[10] Ibid, p. 162.

a collective of infection within which there existed the possibility of different strains with specific local connections. By accident, or design, these strains might be carried beyond their local ecological niche into the wider world, to establish enclaves of infection thousands of miles from home. Indeed, we may speculate around issues of evolution and mutation, and the possibility that there had once been an indigenous British strain of typhoid that was augmented over centuries by imports from Europe, India, Africa and the Americas.

If phage-typing established a newly complex international identity for typhoid, the older bacteriological methods of identification also provided evidence of the consequences of the globalisation of trade and transport in the years after 1940 for the distribution of the lesser salmonellas.[11] In this respect, the experience of England (and Wales) offers a telling story of changing disease ecologies, of a relatively stable bacteriological environment that came under siege from alien invaders, and of bacteria that in the course of a few decades took on a global future.

The history of salmonella infections in England and Wales can be charted with some degree of confidence from the mid-1920s. By this date serological methods of identification had been reliably established, and the Ministry of Health's small bacteriological laboratory was undertaking the monitoring of food poisoning outbreaks, identification of causal organisms, and laboratory research into the natural history of the salmonellas. For William MacDonald Scott, one of the laboratory's two permanent scientific staff, the study of the salmonellas was an enduring commitment.[12] Among his scientific contributions on this score was the elucidation of the mechanisms of salmonella infection in (duck) eggs, and the demonstration of

[11] This point is also made in Frank Fenner, "Infectious disease and social change", *Medical Journal of Australia*, 2, 1, 1971, pp.1043-1047, p. 1044.
[12] Anon, "W M Scott, MD", *BMJ*, 1, 1941, p. 735.

significant levels of salmonella infection in pigs before slaughter.[13] He was also involved in confirming the links between specific species of salmonella and food poisoning outbreaks, often for the first time.[14] Together with his colleague Frederick Griffith, Scott operated a singular service out of a tiny, poorly equipped and chaotic facility initially n Carlisle Place, behind Westminster Cathedral and close by Victoria Station, later in Endell Street, Covent Garden.[15] Described as men who "could do more with a kerosine tin and a primus stove than most men could do with a palace", they offered a 'compendium of knowledge, a wealth of experience and above all, a willingness to help, at whatever cost of time and trouble' that was said seldom to be found elsewhere.[16] Visitors came to them from all over the British Isles, and from abroad, and Scott was an integral part of an international correspondence network centred on salmonella taxonomy.[17] Given this background, and with the reservations that must always surround the reporting of salmonella infections,[18] it seems reasonable to accept as indicative the official record that, prior to 1940, just 14 salmonella serotypes had been identified in England and Wales. These included, besides the generic typhimurium and enteritidis, what were termed the 'indigenous' types, isolated for the first time in Britain and Ireland: thompson, newport, dublin, derby, eastbourne, stanley, london and aberdeen.[19] In fact, thompson later turned out not to be British at all, as will be shown shortly,

---

[13] W M Scott, "Food poisoning due to eggs", *British Medical Journal*, 2, 1930 pp. 56-59
[14] See for example, WM Scott ,"Food poisoning due to Bacillus suipestifer (sub-group II)", *Journal of Hygiene*, 25, 1926, pp. 406-414.
[15] H D W, "A fitting memorial", *Lancet*, 1, 1941, pp. 588-89; Anon, 1941, p. 735", described the Ministry laboratory as 'fantastic in discomfort and lack of good equipment'.
[16] H.D.W, 1941, p. 588.
[17] See Anne Hardy, "The salmonella connection: science, network and knowledge, 1900-1939" in Astri Andresen and Tore Gronlie eds, *Transferring public health, medical knowledge and science in the 19th and 20th Century*, Bergen: Stein Rokkan Centre, 2, 2007, pp. 67-77.
[18] See http://www.epigis.dk/frontpage.html, accessed 28.05.09.
[19] Medical Research Council Special Report Series, "The bacteriology of spray-dried egg; with particular reference to food poisoning", 260, 1947, p.62.

This baseline of 14 serotypes says something about the nature of Britain's import trade in organic materials before 1940, as well as about the nature of British agriculture, and British eating habits, notably the preference for well-cooked meat, and domestic economies so structured as to make left-over cooked meat dishes a rarity. [20] Although long distance trade in organic materials had existed before World War II, the post-war years saw an explosive increase in the level and complexity of such trade.[21] In this respect, the salmonella record reveals World War II as something of a watershed. From being islands with a largely self-contained salmonella ecology, Britain precipitately entered a global bacterial universe. War-time provisioning provided bacterial opportunity. As the Ministry of Food later explained, Britain's food situation had become critical by 1941. European sources of supply were no longer available; shipping losses, especially of vessels with refrigerator space, were heavy; and imports of animal feeding stuffs had been drastically reduced. To maintain food supplies "at the highest level" it became "imperative" to achieve maximum efficiency of shipping space.[22] The Ministry's solution was to turn to America for supplies of spray-dried egg. Spray-dried eggs well outstripped boneless beef, dried milk, canned corned beef and shell eggs in terms of nutritional value; they were easy to reconstitute and were thought to have "good palatability"; unlike shell eggs, they could be shipped without refrigeration; and they were 'most economical' of space and packaging material. Unfortunately, this was virtually a new industry, if a rapidly developing one, and although the heat treatment used was known to be relatively mild, the Ministry

[20] A Ginsberg and A Robertson, "Meat hygiene", *Veterinary Record* 61, 1949, pp. 9-10, p. 9.
[21] Editorial, "Food spread disease", *Medical Officer*, 80, 1948, p. 225. For these developments see Yrjo Kaukianen, "Growth, diversification and globalisation: Main trends in international shipping since 1850" in Lewis R Fischer and Even Lange eds, *International merchant shipping in the nineteenth and twentieth centuries: The comparative dimension*, St John's Newfoundland: International Maritime Research Association, 2008, pp. 1-56, especially Fig 3, p.11.
[22] MRC, "Bacteriology of spray-dried egg", Appendix by the Ministry of Food, 1947, p. 65.

deemed the adoption of bacteriological standards for spray-dried egg impracticable.[23] Post-war investigations revealed, however, that the conditions in which American spray dried egg was prepared were far from satisfactory, and offered every opportunity for contamination previous to drying. The eggs, still dirty from the farm, were first transported to the processing plants, where they were in some cases washed, in others not. They were then broken by hand onto wire netting and spray dried, but the temperature used was insufficient to kill bacteria.[24]

The first consignments of spray-dried egg arrived in Britain towards the end of 1941. Distribution was initially limited to bakers, confectioners and caterers, but from July 1942 until January 1946, supplies were made available to the general public in monthly issues of 5 oz packets, each containing the equivalent of 12 eggs.[25] From July 1942, there was a sudden and unprecedented rise in the number of food poisoning outbreaks. Whereas a total of 428 incidents had been reported between 1923 and 1939, in 1944 alone the number reached 454. Significantly, most of the isolations made from the latter part of 1942 were of salmonella types new to Britain. In 1942 ten new species of salmonella were recorded in the country's laboratories; in 1943 a further 8; and in 1944 another 6, making 24 in all. And the commonest occurring were, in descending order, oranienburg, montevideo, meleagridis (an avian salmonella), anatum (usually a sub-clinical infection of waterfowl), tennessee and bareilly. Oranienburg had previously been recorded just once in Britain, in 1935.[26] As the MRC noted, 'the suggestion is very strong that during the latter part of 1942 some

[23] Ibid.
[24] P S Waters and S. Mander, "An investigation into the incidence of salmonellae in South Australian eggs and egg pulp", *Monthly Bulletin of the Ministry of Health*, 12, 1953, pp. 127-129, p. 127.
[25] MRC, 1947, pp 61, 41. Imports ceased when the Lend Lease arrangement came to an end in 1946, and it became important to economise on dollar imports: See Ina Zweiniger-Bargielowska, *Austerity Britain*, Oxford: Oxford University Press, 2000, p. 214.
[26] TV Cooper, "A case of fever due to infection with bacillus oranienburg", *Monthly Bulletin of the Emergency Public Health Laboratory Service* (MBEPHLS), 3, 1944, p. 40.

new source of infection was introduced into this country'.[27] By 1948, the public health community was finding the spread of food borne infections "highly disquieting", describing them as "escapes" from sanitary control.[28]

In the post-war world, this pattern continued. By 1960, the number of salmonella types reported in England and Wales was getting on for 200, although only a few of the imported ones had become relatively common –particularly heidelberg, anatum, oranienburg, and montevideo, each of which had caused more than 200 outbreaks. Of the rest, fifty had been isolated from just one food poisoning incident, and a further 50 from fewer than 5 incidents.[29] As the local public health authorities noted, their task was often complicated by not knowing which of all these newly introduced varieties were pathogenic to man, although from the perspective of global spread this is less of a consideration. More interesting is the speed with which some of these invaders became established, and the routes by which their incursions were made.

It did not need to take long for 'foreign' salmonellas to become well established in new territory, although with some it may have taken persistent introductions over a period of time to establish a viable indigenous base for individual bacteria in the community. Thus salmonella thompson, which had first been recorded in 1924 in England, was deemed by the Medical Research Council in 1947 to be one of the country indigenous varieties, although it subsequently appeared to have been a variety introduced from China. S. heidelberg, introduced in 1953, entered the table of top ten isolated varieties in England and Wales within two years, and by the later 1960s was

[27] Ibid, p. 62.
[28] Editorial, 1948, p. 225.
[29] A McGregor, AH Fairlamb, and A Hutchinson, "A family infection due to salmonella abony", *Med. Off.* 102 (1959), p. 329.

established in breeding flocks of British poultry.[30] In March 1970, s. agona entered Britain in imported fish meal. By early June human infections linked to the consumption of ready-roasted chickens and contact with day-old chicks had been recorded; in July there was a milk-borne outbreak, and turkey poults sold on from a hatchery were dying in several; it was now being isolated from broiler chickens, turkeys, cattle and pigs. Agona was isolated from just one further consignment of fish-meal, but rapidly established itself in the breeding flocks, where, it was observed, it might be expected to maintain itself indefinitely.[31]

By contrast with the history of heidelberg and agona, the case of s.thompson illustrates how the foothold of some salmonellas could be more tenuous. Once again, eggs played a critical part in the salmonella thompson story. At the time when thompson was identified, England was importing considerable quantities of frozen tinned egg from China – some 40,000 tons of Chinese frozen liquid egg and 1,000 tons of Chinese egg albumen found their way to British markets every year, primarily for use in the baking and catering trades.[32] From 1924 until 1941, thompson was the third most frequently isolated among English food poisoning salmonellae. Between 1941 and 1945, however, incidents dropped away, but the serotype resumed its position in the league tables in 1946. At this period, although salmonella infection in duck eggs had been established, it was generally thought not to be a problem in hen eggs and the source of the thompson infection was regarded as a mystery. Despite its indigenous status, it was very rarely met with in cattle, sheep, pigs, rats or mice; and only very occasionally in Britain in poultry.

---

[30] J H McCoy, "Trends in salmonella food poisoning in England and Wales 1941-72", *Jnl Hyg* 74, 1975, pp. 271-282, p. 277-9.
[31] Ibid, p. 280.
[32] Public Health Laboratory Service, "The contamination of egg products with salmonellae, with particular reference to salm. paratyphi B", *MBPHLS*, 17, 1958, pp. 36-51, p. 48.

Throughout the interwar period, however, outbreaks of food poisoning were regularly associated with the consumption of bakery products filled with synthetic cream; during the war there was a lull in such incidents; after the war they resumed. In 1952 series of eight outbreaks of paratyphoid fever in South Wales, all caused by a single phage-type, were all associated with bakeries selling cream cakes. The firm supplying the cream was also sending it to other parts of the country where no paratyphoid was occurring, and the search was on for another ingredient commonly used by all the bakeries. By a painstaking process of elimination processed egg and egg products were identified as the source of infection. It was only then realised that a great deal of food poisoning and many recent outbreaks of paratyphoid fever had been directly or indirectly associated with contaminated egg products.[33]

The alert on egg products threw fresh light on the source of salmonella thompson. As the third most frequently isolated serotype in the 1950s, thompson was the most successful of the bacterial invaders at this time, and bacterial analysis linked it to frozen Chinese egg. Displaced in the salmonella hierarchy when its medium of transmission ceased to be imported during the war, it returned in force with the resumption of trade in Chinese egg products after 1945. It was circumstantial evidence, but it was strong enough to be incriminating.[34]

The thompson case none the less raised issues of prevention. Diplomatically, the Public Health Laboratory Service made no attempt to indicate the administrative measures "that might be taken on the basis of these findings" to prevent illnesses caused by contaminated egg products.[35] Although the Medical Officers of the great seaports would, it seems, have taken action with alacrity, since the episode produced "convulsion" in their food inspection systems, they found no political support for

[33] Ibid, pp. 36-7.
[34] Ibid, p. 44; Table 6, p. 45.
[35] Ibid, p. 51.

action. On the one hand, the product was of "urgent commercial use", and the bakery trade found pasteurised egg unsatisfactory to use, on the other there was a clear political steer that Chinese egg products "should have a special place in our favour" – they represented the opening of trade between Communist China and Great Britain.[36] And so, the Port Medical Officer of Liverpool, Andrew Semple, noted, "statesmen and salmonellas were to compete for our attention". [37] And it was an issue that rumbled on. Questions were asked repeatedly in the House of Commons, and as late as May 1963, the Ministry of Health representative answered 'No' to a direct query from MP Dick Taverne, as to whether the Ministry would ban imports of frozen liquid egg until regulations had been made for their compulsory pasteurisation.[38] As a pasteurisation technique resulting in a satisfactory product was developed around that time, however, regulations were made later that year, to come into force on 1 January 1964.[39]

Egg products were by no means the only imports carrying significant bacterial loads in these early years of resumed and globalising peace time trade. Two other foodstuffs, likely and unlikely, raised concerns around 1960, imported shellfish, and dessicated coconut. Japanese prawns and Chinese shrimp were not specifically associated with salmonella, indeed Semple deliberately did not test them for salmonella, but they none the less supplied another instance of the problem "of deciding the most effective point of compromise between the needs of commerce and the needs of public health", a point of special difficulty where the infective significance of certain bacteria to the human organism was not fully known.[40] Japanese prawns first attracted attention in January 1959, when samples taken by the

[36] A B Semple, "Some recent problems of imported foods", *Med. Off.*, 104, 1960, pp. 101-105, p. 101.
[37] Ibid.
[38] Parliament and Public Health, "Paratyphoid and imported liquid egg", *Med. Off*, 109, 1963, p. 339.
[39] Anon, "Pasteurised liquid egg", ibid, 110, 1963, p. 185.
[40] Semple, 1960, p. 102.

Medical Officer for the Port of London were found to be "very unsatisfactory".[41] The Ministry of Health was advised, and their response, again to quote Semple, "very delicately [summarised the problem] in the phrase 'the difficulty of basing executive action on provisional bacteriological standards'". This was another new area of trade, and was clearly not to be actively discouraged. Further investigation confirmed that the health authorities were dealing with "a very carelessly prepared commodity", with bacterial plate counts generally exceeding 250,000 per gram. Liverpool at least, worked out its own effective methods for managing the problem, and the only consignment of prawns received at the port in the first five months of 1960 gave a plate count of just 15 organisms. But just as the problem of Japanese prawns seemed to be "resolving" nicely, it metamorphosed into a problem of Chinese shrimp.[42] Keeping up with the development of new global trading opportunities presented significant problems to port health authorities in the 1950s and 1960s.

On the face of it, dried coconut seemed an unlikely vehicle for food poisoning. This was an old established luxury trade run out of Ceylon (modern Sri Lanka) by English merchants, with a very well organised trading association. In this case, policing of the operation was made easier by the co-operation of the trade, who accepted a system of coding packages of the product to help identify contaminated consignments.[43] The problem was, however, extensive, involving a great range of salmonella types. As Semple, observed, and his phraseology is telling in its recognition of the wider problem:[44]

[41] Ibid.
[42] Ibid.
[43] Ibid, p. 103.
[44] Ibid.

> We have detected *Salmonellae paratyphi-B, typhimurium, thompson, bareilly, hvittingfoss, waycross, Newport, muenster, Poona, angoda, perth* and *seftenberg*, quite a global assembly!

In all, 33 different serotypes were identified in dessicated coconut, their nomenclatures suggesting origins from Norway and Scotland to Africa, and south-east Asia.[45] The question of how all these serotypes arrived in Ceylon, which presumably had once had a contained salmonella ecology, goes unanswered, but it was clear how they were enabled to continue on their travels. When the nuts were harvested, they were collected and stored on the ground for a month or so before being delivered to the mills. Farmyard manure was used as a fertiliser for the palm trees, and domestic cattle grazed among them. Contamination of the husks could lead to infection of the workers and their equipment, and to contamination of the broken nuts. Tank washing water could be infected at source, or by the men who went into the tanks to clean them.[46] The problem was confounded, as in bakeries, when infected raw material became lodged or deposited in the processing machinery, allowing the contamination of clean supplies. Despite the coding system, the health authorities were forced to conclude that they should only accept consignments in which repeated sampling had not detected any contamination.[47]

At much the same time as dried coconut was demonstrating its ability to carry a global assembly of salmonellae around with it, a rather different product was opening up another trajectory of microbiological expansion. The establishment of an international network of salmonella centres, devoted to the isolation and study of

[45] A Semple, A J Graham and E M Dutton, "A review of the sampling of imported dessicated coconut", *Med. Off*, 105, 1961, pp. 59-60.
[46] Ibid, p. 60.
[47] Ibid.

these organisms, had taken place just before World War II.[48] As a result, the pace of identification and investigation quickened globally in the post-war world. Partly as a result of the spray-dried egg affair, the question of how the salmonella got into the egg, or into the chicken that produced the egg that contaminated the spray-dried egg, attracted scientific attention. The large scale commercial production of eggs and poultry had been developed in the United States during the interwar years, and part of the regime of these programmes of intensive production was the feeding of birds with high protein feedstuffs.[49] The use of such feeds in animal husbandry provoked popular disgust and dismay in Britain and elsewhere when the blaze of publicity surrounding bovine spongiform encephalitis broke in the mid-1980s, but the use of these feeds dated back into the nineteenth-century. Feeds of bone-meal, dried meat residue, blood and bone, and fish-meal, were being advocated by animal feeds experts from later nineteenth century, and were increasingly used in the United States from around 1920.[50] In Britain their use was more limited before 1940; it was, for example, only with the feeding stuff shortages of World War II that British farmers began using fish meal.[51] Concerns about the use of high protein feeds, from a microbiological point of view, began to surface in the mid 1950s. In 1957, for example, the head of the International Salmonella Centre in Copenhagen, Felix Kauffmann, writing to an Israeli colleague on the subject of s. infantis, noted that the Scandinavian countries had a problem with bone-meal and fish-meal in this connection.[52] A year later, a researcher at the Hygiene Institute in Hamburg had identified 11 different 'American' type salmonellas from Angolan fish-meal.

[48] See Hardy, 2007, pp. 67-77.
[49] See below, note 55.
[50] Herbert H Cousins, *Farm foods or the rational feeding of animals* (translated from the German), London: Gurney and Jackson, 1895, pp. 203-06; see below, note 55.
[51] J C B Ellis, *The feeding of farm live stock*, London: Crosby,. Lockwood & Son. 1937; James A Kerr, "Fish inspection and modern development", *Medical Officer*, 34, 1935 pp.: 165-66.
[52] Fritz Kauffmann, *Erinnerungen eines bakteriologen* , Copenhagen: Munksgaard, 1969, p. 240.

Kauffmann wrote to his contact, Philip Edwards, at the US Communicable Disease Center in Chamblee, Georgia, to ask when imports of African fish-flour to the USA began, and whether American turkeys were fed on it.[53] In an additional note he further observed:

> the Angolan product is terrible, it is the greatest reservoir of salmonella types in the world. A German colleague who wanted to isolate new *Salmonella* types bought a sack of fish-meal and had a new type at once.[54]

The response from Georgia was confirmed that the trade in Angolan fish meal had existed before World War II, and there was a "distinct possibility" that that it might have been responsible for many outbreaks of disease among fowls in the USA. Fish-meal was one of the prime sources of alpha tocopherol (Vitamin E), and the meal was heated as little as possible during manufacture to avoid damaging the alpha tocopherol content.[55] Edwards considered "that the *Salmonella* content of fish meal is entirely a problem of plant sanitation, which I understand is really horrible".[56] As with American spray-dried egg, Chinese and Japanese crustaceans and Ceylonese coconut, the conditions in which Angolan fish-meal was manufactured ensured a simultaneous manufacture of salmonella infections. Ominously and presciently, Edwards noted that there had been an intensive search for sources of protein, especially animal protein, in America, and substances were now being added to animal feeds "which were never thought of when I was an agricultural student". Thus

[53] Ibid, p. 251.
[54] Ibid, p. 254.
[55] Ibid, p. 255.
[56] Ibid, pp. 255-6.

he had recently received a number of salmonella cultures isolated from a feed additive composed of the feathers and entrails of fowls. Within the past two years, he noted,[57]

> I have been quite concerned about the continuous seeding of our animal population with *Salmonella*. So long as present feed producing practices prevail, and until such time as plant sanitation is enforced, I cannot see how the reservoir of *Salmonella* infections in domestic animals can be reduced.

Edwards' concerns about the raw materials and practices used in the animal feedstuffs industry foreshadow the later issues around BSE, of course. But below the surface of the Angolan fish-meal problem, which was, essentially, that it was responsible for distributing a wide range of salmonella serotypes among populations of domestic fowls, lay further evidence of global interconnection. These were types that had originally been designated as 'American', having been identified by American researchers, and (mostly) being named for American places. In April 1958, Kauffmann wrote to Edwards, advising him that 20 so-called American serotypes were being isolated in Africa, especially in the Belgian Congo.[58] Like S. thompson, originally considered English but revealed as Chinese, the American salmonellas kentucky, pomona, california, saint-paul, georgia, richmond, miami, waycross, berkeley and urbana, among others, seemed after all to have originated in Africa. The cycles of export trades in fish meal from Africa to America, and in egg products from America to England, had equally promoted the export and global voyaging of a host of microbial parasites.

The global voyaging of salmonella bacteria had, as the observations of Kauffmann and Edwards suggest, yet another dimension. Once they established themselves in bird and animal populations, these continued to act as reservoirs which

[57] Ibid, p. 256.
[58] Ibid, p. 252.

could disseminate the bacteria as the opportunity arose. This situation had already occurred with American poultry flocks, and was shortly to be replicated in poultry flocks elsewhere. By 1963, for example, it was noted in England that 'there is much published evidence showing that imported foodstuffs (egg products, dessicated coconut, meats and animal feeding stuffs) have given rise to a vast reservoir of salmonella infection in this country'.[59] In this case the newly established reservoirs clearly included humans, but the sheer numbers of salmonellas that were now being discovered argued for a very widespread distribution – and ancient origin – of reservoirs of these bacteria in disparate animal and bird populations across the globe.

In the years after 1920, the microbiologists gradually uncovered and explored numerous potential and actual reservoirs of salmonella infection, some plausible some apparently unlikely. Rats and mice, for example, were initially popular suspects for endemic infections, although the role of rats proved harder to substantiate.[60] Cattle had been known to harbour salmonella since the 1880s, and pigs also were quickly fingered. Other species were more unexpected. An investigation in 1953 showed that tortoises, which were then imported into Britain in substantial numbers as pets (about 300,000 a year at this period), largely from the Mediterranean, carried at least 17 different types of salmonella.[61] Thus when in July 1958 a family in Kingston upon Hull were found to be infected with the uncommon salmonella abony (Hungary), the family tortoise, the children's school tortoise, and that belong to a neighbouring family which was said to be "a frequent visitor" to the family garden, fell under

[59] Wilfrid H .Parry, "The problem of salmonella food poisoning", *Med. Off,* 110, 1963. pp.:27-32, p. 31.
[60] William G. Savage and William J Read, "Gaertner group bacilli in rats and mice", *Jnl Hyg,* 13 , 1913-14, pp. 343-352; William G. Savage, "The sources of infection in food poisoning outbreaks", ibid., 17, 1918, pp. 30-33, pp. 8-9; Ahmed M Khalil, "The incidence of organisms of the salmonella group in wild rats and mice in Liverpool", ibid., 38, 1938 pp. 75-78.
[61] JA Boycott, Joan Taylor and S Hilda Douglas, "Salmonella in tortoises", *Journal of Pathology and Bacteriology*, 65, 1953, pp.401-11.

suspicion. All three were taken to the Public Health Laboratory for bacteriological investigation. The family tortoise and the school tortoise were innocent of salmonellae, but the neighbours' tortoise was excreting both s. potsdam and s. rubislaw (Scotland). The source of the abony infection remained obscure, although it had been found in the 1953 tortoise investigation and was thus known to exist in these creatures.[62] Reptile infections, from tortoises, turtles, snakes, geckos, iguanas, horned toads, lizards and chameleons, remain a frequent source of human salmonellosis to this day – to the extent that the American Centers for Disease Control devote a whole feature to them on their website, an honour not accorded to any other animal group.[63]

An examination of the twentieth-century literature on the salmonellas thus reveals a global canvas of habitats, and multitudinous strands of migration, transportation and exchange. In the years after World War II, it seems that changes in technology, and in levels of human trading and transport activity, resulted in the opening up of multiple transit routes for this group of organisms. The example of England at least, suggests that at this time hitherto stout borders against minor microbial invasion crumbled. Within the dizzy constellation of international salmonella exchange that then emerged, nothing was stable. Different serotypes dominated in different parts of the world, and their dominance too could change over time. In 1958, for example, it was noted of Britain that salmonella typhimurium had been responsible for 75% of cases of human salmonellosis since 1945, with enteritidis and thompson running second and third respectively. Since circa 1950,[64] however, heidelberg had been bidding fair to displace both thompson and enteritidis as

[62] A.Mcgregor, AH Fairlamb, and A Hutchison, "A family infection due to salmonella abony", *Med.Off.*, 102, 1959, pp. 329-30.
[63] http://www.cdc.gov/Features/ReptilesSamonella. Accessed 28 May 2009.
[64] Since 1953, in fact. See note 30 above.

important causes of infection in humans.[65] The factors determining these shifts in the league tables are not always clear, or easy to tease out, but it was noted during the first half of the twentieth century, that s.typhimurium was the dominant infection in Britain, while enteritidis reigned on the Continent of Europe – a difference thought to originate in the Continental preference for lightly cooked meats as against the British for meat extremely well done. [66] In the post 1945 world, things became more complex, as agriculture commercialised, transport networks facilitated speedy long-distance movements of goods, animals and people, human foodways changed, and hundreds of salmonellas took to the road. Between 1968 and 1998, of the top 100 reported isolates of salmonella serotypes in the United States, typhimurium and enteritidis ranked numbers one and two, but heidelberg came in at number 3. [67] By 2007, heidelberg was down to sixth position in the US league table.[68] In the European Salmonella Atlas published in May 2009, enteritidis and typhimurium came in at numbers one and two respectively (in reverse order to the US) as the serotypes most frequently notified; heidelberg came in at number 10.[69]

When I began to construct this paper, I had no idea that s. heidelberg would emerge as a leading actor in my story. It is now a widespread infection in animal populations, and is transmitted to humans through contaminated foodstuffs. It is clear that the globalisation of trade following World War II provided this serovar, and so many of its cousins, with unprecedented opportunities to travel and find congenial habitats abroad. Above all, their journeying and successful acts of colonisation were enabled by trade in contaminated commodities. If trade and transport played their part

[65] V D Allison, "Crowd diseases", *Public Health*, 72, 1958, pp. 43-53, p. 52.
[66] A Ginsberg and A Robertson, "Meat hygiene", *Veterinary Record*, 61, 1949, pp. 9-10, p.9.
[67] Centers for Disease Control, *An atlas of salmonella in the United States 1968-1998*, Atlanta, GA, 2001, CD-Rom version.
[68] http://emedicine.medscape.com/article/228174-overview, accessed 19 August 2009
[69] http://www.epigis.dk/frontpage.html; accessed 28 May 2009.

in creating a global future for these micro-organisms, so did contamination. From egg-canning plants in China, to egg-drying plants in the US, to coconut processors in Ceylon, Japanese prawn producers and African fish-meal manufacturers, the global futures of these bacteria were shaped by a lack of concern for purity in the processes of industrial food production that was at once local and global in nature. Human activity – economic, social, cultural and political – enables the establishment of new disease reservoirs and new disease ecologies.

Locally named and globally distributed, the salmonellas adapt happily into new ecological niches. They are resourceful and resilient; they do not respect human boundaries. As long as there is animal life on earth, the salmonellas have a future.