Medical History, 1998, **42:** 328–346

On the Cusp: Epidemiology and Bacteriology at the Local Government Board, 1890–1905

ANNE HARDY*

There is a well-established tradition in the history of epidemiology that, after a promising start with the foundation of the London Epidemiological Society in 1850 and the work of John Snow and William Budd, the discipline suffered an eclipse with the coming of bacteriology. "Beginning in 1870 and until 1910", wrote David Lilienfeld in 1979, "the Bacteriological Era overshadowed epidemiology. During these 40 years epidemiology hibernated . . .".[1] It is a view which still holds good, and which is taken from the standpoint of the development of concepts and methods in the discipline, and depends on the tradition that with the discovery of bacteria epidemiology vanished in the pursuit of the specific agents of disease.[2] Both these criteria are flawed: the first represents an overly theoretical approach to the discipline which ignores the reality of a long and honourable practice of field epidemiology between 1870 and 1914; the second has complex historical roots, which bear little relation, in England and Wales at least, to the continuing vitality of epidemiological investigation in this period.[3] By the last decade of the century, indeed, practitioners of epidemiology had begun to perceive bacteriology as a threat to their own discipline and, by extension, to preventive medicine. This particular disciplinary anxiety can be detected between 1894 and 1906, between Emil Roux's announcement of the anti-toxin therapy for diphtheria in the summer of 1894, and Klinger's demonstration of the existence of the healthy typhoid carrier in 1906.

Scattered references in the contemporary literature testify to the growing unease of the epidemiological community in the years after 1894. It was perhaps articulated most clearly by an anonymous contributor to the *Journal of State Medicine* in 1896, who began by deploring the dullness of epidemiology when compared to the "joys of modern science"—the "manipulation of bacilli, the extraction of toxines and their antidotes, with

*Anne Hardy, MA, DPhil, Wellcome Institute for the History of Medicine, 183 Euston Road, London NW1 2BE.

[1] David E Lilienfeld, '"The greening of epidemiology": sanitary physicians and the London Epidemiological Society (1830–1870)', *Bull. Hist. Med.*, 1979, **52**: 503–28, p. 527.

[2] Ibid., pp. 527–8. See also David E Lilienfeld and Paul D Stolley, *Foundations of epidemiology*, 3rd ed., Oxford University Press, 1994, p. 28; Mervyn Susser, 'Epidemiology in the United States after World War II: the evolution of technique', *Epidemiologic Reviews*, 1985, **7**: 148–49. William Coleman notes that after 1880, an "all-encompassing biomedical orientation now entered epidemiology" and the role of environmental investigation was greatly reduced: *Yellow fever in the north: the methods of early epidemiology*, Madison, University of Wisconsin Press, 1983, p. 173.

[3] There are good grounds for suggesting that the English and American experiences differed significantly in this respect: see Elizabeth Fee and Dorothy Porter, 'Public health, preventive medicine and professionalization: England and America in the nineteenth century', in Andrew Wear (ed.), *Medicine in society*, Cambridge University Press, 1992, pp. 249–75.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

the off-chance of startling the world by announcing a perfect cure for everything". Since Roux's "famous bombshell", he continued,

> Preventive Medicine has become more and more lost in Bacteriology. To many a micro-organism is all-sufficient; they would summarily dispose of Diphtheria in three simple steps—examine all mouths, find Klebs-Loffler bacillus, isolate the subject. Others would inoculate with anti-toxin as a perfectly satisfactory method of dealing with the whole subject . . . these things . . . are not Preventive Medicine in the best sense, nor even on the road to it; and however great may be the benefits eventually conferred by Bacteriology, it can never lessen the need for the broader study of disease in its general relations on which the Science of Public Health mainly rests.[4]

As immunology began to reveal its potential with the production of cholera and typhoid vaccines by 1900 (however dubious their initial effectiveness), and as bacteriology confirmed the hypothesis of the healthy carrier for a number of diseases, the horizons of epidemiology appeared to contract, in the mind of many of its practitioners at least. In 1917, Major Greenwood noted the prevalence of the idea that "epidemiology is an occupation for the leisure moments of a bacteriologist", at the same time as William Hamer, then Medical Officer of Health for the County of London, recorded his "gloomy" supposition that epidemiology was "finally destroyed . . . when the healthy carrier hypothesis was formulated".[5] It was Hamer (1862–1936) in particular, a generation older than Greenwood (1880–1947), who largely created the idea of "the old epidemiology" and the myth of its eclipse by bacteriology.[6]

How far the threat to epidemiology was real in the years up to 1906 remains debatable. Christopher Hamlin has demonstrated the limitations of bacteriology in respect of water analysis, and the scepticism with which epidemiologists and public health officials continued to regard its utility in the years to 1900.[7] The failure of bacteriology to deliver a "simple and readily discoverable signature of deadly water",[8] inevitably coloured these specialists' attitude towards other applications of bacteriology in their field and re-enforced their sense of the value of the established, broadly environmental approach. On the other hand, anti-toxin therapy had an undeniable impact, and presented an unmistakable indication of the promise of bacteriology for the future. British epidemiologists trod their way through the 1890s in doubt as to the practical uses of bacteriological techniques yet in alarm at the apparently increasing tendency to reduce the problems of infectious disease to the simple detection of causal bacteria by bacteriological means; and increasingly too, they began to explore the possibilities of a bacteriological approach to the problems of field epidemiology. It was less an overshadowing of epidemiology by bacteriology than the gradual emergence of a new—if short-lived—epidemiological method.

[4] Anon., 'Diphtheria and elementary schools', *J. State Med.*, 1896, **4**: 245.

[5] Major Greenwood, 'The outbreak of cerebro-spinal fever at Salisbury in 1914–15', *Proc. R. Soc. Med.*, 1917, **10** (2): 55; W H Hamer, 'The epidemiology of cerebrospinal fever', ibid., p. 17.

[6] The theme can be traced in the great majority of Hamer's numerous contributions to specialist journals circa 1912–1932, and in his *Epidemiology old and new*, London, Kegan Paul, Trench and Trubner, 1928.

[7] Christopher Hamlin, *A science of impurity: water analysis in nineteenth-century Britain*, Bristol, Adam Hilger, 1990, chs 9, 10.

[8] Ibid., p. 300–1.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

The relationship between epidemiology and bacteriology, and the emergence of the new epidemiology can best be explored through the work of the Medical Department of the Local Government Board, which, as Hamlin noted, had been deeply sceptical of the relevance of bacteriology for epidemiology into the early 1890s. The department was one of only two professional strongholds of epidemiology in Victorian England (professional in the sense that its staff were employed to engage in epidemiological investigations), the other being the General Register Office (GRO), where the successive Statistical Superintendents, William Farr, William Ogle, John Tatham and T H C Stevenson, utilized the ample statistical materials generated by the registration system.[9] Outside these two departments, most of those engaged in the study of epidemic diseases in the later nineteenth century were essentially amateurs, whose real professional work lay elsewhere. Medical officers of health, army and naval medical officers, and general practitioners, for example, bulked large among those who contributed to the activities of the Epidemiological Society after its foundation in 1850. As a recognized academic discipline, epidemiology became established only after the First World War.[10]

The Medical Department was created out of the old Chadwickian Board of Health in 1858, initially reporting to the Privy Council, but after 1871 to the Local Government Board (LGB).[11] Between 1858 and 1872, John Simon, as Medical Officer, established the three principal areas of inquiry within the Department's remit: first, the progress and prospects of pandemics of disease such as cholera, influenza and rinderpest; secondly, the investigation of local epidemic outbreaks which might contribute to the sum of knowledge of the diseases involved; and thirdly, after 1865, the generally named but specific in character "auxiliary scientific investigations", which took place in the laboratory. The character of these scientific investigations was determined by the Medical Officer, although the allocation of the work was done by Emanuel Klein (1844–1925), the Viennese-trained histologist who pioneered bacteriology in Britain.[12] This tripartite pattern of interest was continued in the Department for many years after Simon's departure. In the 1870s and 1880s, the three types of investigation ran very much in parallel, but in the 1890s the pressures of modernizing science forged new links between them.

[9] See John Eyler, *Victorian social medicine: the ideas and methods of William Farr*, London and Baltimore, Johns Hopkins University Press, 1979; Simon Szreter, 'The GRO and the public health movement in Britain, 1837–1914', *Soc. Hist. Med.*, 1991, **4**, special issue: 435–63.

[10] With the creation of the Department of Epidemiology and Vital Statistics headed by Major Greenwood, at the London School of Hygiene and Tropical Medicine, 1927. The discipline of *public health*, however, had a longer university career, beginning with the establishment of the BSc degree at Edinburgh, and diplomas at Dublin, Edinburgh and Cambridge in the 1870s: see E W Hope, 'The influence of universities upon the advancement of public health', *Proc. R. Soc. Med.*, 1925–26, **19** (1–2): 22–9.

[11] See Public Health Act 1858, 21 & 22 Vict. c. 97; Royston Lambert, *Sir John Simon and English social administration*, London, McGibbon and Kee, 1963, ch. 14, especially pp. 311–17.

[12] See 'Edward Emanuel Klein', *Br. med. J.*, 1925, i: 388. As suggested here, this was contract science: Klein "allotted particular pieces of investigation commissioned by the Local Government Board" to his co-workers and pupils, for "practical distribution of the annual research grant by the Board was usually left to him".

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

Epidemiology in the 1890s

Late Victorian epidemiology had some confidence in its established methods and procedures. The statistical tools developed by William Farr remained those standardly in use, for it was only after Greenwood's appointment to the Lister Institute in 1910 that the introduction of Pearsonian statistical methods initiated movement towards the highly sophisticated mathematical techniques of the modern discipline.[13] The advent of bacteriology meanwhile had little immediate impact on the nature of epidemiological investigation, although it did introduce the idea of diseases as discrete clinical entities with some finite number of routes of transmission. The identity of diseases involved in epidemic outbreaks was still rarely confirmed in the laboratory, and field investigations generally revolved around statistical analysis and the assessment of local environmental factors. Field studies, based on observation and the judicious deployment of statistics, formed the back-bone of nineteenth-century epidemiology, and were the methods characteristic of the century's three classic epidemiological studies; Peter Ludwig Panum's study of measles in the Faroe Islands, John Snow's analysis of London cholera fields, and William Budd's account of typhoid fever at North Tawton in Devon.[14] These were also the standard methods employed by those who continued to study the behaviour of the infectious diseases, among whom were the medical inspectors of the Local Government Board.

The work of the Medical Department in the 1890s continued very much in the established tradition, but achieved a sharper focus. On the international front, three infections were of over-riding concern: influenza between 1890 and 1892; cholera between 1892 and 1898; and plague between 1898 and 1900. On the domestic front, the central concern of the Department in this decade was typhoid, or enteric fever, as it was often known. Of the thirty-five reports relating to endemic infections which were published by the Department in this period, eighteen, or just over half, were devoted to typhoid.[15] Next in line was diphtheria, with a total of six published reports.[16] At the same time, the auxiliary scientific investigations, which had previously dealt with a range of interests, from the tuberculosis and anthrax bacilli and the habits of streptococci to the composition and effectiveness of disinfectants, became much more tightly focused on the etiology of typhoid and on the behaviour of the typhoid bacillus and associated micro-organisms in the natural environment.

[13] Victor L Hilts, 'Epidemiology and the statistical movement', in A M Lilienfeld (ed.), *Times, places and persons: aspects of the history of epidemiology*, Baltimore, Johns Hopkins University Press, 1980, pp. 43–51, p. 50. For Farr's methods see Eyler, op. cit., note 9 above.

[14] Peter Ludwig Panum, *Observations made during the epidemic of measles on the Faroe Islands in the year 1846*, New York, Delta Omega Society, 1940; John Snow, *On the mode of the communication of cholera*, London, J Churchill, 1849; William Budd, *On the propagation of typhoid fever*, London, T Richards, 1861.

[15] The Department undertook a great many investigations each year, of which the reports of only a selection were published in the Medical Officer's annual report. Nor does the figure of 18 include the separate inquiry into the outbreak of typhoid at Maidstone in 1897, undertaken under the auspices of the Local Government Board, but not of the Medical Department: see 'Borough of Maidstone. Report of inquiry on epidemic of typhoid fever, 1897', British Parliamentary Papers (hereafter PP) 1898, xl, pp. 499–613.

[16] The remainder were concerned with scarlet fever (3 reports), "meningitis" (3), measles (2), pneumonia (1), typhus (1), and smallpox (1). Reports of minor departmental investigations into local outbreaks of disease (generally easily "resolved") were often printed in the *Journal of State Medicine*.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

Concern over typhoid was, of course, nothing new. Already in 1867, John Simon had noted sourly that again to detail the dependence of the disease on "conditions which it is disgusting to specify" might seem superfluous, but that for as long as the disease continued to cause 15,000 to 20,000 deaths a year, and until the legislature had quite exhausted its powers in "fruitless endeavours against the evil", he would continue to do so.[17] In the 1870s and 1880s, however, death-rates from the disease declined, and by 1890 it was in retreat throughout England and Wales, although pockets of exceptional incidence were not uncommon.[18] Ever since the disease had been clinically distinguished from typhus by William Jenner in 1847, typhoid had been recognized as, in the words of Charles Murchison, "the endemic fever of England", and it was considered to be a "sensitive but trustworthy test of sanitary condition".[19] It was therefore a matter for self-congratulation to the preventive establishment that the annual death-rate from typhoid and associated diseases had declined from a maximum of 390 per million living in 1875 to the minimum yet reached—172 per million—in 1890.[20] The satisfactory national picture masked less satisfactory local conditions, however. In his report for 1890, for example, the Registrar-General noted that the disease still appeared to be unduly prevalent in "that tract of ground that runs along the east side of Northumberland and Durham, from Morpeth on the north to Middlesborough and Guisborough on the south".[21] Here the death-rate in 1890 had been 394 per million living: more than the national average, and almost twice that of the twenty-eight great towns, and greater than any one of these with the sole exception of Salford. Examination of the records of the previous three years showed that this excess typhoid mortality was not exceptional.

The Registrar-General's observations presaged an ominous development. In the autumn of 1890, statisticians at the General Register Office became aware of an unusual prevalence of typhoid in the registration districts of Stockton, Darlington and Middlesborough, the three principal industrial towns of the lower Tees valley, lying in the southern part of that tract of land singled out by the Registrar-General as especially notorious. According to their usual practice, the General Register Office notified the Medical Department, who dispatched an inspector, F W Barry, to investigate. Barry's report was a classic piece of epidemiological investigation, the first of the series on typhoid published by the Department in the 1890s, and the first to lay bare inadequacies in the sanitary provision of provincial and rural England which became of vital concern in the context of the seventh European cholera pandemic.

The Epidemiology of Typhoid

The nature of England's typhoid problem had changed significantly since the creation of the Medical Department in the late 1850s. It was no longer a universal hazard of English life, producing consistently high death-rates in particular places amid generally

[17] Medical Officer's Annual Report, Local Government Board (hereafter MOAR LGB), PP 1867–68, xxxvi, pp. 421–2.

[18] Thus in Nottingham typhoid mortality rose by 15% between 1889–93 and 1899–1903: Martin Daunton, *House and home in the Victorian city*, London, Edward Arnold, 1983, p. 254.

[19] Charles Murchison, *A treatise on the continued fevers of Great Britain*, 3rd ed., London, Longmans, Green, 1884, p. 441; *Lancet*, 1888, i: 785. It may be noted here that typhoid was first distinguished from typhus by William Gerhardt of Philadelphia in the 1830s. The British characteristically only began to pay the distinction attention when Jenner rediscovered it.

[20] Registrar-General's Annual Report (RGAR), **53**, PP 1890–91, xxiii, p. 12.

[21] Ibid.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

terrible sanitary conditions; increasingly, it manifested itself as a relatively isolated and apparently random departure from normally healthy conditions. Even in the north-east, the actual number of typhoid deaths remained small. Nevertheless, the fact that the disease, like cholera, was water-borne, and that it could still, despite the proud achievements of England's public health service, produce notable outbreaks, indicated that the country was still highly vulnerable to epidemic cholera should it break through the cordon of the port sanitary authorities. The epidemic at Stockton, Darlington and Middlesborough had occurred in two waves over a period of some five months between September 1890 and February 1891, and it featured in the Medical Department's report for 1891–2. In the next two years, six further investigations were published into typhoid outbreaks in the West Riding; in Ryedale, North Yorkshire; along the course of the river Trent; and at the towns of Worthing, King's Lynn and Chester-le-Street. As Richard Thorne Thorne noted in 1894, all these investigations revealed the widespread distribution of the disease by contaminated water supplies: the subject was

> one of pre-eminent importance to England, not only because, on the Continent of Europe, we are still face to face with cholera, a disease which owes its widest and most fatal distribution in this and other temperate climates to the agency of water contaminated with human excreta; but because even when the contaminating agent is insignificant in amount, and the point of contamination strictly localised, yet the resulting mischief is often as wide in its distribution as is the area of the particular water supply.[22]

This observation was to be reinforced by the subsequent investigations of typhoid outbreaks at High Wycombe, Marsh Wycombe, Bicester, Loddon, Chichester, Camborne, Falmouth, and Newport, Isle of Wight, in the years up to 1900, and indeed, by several notable outbreaks in the years after 1900.

The epidemiological techniques employed in investigation of these outbreaks varied little. Late Victorian epidemiologists knew enough about typhoid to undertake investigations with a fair degree of confidence, but it was a confidence based on past experience of the disease and on epidemiological discoveries, not on bacteriology. The typhoid bacillus had been identified by the German bacteriologists Carl Eberth and Edwin Klebs in 1880; in 1884, Georg Gaffky had succeeded in culturing it; and it was later isolated from the stools of typhoid victims. Although these achievements were followed by failure to induce the disease experimentally in animals (the typhoid bacillus seems to be adapted to human beings alone), the bacillus had, by the 1890s, come to be regarded as "in all probability" the essential cause of typhoid.[23] The identification of the bacillus did not, however, facilitate epidemiological investigation, especially where water supplies were the suspected vehicle of infection, because the bacillus was difficult to detect in water, where its presence is often ephemeral. Indeed, the great Robert Koch himself, at this date, not only doubted the ability of most people to identify the bacillus in excreta with any certainty, but viewed the claims of various observers to have demonstrated the presence of the bacillus in drinking water with considerable suspicion.[24]

[22] MOAR LGB, PP 1894, xl, p. 375.

[23] See Emanuel Klein's discussion, 'The etiology of typhoid fever', in MOAR LGB PP 1894, xl, Appendix B no. 1, pp. 345–57.

[24] Ibid., p. 346.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

Bacteriology was thus not a resource of first call to the LGB's epidemiologists in the study of typhoid outbreaks. They relied on knowledge accumulated through clinical observation and investigation of the behaviour of the disease in discrete outbreaks. They knew that the disease was transmitted by the faecal-oral route, and that it could be communicated in polluted food, milk and water; they still considered there to be a possibility that it was communicated by foul air escaping from drains and sewers, or from accumulated filth. They knew that the incubation period was commonly 14 to 21 days and might be longer, and that infected individuals carried the infection about with them and distributed it in their stools. They knew that the clinical manifestations of the disease were various—some individuals were prostrated, others only slightly affected.[25] Although the concept of the healthy carrier was not established until 1906, knowledge of the dangers of infection presented by very mild, or "ambulant", cases to some extent made up for this. They also recognized the marked seasonal incidence of typhoid, which reached a peak of fatality in August and September, and declined rapidly as autumn advanced and cooler weather set in.[26]

The pattern of all the Medical Department's typhoid investigations thus followed a fairly standard formula. A careful chronological and spatial analysis of attacks and deaths from the disease was followed by an equally careful survey of all the possible causal factors. General sanitary conditions, sewerage and drainage, the disposal of excreta and household refuse, milk runs and water supplies each received careful consideration. Meteorological factors were also considered where likely to be relevant, as were the movements of persons and any recent changes in local sanitary or environmental features which might influence the course of public health. The great bulk of the typhoid investigations (thirteen out of eighteen) were carried out by three inspectors: Robert Bruce Low (King's Lynn, Ryedale, Trent, Camborne); Theodore Thomson (Rotherham, Worthing, Swinton and Pendlebury, Bicester, Newport, Chichester); and George Seaton Buchanan (Wycombe Marsh, Essex and Suffolk, Falmouth). All three were medically qualified, and all three made their subsequent careers entirely within the Medical Department.[27] F W Barry, who conducted the Teesdale inquiry, and who was the

[25] 'Signs and symptoms so distinctive of typhoid as to render a clinical diagnosis secure are present only in a minority of patients', C W LeBaron and D W Taylor, 'Typhoid fever', in Kenneth F Kiple (ed.), *The Cambridge world history of human disease*, Cambridge University Press, 1993, pp. 1071–6, p. 1073.

[26] Information in this paragraph has been garnered from a wide range of English writings on the disease dating from these years. The very familiarity of the typhoid problem meant that the disease did not receive individual monograph treatment in England at this period, in contrast with the situation in the United States. Thus in 1906, the editor of the *British Medical Journal* responded to a request from "Ajax" for a book dealing exhaustively with the etiology of typhoid, by saying that he did not know of one: *Br. med. J.*, 1906, ii: 667. For an outline of late Victorian ideas on typhoid aetiology see W H Broadbent, 'Typhoid fever', in Richard Quain (ed.), *A dictionary of medicine*, 2nd ed., London, Longmans, Green and Co, 1894, vol. 2, pp. 1993–5; *idem*, 'Typhoid fever', in H Montague Murray (ed.), *Quain's dictionary of medicine*, London, Longmans, Green, 1902, pp. 1764–5.

[27] Robert Bruce Low (1846–1922), in general practice in Yorkshire, 1868–87. In 1879 he obtained the Cambridge DPH and joined the Department in 1887, becoming Assistant Medical Officer in 1900. Theodore Thomson (1859–1916), qualified DPH at Cambridge in 1888. From 1888 to 1891 he was Medical Officer of Health for Aberdeen and then for Sheffield; he joined the Department in 1891. George Seaton Buchanan ((1869–1936), the elder son of George Buchanan, LGB Medical Officer 1880–1892, joined the Department in 1895, two years after qualifying MD, and became a senior medical officer in the new Ministry of Health in 1919. After the war, his interest in epidemiology waned as he became a dedicated promoter of co-operation in international health.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

Department's acknowledged statistical wizard and "head of intelligence", was thereafter deputed to the study of the cholera pandemic, which then absorbed his energies in the years up to his untimely death in 1897.

Typhoid in Teesdale, 1890–1

Barry's Teesdale inquiry set the pattern for those that followed, however, and well illustrates the style in which these investigations were conducted. Although Barry's attention had been directed to the registration districts of Darlington, Stockton and Middlesborough, he extended the scope of his inquiry to a further seven registration districts in order to establish the range and limitations of the epidemic. Using information collected under the Infectious Diseases Notification Act and from local practitioners, he was able to obtain what he regarded as a pretty accurate account of the number of cases of typhoid, and his data established the same epidemic pattern as the GRO's cause of death data.[28] The statistical analysis was conducted at three levels: the registration districts; the thirty-two smaller sanitary districts comprised in them; and the municipal wards of the three boroughs. Although the epidemic had manifested itself in two six-week waves, Barry followed his case and mortality data through from January 1890 to the end of March 1891.

At each level, Barry's analysis demonstrated that something very untoward was happening. Across the whole period January 1890 to March 1891, 89 per cent of the registration district typhoid cases had occurred in Stockton, Darlington and Middlesborough. Of the attacks occurring within the three month total of the two epidemic periods, 91 per cent occurred in the same three districts. As Barry noted—it is possible to detect a note of indignation—during the whole sixteen month period, the typhoid attack-rate in Stockton, Darlington and Middlesborough, "collectively and severally . . . outrageously exceeded" that in the remaining seven registration districts. A similar exercise performed on the sanitary district data simply refined and reinforced this observation,[29] while the distribution of the disease in the municipal wards of the three towns showed a universality of incidence, despite great local differences in housing density and population, that pointed very strongly to the operation of some common cause.

Having established the likelihood of a common cause, Barry proceeded to a painstaking analysis of the various possible culprits. The milk supply he was able to dismiss at once as a possible vehicle of infection: the number of towns and districts involved indicated the existence of several hundred sources of supply and no possible community of cause. For the rest, he took the ten affected sanitary districts, which included both urban and rural areas, and gave a sober account of the sanitary circumstances of each. In general, these were very similar. Some three-quarters of the housing in the towns was working class (i.e. rated at under £10 per annum). The towns were sewered (this did not mean that water closets were

[28] Supplement to the MOAR LGB, PP 1893–94, xlii, pp. 275–528: F W Barry, *Report to the Local Government Board on the epidemic prevalence of enteric fever during 1890–91 in certain sanitary districts situate in the valley of the river Tees in south Durham and north Yorkshire*, pp. 275, 282–3, 287.

[29] 10/32 sanitary districts had 89.5% typhoid cases in 16 months; 92% in the 12 epidemic weeks: ibid., p. 299.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

provided) and piped water was supplied to houses throughout, and to most of the rural districts. In both town and countryside, the "privy midden" in one form or another constituted the main form of conservancy, and while corporate and private efficiency in emptying and cleansing these varied, the nightsoil was everywhere disposed of to local farmers who used it to fertilize their fields. Considering this information, and noting that the local incidence of the disease everywhere was quite independent of the type of housing or its conservancy status, Barry concluded that the water supply furnished the only common circumstance that could explain the typhoid outbreak. Both the Darlington Corporation Waterworks and the Stockton and Middlesborough Water Board drew their water from the Tees, and their works lay side by side on the river at Tees Cottage, two miles west of Darlington. Indeed, working with figures provided by the Water Boards, Barry was able to show that in the entire twelve week epidemic period, the typhoid attack-rate per 10,000 population was 5 among Tees-water drinkers; among non-Tees users it was 1. In the first epidemic wave the rates were 11 and 1 respectively; in the second, 28 and 1.[30]

The arrangements at the waterworks offered little clue, however, so Barry pursued his way upstream from Tees Cottage to see if he could track down any unusual source of pollution of the river at about the time of the two epidemics. As he went upstream, his horror mounted, for the deeper he penetrated into Teesdale, the more numerous and appalling were the sources of pollution he discovered. Finally, arriving at Barnard Castle, a small market and resort town some seventeen miles upstream, he discovered that not only did the town's main sewer discharge directly into the river below the town, but that numerous sewers and domestic privies discharged their contents directly over the river at the level of the town itself and deposited their contents on the river foreshore. From time to time the river flooded and carried the filth away, but at the time of Barry's visit, in December 1890, the accumulation was considerable. "In the whole course of my experience as an inspector", he recorded, "I had not before encountered, in comparatively small compass, such a mass of stinking abominations as was in existence at the time of my visit on the Barnard Castle foreshore".[31]

As Barry proceeded with his investigations, it became clear that the foreshore was a new phenomenon at Barnard Castle. A weir had formerly stood across the river at the lower end of the town, and above this the river bed had always been covered by water, so that sewer and privy discharges were immediately carried away by the river. But during the winter of 1888 a flood had breached the weir, which was then gradually swept away until, when Barry visited it, only a few stones remained. Following the disappearance of the weir, the foreshore appeared, and it was only at time of flood that it was relieved of its insanitary burden. As Barry concluded firmly,

> There can be no doubt at all that a vast amount of excremental filth from one and another source is being continually poured into the river Tees; or that the amount of polluting matter so carried down is enormously increased during periods of heavy rainfall, when the river is in flood.

The Barnard Castle rainfall records revealed that the river had been seriously flooded on 13 August and again on 1 December 1890. If allowance was made for the incubation period of typhoid (which in water-borne outbreaks is typically between 14 and 30 days),

[30] Ibid., Tables xxvii, xxviii, pp. 355–7.

[31] Ibid., p. 423.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

then these floods exactly paralleled the outbreak of the disease in all the districts supplied with Tees water on 7 September and 28 December respectively.[32]

Typhoid in the English Provinces

Barry's account of typhoid in Teesdale revealed several features of local typhoid ecology which reappeared in subsequent investigations. One was the regular gross pollution of local water sources, whether rivers or gathering grounds, with faecal matter; another was the insularity of local communities, who considered their own sanitary convenience without thought to the consequences for others. Meteorological conditions—that is, heavy rainfall—were often instrumental in precipitating epidemic outbreaks which had been "waiting to happen"; while the alteration of some local feature (like the weir at Barnard Castle) could have significant consequences for the local typhoid ecology. Of the eighteen typhoid investigations published in the 1890s, all but four directly implicated local water sources. Of these, polluted rivers and gathering grounds were responsible in ten instances; and a polluted well in one other. In two cases supplies were infected by accidental environmental circumstances—the raising of a dam at Wycombe Marsh, and the sinking of a new deep well-shaft through polluted subsoil at Worthing.[33] In two of the four cases where water was not directly implicated, it played an indirect part—at Chichester, and in the contamination of oyster-beds in Essex and Suffolk.[34] At High Wycombe and the Manchester satellite towns of Swinton and Pendlebury, a great many unwholesome conditions were found, but none to which specific blame could be attached.[35]

These different investigations threw up many various pieces of information which gave both epidemiologists and public health officials pause for thought. Over most of the north and east of England, for example, the practice of selling "town manure" to farmers was widespread, and had become more so with the agricultural depression of the 1880s, since nightsoil was cheaper than artificial fertilisers.[36] Agricultural land along the course of the Trent, the third longest river in England, received nightsoil from the towns of Huddersfield, Sheffield, Lincoln and Nottingham: Nottingham alone, where the pail system of conservancy operated, had to dispose of more than 1,000 tons of nightsoil a week in the early 1890s.[37] Local authorities, from parish committees to town councils, were frightened of the financial implications of new sanitary works, and even where they

[32] Ibid., p. 425–6.

[33] See Theodore Thomson, 'An epidemic of enteric fever in the borough of Worthing and the villages of Broadwater and West Tarring', MOAR LGB, PP 1894, xl, Appendix A no. 6, pp. 449–526; and G S Buchanan, 'On an outbreak of enteric fever in the village of Wycombe Marsh', MOAR LGB, PP 1896 xxxvii, Appendix A no. 7, pp. 379–90.

[34] For Chichester, see below; see also G S Buchanan, 'The occurrence of certain cases of enteric fever in six sanitary districts of Essex and Suffolk, and upon oysters in relation thereto', MOAR LGB, PP 1898, xl, Appendix A no. 6, pp. 73–91.

[35] See S W Wheaton, 'The sanitary condition of the borough of Chipping Wycombe', MOAR LGB, PP 1896, xxxvii, Appendix A no. 6, pp. 375–8; Theodore Thomson, 'Report on the persistence of enteric fever in the Swinton and Pendlebury Urban District', MOAR LGB, PP 1899, xxxviii, Appendix A no. 14, pp. 217–31.

[36] R Bruce Low, 'The circumstances of the river Trent in Lincolnshire and part of Nottinghamshire', MOAR LGB, PP 1894, xl, Appendix A no. 9, pp. 551–600, see p. 553.

[37] Ibid., p. 552.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

were willing to undertake them, might be prevented from doing so by their ratepayers: at King's Lynn, for example, a proposal to pipe water from local springs rather than draw it from the Gaywood river, was repeatedly defeated by ratepayer action between 1884 and 1894.[38] Even where local people were willing to take action, they often found that they had no powers to remedy pollution at source, especially where water was drawn from catchment areas.[39] And where the control of local epidemics was concerned, most rural areas and small provincial towns did not enforce the notification of infectious diseases, and even where they did, the local authorities either had no infectious disease hospital in which to isolate cases, or made inadequate provision: at Chichester, there were eight beds for a population of between 8,000 and 10,000.[40]

Circumstances such as these were of great and growing concern to the Medical Department, as it struggled to eliminate the potential for random typhoid outbreaks. Already in 1893, a hot dry summer had been followed by the widespread diffusion of typhoid, and a mortality unprecedented since 1884.[41] The disease became epidemic in towns all over the Midlands and the north—in Barnsley, Sheffield, York, Sunderland, Leicester, Grimsby, and Nottingham among others.[42] On a county basis, however, wide variations were found to exist. While the average typhoid death-rate for England and Wales was 226 per million living, Oxfordshire, Berkshire, Buckinghamshire and Wiltshire registered rates between 58 and 73, and London a modest 160. The highest rates were in Lancashire (320), the North Riding (326), Sussex (448: the figure was extraordinarily inflated by the Worthing outbreak), the East Riding (487) and Durham, which headed the list with a mortality of 565 per million. This pattern was later demonstrated to have a wider relevance. In 1896, as part of his investigation of typhoid in Chichester, H Timbrell Bulstrode prepared maps of typhoid distribution in England and Wales in the 1870s and 1880s. Thorne Thorne noted that despite the enormous reduction of deaths from the disease in the country as a whole the areas of both maximum and minimum incidence had remained practically the same over the twenty year period, and this despite the great improvements which had taken place in the sanitary circumstances of towns and villages across the country.[43]

As the figures stood, the typhoid death-rates for England and Wales had been 43 per 100,000 living from 1871 to 1880, and 22 from 1881 to 1890. But in the first decennium, rates in Durham, South Wales, the Yorkshire Ridings, Lancashire, Nottinghamshire and Northumberland had ranged from 73 down to 50, while at the bottom of the heap the combined counties of Surrey, Sussex and Dorset had a typhoid death-rate of no more than

[38] R Bruce Low, 'An outbreak of typhoid fever in King's Lynn', MOAR LGB, PP 1894, xxxix, Appendix A no. 8, pp. 67–82, see p. 80. See also Low, op. cit., note 36 above, pp. 574–9.

[39] MOAR LGB, PP 1899, xxxviii, p. 20. Legal niceties could defeat the best intentions. According to Thorne Thorne, this was especially the case "where statutory powers are acquired . . . by companies and other local bodies to collect and impound waters derived from gathering grounds the proper control of which, in so far as the wholesomeness of the water is concerned, has not been acquired by purchase or otherwise. In such instances both the purveyors and the consumers of the water are often alike helpless, even though the most superficial examination of the catchment area may give obvious indication of multiple sources of pollution of the gravest sort".

[40] H Timbrell Bulstrode, 'The prevalence of enteric fever in the city of Chichester', MOAR LGB, PP 1897, xxxvii, Appendix A no. 9, pp. 137–64, see pp. 137, 143.

[41] *Public Health*, 1894–95, **7**: 234.

[42] *Lancet*, 1893, **ii**: 1144.

[43] MOAR LGB, PP 1897, xxxvii, p. 13.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

25. From 1881 to 1890, the same eight counties took the top eight places: although the rankings had changed a little, Durham remained in first place. While still in the lower echelons, Surrey, Sussex and Dorset had been displaced by Rutland, Bedfordshire and Herefordshire.[44] Meanwhile it was becoming clear, by the mid 1890s, that the reduction in the national typhoid death-rate, which had been going on since the 1870s, was coming to a halt.[45] Much of the reduction, according to Thorne Thorne, had been associated with the improvement of water services; the Medical Department's investigations showed that much of the continuing persistence of the disease was associated with local habits of excrement disposal which resulted in organic pollution of the soil. In 1896, he called for the systematic study of local conditions which resulted in typhoid persistence in particular areas, and for more investigation of the practical and scientific aspects of soil pollution.[46]

Epidemiological Conundrums

It soon became clear, however, that the traditional type of local epidemiological investigation was not going to resolve the problem of typhoid endemicity. Two surveys in Chichester, conducted by Bulstrode in 1896 and by Thomson with Colonel J T Marsh of the Royal Engineers in 1898, strongly indicated the limitations of such inquiries. Chichester, a market and cathedral town, situated on a low-lying plateau between the South Downs and the sea in Sussex, was a notable exception to that county's otherwise admirable typhoid record. From the 1860s at least, the disease had been constantly present in the city, and at times it escalated into epidemics. The Registrar-General's records for the 1870s and 1880s showed that its death-rates from typhoid and continued fever exceeded those of any other district in Sussex in those two decades; indeed in the period 1881–90, only sixteen registration districts in England and Wales had suffered more typhoid than Chichester.[47] In sanitary terms, the town was in the process of modernization but, for all that, neither Bulstrode nor Thomson could satisfactorily account for its typhoid history, except in terms of gross pollution of the soil on which the city was built. A sewage system had recently been constructed, but privy cesspits and cesspools still abounded to the extent that Bulstrode remarked, "the whole of the gravel upon which Chichester is built is riddled with them".[48] Water was supplied partly from deep wells by the local water company, and partly by numerous private shallow wells scattered throughout the city. Neither wells, cesspits nor cesspools were water-tight, and were often close neighbours.[49] Bulstrode's initial conclusion was that the soil and subsoil of the city must be thoroughly polluted, and that until the cesspits and pools had been done away with, and their sites fully cleansed, there was no hope of eradicating typhoid.[50]

Meanwhile, some of the city's residents despaired. In 1897 they petitioned the Mayor to obtain an independent inquiry by "an experienced engineer" and a medical officer. In 1898 they petitioned again, this time directly to the LGB, calling urgently for an inquiry. "Not", they noted, "a formal inquiry, costing money and stirring up strife, but a walking [*sic*] inquiry by scientific experts accessible to all and anxious only to arrive at the

[44] Ibid., pp. 13–14.
[45] Ibid., p. 14.
[46] Ibid.
[47] Bulstrode, op. cit., note 40 above, pp. 144–6.
[48] Ibid., p. 141.
[49] Ibid., pp. 139–41.
[50] Ibid., p. 150.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

truth".[51] The LGB dispatched Theodore Thomson, with Colonel Marsh of the Royal Engineers. Thomson and Marsh found that considerable progress had been made on the sanitary front since 1896, but they could still establish no specific source of typhoid. They noted with interest the changing distribution of the disease in the city since 1870—between 1870 and 1884 all areas had suffered much the same, but since 1884 four areas outside the old city walls had borne the brunt of visitations—but could make no sense of it. Like Bulstrode, they felt that soil pollution offered a possible explanation, but they had to admit that it did not explain the distribution of the disease either by time or place within the city.[52] Indeed, the limitations of the type of inquiry they had conducted were clear to them. What was needed, they observed, was a careful comparison between local conditions in typhoid endemic areas and conditions in places that suffered little from the disease. Significantly, they added a rider: "Such investigations would need to be supplemented by skilled research on the part of the statistician, the geologist, the chemist, and the bacteriologist, and would entail prolonged and arduous labour in all their aspects".[53] While all these experts except the bacteriologists had, of course, been involved in public health since the mid-century, the emphasis on "skilled research" and "long and arduous labour" suggested a newly serious approach with broader dimensions than had been the case in the past. Epidemiological inquiry by doctors and engineers, they now recognized, could no longer be accepted as the best available service. The skills of the trained observer must be supplemented systematically from other relevant disciplines if the conundrum of typhoid was to be resolved.

Science at the Local Government Board

This awareness of the need for the greater integration of epidemiology with other scientific disciplines was reflected in the direction taken by the auxiliary scientific investigations in the 1890s. Here the sense of a new relationship between the two types of inquiry surfaced somewhat earlier. From mid-1892 onwards, as Buchanan's successor Thorne Thorne and the Department began to take in the implications of the inquiries at Teesdale, Rotherham and King's Lynn, the attention of the scientific division began to turn to the typhoid bacillus and associated micro-organisms. The annual report for 1892–93 contained two reports, one by Emanuel Klein on the etiology of typhoid, the other by Edmund Cautley into the micro-organisms to be found in the small intestine.[54] These reports were significant for the epidemiologists, because they turned on the disputed question of whether the typhoid bacillus was the true cause of typhoid, and whether it was

[51] Theodore Thomson and Col. J T Marsh, 'Enteric fever in the city of Chichester', MOAR LGB, PP 1900, xxxiv, Appendix A no. 7, pp. 52–88, on p. 52.

[52] Ibid, p. 75.

[53] Ibid.

[54] Emanuel Klein, 'On the etiology of typhoid fever', MOAR LGB, PP 1894, xxxix, Appendix B no. 1, pp. 345–66; Edmund Cautley, 'On micro-organisms found in the small intestine', ibid., Appendix B no. 4, pp. 413–18. Klein was the Department's principal micro-biologist, originally recruited by John Simon; Edmund Cautley (1864–1944) did not pursue his career in micro-biology but became a leading paediatrician, associated with the Belgrave Hospital for Children. By this date, although the subjects for scientific investigation were determined by the Medical Officer, it was Klein who allocated the various pieces of investigation among his students and co-workers: 'Edward Emanuel Klein', *Br. med. J.*, 1925, i: 388.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

a distinct organism or merely a sub-species of bacillus coli communis (E. coli), as various French bacteriologists had argued.[55]

Klein was able, by careful experiment, to establish that although the two bacilli had certain points in common they also exhibited well defined differences both morphologically and in culture, to a degree that justified their classification as separate species. B. coli was "shorter, less actively mobile", produced gas bubbles in shake culture, curdled milk, and gave the Indol-reaction. The typhoid bacillus, on the other hand, was longer and more active, it did not form gas bubbles in shake culture, or curdle milk, or give the Indol-reaction.[56] Furthermore, close observation by Klein of fifteen typical typhoid cases in the wards of St Bartholomew's Hospital (where he was director of the bacteriological department) showed that while large numbers of typhoid bacilli were to be found in the mesenteric glands and spleen during the second and third weeks of illness, and in tissues in pure culture, no B. coli were present in those organs.[57]"There is then the strongest presumption short of actual proof", stated Klein, "that this particular organism called the typhoid bacillus, which differs essentially from the bacillus coli, stands in an intimate relation to the disease enteric fever".[58] Cautley, similarly, reached the conclusion that the two bacilli were not identical, nor could one be developed from the other by artificial culture.[59]

These researches were important for the epidemiologists partly because they confirmed the plausibility of bacteriological assessment of water quality, and partly because they paved the way for further laboratory research into the behaviour of the typhoid bacillus in various natural media—in water, sewage, soil and foodstuffs—and so promised a better understanding of the often erratic behaviour of typhoid in the natural environment. These researches were not unproblematic, however, and at times threatened to overturn accepted epidemiological values, as well as to increase the sum of knowledge. In 1894, for example, Frederick Andrewes, a notable pathologist who had worked with Klein for some years on pyogenic bacteria, began to work on the behaviour of the typhoid bacillus and B. coli in sewage.[60] He found that when kept in "ordinary fluid sewage", the bacteria did not increase but actually diminished in numbers and eventually died out. These findings were corroborated by the work of J Parry Lawes; and Lawes and Andrewes then submitted their findings as reports to the London County Council.[61] The findings seemed significant, for they appeared to indicate that the danger of contracting typhoid from sewage polluted water, axiomatic among epidemiologists since the days of William Budd, was at least questionable.

On a wider canvas, the significance of these findings was diminished by the enormous range of results on the survivability of typhoid bacteria in different media recorded by different researchers, but this action may have led to a breach between Klein and Andrewes, for the latter did not publish further reports for the LGB in the 1890s. Klein,

[55] Emanuel Klein, 'The etiology of typhoid', MOAR LGB, PP 1894, xl, Appendix B no. 1, pp. 345–66, see pp. 345–7.
[56] Ibid., p. 350.
[57] Ibid., pp. 356–7.
[58] Ibid., p. 357.
[59] Cautley, op. cit., note 54 above, p. 418.
[60] Sir Frederick Andrewes (1859–1932) made his career in pathology at St Bartholomew's Hospital, where Klein was employed at the Medical School: see 'Sir Frederick William Andrewes', *Obituary notices of Fellows of the Royal Society*, **1**: 37–44.
[61] J P Lawes and F W Andrewes, *Report on the result of investigations on the micro-organisms of sewage*, London County Council, 1894.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

although he confirmed the observation personally, had little trouble in disposing of its supposed significance. "It has to be borne in mind", he observed, "that the vitality of the typhoid bacillus in sewage may not be parallel to its vitality in sewage *plus* water of one sort or another".[62] He proceeded to add nitrates to his experimental sewage, "as representing for experimental purposes salts to be found alike in sewage polluted drinking water and in soil through which sewage matters percolate", and discovered by careful measurement that typhoid bacilli showed "incomparably greater" vitality in "nitrate sewage" than in "pure sewage". In the former they distinctly increased in numbers, in the latter they decreased. For good measure Klein also checked the behaviour of the cholera bacillus in similar circumstances and found that it rather preferred pure sewage to nitrate sewage.[63]

Having thus disposed of the challenge to accepted epidemiological orthodoxy, Klein moved on to expose the inadequate methods of scientists who disputed the ability of pathogenic bacteria like the typhoid bacillus to survive in water. Percy Frankland, for example, son of the distinguished analytical chemist Edward Frankland, claimed to have been unable to find either bacillus coli or the typhoid bacillus in samples of unfiltered water drawn from the Thames at Hampton.[64] Robert Koch, too, as noted earlier, was sceptical of claims that the typhoid bacillus had been found in drinking water. Klein, who in 1893 had demonstrated the presence of typhoid bacilli in the well-shaft at Worthing, proceeded to detail his methods, demonstrating by a series of elegant experiments that pathogenic microbes could be demonstrated in water only if a sufficiently large sample was taken for analysis. Percy Frankland commonly took samples of only 0.5–1 cc for examination; Klein found that in samples of between 1000 and 2000 cc an abundance of both B. coli and B. typhosus could be demonstrated in Hampton water.[65]

Thus far, the work of Klein and his collaborators had essentially been directed to elucidating the scientific reality behind epidemiological phenomena already established by observation in previous decades. Increasingly, however, from the mid-1890s, the laboratory investigations focused on the current problems being experienced by the field epidemiologists. In particular, the behaviour of the typhoid bacillus in soil—a question with clear relevance to the agricultural use of nightsoil in the North, and to the problems of cities like Chichester—became an important activity. Between 1895 and 1900, the pathologist Sidney Martin, a specialist in the physiology and pathology of digestion,[66] produced a series of reports on this subject, while Alexander Houston, who was to become, in Walter Fletcher's words, a "big biological engineer",[67] applied some of his energies to the bacteriological and chemical examination of soils, with special reference to the amount and nature of organic matter and the number and character of bacteria

[62] Emanuel Klein, 'The behaviour of the bacillus of enteric fever and of Koch's vibrio in sewage', MOAR LGB, PP 1895, li, Appendix B no. 2, pp. 855–88, on pp. 886–7.

[63] Ibid.

[64] See Emanuel Klein, 'The ability of certain pathogenic microbes to maintain existence in water', MOAR LGB, PP 1895, li, Appendix B no. 3, pp. 889–906, on p. 906. For the wider context of Frankland's work, see Hamlin, op. cit., note 7 above, pp. 250–64, 273–7, 302.

[65] Ibid.

[66] Sidney Martin FRS (1860–1924), Professor of Pathology at University College Hospital, see *Lancet*, 1924, **ii**: 680.

[67] Sir Alexander Cruickshank Houston (1865–1933), Director of Water Examination for the Metropolitan Board of Works from 1905. Fletcher's phrase is cited in 'Sir Alexander Cruickshank Houston', *Obituary notices of Fellows of the Royal Society*, **I**, p. 343.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

contained in soil "washings"—the thin layers of topsoil removed by natural drainage processes during heavy rainfall.[68] In 1899, Houston performed a chemical and biological analysis of soils from the so-called "fever" and "non-fever" areas of Chichester: an investigation which proved thoroughly inconclusive in that no discernable difference between the two could be found.[69] Yet again, bacteriology had failed to provide the answer to a specific epidemiological problem.

By the turn of the century, however, the auxiliary scientific investigations were beginning to suggest that soil pollution was not as serious an element in typhoid endemicity as the field epidemiologists had tentatively concluded. In 1900, Sidney Martin ended his series of reports with an investigation into the "nature of the antagonism of the soil to typhoid bacilli", in which he deduced that the bacillus commonly had only a short life in soil and was destroyed by the products of the putrefactive bacteria which exist in most cultivated soils.[70] A year later, Houston, who had been experimentally treating soil with sewage, reached much the same conclusion, although he warned that, "extreme caution must be exercised in distinguishing between the surface and deeper layers of soil and between the *relative* and the *actual* death of bacteria".[71] The laboratory scientists' failure to confirm the field observers' theory of last resort on typhoid endemicity suggests again the delicate balance of expertise within the Medical Department. Bacteriology could not confirm epidemiological theory, but neither would it conclusively deny it—and there was no comfort in theories which were open to such tentative assessments. As the case of typhoid showed, epidemiological theory was henceforth to be qualified by the laboratory's assessment of scientific plausibility. The field epidemiologists were themselves aware of this, as Theodore Thomson's call for interdisciplinary studies of endemic typhoid showed.[72]

At a deeper level, however, the realization of the need for co-operation between epidemiology and bacteriology masked a growing uneasiness among the epidemiologists. Already in 1895, in the context of a parallel series of investigations into the relationship between typhoid incidence and oyster consumption, Thorne Thorne had noted that epidemiological evidence which previously would have established a case, was now subject to confirmation or modification by bacteriology.[73] He went on to stake out the older discipline's corner.

[68] Sidney Martin, 'Preliminary report on the growth of typhoid bacilli in the soil', MOAR LGB, PP 1897, xxxvii, Appendix B no. 2, pp. 291–302; *idem*, 'Growth of typhoid bacillus in soil', PP 1898, xl, Appendix B no. 3, pp. 308–418; *idem*, 'Growth of typhoid bacillus in soil', PP 1899, xxxviii, Appendix B no. 3, pp. 460–90; *idem*, 'Growth of typhoid bacillus in soil', PP 1900, xxxiv, Appendix B no. 5, pp. 525–48; A C Houston, 'The chemical and bacteriological examination of "washings" of soils', MOAR LGB, PP 1899, xxxvii, Appendix B no. 5, pp. 525–75; *idem*, 'The chemical and bacteriological examination of samples of soil obtained from the "fever" and "non-fever" areas of Chichester', PP 1900, xxxiv, Appendix B no. 6, pp. 549–73.

[69] A C Houston, 'The chemical and bacteriological examination of samples of soil obtained from the "fever" and "non-fever" areas of Chichester', MOAR LGB, PP 1900, xxxiv, Appendix B no. 6, pp. 549–73.

[70] Sidney Martin, 'The nature of the antagonism of the soil to the typhoid bacillus', MOAR LGB, PP 1901, xxvi, Appendix B no. 5, pp. 487–610, on p. 510.

[71] A C Houston, 'The inoculation of soil with sewage', MOAR LGB, PP 1902, xxxvii, Appendix B no. 7, pp. 939–75, on p. 975.

[72] It can be noted that William Hamer wrote retrospectively that epidemiology and bacteriology seemed completely to have lost touch "at the end of the last century", see Hamer, op. cit., note 6 above, p. 14. However, the type of epidemiology being practised at the LCC in the 1890s differed from that at the LGB.

[73] For the background to these oyster investigations see John M Eyler, *Sir Arthur Newsholme and state medicine, 1885–1935*, Cambridge University Press, 1997, pp. 119–27; Robert Neild, *The English, the French and the oyster*, London, Quiller Press, 1995, pp. 105–17.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

The bacteriologist can . . . tell us of impurity and hazard—often, indeed, of the precise nature of specific hazard—but not of purity and safety. For information about these we must go, with the aid of what the bacteriologist has been able to teach us, in search of the conditions surrounding and affecting the storage and culture of oysters along our coasts.

In other words, the specifically epidemiological perspective must not be abandoned. Observation—"the accumulation of observed facts as to the conditions under which epidemic and preventable diseases occurred"—had, he declared, provided the evidence which had shaped the progress of preventive medicine in the past and on such evidence "our future measures of prevention must necessarily be based".[74]

The challenge from the bacteriologists was not overt, but some bacteriologists at least clearly perceived a significant shift in balance between the two disciplines. Alexander Houston, seven years younger than Percy Frankland, was to become Britain's foremost expert on water purification. After medical training at Edinburgh, culminating in the DSc in Public Health in 1892, Houston's first project was an investigation of water from moorland gathering grounds in the North of England, for the Local Government Board. His entire career was spent in research work related to water supplies, and after about 1900 a large part of his work was devoted to "perfecting the analysis of water by the application of bacteriology on thoroughly scientific lines".[75] Concluding a chemical and bacteriological investigation of the Chichester well waters, and finding them distinctly contaminated with B. coli, Houston staked a vigorous claim to equality with the traditional investigators:

Epidemiologists in the past have shrunk from accepting too readily the unsupported testimony of bacteriologists . . . Nor is this on the whole to be wondered at. Yet when the modern methods of responsible bacteriologists of *measuring* the probable degree of potential danger to health by means of *quantitative* and *qualitative* biological tests of proved efficiency are considered, the scepticism of the epidemiologist is less reasonable than in the past. It requires no seer to prophesy that the future progress of preventive medicine . . . lies in a happy recognition of the claims not only of the epidemiologist but also of the bacteriologist . . . [76]

Houston was speaking of water supplies and water-borne disease, but, given the rate at which the causal organisms of all kinds of diseases were being identified in the 1890s, it needed no leap of the imagination to extend the claims of laboratory science on the practice of epidemiology almost indefinitely. Indeed, it seems that certain interest groups also had begun to perceive in bacteriology a useful counter-balance to epidemiology. By 1902, oyster growers in Hampshire were seeking to escape epidemiological conclusions by calling for additional bacteriological investigations. Bulstrode, who was in charge of this investigation, refused in words eloquent of the determination to establish the independent credentials of epidemiology. "No negative testimony, either bacteriological

[74] 'Oyster culture in relation to disease', Supplement to the 24th MOAR LGB, PP 1896, xxxvii, introduction, pp. 18–26, p. 26. Thorne was deliberately paraphrasing the words of his predecessor, George Buchanan, on the relations between chemistry and epidemiology, in 1882.

[75] 'Houston', op. cit. note 67 above, p. 337.

[76] A C Houston, 'The chemical and bacteriological examination of Chichester well-waters', MOAR LGB, PP 1902, xxxvii, Appendix B no. 7, pp 978–1033, on p. 1027. For the research substantiating Houston's claims see 'Houston', op. cit., note 67 above, pp. 377–8.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

or chemical, would undo the fact that the oysters were laid down within a few yards of the main sewer. Moreover a positive result would in either case be superfluous." And, he added: "This attitude I adopted because I considered that if this outbreak of enteric fever had been caused by the . . . oysters, the science of epidemiology should be competent to demonstrate the fact".[77]

Conclusion

The publications of the Medical Department of the Local Government Board in the closing years of the nineteenth century provide a notable picture of the methods of Victorian epidemiology, of its limitations and of the opportunities these offered to bacteriology to enter into a new working partnership with the field observers, in attempting to resolve problems and in testing the probabilities of epidemiological theory. Thirty years after John Simon had first envisaged the Medical Department contributing to the establishment of new principles for the prevention of disease through abstract scientific research,[78] a closer, if uneasy, relationship between science and prevention was becoming established. The epidemiologists might doubt the utility of bacteriology in respect of water supplies, but they often accepted the need for chemical and bacteriological tests in their investigations. The development of this relationship was, however, already coloured by a growing competition between the two disciplines, in which the practitioners of traditional epidemiology explicitly resisted the encroachment of bacteriology on their established professional authority. This direct competition was to be relatively brief: after 1905 the challenge to traditional English epidemiology was broadened and diversified both by the discovery of healthy human carriers, which reinforced the authority of bacteriology, and by the entry into epidemiological territory of several advocates of new and more sophisticated statistical methods.[79]

The wider context of this disciplinary conflict between epidemiology and bacteriology merits some attention. There are preventive ironies in the continuing water-borne typhoid outbreaks, despite water supplies which had apparently met currently accepted standards, and in the growing suspicion that Edwin Chadwick's greatest dream, of efficiently and economically re-cycling human sewage as farming manure,[80] had proved the vehicle for spreading typhoid fever. Yet the Medical Department's reports reveal not only the changing ideologies of public health and preventive medicine, but also the continuities of epidemiological concern in the period up to 1914. The anxieties over the stagnation of the typhoid death-rates and the persistence of long-term geographical variations in the

[77] H Timbrell Bulstrode, 'Report upon alleged oyster-borne enteric fever and other illness following the Mayoral banquets at Winchester and Southampton', MOAR LGB, PP 1904, xxvi, Appendix A no. 9, pp. 129–89, on p. 151.

[78] Lambert, op. cit., note 11 above, p. 400.

[79] For the impact of the discovery of the carrier in the United States, see Judith Waltzer Leavitt, '"Typhoid Mary" fights back. Bacteriological theory and practice in early twentieth-century public health', *Isis*, 1992, **83**: 608–29; *idem*, *Typhoid Mary: captive to the public's health*, Boston, Beacon Press, 1996. The statistical practitioners in question were Ronald Ross, John Brownlee and Major Greenwood. Greenwood's application of Pearsonian methods led eventually to a profound alteration in the methods and practice of epidemiology.

[80] For Chadwick's proposals see E Chadwick, *Sewage manure*, London, Reynell and Weight, 1849. For the wider relevance of such ideas in Victorian society see Christopher Hamlin, 'Providence and putrefaction: Victorian sanitarians and the natural theology of health and disease', *Vic. Stud.*, 1985, **28**: 381–411, especially pp. 392–400.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press

distribution of high and low mortality prompt speculation about the real reasons for these patterns, and the regional conditions that determined them. The history of typhoid in Teesdale and along the valley of the Trent, for example, raises questions about the relationship between Victorian cities and their hinterlands, and the exchanges of disease between them. The Victorians tended to think that the industrial towns and the great cities bred their own evils, and historians often incline to the same opinion. But in Teesdale, and in many other areas, the epidemiological investigations of the 1890s revealed the health of the towns as hinging at times on what went on in the sanitarily unregenerate rural communities around them. Thus the various pieces of legislation, dating from the 1870s, which were intended to prevent the gross pollution of watercourses, and to ensure the provision of wholesome water supplies in rural areas, were widely ignored, or even flouted.[81] Even a cursory survey of the Local Government Board investigations into typhoid in the years before 1914 extends and reinforces Christopher Hamlin's observation that urban improvement long remained a matter of staggering complexity; in many towns efforts at sanitary reform were hindered by financial considerations, by antagonistic ratepayers, or by inadequate legal powers, and were always vulnerable at times to being undercut by events or conditions beyond their control.[82] The epidemiological inquiries into typhoid outbreaks in the 1890s made anxious reading for practitioners of public health.

[81] See Public Health Act 1875, 38 & 39 Vict c 55 sections 17, 69; Rivers Pollution Prevention Act 1876, 39 & 40 Vict c 75, part 1; Public Health (Water) Act 1878, 41 & 42 Vict c 25, section 3.

[82] Christopher Hamlin, 'Muddling in Bumbledom: on the enormity of sanitary improvements in four British towns, 1855–1885', *Vic. Stud.*, 1988, **32**: 55–83, pp. 82–3.

https://doi.org/10.1017/S0025727300064012 Published online by Cambridge University Press