The Incentives of Scientific Experts: Evidence from the History of Public Health

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Abstract

I examine the dual roles of experts – on one hand, as scholars responsible to a community of their expert peers, and on the other hand, as interested advisors to policymakers – using evidence from a large corpus of nineteenth-century medical research on cholera. Experts with links to Britain's overseas trade sector were less likely than experts without such connections to advance theories that cholera was a contagious disease (which had costly implications for British commerce). This difference is driven by the early part of the century, when a scientific consensus around how cholera spreads had not yet solidified. I argue that conflicted experts are more likely to act on their bias in low-information environments, when revealing new information can have a larger impact on policy. As a consensus forms, the value of hiding unfavorable information decreases, and even conflicted experts will reveal what they know to gain scientific credit.

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1 Introduction

A substantial theoretical literature suggests that experts can be affected by principal-agent problems that cause them to shade, spin, or withhold policy-relevant information. In practice, it can be difficult to systematically observe the effect of expert conflicts of interest on information revelation (and the resultant scientific progress) without the benefit of a good deal of hindsight about the "true state of the world," as ongoing debate within a scientific community can make it difficult to pin down the degree to which conflict skews views relative to the eventual consensus. A historical case study is especially well-suited for this task, as it allows us to examine the full trajectory of a scientific community's response to a novel, policy-relevant problem, from first encounter to eventual consensus formation.

This paper leverages such a historical case study from the history of public health. Cholera, a bacterial disease, spread from its origin in the Indian subcontinent to Eurasia for the first time in the 1810s, reaching the British Isles in 1831. Throughout the rest of the nineteenth century, periodic new waves of the pandemic caused millions of deaths across the globe (Durey 1979; Baldwin 1999). A crucial question for both scientists and the broader public was how cholera spread. If it was transmitted by human carriers from place to place, quarantines could provide an effective but costly way to combat disease risk. The costs of these policies fell most heavily on the shoulders of people and industries dependent on the free flow of trade, which would be interrupted by quarantines. If, on the other hand, cholera was caused by weather patterns, pollution, or other factors not directly linked to the movements of infected individuals, quarantines were pointless and could be dismissed as a potential policy choice.

Drawing on a dataset of nineteenth-century scientific research, I show that British scientists with known links to trade – for instance, those employed by the British East India Company or working in Britain's overseas colonial administrations – were more likely than their peers to attack the theory of contagious cholera. This pattern is driven by scientists active in the

early nineteenth century, when there was not yet a firm scientific consensus about cholera transmission and scientific methodologies were unsophisticated. By the latter half of the century, new scientific methodologies, including advances in microscopy and the development of controlled and natural experimental methods, tightened the consensus considerably, reducing both support for the "anti-contagionist" hypothesis overall and its disproportionate support among scientists whose careers were linked to British trade.

This paper contributes to a body of literature on epistemic communities, defined by Haas (1992b) as a "network of professionals with recognized expertise and competence in a particular domain and an authoritative claim to policy-relevant knowledge within that domain or issue areas" (3). The policy preferences of expert communities have been examined in the context of ecological policy (Haas 1989, 1992a; Peterson 1992; Allan 2017); telecommunications standards (Cowhey 1990); international aid (Hopkins 1992); global security (Adler 1992; Mendelson 1993); economic policy (Chwieroth 2007); and public health (Vogel 2013). In general, however, this strain of literature examines the behavior of such expert communities once a scientific consensus, and the resultant policy platforms, have already formed. I instead look at how policy considerations can interfere with the formation of a coherent consensus within a scientific community. Conflicted experts – those who benefit when others hold a certain belief about the state of the world – may choose to hide inconvenient information when the scientific consensus is weaker and when revealing it could lead to negative policy consequences. Under these conditions, experts who have uncovered information that is "bad" for their preferred policy position may suppress such information and pool with those who have no useful information to reveal. As a scientific consensus forms, the marginal effect of revealing information falls and the value of suppressing information decreases, so that even experts with a known conflict of interest are more likely to reveal what they know in order to capture scientific credit. Conflicted experts will not remain the last holdouts to an outdated but convenient theory; they can only gain from acting on their conflict of interest as long as there is sufficient uncertainty amongst their peers.

The COVID-19 pandemic demonstrates the importance of examining the motivations and actions of experts through a political lens. Policy-motivated experts – even those who are motivated by purely altruistic goals to improve welfare – may have reasons to shade their scientific pronouncements. There is evidence to suggest that in the face of initial uncertainty about the virus and its characteristics, policymaker-scientists allowed their policy goals to color their messaging about scientific evidence. For instance, in late February 2020, Jerome Adams, the Surgeon General of the United States, requested that Americans stop buying face masks in response to the threat of the virus, writing, "They are NOT effective in preventing general public from catching #Coronavirus, but if healthcare providers can't get them to care for sick patients, it puts them and our communities at risk!"[1] A year later, he expressed regret for having made such a "definitive message" but explained his earlier statement by stating that "what I said and what Tony Fauci really were saying was save the medical mask for the medical workers."[2] This incident is consistent with a model in which scientific uncertainty about the efficacy of masks allowed interested experts more latitude to optimize policy, highlighting the continued importance of considering experts as political actors.

2 Historical Context

2.1 A Scientific History of Cholera

Cholera, historically endemic to the Indian subcontinent, is a bacterial disease caused by the microscopic *Vibrio cholerae*. The first global cholera pandemic originated with an 1817 outbreak that followed trade routes with the movement of travelers (Selwyn 1977). For the rest of the nineteenth century, periodic cholera pandemics swept across Europe and beyond,

[1] Asmelash, Leah. March 2, 2020. "The surgeon general wants Americans to stop buying face masks." CNN Health. https://www.cnn.com/2020/02/29/health/face-masks-coronavirus-surgeon-general-trnd/index.html.

[2] CBS News. July 25th, 2021. "Transcript: Jerome Adams on 'Face the Nation."' https://www.cbsnews.com/news/transcript-surgeon-general-jerome-adams-on-face-the-nation-july-25-2021/. Note that per a January 2023 Cochrane Review, the efficacy of face masks with respect to slowing the spread of respiratory viruses remains unclear; see Jefferson et al. (2023).

causing particularly high mortality spikes in dense, unsanitary urban centers. The typical transmission mechanism of cholera is drinking water contaminated with fecal matter from infected individuals, some of whom may not show distinctive symptoms. Thus, cholera can be carried across long distances, but it does not usually spread through direct person-to-person contact. This indirect mechanism made cholera a medical mystery to European scientists for several decades after their first contact with the disease (Durey 1979; Johnson 2006).

The mysterious transmission mechanism of cholera created high stakes for policy. One of the few tools in the rudimentary public health arsenal, developed during past experiences with bubonic plague, was quarantine – of national borders, localities, households, or individuals (Baldwin 1999). If cholera was contagious in any sense of the word, quarantines could be a helpful countermeasure, although they required paying an economic cost. If it was spread by some mechanism that did not involve infected carriers, quarantines would be useless, imposing economic costs and infringing on freedom of movement with no health benefits.

Specific theories about cholera's transmission mechanism varied widely in the early decades of the nineteenth century. Roughly speaking, they could be divided into two camps – "contagionist" theories and "anti-contagionist" theories, to use contemporary parlance (Koch 2005). Contagionist theories of cholera included any explanations that hinged on cholera being spread by the movement of infected carriers. Some researchers, observing that cholera outbreaks followed common transportation routes, staked out a contagionist position without making any claims about the specifics of how transmission occurred. The anti-contagionist camp included many alternative theories. Some anti-contagionists focused on weather and climate conditions, pointing to atmospheric phenomena, electrical fields, or volcanic emissions as factors that could affect the course of a cholera epidemic. Others theorized that the disease was caused by deteriorating urban living conditions that produced dangerous "miasma" (or "bad air").

At first contact with cholera, scientific techniques were still primitive, especially in the

subfields of biology and medicine. Controlled experiments were in their theoretical infancy and, when put into practice, generated noisy results because scientists did not yet have a framework for thinking about confounding variables (Tröhler 2005). Tools for observing the natural world at the microscopic level were rudimentary. Formal medical training still incorporated ideas, such as the "four humors," that were inherited from antiquity. Mokyr (2011) describes "tight" knowledge as knowledge that is characterized by "confidence and consensus" (6). Medical science at the beginning of the nineteenth century was characterized by neither. There was justifiably low confidence in tools and methodologies, and as a result, weak consensus on many questions, including that of how cholera spreads.

In the absence of good tools to study cholera, it is easy to understand how the anti-contagionist position could have gained support. Early scientists not only lacked the ability to distinguish correlation from causation but also lacked a clear framework for distinguishing causal mechanisms from mediating and moderating factors. They could, however, observe the clear spatial and demographic correlation between cholera outbreaks and urban poverty (Koch 2005; Johnson 2006). Fecal contamination of drinking water – enabled by poor infrastructure in decaying cities – is a necessary condition for a major cholera outbreak, but poor nutrition and pre-existing disease loads make people particularly susceptible to illness and death conditional on ingesting the bacterium (Richterman et al. 2018). In the sense that cleaning up cities could mitigate disease outbreaks, anti-contagionists were not wrong, although they misunderstood the proximate mechanism of disease. Even more tenuous theories tended to have some basis in observational evidence and reasoning. The 1815 eruption of Mount Tambora spread a layer of volcanic ash over Europe, blocking out the sun and damaging crop yields. This natural disaster predated the first Eurasian cholera outbreak by only a few years, and its impact on the food supply may have indirectly made people more susceptible to disease (Oppenheimer 2003).

In 1848, John Snow, a prominent London surgeon already well-known for his innovations in

anesthetics, published his theory of waterborne cholera (Johnson 2006). Several years later, he published causally identified evidence for the theory using the famous natural experiments that leveraged variation in access to safe vs. contaminated water in London households. Meanwhile, improvements in microscopy made it possible to observe microscopic organisms and theorize about their causal role in spreading disease. The cholera bacterium was first identified by Filippo Pacini in 1854. Louis Pasteur's experiments disproving the theory of spontaneous generation of living organisms in 1859 and his subsequent development of pasteurization in 1865 provided additional evidence against variations of the miasmatic theories that held poor living conditions themselves to be the ultimate cause of spontaneously-arising disease. Robert Koch's 1884 confirmation of Pacini's earlier findings are usually credited as providing definitive proof of cholera's bacterial origins. Mokyr (2011) states that "the germ theory prior to Pasteur and Koch was untight. It might be true, but for contemporaries there was no way of knowing for sure [how diseases spread]. The triumph of the germ theory after 1865 should be regarded above all as a victory of scientific persuasion in which brilliant scientists were able to combine scientific insights with considerable academic prestige and a good understanding of how power and influence in the scientific community work" (184).

2.2 Cholera and Policy in Britain

Britain escaped the first Eurasian cholera pandemic of the 1820s, but by the summer of 1831, a raging epidemic in Continental Europe prompted worries that the disease could soon impact Britain. Following the precedent set by previous disease outbreaks, the government assembled a Board of Health comprised of prominent members of the medical community. In October 1831, acting on the Board's advice, top government officials enforced a quarantine of national borders, along with other measures to treat any sign of an outbreak as a contagious threat. These steps met with wide support in Parliament, not because there was a consensus that cholera was contagious, but because of the potential for a disaster if it were contagious and no preventative measures were taken. However, those members of Parliament who objected were

members of the pro-trade Whig party, while support for the quarantine among Conservatives, who tended to align with rural interests and of the landowning aristocracy, was universal.[3]

Despite these precautionary policies, the quarantine of the national borders was imperfectly administered, and the first case of cholera in Britain was reported in October 1831. By the time the epidemic had run its course in the autumn of 1832, about 30,000 people had succumbed. Further epidemics followed in 1848-1849, 1853-1854, and 1866.[4] Aside from its contribution to mortality in poor urban areas, the main symptom of cholera – gastrointestinal distress leading to death through dehydration – captured the public imagination; as an anonymous medical practitioner in Glasgow in the 1840s wrote, "There is no subject that excites so much interest in Glasgow as Cholera... Even if it were useful to prevent us from thinking of Cholera, it is impossible: for how can we help thinking of it, when our neighbours and friends are dying so suddenly around us?"[5]

Politically, an anti-contagionist interpretation of cholera benefited two constituencies. First, it was useful to those who depended on the free flow of trade and commerce and thus most directly bore the economic costs of quarantine policies. A contagious theory of cholera potentially justified not only quarantines of Britain's own borders, like those that had been enacted in 1831-1832, but international agreements to monitor ships across the globe for signs of disease and subject them to onerous holding periods. Secondly, it helped those who supported a broad agenda of welfare spending, infrastructural investment, and urban cleanup to reduce poverty and promote social reform. A theory of generally poor living conditions generating disease justified funding for improvements to cities, while one that pared down the link between poverty and cholera to a single factor such as contaminated water did less to justify a broad program of reform.

In fact, it was a desire to improve urban living conditions – specifically, those of Britain's

[3]Source: Author's tabulation of Parliamentary speeches from Hansard Parliamentary Database.

[4]Figure A1 shows mortality data for a selection of British cities during the years of major cholera epidemics.

[5]Quotation from an unnamed medical practitioner in *Asiatic cholera : its history and nature, with directions for its prevention and cure*, 1849.

ruling elite – that eventually brought cholera under control in London. In the summer of 1858, water and air pollution in the Thames became intolerable, disrupting the operations of Parliament. The "Great Stink" forced Parliament to allocate funds to a rebuilding of London's sewerage system. This major investment, coupled with improvements to water supply infrastructure, largely solved the problem of waterborne diseases in the capital city, as demonstrated by the comparatively low mortality rate of the 1866 epidemic (Luckin 1977).[6] Similar investments in other cities had a comparative effect on eradicating these public health threats.[7]

Despite these advances in domestic public health policy, Britain still needed to reckon with the implications of quarantine policy as a matter of global health cooperation. Beginning in 1851, a series of International Sanitary Conferences provided a venue for different countries to send representatives to debate and formulate international quarantine standards (Howard-Jones 1975).[8] Having made strides in attacking its disease problems with public investments in water and sanitation, and given its economic dependence on global trade, British authorities had even more reason to shy away from stringent quarantine standards, and the writings of British government officials express their dismay at the quarantine regulations that resulted from the meetings. For instance, an 1866 proposal to quarantine ships transiting from Asia to Europe for a period of ten days was passed over the objections of both British representatives to the conference, with nearly-unanimous consent from all other countries.[9]

[6]Most of these deaths occurred in an area of London where water infrastructure was improperly maintained.

[7]For quantitative evidence on the role of sanitation infrastructure (including both sewerage and chlorination) and clean water on reducing urban mortality, see Chapman (2019) and Aidt, Davenport, and Gray (2023) in Britain; Cutler and Miller (2005), Ferrie and Troesken (2008), and Alsan and Goldin (2019) in the United States; Gallardo Albarrán (2018) in Germany; Kesztenbaum and Rosenthal (2016) in France; and Ogasawara, Shirota, and Kobayashi (2018) in Japan.

[8]These conferences eventually developed into a permanent standing body, the World Health Organization.

[9]"Report on the cholera epidemic of 1866 in England" (William Farr, 1868).

3 Theory

3.1 The Motivations of Experts

What do experts want? This question underpins a substantial literature on expertise, technocracy, and principal-agent problems between policymakers (or the public) and the experts upon whom they rely. Historians and philosophers of science whose main goal is to explain scientific progress tend to take a particularly charitable view towards the experts class, assuming that they are motivated by intrinsic curiosity (Kuhn 1970; Popper 1972). This supposition likely does bear some weight: surveys and longitudinal studies show that those who select research careers tend to be more intrinsically motivated and open to new ideas (Lounsbury et al. 2012; Fosse, Freese, and Gross 2014). Other models of expert behavior assume they are motivated primarily desire for a good reputation amongst their fellow experts or their managers (Scharfstein and Stein 1990; Effinger and Polborn 2001; and Ottaviani and Sørensen 2001), or by a desire to affect the choices or behavior of those who hear the expert's pronouncement. In general, models in this latter family focus on how an audience can extract maximal information from experts when that expert may have some reason to withhold or misrepresent it – see, for instance, Crawford and Sobel (1982); Gailmard and Patty (2013); Backus and Little (2020); Andrews and Shapiro (2021); and Hoffman and Yoeli (2022). In turn, policymakers may not be interested in an expert's information *per se* so much as their ability to legitimate to policymakers' choices, particularly if policymakers have fallen prey to moral hazard with respect to the public (Downs and Rocke 1994; Fearon 1999; Canes-Wrone, Herron, and Shotts 2001; Majumdar and Mukand 2004; Ashworth 2012).

Empirical research demonstrates how policy incentives can create a dramatic departure the outcomes one would expect if experts (and, in particular, scientists) were motivated purely by curiosity. Carpenter (2003) finds that risk aversion with respect to public judgment causes the Food and Drug Administration to be overcautious when approving new pharmaceutical

products. Youde (2005) gives an account of South African public health researchers who promoted a narrative that focused on poverty as the ultimate cause of the African HIV/AIDS epidemic, in contrast to the Western focus on proximate medical risk factors for disease transmission. Youde attributes this difference to the policy context in which South African researchers worked: poverty alleviation was a major priority for the South African government, and South African public health scientists therefore tailored their messaging in order to maximize their impact with relevant policymakers.

First-hand accounts discussed in Section 2 discuss the policy considerations of scientists working on public health issues like cholera. At the same time, however, the British medical community clearly rewarded contributions to the stock of knowledge in the form of awards, election to prestigious honor societies, and friendly citations by other scientists. Experts working in this environment were rewarded for having a "good reputation" (making novel, defensible claims about important research questions) and, like modern scientists, can be assumed to have enjoyed intrinsic satisfaction for making new discoveries, but some also had policy implications on their minds. The rest of this paper examines the conflict between the policy incentives and the incentives for credit and discovery.

3.2 Elucidating Expert Incentives

When experts have both an incentive to garner professional credit by making discoveries and a policy incentive, how should we expect them to behave? In this section I lay out a theory that is formalized via a simple model in Appendix Section B.

I assume that experts are motivated to pursue discovery by professional incentives, both material (monetary compensation, career advancement) and immaterial (prestige). Experts who work on a policy-relevant topic may also have personal preferences over what policy is selected. In the public health setting explored in this paper, all experts studying public health benefit professionally by making a discovery and publicizing it; some – those working for

overseas trade interests – are harmed (via their employer) by the imposition of a quarantine that is disproportionately costly to their group and are thus conflicted.

Unconflicted experts have a maximal incentive to pursue their scientific research under all circumstances, as their payoffs are only affected by the number of discoveries they publicly reveal for credit. But consider a conflicted expert who makes an "inconvenient" discovery – evidence for contagious cholera that could be used to justify a quarantine. Will such an expert reveal or hide his discovery?

Hiding the discovery only benefits the expert if the professional incentives (credit for discovery) do not outweigh the downside of potentially supporting a "bad" policy. If the community's consensus is already sufficiently strong in the direction of contagion (the only position that would suggest quarantine), even a conflicted expert should reveal evidence in favor of contagion, since doing so could yield professional credit but could not meaningfully affect policy. If the consensus is strongly *against* contagion, the expert may also be willing to reveal some (weak) evidence in favor of contagion, since doing so could yield credit and shift public opinion somewhat in the direction of the inconvenient contagion hypothesis, but not by enough to trigger a policy change. It is only when the prior consensus over contagion is relatively weak – when the community prior is relatively balanced between the two states of the world – that a conflicted expert may withhold information and forego gaining professional credit to affect the policy outcome.

Under these theoretical assumptions and conclusions, we should expect experts' professional biases to be most salient in shading their behavior with respect to the withholding or disclosure of scientific results when uncertainty is high. This contrasts with other accounts of expert bias, such as that of Oreskes and Conway (2011), which study the behavior of a small set of experts who act on their biases to influence policy despite an existing scientific consensus. However, Oreskes and Conway (2011) explicitly discounts the constraining role of professional reputation, as they focus on "scientists in their twilight years who had turned to fields in

which they had no training or experience" out of financial or political self-interest (36). In contrast, I focus on active researchers who have something to lose if they forego making a contribution to the stock of community knowledge.

3.3 Connection to Case Study

During Britain's first domestic cholera epidemic in the 1830s, the government initially attempted to protect the nation with a quarantine of incoming ships, holding them for a period of two weeks at the border before they were allowed to proceed to British ports. Due to imperfect enforcement (or cases of long-duration asymptomatic cases on board ships), however, this strategy did not prevent cholera from eventually reaching a port city and spreading throughout the country. After domestic cholera cases began to appear, the government switched its focus to mitigation measures, including emergency funding for hospitals and programs to alleviate the economic effects of mass disease on the working poor. What role quarantines would play should future epidemics arise, however, was not obviously clear. This question was posed both at the domestic level (with respect to quarantines at British borders or within the country during times of disease) and at the international level (determining international agreements on policies regarding ships transiting through areas where disease was known to exist). Pro-trade interests bore the burden of paying the costs of these quarantines when belief in their efficacy was sufficiently high to justify the policy. As evidence for the contagious transmission mechanism accumulated – in Mokyr's terminology, as science became "tighter" – the consensus converged on the contagion explanation, and the incentive for trade-linked scientists to withhold information vanished.

Simultaneously, cities in Europe began to invest in more public goods in urban areas, including better water and sanitation infrastructure. These investments were driven both by growing recognition of the link between sanitation and health and by the increased political power of cities that could be leveraged for redistributionary policies to improve the quality of urban life (Lizzeri and Persico 2004; Aidt, Daunton, and Dutta 2010). They may have decreased

the policy costs to trade if the public accepted the contagion hypothesis: while quarantine policies could still hamper the flow of global trade, they were no longer a necessary strategy at Britain's own borders when sanitation effectively prevented the spread of disease at home.

Empirically, the theory outlined above therefore predicts that during the early cholera epidemics, experts with a political incentive to avoid a quarantine will avoid publishing information that could be used to support one, biasing the observed sample relative to the true state of (private) knowledge; this bias is not present when authors are incentivized to always share their information to maximize professional gains. Experts who find information that could be politically damaging to their interests will consciously discard this information (the "file drawer effect") or (perhaps unconsciously) find a way to interpret it more favorably. As it is impossible to see what information a given expert chooses *not* to publish, or the degree to which their interpretation of evidence was shaded by their policy preferences, I infer evidence of an expert's bias from the conclusions reached in existing publications that *are* released into the public record.

4 Data and Research Design

4.1 Data

Data on the stated views of the scientific community about cholera comes from the Medical Heritage Library (MHL), a digitized archive of the holdings of a consortium of universities, museums, and research institutions focused on the history of medicine. This ongoing effort is coordinated by curators at the U.S. National Institutes of Health, Harvard University's Countway Library, and others, who ensure that at any given time, the digitized sample is as representative as possible of the summed physical collections of contributors. Each file is available as both a PDF and an OCR-generated text file. This database is accompanied by a substantial body of metadata that typically includes, for each item, the author, title,

year of publication, city of publication, and a series of topic tags created by the contributing institutions. I restrict my attention to English-language publications from authors based in Britain (including British colonies) between the years 1800 and 1900 in order to obtain a sizable sample of authors who form a unified professional community. I identify 418 qualifying items that are *primarily* about cholera with individual attributable authorship (vs. institutional authorship) based on contributor-supplied subject tags in the metadata (supplemented with other untagged documents that contain very frequent use of the term "cholera.") Some documents are authored by multiple individuals. MHL metadata and/or the document itself typically offer basic biographical data about authors: the year of an author's birth and death; relevant academic degrees; and professional affiliations with various medical research societies. I verify and extend these author-level data using secondary sources (dictionaries of national biography and obituaries published in major medical journals). Appendix E (p. A7) gives an example of an original document included in the sample. Appendix F (p. A8) gives details of corpus construction.

Figure 1 shows the number of documents for each year within the sample. These documents include complete books, monographs, pamphlets, transcribed lectures, individual journal articles, and policy documents about cholera authored by individuals with scientific credentials. Advertisements, complete journal editions containing multiple works by multiple authors, and non-scientific works are excluded. The MHL archival sample is useful because it forms a representative sample of work that legitimate contemporary institutions believed was worth collecting and preserving. Almost all articles were written by people who, although the prestige of their professional affiliations may have varied, had a claim to expertise by the standards of the nineteenth century. Altogether, the database includes contributions from 323 unique authors, 64 of whom contribute to more than one article (see Appendix Figure A6 for the total distribution of count of articles by author). There are 30 co-authored articles; the rest are single-authored.

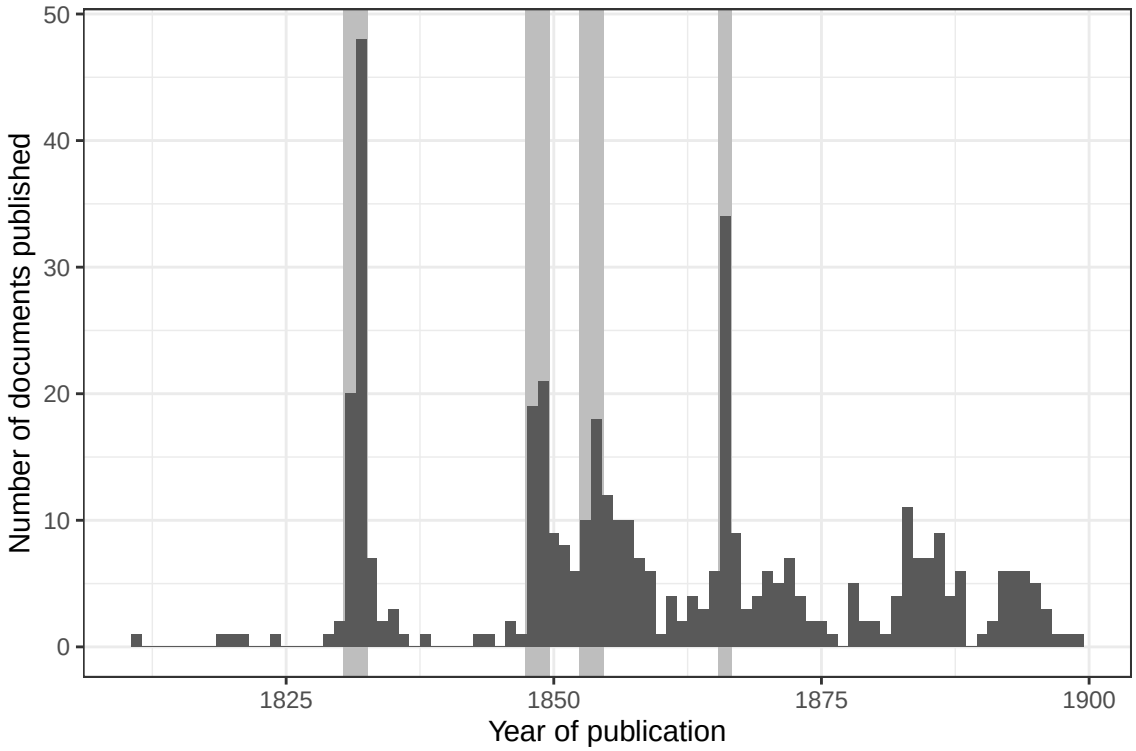

Figure 1: Number of documents in MHL sample by year of publication. Grey bars denote epidemic years.

4.2 Research Design

4.2.1 Explanatory Variable

Section 3.3 suggests a testable hypothesis: conflicted experts will act on their source of conflict, but only in when the environment is epistemically "loose" rather than "tight" (in the terminology of Mokyr 2011). As documented in Section 2.2, *policymakers* clearly understood the *costs* of following a contagionist framework for handling cholera, even if they relied on experts to inform them of the potential benefits. In the absence of sufficiently "tight" knowledge, experts who had personal reasons to avoid a strict quarantine policy might inject political considerations into their work. That beliefs about science were biased by concerns for Britain's trade economy was taken for granted by contemporary and retrospective observers. Writing in 1866, Edwin Lankester, a scientist involved in policy advisory, stated that "... there is evidence to show that... the contagiousness of certain diseases is not altogether unconnected

with political views. Thus during the early part of this century the free-trade party were most earnest in their efforts to induce the governments of Europe to abandon the quarantine of vessels proceding from infected ports."[10] Pelling (1978) writes that the landmark 1848 Board of Health Report, a major government-sponsored investigation into the poor living conditions in British cities, was characterized by "the subservience of every statement to the single aim of abolishing quarantine," (67) and that "a climate of anticontagionism was a *fait accompli*" (69).

I therefore focus on the free flow of trade as the primary policy consideration affecting an expert's potential to be policy-motivated. As policy preferences are impossible to observe directly, I rely on a proxy measure that simultaneously captures experts' proximity to policy considerations and their incentive to hold a particular political view: their association with interest groups linked to overseas British trade. I classify an author as "trade-linked" if he was employed as a medical expert by a private British trade interest group (such as the East India Company) or by a British colonial overseas administration. This variable captures both authors who were employed at the time of writing and those who identify themselves in in their work as having been formerly employed by a trade-linked organization (an indication that they retained this association as a primary part of of their professional identity). British colonial administrations were entangled with private British commercial interests; for instance, the British East India Company (EIC) effectively governed India on behalf of the British government in the first half of the nineteenth century and officially merged with the colonial government in the latter half, and some experts in the sample held dual appointments with both a colonial administration and the EIC. Twenty-seven documents were written by authors professionally based in overseas British colonies but did not have an identifiable role employed by a trade-linked organization; instead, they were employed in a private capacity as clinicians, hospital physicians, or independent researchers. I separately categorize these documents as "Colonial" but not necessarily "trade-linked." While it is possible that the authors who fall

[10]Lankester, Edwin. (1866) "Cholera: What Is It? And How to Prevent It." MHL ID b21472324, 15.

into this category have undocumented direct ties to British trade, or were socially influenced to fit into a professional milieu that included many trade-linked colleagues, I expect these experts to be less susceptible to the kinds of incentives that characterized the trade-linked experts.

An obvious question is whether one should expect an affiliation with the British government overseas to actually influence the scientific work or conclusions of experts who, after all, also benefited from increased reputational standing – and, for that matter, had at least some incentive to get the science right for the sake of saving lives. Retrospective historians (Watts 1997; Harrison 2018) tend to echo Lankester's assessment that the trade issue colored cholera politics, especially in crucial trade zones like British India. Certainly, not everyone always agreed with Lankester's grim assessment all of the time. In 1831, at the very start of the first British epidemic, a contagionist physician gave credit to the East India Company's efforts to gather data on the disease, writing that "The large majority of the facts contained in this volume are taken from the Reports on Cholera, which were compiled in India by order of the East India Company. For the opportunity of laying them before the public, I am indebted to the liberality of the Honourable Court of Directors, which has ever shewn an earnest desire to promote the interests of science."[11] However, in 1831, the perceived payoffs to getting the cholera question right were arguably higher than at any point in history, since it was not yet apparent exactly how severe an uncontained pandemic might be, and the eagerness of even the EIC to put aside any political and economic agenda mirrors the willingness of even pro-trade British Members of Parliament to accept quarantine laws until they realized how costly it would be. (For an analysis of the attitudes of MPs towards cholera and quarantines, see Appendix D on p. A6.)

[11]Kennedy, James. (1831) "The history of the contagious cholera: with facts explanatory of its origin and laws, and of a rational method of cure." MHL ID b21306345, 11.

4.2.2 Outcome Variable: Views on Contagion

The relevant outcome variable for understanding the link between policy and science in this setting must capture the implications of a scientist's stated view on cholera transmission with respect to quarantines. Thus, the most important feature to measure is whether or not an author's proposed transmission theory involves human carriers of disease who move from place to place, passing the disease to others as they go. I classify documents that argue for human-to-human transmission, directly or through some other medium (including water) as "contagionist." Documents that argue that cholera is spread by other mediums, such as weather patterns or environmental pollution, are classified as "anti-contagionist."[12]

I rely on keywords to identify relevant sections of each document but classify documents by reading the author's statements in their context within the document, an approach that precludes automated classification. To construct an outcome variable, I extract sections of text from each document that are within a 500-word bandwidth around any mention of one of five relevant keywords: "contagion," "infection", "communicable," "transmissible," and "propagate" (as well as any associated word stems). I then evaluate whether each section reflects a contagionist or anti-contagionist viewpoint. Documents containing none of these relevant keywords are hand-inspected in their entirety to make sure they do not touch on the topic of contagion using other vocabulary.[13]

Most documents explicitly adhere to a contagionist or anti-contagionist view. A minority do not fall neatly into one category or the other. Some acknowledge the debate over contagion but take an explicitly neutral view on the question or express the author's lack of certainty.

[12]Nineteenth-century medical vocabulary was neither as precise nor as standardized as it is today; the meaning of some relevant words shifted during the sample period or differed according to different authorities' precise definitions. Some scientists differentiated, for instance, between "infectious" diseases (those spread by direct contact with other victims) and "contagious" diseases (those that could be spread by indirect contact, for instance, via the clothing or bedding of patients). Appendix C gives further details of these subtleties, but for the sake of clarity, I will use the words "contagious" and "anti-contagious," as these were the terms most often used to capture the main theoretical division between the two camps.

[13]All documents that discussed contagion were independently re-coded for verification. For details on inter-coder reliability, see Appendix H.

Others do not mention the debate over contagion at all. Some documents that ignore the issue do so because they are focused on other aspects of cholera, for instance, diagnostics or therapeutic treatments, rather than speculating about its origins. Other authors may ignore the contagion question if they feel it is settled science and does not bear mentioning. Table 1 shows examples of each category (contagionist, anti-contagionist, and neutral), and Figure 2 shows the change in scientific opinion over time. For tractability, documents are categorized according to the epidemic with which they are associated.

Category	Source	Example
Anti-Contagionist	John Lizars (1832) "Substance of the investigations regarding cholera asphyxia : with cases and dissections" (MHL ID b21978499)	"[The author] hopes that their conjoint results will prove to the Medical Profession, and the Public at large, that Cholera is not a mysterious, but an explicable disease, and that it is as **free of contagion as a cut finger or an amputated limb** (Preface)."
Anti-Contagionist	Edmund Skiers (1849) "A sketch of a popular and a novel treatment for diarrhoea, dysentery, and English and Asiatic cholera" (MHL ID b21975620)	"**The cholera not being contagious**, there can be no excuse against assiduous, intimate, and close assistance being given, when that assistance might be simple, efficient, and attainable, in every community, in every family (2)."
Neutral	William Scot (1840) "Report on the epidemic cholera as it has appeared in the territories subject to the presidency of Fort St. George" (MHL ID b30459564)	"It may consequently be inferred, that the disease has **either been propagated by infection or contagion; or, that its progress is owing to circumstances beyond our knowledge**, thus ranking cholera amongst many other epidemics, the **cause of whose origin and progress are equally unintelligible and unknown** (xlvii)."
Neutral	John Taylor (1849) "On the mode of origin and propagation of the epidemic cholera, in Huddersfield, and the neighbourhood, in the autumn of 1849" (MHL ID b21481829)	"Thus, a careful analysis of the cases leads me to the conclusion that in some of them the disease **certainly was not propagated by contagion**, and that it **probably was not in most of them**. In others, again, **it may have been**, and in a **few it is not improbable (although it is by no means certain) that it was propagated by contagion** (28)."
Contagionist	Francis Bisset Hawkins (1831). "History of the epidemic spasmodic cholera of Russia" (MHL ID 39002086311710.med.yale.edu)	"The first clause states the necessity of pre-cautions on account of the **strong evidence of the contagious character of the disease** (13)."
Contagionist	John Murray (1884) "On the treatment of cholera epidemics in India" (MHL IDb22272999)	"The **question of the communicability of cholera through human intercourse** was answered by the Official Reports of the Hurdwar epidemic of 1867, when an assemblage of 2,800,000 pilgrims was attacked in April, and **in returning to their homes progressively spread the disease in all directions** for 500 miles over the N.W. provinces and the Punjaub, and extended it for 500 miles further to the west in Scinde (5)."

Table 1: Examples of "contagionist," "anti-contagionist," and "neutral" scientific views with regard to cholera's transmission mechanism.

At first contact with the disease, roughly half of the scientific community agreed that, on the basis of known evidence, cholera exhibited the characteristics of a contagious disease.[14] With each successive wave of disease, fresh experiences caused a decrease in their adherence to "anti-contagionism" (represented by the uppermost segment of the bar plot in Figure 2), while an increasing proportion do not mention contagion at all (consistent with an expectation that as a consensus forms, some authors will refrain from stating the obvious; very few documents dating from the first wave do not mention the contagion debate).

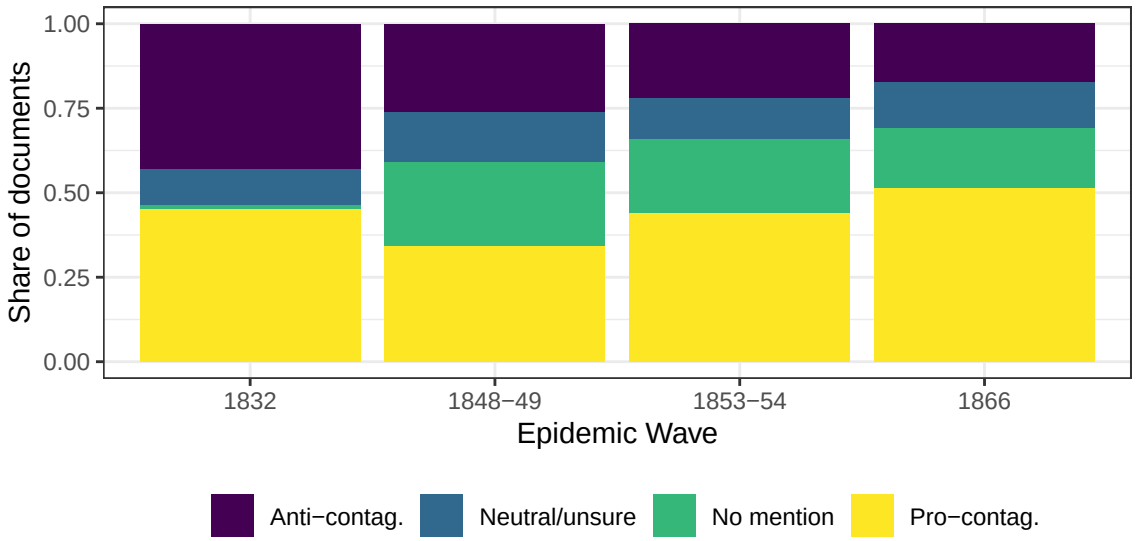

Figure 2: Share of nineteenth-c. British scientific articles pro-contagionist, anti-contagionist, or neutral view, by wave. While the share of researchers expressing pro-contagionist views remains roughly steady over time, the share expressing anti-contagionist views shrinks.

4.2.3 Adjusting for Confounders

In order to better understand the relationship between exposure to the trade economy and scientific views on quarantine, and to adjust for potential confounders in regression specifications, I gather various additional data on the documents and authors in the sample beyond the basic metadata provided by the MHL (which includes date and place of publication). Pelling (1978) suggests that experts may have written differently about cholera when communicating with

[14]This roughly mirrors the breakdown of an 1832 poll of the Westminster Medical Society, in which 24 out of 46 members voted to support an anti-contagionist statement and 22 declined. See *The Lancet* Volume 2, Issue 453, published May 5th, 1832.

other experts versus members of the public in order to maintain clarity of messaging: "It was, perhaps, not in [their] power to admit that cholera was contagious under some circumstances, that is, contingently contagious. These were not the terms in which the public could be instructed" (57). Accordingly, I create a document-level dichotomous variable that captures whether a publication was addressed to a technical scientific audience or the general public. Documents intended for public consumption typically mention the intended audience in the title (for instance, Gilbert Blane's 1831 "Warning to the British public against the alarming approach of the Indian cholera") and use simpler language than those intended for a scientific audience.

Author-level covariates, like author-level trade link data, come from two kinds of sources: the documents themselves, in which authors often provide information about their personal backgrounds, and from secondary sources that capture information about both well-known and lesser-known scientists working in Britain and British colonies during the nineteenth century. These secondary sources include dictionaries of national biography, membership rolls of professional associations, and obituaries published in medical journals. From these sources, I create measures of authors' seniority in the medical community and the density of their connections with the rest of the medical community, which could affect their awareness of others' research.

Seniority is captured by two variables: age at the time a work was published, obtained from MHL metadata and/or biographical and obituary information, and whether an author had been elected a Fellow of the Royal College of Physicians of London (denoted by the post-nominal "FRCP").[15] This accolade, originally available only to medical graduates of English universities who had been voted to the position, was eventually opened to graduates of other medical schools in the British Isles and Europe. A complete list of FRCPs, called Munk's Roll after its original compiler William Munk, is maintained by the College; I cross-reference

[15]Some books are later editions published after their authors' deaths; I retain these in the sample since republication is a sign of continued relevance.

all authors against this list.

I measure density of social connections with other scientists by counting the number of memberships and associations with other British medical, scientific, and learned societies: the Royal College of Physicians of London (counting memberships, which were less prestigious and easier to obtain than fellowships); the Royal College of Surgeons of England; equivalent societies in Edinburgh, Glasgow, and Ireland, and the London-based Epidemiological Society. Appendix Table A1 (p. A10) shows summary statistics and covariate balance across documents authored by trade-linked vs. non-trade-linked experts.

5 Analysis

5.1 Trade-Linked Experts Are More Likely to Be Anti-Contagionist

The share of articles espousing an anti-contagionist view by trade-linked scientists is 0.39, while of the articles written by scientists without political links to trade, that share is 0.22. However, a more rigorous examination requires controlling for potential confounding variables. To test the relationship between political interest group and scientific pronouncement, I run the following linear probability model:

$$\mathbb{1}_{anti_{ij}} = \alpha + \beta \mathbb{1}_{trade_j} + \mathbf{X}\gamma + \epsilon_{ij} \tag{1}$$

where $\mathbb{1}_{anti_{ij}}$ is a dichotomous variable capturing whether document i written by author j expresses an anti-contagionist position; α is an intercept term; $\mathbb{1}_{trade_j}$ is a dichotomous variable capturing whether an author j is associated with British overseas trade interests; and $\mathbf{X}$ is a matrix of document- and author-level control variables defined in Section 4.2, including both linear and quadratic time trends to account for potential non-linear time trends in view.[16]

[16] I use anti-contagionist documents as the outcome category of interest, pooling contagionist, unsure

If an author of a document is politically associated with trade, that document is substantially more likely to espouse an anti-contagionist hypothesis of cholera transmission after adjusting for other variables. Columns 1-4 of Table 2 compare trade-linked experts to non-trade-linked experts; colonial experts who reside in British overseas trade hubs but have no link to a trading company or a colonial administration are included in the comparison group. Columns 5-6 pool together trade-linked experts and other non-trade-linked colonial experts; consistent with the expectation that the former have more reason to be policy-motivated than the latter; the point effect is somewhat attenuated compared to that of Columns 1-4. (Accordingly, in further specifications below, I compare trade-linked experts to all other experts, including the Colonial experts with the control group.) Introducing age as a covariate increases the point effect on trade (Column 3), although data on age is unavailable for a number of observations in the sample.

and "does not mention" into the reference category, due to the ambiguous interpretation I discuss above; a document may not mention how cholera is transmitted because it is unimportant to the author's focus or because the author believes the mechanism to be obvious. Each observation is a document-author pair; documents with multiple authors are assigned a weight $\frac{1}{N}$ where N represents the number of authors who share credit, and standard errors are clustered at the author level. Appendix I (p. A11) shows a variation of the specification in which pro-contagion views are compared to a reference category that pools anti-contagionist, neutral/unsure, and does-not-mention views, and to one in which the sample is restricted to omit neutral/unsure and does-not-mention, as well as other checks on the robustness of the results to alternate specifications, such as removing author weights to assign the full weight (or responsibility) of each view to each of its multiple authors.

Table 2: Regression results for a linear probability model demonstrating the overall differences in scientific opinion between trade-linked and non-trade-linked experts.

	Dependent variable:					
	Anti-contagionist stance					
	(1)	(2)	(3)	(4)	(5)	(6)
Trade link	0.168^{***}	0.213^{***}	0.184^{***}	0.197^{***}		
	(0.058)	(0.056)	(0.058)	(0.062)		
Trade link or other Colonial					0.136^{**}	0.152^{***}
					(0.056)	(0.058)
Date		−0.288	−0.262	−0.321	−0.247	−0.300
		(0.190)	(0.212)	(0.210)	(0.216)	(0.215)
Date^2		0.0001	0.0001	0.0001	0.0001	0.0001
		(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
FRCP			−0.112	-0.133^{*}	-0.118^{*}	-0.140^{**}
			(0.071)	(0.068)	(0.072)	(0.070)
Memberships			0.023	0.027	0.025	0.029
			(0.030)	(0.029)	(0.030)	(0.029)
Public audience			0.063	0.021	0.053	0.010
			(0.101)	(0.101)	(0.105)	(0.106)
Age				0.004^{*}		0.004^{*}
				(0.002)		(0.002)
Observations	448	448	426	402	426	402
Adjusted R^2	0.024	0.083	0.056	0.081	0.045	0.070
Note:						*p<0.1; **p<0.05; ***p<0.01

5.2 Difference in Views Is Driven By Early Part of Sample

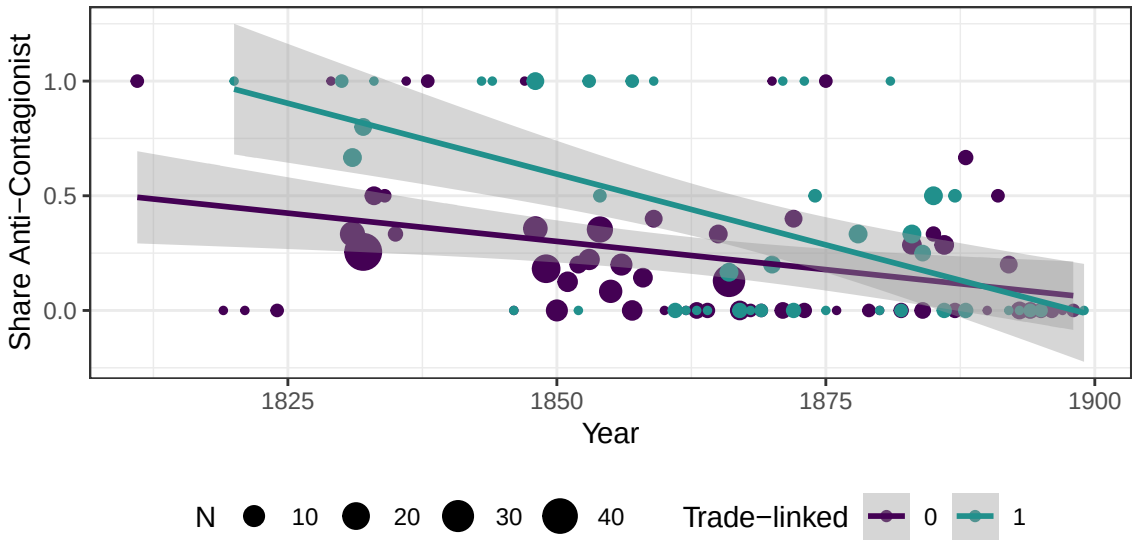

Figure 3: A graph showing the convergence of trade-linked vs. non-trade-linked scientists' views about how cholera spreads. The unit is the document-author pair, where the size of points reflects the number of document-author pairings by publication year.

The main results presented in Table 2 demonstrate that trade-linked experts are more likely than their peers to draw anti-contagionist conclusions about cholera. However, the hypothesis put forth in Section 3.3 predicts that trade-linked experts will be more motivated towards anti-contagionism in the early part of the nineteenth century, before major scientific innovations significantly narrowed the scope of plausible belief about how cholera spread. Figure 3 shows evidence in favor of this prediction. More formally, I run the following specification:

$$\mathbb{1}_{anti_{ij}} = \alpha + \beta_0 \mathbb{1}_{trade_j} + \beta_1 \mathbb{1}_{date_i > 1854} + \beta_2 \mathbb{1}_{trade_j} \times \mathbb{1}_{date_i > 1854} + \mathbf{X}\gamma + \epsilon_{ij} \tag{2}$$

where $\mathbb{1}_{trade_j}$ is a dichotomous variable capturing a link to trade; $\mathbb{1}_{date_i > 1854}$ is a dummy variable for a publication date post-1854; and $\mathbb{1}_{trade_j} \times \mathbb{1}_{date_i > 1854}$ is an interaction of the two. I choose 1854 as the cut-off date between the "loose-knowledge" early period and the "tight-knowledge" late period because it is both the year in which Snow used experimental methodologies to provide empirical evidence of his waterborne theory and when Pacini

published first evidence of the *Vibrio cholerae* bacterium.[17] As an alternate specification, I re-run Equation 1 on "late" versus "early" subsamples. In the early sample, the share of trade-linked experts is 0.15; in the late sample, the share is 0.29.[18] Consistent with the hypothesis, Table 3 shows that the preference of trade-linked experts for anti-contagionist explanations is driven by the early period, in which results are positive, large, and statistically significant.[19]

The convergence between contagionists and non-contagionists by the late nineteenth century is qualitatively similar to results of a formal poll of British scientists working in India conducted in or around 1870. Of 481 scientists who responded to the poll, 456 believed that cholera was communicable in some way (either by personal contact or through some other medium, including via water or sewerage).[20]

[17]Appendix Figure A7 on p. A13 shows the increase in vocabulary associated with modern bacteriology at this juncture, reflecting the transformation in scientific understanding of how infectious diseases are spread. Although Mokyr (2011) dates the "tightening" of epistemic knowledge around germ theory to the 1860s, Snow's findings a decade earlier were well-known in England, and awareness of the new science of germ theory was clearly already on the rise.

[18]Although it is uncertain what share of medical researchers practicing in Britain and British colonies in the early vs. latter part of the century were affiliated with British overseas governments or were otherwise involved directly with Britain's global trade interests, the higher share of experts in the latter half of the sample is reminiscent of the selection effect that drives the intuition behind the model.

[19]In these specifications and in others, adjusted R^2 measures are relatively small, a characteristic typical of a regression with a dichotomous outcome.

[20]The poll is cited in Edward Balfour's 1870 Statistics of Cholera, MHL ID b20398451.

Table 3: Results for specifications showing the interaction effect of a trade link with a variable denoting post-1854, as well as regressions run on pre- vs. post-1854 subsamples of the data.

	Dependent variable:					
	Anti-contagionist stance					
	Interaction	Interaction	Early Only	Early Only	Late Only	Late Only
	(1)	(2)	(3)	(4)	(5)	(6)
Trade link	0.081	0.100	0.440***	0.458***	0.082	0.102
	(0.065)	(0.067)	(0.105)	(0.109)	(0.066)	(0.069)
Date<1854	−0.001	0.016				
	(0.080)	(0.075)				
Trade link × date <1854	0.377***	0.383***				
	(0.130)	(0.135)				
Date	−0.201	−0.217	0.990	1.519	0.006	−0.043
	(0.224)	(0.223)	(2.893)	(3.096)	(0.723)	(0.735)
$Date^2$	0.0001	0.0001	−0.0003	−0.0004	−0.00000	0.00001
	(0.0001)	(0.0001)	(0.001)	(0.001)	(0.0002)	(0.0002)
FRCP	−0.101	−0.121*	−0.136	−0.160	−0.106	−0.120
	(0.071)	(0.069)	(0.131)	(0.128)	(0.078)	(0.078)
Memberships	0.018	0.022	−0.051	−0.037	0.040	0.038
	(0.029)	(0.029)	(0.050)	(0.054)	(0.030)	(0.029)
Public audience	0.050	0.007	0.093	0.084	−0.035	−0.100
	(0.099)	(0.098)	(0.138)	(0.143)	(0.111)	(0.082)
Age		0.004*		0.002		0.005
		(0.002)		(0.003)		(0.003)
Observations	426	402	165	152	261	250
Adjusted R^2	0.077	0.102	0.115	0.126	−0.001	0.031

Note: *p<0.1; **p<0.05; ***p<0.01

5.3 Considering Other Mechanisms

Because these data are observational, it is helpful to consider other factors that might differentiate trade-linked and non-trade-linked scientists and drive them to different conclusions. Scientists with trade links were about the same age, on average, as those without trade links when they published (47.36 vs. 47.76 years at age of publication, respectively), although they tended to have fewer professional memberships than the latter (0.702 vs. 0.976) and to be less likely (4.3% vs. 9.3%) to hold an elite status as a Fellow of the Royal Society of Medicine (Appendix G). Does this suggest that experts with trade linkages were of poorer professional quality in a way that led to systematic error in how they understood cholera? I argue that there is no particular reason to think so. A doctor or scientist who chose a career that took him out of Britain necessarily gave up some opportunities, i.e., to join professional societies based in large cities like London and Edinburgh.

Examining their educational backgrounds, before they selected into a career track overseas or at home, reveals no reason to think that trade-linked experts were of worse quality. Because scientists received their medical training before they made the decision to undertake a career in a trade organization or colonial administration, looking for differences in educational attainment could reveal information about latent quality of experts in the sample (Appendix K). In the nineteenth century, Scottish universities were widely acknowledged as the forefront of empirical medical training and research, playing a significant role in the Scottish Enlightenment; by the mid-18th century, the University of Edinburgh was "the primary academic site in Europe for medical education, which in turn transformed Edinburgh into one of the leading centres for the cultivation of natural knowledge in the Atlantic world" (Wood 2003, 95–96). Trade-linked experts with known educational backgrounds were slightly more likely to be graduates of Scottish universities and thus to have received a top-tier empirically-oriented medical training. On the other hand, trade-linked experts are slightly *less* likely to appear in dictionaries of national biography or to have obituaries appear in major medical journals

(the source of educational data). Neither difference, however, is statistically significant, and on the whole, there is no reason to think that there was any particular quality difference between the trade-linked and non-trade-linked scientists at the start of their careers.

Another possible explanation for differing opinions is that cholera simply behaved differently in colonies, especially India, than in the British Isles, due to physical geography, infrastructure, or characteristics of the population. However, the basic pathways of transmission – waterborne, though poor-quality drinking water, affecting dense population areas and individuals with poor living conditions and poor nutrition – did not differ from place to place. From the scant mortality statistics available, the mortality rate of cholera in India at the start of the pandemic were comparable to those in the hardest-hit British cities: 70 per 10,000 inhabitants in Bombay and Nellore and 110-120 per 10,000 inhabitants Madras, compared to about 30-150 deaths per 10,000 in Glasgow, Liverpool, and London (Arnold 1986, 121–22).[21] From historical accounts, it was qualitatively similar, too: "the seemingly arbitrary way in which the disease chose its victims, the rapidity of its advance, the high fatality among those attacked and the violent nature of their death all contributed to making cholera in India, as in the west, a singularly frightening disease, and one amenable to few customary controls" (Arnold 1986, 119).

5.4 Discussion

These results show that trade-linked scientific experts were more likely to espouse anti-contagionist scientific views in the early nineteenth-century, but that the gap between them and their peers narrowed post-1854 as scientific advances were made. Scientific advances – microscopes, improvements in experimental theory and methodology, and more practical observational experiences – gave scientists tools they previously lacked to investigate the origins of disease. This chipped away at the anti-contagionist bloc within the scientific community over time (Figure 2), although some holdouts remained until the very end of the

[21] See Appendix Figure A1 for British data.

century. However, by the end of the century, the trade sector did not disproportionately contribute those holdouts. While the model stylizes the view of the scientific community as single probability that may be weaker or stronger, a more realistic view allows for a diversity of opinions and, resultantly, higher or lower barriers of proof for different individuals to be convinced to change their minds. What matters is that those hold-outs are not disproportionately trade-oriented.

As noted in Section 3.3, a potential alternate explanation for why trade-linked scientists abandoned the bias they held in the early period runs through the development of better sanitation that obviated the need for quarantines: a better understanding of how cholera spread may have led to better public health policy that accommodated the free flow of trade, rather than the blunt instrument of a general quarantine. However, as I discuss in Section 2.2, cholera quarantine policy continued to hamper British international interests into the second half of the nineteenth century, as many places around the globe that were crucial to British trade did not have the ability to make the kinds of large-scale infrastructural investments that eliminated the disease threat domestically.

To understand the implications of these findings for our understanding of the development of science in the nineteenth century, it is crucial to recall the context in which scientists were studying cholera. As noted in my discussion of the case study and data, many anti-contagionists focused on the role of poor urban sanitation, crowded living conditions, local geography, weather patterns, and poor diet in spreading the disease. In fact, all of these factors are potential mediators and moderators for the spread of cholera germs. It is harder to keep water supplies clean when sanitation infrastructure is poorly constructed and stressed by huge populations crammed into dense urban centers. Low-elevation areas are affected by the runoff of higher-elevation areas, increasing the chances of encountering dangerous waste. Warm weather helps incubate bacteria, accounting for seasonal swings in cholera case rates. A poor diet makes people more susceptible to many kinds of diseases, and poor-quality food

may also be contaminated food. Investments in sanitation, not quarantines, were ultimately responsible for the substantial mortality reductions of the nineteenth and 20th centuries. While the anti-contagionists were wrong about how cholera was spread in the early part of the century, their empirical observations about the correlates of cholera were correct, and their ideas were ultimately the ones that solved the crisis of urban disease in an increasingly mobile world.

One should note that the findings in this paper focus on the internal dynamics of the scientific community itself, not on policymakers' selection of particular scientific experts as advisors, which is out of scope for this particular research. However, it is a promising line for future inquiry. Evans (2005) examines the case of Hamburg's 1892 cholera epidemic, which killed roughly 10,000 residents. Hamburg's public health policy was coordinated by Max Joseph von Pettenkoffer, a well-regarded scientist who nevertheless adhered to a theory of cholera transmission that focused on local geographic conditions rather than germs and contagion. Hamburg's sanitation system was poor and degraded, putting the city at great risk if germs entered the water supply via carriers from an outbreak further East. As Hamburg was a major trade city dependent on the transportation industry, Pettenkoffer's resultant decision not to restrict transit into the city was supported by local political elites. (As a result of the outbreak, Pettenkoffer was removed from his office and replaced by Robert Koch, who had already become famous for his work on the bacteriology of cholera.) Pettenkoffer himself was likely a true believer in outdated cholera theories, even going so far as to experiment with drinking contaminated water to gather evidence for his views. However, his idiosyncratic views were convenient for a policymakers who needed to bolster support for their preferred policies.

6 Conclusions

I examine the dual role of experts as scientists who search for answers to scientific questions and as private actors with a preference over policy. I show how, and the circumstances under which, policy goals can interfere with the progress of science. I theorize that conflicted experts will be more inclined to suppress information that contradicts their policy goals when there is no clear consensus on scientific fact and when scientific methodologies are weak, producing inconclusive results. As methodologies improve and a consensus forms, even conflicted experts will be more forthcoming, since they have little chance of swaying policy but can reap the rewards of publicizing their discoveries.

I find evidence to support this theory using data from public health in the nineteenth century. At the beginning of the century, weak scientific techniques and methodologies hampered scientists' ability to understand how cholera, a novel disease, spread. If policymakers accepted the premise that it was a contagious disease, the findings could support a quarantine policy, which would have disproportionately high costs for the parts of the British economy (and those of British overseas colonies) linked to trade. I find that experts who were professionally linked to Britain's trade economy tended to oppose "contagionist" theories of cholera transmission - but only in the beginning of the century. By the end, their views had converged to that of the rest of the scientific establishment.

The findings of this paper present a contrast to other studies of conflicts of interest in science, particularly that of Oreskes and Conway (2011), which specifically focuses on the behavior of experts who have no reputational concerns (due to being in the late stage of their careers), leading to a situation in which the most biased actors are the last holdouts to an outdated theory (in the case of Oreskes and Conway 2011, about climate change, pollution, and secondhand smoke). In contrast, consistent with a theory in which experts weigh both career concerns and policy impact, I find that conflicts of interest best predict scientific views when

a research question is first studied scientifically and no clear consensus yet exists.

The bulk of today's scientific and medical capabilities date from the huge gains in scientific progress dating to the late nineteenth and twentieth centuries. Modern scientific communities have the benefit of a far more advanced toolbox than did their predecessors. Nevertheless, as the COVID-19 pandemic has demonstrated, experts are still capable of being taken by surprise – and they are still required to juggle policy priorities with their discovery goals, underscoring the importance of understanding how scientific and policy incentives interact.

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//doi.org/10.1177/0047117805058533.

Appendix for The Incentives of Scientific Experts: Evidence from the History of Public Health

A Cholera Mortality

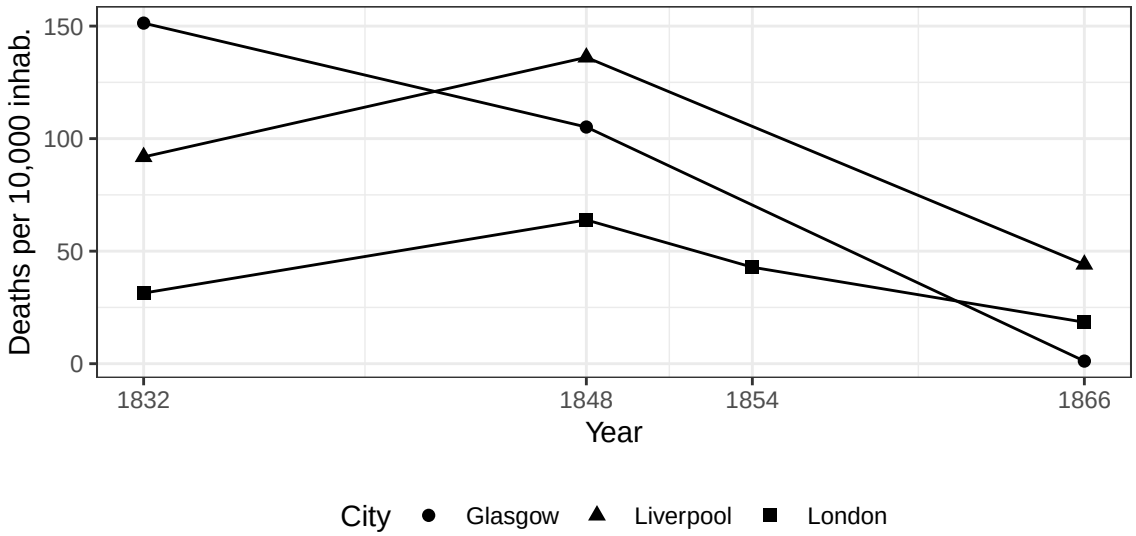


Figure A1: Mortality rates for selected cities during nineteenth-century cholera pandemics. Data for Glasgow comes from Underwood (1948) and University of Portsmouth (2017); for Liverpool from Underwood (1948) and Battersby (2017); and for London from Underwood (1948), Royal Society of Arts (1878), and Haughton (1867). In the context of British mortality, cholera stood out as a threat in urban centers, though its overall impact on national mortality rates was more muted. No epidemic had caused a major spike in deaths for several generations (the most recent was an outbreak of typhus in 1740) (Wrigley and Schofield 1989).

B A Simple Model of Experts' Dual Incentives

Suppose that a scientific community is interested in knowing the the state of the world $S \in \{-1, 1\}$. They have a common prior $\pi = P(S = 1) = 1 - P(S = -1)$.[22] An expert searches for new evidence, which takes the form of a signal $\sigma \in \{-1, 0, 1\}$. With probability p, the expert uncovers a signal $\sigma \in \{-1, 1\}$. The signal σ and the associated probability $P(S|\sigma)$ satisfy the monotone likelihood ration property such that σ gives useful, but not infallible, information about S: for $S \in \{i, j\}$, $1 > P(\sigma = i|S = i) > P(\sigma = j|S = i) > 0$, so it is possible to observe a "mistaken" signal that does not match S. With probability $1 - p$, the expert fails to find any useful evidence, returning a signal $\sigma = 0$.

After searching, the expert transmits a message m to the scientific community. An expert who finds a signal $\sigma \in \{-1, 1\}$ has the opportunity to reveal it or can reveal nothing (i.e., $m = 0$, sending an "empty message"). However, the expert cannot falsify evidence he or she did not find; if the expert found no evidence ($\sigma = 0$), he or she can only send an empty message, and an expert who received a signal $\sigma = -1$ cannot send a message of $\sigma = 1$ (or *vice*

[22]This prior can be thought of as representing either the community's certainty or, if it represents the average view, the degree of consensus.

versa).[23] Experts cannot commit *ex ante* to any strategy to reveal or hide their signals. After observing the contents of the expert's message, the community updates its common prior to a new posterior π^* (the community's process for updating its belief is described below).

There are two types of experts: "unconflicted experts" ("he") and "conflicted experts" ("she"). An unconflicted expert's payoff is solely a function of whether he sends a non-empty message, which represents, for instance, rewards to career advancement or social status from publishing a scientific finding:

$$\hat{U} = \begin{cases} 0 & m = 0 \\ \alpha & m \in \{-1, 1\} \end{cases} \tag{A1}$$

A conflicted expert receives the same payoff as an unconflicted expert for sending a non-empty message. However, she also gets a payoff when the community's belief is to the right of a particular threshold θ (at which point, for instance, a certain policy preference that she favors is adopted):

$$\tilde{U} = \hat{U} + \begin{cases} 0 & \pi^* \leq \theta \\ \beta & \pi^* > \theta \end{cases} \tag{A2}$$

Any expert's type is common knowledge, as are the values of the parameters α and β.[24] However, the community may have imperfect information about whether an expert who sent an empty message actually has no information to transmit.

The solution concept is a perfect Bayesian equilibrium in which an expert and a policymaker each update their beliefs in a manner that is sequentially rational and consistent on the equilibrium path. I focus on identifying the conditions under which a "truth-telling equilibrium" exists, where any expert, including a conflicted one who receives an unfavorable signal $\sigma = -1$, reveals a message that matches his or her signal.

An unconflicted expert who sends an empty message gets a payoff of $\hat{U} = 0$ vs. a payoff of $\hat{U} = \alpha$ when he sends an informative message. Thus, he has a dominant strategy of sending an informative message whenever he can. Additionally, a conflicted expert has a dominant strategy of always conveying a message containing a "convenient" signal ($m = 1$) whenever possible, since doing so yields a minimum payoff of α without any chance of negative consequences for her policy preference.

Under what circumstances will a conflicted expert send the message $m = -1$? There are some situations, based on the values of the primitives α, β, θ, and π, in which even a conflicted expert's strategy is to reveal a message consistent with $\sigma = -1$. If $\pi < \theta$, the

[23] Practically speaking, experts can present information they know to be false, but the professional penalties for doing so tend to be very high.

[24] Making type common knowledge diverges from a classic family of signalling models, such as Spence (1978), in which experts' type is hidden, but in the context of experts with potential conflicts of interest, the conflict is often obvious. For instance, experts employed by a pharmaceutical company seeking FDA approval for a new product are know to be conflicted in that they benefit when the product is approved.

community's prior belief is already to the left of θ. An expert who suppresses a signal $\sigma = -1$ cannot move the belief to the right of the threshold. The expert thus reveals $m = \sigma = -1$ for a payout of $\tilde{U} = \alpha$ (vs. $\tilde{U} = 0$ if she does not reveal). If π is sufficiently far to the right of θ, the expert can safely reveal $m = \sigma = -1$ without causing π^* to fall below the threshold.

In a truth-telling equilibrium, if the community observes a message containing a signal $\sigma = -1$, they update their common prior in a typical Bayesian fashion, since signals cannot be falsified:

$$\pi^*_{m=-1} = P(S = 1|\sigma = -1) = \frac{P(\sigma = -1|S = 1)\pi}{P(\sigma = -1|S = 1)\pi + P(\sigma = -1|S = -1)(1 - \pi)} \tag{A3}$$

if $m = -1$.[25]

Setting Equation A3 equal to θ and solving for π yields

$$\omega = \frac{\theta P(\sigma = -1|S = -1)}{(1 - \theta)P(\sigma = -1|S = 1) + \theta P(\sigma = -1|S = -1)} \tag{A4}$$

As long as $\pi > \omega$, the expert can reveal an "inconvenient" signal without risking policy consequences. She will reveal $m = \sigma = -1$ for a payoff of $\alpha + \beta$ (vs. β if she did not reveal).

On the other hand, if $\theta < \pi < \omega$, a conflicted expert who has a signal $\sigma = -1$ may be forced to choose between her two sources of utility. Revealing her signal yields $\tilde{U} = \alpha$, since $\pi^* < \theta$. Can she improve her outcome? Not if $\alpha > \beta$; in this case, she is better off revealing the signal to get credit for the discovery.

To summarize, at least one of the following conditions must hold for a conflicted expert to always share a signal $\sigma = -1$:

1. $\pi < \theta$ or $\pi > \omega$: either the audience must put sufficient weight on the possibility that $S = -1$ so that the expert's preferred solution is out of reach, or the audience must put sufficiently little weight on the possibility that $S = -1$ that the expert can safely reveal inconvenient information without risking policy consequences.
2. $\alpha > \beta$: the expert gets high enough credit for revealing a signal than it outweighs the benefits of her preferred policy.

[25] Analogously, if $m = 1$,

$$\pi^*_{m=1} = P(S = 1|\sigma = 1) = \frac{P(\sigma = 1|S = 1)\pi}{P(\sigma = 1|S = 1)\pi + P(\sigma = 1|S = -1)(1 - \pi)}$$

.

C Medical Vocabulary in the Nineteenth Century

Medical vocabulary in the nineteenth century was non-standardized; definitions of even fundamental terms varied over time and, sometimes, between authors active at the same time. Of key importance to our setting is what scientists meant when they used the terms "contagious" and "infectious" – two of the most common words used to describe disease transmission. (Other common words, such as "communication" and "transmission," are clearly comprehensible to a layperson and avoid complicated ambiguity.)

Some authors used "contagious" and "infectious" interchangeably to denote a disease that is spread by its carriers. Others drew a distinction between "contagion," which usually (but not always) meant transmission via direct contact, and the broader category of "infection" which usually (but not always) encapsulated indirect contact (such as via objects that had been touched by a patient, or waterborne disease transmission by way of a patient's "effluvia"). For instance, an early government report in India in 1824 could state, "It may consequently be inferred, that the disease has either been propagated by infection or contagion; or, that its progress is owing to circumstances beyond our knowledge, thus ranking cholera amongst many other epidemics, the cause of whose origin and progress are equally unintelligible and unknown,"[26] differentiating between the two, while a different author could write that "I shall endeavour to prove that malignant Cholera is infectious, according to the important principle laid down by Sydenham, 'that all epidemic diseases of a malignant nature are capable of propagating themselves by contagion,' "[27] using "contagious" and "infectious" more or less interchangeably. This highlights the importance of coding outcomes based on the author's view of the key issue at stake: whether cholera was transmitted (directly or indirectly) by the movement of people.

Some other common words used to discuss cholera include "epidemic" – which in its nineteenth-century context usually referred to a disease that affected people in a specific geographic area, without necessarily indicating anything about its transmission mechanism (for instance, a "miasmatic" disease could be "epidemic" in a particular area) – or "pestilential," by analogy to the plague, which was generally considered to be contagious. "Zymotic" is a term arising from early forays into microbiology to refer to potentially disease-bearing microscopic organisms, sometimes in the context of a theory that linked their multiplication to a fermentation process. Below, I give definition for these terms from two medical dictionaries, an 1839 edition of Robert Hooper's *Lexicon Medicum* and an 1887 edition of Richard D. Hobyln's *A Dictionary of Terms Used in Medicine and the Collateral Sciences* to give a greater sense of how medical vocabulary was used and evolved.

Contagion

Hooper 1839: CONTAGION. (Contagio , onis, f. ; from con and tango, to touch.) The term contagion has been used in several acceptations. 1. It has been employed to signify the

[26]Scot, William. (1824) "Report on the epidemic cholera as it has appeared in the territories subject to the presidency of Fort St. George," xlvii

[27]Sadler, Michael Thomas. (1848) "A few thoughts on cholera: in reference to its origin, the nature of the exciting cause, and the principle of treatment," 4.

communication of a disease by personal contact with the sick. 2. It has been employed to signify the communication of a disease, either by personal contact with the sick, or by an effluvium evolved from the body of the sick. 3. It has been employed as generic term, embracing all atmospheric and morbid poisons ; all the effluvia, miasmata, infections, and poisons that cause fevers, of whatever kind ; and those poisons which uniformly excite the diseases which give birth to them, as the venereal disease, the itch, tinea capitis, &c. The second of these senses is the one which it is now generally employed. Attempts have been made to distinguish between contagion and infection, the former being restricted to the communication of disease by direct contact, and the latter to that by effluvia arising from the body of the sick, and communicated through the medium of the atmosphere; but this distinction is now properly discarded by the majority practical writers, and the two words are considered as synonymous

Let it be understood, then, that contagion, [like] infection means the communication of a disease by personal contact with the sick, or by means of an effluvium arising from the body of the sick... (452)

Hobyln 1887: CONTAGION (contagiim, for contagio, a touching, from contingere, to touch, take hold of). This term, and Infection, generally denote the transmission of a poisonous principle. When the transmission is effected by a material substance, and is brought about by actual contact, the term contagion (immediate contagion) is employed; but when transmission is effected through the agency of the winds, and at a distance, the mode of communication is called infection (mediate contagion). In other words, when the poisonous principle is volatile and communicable through the medium of the atmosphere, it is infectious; when this diffusibility is absent, it is contagious. (168)

Epidemic

Hooper 1839: EPIDEMIC. (Epidemicus, from [epi], upon, and [demos], the people.) Applied to a disease which attacks a multitude of persons at the same time and in the same place. Epidemic diseases may arise from contagion or from some atmospheric cause, or from both combined, which last is probably the case for most instances. (591)

Hoblyn 1887: EPIDEMIC ([epidemios], prevalent among a people). An epithet for a popular, prevailing, but not native disease, arising from a general and temporary cause, as excessive heat. (See *Endemic.*) The phrases "Epidemic constitution," "Epidemic influences" belong to the earlier ages of physic, and are suggestive of mystical notions. "It is the disease that constitutes the epidemic, and not the epidemic the disease. The evil always remains the same, the number of those affected being alone increased."' (244)

Infection

Hooper 1839: See Contagion. (763)

Hobyln 1887: INFECTION (inficere, to stain). A general term for the contamination of the atmosphere by malaria, by matter of contagion, by effluvia arising from putrid animal and vegetable substances, &c. See *Contagion.* (365)

Pestilential/Pestis

Hooper 1839: PESTILENTIAL. (*Pestilentialis*; from *pestis*, the plague.) A disease which is epidemic and malignant, partaking of the nature of a plague, is called a pestilential disease. (1011)

From the entry for PESTIS: The plague is by most writers considered as the consequence of a pestilential contagion, which is propagated from one person to another almost solely by *contact* with a diseased person, or with fomites embued with the specific poison. (1012)

Hobyln 1887: PESTIS: This term is seldom used by good authors to signify an infectious disorder. It generally denotes a noxious atmosphere, destruction, curse, &c. (536)

Zymotic

Hooper 1839: No entry.

Hobyln 1887: ZYMOTIC DISEASES ([zymotikos], causing to ferment, from [zyme], leaven). A term suggestive of a fermentation in the blood, occasioned by the introduction into the system of a specific or peculiar virus. The term is used synonymously with "acute specific diseases." (806)

D Cholera in Parliament

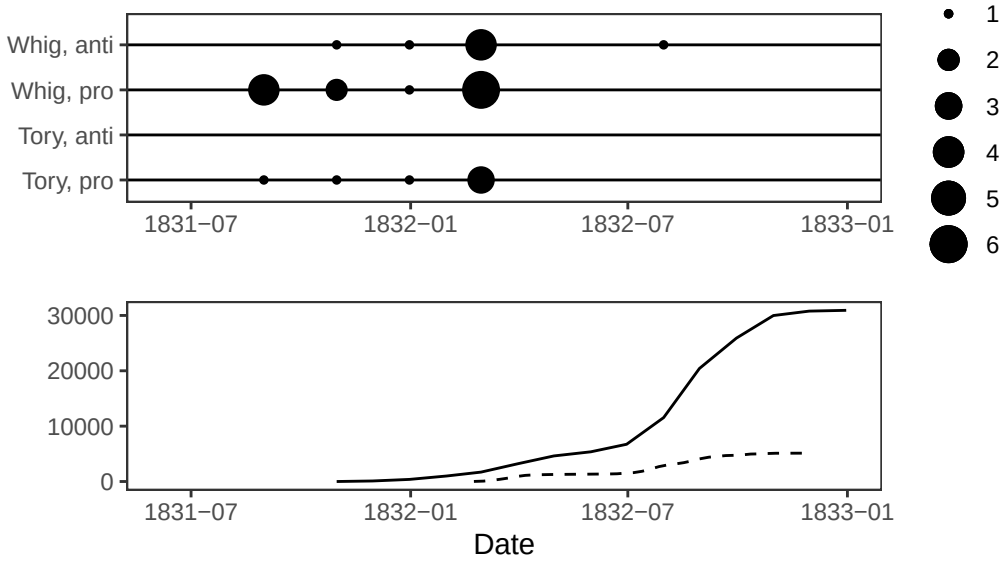
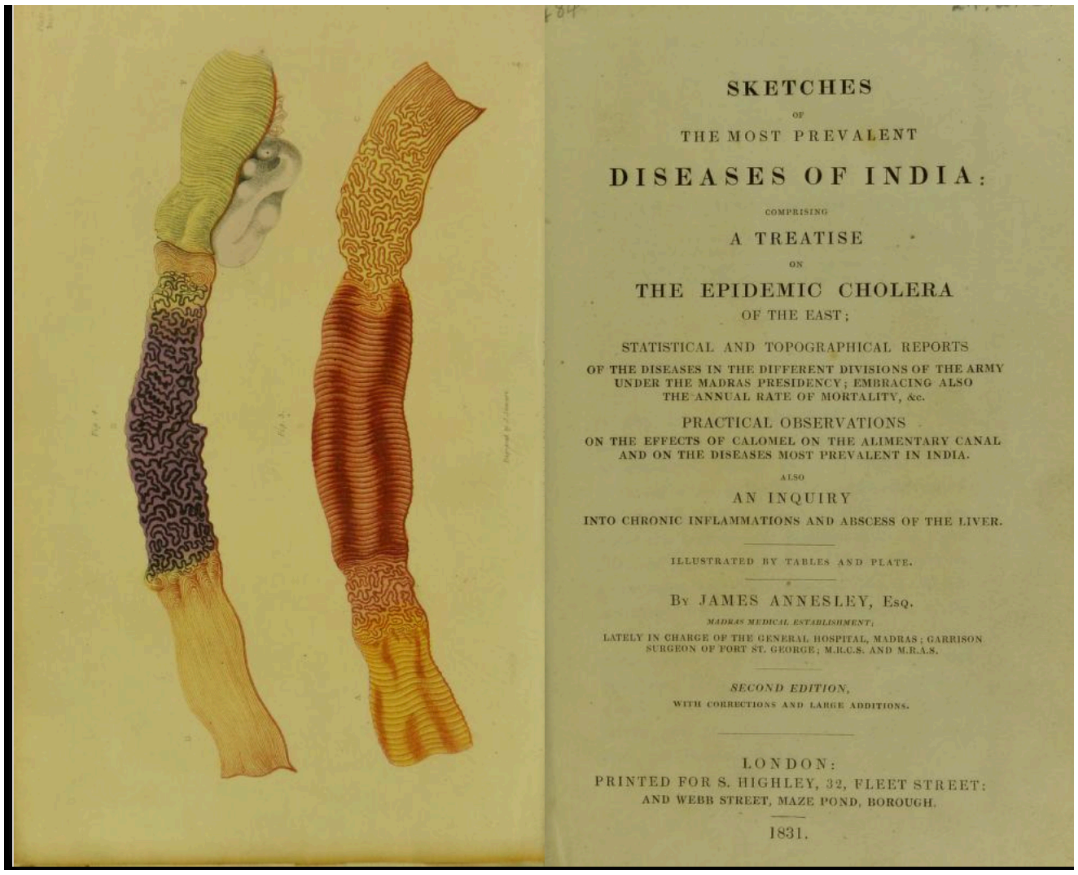


Figure A2: The top panel shows the number of pro- and anti-quarantine speeches made in Parliament by members of the Whig party (and allied Liberal and Radical members) versus the Conservative (Tory) party by date, contemporaneously plotted against all-Britain (solid) and London (dotted) cumulative cholera deaths in the bottom panel. Parliament had no further meetings after August in 1832.

E Raw Data



SKETCHES

OF

THE MOST PREVALENT

DISEASES OF INDIA:

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A TREATISE

ON

THE EPIDEMIC CHOLERA

OF THE EAST;

STATISTICAL AND TOPOGRAPHICAL REPORTS

OF THE DISEASES IN THE DIFFERENT DIVISIONS OF THE ARMY UNDER THE MADRAS PRESIDENCY; EMBRACING ALSO THE ANNUAL RATE OF MORTALITY, &c.

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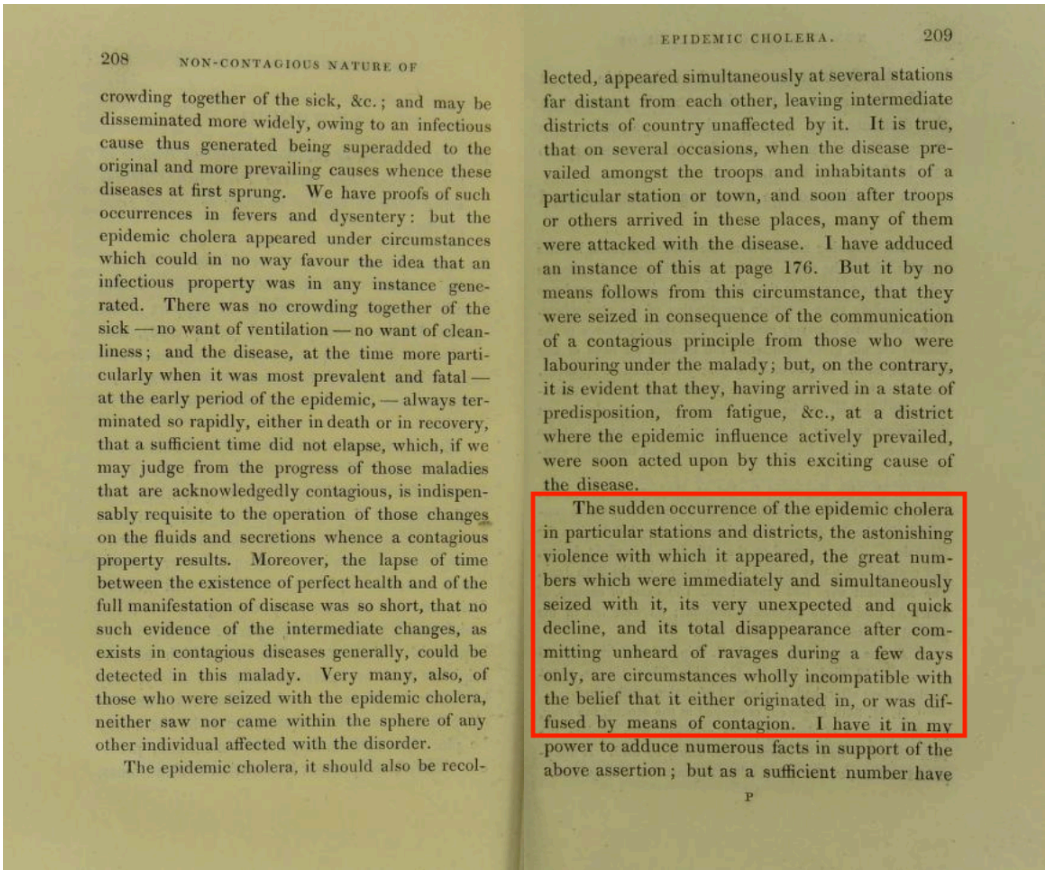
1831.

Figure A3: The frontispiece of MHL document b21306333 by James Annesley, a surgeon working in British India, in 1831.



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crowding together of the sick, &c.; and may be disseminated more widely, owing to an infectious cause thus generated being superadded to the original and more prevailing causes whence these diseases at first sprung. We have proofs of such occurrences in fevers and dysentery: but the epidemic cholera appeared under circumstances which could in no way favour the idea that an infectious property was in any instance generated. There was no crowding together of the sick — no want of ventilation — no want of cleanliness; and the disease, at the time more particularly when it was most prevalent and fatal — at the early period of the epidemic, — always terminated so rapidly, either in death or in recovery, that a sufficient time did not elapse, which, if we may judge from the progress of those maladies that are acknowledgedly contagious, is indispensably requisite to the operation of those changes on the fluids and secretions whence a contagious property results. Moreover, the lapse of time between the existence of perfect health and of the full manifestation of disease was so short, that no such evidence of the intermediate changes, as exists in contagious diseases generally, could be detected in this malady. Very many, also, of those who were seized with the epidemic cholera, neither saw nor came within the sphere of any other individual affected with the disorder.

The epidemic cholera, it should also be recol-

EPIDEMIC CHOLERA. 209

lected, appeared simultaneously at several stations far distant from each other, leaving intermediate districts of country unaffected by it. It is true, that on several occasions, when the disease prevailed amongst the troops and inhabitants of a particular station or town, and soon after troops or others arrived in these places, many of them were attacked with the disease. I have adduced an instance of this at page 176. But it by no means follows from this circumstance, that they were seized in consequence of the communication of a contagious principle from those who were labouring under the malady; but, on the contrary, it is evident that they, having arrived in a state of predisposition, from fatigue, &c., at a district where the epidemic influence actively prevailed, were soon acted upon by this exciting cause of the disease.

The sudden occurrence of the epidemic cholera in particular stations and districts, the astonishing violence with which it appeared, the great numbers which were immediately and simultaneously seized with it, its very unexpected and quick decline, and its total disappearance after committing unheard of ravages during a few days only, are circumstances wholly incompatible with the belief that it either originated in, or was diffused by means of contagion. I have it in my power to adduce numerous facts in support of the above assertion; but as a sufficient number have

P

Figure A4: P. 209 from Annesley's work, in which he writes that 'The sudden occurrence of the epidemic cholera in particular stations and districts, the astonishing violence with which it appeared, the great numbers which were immediately and simultaneously seized with it, its very unexpected and quick decline, and its total disappearance after committing unheard of ravages during a few days only, are circumstances wholly incompatible with the belief that it either originated in, or was diffused by means of contagion.' The document is accordingly coded as anti-contagionist.

F Sample Construction

Figure A5 shows the step-by-step process of cleaning and constructing the final sample of corpus documents. As of the time when data was scraped from the Medical Heritage Library online archive, there were over 127,000 documents with dates of publication in the nineteenth century (although the archive is continuously growing in size). Of these, just over 80,000 are in English. Each document includes one or more contributor-supplied metadata tags. 1,049 documents – about 1.3% of the entire sample – included "cholera" as such a metadata tag. I supplement these with an additional 113 documents that, while not containing a relevant metadata tag, frequently mentioned the word "cholera" in the actual text, which I define as greater than or equal to a frequency of 3.789 mentions per thousand words (the 10th decile frequency of the metadata-tagged part of the corpus). Together, these generated 1,162 documents. Eliminating English-language documents from outside of Britain

(mostly from the United States) and documents authored by non-experts or those not attributable to individual authors yielded a final sample of 418 documents.

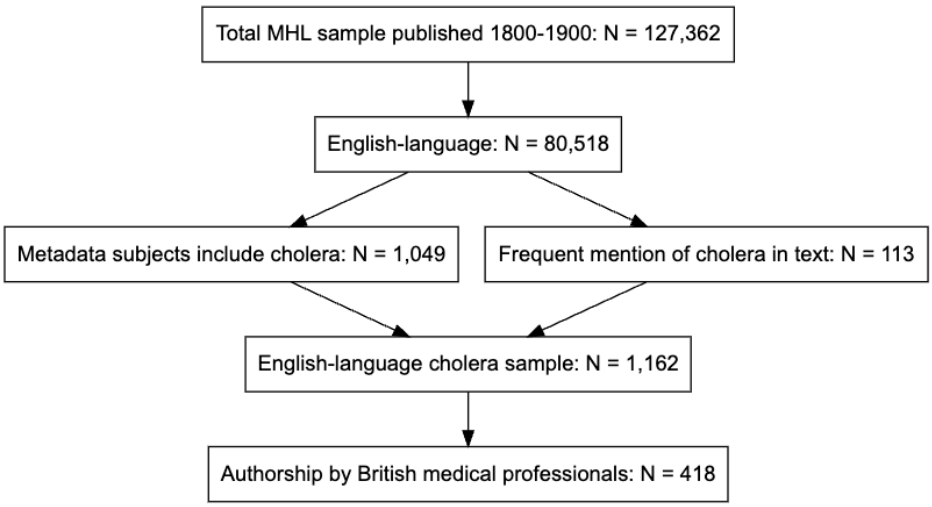

Figure A5: Flowchart showing construction of final corpus from Medical Heritage Library archival materials.

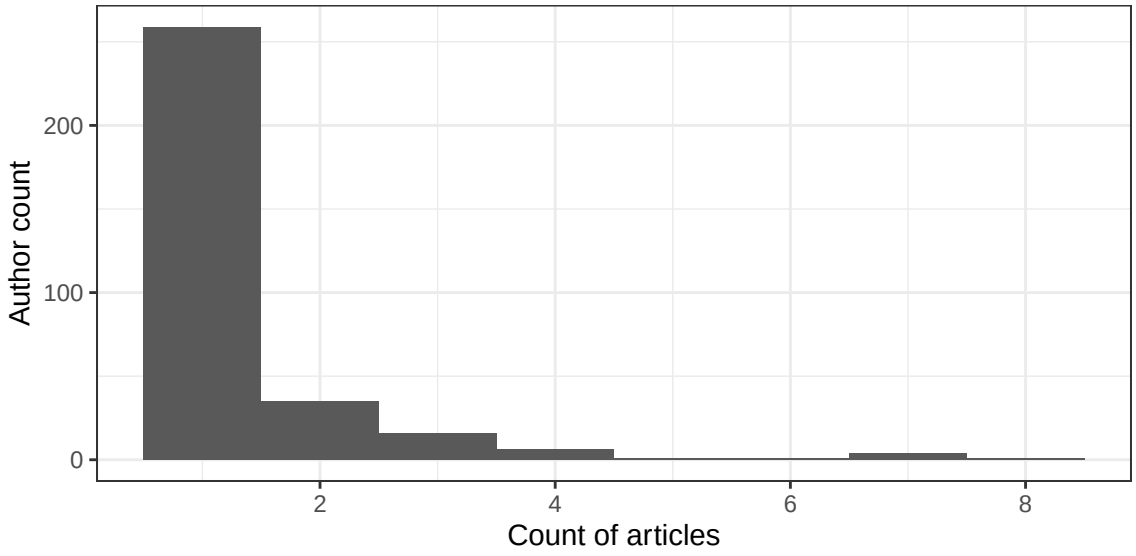

Figure A6: Count of authors by number of articles contributed to final sample.

G Summary Statistics

Tables A1, A2, and A3 give summary statistics for trade-linked vs. non-trade-linked document-author observation pairs used in the main specifications. Consistent with the specifications used to generate the main results, the differences-in-means are weighted by the inverse of the number of authors to which a document is attributed.

Table A1: Summary statistics for trade-linked and non-trade-linked experts, full sample.

	Trade=1	(N=103)				Trade=0	(N=345)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0.043	0	0.203	0	1	0.093	0	0.291	0	1	0.109
Prof. orgs.	0.702	0	0.971	0	4	0.976	1	1.158	0	7	0.035
Public audience	0.078	0	0.269	0	1	0.087	0	0.282	0	1	0.833
Age	47.363	47	12.824	24	86	47.76	45.5	14.232	20	100	0.948

Table A2: Summary statistics for trade-linked and non-trade-linked experts, pre-1854 sample.

	Trade=1	(N=26)				Trade=0	(N=153)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0	0	0	0	0	0.069	0	0.254	0	1	0.238
Prof. orgs.	0.7	1	0.733	0	2	0.814	1	0.874	0	4	0.597
Public audience	0.154	0	0.368	0	1	0.124	0	0.331	0	1	0.632
Age	43.222	41	15.299	24	82	44.548	41	13.819	20	100	0.679

Table A3: Summary statistics for trade-linked and non-trade-linked experts, post-1854 (and inclusive of 1854) sample.

	Trade=1	(N=77)				Trade=0	(N=192)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0.054	0	0.228	0	1	0.112	0	0.317	0	1	0.136
Prof. orgs.	0.703	0	1.03	0	4	1.102	1	1.326	0	7	0.018
Public audience	0.052	0	0.223	0	1	0.057	0	0.233	0	1	0.874
Age	48.384	49	12.04	29	86	50.209	49	14.092	25	85	0.484

H Inter-Coder Reliability of Outcome Variable

All documents that mentioned cholera were independently re-coded by a research assistant to ensure that views could be extracted from the documents with reasonable reliability. Table A4 shows the resultant confusion matrix. In cases of disagreement, the main coder (the author) re-examined each discrepant observation without reference to either the original coding or independent coding and issued a new revised classification. Overall, at the end of this process, the two coders agree 87.68% of the time. In cases in which neither coder categorized an entry as "neutral," the two are in agreement 92.93% of the time. Table A5 shows that the main results change little if the second coder's outcomes are used.

Table A4: Confusion matrix showing original coding (left) and second independent coding (top).

	Anti	Neutral	Pro
Anti	97	4	5
Neutral	6	43	4
Pro	15	9	166

I Robustness Checks

Table A5 shows robustness checks for the results shown in Table 2. Column 1 swaps the category of interest in the outcome to contagionist, pooling anti-contagionist, neutral/unsure, and does-not-mention into the reference category, while Column 2 subsets the sample to only documents that are coded as clearly contagionist or anti-contagionist, omitting the neutral/unsure and does-not-mention categories. The smaller and noisier point effect in Column 1 is unsurprising, given that many articles that do not mention cholera do so precisely the pro-contagionist consensus was established. Column 3 presents a logistic regression. Column 4 omits the author-per-document weights. Column 5 uses the second independent coder's outcome.

Table A5: Robustness checks for Table 2.

	Dependent variable:				
	Pro-contagionist stance	Anti-contagionist stance			
	Swap outcome	Subsample	Logistic	Unweighted	Alternate coder
	(1)	(2)	(3)	(4)	(5)
Trade link	−0.109	0.200**	1.023***	0.187***	0.177***
	(0.076)	(0.079)	(0.316)	(0.056)	(0.060)
Date	−0.537**	−0.014	−0.772	−0.216	−0.216
	(0.253)	(0.281)	(1.270)	(0.202)	(0.223)
Date^2	0.0001**	0.00000	0.0002	0.0001	0.0001
	(0.0001)	(0.0001)	(0.0003)	(0.0001)	(0.0001)
FRCP	−0.026	−0.120	−0.729	−0.110	−0.169**
	(0.122)	(0.117)	(0.535)	(0.069)	(0.071)
Memberships	0.012	0.018	0.134	0.023	0.024
	(0.030)	(0.039)	(0.166)	(0.028)	(0.023)
Public audience	0.004	0.030	0.284	0.057	0.066
	(0.094)	(0.121)	(0.483)	(0.099)	(0.097)
Controls	Core	Core	Core	Core	Core
Observations	426	299	426	426	426
Adjusted R^2	0.014	0.047		0.052	0.047
Log Likelihood			−222.932		
Akaike Inf. Crit.			459.864		
Note:					*p<0.1; **p<0.05; ***p<0.01

J Use of Bacteriological Vocabulary

Figure A7 shows the share, by year, of documents using at least one word stem that references bacteriological concepts ("animalcul-", "vibrio-", "bacil-", or "bact-"). The dashed line denotes 1854 (the year Snow published proof of the waterborne nature of cholera and Pacini identified the relevant bacterium under a microscope). The size of each point is scaled to reflect the number of articles published by year. While some early research theorized about the role of invisibly small life forms in the spread of disease (typically termed "animalcules"), these theories could not be evaluated until advances in microscopy allowed for direct observation. The work of Snow and those who followed in his footsteps helped to elucidate the causal (vs. correlative) role of microscopic organisms in the spread of disease.

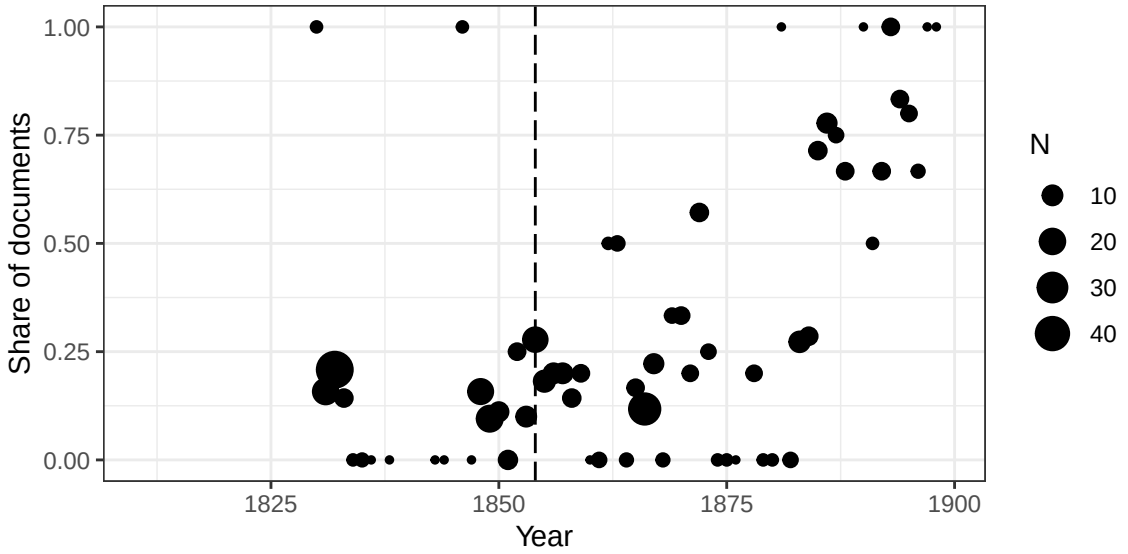

Figure A7

K Author Characteristics

Table A6

	Dependent variable:			
	Trade link			
	(1)	(2)	(3)	(4)
School unknown	0.059	0.055		
	(0.045)	(0.044)		
Scottish university			0.052	0.052
			(0.052)	(0.051)
Date		−0.227		−0.225
		(0.186)		(0.186)
Date^2		0.0001		0.0001
		(0.00005)		(0.0001)
Observations	323	323	323	323
Adjusted R^2	0.002	0.042	0.00000	0.040
Note:				*p<0.1; **p<0.05; ***p<0.01