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 reputation for scientific accuracy and their ability to shape policy – goals that sometimes conflict, particularly when experts have personal preferences over a contentious policy choice. To explore this duality, I use evidence from public health interventions during 19th-century cholera pandemics in Britain. 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 scientific methodologies were primitive and the scientific consensus was weak. I argue that biased experts are more likely to act on their bias in low-information environments, when they can convincingly pool with uninformed actors. As a consensus forms and the quality of scientific evidence improves, they are more likely to reveal information as the pooling strategy becomes less plausible.

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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?

This paper presents a model that describes the conditions under which a politically-motivated expert will suppress information that is inconvenient for convincing others to adopt their policy preference. In this model, motivated experts – experts who benefit when others hold a certain belief about the state of the world – will hide inconvenient information when the scientific consensus is weaker, scientific tools and methodologies are less informative, or there exist higher levels of uncertainty about the true "state of the world." When these conditions are met, experts who have uncovered information that is "bad" for their preferred policy position can convincingly pool with those who have no useful information to reveal. As a consensus forms and methodological precision improves, motivated experts who claim to have no scientific information to reveal are suspected of suppressing information and are punished by their peers for acting on their personal preferences.

I support this theoretical framework 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. For 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, it was an environmental (or "miasmatic") disease caused by weather patterns, pollution, or other factors not directly linked to the movements of individual infected carriers, 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 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.

The COVID-19 pandemic has made clear 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 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 carriers of the cholera bacteria, some of whom may be asymptomatic. 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 infectious 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 were contagious in any sense of the word, quarantines could be a helpful counter-measure, although they required paying an economic cost. If cholera were spread by some mechanism that did not involve infected carriers, quarantines would be useless, imposing economic costs and infringing on freedom of movement to no health benefit.

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).[1] Contagionist theories of cholera included any explanations that hinged on cholera being spread

[1]Medical terminology was fairly non-standardized at this time, and different writers sometimes used vocabulary in different ways.

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. Tools for observing the natural world at the microscopic level were rudimentary (Tröhler 2005). Formal medical training still incorporated ideas, such as bodily 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, they 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 – made possible 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 ill-

ness 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, predated the first Eurasian cholera outbreak by only a few years (Oppenheimer 2003). Its damage to the food supply may have made people more susceptible to disease.

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). He backed up this theory several years later with evidence from his famous natural experiment 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 spontaneous generation 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 worry that it could somehow cross the English Channel. 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.[2]

Despite these precautionary policies, 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?"[3] Cholera had become part of the broader landscape of urban health hazards.

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

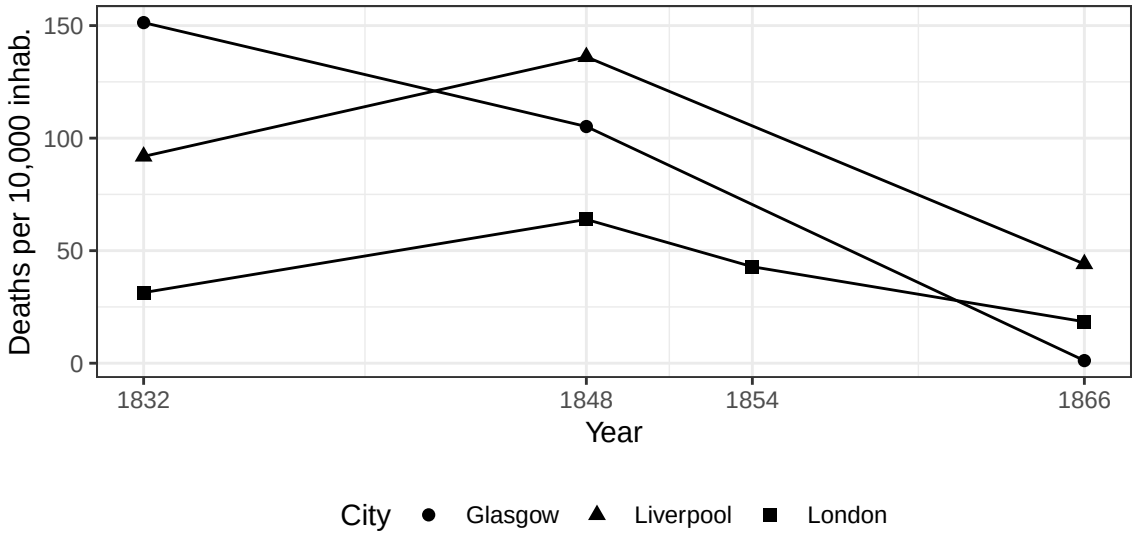

Figure 1: Mortality rates for selected cities during 19th-century cholera pandemics. Data for Glasgow comes from Underwood (1948) and University of Portsmouth (2017); for Liverpool from Underwood (1948) and Battersby (2017); and for London from Underwood (1948), Royal Society of Arts (1878), and Haughton (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). For comparison, the COVID-19 mortality rate for New York City during the first year of the COVID-19 pandemic was approximately 35.6 people per 10,000.

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.

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).[4] Similar investments in other cities had a comparative effect on eradicating these public health threats.[5]

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

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

[5]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.

(Howard-Jones 1975).[6] 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.[7]

3 Theory

3.1 The Motivations of Experts

Scholarship from the philosophy, and sociology of science tends to assume that scientific experts exert effort to learn about the world because they are motivated by intrinsic curiosity (Kuhn 1970; Popper 1972). A literature on expert predictions from economics assumes a more pedestrian 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."[8]

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, 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,

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

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

[8] 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.

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.[9] 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 later standards, but it was evidence nonetheless. There was no moment of clear state validation about cholera's transmission mechanism, rather, a gradual 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 make the assumption 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 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

[9]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).

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.[10] 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) from 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 a reputation for bias rather than a reputation for incompetence. 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 African researchers worked: poverty alleviation was a major priority for the South African government, and South African public health sci-

[10]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).

entists 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

Building on the notion of epistemic "tightness" from Mokyr (2011), I propose the following 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 $\pi = P(S_1) = 1 - P(S_0)$.[11] An expert, or scientist, searches for new evidence. Upon searching, there is probability p that the scientist uncovers evidence about S, which takes the form of 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, but it is more likely that a signal that does match the state of the world.

An expert who finds a signal has the opportunity to reveal it. Alternately, he can claim to have found nothing. However, he cannot falsify evidence he did not find. If the expert reveals his signal, the community updates its common prior in a typical Bayesian fashion:

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

if $\sigma = \sigma_1$ and

$$\pi^* = P(S_1|\sigma_0) = \frac{P(\sigma_0|S_1)\pi}{P(\sigma_0|S_1)\pi + P(\sigma_0|S_0)(1-\pi)} \tag{2}$$

if $\sigma = \sigma_0$.

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

There are two types of experts: "unconflicted experts" ("he") and "conflicted experts" ("she"). An unconflicted expert's payoff is solely a function of how far he moves the community's prior after he shows them a signal:

$$U_{\text{unconflicted}} = \alpha|\pi - \pi^*| \tag{3}$$

An unconflicted expert will always reveal any signal he receives, as doing so always causes the community's belief to shift and give him positive utility.

A conflicted expert receives the same payoff as an unconflicted expert for changing the community's belief. 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):

$$U_{\text{conflicted}} = \alpha|\pi - \pi^*| + \beta(\mathbb{1}_{\pi^* > \theta}) \tag{4}$$

Thus, a conflicted expert will not necessarily always *act* on her bias. Any expert's type is common knowledge, as are the values of the parameters α and β are common knowledge.[12] An expert of either type who reveals no information (either because he or she obtained no signal or because he or she obtained one and chose not to reveal it) gets no payoff for changing the community's belief (although, as I show later, there are cases in which the community's belief may change even when no information is revealed).

If an unconflicted expert reveals no signal, the community knows he received no useful information. They do not update their prior, retaining the belief $P(S_1) = \pi$, and the unconflicted expert receives a payoff of 0. If the unconflicted expert reveals a signal, the

[12]This diverges from a classic family of signalling models, such as Spence (1978), in which experts' type is hidden, but I argue that in the context of experts with potential conflicts of interest, the conflict is typically 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.

scientific community updates their beliefs according to Equations 1 and 2, and the expert receives the payoff $\alpha|\pi^* - \pi|$.

If a conflicted expert reveals a signal, the community updates their belief according to Equations 1 and 2, since experts cannot falsify signals. If she does not reveal a signal, two possibilities exist: either she received no signal (an event that occurs with probability p conditional on the expert having searched), or she received a signal and chose not to reveal it.

A conflicted expert who receives the signal σ_1 will always reveal it, because that signal is compatible with both parts of her utility function. Because it cannot be falsified, it causes the community to update their prior according to Equation 1, increasing U_{biased} by $\alpha|\pi^* - \pi|$ and, if $\pi^* > \theta$, by β as well.

Should, and by how much, will the community update their belief if a conflicted expert claims to be uninformed? First, consider the case of $\pi < \theta$. The prior belief is already to the left of θ, and the expert only receives β when it is to the right. If the community could verify that the expert is uninformed, their posterior belief would equal their prior, and the expert would receive a payout of 0. If the community could verify that the signal is s_0, they would update to a posterior further to the right of π, and the expert would receive a payout of $\alpha|\pi^* - \pi|$. Thus, an expert who claims to be uninformed when $\pi < \theta$ is credible, and the community retains a belief that $P(S_1) = \pi$.

On the other hand, suppose the community's prior is within the expert's preferred range ($\pi > \theta$). The community's assessment of the expert's credibility depends on whether their posterior belief would still be in the preferred range if they knew the expert had received a signal of σ_0. Setting Equation 2 equal to θ and solving for π yields

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

If $\pi > \omega$, then $\pi^*|\sigma_0 > \theta$. An expert who has a signal σ_0 can reveal it and get $U_{\text{biased}} = \alpha|\pi^* - \pi| + \beta$, the maximum possible payoff given π. Thus, if $\pi > \omega$ and a conflicted expert claims to be uninformed, they are credible, and the community will maintain the belief that $P(S_1) = \pi$.

On the other hand, if $\pi < \omega$. Suppose the expert reveals no information. The community knows she either received σ_0 or no signal but cannot verify which (since she always reveals σ_1). They also know that the probability of receiving a signal is p and of receiving none is $1 - p$. They will adopt a different estimated posterior, $\pi^\dagger$, when a conflicted type shows them no signal:

$$\pi^\dagger = p[\pi^*|\sigma_0] + (1 - p)\pi \tag{6}$$

Although the community has updated its prior in this probabilistic manner, the expert will receive no payoff associated with α since she has not actively contributed to scientific discovery. Thus, if the conflicted expert who sees σ_0 when $\pi < \omega$ reveals nothing, she receives $U_{\text{conflicted}} = \beta(\mathbb{1}_{\pi^\dagger > \theta})$. If she reveals it, she receives $U_{\text{conflicted}} = \alpha|\pi^* - \pi|$. If $\alpha|\pi^* - \pi| > \beta$, she will reveal. Extending this line of reasoning, if $\alpha|\pi^* - \pi| > \beta$, an expert who does *not* reveal must be truly uninformed (which in turn gives the community the information they need to form a posterior belief). On the other hand, if $\beta > \alpha|\pi^* - \pi|$, the conflicted expert will pool with "no signal" for a payoff of β and the community will update its belief to $\pi^\dagger$. The intuition behind these results is visualized in Figure 2.

To summarize, the conditions that must hold for a conflicted expert to suppress her information and pool with the uninformed are

1. $\theta < \pi < \omega$: π is to the left of θ (the audience is already convinced to reject the range to the right of θ) or to the right of ω (revealing information would sway the audience away believing $S = S_1$ but would not make the posterior fall below θ once updated).

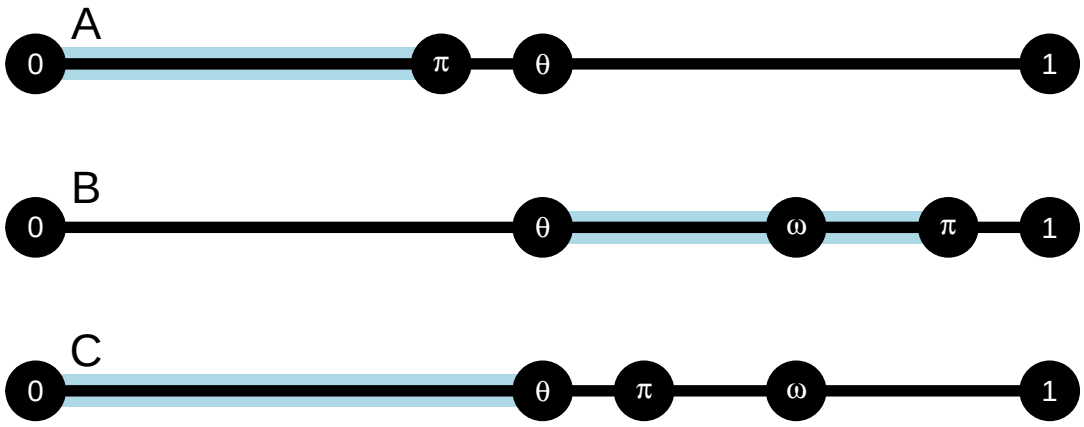

Figure 2: This figure shows the range of possible posterior beliefs (represented by shaded regions) when (A) $\pi < \theta$; (B) $\theta < \omega < \pi$; and (C) $\theta < \pi < \omega$. A conflicted expert will reveal an unfavorable signal σ_0 in Cases A and B but may suppress it in Case C.

2. $\pi^\dagger > \theta$: the belief about $P(S_1)$ to which the audience will default if they see no signal is not larger than θ

3. $\beta > \alpha|\pi^* - \pi|$: the expert gets a higher reward from the community's belief remaining to the right of θ than she does from changing the community's belief

The notion espoused by Mokyr (2011) of epistemic "tightness" is captured by several different parameters. First, tightness is represented by a stronger prior that is further from 0.5. Second, it appears in the informational content of the signal. When $P(\sigma_i|S_i)$ is relatively high, and $P(\sigma_i|S_j)$ relatively low, σ is a better predictor of S. This captures the level of sophistication and predictive power of scientific techniques or methodologies. Additionally, higher levels of epistemic tightness suggest a lower value of p: the community becomes more certain about *how* to do research and therefore expects a lower rate of ambiguous results.

Changes in these parameters do not affect the strategy of a unconflicted expert, who always reveals his information when he has it, but they matter for the equilibrium strategy of an conflicted expert. As π becomes stronger, moving closer to either 0 or 1, while θ remains

fixed, it becomes increasingly likely that Condition 1 will not be met. On the other hand, a stronger π decreases the returns to revealing a signal (because π^* will be closer to π), meaning that an expert can afford to reveal information without affecting policy except when π is quite close to θ.

Now consider the effects of changes in $P(\sigma_0|S_0)$ and $P(\sigma_0|S_1)$ on ω:

$$\frac{\delta\omega}{\delta P(\sigma_0|S_0)} = -\frac{(\theta-1)\theta P(\sigma_0|S_1)}{(\theta(P(\sigma_0|S_0)-P(\sigma_0|S_1))+P(\sigma_0|S_1))^2} \geq 0$$

$$\frac{\delta\omega}{\delta P(\sigma_0|S_1)} = \frac{(\theta-1)\theta P(\sigma_0|S_0)}{(\theta(P(\sigma_0|S_0)-P(\sigma_0|S_1))+P(\sigma_0|S_1))^2} \leq 0$$

As $P(\sigma_0)$ gets larger or $P(\sigma_0|S_1)$ gets smaller, ω moves further towards 1, narrowing the window in which π can fall such that the expert suppresses her information according to Condition 1. Furthermore, as the signal's informational value increases, $|\pi^* - \pi|$ is larger when a signal is revealed. Because $\pi^\dagger$ is a function of π^*, so to is $\pi^\dagger - \pi$. This means that the audience's default assumption when they see no signal will be closer to 0, decreasing the incentive to hide information via Condition 2.

Finally, as p, the probability of getting a signal, increases, an informed expert's ability to pool with the uninformed decreases because of their lower prevalence in the population, also decreasing the incentive to hide information via Condition 2.

3.3 Connection to Case Study

The model outlined above predicts that in general, conflicted experts will be more willing to reveal inconvenient information in a "tight world" because if they don't, their peers will be more apt to believe the worst – that they are hiding something, rather than being uninformed. Mapping this model to evidence from the first cholera pandemic generates the hypothesis that experts who have a reason to be conflicted will be more likely to espouse anti-contagion views relative to unconflicted experts - but only in the early part of the sample period, before

scientific techniques were well-developed and when prior beliefs were still weak, and when conflicted experts' preferred policy was politically within reach. The remainder of this paper tests the validity of that hypothesis.

4 Data and Research Design

4.1 Data

Data on the stated views of the scientific community about public health comes from the Medical Heritage Library (MHL), an online digitized archive 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 that can be used for quantitative text analysis. 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 unified professional communities. I identify 548 qualifying items that are *primarily* about cholera, based on contributor-supplied subject tags in the metadata (supplemented with other untagged documents that contain very frequent use of the term "cholera.")

Figure 3 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 legitimate claim to expertise by the standards of the 19th century. Furthermore, based on my conversations with curators, there is no reason to believe that the MHL collection was purged after the fact.

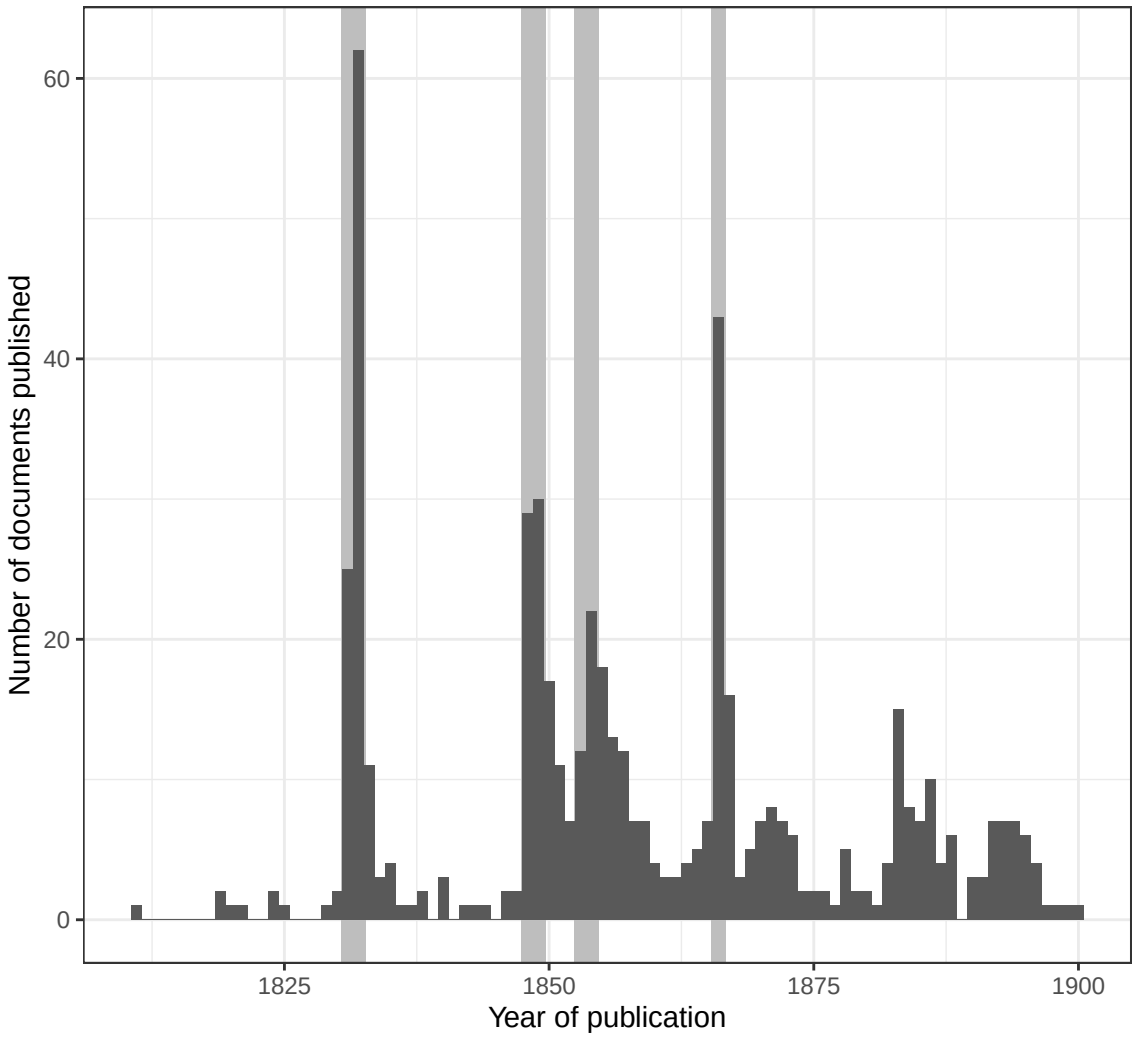

Figure 3: Number of documents in MHL sample by year of publication. Grey bars denote epidemic years.

The 548 observations in the corpus include both documents with named authors and documents attributed to institutions (usually government bodies; some documents with institutional affiliations also have named authorship). Some observations are authored by multiple

individuals. For named authors, MHL metadata and/or the document itself typically offer basic biographical data: the year of an author's birth and death; relevant academic degrees; and professional affiliations (such as membership in the Royal Society or Royal College of Surgeons). I verify and extend these author-level data using secondary sources (dictionaries of national biography and obituaries published in major medical journals). Table 1 shows the share of all documents by institutional affiliation with colonial institutions, local governments, or central governments.

4.2 Research Design

Section 3.3 suggests a testable hypothesis: conflicted experts will act on their source of conflict, but only in an epistemically weak 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 fear a strict quarantine policy might fall prey to injecting 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 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 affects an expert's potential to act on a conflict of interest. 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 professional association with interest groups linked to overseas British trade. I classify an author as "trade-linked" if he was based in a British overseas colony at the time of authorship.[13] Most authors based in overseas colonies were directly employed by the British government on behalf of either a civilian or military administration. These overseas 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. Given the degree to which these institutions were intertwined, I do not differentiate between the two. A small number ($N = 5$) authors have no stated affiliation with the government or trade interests per se but were located in a British overseas colony when they contributed their scholarship; I count these as "trade-linked" as well.

I gather additional data to adjust for various potential confounders in my specifications. I include linear and quadratic terms for date in all specifications. I control for whether a scientist was a member of the medical elite – a Fellow of the Royal Society of Physicians of England (FRCP) – as well as for the number of other professional memberships, which proxies 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

[13]The MHL sample is composed almost entirely of male authors; the sole exception is an article by Florence Nightingale.

[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.

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. Table 2 shows summary statistics of document-level characteristics and 1 of document-level characteristics.

Table 1

Statistic	N	Mean	St. Dev.	Min	Pctl(25)	Pctl(75)	Max
Anti-contagionist	359	0.376	0.485	0	0	1	1
Author has trade link	359	0.206	0.405	0	0	0	1
Date	359	1,857.454	20.757	1,811	1,834.5	1,871	1,898
Author's age	251	49.490	14.059	22.000	39.000	57.000	100.000
Public audience	359	0.103	0.304	0	0	0	1

Table 2

Statistic	N	Mean	St. Dev.	Min	Pctl(25)	Pctl(75)	Max
FRCP	364	0.069	0.253	0	0	0	1
Memberships	363	0.736	0.935	0.000	0.000	1.000	7.000

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 better reflects a contagionist or anti-contagionist viewpoint. 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. 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 3 shows examples of each category.

Figure 4 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 with the government's

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

expert advisors that, on the basis of known evidence, cholera exhibited the characteristics of a contagious disease. With each successive wave of disease, fresh experiences caused experts to steadily decrease in their adherence to "anti-contagionism" (represented by the uppermost segment of the bar plot in Figure 4). 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 are more focused on other aspects of disease management, such as documenting individual clinical cases, while others may consider the question of transmission to be settled science and do not bother to engage.)

5 Analysis

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

The share of articles espousing an anti-contagionist by trade-linked scientists 0.46, while of the articles written by scientists without political links to trade, that share is 0.35. However,

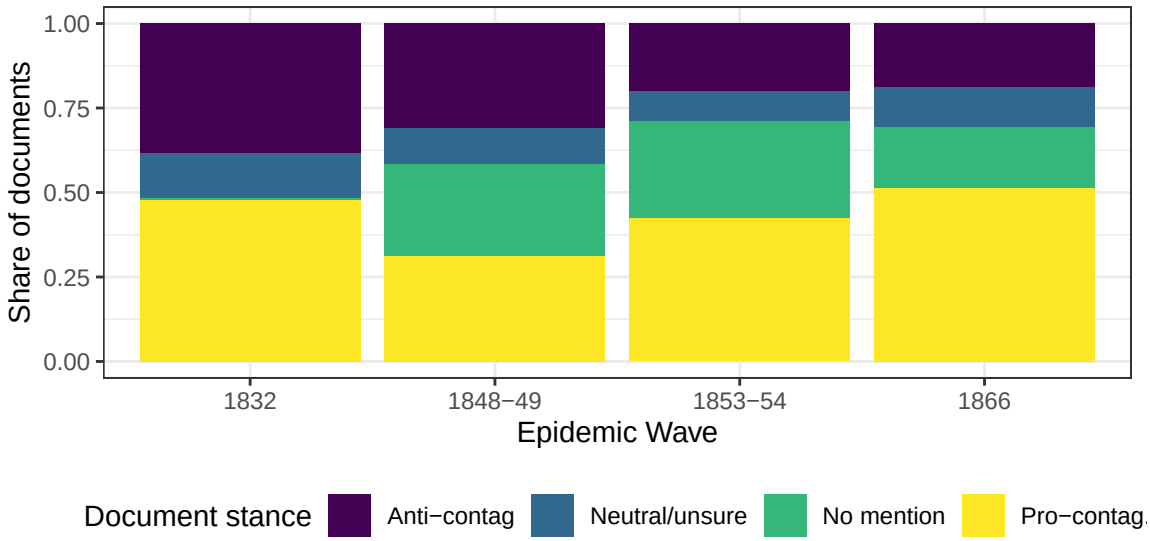

Figure 4: Share of 19th-c. British scientific articles pro-contagionist, anti-contagionist, or neutral view, by wave. While the share of researchers expressing pro-contagionist views remains roughly steady over time, the share expressing anti-contagionist views shrinks.

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:

$$\mathbb{1}_{anti_i} = \alpha + \beta \mathbb{1}_{trade_i} + \mathbf{X}\gamma + \epsilon_\mathbf{i} \tag{7}$$

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. The regression is run on the sample of documents with named authors (vs. strictly institutional documents) at the document-author level. 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 (to accommodate authors who contribute multiple documents).

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. Anti-contagionist views, always in the minority and increasingly out-of-consensus over the sample period, are more naturally analyzed in comparison to all other views.

Table 4

	Dependent variable:		
	Anti-contagionist stance		
	(1)	(2)	(3)
Trade link	0.116*	0.116*	0.098
	(0.069)	(0.070)	(0.089)
Date	−0.190	−0.190	−0.122
	(0.227)	(0.232)	(0.286)
Date^2	0.0001	0.0001	0.00003
	(0.0001)	(0.0001)	(0.0001)
FRCP			0.008***
			(0.003)
Memberships		−0.080	−0.118
		(0.144)	(0.147)
Age		0.005	0.001
		(0.039)	(0.039)
Public audience		0.048	−0.235*
		(0.111)	(0.124)
Constant	180.416	180.444	118.111
	(211.322)	(216.190)	(266.420)
Controls	Date only	Core	Extended
Observations	359	359	251
Adjusted R^2	0.024	0.019	0.046
Note:		*p<0.1; **p<0.05; ***p<0.01	

If an author of a document is politically associated with trade, either via direct employment for an overseas trade company, direct employment in an arm of the Britain's overseas colonial administrations, or residence in an overseas British colony, that document is about 10-11% more likely to espouse an anti-contagionist hypothesis of cholera transmission. The coefficient on trade linkages is significant at the 10% level in Columns 1 and 2 of Table 4. 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 4 demonstrate that trade-linked experts are more likely than their peers to draw anti-contagionist conclusions about cholera, although the effect is somewhat noisy by the standards of classical statistical significance. 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:

$$\mathbb{1}_{anti_i} = \alpha + \beta_0 \mathbb{1}_{trade_i} + \beta_1 \mathbb{1}_{date_i > 1854} + \beta_2 \mathbb{1}_{trade_i} \times \mathbb{1}_{date_i > 1854} + \mathbf{X}\gamma + \epsilon_{\mathbf{i}} \tag{8}$$

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. As an alternate specification, I re-run Equation 7 on "late" versus "early" subsamples. In the early sample, the share of

trade-linked experts is 0.13; in the late sample, the share is 0.26.

Table 5

	Dependent variable:					
	Anti-contagionist stance					
	Interaction	Interaction	Early Subsample	Early Subsample	Late Subsample	Late Subsample
	(1)	(2)	(3)	(4)	(5)	(6)
Trade link	−0.004	0.026	0.373***	0.384*	0.003	0.036
	(0.079)	(0.093)	(0.118)	(0.216)	(0.084)	(0.102)
Date<1854	0.025	0.098				
	(0.100)	(0.115)				
Trade link × date<1854	0.397***	0.379				
	(0.140)	(0.232)				
Date	−0.050	0.109	−0.097	0.039	0.273	0.243
	(0.242)	(0.314)	(1.617)	(2.684)	(0.911)	(1.093)
$Date^2$	0.00001	−0.00003	0.00003	−0.00001	−0.0001	−0.0001
	(0.0001)	(0.0001)	(0.0004)	(0.001)	(0.0002)	(0.0003)
Age		0.008***		0.004		0.011**
		(0.003)		(0.004)		(0.004)
FRCP	−0.067	−0.097	0.048	0.0004	−0.119	−0.162
	(0.145)	(0.149)	(0.202)	(0.221)	(0.194)	(0.179)
Memberships	0.005	0.001	−0.069	−0.052	0.040	0.020
	(0.039)	(0.039)	(0.054)	(0.062)	(0.050)	(0.047)
Public audience	0.063	−0.194	0.038	−0.148	0.123	−0.220
	(0.103)	(0.122)	(0.122)	(0.153)	(0.201)	(0.201)
Constant	48.259	−99.602	91.899	−34.260	−254.880	−224.665
	(225.608)	(293.360)	(1,485.869)	(2,468.028)	(855.071)	(1,025.274)
Controls	Core	Extended	Core	Extended	Core	Extended
Observations	359	251	155	102	204	149
Adjusted R^2	0.039	0.060	0.058	0.015	−0.017	0.058

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

Table 5 show the results of this exercise for both the "core" and "extended" samples, as defined above. Consistent with the hypothesis, the preference of trade-linked experts for anti-contagionist explanations is entirely driven by the early period.

5.3 Discussion

The analysis above shows 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. In this section, I discuss how to interpret this finding in light of the model presented in Section 3.2.

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 4), 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.

A potential alternate explanation for why trade-linked scientists abandoned the bias they held in the early period runs through the α and β parameters in the model: 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. 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 entry 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 try to gather information about the world and as advocates for a policy position. 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 hampers 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, they might 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.

7 References

Alesina, Alberto, and Guido Tabellini. 2007. "Bureaucrats or politicians? Part I: A single policy task." *American Economic Review* 97 (1): 169–79. https://doi.org/10.1257/aer.97.1.169.

Alsan, Marcella, and Claudia Goldin. 2019. "Watersheds in child mortality: The role of effective water and sewerage infrastructure, 1880–1920." *Journal of Political Economy* 127 (2): 586. https://doi.org/10.1086/700766.

Andrews, Isaiah, and Jesse M. Shapiro. 2021. "A Model of Scientific Communication." *Econometrica* 89 (5): 2117–42.

Ashworth, Scott. 2012. "Electoral accountability: Recent theoretical and empirical work." *Annual Review of Political Science* 15: 183–201. https://doi.org/10.1146/annurev-polisci-031710-103823.

Avery, Christopher N., and Judith A. Chevalier. 1999. "Herding over the career." *Economics Letters* 63 (3): 327–33. https://doi.org/10.1016/s0165-1765(99)00039-7.

Backus, Matthew, and Andrew T. Little. 2020. "I Don't Know." *American Political Science Review* 114 (3): 724–43. https://doi.org/10.1017/S0003055420000209.

Baldwin, Peter. 1999. *Contagion and the State in Europe, 1830-1930.* Cambridge: Cam-

bridge University Press.
Battersby, Stephen. 2017. "Duncan of Liverpool: The first Medical Health Officer." In *Pioneers in Public Health: Lessons from History*.
Brandenburger, Adam, and Ben Polak. 1996. "When Managers Cover Their Posteriors: Making the Decisions the Market Wants to See." *The RAND Journal of Economics* 27 (3): 523–41. https://doi.org/10.2307/2555842.
Canes-Wrone, Brandice, Michael C. Herron, and Kenneth W. Shotts. 2001. "Leadership and Pandering: A Theory of Executive Policymaking." *American Journal of Political Science* 45 (3): 532. https://doi.org/10.2307/2669237.
Carpenter, Daniel P. 2003. "Why do bureaucrats delay? Lessons from a stochastic optimal stopping model of agency timing, with applications to the FDA." In *Politics, Policy, and Organizations: Frontiers in the Scientific Study of Bureaucracy*, edited by George A. Krause and Kenneth J. Meier, 23–40.
Crawford, VP, and J Sobel. 1982. "Strategic information transformation."
Cutler, David, and Grant Miller. 2005. "The role of public health improvements in health advances: The twentieth-century United States." *Demography* 42 (1): 1–22.
Downs, George W., and David M. Rocke. 1994. "Conflict , Agency , and Gambling for Resurrection : The Principal-Agent Problem Goes to War Author (s): George W . Downs and David M . Rocke Source : American Journal of Political Science , Vol . 38 , No . 2 (May , 1994), pp . 362-380 Published by : M." *Political Science* 38 (2): 362–80.
Durey, Michael. 1979. *The Return of the Plague: British Society and the Cholera 1831-1832*. Dublin: Gill; Macmillan Ltd.
Effinger, Matthias R., and Mattias K. Polborn. 2001. "Herding and anti-herding: A model of reputational differentiation." *European Economic Review* 45 (3): 385–403. https://doi.org/10.1016/S0014-2921(00)00070-2.
Evans, Richard J. 2005. *Death in Hamburg: Society and Politics in the Cholera Years*. Penguin.
Fearon, James. 1999. "Electoral Accountability and the Control of Politicians: Selecting Good Types verus Sanctioning Poor Performance." In *Democracy, Accountability, and Representation*, edited by Adam Przeworski, Susan C. Stokes, and Bernard Manin, 55–97. Cambridge: Cambridge University Press.
Ferrie, Joseph P., and Werner Troesken. 2008. "Water and Chicago's mortality transition, 1850-1925." *Explorations in Economic History* 45 (1): 1–16. https://doi.org/10.1016/j.eeh.2007.06.001.
Flinders, Matthew. 2020. "Gotcha! Coronavirus, Crises and the Politics of Blame Games." *Political Insight* 11 (2): 22–25. https://doi.org/10.1177/2041905820933371.
Fox, Justin, and Stuart V. Jordan. 2011. "Delegation and accountability." *Journal of Politics* 73 (3): 831–44. https://doi.org/10.1017/S0022381611000491.
Fox, Justin, and Richard Van Weelden. 2012. "Costly transparency." *Journal of Public Economics* 96 (1-2): 142–50. https://doi.org/10.1016/j.jpubeco.2011.08.007.
Gailmard, Sean, and John W Patty. 2013. "Stovepiping." *Journal of Theoretical Politics* 25 (3): 388–411. https://doi.org/10.1177/0951629813477277.
Gallardo Albarrán, Daniel. 2018. "Sanitary infrastructures and the decline of mortality in Germany, 1877-1913." 176th Series. University of Groningen.
Haughton, Samuel. 1867. "A Scientific Inquiry Into Some of the Causes Alleged to Produce

Asiatic Cholera." *The Medical Times and Gazette* 1: 713–14.

Hill, Ryan, and Carolyn Stein. 2021. "Race to the Bottom : Competition and Quality in Science."

Howard-Jones, N. 1975. "The scientific background of the International Sanitary Conferences, 1851-1938." *History of International Public Health* 1. https://doi.org/10.1136/bmj.2.6035.592.

Johnson, E. 2012. "Political Correctness." *Encyclopedia of Applied Ethics* 109 (2): 496–504. https://doi.org/10.1016/B978-0-12-373932-2.00287-8.

Johnson, Steven. 2006. *The Ghost Map: The Story of London's Most Terrifying Epidemic–and How It Changed Science, Cities, and the Modern World*. Penguin.

Kesztenbaum, Lionel, and Jean-Laurent Rosenthal. 2016. "Sewers' diffusion and the decline of mortality: The case of Paris, 1880–1914." *Journal of Urban Economics*, 1–13.

Koch, Tom. 2005. "Mapping the Miasma: Air, health, and place in early medical mapping." *Cartographic Perspectives*, no. 52: 4–27. https://doi.org/10.14714/CP52.376.

Kuhn, Thomas S. 1970. *The Structure of Scientific Revolutions*. 2nd ed. Chicago: University of Chicago Press.

Levy, Gilat. 2004. "Anti-herding and strategic consultation." *European Economic Review* 48 (3): 503–25. https://doi.org/10.1016/S0014-2921(03)00019-9.

Luckin, Bill. 1977. "The Final Catastrophe: Cholera in London, 1866." *Death and Survival in Urban Britain*, 9–12. https://doi.org/10.5040/9780755621576.ch-004.

Majumdar, Sumon, and Sharun W. Mukand. 2004. "Policy Gambles." *American Economic Review* 94 (4).

Mokyr, Joel. 2011. "The Gifts of Athena." In *The Gifts of Athena*. Princeton University Press.

Ogasawara, Kota, Shinichiro Shirota, and Genya Kobayashi. 2018. "Public health improvements and mortality in interwar Tokyo: a Bayesian disease mapping approach." *Cliometrica* 12 (1): 1–31. https://doi.org/10.1007/s11698-016-0148-3.

Oppenheimer, Clive. 2003. "Climatic, environmental and human con sequences of the largest known historic eruption: Tambora volcano (Indonesia) 1815." *Progress in Physical Geography* 27 (2): 230–59. https://doi.org/10.1191/0309133303pp379ra.

Ottaviani, Marco, and Peter Sørensen. 2001. "Information aggregation in debate: Who should speak first?" *Journal of Public Economics* 81 (3): 393–421. https://doi.org/10.1016/S0047-2727(00)00119-5.

Ottaviani, Marco, and Peter Norman Sørensen. 2006. "Reputational cheap talk." *RAND Journal of Economics* 37 (1): 155–75. https://doi.org/10.1111/j.1756-2171.2006.tb00010.x.

Pelling, Margaret. 1978. *Cholera, fever and English medicine, 1825-1865*. Oxford: Oxford University Press.

Popper, Karl. 1972. "The logic and evolution of scientific theory." In *All Life Is Problem Solving*, 3–22. New York: Routledge. http://library1.nida.ac.th/termpaper6/sd/2554/19755.pdf.

Richterman, Aaron, Duarxy Rodcnel Sainvilien, Lauren Eberly, and Louise C. Ivers. 2018. "Individual and household risk factors for symptomatic cholera infection: A systematic review and meta-analysis." *Journal of Infectious Diseases* 218 (Suppl 3): S154–64. https://doi.org/10.1093/infdis/jiy444.

Royal Society of Arts. 1878. *National Water Supply: Notes on Previous Inquiries, Prepared in Connexion with the Congress, Held Tuesday and Wednesday, 21st and 22nd May, 1878*. London: George Bell; Sons.

Scharfstein, David S., and Jeremy C. Stein. 1990. "Herd Behavior and Investment." *American Economic Review* 80 (465-479).

Spence, Michael. 1978. "Job Market Signaling." In *Uncertainty in Economics*, 281–306. Elsevier.

Tröhler, U. 2005. "Lind and scurvy: 1747 to 1795." *Journal of the Royal Society of Medicine* 98 (11): 519–22. https://doi.org/10.1258/jrsm.98.11.519.

Underwood, E Ashworth. 1948. "The History of Cholera in Great Britain." *Proceedings of the Royal Society of Medicine* 41 (3): 165–73. https://doi.org/10.1177/003591574804100309.

University of Portsmouth. 2017. "A Vision of Britain Through Time." https://www.visionofbritain.org.uk.

Wrigley, Edward Anthony, and Roger S Schofield. 1989. *The population history of England 1541-1871*. Cambridge University Press.

Youde, Jeremy. 2005. "The Development of a Counter-Epistemic Community: AIDS, South Africa, and International Regimes." *International Relations* 19 (4): 421–39. https://doi.org/10.1177/0047117805058533.