

Commentary: Behind the Broad Street pump: aetiology, epidemiology and prevention of cholera in mid-19th century Britain

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Introductory epidemiology text books and courses generally contain little epidemiological history, but an exception is made for the story of John Snow, the water-borne transmission of cholera, and the handle of the Broad Street pump.^{1–5} Snow's 1855 book, *On The Mode Of Communication Of Cholera*,⁶ is indeed a beautiful demonstration of 'the epidemiological imagination'⁷ in action, and continues to provide example and inspiration to people entering the discipline. However, it appeared amidst a veritable spate of speculation, experiment, investigation and recommendations regarding cholera, and some of these less celebrated (at least now) contributions remain instructive. Therefore, in the current issue of the *International Journal of Epidemiology* we reprint a section of Dr John Sutherland's report for the General Board of Health on the 1848–1849 British cholera epidemic (Figure 1), together with a series of commentaries.^{8–10} The extracts from Sutherland's report include his investigation of the effect of water source on cholera risk in Salford, Manchester, which was briefly referred to by Snow⁶ and has occasionally been recognized as a seminal investigation.^{11,12} The discussion by Sutherland of the implications of his findings are clearly at variance with those of Snow, who more strongly emphasized the necessary transmissible element in generating cholera (and thus in triggering epidemics), but Sutherland's utilization of quantitative data is striking.

As with (virtually) all scientific advances, Snow's work did not emerge from a vacuum, and this background has been explored from various perspectives.^{13–23} The proto-epidemiological approaches to cholera in the mid-19th century have continuing implications for epidemiological theory and practice, and this does not only apply to the investigations now seen to have contributed to us reaching the correct conclusions. The efforts of many of Snow's predecessors and contemporaries were seen, at the time, as of at least (and often greater) importance than those of Snow.^{13,14,24,25} The contributions of those who are now excluded from potted histories of epidemiology are certainly worth revisiting.

Epidemiology and prevention of cholera before 1848

Cholera first reached Britain from continental Europe in October 1831 and during the subsequent year resulted in over 30 000 deaths.²⁶ The disease had probably been endemic in India²⁷ and from 1817 onwards spread inexorably towards

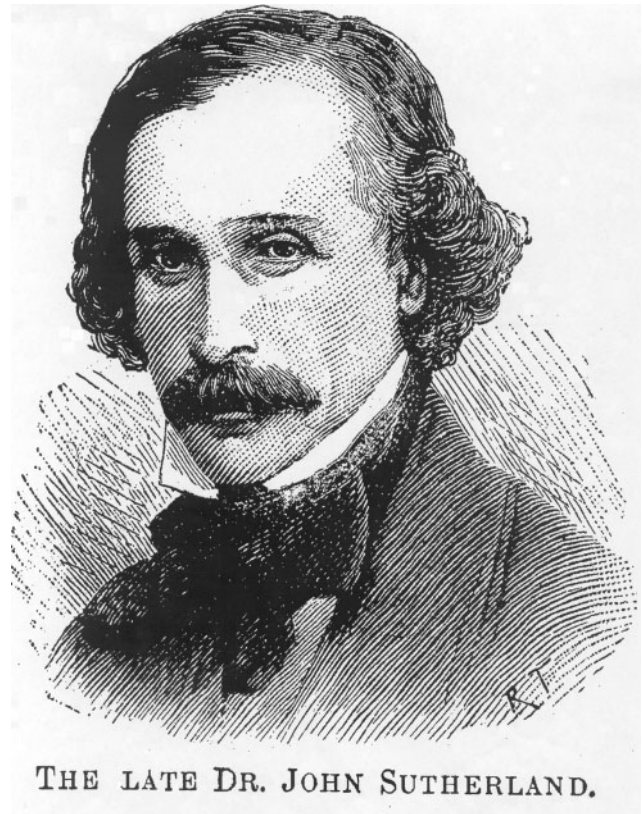


Figure 1

Britain. The *Lancet* devoted 44 pages of its 19 November 1831 issue to the arrival of cholera in Britain.²⁷ Regarding the origin of cholera, the *Lancet* concluded that:

We can only suppose the existence of a poison which progresses independently of the wind, of the soil, of all conditions of the air, and of the barrier of the sea; in short, *one that makes mankind the chief agent for its dissemination*.²⁷ (Box 1)

Others pointed out that cholera in India followed the paths of rivers. After indicating that they were using contagion and infection synonymously (because some authorities used these terms to designate distinct transmission modes¹³), the *Lancet* also discussed evidence against the contagious nature of cholera, but they dismissed this and advocated quarantine and sanitary measures. The viewpoint of the *Lancet* was not one

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Box 1

PROOFS OF COMMUNICATION OF CHOLERA BY MAN

We shall classify our proofs of communication under the following heads of:

1. The coincidence between the irruption of the disease in previously-uninfected places, with the arrival of ships, of caravans of fugitives or pilgrims, of individuals, and with the progress of armies.
2. Examples of the coincidence between the occurrence of cholera in individuals, and their contact with others actually labouring under the disease.
3. Examples of immunity afforded by seclusion in the midst of an unhealthy district.

The *Lancet*, 19 November 1831.²⁷

widely shared in the medical and related professions, and a variety of non-contagious aetiologies—generally glossed as ‘miasma theories’—were advanced.^{13,26,27} Several studies of local outbreaks were carried out, and while some hinted at both contagion and transmission by water, most of published professional opinion remained that the disease was non-contagious and arose through miasmatic processes.^{26,28}

In the US, where cholera arrived in 1832, a similar picture emerged.²⁹ Charles Rosenberg surveyed the published views of 109 physicians between 1832 and 1834 and found that the large majority did not consider the disease to be at all contagious and only 5 considered it to be primarily contagious.²⁹ The public were, however, little influenced by the views of these physicians and there was a strong sense that the disease was indeed contagious. This was illustrated in several ways: local residents mounted considerable opposition to the building of cholera hospitals near where they lived; people believed to be carrying the cholera ‘poison’ were attacked; and wealthy New Yorkers left the city to avoid the disease, or bought in water from outside the city.²⁹ Furthermore, quarantine measures were imposed in both the US and Britain along with recommendations for improved hygiene, including the avoidance of raw vegetables and fruits, in tandem with traditional approaches to miasmas—such as the burning of tar and pitch to purify the atmosphere.

During the outbreak of cholera in Exeter in 1832 the chairman of the local Board of Health (who was also the Mayor of Exeter) released a hand bill in which cholera was referred to as a contagious disorder and quarantine regulations were announced. Dr Thomas Shapter, who saw the first case of the outbreak and who in 1849 published *The History of the Cholera in Exeter in 1832*³⁰ (Figure 2), stated that many medical men thought the disease was ‘solely propagated from man to man by the communication of a “materies morbi”, of which we also neither know the nature nor the medium’. Shapter, himself, considered cholera ‘essentially an epidemic, originating in, and chiefly due to, aerial influences, but capable, under peculiar and rare conditions, of being transmitted from man to man’. Shapter included a detailed map showing all the cases of cholera in Exeter in 1832 and John Snow obtained from him additional information regarding sewers and water supply, which he took to provide evidence of the water-borne nature of cholera.⁶

THE HISTORY
OF THE
CHOLERA IN EXETER

1832.

BY
THOMAS SHAPTER, M.D.,
PHYSICIAN TO THE DEVON AND EXETER HOSPITAL; THE ST. THOMAS'
HOSPITAL, NEAR EXETER, FOR LUNATICS; THE LYING-IN-
CHARITY, &c. &c.

LONDON:
JOHN CHURCHILL, PRINCES STREET, SOHO.
EXETER: ADAM HOLDEN, HIGH STREET.
MDCCLXXXIX.

Figure 2 Front piece of Thomas Shapter's *The History of the Cholera in Exeter in 1832*

In the 1830s epidemiological and public health approaches to cholera were being developed in the context of some understanding of the contagious nature of some diseases, in particular smallpox and syphilis, but with little agreed differentiation of the fevers. Benjamin Rush's late 18th century doctrine that ‘there was but one fever in the world’³¹ was widely shared. The ‘exciting factor’ for epidemic cholera was sometimes viewed as shaping existing fevers into its own image, and it was questioned whether the arrival of cholera actually coincided with an increase in mortality, or just transferred deaths between categories (although demonstrations of an increase in overall death rates proved problematic for this view).²⁹ The causes of fevers were often discussed in terms of predisposing and exciting (or localizing) causes.^{9,10,13,32} In general predisposing causes were factors such as inadequate diet, overwork, poverty, inadequate housing or ventilation, debilitation through alcohol and mental exhaustion. The exciting causes were those which drove the debilitation due to predisposing causes towards a particular pathological form, such as the specific atmospheric factors seen as leading to cholera outbreaks.

This formulation may look familiar to contemporary epidemiologists, in reflecting the triad of host, agent and environment. Host susceptibility—influenced by the predisposing causes—meets the agents (the exciting factors) under the constraints of the broader environment. The model clearly reflected a multifactorial theory of the origins of cholera, similar in some ways to the multifactorial models of conditions such as coronary

heart disease in contemporary epidemiology. However, acceptance of a predisposing/exciting causes distinction did not restrict an author's range of potential explanations. Thus for some it was possible that continued exposure to a set of predisposing causes could lead to accumulated damage, which finally resulted in disease, without the necessary intervention of a final exciting cause. For others, a specific exciting cause was a necessary trigger of disease. Further confusion followed from disagreements about what constituted predisposing or exciting causes. For example, in a later publication than the one we excerpt here, John Sutherland described how some commentators thought that water contained the specific poison of cholera, while some thought that water containing rotting organic matter served as a powerful predisposing cause of the pestilence, in the same way as the atmosphere or bad food could predispose to disease.³³ Others considered that the atmosphere at the time of epidemics contained the exciting cause. The flexibility of models of predisposing and exciting causes certainly meant that such formulations could accommodate virtually any pattern of observed data.

Methods of prevention advanced on the basis of an understanding of the predisposing causes of cholera involved a wide variety of (perhaps familiar) lifestyle advice, particularly focussing on avoiding alcohol (Box 2), together with advocacy for avoiding filth and squalor.

As Charles Rosenberg points out, the flexibility of the disease concept also meant that 'it was only natural that mental and moral factors should be presumed to play a role in its causation'.²⁹ As with peptic ulcer from the 1940s to the 1970s, and coronary heart disease today, what would currently be called stress was seen to be an important determinant of cholera, with the physiological consequences of such stress considered a major predisposing factor. Indeed many authorities followed William Beaumont in thinking that such factors underlay 'the greater proportional number of deaths in the cholera epidemics'.²⁹ Rather than our contemporary approaches to stress, however, prayer and an unconditional belief in God were prescribed for its control.²⁹ As the clergy of the city of Exeter explained in a hand bill, 'It has been observed that a calm and even temper, such as arises from a right trusting God, renders those who are blessed with it less liable to take infection.'³⁰

Box 2

Hand bill from the New York Board of Health, 1832²⁹

Notice

Be temperate in eating and drinking,
avoid crude *vegetables* and *fruits*;
abstain from *cold water*, when heated;
and above all from *ardent spirits* and

if habit have rendered it indispensable, take much less
than usual.

Sleep and clothe warm

Avoid labor in the heat of day.

Do not sleep or sit in a draught of air when heated.

Avoid getting wet

Take no medicines without advice

The cause and prevention of cholera, 1848–1857

Between 1832 and 1848 discussion of the causes and prevention of cholera subsided, in response to the retreat of the disease. An outbreak of 20 cases on the hospital ship 'Dreadnought' in October 1837 was investigated by George Budd (brother of William Budd) and George Busk,³⁴ who concluded aspects of the outbreak 'militate against the idea of its contagious nature'.³⁴ Busk was a keen microscopist and examined cholera evacuations on the Dreadnought with negative findings.¹³ George Budd was prompted by the 1837 outbreak to re-examine records of cholera on the Dreadnought during the 1832 epidemic. He concluded that the distribution and pattern of cases was 'very unfavourable to the supposition that the disease is contagious'.³⁵

The return of epidemic cholera to Britain in 1848 and the US in 1849 rekindled interest in the disease for obvious reasons, and also provided possibilities for testing aetiological theories. Initially the main evidence related to the apparent spread of cholera towards Europe and the Americas. Reviewing this in November 1848, SH Dickson wrote that his:

train of thought leads philosophically and by logical necessity, to the doctrine of infection—of contagious propagation. The same causes produce the same effects. From the choleric patient is derived the supply of that agent which affected him with the disease. *He* presents the only similar contingency, in his personal condition, which can be traced when we endeavour to connect effect with cause, and ask why cholera, which in January was in Moscow, is in September at Hamburg; which, thirty years ago ravaged Hindostan, and now threatens the crowded cities of England.³⁶

This conclusion came from careful reasoning about the patterns of cholera outbreaks, who was affected and who was spared during the epidemics, where and when epidemics occurred. It was not merely the repetition of dogma.

Although equally trenchant (and reasoned) opposing opinions appeared, other authors wrote along the same lines as Dickson, but the only publications that are now celebrated are the 1849 pamphlets by John Snow³⁷ and William Budd,³⁸ the latter appearing within a month of the former. Both contained physiological reasoning as to why the gastrointestinal tract was the likely port of entry and port of exit for the cholera agent, together with clinical observations and details of several outbreaks which could only plausibly be explained by the transmission of an infectious agent of disease. Snow reported crude death rates from cholera by London district, and drew attention to the higher rates in South and East London, which he attributed to differences in water supply. In discussing an outbreak in Albion Terrace he concluded that it:

is not here implied that all the cases in Albion Terrace were communicated by the water, but that far the greater portion of them were; that, in short, it was the circumstance of the cholera evacuations getting into the water which caused the disease to spread so much beyond its ordinary extent.³⁷

Budd discussed the first violent outbreak of cholera in Bristol, during which more than 40 people died in one small locality,

while in closely proximal areas there were no cases. He reasoned that any atmospheric element must have been shared between these places, whereas the water that was drunk was different, and if one supposed 'the poison to have been introduced into the bodies of the persons who took the disease, in the water they drank ... what was before a mystery is now clear as day'.³⁸ Budd's summary of his thinking regarding the cause of cholera is given in Box 3.

There are two main differences between the views of Snow and Budd. Firstly, although Snow thought that water was not implied in all the cases of cholera, he did not mention the possibility of transmission in the atmosphere, while Budd allowed for this as a secondary, but less important, mode of communication. Secondly, and more crucially, Budd considered that he had identified the agent of cholera—a fungus-like element in the water, which he and others detected microscopically in the water from Redcross Street, the site of the first Bristol outbreak (Figure 3). The fungus theory attracted considerable controversy¹³ and various other investigators—including George Busk, a colleague of William Budd's brother—failed to substantiate the original claims, which led to William Budd's work becoming discredited.¹³

Snow concluded his pamphlet by accepting that he had not produced sufficient evidence to support his thesis, but pleaded pressure of other work. Budd added a footnote to his pamphlet saying that Snow's 'ingenious pamphlet on cholera fell into my hands while these materials were preparing for publication' and that Snow, therefore, had priority. The generous acknowledgement, however, ended with the ambiguous statement that in Snow's work:

there is, besides, much that is so apt, and in such entire accordance with the truth, that the detection of the actual

Box 3

From *Malignant Cholera: Its Mode of Propagation and its Prevention*. By William Budd, MD, physician to the Bristol Infirmary³⁸

1. That the cause of malignant cholera is a living organism of distinct species.
2. That this organism—in shapes hereafter to be described—is taken by the act of swallowing into the intestinal canal, and there becomes infinitely multiplied by the self-propagation, which is characteristic of living beings.
3. That the presence and propagation of these organisms in the intestinal canal, and the action they there exert, are the cause of the peculiar flux which is characteristic of malignant cholera; and which, taken with its consequences, immediate and remote, constitutes the disease.
4. That the new organisms are developed only in the human intestine.
5. That these organisms are disseminated through society, (1) in the air, in the form of impalpable particles; (2) in contact with articles of food; and (3) and principally, in the drinking-water of infected places.

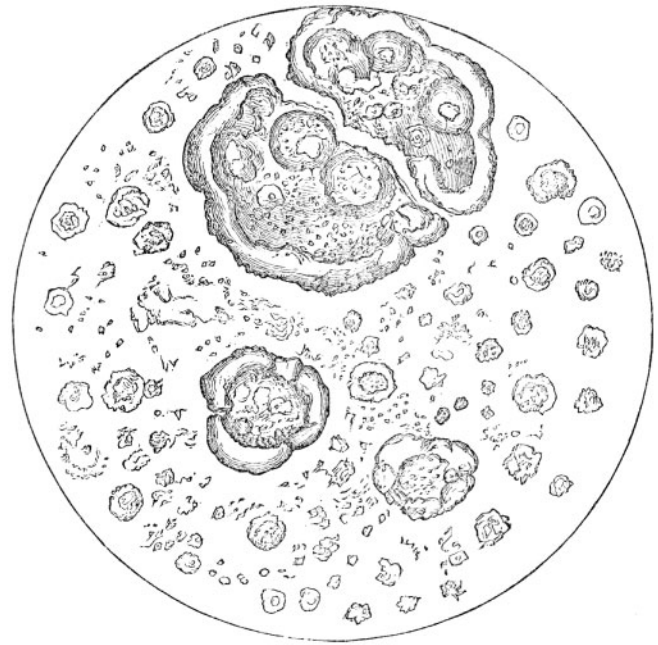


Figure 3 Illustration of the organisms thought to cause cholera, in various phases of development, from William Budd's 1849 pamphlet³⁸

cause of the disease, and the determination of its nature, were all that was wanting to convert his views into a real discovery.³⁸

Budd considered the identification of the agent of cholera the crucial element in uncovering its mode of transmission, and thus prevention.

The opinions of Budd and Snow were widely discussed, along with other views as to the cause, transmission and prevention of cholera. An editorial in *The Medical Examiner* in November 1849 cited Budd as maintaining that cholera could be prevented by supplying water from an uninfected district, 'as water is the principal channel through which this poison finds its way into the human body'.³⁹ Later it seems the water theory was mainly associated with Snow. His friend and biographer, Benjamin Ward Richardson (a prominent member of The Epidemiological Society of London), wrote that the theory was referred to as 'Dr Snow's theory'.⁴⁰ Snow was particularly concerned with priority, a typical example being a letter in the *Lancet* in 1856 complaining that a paper had attributed the water-borne theory to William Budd.⁴¹ Snow stated that Budd had adopted his views, but that he (Snow) had:

not made the above remarks in the way of complaint; but as my researches respecting cholera were conducted with great labour, and very much to the detriment of my more immediate interests, I feel it a duty not to allow the credit of them to pass from me by a mere mistake.⁴¹

Others were chided by Snow for ignoring his crucial contribution.⁴²

In 1849 claims for priority lay in the future, and both Snow and Budd lacked convincing epidemiological data to support their views. Certainly none of the evidence they cited could

begin to quantify the magnitude of the risk associated with water that could transmit cholera. Such reports soon began to appear, however, initially in primitive form. A report from the General Board of Health (of which Edwin Chadwick and Southwood Smith were authors) published in 1850 quoted the evidence from Sutherland's report, which we excerpt in this issue of the *International Journal of Epidemiology*, and other findings that implicated unwholesome water. For example:

Five houses in Windmill Square, Shoreditch, occupied by 22 inhabitants, were supplied with water from a well into which surface refuse and the contents of cesspools percolated. Of the inhabitants of these houses 11, that is, one half of the whole number, died of cholera within a few days.⁴³

Dr Gavin of Hackney investigated diarrhoea (which with increasing severity was seen to merge with cholera) and found that of 22 inhabitants who did not use a particular well all remained free from the disease, while of 63 inhabitants who used the well 46 were attacked.⁴³

In the Hope Street, Salford investigation of John Sutherland, included in his report as an inspector for the General Board of Health, there was a remarkably strong association between household water supply and the occurrence of cholera. Diarrhoea showed a similar, but less marked, association with water supply. Sutherland concluded that with respect to water obtained from wells into which the contents of sewers or privies had escaped the 'predisposition occasioned by the continued use of such water is perhaps the most fatal of all'. This unequivocal statement was, however, buried in a report of 164 pages, in which many other predisposing and localizing causes were discussed, and the importance of unwholesome water was not even mentioned in the conclusions to the report.

In the US similar investigations were being carried out. John Lea noted that the use of rain water or boiled water, rather than unwholesome water, protected people against cholera, and communicated his discovery to the British government in July 1848.⁴⁴ In Cincinnati in 1849 Lea investigated water supply and cholera, producing a map of cholera cases and the water supply of a group of intermingled houses. Of 17 houses, 12 used water from a spring and five used rain water. In the 12 houses using spring water there were 32 deaths, while in the five houses using rain water there were two deaths. Lea assumed 6 people per household to produce an overall mortality proportion of two out of 30 for the people using rain water and 40 out of 72 for those using spring water, about an eightfold difference.

Lea quoted a similar investigation by SO Butler in St Louis, 1849, during which 90 cholera deaths occurred in 25 houses using water from a particular pump, while no deaths were seen in two houses not using the pump water. For these tenement houses Lea allowed eight people to each house to calculate a cholera mortality proportion of around one-half among people using the pump water. He went on to report—in anticipation of the symbolic act in Broad Street—that the Board of Health of St Louis had the handles removed from those pumps considered to provide water leading to cholera. This action was said to produce an immediate improvement in the health of the city, but whether (as with the Broad Street pump handle removal) this was because of an already subsiding epidemic (Figure 4) is unclear.

Lea supported his findings by reference to a review article in the October 1850 issue of *The Quarterly Review* (a publication from London) which quoted Sutherland's Hope Street investigation.⁴⁵ He considered that 'rain water was a surer prophylactic against cholera than vaccination against small pox'.⁴⁶ The mineral content of water was the element Lea thought determined whether it predisposed to cholera or not.

After producing his 1849 pamphlet, John Snow continued working on cholera, referring, at least initially, to the microscopic fungi Budd had detected,⁴⁷ to the additional data he had obtained from Dr Thomas Shapter regarding the outbreak of cholera in Exeter in 1832,^{48,49} and to John Sutherland's investigation of Hope Street, Salford.^{50,51} In reporting the data from Salford he attributed this to the General Board of Health, not to John Sutherland,⁵⁰ but he criticized Sutherland by name⁵¹ for assuming a potential atmospheric transmission route for cholera. He also discussed the work of John Lea, saying the 'connection which Mr. Lea has observed between cholera and the water is highly interesting, although it probably admits of a very different explanation than the one he has given'.⁶

The 1854 cholera outbreak in London allowed John Snow to instigate the extensive empirical investigations outlined by Stephanie Snow in this issue of the *International Journal of Epidemiology*.⁸ The background to these were the small-scale studies discussed above, which linked cholera to water supply, investigated situations where contiguous houses received a different water supplier, and had utilized crude quantification. John Snow's investigations were, however, of a different scope and with different intention than the work they built on, and clearly justify their status as *locus classicus* of 19th century epidemiology. Snow initially utilized data produced in the General Register Office under the auspices of William Farr to demonstrate the association between water supply and cholera cases,^{6,25,52} indeed in John Sutherland's report on the 1854 cholera epidemic these data are referred to as 'enquiries instituted by the Registrar-General' (Box 4).

Snow stimulated Farr to produce further reports on cholera mortality by source of water supply, and organized additional data collection to supplement these. In his most celebrated work—the 'experiment on the grandest scale'⁶—he described a

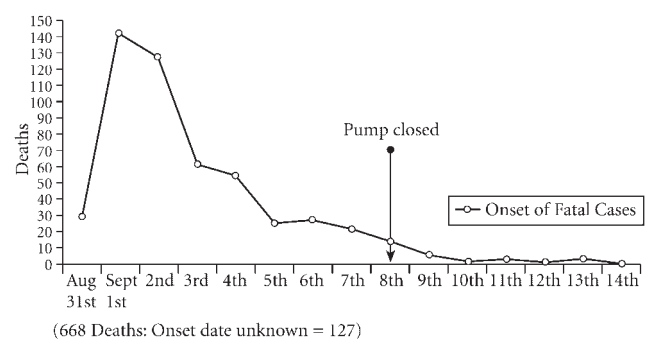


Figure 4 Cholera outbreak in Golden Square, Broad Street, London 1854. The pump handle was removed when the epidemic was waning and appears to have had no effect, although the Reverend Henry Whitehead, who produced these figures, thought that the closure of the pump may have prevented recurrence of the epidemic¹⁰⁸

natural experiment in which water supply was related to cholera risk among intermingled houses that otherwise were similar and inhabited by comparable people. He considered, 'it was obvious that no experiment could have been devised which would more thoroughly test the effect of water supply on the progress of cholera than this'.⁶ A recent introductory epidemiology book concurs, explaining that:

Snow conducted his study within specific neighbourhoods in London where the pipes from these two water companies were intermingled. In other districts, there was less intermingling of pipes from the various water companies that supplied water to dwellings. Comparing the attack rates across various districts of London would have been a less persuasive way to evaluate the effect of the water supply because many factors differed from one district to another. Within the area in which the pipes of the Southwark and Vauxhall Company and those of the Lambeth Company were intermingled, however, Snow saw that there was little difference between those who consumed water from one company or the other, apart from the water supply itself. Part of his genius was identifying the precise setting in which to conduct the study.⁵

The textbook goes on to reproduce data from Table 11 of Snow's 1855 book, demonstrating an attack rate many times higher in those receiving water from the Southwark and Vauxhall company than those receiving it from the Lambeth Company.

Box 4

From John Sutherland's report on the 1854 cholera epidemic in London³³

So far as the inquiries instituted by the Registrar-General go, they certainly exhibit some striking results, which are thus stated.

'In 26 107 houses that derived water from Ditton, 313 deaths from cholera occurred in 10 weeks. In the 40 046 houses that received the impure water from Battersea, 2443 persons it was ascertained died from cholera in the same time. The deaths in the latter districts exceeded by nearly 2000 deaths that would have occurred if cholera had only been as fatal as it was in the houses that derived their water from Ditton. The Registrars were probably in some cases misinformed, but there is reason to believe that no undue proportion of deaths is referred to houses that the Southwark Company supplies.'

It would thus appear that the mortality in a given number of houses supplied by the Southwark and Vauxhall Company when compared with the mortality in the same number of houses supplied by the Lambeth Company would be about as 5 to 1.

When it is considered that the sanitary condition of the population does not materially differ, except in the quality of the water supplied by the two companies, it is difficult to resist this statistical evidence of the predisposing effect of the Battersea water, and of the loss of life which has arisen from its use.

The presentation of the study that Snow gives is, in fact, difficult to follow, but reveals that it was not as summarized above. As a contemporary reviewer, the distinguished hygienist EA Parkes, wrote:

on first reading ..., we thought that the deaths referred to took place only in the district with the intermingled supply, and that this was the answer to the 'experiment on a grand scale', so laboriously inquired into by Dr Snow. But, on re-perusing the passage and its context, we found that these deaths had taken place in *all* districts supplied by the two companies, separately or conjointly. If this reading be correct, we doubt if the comparison can be safely made, for the Lambeth Company supplies, to a considerable extent, a good neighbourhood on elevated ground ... while the Southwark and Vauxhall Company supplies the greater part of the poorest, lowest, and marshiest district in London.⁵³

Parkes' reference to the fact that the Southwark and Vauxhall Company supplied the poorest and lowest parts of London reflected the thinking of William Farr. Farr had shown that those parts of London which had high overall mortality in the years before the cholera epidemic tended to have high death rates from cholera during the epidemic, and attributed this to environmental factors which increased the risk of both cholera and other causes of death.⁵² He also found that elevation above the high-water mark of the Thames was inversely related to cholera mortality rates, and produced a mathematical formula quantifying this relationship.⁵⁴ Snow dealt with the (unacknowledged) criticisms of Parkes by later obtaining data on the water supply for all cholera cases that occurred in districts which were supplied by both companies, together with the number of houses each company supplied in the districts, from which he estimated the population.⁵⁵ These findings supported his less robust original comparison, although it is possible to show that the effects were somewhat reduced—e.g. a ratio of 6.9 during the first 7 weeks of the 1854 epidemic comparing those receiving water from the Southwark and Vauxhall company to those receiving water from the Lambeth company when the analysis is restricted only to districts receiving an intermingled supply from both companies, compared to a ratio of 8.5 as published in his 1855 book (comparing the recipients of either water supply across all districts of London)—suggesting that to a small degree the characteristics of the areas could have confounded the association. Finally, Snow anticipated later epidemiological considerations of how measurement error dilutes the strength of associations. A General Board of Health report found a weaker association between water company and cholera risk than Snow did, which he attributed to the fact that the Board of Health misclassified houses according to source of water supply.⁴²

Knowledge and action

The 19th century cholera epidemics focussed attention on how to prevent disease, with the various studies discussed above explicitly addressing this question. Some commentators thought that an exact understanding of disease mechanism was unnecessary for the prevention of cholera. For example, John Sutherland, in his report on the 1854 epidemic comparing those receiving water from Southwark and Vauxhall company with

those receiving water from the Lambeth company, stated with regard to the dispute over whether impure water was a predisposing or direct cause of cholera that if 'the use of impure water is dangerous to the public health, the manner of its action is of very secondary importance, at least for practical purposes'.³³ Initially John Snow seemed to agree, indicating that those like Sutherland who saw bad water as a predisposing cause, but not one containing a specific cholera agent, held 'a view which, in a hygienic sense, is calculated to be to some extent as useful as the admission of what I believe to be the real truth',⁴⁹ but he later came to think that we 'cannot hope to prevent any disease unless we have a correct knowledge of its causes',⁵⁶ directly criticizing Sutherland for his views.⁵⁷ Chadwick's policy of flushing the sewers and draining cesspools, Snow maintained, increased the contamination of drinking water.⁵⁷ Chadwick explicitly defended his action, saying that a small increase in the pollution of the Thames was better than allowing the sewers to continue giving off the pestilent exhalations into the atmosphere.⁵⁸

It is difficult to attribute what would now be seen as progressive or regressive approaches to disease prevention strictly according to the points of view of their promoters regarding the cause of cholera, or the contagious nature of diseases in general.^{32,59–61} For example, the elite population of Britain instigated changes in hygiene practices, child rearing and disease avoidance from the mid-17th century onwards, that were accompanied by increasing improvement in their health when compared to the rest of the population, before there was what would now be considered accurate knowledge of disease aetiology.⁶² Similarly the mortality experience of British soldiers in the tropics improved dramatically from the early 19th century onwards, apparently through activities intended to improve well-being.⁶³ It is not possible to read backwards from current evaluations of the 'correctness' of contemporary aetiological knowledge to the value of attempts at health improvement. Thus together with a comprehensive list of (in retrospect sensible) interventions—with respect to personal hygiene, changes in working and living arrangements, improvements in the infrastructure of water supply and sanitation⁶—Snow advocated the discontinuance of water closets,⁶⁴ which would not now be seen as a progressive move.

In whose interests?

The 19th century debates about the causes of cholera were often acrimonious,^{32,60} it being clear to the contributors that more was at stake than the niceties of medical research. Thomas Watson, one of the leading physicians of his time and a mentor of William Budd, was quoted by Budd as having said, with respect to beliefs regarding whether certain fevers were contagious or not, 'that the opinions which different men entertain upon it seem to bear some relation to their views on other questions'.⁶⁵ Watson noted 'that all the anti-contagionists he has met with held what are called liberal views in politics and religion'.⁶⁵ Similarly John Snow wrote:

The question of contagion in various diseases has often been discussed with a degree of acrimony that is unusual in medical or other scientific inquiries. The cause of the warmth of feeling that has been displayed has, in most cases,

probably been unknown to the disputants. It is the great pecuniary interests involved in the question, on account of its connection with quarantine.⁶⁶

The notion that views on the contagious, or otherwise, nature of disease reflected views on the appropriate form of political or economic ordering of society was a strong presence in the writings of the participants to these debates, and was later formalized in an influential paper by the medical historian, Erwin H Ackerknecht, in 1948.⁶⁷ One version of his argument—perhaps cruder than the subtlety of his writing merits—was that there existed a strict division between holders of 'contagionist' and 'anti-contagionist' views; that the latter considered atmospheric miasmas to be the principal determinant of epidemic disease; and that the viewpoints people held reflected their political affiliations. Economic liberals desired free trade, (which would be threatened by quarantine and other limitations to the movement of goods and population). Conservatives viewed agricultural interests as paramount, and they were pro-quarantine in exactly the same way as they were pro-Corn Laws and other forms of state interference in the economy. The (generally liberal) rising industrial bourgeoisie were, in this schema, anti-contagionists, and thus the paradox of the rise of such an apparently anti-scientific viewpoint during the first half of the 19th century can be seen as an automatic reflection of the rise in influence and power of this class formation.

Conventional glosses on 19th century epidemiology (and its implications for 20th and 21st century epidemiology) have tended to ignore the political, economic and social origins of disease theory,⁶⁸ instead seeing a struggle over falsity. Equally, however, the crude reductionism of seeing ideas emerging directly from their economic, political and social foundations has failed to survive a detailed analysis of primary sources.^{13,32,60,69} Many individuals held political views that were the opposite of those predicted from their views of cholera; the distinction between contagionist and anti-contagionist thought is too binary, with many theoretical varieties containing elements of both (sometimes termed contingent-contagionism) being widespread. Further, views on politics and disease were mutually constitutive—a recognition that fevers were contagious could influence views on fiscal law, rather than there being a one-way street from the economic base to theories of disease. Moreover, just as theories of disease contained nuances that a binary opposition ignores, political, economic and social beliefs were not simply polarized between two or three cores. Certainly William Budd thought that rigid anti-contagionist views did not sit easily with the generally progressive nature of a rising class when he said, with respect to anti-contagionism:

I have no means of knowing whether the many eminent men who have lately taken this side are radicals and free-thinkers, but I feel very sure that in one important article of their belief they have departed from the true faith.⁶⁵

Reading contemporary accounts makes it clear, however, that at the very least, views on cholera transmissibility and on the economy were refracted through each other.

At a less macro-level, some economic and personal interests were clearly reflected in views on cholera. Snow protested in 1849 that if his 'opinions be correct, cholera might be checked

and kept at bay by simple measures that would not interfere with social or commercial intercourse',³⁷ arguing that sensible approaches to contagious disease and liberal forms of fiscal policy could go together. Recently David Lilienfeld republished Snow's 1855 testimony to a Select Committee of the British parliament considering the 'Nuisances Removal and Diseases Prevention Amendments Bill', intended to increase the salubrity of the environment.⁷⁰ Snow testified that he thought the atmospheric products of bone-boiling and similar trades did not produce disease. Perhaps, Jan Vandenbroucke suggests, Snow was again trying to demonstrate that his theory of disease transmission would not obstruct the interests of industrial capitalists.⁷¹ Certainly Snow took this issue seriously and utilized data from the Registrar-General to demonstrate that the mortality rates amongst those working in the so-called offensive trades were actually lower than those of the general population (Table 1).⁷²

He coupled these data with a discussion of exposure characteristics—a man working with his face one yard from offensive substances would breathe ten thousand times as much of the gases given off, as a person living a hundred yards from the spot⁷²—and the implication of the fact that no effect was seen in this very highly exposed group for notions that such gases damaged health. He also anticipated the contribution of William Ogle⁷³ in recognizing what is now termed 'the healthy-worker effect'⁷³ when stating:

I of course attribute no benefit to offensive smells; and the reason why the persons employed in the callings I am treating of enjoy a greater longevity than the average, is probably because they are less exposed to privation and less addicted to intemperance than men following many other occupations, and because, as a general rule, they do not lead a sedentary in-door life.⁷²

In his empirical work on mortality among men working in the offensive trades, Snow addressed Sandler's claim⁷⁴ that he

produced no evidence to show such workers were not dying or suffering ill effects from their exposures, a century and a half before it was made.

Snow's views on the appropriate agents of disease prevention reflected his interests as a physician. He claimed that:

it is to the improvement of the science of Medicine, by the study and observation of Medical men, that society must look for the diminution of mortality; and not to the ill-directed efforts of benevolent individuals among the non-medical part of the community.⁷⁵

His status as a near-teetotaler, however, was not reflected in his views on the cause of cholera. At a time when it was commonplace to blame drunkenness for cholera—Sutherland included a section on this in the full report from which we excerpt extracts in this issue of the *International Journal of Epidemiology*—Snow's comments were few and mild, indeed he famously attributed the lack of cholera in brewery workers to the fact that they drank only beer.⁶

John Sutherland, on the other hand, clearly had very personal interests when he acted to prevent fever hospitals being built near his home, at the instigation of his neighbours who anticipated a fall in property values as fear of cholera spread.⁷⁶ Sutherland stated that he had no fear of the contagion spreading⁷⁶ and had complained that quarantine was medically useless (although commercially significant), but still led a lobby of local residents taking petitions to London against siting the fever hospitals near his home.⁷⁷

The water companies, naturally, had a particular interest in cholera. Thomas Shapter documents in considerable detail how Mr Goldsworthy, the proprietor of the main water company in Exeter in 1832, profited from providing water during the 1832 epidemic.³⁰ Snow reports how a Mr Main, of a Gateshead water company, carried out a study he claimed exonerated his company's water, while Snow found in the evidence Main collected

Table from Snow, 1856⁷²

| Occupations of males, aged 20 and upwards | Living in London at the Census of 1851 | Deaths in 18 months, ending June 28 1856 | Deaths per annum in 10 000 living in 1851 |
|--|--|--|---|
| Tripe dealer, dresser | | 194 | 9 |
| Tallow chandler | 1239 | 42 | 226 |
| Comb maker | 398 | 16 | |
| Soap boiler | 338 | 6 | |
| Music-string maker | | 87 | 1 |
| Bone gatherer | 34 | 3 | |
| Bone worker | 52 | 2 | |
| Currier | 2649 | 79 | 195 |
| Tanner | 1314 | 35 | 177 |
| Fellmonger | 202 | 5 | |
| Grease dealer | 67 | 1 | |
| Cats' meat purveyor | | 60 | 3 |
| Skinner | 170 | 5 | |
| Parchment maker | 75 | 5 | |
| Glue and size maker | | 64 | 2 |
| Total of offensive trades | 6943 | 214 | 205 |
| Total working and dealing in animal substances | 40 004 | 1210 | 201 |
| Total of males aged 20 and upwards | 632 545 | 22 889 | 241 |

that all the cholera cases occurred in houses supplied by Main's company.⁶ Writing a decade later William Farr explained why the atmosphere may have been blamed for cholera, rather than water:

As the air of London is not supplied like water to its inhabitants by companies the air has had the worst of it ... For air no scientific witnesses have been retained, no learned counsel has pleaded; so the atmosphere has been freely charged with the propagation and the illicit diffusion of plagues of all kinds; while Father Thames ... has been loudly proclaimed immaculate and innocent ...⁷⁸

Poverty and cholera

The apparent success of mid-19th century contagionism⁷⁹ must be judged against the fact that cholera is still an endemic disease in many poor parts of the world.⁸⁰ Knowledge of how to prevent cholera has not been translated into action. In mid-19th century Britain, cholera was seen to be a disease of the poor. This perception was shared by those who considered cholera a disease caused by a specific agent, and those who saw it as the result of a broader set of environmental causes. Common too was the notion that cholera amongst the poor was a threat to the health of the wealthy. The notion that the poverty stricken areas of cities served as epicentres from which disease could spread to the better off areas led to a fear of the poor and to calls that something must be done to alleviate their lot.⁸¹ William Budd concluded his 1849 pamphlet by:

remarking how important it is—even in regard to their own interests—for the Rich to attend to the physical wants of the Poor. To do this is one of our first and plainest duties. The duty itself we may evade, but we cannot evade the sure penalties of its neglect. By reason of our common humanity, we are all more nearly related here than we are apt to think. The members of the great human family are, in fact, bound together by a thousand secret ties, of whose existence the world in general little dreams. And he that was never yet connected with his poorer neighbour by deeds of Charity or Love, may one day find, when it is too late, that he is connected with him by a bond which may bring them both, at once, to a common grave.³⁸

The passage has striking resonances with some current thinking on the role of income inequality in health. Richard Wilkinson has argued that higher levels of inequality within societies are not just associated with worse health amongst the poor, but with overall worse health, affecting the poor and rich alike in highly unequal societies.⁸² In this formulation it is a noxious psychosocial environment that translates into worse health for the rich in societies ignoring the needs of the poor, as opposed to the more directly material notions of the 19th century, although the descriptions of atmospheric miasmas share linguistic similarities with contemporary accounts of adverse psychosocial environments.

John Snow gave a striking example of how unfairness to one's fellows could be rewarded by ill-health. He documented the case of a landlord whose tenants complained that drainage from cesspools was entering their water supply. The landlord

sent an agent who maintained that there was no problem, but with the tenants still complaining:

the owner went himself, and on looking at the water and smelling it, he said that he could perceive nothing the matter with it. He was asked if he would taste it, and he drank a glass of it. This occurred on a Wednesday; he went home, was taken ill with the cholera, and died on the Saturday following, there being no cholera in his own neighbourhood at the time.⁶

Unifactorial or multifactorial?

With respect to cholera in the mid-19th century the distinction between unifactorial and multifactorial approaches has several dimensions. Firstly, as discussed above, in the early part of the century the question of whether cholera was itself a disease entity or merely one form by which an underlying fever could manifest itself was a matter of contention. By the mid-19th century this was less of an issue. Secondly, if disease agents borne by water were responsible for cholera, did this reflect a general, but previously unrecognized, mode of transmission? John Snow certainly thought so, concluding in his 1855 book that the water-borne theory could also apply to plague, yellow fever, dysentery, typhoid, ague (malaria) and other intermittent fevers, including typhus.⁶ Snow quoted some preliminary evidence to support these claims. William Budd, in 1849, generalized the notion of transmission by infectious agents, but in a way that would now be seen as more reasonable, concluding that dysentery was a disease of the same class as cholera and propagated in the same way—i.e. by water—while whooping cough and influenza:

are diseases of the same order; produced, that is, by the growth and propagation of microscopic beings at the expense of the materials of the human body, but drawing these materials from the lining membrane of the air-tubes, and transmitting their germs through the air.³⁸

Most discussion of the unifactorial/multifactorial distinction with respect to cholera in the 19th century has considered Snow to be the convinced unifactorialist, who thought multifactorial causation 'a metaphysical abstraction assumed to account for the facts'.^{83,84} Some writings about the triumph of Snow's approach have⁷⁹—or at least have been taken to⁸⁵—view this as his primary achievement, and have proposed that modern epidemiology could learn from the mistakes of the anti-contagionists of the mid-19th century.

Vandenbroucke considers current approaches that link lifestyle and generic socio-environmental factors to disease, with no understanding of mechanism, as akin to the miasma theories of cholera,⁷⁹ while Loomis and Wing⁸⁵ consider that abstracting single disease agents from their socio-cultural background will lead to an epidemiology that cannot contribute to understanding the determinants of disease rates and distribution in populations. To what extent did Snow perform such abstractions? Certainly he searched for the proximal agents of disease causation and was critical of approaches that attributed disease to general aspects of the environment. This did not only apply to cholera or diseases he considered contagious. Thus

when discussing the cause of rickets, he noted that the disease had generally been attributed to causes:

of a somewhat general nature, such as vitiated air, want of exercise and nourishing food, and a scrofulous taint. These explanations, however, did not satisfy me, as I had previously seen a good deal of practice in some of the towns in the north of England, where the over-crowding and the other evils above mentioned were as great as in London, whilst the distortion of the legs in young children was hardly present.⁸⁶

Instead he thought the disease was due to a deficiency of calcium phosphate, which the adulteration of bread with alum, through rendering the mineral unavailable for utilization, exacerbated.

Snow considered that the simple attribution of cholera to socio-environmental factors was unhelpful. As a thought experiment he suggested that if an investigation of scabies—‘the itch’—were carried out ‘a far greater association would be found between impure air and itch, than between impure air and cholera, and yet we know that impure air has no share in causing the itch’.⁸⁷ Even the most convinced anti-contagionist accepted that scabies was contagious, and Snow clearly hoped that his example would make them think harder about the causes of cholera.

Snow’s approach did not, however, abstract the cholera-causing agent from its environment. Although generally known as the person who demonstrated that cholera was water-borne, much of Snow’s discussion of the disease make it clear that he also considered that transmission occurred through contaminated food or other faecal-oral routes.⁶ In his words:

I believe no one ever supposed that impure water was the sole cause of cholera, and, for my part, I do not consider it a cause at all, but only a frequent medium or vehicle of the one true cause of the disease, namely, the reproductive cholera poison.⁸⁷

He explained how poverty and overcrowding forced people into situations where evacuations containing the cholera agent were involuntarily transmitted.^{6,88} Those who could afford hand-basins and towels, and separate rooms for cooking, eating and sleeping—i.e. the rich—were protected. Thus cholera was highly dependent on social circumstances, but through determining patterns of exposure, rather than through lowering general health, reducing the strength of people and thus increasing their susceptibility.⁸⁸ As Snow pointed out, genteel people contracted cholera if exposed to contaminated water.⁶ Wealthy people could generally avoid this, however, making their experience of cholera considerably more favourable to that of the poor.

In this formulation, understanding the determinants of disease levels in populations, and the distribution of disease within populations, requires knowledge of the historical, political, social and economic forces that lead some populations and sub-populations to be subjected to noxious exposures, while other groups can escape them.^{89–91} The element sometimes missing from this viewpoint is the importance of human agency in struggling for a salubrious environment. A striking phenomenon in the social inequalities in health field is that the most

important causes of ill health and death tend to demonstrate the most marked socioeconomic gradients, and as a particular cause rises in prominence as a contributor to overall levels of ill-health, its social gradient becomes more marked, sometimes reversing from an initial preponderance amongst the better off.⁹² An important element here is lay understanding of what is detrimental to health, and a remarkable aspect of the early and mid-19th century literature on cholera is how even when published professional opinion was against a contagious nature for the disease, or against impure water as an important vehicle of disease, popular opinion thought otherwise. It was, however, only the wealthy who could translate this knowledge into ways of avoiding exposure. The importance of what has more lately been referred to as lay epidemiology⁹³ in mediating between socioeconomic determinants and their ultimate expression in disease rates is largely absent from the history of epidemiology, although not from social history.

Individual and population health

The model of cholera transmission advanced by John Snow was one in which exposures led to disease through particular processes, but the distribution of such exposures—and the underlying factors influencing this distribution—determined the population manifestation of disease. In this model determinants can differ between particular situations. For example, Snow discussed how amongst underground miners poor hygiene and transmission through food was paramount, whilst elsewhere water-borne transmission was of greater importance. This has resonance for contemporary thinking about enteric infections: for childhood diarrhoea, transmission is probably more influenced by lack of water for washing and general hygiene purposes, rather than by poor water quality.⁹⁴ With respect to cholera in different situations the disease can be mainly transmitted through contaminated water (as in Snow’s time), through food, or through other faecal-oral routes.⁹⁵ The demographic groups affected most and the patterns of disease spread would be different according to predominant transmission mode. This is against a background of important climatic influences on cholera^{96,97} and the still imperfectly understood mechanisms through which temperature and salinity of water can influence the survival of cholera *vibrios*, and thus the potential for epidemics. These climatic influences probably account for the simultaneous outbreaks of cholera in unconnected foci,^{98,99} the existence of which provided evidence in support of anti-contagionist theories in the 19th century.

Opponents of Snow, Budd and their co-thinkers referred to the observation that many people who were exposed to the putative cholera agents did not contract the disease, and they utilized this observation when denying the existence of such agents. The same thinking could be applied to smoking and lung cancer today; indeed it was a frequent argument advanced by those denying a causal link between smoking and lung cancer. Missing from this reasoning is the fundamental insight of social epidemiology, that to influence disease levels in populations, the determinants of population levels of disease need to be addressed, not the potentially different determinants of who gets the disease when a population is subjected to a homogenous environment.^{100,101} In public health terms there is no need to understand why one particular cigarette smoker will develop lung cancer and another

will not, given that if cigarette smoking were abolished 90% of the lung cancer within a population would disappear.

In many cases popular opinions of what is a pleasant environment coincide with what is a healthy environment. The fact that rates of mortality for many diseases began to decline well before there were effective medical interventions¹⁰² attests to this, with the particular contribution of motivated attempts to increase the salubrity of the environment, by non-medical and medical interests, making an important contribution.¹⁰³ But this is not automatically the case—cigarette smoking was adopted initially by the better off, who also used to be more obese than the poor. Being a smoker and being obese were characteristics people desired before understanding of their health-damaging nature developed (or, in the case of obesity, before the balance of influences on disease patterns within populations changed, such that over-nutrition had a net adverse effect). Identification of exposures determined by adverse social conditions will not occur if the focus is only on broader—or even abstracted—environmental factors. This was recognized by Snow when, as quoted above, he referred to his thought experiment for a study on scabies. Improving the atmosphere without removing the mite would not reduce scabies rates. The approach to peptic ulcer—one of the epidemic diseases of the mid-20th century—shows similarities to approaches to cholera in the mid-19th century, with a large array of putative causal factors—in particular dietary patterns and stress—being identified, as was the socioeconomic distribution of the disease. This identification produced little benefit to either population or individual health—trials of dietary modifications or stress management failed to improve health in ulcer patients. Identification of *Helicobacter pylori* infection as the proximal causal agent—which was consistent with epidemiological investigations that demonstrated important birth cohort effects by Mervyn Susser and Zena Stein¹⁰⁴ (who also recognized that the apparent multifactorial aetiology of peptic ulcer—with supposed contributions from diet, alcohol, cigarette smoking as well as stress, personality and genotype—did not ‘exclude the possibility that a major single causal factor waits discovery’).¹⁰⁵ Peptic ulcer rates declined in a cohort-specific manner in line with improvements in early-life social circumstances, in particular with respect to housing, sanitation and means of maintaining adequate hygiene practices, which in turn would have led to cohort-specific declines in *H.pylori* infection.¹⁰⁶ Improvements in living conditions were not, of course, introduced to influence peptic ulcer rates many years later, but this was a beneficial unintended consequence of improving the environment in ways which were obviously considered favourable to those experiencing (and agitating for) such improvements.

There are many examples of conditions in which a wide variety of socioenvironmental factors were identified by epidemiological or proto-epidemiological studies—including scurvy, pellagra, stomach cancer, AIDS and cervical cancer—where it is clear that these associations were due to the factors determining a particular exposure (or in some cases factors determining lack of treatment), rather than through influencing susceptibility (in the 19th century sense, through being predisposing causes). This does not, of course, mean that the appropriate approach to these conditions is to identify the proximal agents of disease and focus on these. While niacin deficiency is the proximal factor leading to pellagra, finding this out without understanding the

socioeconomic constraints on nutritional intake would not—and did not—alleviate population rates of disease, just as a much greater understanding of aetiology of cholera than that held by John Snow has not translated into absence of cholera for the poorer people in the world today.⁸⁰

Sylvia Tesh⁵⁹ suggests that both so-called contagionists and anti-contagionists of the 19th century were, as supporters of capitalism in at least some of its guises, intent on focussing on the most proximal cause of cholera which their understanding allowed—on drains, rather than on the exploitative economic system which ensured that the poor remained poor. Far from being interested in ‘social factors’ (as heroic histories of public health in the 19th century have sometimes contended), those involved were concerned with eliminating either miasmas or the direct agents of cholera in the way that was least disruptive to the economic system. This is well argued—and clearly has resonances for approaches to socioeconomic inequalities in health today^{92,107}—but should not distract us from understanding that knowledge and power intimately intersect, and the best armament for improving population health is an understanding which links socioeconomic determinants, through proximal mechanisms, to their ultimate expression in population health.

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