

Commentary: John Sutherland's Epidemiology of Constitutions

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To a modern epidemiologist, these extracts from John Sutherland's *Report to the General Board of Health on the Epidemic of Cholera of 1848 and 1849* will likely seem both familiar and strange.¹ Sutherland plots and counts cases; he is interested in cholera in time and space, searches for empirical generalizations ('fixed laws'), and will suggest effective measures to respond to the epidemic. In particular, his discussion in section three of the effects of 'unwholesome water' may seem intriguing in light of John Snow's concurrent and rightly celebrated demonstrations of the waterborne character of cholera.

And yet Sutherland fails to take much interest in his findings with regard to water. He relegates impure water to the status of predisposing cause, and seems uninterested in analysing the phenomena of the epidemic to exclude plausible cholera causes. Here I explore the unfamiliar in Sutherland's epidemiology, and consider how the familiar fits into it. I will suggest that we need to think about the work of Sutherland and most of his contemporaries, not in terms of groping toward modern knowledge or in terms of rival theories of disease causation (commonly designated as miasma versus contagion), but at a more fundamental level—of what a causal account of an epidemic entails. The quite different paradigm in which Sutherland worked had classical antecedents in Galen, though it had passed through multiple reconfigurations by 1850.^{2–4} It is exemplified in his dual use of 'constitution': as a state, probably of atmosphere, associated with cholera, and also to refer to the differing conditions of people exposed to it.

Sutherland and the Chadwickian Project

First, context. These extracts form roughly half of the first 28 pages of Sutherland's 185-page report—under 10%. In the remaining pages, there is much concern with treatment and prevention, but also much more on other local causes. That report in turn is embedded in a general summary report by Edwin Chadwick, secretary to the new General Board of Health in England (only created in late 1848), and is accompanied by another lengthy report (Appendix B) on cholera in London, which has its own appendices. In all, nearly 600 pages of the 21st volume of the House of Commons sessional papers for 1850 deals with the second, or, depending on who is counting, third pandemic of cholera, which began its westward spread in 1839 and reached Europe in 1848. A reader of the full report will probably find the extracts representative, and yet it is important to keep in mind the sheer quantity of data these indefatigable sanitarian empiricists generated. One way of understanding the sanitarians' failure to follow up the marked association of cholera with water is that they were swamped with other suggestive associations.

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Sutherland (1808–1891), was one of a number of Edinburgh-educated physicians (MD 1831) prominent in public health.^{5,6} He had become active in the health of towns movement by 1845. At the time, he was already an important Liverpool practitioner and senior dispensary physician. In the next few years he became editor of two short-lived journals, *The Liverpool Health of Towns Advocate* (1846) and *The Journal of Public Health and Monthly Record of Sanitary Improvement* (1847–1849), and was one of the most vigorous campaigners for establishment of a full-time medical officership for Liverpool. The post would be occupied by William Henry Duncan, whose handling of the job was insufficiently activist for Sutherland.⁷ While Edwin Chadwick was happy enough to have Sutherland as a consultant, the two had quite different views of the role of medicine in public health. Chadwick saw public health as administration and engineering, while Sutherland saw medicine as central.^{7,8} Both worried about the implications of the view that disease was largely a function of poverty and preventable by an entitlement to a higher standard of nutrition and medical care. Such a view would promote the medicalization of poverty and provide a basis to access public relief outside the strictures of the new poor law.^{9–11} Rather, they looked to sanitation broadly conceived as an approach that would not disrupt the structures of incentive and individual responsibility upon which liberal society was to operate: midway through section one, Sutherland takes pains to point out that cholera does not prey exclusively on the weak, but rather on the 'robust' and those in the 'prime of life'. It was important to reiterate that disease was cause, not consequence of poverty. Following Chadwick's expulsion from the leadership of English public health in 1854, Sutherland became increasingly involved in army hygiene. He investigated camp conditions in Crimea and was later one of Florence Nightingale's allies in her campaign for airy hospitals.^{6,12}

Sutherland's job on the cholera report was to roam Britain (beyond London), to accumulate information and analyse it, and also to admonish and advise. The 1848–1849 cholera was Britain's second experience with cholera, the first occurring in 1831–1832. The epidemic was initially relatively mild in the winter of 1848–1849, waned, and then returned more virulently in the summer of 1849. The General Board of Health policy was to try to prevent those exposed—which it presumed to be almost everyone in greater or lesser degree—from developing active cholera symptoms. This it did by promoting public and private cleanliness, but also by urging a regimen of diet and habit, which, it was complained, was unrealistic for most working people to follow. Its aim here was to forestall a 'premonitory diarrhoea', which, during the prevailing epidemic 'constitution', would often develop into a full case of cholera.^{13–16}

Below I develop Sutherland's framework by reviewing these extracts in order.

Constitution and localization

Early in section one Sutherland asserts his epidemiological manifesto: cholera is not ‘capricious’, its ‘propagation’ reflects ‘fixed laws’. And yet the most basic questions are unresolved. Is this cholera mainly a temporal phenomenon, an occasional state of nature? or an entity that moves from place to place? Or, is it always present but not always manifest? Is it of equal strength everywhere? And, whatever it is, is it at all susceptible to human action? It may seem hard to reconcile such vast uncertainty with Sutherland’s assertion of ‘fixed laws’, but the laws he is referring to—the ‘modifying conditions’—operate in a different way and on a different level.

We need here to recognize the dual usage of ‘constitution’, evident already in the first two paragraphs. The grand questions above concern the ‘epidemic constitution’. Sutherland is exploring answers to an epidemiological question that was effectively marginalized by the fascination with germs that arose in the last third of the nineteenth century. This is the question not of what causes a *case of disease* (in turn, a more complicated question for him than for his successors), nor even of how a causal agent reaches its victim, but of what occasions the *epidemic*. That there is an epidemic is empirically clear from the frequency and uniformity of the series of symptoms he lists: these constitute cholera, a disease endemic in India, but how to explain its pandemicity? Or that of plague or smallpox? Modern answers to these questions involve the immunological status of populations, random and induced changes in the virulence of agents, and various environmental factors. While in recent decades we have developed abilities to confirm some of these changes independently of their purported effects, in earlier decades such assertions had mainly placeholder status. Sutherland remains here a Newtonian empiricist, feigning hypotheses only reluctantly. If there is changed effect, there must be changed cause, but where in the complicated structure of antecedents that change lies, he will not say. In the 1840s, ‘epidemic constitution’ most commonly referred to states of undetermined atmospheric variables, but it need not be so limited. All that was required was that a hypothesis be plausibly environmental at the level of generality dictated by the spatial and temporal character of the epidemic. One may note that the question of the occasion of the epidemic is one that the familiar hypotheses of miasma and contagion do not directly answer. Sutherland’s alternatives—even of the movement of the epidemic from place to place—are consistent not only with the language of contagion, but also with sophisticated versions of local miasms.

It is at the local level that progress is possible. The laws that can be known are of those ‘special’ or ‘modifying conditions’. These conditions modify not what we would call the agent of the disease, but its recipient, the person in whom the disease arises. Here ‘constitution’ summarizes the immunological state of the individual with respect to the particular epidemic. It is responsive to factors that are effectively original and might include matters of sex, age, and heredity, and to external factors that will leave their stamp on anyone exposed to them. These together determine the state of predisposition of each individual: ‘by virtue ... [of them] the epidemic obtains such power over the resisting vital forces of individuals as to produce that class of phenomena usually ranked under the general designation of cholera.’ These predisposing causes are also the site of prevention: by strengthening the constitution, the vital forces may resist the

epidemic constitution. There is no need here even to notice a question about the nature of the causal agent, if indeed there is one.^{3,17} The epidemic constitution accounts for the occasion and character of the epidemic; the local factors determining predisposition account for its differential effects and provide the basis for fighting the epidemic. While Sutherland can refer to terms like ‘atmospheric impurity’ or ‘contagion’, they play no very large role in his account, and he sees no essential antagonism between them. In part, he can do this because he conceptualizes cholera as analogue rather than digital, in terms of degree, rather than kind. If disease were a species it might make sense to ask about its special cause or agent or essence, but for Sutherland it is distinct only in the most nominal way. His reference to ‘that class of phenomena usually ranked under the general designation of cholera’ indicates this: ‘cholera’ is a construct marginally useful in designating a certain set of symptoms and a disease course.

The epidemiologist as clinician

Sutherland’s focus on cholera as a matter of degree brings a concern for its course. Why, he wonders, does the course of symptoms outlined in paragraph two sometimes lead to the deadly sequelae in paragraph ten and sometimes resolve safely? From Hippocratic times onward, physicians had recognized that in many diseases certain signs, those on so-called crisis days, indicated a positive or negative prognosis. Fortunately, the constitution that produced those signs could be changed by the physician’s armamentarium of non-specific therapies, and the prognosis improved. Sutherland, in common with contemporary fever specialists (and, for that matter, those focussing on any other disease), recognized that the crucial alterations of constitution were not limited to those effected by items in the Pharmacopoeia. For one thing, some of these alterations were best achieved before one was brought (voluntarily or otherwise) to the physician as a victim of disease: hygiene, public and private, blended with therapy. Factors that influenced the course of disease, then, were causes, not of the disease itself in a modern sense, but of its course and outcome (death or recovery), which was, after all, from the patient’s point of view, the most important matter. Because it involved seeking general knowledge of how various factors affected outcomes, the clinician’s mission of studying, treating, and otherwise managing individual cases merged with the epidemiologist’s problem of determining their causes. It was such an approach of managing the course of disease rather than the attacking its ulterior causes that Sutherland would later share with Florence Nightingale, at a time when it had been rendered heterodox to medical thought.

It may seem that there is a truistic and even tautological quality to Sutherland’s reasoning here; that in admitting so many independent, indistinguishable, and unconfirmable factors to operate as causes, it resolves itself simply into a statement that ‘those in whom the forces of cholera are sufficient to overcome the vital forces get cholera’ or ‘those in whom the causes are sufficiently severe, die from cholera’. Sutherland, however, is aware of such a tendency in this causal framework and resists it. He notes, for example, that in places without distinct localization of the epidemic (i.e. where the processes of contagion or miasmatic transmission are unlikely to operate), populations ‘apparently’ similarly predisposed do not always suffer similarly. That suggests

that there are differences in the intensity of the epidemic constitution, and it means, he admits, that what will suffice in one place may not suffice in others. Cholera, it seemed, was not as 'general' as the General Board of Health would like it to be.

Cholera as moral failing

Having inferred some characteristics of the epidemic constitution, Sutherland goes on to consider whether one can say anything about those who die from cholera. Here the language jars, for Sutherland asks the teleological question 'what is cholera for?' It is not, he assures us, for the extermination of all in its region of influence, although it could have been. It is, he argues, 'corrective'. For contemporaries the term would have recalled the 'positive checks' to population of Thomas Robert Malthus, who had borrowed from the 18th century physician Thomas Short the notion of deadly epidemics as among the 'great correctives' of overpopulation. By 1850 Malthusianism had waned, and while Sutherland's apology continues to deploy the Malthusian themes of the emergence of mind and moral qualities through hardship, the links are more immediately to the Unitarian necessarianism of Thomas Southwood Smith, who was the closest thing to a medical member of the General Board of Health. Smith, following Joseph Priestley, held that epidemics, like other misfortunes, were necessary components of God's Providence. They would somehow improve us, in this case by forcing us to combat 'sanitary evils'. What made these 'evils', however, was not that they contributed to disease, but that they led to 'ignorance, vice, and crime'.^{9,18}

Sutherland follows this point by claiming that epidemics frequent those areas where their purifying influence is most needed, and then, almost immediately, turns from geography to social anthropology: In the haunts of epidemics is found a disturbing, 'hardly to be forgotten' human type, those who preponderantly die from epidemic cholera. He uses the idiom of case history, not class, but class is implicit. The characteristics of these people are anxiety, apathy, and resistance, which at first glance seem hard to reconcile. While he is not explicit, Sutherland hints that this mien is not simply prognostic but causal: responding in these ways will cause those suffering from fever or other epidemic disease to 'fall rapidly into hopeless collapse'. For an Edinburgh-trained physician to find a significant psychosomatic component in cholera predisposition is not odd.^{5,19} More interesting is the tone and the identity of the attributes. Replacing a sympathetic rendering of the despair and depression that lead to fever is Sutherland's exhortation: bravely pitch in and co-operate and you will live. In Charles Kingsley's novel *Two Years Ago*, set in the next (1853–1854) cholera epidemic, these themes loom large. Those who succumb (or at least some of them) are those who briefly drop their trust in God and duty to others to indulge their fear (or escape it with other indulgences).²⁰ Without impeaching any empirical or theoretical grounds Sutherland may have had for associating death from cholera with anxiety and apathy, one might wonder about the direction of causation: surely if one doubts the motives and competence of the state and the medical profession, sees one's neighbours dying no matter what is done, unco-operation, anxiety, and apathy is quite understandable.^{21,22} On the one hand, Sutherland was appealing to a very old motif: that our state of grace (or in Calvinist terms, election) is manifest by fearlessly

doing God's work in the face of danger. (Notably, his discussion of the sudden, 'struck by God', character of cholera immediately follows this discussion.)²³ On the other, he and other sanitarians were fully persuaded of the centrality of self-help. In this and other matters people must somehow—acting both communally and individually—deliver themselves from their situation.

Cholera from 'a social point of view'

The final portion of section one juxtaposes what may seem a number of disparate determinants of the disease. First comes a relatively weak association of cholera attacks with night-time. This is consistent with the landslide model Sutherland employs—of predisposing causes mounting until the body is precipitated into full blown cholera—but it does not clearly contribute anything to prevention: we can't avoid night. Next comes the compendium of social and structural sanitary failings—'overcrowding', 'dampness', 'filth' (a euphemism that defeats further precision), 'want of ventilation and atmospheric pollution' (which might be seen to act quite differently to effect a common outcome), 'nuisances' (which include graveyards, pigsties, and 'offensive sewers'), then qualities of the natural and built environment, including bad water and 'confined' neighbourhoods. In a futile search for inclusivity, Sutherland gets ever vaguer, and ends with 'other similar causes'. It is here that a modern epidemiologist is likely to become impatient. Sutherland lumps, where any effective preventive will require splitting, so we can concentrate on the most important factors. In any 19th-century town, and in much of the countryside as well, one could hardly fail to find one of these cholera causes. The standpoint of predisposition, however, while it may in principle suggest that there are greater and lesser influences, does not imply that some (or even one) will be guilty and the others innocent. What Sutherland finds important is that while all these will always undermine health and cause weakness, during this epidemic, they are also killing *healthy* adults. And even those who do not live in such areas may come down with cholera upon visiting them, even if they never come in direct contact with victims of the disease.

The generalization with which he closes is both a model of a pathological process and an admonition about the interconnectedness of society. The model is the zymotic model that William Farr was developing from the organic chemistry of Justus von Liebig. It would be the dominant model of pathological process well into the 1870s. 'It appears as if some organic matter, which constitutes the essence of the epidemic ... has the power of changing the condition of ... [other organic matter, living or non-living] as to impress it with poisonous qualities of a peculiar kind similar to its own.' The metaphor linked the sanitarians' concern with rotting (or 'rottable') matter with the phenomenon of the apparent multiplication of the poison, which was observed in epidemics of contagious disease and later held as a proof of an organismic agent. Sutherland poses it as an alternative to an inorganic poison where the relation of response to dose will be simpler. As admonition, the metaphor makes clear that any hope (that the literate classes, who presumably will be the readers of his report, may have) of escaping the influence of the poison is illusory. One cannot rely on the dilution that would come with distance, since, in appropriate environments, the poison is constantly 'multiplying its force of

attack', and subverting society. In the hands of later writers, this special something, this temporary state of pathological replication, would increasingly be materialized, first as a ferment, then as a germ, finally as a microbial species.^{12,16,24}

Bad water

At the beginning of section three Sutherland tells us that 'a number of the most severe fatal outbursts of cholera were referable to no other cause except the state of the water-supply', and a sentence later that 'the predisposition occasioned by the continued use of such water is perhaps the most fatal of all.' He then reviews the case of a Salford neighbourhood, which seems almost to have been designed to test the hypothesis of water-borne disease: 90 houses, a third using water from a single well, suspected by the residents themselves, the others using other water sources. Among those using the suspect water there were 19 diarrhoea cases, 26 cholera cases, and 25 deaths; among those using other waters there were 11 diarrhoea cases and no cholera. Edwin Chadwick, in his portion of the cholera report (ref. 1, pp. 59–62), cited a similarly remarkable case in Hackney, where several cesspools were dug near a well used by 85 people. All of those who did not use that well water (22) remained healthy, while 46 of the 63 who did were struck by diarrhoea. And yet, both Sutherland and Chadwick saw water as only a predisposing cause.

It does not take elegant statistics to see a strong relationship here. Surely the rational thing for Sutherland to do will be to ask if bad water may also be responsible in other cases. But he does not. His reasons should be evident by this time, but let me develop the structure of Sutherland's reasoning with regard to this case. As noted, Sutherland is interested in the interaction of two constitutions: about the epidemic constitution, he can say very little definitive, beyond that it is the underlying entity whose changes are manifest in the appearance and disappearance of cholera. Its existence is required on the assumption that an effect must have a cause. A non-constant effect (cholera) cannot have a constant cause without violating that canon of causation. Bad water, more constant and widespread than cholera, cannot therefore be the cause of cholera (unless one appeals to the arbitrary auxiliary hypothesis of some temporary and unmeasurable kind of badness to allow the tautology that water causes cholera when it contains cholera matter). But Sutherland seems oblivious to this option. Instead, he is concerned with the 'continued use' of water whose impurity is plain to him—and to local residents—even if not to chemists.²⁵

The other constitution is that of the individual. Sutherland is interested in the people who take cholera. Their cholera, and in particular, its course, is the only important register of the causes to which they have been exposed. The problem of correlating rates of exposure to certain causes with rates of cholera is relatively uninteresting to him. The reason is that all pathological causes are presumed to produce the same effect: a weakening of vitality, which, on the occasion of the epidemic constitution of cholera, will appear as a case of that disease. Thus kinds of causes are not important, but only their relative strength. Water's danger comes from its strength as a cause of *death* not as a cause of cholera: 'the proportion of deaths to attacks has generally been much greater in epidemic seizures resulting from it than from any other predisposing cause.'

The alternative approach, associated with John Snow, would focus on infective agents moving through particular media and acting as single causes. It would privilege a level of explanation intermediate to the two that Sutherland recognizes, and, initially, would require chains of *ad hoc* resolutions to the questions Sutherland finds important of the occasion of the epidemic and the variety of individual responses. To the question of how the agent comes to be in the water one might respond, as Snow did, that it came from evacuations from previous sufferers. As to why it should not be permanent, one can appeal to its inherent impermanence or mutability (though one then has to figure out how it starts up again some years later). As to why the agent does not identically injure all who drink a contaminated water (as any good Newtonian force should do), one can posit its particulate character, which makes exposure probabilistic, the necessity of a threshold dose, and certain occult qualities of resistance: i.e. immunological status. And, to explain cholera among those who have not consumed the water, one can posit supplementary modes of transmission through person to person contact or fomites. We may recognize that all of these suppositions are more or less readily capable of confirmation. In retrospect, having confirmed them, we may conclude that it is rational to expect to confirm them, and thus to hold a hypothesis that requires such suppositions. At the same time, we should recognize that some would be wary of embarking on a trajectory of explanation that demanded so many supplementary *ad hoc* explanations. One may complain that Sutherland's epidemic constitution is *ad hoc*. It is, but only insofar as he endows it with particular properties, and this Sutherland avoids as much as possible: the epidemic constitution is less explanation than empirical constant—it must exist for the epidemic to occur, but we know very little about it.

Overdetermination and sanitary reform

My final point concerns Sutherland's reasoning in section seven on 'Defective Sanitary Alterations'. There he relates the case of a Bristol neighbourhood of crowded back-to-back houses adjacent to an overused graveyard, with bad water, ventilation, and sewerage, and concludes that 'it would be difficult for human ingenuity to contrive and arrange a set of conditions more thoroughly unhealthy, or more likely to predispose the inhabitants to epidemic disease'. And yet, as part of a diatribe against a form of drainage system Chadwick had rejected (the conduct of a drain beneath a dwelling), Sutherland tries to explain differential mortality on the two sides of Wellington Court. He notes that a 'death took place in every house under the floor of which a drain passed'. In fact there are only four such dwellings among 66; cholera cases and deaths were certainly not preponderantly in these four. The problem with classical epidemiology is that while it warranted many actions to improve health it privileged none. Where the total environment is implicated in disease causation, decisions about what parts of it to alter will reflect assessments of probity more than efficacy (equally, it will be hard to assign blame or liability, if one can always point to multiple factors). Chadwick would focus public health on so-called 'physical causes', implicitly distinguishing these from social, economic, or political causes that were not subject to sanitarian interference.^{9,26,27} Among physical causes he privileged sewers and water (and the water was mainly to

keep the sewers flushed). By contrast, the overcrowded housing that many physicians saw as a larger obstacle to overall health remained on the margins of the public health agenda, with a few notable exceptions: housing was a market phenomenon; in the absence of subsidy for affordable satisfactory housing, to relieve crowding in one place only exacerbated it in another.²⁸

Conclusion

The general issues with which Sutherland struggled persist. Epidemiology, in its intersection with public health, continues to be both philosophical and political. The work of Wilkinson and others reminds us that health status indeed reflects the integration of multiple factors, manifested, for example, in social inequality.²⁹ Risk factors have replaced predisposition in many domains (even though probability is a quite different concept from partial causation). The several questions of epidemiology have become more clearly distinct as distinct means to address them have evolved. There is still tension between what is scientifically warranted and what is politically feasible, but, given the power of public health as a scientific imperative, the boundaries of feasibility can and do shift.^{30,31}

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