Commentary: Nobody loves a critic: Edmund A Parkes and John Snow’s cholera

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Accepted 20 June 2013

Science isn’t about being right. It is about convincing others of the correctness of an idea through a methodology all will accept using data everyone can trust. New ideas take time to be accepted because they compete with others that already have passed the test. New thinking needs a strongly favoured methodology and an iron-clad application if it is to triumph, replacing the old.

Journal critics are the first line of defence against ideas and research projects that seem promising but have yet to be vetted, their methods analysed carefully. Despite the importance of that service, the critic’s role is typically disparaged because—let us be frank here—nobody likes critics. If they praise something they’re assumed to be sycophants and if they disparage published work they’re dismissed as merely grumblers.

History is not kind to critics. Its writers typically dismiss where they do not simply ignore those whose careful reviews argue caution in the face of works destined to become, in the future, classics. Think, here, Prince Peter Kropotkin whose naturalist studies focused upon the limits of Charles Darwin’s evolutionary theory and the direction in which research based upon it would be best directed.1 Only today—more than 130 years later—is the importance of his critique being acknowledged.2

There are good critics, of course, even great ones. The best are not only prominent in their field but also stylish essayists whose careful insights educate the general and the professional reader alike. Harvard biologist Richard Lewontin is a current example, an essayist who enfolds each review within an erudite recital of the state of the science being discussed. The result leaves the reader (and author) gasping: ‘I wish I had said that.’3

As an example of a good critic unfairly dismissed by history think Edmund A Parkes, the British physician and researcher who reviewed John Snow’s famous 1855 opus, On the Mode of Communication of Cholera.4 In a seven-page, approximately 7800-word essay, Parkes carefully considered and found wanting Snow’s argument that cholera (and plague, and typhoid fever) was solely waterborne.5 Although the myth of Snow’s brilliance insists his critics were wrong, a careful reading of Parkes’ concerns insists that the myth of Snow is overstated6. Yes, cholera is a waterborne disease. But were we to read Snow’s work with attention but without foreknowledge we, too, would find its argument incomplete.7

This review of the 19th century debate over cholera has more than historical significance. It pits a simplistic, focused explanation against one that was broad and multifactorial. And, too, it demands attention be paid to the researcher’s methodologies and their sufficiency, not just results. Finally, it pits the myth of the lone researcher against the reality of science as a complex, communal, interactive process. In a time of rapidly evolving, epidemic zoonotics, the lessons of that earlier debate are as contemporary as the evolving state of the mutating coronavirus that so concerns us today.

Edmund A. Parkes

The choice of Edmund Parkes assured a knowledgeable and informed review of Snow’s privately published 1855 text. A former military physician with ‘considerable experience of tropical diseases’, including cholera, in 1849 Parkes was elected a special professor of clinical medicine at University College where he was also appointed as a physician.6 He had published at least three works on cholera: ‘On Asiatic and Algide cholera’, written during his service in India, was published in 1847; ‘Intestinal discharges in cholera’ and then ‘Early cases of cholera in London in 1848’. A frequent contributor to the medical journals of the day, he also authored several well-regarded textbooks. And, from 1852 to 1855, he was editor of the then prestigious British and Foreign Medico-Chirurgical Review in which his review of Snow’s work was published.

Expert in both chemistry and physiology, he was an authority in the area of ‘modern hygiene’ which today we call public health. His textbook, the Manual of Practical Hygiene, went through four editions in his lifetime. An eighth edition, published in 1891, was translated in several European languages.
And so, like Snow, Parkes was an extremely well-published medical author, researcher and sanitarian. He was similarly engaged in the study of cholera and published in the field before Snow’s first monograph on cholera in 1849.9 It is hard to think of anyone better qualified to review Snow’s 1855 tome.

**John Snow**

Snow’s work, as G Davey Smith so nicely put it, ‘appeared amidst a veritable spate of speculation, experiment, investigation and recommendations regarding cholera’.10 Following the first global pandemic, which began in India in 1817,11 a generation of researchers assembled and reviewed a mass of evidence.12 On that basis they debated from the 1830s through the 1860s the nature of cholera, the mechanisms of its local transmission and the general means of its broad diffusion. Snow was not the only author to propose a waterborne disease. William Budd was another.13 Most, however, did so within a more or less multifactorial context that acknowledged then prevalent miasmatic theories of airborne disease generation and diffusion.

For others, water was part of the cholera story but for Snow it was the whole story. Thus he proposed not only a novel theory of cholera as a water- rather than airborne disease but advanced simultaneously a general theory of waterborne disease that broke with accepted theories of disease as fundamentally airborne. The onus was on Snow, therefore, to make the case for his theory through his studies of cholera.

In 1849 Snow published a monograph arguing cholera as solely waterborne. His argument was based upon what recent biographers have called ‘a complex blend of epidemiological evidence, pathological observations, and bold analogies’.14 Two local outbreaks, one at Surrey Court, Horsleydown, and another at Albion Terrace, were the epidemiological centerpiece of this publication. In both microstudies, Snow concluded contaminated local water sources were the source. Other investigators drew different conclusions based on an air- rather than waterborne disease theories.

Snow admitted his evidence was ‘scattered and general a nature’, and his theory was thus offered ‘not as matters of certainty, but as containing a greater amount of probability in their favor than any other’14 (quoting *Snow on the Mode of Communication Cholera*). Almost immediately, he published two further papers attempting to flesh out the evidence and between 1849 and 1856 published frequently in the field, arguing time and again that cholera was solely the result of contaminated water and transmitted by oral-faecal contamination. Period.

**The review**

Parkes began his review this way: ‘None of our readers can be ignorant of the opinions of Dr Snow on the communication of cholera by means of drinking water, nor the perseverance and energy with which he has sought for facts to corroborate this view’.3 The text under review, Parkes added, recapitulated ‘all the evidence [he] has hitherto published, with the addition of certain facts lately acquired’. The first part of Parkes’ essay covered the older data, carefully reviewing 11 separate cases of localized cholera outbreaks described as evidentiary by Snow. Time and again, Parkes complained that Snow continually stated possibilities as certainties and suppositions as fact. In a review of the Horsleydown data, for example, ‘instead of leaving the origin of the first case uncertain’, Parkes writes, Snow assumed without conclusive evidence its source; at Albion Terrace he insisted without proof that cholera ‘must’ have entered local drainpipes in the water even though other investigators argued different explanations.

‘The point’, Parkes wrote in exasperation, ‘is to prove the fact of water being the agent, and not to assume it, and then to seek for some other explanation of these cases for which the presumed contamination cannot account’.3 And here Snow’s methodology, and the data it required, were wanting. Time and again, relevant data by which Snow’s theories might have been rigorously considered within the science of the day were lamentably absent. ‘Now, certainly in no less than seven of the eleven cases’, Parkes concluded, ‘the evidence to prove the effect of the water is so loosely stated, and the accessory circumstance of the outbreaks are so utterly disregarded, that we do not think any one can feel that even a tolerable case is made out in favor of Dr Snow’s opinion’.5 In the remaining four cases the evidence was stronger but not conclusive.

**New cases**

Snow’s 1855 text included two critical new studies. The first was the ferocious, if localized, Broad Street outbreak and the second his study of the South London epidemic, both occurring in 1854. In the latter study, Snow proposed a ‘natural experiment’ in which cholera mortality would be related at various scales to different water sources in the South London cholera epidemic of that same year. This ambitious idea was later advanced as a rationale for his epidemiological fame.

**Broad Street**

Perhaps the most famous of Snow’s examples, his analysis of the Broad Street outbreak exemplified for Parkes the author’s conceptual and methodological limits. Snow employed two very different types of analysis, both then common in disease studies. The first included a recitation of case reports and the second was cartographic. Famously, Snow applied for the mortality reports from the General Register...
Office and had the first weeks of the outbreak’s mortality included on a map of the Broad Street area with, of course, the location of water pumps that provided district water. ‘It would clearly appear’, agreed Parkes, ‘that the center of the [cholera] outburst was a spot in Broad-street, close to which is the accused pump; and that cases were scattered all round this nearly in a circle, becoming less numerous as the exterior of the circle approached’. 5

But to Parkes the centric distribution appeared more likely to present the image of an airborne rather than waterborne pattern of disease diffusion. ‘If it were owing to the water,’ he asked, ‘why should not the cholera have prevailed equally everywhere the water was drunk?’ And whereas the epicentre of the outbreak was clearly in the vicinity of the pump ‘there are, indeed, so many pumps in this district that wherever the outbreak had taken place, it would most probably have had one pump or other in its vicinity’. 5

Snow provided neither the type of comparative nor even descriptive statistics, then widely employed, that might have strengthened his mapped argument. In the 1854 Broad Street study the local curate, Rev. Henry Whitehead, published in 1854 a monograph replete with descriptive statistics of the outbreak and its relative impact on citizens. 15 Without that type of analysis Snow’s mapped argument was open to multiple interpretations.

Recognizing this deficit, perhaps, in an 1855 report to a parish inquiry Snow remapped the data, correcting several small errors, and included an irregular polygon based on greater proximity to the Broad Street well than to all others (Fig. 1). 16 He did not, however, use this to calculate relative mortality among persons living within this area. Nor did he create other polygons around other pumps in a manner that would permit comparisons of mortality based on population between the Broad Street and other pump regions. As a result the map was inconclusive and, in Snow’s study, statistics that might have strengthened his case were largely absent.

Parkes summed up the more general limits of Snow’s argument this way: ‘The weak points in this array of evidence are, I 81, the want of proof of

Figure 1 In a later map Snow included a ‘nearest neighbor’ irregular polygon to enclose the population closest to the Broad Street pump. He did not, however, use this to calculate comparative mortality ratios that would have helped his case
contamination of water, or rather, the evidence in favour of its purity; 2nd, the deficiency in negative evidence, that there was no other local cause which produced the partial outbreak; and 3rd, the fact that the disease ran rapidly to its acme, and then declined, while the water supply remained the same.5

These were serious deficiencies. Indeed, Snow admitted that, examining the pump’s water, ‘I found so little impurity in it of an organic nature that I hesitated to come to a conclusion’. In 1854 and early 1855, available evidence did not support the idea that the well itself had been contaminated. ‘It will have been observed’, wrote Parkes, ‘that the contamination of the pump water with drains, or by any other method, is not even attempted to be proved….’ If the reasons for the outbreak’s rapid onset were unclear so, too, was any rationale for its decline.

Snow’s stated goal, Parkes lectured, was ‘not to prove that bad water acts as a predisposing cause, but that the water contains itself the cause of cholera. To prove so weighty a fact, we require not only positive, but negative evidence…that no other circumstance existed which could explain the attack except the contaminated water’.5 Snow briefly considered but as briefly dismissed then current arguments implicating the foul-smelling, new sewer lines added to the district in 1851 or ‘bad airs’ emanating from the 1665 plague burial sites that had been punctured by the new sewers. Indeed, Snow’s map did not include the sewer lines—included in three other contemporary maps—and misrepresented both the size and location of the former plague burial pit whose southeast corner was a short block from the Broad Street pump (Fig. 2).17

Snow’s report was published before an 1855 engineering study that revealed a break in the bricking of the Broad Street well and visual evidence that materials flowed from a local cesspool into it.18 Also in 1855, Reverend Whitehead identified the index case in his report for a parish inquiry into the outbreak, a family that lived near and used that cesspool for its wastes. Had Snow not published prematurely, before those data were available, and had he added even minimal comparative and descriptive statistics, his case would have been more persuasive.

The South London epidemic

Snow’s study of the South London epidemic, affecting thousands rather than hundreds of Londoners, was similarly deficient. Ambitiously, he attempted to distinguish mortality on the basis of two different water company jurisdictions, the ‘good’ waters of Lambeth Water Company and the ‘bad’ waters of the Southwark and Vauxhall Company that drew its water from Battersea in an area of Thames River pollution.

Figure 2 In the Broad Street study Snow located the 1665 plague burial site as a small oval at the northwest of the map. It was, however, a large area whose southeast corner was a block from the Broad Street pump. New sewer lines had been added to the district in the early 1850s.
In service of this study, Snow had drawn a map of the epidemic area of South London in which the water service jurisdictions were identified. He then attempted to argue that the mortality in areas supplied by the Southwark and Vauxhall Company was far greater than in the area supplied by the Lambeth Water Company, which drew its waters from the cleaner area of Ditton. Unfortunately, when he was writing the book, he later admitted, ‘I was unable at the time to show the relation between the supply of houses in which fatal attacks took place, and the entire supply of each district and subdistrict, on account of the later circumstances not being known’.19 Parkes made very clear that without those data Snow’s experimenta cruces could not go forward. ‘Snow endeavours to meet this difficulty by giving, from the Parliamentary return, the number of houses supplied respectively by the two companies. But this return applies to the entire districts, and not to the special district where the supplies are intermingled, so that really we are in doubt whether the Ditton water is supplied to half of this special district, or to a quarter or a tenth part’.

The required data became available after Snow’s book went to press. They were then carefully analysed by the Board of Health’s Dr John Simon in a study ‘presented to both houses of Parliament by Command of her Majesty’ and published in 1856.20 In a statistical tour de force he found that there did appear to be, as Snow predicted, a 3:1 difference in mortality ratios at the scale of general water supply jurisdictions (Fig. 3). But that difference disappeared when data were analysed at finer registration district and subdistrict levels. Without better methods of statistical analysis or a definitive identification of a cholera agent, Simon concluded, water was implicated as at least a contributing source, but its acceptance as the sole source of the outbreak could not be proven.

Snow responded to Simon’s paper with his own. But Simon was right. Absent a kind of Bayesian analysis, and the statistics available were insufficient to provide conclusive proof;21 and without the identification of and a means of testing for what we now know is the bacterial agent, the quality of the water could not be adequately ascertained.

The result to Parkes, and most of his contemporaries, was conclusive: ‘We were unable to do more than conclude that he [Snow] had rendered the transmission of cholera by water an hypothesis worthy of inquiry; we cannot draw any other conclusion from his researchers on water supply that the predisposing effects of impurity of water are also rendered highly probable’.5

Discussion

Figure 3 In his analysis Dr John Simon found that, at a general level, cholera mortality could be related to different water suppliers. At a finer scale of analysis this relationship broke down, however. Therefore, he believed the findings suggestive but not conclusive

Clearly, Snow did not prove his case. In both the Broad Street and the South London studies, Snow failed to include the type of carefully constructed mortality ratios based on population that would have permitted comparison between the areas of ‘good’ and ‘bad’ water. In neither case did he vigorously address alternative disease theories, even where the geography of the outbreak insisted they be seriously considered. This was especially galling in Snow’s South London study where, Parkes noted, Snow paid no attention to ‘Farr’s Law’ demonstrating an inverse relation between altitude above sea level and cholera mortality. ‘He alludes, indeed, to, but speedily dismisses the important law of elevation’, wrote Parkes, ‘demonstrated by the Registrar-General; and refused, indeed, to admit the effect of elevation and refers the differences of prevalence entirely to the water supply’.5

Some of the faults of Snow’s 1855 tome could have been easily avoided. Had he constructed other, distance-based polygons around other pumps in the Broad Street area, he might have constructed relative mortality ratios that implicated the central pump more strongly. Had he delayed publication until the pump’s casing could be carefully investigated—and the index case identified—the contamination of the Broad Street pump would have been evident rather than deduced. And as he admitted later, his ‘grand experiment’ failed because he rushed to publication before the required data were
available. When the data from the General Register office were made available they were, as Dr Simon correctly noted, suggestive but inconclusive using the analytical tools of the day.

Parkes did what a good reviewer is supposed to do; he carefully considered the evidence presented and weighed its presentation and relevance given the author’s objectives. The myth of Snow’s brilliance is thus transformed from a hero story into a cautionary tale. Snow had a good idea. Indeed, he had a great idea. Cholera is waterborne, after all. But in presenting this idea, and a disease theory tied to it, he failed to employ the best methodologies of the day (cartographic or statistical) and in his enthusiasm rushed to print, again and again, before necessary data were available. In arguing his theories he rarely gave more than grudging attention to other disease theories or to the data that seemed to support them. He refused to modify his arguments or change his methodology, when presented with the work of others that seemed at odds with his own.

The practical lessons of Snow’s failure to convince his contemporaries is a warning to those who, convinced of their theories, seek to publish prematurely or to promote bolsterously research whose proofs are only partial. Of equal and perhaps greater importance is the necessity of a defence of Snow’s critic, an argument against the near universal myth of Snow the great and lonely hero. At a 2013 conference celebrating the 200th anniversary of Snow’s birth, I argued the obvious limits of Snow’s work and the appropriateness of his contemporaries’ criticisms and cautions. And, too, I argued the falsity of the claims made on Snow’s behalf by modern historians. Snow did not create ‘show leather’ epidemiology or mapped cartography. He did not create the experimenta cruces. He did not prove cholera was waterborne. Other contemporary researchers—Reverend Henry Whitehead, William Farr, John Simon etc.—were more exacting and better epidemiological researchers than Snow. And yet we persist in the modern myth of Snow, a myth born in a 1901 textbook on the Principles of Sanitary Science and Public Health, and later expanded in the 1930s as a broad truth.

But, ‘We need heroes, don’t we?’ a member of the conference audience asked plaintively. It does not matter if Snow was a hero or a chump, in other words. We need to believe in the solitary genius as we face the disease challenges of our own time. Snow is a symbol, in other words, and the truth of his work as a researcher does not matter. My reply is a suitable conclusion to this essay and its relevance: ‘Yes, we need heroes. But we don’t need a Lone Ranger who singlehandedly saves the day.’ When we make the hero a solitary figure we forget the cooperative nature of medicine and public health. There are no solitary heroes in the struggle with endemic and pandemic disease, just the many who struggle to treat them and understand their nature.

If we are to learn anything from Parkes’ careful review and from Snow’s passionate if incomplete work, it is this: research is collaborative and cooperative, not individualistic. It includes our fiercest critics and most ardent supporters. In the dilemma that is endemic or epidemic disease, it’s not about us, our ideas, but about the disease and our communal means of understanding these conditions. To ignore Snow’s failings because we want a simple hero is to assure the failure of the science we promote and practise. It is to assure that public health disasters will follow.

Conflict of interest: None declared.

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