Original Article

Cholera, canals, and contagion: Rediscovering Dr Beck's report

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Abstract Cholera first appeared in North America (in Montreal and Quebec) in 1832 and spread rapidly across the eastern half of the continent. The dispatch of American disease control experts to Lower Canada in anticipation of cholera's spread implies that medical professionals expected spread, possibly from contagion, even though the notion that cholera was contagious was disparaged in medical writings of the time, and would be until John Snow's landmark work in London in the 1850s. Snow's insights derived largely from his observations on spatial and temporal patterns of cholera cases. We discuss a document from the 1832 epidemic, the report of Dr Lewis Beck to New York's Governor Throop, which anticipates Snow in presenting geospatial data that imply cholera's contagiousness. Beck shows that the movements of immigrants along the newly completed New York state canal system resulted in sequential cholera outbreaks along the canal's path. Although aware of the degree to which this suggested contagion, Beck argues strenuously against the contagiousness of cholera. We explore the social context of early nineteenth-century medicine that probably led Beck to disbelieve his own observations, and to favor a medical model inconsistent with his data. Themes that emerge from our inquiry include belief in disease as a physical manifestation of defective morality, stigmatization of the poor and immigrant groups, and reluctance to overturn prevailing medical models that themselves reflected the economic position of medical practitioners. We show that these themes continue to serve as obstacles to innovation in medical and public health practice today.
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Introduction

Cholera remains a major global public health threat. A severe diarrheal illness, caused by toxigenic strains of *Vibrio cholerae*, cholera is endemic throughout much of the world,[1] and causes sporadic outbreaks in regions with inadequate access to safe drinking water and sanitation.[2,3] Even in the modern era, cholera can have a case-fatality rate in the range of 14 to 23 per cent.[4,5] Cholera has pandemic potential: antigenic shifts in dominant *V. cholerae* strains are associated with the emergence of global epidemics with high case-fatality rates. The seventh known cholera pandemic started in 1961 and is still ongoing. The causative organism, *V. cholera* O1 biotype El Tor, has replaced the classical strain as the leading cause of endemic cholera worldwide.[6] As events in Haiti in the autumn of 2010 have demonstrated, lack of universal access to clean water and sewage treatment in low-income countries means that this disease remains a major public health priority.[7,8]

While cholera continues to be a major source of mortality in low- and low-middle income countries, it may also threaten higher income countries via disease importation.[6,9] Khan and colleagues have recently commented on continued importance of global 'connectedness' to disease emergence and spread, particularly given modern air travel.[10] A single person with the disease may spark a local epidemic in an area previously free of that pathogen. Travelers import cholera into Canada and the United States with some frequency (<12 cases per year between 1996 and 2007)[11] but the relative wealth of water- and sewage-treatment infrastructure in these countries appears to limit epidemic spread.[12]

Travel's role in diffusion of infectious diseases predates air travel. Indeed, the way infectious disease epidemics spread tells much about indirect connections between populations with little awareness of one another's existences. Fenn, for example, demonstrated that smallpox epidemics in the late eighteenth century showed a network of contacts spanning the American continent decades before Lewis and Clark's cross-continental expedition.[13] Late nineteenth-century American disease control experts recognized the importance of travel and transit in the genesis of cholera epidemics. In their efforts to forestall or prevent cholera epidemics in US cities, they focused on Hamburg, then a major embarkation point for people crossing the Atlantic.[14]

Recognition by mainstream medical practitioners of the transmissibility of cholera emerged largely from John Snow's work on London

cholera epidemics of the 1840s and 1850s,[15–17] and lagged behind the widespread popular belief that the disease was contagious.[18] During the cholera epidemics of the early nineteenth century, physicians in Europe and North America disparaged fear of contagion as superstition. They admonished the public to attend to its 'moral constitution' to prevent disease. As Richardson notes, cholera was 'hardly regarded as a biological entity but a means of retribution upon the morally suspect'[19] –a product of the interaction between a corrupted atmosphere (a 'miasma') and exciting characteristics in the victim. Drunkenness, gluttony, sexual depravity, and any other form of 'intemperance', were held to increase vulnerability to cholera,[20,21] as were both fear of cholera[22] and lack of fear of cholera.[23]

Economic liberalism, prevalent in professional classes, may have encouraged physicians to disparage the notion of cholera as contagious. Designating cholera as contagious would have, as a corollary, implied that disruption of trade and travel with via quarantine would be necessary to control the disease.[24,25] State-directed disruption of individuals' economic activities was unattractive to an emerging and increasingly independent-minded professional and merchant class.[24,25]

We discuss a document about the 1832 cholera epidemic, presenting geospatial data that imply cholera's contagiousness. These data might have allowed the report's author to intuit the transmissibility of cholera well in advance of Snow. And we explore the social context that led the report's author to disbelieve his own observations and favor a medical model inconsistent with his data.

Spatial Diffusion of Cholera in 1832: Canals and Commerce

The 1832 cholera epidemic was the first recorded appearance of cholera in North America. It's first victims were immigrants who died of cholera after disembarking in Quebec City and Montreal.[22,26] Cholera spread south and west largely along waterways, the major commercial arteries in the 1830s. New York State's newly completed Northern and Western (Erie) canals provided a conduit for diseased individuals to move, bringing cholera with them.

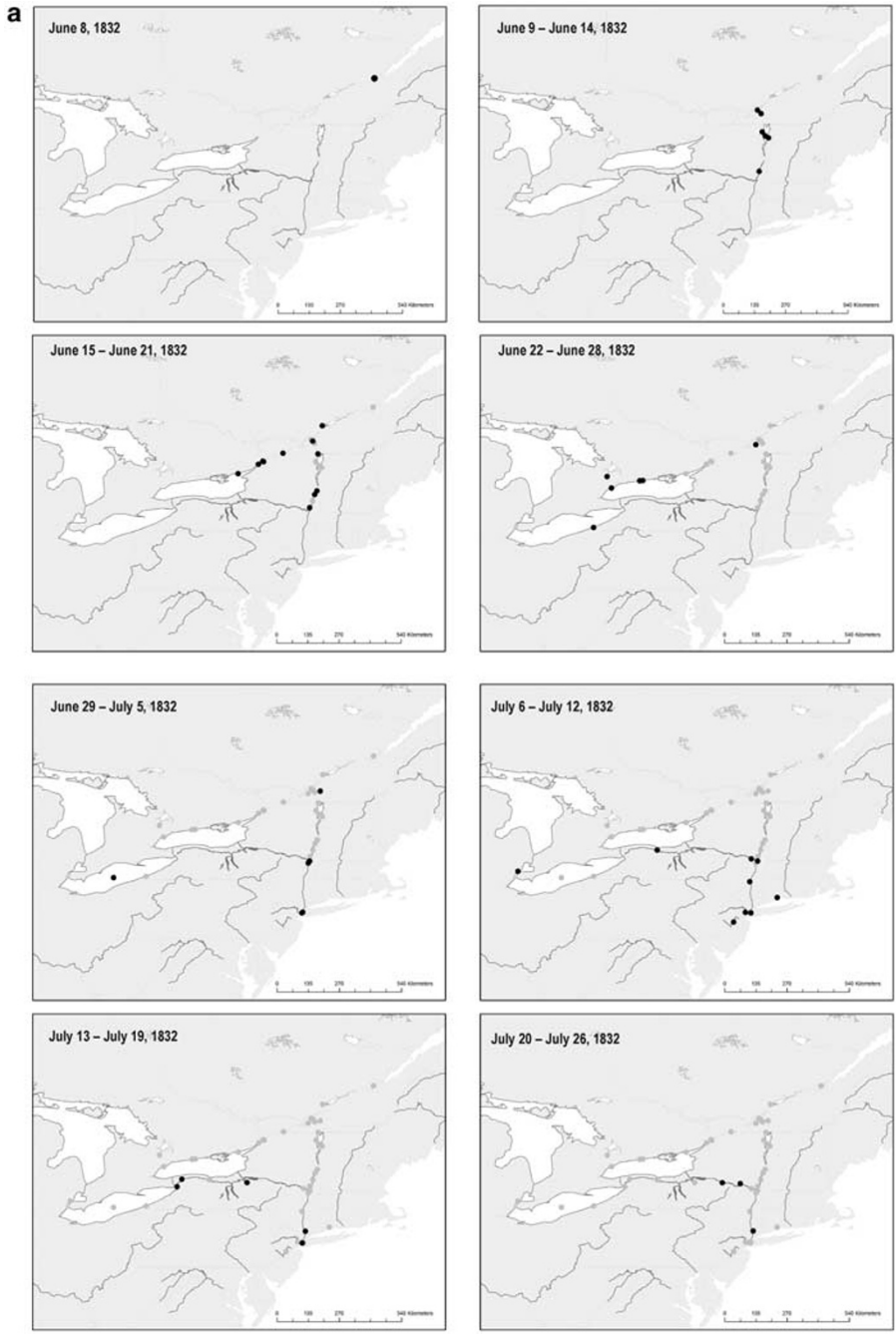
New York State completed its canal system in the mid-1820s, a massive engineering project linking the Hudson River Valley (with a southern terminus in New York City) to the Great Lakes at Buffalo,

and to Lake Champlain via the Northern canal. Laborers, many of them immigrants, cut through old-growth forests, and traversed the formidable Niagara Escarpment to complete the four hundred mile canal in just eight years.[27] The canal's ability to move goods and people between the New York seaport and the state's vast trans-Appalachian interior was credited for New York's rise to economic preeminence. In 1817, Elisha Williams, a politician, presciently described the canal as 'a river of gold [that will] flow into her [New York's] lap'.[28]

Dr Lewis Beck's painstakingly detailed report to Governor Enos Throop describes spatial and temporal patterns of cholera spread in New York and surrounding states.[22] Beck's 1853 obituary in the *The American Journal of Science and Arts* described him as an accomplished physician, botanist, and geologist who wrote an important treatise on the detection of contamination in medications.[29] In 1832, Governor Throop appointed Beck, based in Albany, his advisor on cholera. By August of that year, he had prepared an extensive report on the movement of cholera through the state.

Beck traveled to affected locales, and compiled information and statistics from physicians and local boards of health. The report described not only the dates and locations where cases were first observed, but when available, such details as the total number of cases and deaths, noting other characteristics he deemed to be relevant, including ethnicity, immigrant status, what victims had eaten prior to falling ill, and 'moral constitution'. Beck describes, for example, the first case in Plattsburgh: an immigrant who had 'been much exposed to wet and cold, and had eaten voraciously shortly previous to the attack'. Nearby cases were confined to 'a filthy part of the village, and to persons of irregular habits'. Despite several household members becoming ill, Beck points out that 'clergy, physicians and those who were in attendance on the sick, unless rendered susceptible by over exertion, generally escaped'. In Rochester, Beck noted that cholera cases were 'not confined to the intemperate, or even generally among that class'.

When we displayed Beck's data on a map, we found that outbreaks occurred sequentially, starting with the disembarkation of infected individuals from boats plying the canals and rivers of New York (Figure 1/Movie, http://www.youtube.com/watch?v=mKa7vbsfTic). This pattern was difficult to explain if cholera were not contagious. Indeed, Beck conceded as much, writing that the manner in which cholera moved with immigrants and laborers along waterways 'seem[s] at first sight to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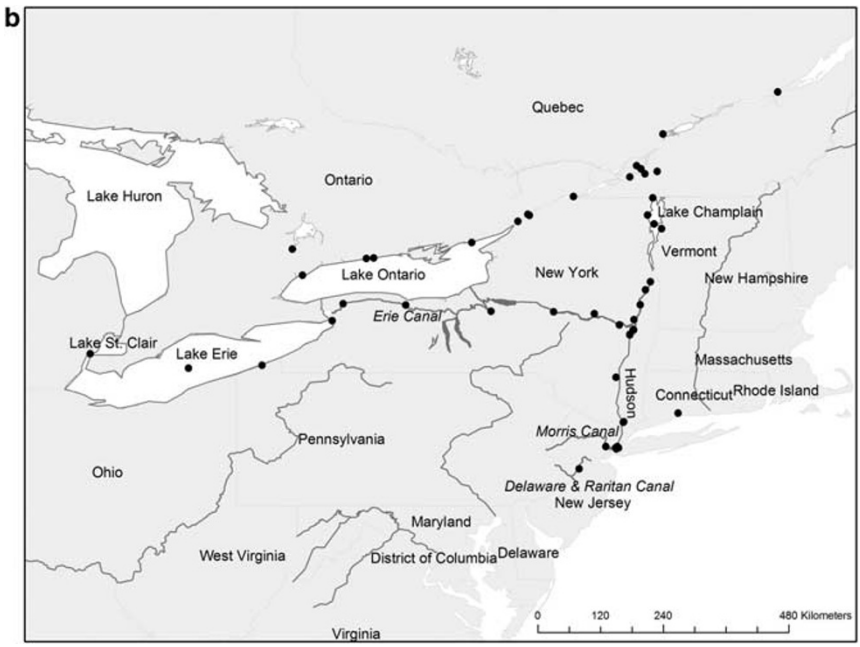

Figure 1: (**a**) Maps showing the weekly progression of the 1832 North American cholera epidemic, as reported in Lewis Beck's Report on Cholera.[22] The first North American cholera cases were reported in Quebec on 8 June and cholera rapidly diffused across the Northeastern United States and Canada, following major waterways. Dates of case occurrence are indicated. On each map, the locations of newly reported cholera cases are indicated by black circles, while locales where cases were reported in previous weeks are indicated by gray circles. (**b**) Map showing all locations reporting cases of cholera. States, provinces, and major waterways are labeled accordingly.

favor the idea that cholera is contagious, or directly communicable from man to man…'. But he continued, 'that cholera is not contagious can be proved by a multitude of facts'.

Beck's Failure of Insight and Prevailing Medical Models

A major line of evidence pointed by Beck, and by contemporaries like Daniel Drake,[30] in refuting the contagiousness of cholera, was the low attack rates among cholera patients' care providers. As Beck writes, '[i]n Quebec I believe only two physicians died of the disease; in Montreal one; in New-York five or six; in Albany not one'. Chilling

numbers by current standards, but mortality due to contagious disease constituted an important occupational hazard among contemporary physicians and nurses.

Beck also noted geographical discontinuities in cholera spread. Arguing against contagion, the disease spared some villages and towns despite close commercial ties to centers where cholera raged.[22] Were Beck and his contemporaries led astray by the variability in the clinical presentation of cholera, and by the likelihood that it would have been under-reported or unrecognized in some jurisdictions? Perhaps, given its stigmatizing nature, intentionally so.

The heterogeneity in severity of cholera cases was a source of confusion as late as 1892, when Max von Pettenkofer, the preeminent German hygienist, and several of his colleagues, drank flasks of *V. cholerae* to disprove Robert Koch's contention that *V. cholerae* was a specific etiologic agent of cholera.[31] Von Petterkofer developed diarrhea, and two of his colleagues developed cholera, but the heterogeneity in presentation was sufficient for von Pettenkofer to claim vindication.

Beck's report preceded John Snow's classic work on the transmissibility of cholera by over two decades. Yet basic elements of Snow's theory, based on spatial and temporal patterns of cholera deaths, were present in Beck's report. Snow saw that cholera deaths, which clustered in space and time, were not distributed in a manner that one would have expected if the disease occurred as a result of miasma. Snow's studies of gasses (derived from his career as a pioneer anesthetist) gave him a profound understanding of the expected behavior of a 'miasma', against which he was able to compare empirical data on cholera deaths in London.[32] That Beck began his discussion by emphasizing that cholera is not contagious, despite appearances, underlines the striking degree to which the patterns he recorded suggested that it was.

Beck's contemporaries, including Drake and Amariah Brigham, emphasized the movement of cholera epidemics along shipping routes as evidence contradicting the miasmatic nature of cholera, with Brigham suggesting that such patterns supported contagion.[21,30] Drake, ingeniously, posited that invisible microbes could be responsible for the spread of cholera. While he argued against contagion, he thought transmission could occur by 'poisonous, invisible, aerial insects, of the same or similar habits with the gnat', and drew an explicit analogy to 'intermittent fever' (malaria).[30]

Why was Beck unable to make the intellectual leap that would have allowed him to intuit the mechanism responsible for cholera's movement across the state? He had no knowledge of microbes, but the same could be said of Drake, and indeed, of Snow, who posited the existence of microbial pathogens based on the patterns of the epidemics they observed.

Ackerknecht examined the intersection of disease etiology theories and political ideology, proposing that an individual physician's stance – contagionist or anti-contagionist – related to his socio-economic position and attitude toward state regulation.[24] Anti-contagionists tended to be more liberal, suspicious of state interventions, and against quarantines and other measures that disrupted trade.[24,33] Beck and his political masters may have been aware of the violent unrest created by the attempts of (contagionist) Russian regions to (unsuccessfully) contain cholera's spread using military cordons.[25]

Beck was a political appointee. Could he be relied on not to disrupt the status quo? Beck's admonitions seem directed at maintaining order and social cohesion in a jittery population. He stresses the impact of fear on susceptibility to cholera: 'those who remain firmly at their posts, in most instances are safe, while those who ignobly desert them, are sometimes among the first victims'. Beck's writing suggests something familiar: the physician who is able to avoid drawing inconvenient inferences that are at variance with the currently accepted medical model.

David Wootton has written extensively on the subject of non-innovation in medicine. He notes that medical training, acquired via substantial personal effort and cost, may result in a reluctance to embrace novel ideas that render these skills and knowledge obsolete.[34] Wootton also notes that the Hippocratic element in medicine was still vital to medical thinking. It regards every case of disease as resulting from a unique interaction between causal influences in the environment and the constitution and 'exciting factors' in an individual. The groundbreaking work of Pierre Louis on phlebotomy for pneumonia, which effectively created clinical epidemiology by 'grouping' patients with similar afflictions, was not published until 1835.[35]

Clusters of cholera cases among the poor and laborers implied that predisposing factors were concentrated in these populations. Moral deficiencies, held to be characteristic of the 'unworthy' poor – those impoverished due to defective morality – fit this model well. Beck noted the large numbers of affected immigrants from Ireland and Wales:

'being often times in the most filthy state, and by their habits and exposures very liable to attacks of the disease'. Cholera struck 'filthy part[s] of the village [and] persons of irregular habits' in Plattsburgh; a 'very intemperate' individual on a Lake Champlain steamer; 'the intemperate' in Albany; a laborer who 'was very intemperate in his habits' in Buffalo, and so on. Gluttony was also an important risk factor: Beck 'witnessed a case at Whitehall … undoubtedly brought on by the eating of a large quantity of green peas'. An outbreak in the 'Dutchess county alms-house occurred after the inmates had eaten immoderately of cucumbers and other vegetables'.

The moralistic underpinnings of medical explanations of cholera were in keeping with the spirit of the times. The 1832 cholera epidemic occurred during the Second Great Awakening, a time of fervent Christian belief that gave rise to many new evangelical denominations.[36] Human perfectibility was a hallmark of the return to fundamental Christian ideas in the 1832 milieu.[37] Disease was a consequence of moral debility and the violation of natural laws, and was independent of broader social and economic conditions; accordingly, the benefits of abstinence from excesses of food, drink, and sex, accrued to both the physical and spiritual self.[37] Benjamin Rush's *Inquiry into the Effects of Spirituous Liquors on the Human Body and the Mind*, with its 'Moral Thermometer' and other late eighteenth-century tracts illustrate graphically the tendency to equate moral consequences with physical health.[38] In this framework, cholera's progress could be conceptualized as the earthly manifestation of God's justice.[20] Consequently the epidemic should not be a cause for alarm to those upstanding and prosperous citizens who lacked predisposition for the disease (Figure 2).

Under such circumstances, cholera in locales where a homogeneous, upright population might be expected to dwell would be problematic. Beck describes cholera in New Jersey is as follows: 'July 11th – Among laborers on the canal between Millstone and Griggstown, New-Jersey'. What is important here is what Beck does not say. Along existing waterways, the midpoint between Millstone and Griggstown lies at Princeton. Princeton was the seat of a Presbyterian university, as well as the affiliated Princeton Theological Seminary. The occurrence of cholera in a locale populated largely by current and future clerical leaders was problematic.

A Princeton minister (James W. Alexander) appears to have been extremely concerned at the implications of a cholera-susceptible

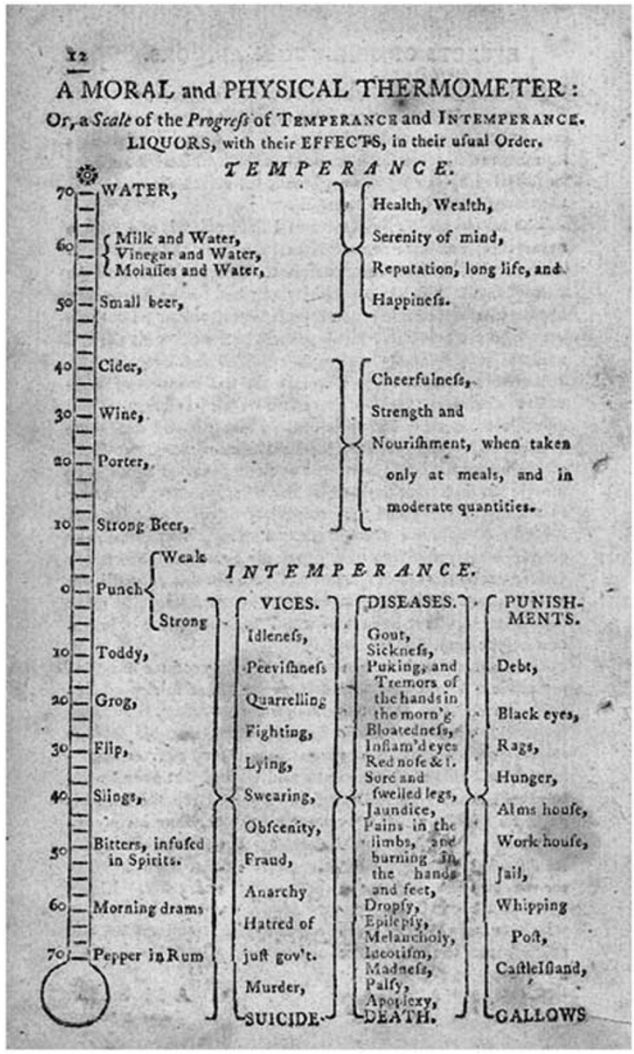

Figure 2: Benjamin Rush's Moral Thermometer, showing the association between different 'liquors' and levels of intemperance. From: Rush.[38]

population in a town full of divines. In a letter to a colleague on 4 August, he wrote: 'By this time perhaps you have seen in the New York papers, that Cholera rages in Princeton. Through Divine Mercy this is not true. There have indeed been three deaths of Irishmen in the

town, and nearly twenty on the neighbouring canal. Great uproar has been occasioned by some cit[izens] who are rusticated here, and who condemned the little Health-Board for having a hospital within the borough'. Again, if cholera is not contagious, why the fuss? 'The disease is at Scudder's mills, 3 miles; and Kingston, 3 miles; all cases Irish Catholics'.[39]

Modern Echoes – Predestination, Risk, and Relative Risk

Beck seems to have chosen to disregard the epidemiological data he collected – data which illustrated the contagious nature of cholera – because alternate models, ones in which moral corruption begat poverty, and moral corruption enhanced cholera risk, allowed him to explain high rates of cholera among the poor, and reaffirmed the correctness of prevailing moral and religious currents. The predictably high toll of cholera deaths in those at the bottom of the social ladder, while unfortunate, could nonetheless reassure their betters that their understanding of the relationship between moral and physical health was indeed correct.

From our current vantage point, this view seems constricted and cruel. But beliefs that gave rise to Beck's misinterpretation of cholera epidemiology remain prevalent, albeit outside mainstream public health and medicine. Perhaps the starkest recent North American example of disease being blamed explicitly on moral failings can be found in pronouncements around the genesis of the HIV epidemic. Evangelical clerics, like Rev. Jerry Falwell, described the AIDS epidemic as 'God's judgment because of the homosexual promiscuity in this land'.[40]

Punitive attitudes persist when risk factors for disease are regarded as a result of uncontrolled impulses. To quote John Kilwein, 'the health promotion movement has taken on the trappings of a new religion, one in which the body is worshipped and good health is often equated with moral superiority'.[41] Epidemiological predisposition may be subtlety conflated with predestination by medical and public health professionals. When epidemiological analyses identify an elevated relative risk of disease or adverse outcomes in a minority group (whether that minority status relates to underlying health conditions or social status), this may reassure the majority and diminish society's culpability for subsequent outcomes.

We propose that such confusion flows from conflation of relative and absolute measures of risk.[42] Conflation of predisposition and predestination was apparent during the recent influenza pandemic; reported deaths were often qualified as having occurred in individuals with 'underlying health conditions'.[43] Deaths in those without identified predisposing factors caused waves of extreme public concern.[44]

Conclusion

Lewis Beck's *Report on Cholera* represents a failure to innovate in public health. The implications of Beck's findings were obvious to him, but he failed to anticipate the later work on contagiousness of cholera by John Snow. The dominant theoretical model of the day was reinforced by contemporary religious beliefs regarding human perfectibility and resultant individual responsibility for health. Beck saw elevated risk as a manifestation of divine will towards sinners; under this framework, it would seem presumptuous for authorities to try to prevent cholera and thwart God's will. While many aspects of Beck's report are atavistic, the over-valuation of theoretical models of causation, and the degree to which predisposition can be conflated with moral judgment, may still challenge contemporary reasoning in medicine and public health.

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References

1. Griffith, D.C., Kelly-Hope, L.A. and Miller, M.A. (2006) Review of reported cholera outbreaks worldwide, 1995–2005. *American Journal of Tropical Medicine and Hygiene* 75(5): 973–977.
2. Gaffga, N.H., Tauxe, R.V. and Mintz, E.D. (2007) Cholera: A new homeland in Africa? *American Journal of Tropical Medicine and Hygiene* 77(4): 705–713.
3. Mintz, E.D. and Guerrant, R.L. (2009) A lion in our village – The unconscionable tragedy of cholera in Africa. *New England Journal of Medicine* 360(11): 1060–1063.
4. Tauxe, R.V., Holmberg, S.D., Dodin, A., Wells, J.V. and Blake, P.A. (1988) Epidemic cholera in Mali: High mortality and multiple routes of transmission in a famine area. *Epidemiology and Infection* 100(2): 279–289.
5. Siddique, A.K. *et al* (1995) Why treatment centres failed to prevent cholera deaths among Rwandan refugees in Goma, Zaire. *Lancet* 345(8946): 359–361.
6. Zuckerman, J.N., Rombo, L. and Fisch, A. (2007) The true burden and risk of cholera: Implications for prevention and control. *The Lancet Infectious Diseases* 7(8): 521–530.
7. Centers for Disease Control and Prevention (CDC). (2010) Cholera outbreak – Haiti, October 2010. *MMWR Morbidity and Mortality Weekly Rep* 59(43): 1411.
8. Chin, C.S. *et al* (2011) The origin of the Haitian cholera outbreak strain. *New England Journal of Medicine* 364(1): 33–42.
9. ProMED-mail. Cholera – Haiti (21): USA(FL). 20101117.4171, http://www.promedmail.org, accessed 10 January 2011.
10. Khan, K. *et al* (2009) Spread of a novel influenza A (H1N1) virus via global airline transportation. *New England Journal of Medicine* 361(2): 212–214.
11. Pan American Health Organization. Number of cholera cases in the Americas (1990–2008), http://www.paho.org/common/Display.asp?Lang=E&RecID=9812, accessed 18 June 2010.
12. Guerrant, R.L. (2006) Cholera – Still teaching hard lessons. *New England Journal of Medicine* 354(23): 2500–2502.
13. Fenn, E.A. (2001) *Pox Americana: The Great Smallpox Epidemic of 1775-82*. New York: Hill and Wang.
14. Bell, W.J. (1987) *The Age of Weir Mitchell. The College of Physicians of Philadelphia: A Bicentenial History*. Canton, MA: Science History Publications, pp. 169–195.
15. Snow, J. (1855) *On the Mode of Communication of Cholera*, 2nd edn. London: Churchill.
16. Snow, J. (1856) *Cholera and the Water Supply in the South Districts of London, in 1854*. London: Richards.
17. Snow, J. (1849) *On the Mode of Communication of Cholera*, 1st edn. London: Churchill.
18. Rosenberg, C.E. (1987) *The Medical Profession I. The Cholera Years*, 2nd edn. Chicago, IL: University of Chicago Press, pp. 65–81.
19. Richardson, R. (1989) *The Act is Uninjurious if Unknown. Death, Dissection and the Destitute*. Harmondsworth, UK: Penguin Books, pp. 219–238.
20. Rosenberg, C.E. (1987) *God's Justice. The Cholera Years*, 2nd edn. Chicago, IL: University of Chicago Press, pp. 40–54.
21. Brigham, A. (1832) *Treatise on Epidemic Cholera: Including an Historical Account of its Origin and Progress, to the Present Period*. Hartford, CT: H. and F.J. Huntington.
22. Beck, L.C. (1832) *Report on Cholera. Transactions of the Medical Society of the State of New York*. Albany, NY: Webster and Skinners.
23. Gilbert, P.K. (2008) *A Sinful Nation: 1832. Cholera and Nation: Doctoring the Social Body in Victorian England*, 1st edn. Albany, NY: State University of New York Press, pp. 17–28.
24. Ackerknecht, E.H. (2009) Anticontagionism between 1821 and 1867: The Fielding H. Garrison Lecture. *International Journal of Epidemiology* 38(1): 7–21.

25. Hays, J.N. (2009) *Cholera and Sanitation. Burdens of Disease*. New Brunswick, NJ: Rutgers University Press, pp. 135–154.
26. Jackson, S., Meigs, C.D. and Harlan, R. (1832) Philadelphia Board of Health Sanitary Committee. Report of the commission appointed by the sanitary board of the city councils, to visit Canada, for the investigation of the epidemic cholera, prevailing in Montreal and Quebec. Philadelphia: Mifflin & Parry.
27. Finch, R. (1925) *The Story of the New York State Canals: Governor Dewitt Clinton's Dream*, 1st edn. New York: New York State Engineer and Surveyor (republished by the New York State Canal Corporation).
28. Hosack, D. (1829) Memoir of De Witt Clinton: With an Appendix, Containing Numerous Documents, Illustrative of the Principal Events of his Life. New York: J. Seymour, http://www.history.rochester.edu/canal/bib/hosack/APP1C1.html, accessed 18 June 2010.
29. Silliman, B., Silliman, B. and Dana, J.D. (1853) Miscellaneous intelligence: Obituary. Death of Dr. Lewis C. Beck. *The American Journal of Science and Arts* XVI(2nd series): 149–150.
30. Drake, D. (1832) *A Practical Treatise on the History, Prevention and Treatment of Epidemic Cholera, Designed both for the Profession and the People*. Cincinnati, OH: Corey and Fairbank.
31. Morabia, A. (2007) Epidemiologic interactions, complexity, and the lonesome death of Max von Pettenkofer. *American Journal of Epidemiology* 166(11): 1233–1238.
32. Vinten-Johansen, P., Brody, H., Paneth, N., Rachman, S. and Rip, M. (2003) *Snow's Cholera Theory. Cholera, Chloroform, and the Science of Medicine: A Life of John Snow*. New York: Oxford University Press.
33. Rosenberg, C.E. (2009) Commentary: Epidemiology in context. *International Journal of Epidemiology* 38(1): 28–30.
34. Wootton, D. (2006) *Bad Medicine: Doctors Doing Harm since Hippocrates*. Oxford, New York: Oxford University Press.
35. Louis, P.C.A. (1835) *Recherches sur les effets de la saignée dans quelques maladies inflammatoires et sur l'action de l'émétique et des vésicatoires dans la pneumonie*. Paris: Librairie de l'Académie royale de médecine.
36. Jortner, A., Summers, M.W., Schweiger, B.B. and Ownby, T. (2007) Cholera, Christ, and Jackson: The epidemic of 1832 and the origins of Christian politics in antebellum America. *Journal of the Early Republic* 27(2): 233.
37. Jimenez, M.A. (1997) Concepts of health and national health care policy: A view from American history. *Social Service Rev* 71(1): 34–50.
38. Rush, B. (1790) *An Inquiry into the Effects of Spirituous Liquors on the Human Body and the Mind*. Boston, MA: Thomas and Andrews.
39. Jones, J.H. (1839) *Outline of a Work of Grace in the Presbyterian Congregation at New Brunswick, N.J., During the Year 1837*. Philadelphia, PA: Perkins & Marvin.
40. Jerry Falwell and Troy Perry debate morality of AIDS, http://archives.cbc.ca/health/public_health/clips/4563/, accessed 4 June 2010.
41. Kilwein, J.H. (1989) No pain, no gain: A Puritan legacy. *Health Education Quarterly* 16(1): 9–12.
42. Estellat, C., Faisy, C., Colombet, I., Chatellier, G., Burnand, B. and Durieux, P. (2006) French academic physicians had a poor knowledge of terms used in clinical epidemiology. *Journal of Clinical Epidemiology* 59(9): 1009–1014.
43. Groopman, J. and Hartzband, P. (2009) What's your underlying condition? *New York Times*, http://www.nytimes.com/2009/11/27/opinion/27groopman.html?_r=2&scp=1&sq=H1N1risk factors&st=cse, accessed 18 June 2010.
44. Picard, A. (2009) Why H1N1 panic is not the answer. *Globe and Mail*, http://www.theglobeandmail.com/life/health/h1n1-swine-flu/why-h1n1-panic-is-not-the-answer/article1341226/, accessed 18 June 2010.