Expert-Policymaker Interactions: Evidence from 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. In this setting, experts must trade off between their professional reputations for scientific accuracy and their ability to shape policy – goals that sometimes conflict, particularly when experts have personal preferences over policy. To explore this duality, I use evidence from public health research on cholera in the 19th century. Drawing on a large, representative corpus of medical publications, I find that experts with links to Britain's overseas trade sector were less likely than their peers without such political connections to advance theories that cholera was a contagious disease spread by trade and travel. This difference is driven by the early part of the 19th century, when the scientific consensus was around how cholera spread had not yet solidified. I argue that conflicted experts are more likely to act on their bias in low-information environments, when revealing inconvenient information can lead to negative policy consequences. As a consensus forms, the value of hiding unfavorable information lessens, and even experts with a conflict of interest will reveal what they know to gain scientific credit.

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1 Introduction

Policymakers often solicit input about technical topics, such as disease control, environmental policy, or financial regulation, from experts with technical subject matter knowledge. These experts are typically people with scholarly credentials whose reputations depend on their standing in scientific communities. However, this does not necessarily mean they are apolitical actors with no personal preferences over policy. When policymakers directly or indirectly delegate policymaking to experts, how do those experts' political preferences affect the progress of science?

I approach this question using evidence from the history of public health. Cholera originally spread from its origin point on the Indian subcontinent, where it was an endemic disease, to Eurasia for the first time in the 1810s. Throughout the rest of the 19th 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 were 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 novel dataset of 19th-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 trade hubs – were more likely than their peers to attack the theory of contagious cholera. This pattern is driven by the early 19th century, when there was not yet a firm scientific consensus 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, had 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.

I consider the conditions under which a politically motivated expert will suppress information that is inconvenient for convincing others to adopt their policy preference. I theorize that conflicted experts – experts 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 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 value of suppressing information drops, and even experts with a known conflict of interest are more likely to reveal what they know in order to capture scientific credit.

The COVID-19 pandemic demonstrates the importance of examining the motivations of experts, including those with scientific expertise, 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. The striking parallels between the how actors juggled these dual roles in the 19th century and the similar struggles of their counterparts in public health in the 21st century highlights the importance of examining experts as political actors.

2 Historical Context

2.1 A Scientific History of Cholera

Cholera is a bacterial disease caused by the microscopic *Vibrio cholera*. Endemic to the Indian subcontinent, it spread across Eurasia for the first time in the 1820s. For the rest of the 19th century, periodic cholera pandemics swept across Europe, 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 by infected carriers, 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; S. 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, or households and 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 19th 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, attributing cholera to atmospheric phenomena. Others theorized that the disease was caused by deteriorating urban living conditions that produced dangerous "miasma" (or "bad air"). Other, more exotic anti-contagionist theories included volcanic emissions and electrical fields generated by new telegraph technologies.

At first contact with cholera, scientific techniques were still rudimentary, 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." Medical science at the beginning of the 19th century had neither. There was justifiably low confidence in tools and methodologies, and as a result, weak consensus on many questions, including that of how cholera was spreading.

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; S. 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 made 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 (S. Johnson 2006). Several years later, he published strong supportive evidence for the theory from his famous natural experiments leveraging 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 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. 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" (p 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 it 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.[1]

Despite these precautionary policies, the quarantine of the national borders was imperfectly administered, 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 had succumbed. Further epidemics followed in 1848-1849, 1853-1854, and 1866. Figure 1 shows mortality data for a selection of British cities during the years of major cholera epidemics. 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?"[2]

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 welfare. A theory of generally poor living conditions generating disease justified funding for general 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.

[1]Source: Author's tabulation of Parliamentary speeches from Hansard Parliamentary Database.
[2]Asiatic cholera : its history and nature, with directions for its prevention and cure, 1849.

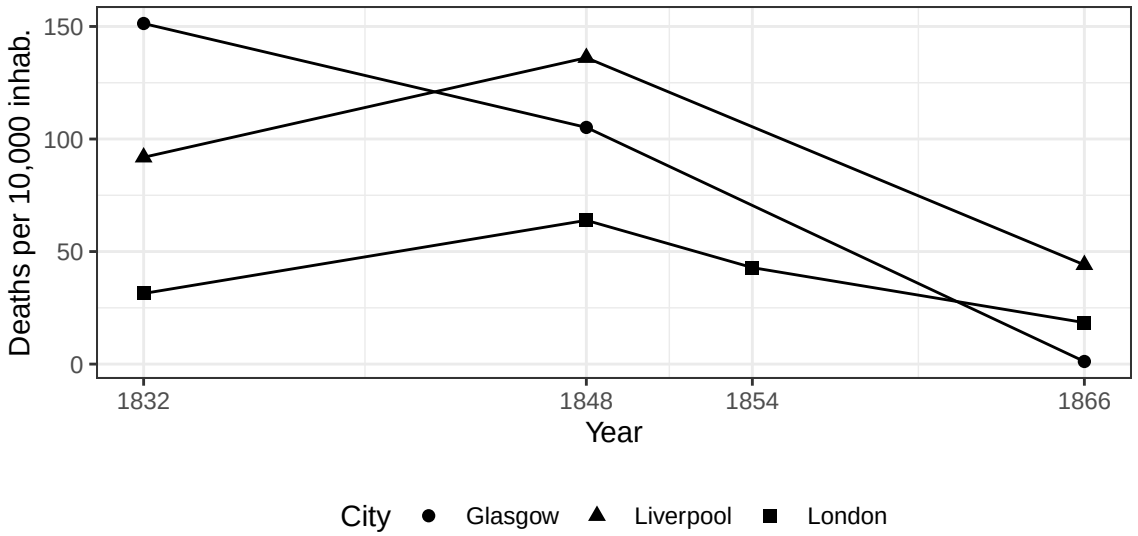

Figure 1: Mortality rates for selected cities during 19th-century cholera pandemics. Data for Glasgow comes from Underwood (1948) and University of Portsmouth (2017); for Liverpool from Underwood (1948) and Battersby (2017); and for London from Underwood (1948), Royal Society of Arts (1878), and Haughton (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).

In fact, it was a desire to improve urban living conditions – specifically, those of Britain's ruling elite – that 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).[3] Similar investments in other cities had a comparative effect on eradicating these public health threats.[4]

Despite these advances in domestic public health policy, Britain still needed to reckon with

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

[4]For quantitative evidence on the role of sanitation infrastructure (including both sewerage and chlorination) and clean water on reducing urban mortality, see 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.

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).[5] 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.[6]

3 Theory

3.1 The Motivations of Experts

Scholarship from the philosophy, and sociology of science tends to assume that scientific experts exert effort because they are motivated by intrinsic curiosity (Kuhn 1970; Popper 1972). A literature on expert predictions from economics assumes a more material motivation: experts' goal is to maximize their reputations (see, for instance, Scharfstein and Stein 1990; Effinger and Polborn 2001; and Ottaviani and Sørensen 2001). In both cases, all else equal, experts are better off "being right" over "being wrong."[7]

How, then, are experts evaluated? Some formal models assume "state verification": an expert's job is to make predictions about the state of the world with respect to some question,

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

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

[7] These models discuss experts in a stylized, generalizable sense, i.e., anyone who is in a position to hold privileged information. Labor economics, for instance, models employees as having private information about their own productivity, and they seek to convince a manager that they are a "high type" in order to gain a reward. However, these frameworks can be easily exported to examine scientific experts in particular. In fact, in many real-life settings, particularly academic ones, scientific experts are "employed" by their peers.

which may be resolved in the future with some probability, upon which experts are rewarded or punished on the basis of how their prediction compares to the revealed state. This stylized scenario does indeed map cleanly to some real-life expert roles. Meteorologists, for instance, are in charge of making concrete predictions about the weather, which will be eventually revealed with certainty. On the other hand, a model may assume that an expert must produce evidence for any claim.[8] Some models (Avery and Chevalier 1999; Effinger and Polborn 2001; Ottaviani and Sørensen 2001; Levy 2004) have the added feature of adjusting payoffs to account for whether an expert's prediction is in vs. out of consensus.

In the setting I examine, the "evidence for credit" is the best theoretical fit. Medical scientists in the 19th century, like those today, were required to provide evidence for their claims – statistical data, case studies, reasoned argument, etc. – when they published in scholarly venues. Some of the evidence they presented may have been weak by modern standards, but it was evidence nonetheless. Methodological innovations and a few key breakthroughs led to an accumulation of evidence that shifted the scientific community's consensus closer and closer to accepting a theory of waterborne germ transmission.

Another branch of literature considers how experts and policymakers interact. In general, these models focus on how policymakers can extract maximal information from experts under the assumption that an expert may have some reason to withhold or misrepresent information (see, for instance, Crawford and Sobel 1982; Gailmard and Patty 2013; Scharfstein and Stein 1990; Brandenburger and Polak 1996; Avery and Chevalier 1999; Ottaviani and Sørensen 2001; and Backus and Little 2020). This departs from the assumption that an expert's chief goal is to maximize the chance of making a correct prediction. Usually, these models assume that experts' information will influence the decisionmaker's choice of action, which in turn has consequences for the expert; they can gain by manipulating the decisionmaker's

[8]Experts may also "overclaim" to enhance their reputations; see Scharfstein and Stein (1990), Brandenburger and Polak (1996), Ottaviani and Sørensen (2006), and Hill and Stein (2021). In other models, experts can transmit false signals, for instance, in the "cheap talk" family of models originating with Crawford and Sobel (1982).

beliefs about the state of the world, or about the outcomes that will result from their choice. Clearly, if an expert always reveals complete and true information, the policymaker does not need to rely on complicated strategies to extract useful knowledge. 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). Flinders (2020) examines how policymakers use experts to deflect blame from the failures of their policy to benefit voters.[9] Andrews and Shapiro (2021) extends this line of thinking, comparing the actions of an expert whose goal is to change a policymaker's action (the goal of most classical models in the literature) to those of an expert whose goal is to change a policymaker's beliefs (which in turn determines action).

I abstract away from policymakers' incentives, taking as given the policy implications of different scientific beliefs in order to put the focus on experts' trade-offs between scientific reputation and policy outcomes. Carpenter (2003) and Fox and Van Weelden (2012) examine how reputational consequences – for instance, outsized downsides to being wrong – can affect an expert's potential to suppress information, even if it is useful in expectation. Conversely, E. Johnson (2012) presents a model in which agents protect their reputations by withholding information to avoid appearing biased. Youde (2005) examines a fracture in the epistemic community of HIV/AIDS researchers between the South African public health community and their counterparts in the Western world. South African public health researchers promoted a narrative that focused on poverty as the ultimate cause of the African HIV/AIDS pandemic, 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

[9]Other work, like Alesina and Tabellini (2007) and Fox and Jordan (2011), focuses on optimal allocation of policymaking tasks to elected officials (who can be motivated by electoral prospects to exert effort) versus expert bureaucrats (who are insulated from effort-based incentives but have higher ability and are motivated by reputational concerns).

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, a scenario that parallels the historical case study on which I focus.

3.2 Model

Suppose that a scientific community is interested in knowing the the state of the world $S \in \{S_0, S_1\}$. They have a common prior $\pi = P(S_1) = 1 - P(S_0)$.[10] An expert, or scientist, searches for new evidence, which takes the form of a signal $\sigma \in \{\sigma_0, \sigma_1, \emptyset\}$. With probability p, the scientist uncovers a signal $\sigma \in \{\sigma_0, \sigma_1\}$. The signal σ gives useful, but not infallible, information about S: for $S \in \{i, j\}$, $P(\sigma_i|S_i) > P(\sigma_j|S_i) > 0$, so it is possible to observe a "mistaken" signal that does not match S. With probability $1 - p$, the scientist fails to find any useful evidence, returning a signal $\sigma = \emptyset$.

After searching, the expert transmits a message m to the scientific community. An expert who finds a signal $\sigma \in \{\sigma_0, \sigma_1\}$ has the opportunity to reveal it or can reveal nothing (i.e., $m = \emptyset$, sending an "empty message"). However, he cannot falsify evidence he did not find; if he found no evidence ($\sigma = \emptyset$), he can only send an empty message, and an expert who received a signal σ_0 cannot send a message of σ_1 (or *vice versa*). 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:

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

$$U_{\text{unconflicted}} = \alpha(\mathbb{1}_{m \trianglelefteq \emptyset}) \tag{1}$$

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):

$$U_{\text{conflicted}} = \alpha(\mathbb{1}_{m \trianglelefteq \emptyset}) + \beta(\mathbb{1}_{\pi^* > \theta}) \tag{2}$$

Any expert's type is common knowledge, as are the values of the parameters α and β.[11] However, the community may have imperfect information about whether an expert who sent an empty message actually has no information to transmit.

If the community observes a message containing a signal σ_0 or σ_1, they update their common prior in a typical Bayesian fashion, since signals cannot be falsified:

$$\pi^*_{\sigma=1} = P(S_1|\sigma_1) = \frac{P(\sigma_1|S_1)\pi}{P(\sigma_1|S_1)\pi + P(\sigma_1|S_0)(1-\pi)} \tag{3}$$

if $\sigma = \sigma_1$ and

$$\pi^*_{\sigma=0} = P(S_1|\sigma_0) = \frac{P(\sigma_0|S_1)\pi}{P(\sigma_0|S_1)\pi + P(\sigma_0|S_0)(1-\pi)} \tag{4}$$

if $\sigma = \sigma_0$.

An unconflicted expert who sends an empty message gets a payoff of $U_{\text{unconflicted}} = 0$ vs. a

[11] 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.

payoff of $U_{\text{unconflicted}} = \alpha$ 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 (σ_1) whenever possible, since doing so yields a net payoff of α without any chance of negative consequences for her policy preference.

Under what circumstances will a conflicted expert send the message $m = \sigma_0$? 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 σ_0. If $\pi < \theta$, the community's prior belief is already to the left of θ. An expert who suppresses a signal σ_0 cannot move the belief to the right of the threshold. The expert thus reveals σ_0 for a payout of $U_{\text{conflicted}} = \alpha$ (vs. $U_{\text{conflicted}} = 0$ if she does not reveal). If π is sufficiently far to the right of θ, the expert can safely reveal σ_0 without causing π^* to fall below the threshold. Setting Equation 4 equal to θ and solving for π yields

$$\omega = \frac{\theta P(\sigma_0|S_0)}{(1-\theta)P(\sigma_0|S_1) + \theta P(\sigma_0|S_0)} \tag{5}$$

As long as $\pi > \omega$, the expert can reveal an "inconvenient'' signal without risking policy consequences. She will reveal $m = \sigma_0$ 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 σ_0 may be forced to choose between her two sources of utility. Revealing her signal yields $U_{\text{conflicted}} = \alpha$, since $\pi^* < \theta$. Can she do better? 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 σ_0:

1. $\pi < \theta$ or $\pi > \omega$: either the audience must put sufficient weight on the possibility that $S = S_0$ so that the expert's preferred solution is out of reach, or the audience must

put sufficiently little weight on the possibility that $S = S_0$ 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.

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. This strategy, however, 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 to alleviating 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 on the domestic level (determining what role quarantines at British borders or within the country during times of disease) and on the international level (determining international agreements on policies applied to 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 – that is, if π (representing consensus belief in the probability that the anti-contagionists were correct) dropped below some threshold θ at which their effectiveness could be justified. As evidence for the contagious transmission mechanism accumulated, π eventually dropped below the θ threshold.

Simultaneously, however, cities in Europe began to invest in better 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 that improved quality of urban life (Lizzeri and Persico 2004; Aidt, Daunton, and Dutta 2010). Doing so may have in part decreased the value of β relative to α: 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.[12]

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 434 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

[12] An additional interesting comparative static leverages the effect of changing $P(\sigma_i|S_i)$ and $P(\sigma_i|S_j)$ on ω. Increasing the "signal reliability" of σ increases the impact of announcing a discovery, potentially widening the interval between θ and ω and increasing the incentive to suppress a strong and inconvenient signal. As a matter of science policy, this could be offset by rewarding an expert more for higher-impact discoveries.

"cholera.")

Figure 2 shows the number of documents for each year within the sample. These documents include complete books, monographs, pamphlets, transcribed lectures, and individual journal articles. 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 19th century.

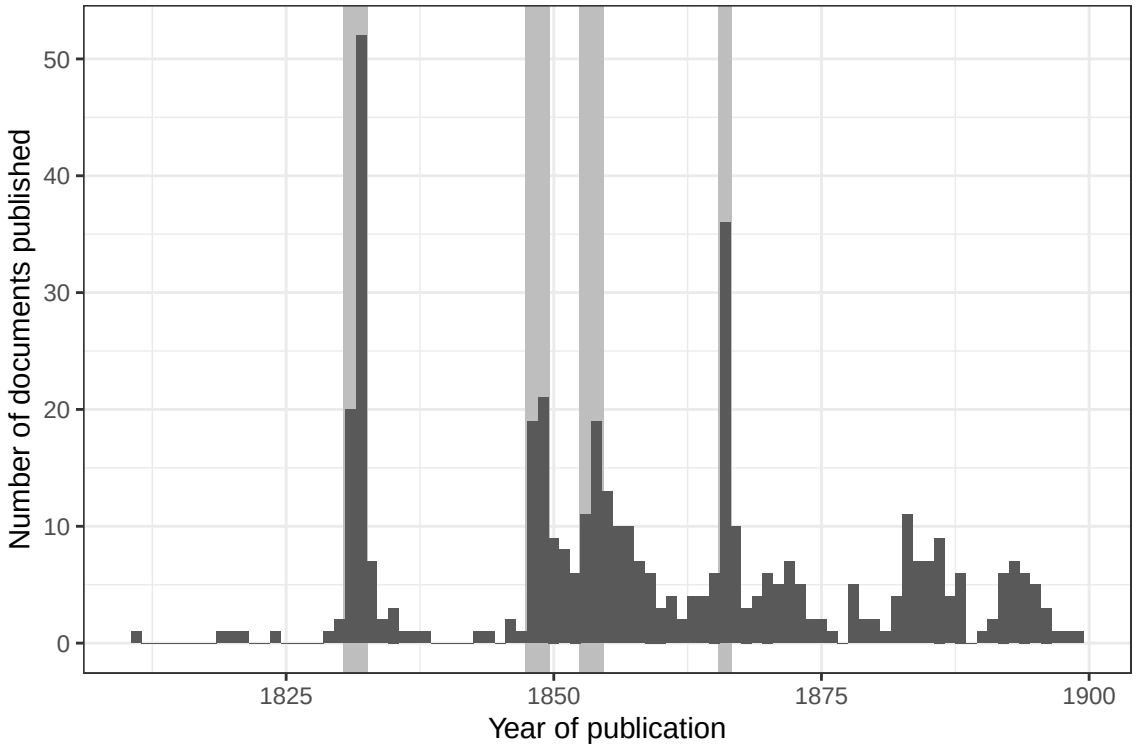

Figure 2: Number of documents in MHL sample by year of publication. Grey bars denote epidemic years.

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).

4.2 Research Design

Section 3.3 suggests a testable hypothesis: conflicted experts will act on their source of conflict, but only in what Mokyr (2011) refers to as an "epistemically loose" environment. As documented in Section 2.2, *policymakers* clearly understood the costs of following contagionist vs. anti-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 advice. 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." 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" (p. 67) and "a climate of anticontagionism was a *fait accompli*" (p. 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 to 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; or who was located in a major center of British colonial trade.[13]

I gather additional data to adjust for various potential confounders, including linear and quadratic terms for date and control variables that capture whether a scientist was a member of the upper echelons of the medical elite (holding a title of "Fellow of the Royal Society of Physicians of England," entitling them to use the post-nominal title "FRCP") as well as for total number of other professional memberships, which proxies for the breadth of experts' social networks and exposure to cutting-edge ideas.[14] For a subsample of MHL authors, date of birth is available, which I supplement when possible with information from authors' obituaries in major British medical journals to construct authors' age at publication.[15] Additionally, Pelling (1978) suggests that government experts shied away from discussing cholera in terms of contagion because doing so might confuse the public: "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." Accordingly, I include a document-level dummy variable that captures whether a publication was addressed to a technical scientific audience or the general public. Appendix Table A1 shows summary statistics and covariate balance across documents authored by trade-linked vs. non-trade-linked experts.

To construct an outcome variable, I extract sections of text from each document that are within a 500-word bandwidth around any mention of a relevant keyword ("contagion," "communicable," "transmissible," or the associated word stems). I then evaluate whether each section reflects a contagionist or anti-contagionist viewpoint. Nineteenth-century medical

[13]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 Crown in the first half of the 19th 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.

[14]These include non-fellow membership in the Royal College of England as well as membership in the Royal College of Surgeons of London and equivalent societies in Edinburgh, Glasgow, and Ireland and the London-based Epidemiological Society.

[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.

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. Therefore, I classify documents using not only authors' word usage but also contextual information, an approach that precludes automated classification. Some documents are "neutral"; their author explicitly acknowledges a lack of sufficient evidence to come to a decision, while others do not mention the contagion debate at all. Table 1 shows examples of each category.

Category	Source	Example
Anti-Contagionist	Reinhardt 1853	"The statistics of its late visit to England are minute and circumstantial, and **prove beyond all doubt**, that the disease spreads by virtue of true epidemic, or **atmospheric quality**, and that **contagion has little or nothing to do with it**."
Anti-Contagionist	Stevens 1832	"**I have not witnessed any facts** which lead me to think the malignant Cholera as it now prevails in the city to be personally contagious. I put forth these opinions as seeming to me probable and subject to change upon further observation.
Neutral	Mussey 1840	"To confirm or disprove this hypothesis **will require far more investigation** than the subject has, as yet, received."
Contagionist	Dixon 1871	"The disease **probably depends on a specific animal poison**, fungus, or contagion germ, which neither the science of chemistry or the use of the microscope has succeeded in detecting."
Contagionist	Lassen 1866	"From the first hour I directed my attention to **contagious diseases**: the oriental plague, the yellow fever, the **Asiatic Cholera**..."

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

Figure 3 shows the change in scientific opinion over time. For tractability, documents are categorized according to the epidemic with which they are associated. 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. With each successive wave of disease, fresh experiences caused a decrease their adherence to "anti-contagionism" (represented by the uppermost segment of the bar plot in Figure 3). The share of documents that definitely espouse a contagionist viewpoint fluctuates around 50% in each period but rises consistently relative to the anti-contagionist views; the difference is due to an increase in the number of documents that do not mention contagion at all. (The interpretation of these

documents is ambiguous; some were more focused on other aspects of disease management, such as documenting individual clinical cases, while others may have considered the question of transmission to be settled science and did not bother to engage.)

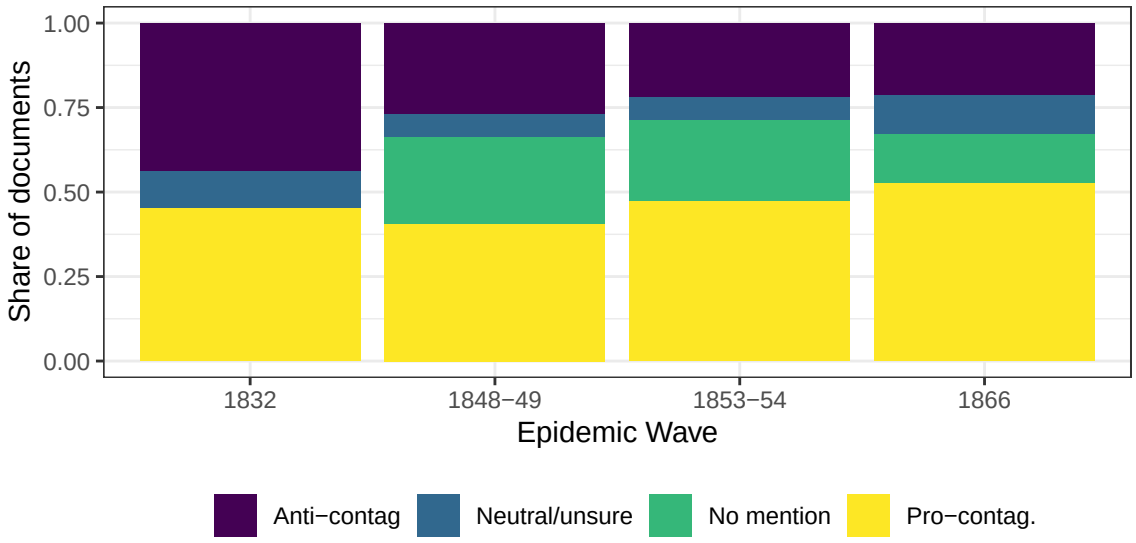

Figure 3: Share of 19th-c. British scientific articles pro-contagionist, anti-contagionist, or neutral view, by wave. While the share of researchers expressing pro-contagionist views remains roughly steady over time, the share expressing anti-contagionist views shrinks.

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 0.38, while of the articles written by scientists without political links to trade, that share is 0.24. 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:

$$\mathbb{1}_{anti_i} = \alpha + \beta \mathbb{1}_{trade_i} + \mathbf{X}\gamma + \epsilon_{\mathbf{i}} \tag{6}$$

where $\mathbb{1}_{anti_i}$ is a dichotomous variable capturing whether a document expresses an anti-contagionist position; α is an intercept term; $\mathbb{1}_{trade_i}$ is a dichotomous variable capturing whether an author is associated with British overseas trade interests; and $\mathbf{X}$ is a matrix of control variables defined in Section 4.2.[16] 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.

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)
Trade link	0.171***	0.137**	0.102
	(0.061)	(0.059)	(0.068)
Date	−0.294	−0.315	−0.369
	(0.179)	(0.229)	(0.252)
Date^2	0.0001	0.0001	0.0001
	(0.00005)	(0.0001)	(0.0001)
FRCP			0.005**
			(0.002)
Memberships		−0.068	−0.069
		(0.089)	(0.095)
Age		0.010	0.017
		(0.030)	(0.030)
Public audience		0.017	−0.183**
		(0.105)	(0.090)
Constant	277.722*	297.359	347.028
	(166.299)	(212.697)	(235.101)
Controls	Date only	Core	Extended
Observations	465	381	287
Adjusted R^2	0.060	0.045	0.044
Note:			*p<0.1; **p<0.05; ***p<0.01

If an author of a document is politically associated with trade, that document is substantially

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

more likely to espouse an anti-contagionist hypothesis of cholera transmission (Columns 1 and 2 of Table 2). Introducing age as a covariate only slightly decreases the point effect on trade (Column 3), although standard errors are larger, consistent with the smaller sample size (due to data on age being unavailable for a number of observations in the sample).

5.2 Difference Is Driven By Early Part of Sample

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 19th century, before major scientific innovations significantly narrowed the scope of plausible belief about how cholera spread. I therefore run the following specification:

$$\mathbb{1}_{anti_i} = \alpha + \beta_0 \mathbb{1}_{trade_i} + \beta_1 \mathbb{1}_{date_i > 1854} + \beta_2 \mathbb{1}_{trade_i} \times \mathbb{1}_{date_i > 1854} + \mathbf{X}\gamma + \epsilon_{\mathbf{i}} \tag{7}$$

where $\mathbb{1}_{trade_i}$ 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 $\mathbb{1}_{trade_i} \times \mathbb{1}_{date_i > 1854}$ 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 6 on "late" versus "early" subsamples. In the early sample, the share of trade-linked experts is 0.17; in the late sample, the share is 0.32.[18] Consistent with the

[17] Appendix Figure A1 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

hypothesis, Table 3 shows that the preference of trade-linked experts for anti-contagionist explanations is entirely driven by the early period.

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 Subsample
	(1)	(2)	(3)
Trade link	0.050	0.274**	0.042
	(0.065)	(0.122)	(0.070)
Date<1854	0.045		
	(0.086)		
Trade link × date <1854	0.290**		
	(0.137)		
Date	−0.184	7.412	0.467
	(0.251)	(4.965)	(0.761)
Date^2	0.00005	−0.002	−0.0001
	(0.0001)	(0.001)	(0.0002)
Age	−0.046	−0.300***	0.068
	(0.088)	(0.063)	(0.108)
FRCP	0.006	−0.054	0.016
	(0.029)	(0.050)	(0.027)
Memberships	0.023	0.045	−0.023
	(0.098)	(0.133)	(0.129)
Public audience	173.847	−6,816.271	−436.131
	(234.473)	(4,570.120)	(714.334)
Controls	Core	Extended	Core
Observations	381	160	221
Adjusted R^2	0.058	0.096	−0.015
Note:			*p<0.1; **p<0.05; ***p<0.01

5.3 Discussion

These results show that trade-linked scientific experts were more likely to espouse anti-contagionist scientific views in the early 19th-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 obser-

involved directly with Britain's global trade interests, the higher share of experts in the latter half of the sample is reminiscent with the selection effect that drives the intuition behind the model.

vational 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 3), although some holdouts remained until the very end of the century. However, those holdouts were not differentially political in any measurable way related to trade. 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 values of α and β: the returns to a trade-linked scientists for an anti-contagionist policy shifted over time. For instance, 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 19th 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.

One should note that the findings in this paper focus on the internal dynamics of the scientific community itself, not to 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 own biases.

6 Conclusions

In this paper, I examine the dual role of experts as scientists who search for scientific information about the world and as private actors with a preference over policy. I show how, and the circumstances under, policy goals can interfere with the progress of science. I theorize that conflicted experts will be less 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 19th 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 the "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 bulk of our scientific and medical capabilities date from the huge gains of the 20th century. 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 scientific goals.

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A 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 p-values on 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=122)				Trade=0	(N=343)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0.062	0	0.243	0	1	0.074	0	0.262	0	1	0.714
Prof. orgs.	0.625	0	0.965	0	4	0.951	1	1.189	0	7	0.011
Public audience	0.074	0	0.262	0	1	0.09	0	0.287	0	1	0.648
Age	47.026	47	11.717	23	75	46.646	44	14.469	20	100	0.763

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

	Trade=1	(N=32)				Trade=0	(N=153)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0	0	0	0	0	0.066	0	0.249	0	1	0.215
Prof. orgs.	0.565	0	0.662	0	2	0.759	1	0.836	0	4	0.208
Public audience	0.125	0	0.336	0	1	0.137	0	0.345	0	1	0.934
Age	41.467	40	12.053	23	65	45.02	42	14.627	20	100	0.235

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

	Trade=1	(N=90)				Trade=0	(N=190)				P-Value
	Mean	Med.	SD	Min.	Max.	Mean	Med.	SD	Min.	Max.	
FRCP	0.082	0	0.277	0	1	0.081	0	0.274	0	1	0.993
Prof. orgs.	0.644	0	1.046	0	4	1.128	1	1.42	0	7	0.009
Public audience	0.056	0	0.23	0	1	0.053	0	0.224	0	1	0.901
Age	48.349	49	11.334	29	75	48.109	46.5	14.234	25	83	0.651

B Robustness Checks

Table A4 shows robustness checks for the results shown in Table 2. Column 1 omits the author-per-document weights. 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. Column 3 presents a logistic regression, and Column 4 swaps the category of interest in the outcome to contagionist, pooling anti-contagionist, neutral/unsure, and does-not-mention into the reference category. The smaller point effect in Column 4 is unsurprising, given that many articles that do not mention cholera may not do so precisely because they already consider the pro-contagionist consensus to be firmly established.

Table A4

	Dependent variable:			
	Unweighted	Anti Subsample	Logistic	Pro Swapped outcome
	(1)	(2)	(3)	(4)
Trade link	0.142**	0.150**	0.763**	−0.086
	(0.056)	(0.076)	(0.313)	(0.073)
Date	−0.292	−0.092	−1.113	−0.392
	(0.218)	(0.279)	(1.345)	(0.268)
$Date^2$	0.0001	0.00002	0.0003	0.0001
	(0.0001)	(0.0001)	(0.0004)	(0.0001)
FRCP	−0.078	−0.086	−0.439	0.007
	(0.082)	(0.131)	(0.529)	(0.126)
Memberships	0.012	0.011	0.063	−0.006
	(0.029)	(0.041)	(0.170)	(0.035)
Age	0.014	−0.023	0.066	0.070
	(0.103)	(0.118)	(0.504)	(0.101)
Public audience	276.559	90.348	1,058.377	362.270
	(202.524)	(259.550)	(1,248.218)	(249.287)
Controls	Core	Core	Core	Core
Observations	381	280	381	381
Adjusted R^2	0.046	0.034		0.005
Log Likelihood			−205.771	
Akaike Inf. Crit.			425.541	
Note:				*p<0.1; **p<0.05; ***p<0.01

C Use of Bacteriological Vocabulary

Figure A1 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.

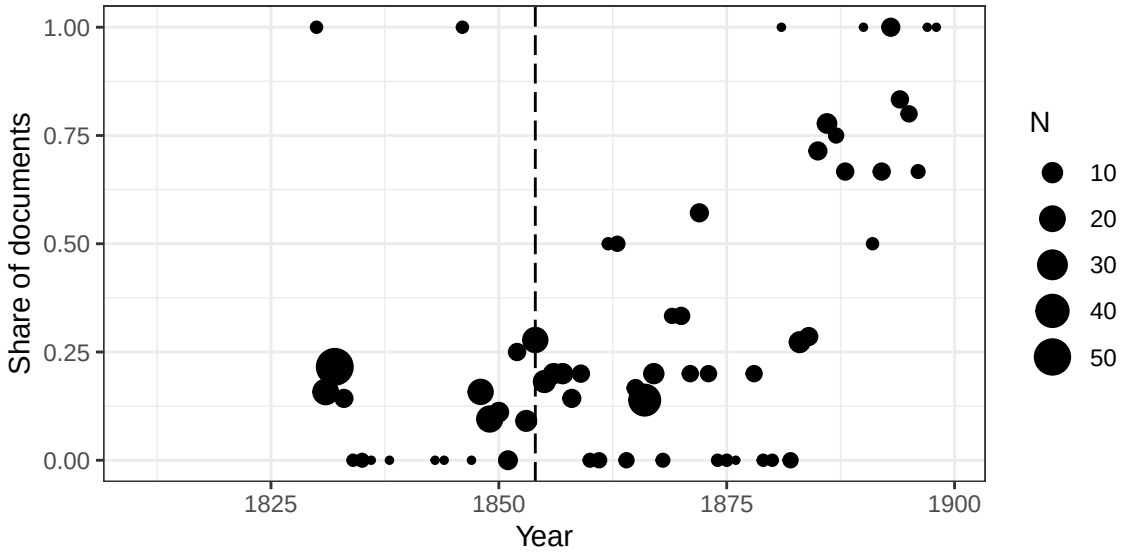

Figure A1