

The curious case of Dr Farr's theory of miasma
and inferential knowledge from falsehood

Nathan Oseroff

Introduction

Consider the slogan, ‘inferential knowledge requires known basis beliefs’ (Audi 2003). This is a plausible slogan to uphold: it would be inappropriate to ascribe inferential knowledge to an epistemic agent if their inferred belief is solely predicated on relevant *unknown* basis beliefs. To admit this would be to invite epistemic anarchy. For the following discussion, let us focus on the condition of truth. Since all theories of knowledge have a condition of truth, the basis belief P must be true if one inferentially knows Q based solely on P .

There are competing explanations for why inferential knowledge requires true basis beliefs. All available explanations are unified by the common assumption that there is a failure to preserve desirable features of deductive inferences. For example, a true belief based on false basis beliefs is not due to epistemically valuable aspects of the epistemic agent, the belief or their present environment. While the resultant inferential true belief Q may be approximate to or share similarities with knowledge, S does not *automatically* inferentially know that Q based on P so long as P is false.

Frederico Luzzi provides one unifying explanation for this intuition:

... the general thesis is that the epistemic pedigree of a conclusion can seemingly be no better than the epistemic pedigree of the [basis belief]...it is not possible for the premise to have a pedigree that falls short of knowledge yet for the conclusion to have a pedigree sufficient for knowledge (Luzzi 2012, p. 32)

. More colloquially, the belief that epistemic pedigree of inferential knowledge and basis beliefs adheres to the commonsense belief that *one cannot squeeze blood from a stone*. We can set out this commonsense belief as follows:

Epistemic counter-closure: S inferentially knows that Q iff S comes to believe and maintain Q solely on the relevant basis belief(s) P , S knows that P entails Q , and S knows that P .

We can call this the *counter-closure thesis*: in order for S to inferentially know that Q , the relevant basis belief(s) P must, at minimum, be *known*. If counter-

closure were accepted, deductive inference can do no more than preserve knowledge, deductively transferring the pedigree of each basis belief on down; thus knowledge cannot be generated from false basis beliefs (Luzzi 2012, p. 9), just as gold cannot be generated from dross.

Several philosophers explicitly assume this thesis (e.g. Armstrong 1973, pp. 198-9; Nozick 1981, p. 231; Stanley 2005, p. 89). Furthermore, it is *prima facie* plausible. Acceptance of counter-closure is the establishment view. Can counter-closure be maintained? I argue it cannot, and for an unexpected reason: certain types of dross in the right epistemic furnace can give us gold. In what follows, I focus on failures to satisfy the condition of truth that provide grounds for rejecting counter-closure. In order to prevent an epistemic free-for-all, the example I provide is one in which a particular method (namely consulting the predictions derived from Dr Farr's zymotic theory of disease transmission) was exceedingly reliable and surprisingly fecund in a number of areas of public health.

This demonstrated reliability provides a suitable desirable condition for contexts in which it would be appropriate to ascribe inferential knowledge to epistemic agents with relevant false basis beliefs. Consequently, the reliability of the method of consulting a false basis belief sets limits on inferential knowledge based on false basis beliefs, thus closing the doors to epistemic anarchy.

1 Three diagnoses for perceptual basis beliefs

Logical entailment preserves what Fitelson calls 'good-making features' of premises such as truth. Accepting counter-closure appears plausible, in part, because the contrapositive appears plausible as well: entailment preserves the 'bad-making' features of premises, such as the preservation of falsity from basis beliefs. However, this is not so: logical entailment fails to preserve 'bad-making' features of premises.

The kernel of the puzzle facing the acceptance of counter-closure is as follows: if counter-closure is maintained, inferential knowledge cannot ever be gleaned from consulting false basis beliefs; however, consider the following two cases:

Case A: I have the perceptual belief is that that you are five feet tall. I

believe that since you are five feet tall, you cannot be taller than eighteen feet. I believe truly that you are not greater than eighteen feet tall, but my perceptual belief is false: *you are four-foot-eleven*.

Case B: I have the perceptual belief is that you are eleven feet tall. I believe that since you are eleven feet tall, you cannot be taller than eighteen feet. I believe truly that you are not greater than eighteen feet tall, but my perceptual belief is false: *you are four-foot-eleven*.

The first plausible explanation for why it would seem without any issue to ascribe knowledge to me in case A and not case B is that my false perceptual belief in A is ‘approximate to’ or ‘nearby’ the truth, while my false perceptual belief in B is not. There is an important mitigating factor in A not present in B: the closeness of the false perceptual belief to the nearby true belief ‘washes out’ any disvalue. We can call this diagnosis *truth proximity*.

The reasons that underlie this closeness to the truth may be different in extenuating circumstances: had I been in an Ames room—a small room in which there is the optical illusion that a person appears to be taller if they stand in one corner and shorter in the other—if my friend Anna should stand in a corner that makes her appear to me to be eleven feet tall, I would have a perceptual belief that is false; however, an explanation that would mitigate the falsity of my belief can be provided as follows: we could also imagine that what is going on is not merely the proximity of my false belief to a nearby true belief, but rather that some local accident prevents me from holding a true basis belief that is nevertheless exculpatory on counterfactual or modal grounds.

Under the second plausible explanation, if we were to consider the counterfactual that had I believed truly rather than falsely, I *would have* believed truly in most nearby possible worlds: given the relevant and specific environmental conditions in play, my perceptual belief *would have* been something more like ‘Anna is five-foot tall’. In the case of Ames room, had Anna stood anywhere else but in the corner, I would instead have a perceptual belief that is true or more truth-like, thus preserving the intuition that I still know even if my basis belief were false. We can call the second diagnosis *modal proximity*.

In this first and second diagnoses, the issue is that there exists a number

of edge cases near the boundaries of acceptability: this explanation, naturally, comes in a matter of degrees, and shares some similarities with more formalised approaches in truth-likeness; however, it is relevant to more 'home-grown' truths such as beliefs about whether one is late for a meeting based on slightly mistimed watches (Warfield 2005, p. 408), assessing that a child has a fever based on a slightly inaccurate thermometer (Hilpinen 1988, pp. 163-4), inductive inferences based on a false belief about the exact number of confirming instances (Saunders and Champawat 1964), epistemic pranksters (Luzzi 0088, p. 2) or inaccurate perceptual beliefs about the shape and size of nearby objects. Consequently, while counter-closure is abandoned, inferential knowledge is not opened up to accepting all cases of inferential knowledge based on false basis beliefs—some specific contexts preserve the relevant 'good-making' features of false basis beliefs.

The third diagnosis runs differently than the first two: counter-closure is still accepted; however, a number of relevant true basis beliefs play a key part in the inference (cf. Coffman 2008, pp. 190-1): this targets the '*sole* basis belief' condition included in the counter-closure thesis by indicating that a toy example isn't a *genuine* violation of counter-closure. By analogy, a bad lead actor may ruin a play, but if the actor is relegated to a secondary or background role and more competent actors take their place, the audience will leave the play having been satisfied, never knowing how the background extra lacked any acting ability whatsoever. We can call the third diagnosis *belief-swamping*.

2 A historical case-study: Dr Farr's miasmatic theory

There exist cases that don't obviously satisfy these three diagnoses of truth proximity, modal proximity and belief-swamping. This fourth diagnosis focuses on a different type of problem of apparent inferential knowledge based on false basis beliefs: beliefs in the existence of regularities, nonexistent entities and causal explanations that are, within a fairly broad domain, reliable in their predictive accuracy, yet based on faulty causal explanations and prognosticatory models. These cases naturally occur in the history of the natural sciences, where such inferences are not uncommon.

This problem can be put as follows: prognosticators in the natural sciences

often make reliable predictions based solely on (in retrospect) mostly or entirely false theoretical systems or models, yet consulting these false theoretical systems when devising concrete and specific plans for action were as reliable in their prognosticatory success as (mostly or approximately) true theoretical systems.

I present a historical case-study of the consultation of Dr William Farr's model of miasmatic theory as satisfying these conditions of repeatedly demonstrated reliability in its predictions in a number of fields. Detailing the extent of the reliability of Farr's model shows that it is at least *prima facie* plausible to ascribe inferential knowledge to epistemic agents like Dr Farr that adopt a reliable (but false) system of basis beliefs, even if the core parts of their theoretical system are not proximate to the truth (failure to satisfy truth proximity), there are no nearby possible worlds in which the relevant parts of the theoretical system are true (failure to satisfy modal proximity), and no relevant truths are available (failure to satisfy belief-swamping).

2.1 Miasmatic theory. What was it? And was it successful?

Miasmatic theory posited that cholera was caused by a nonliving organic poison that was dispersed in the air, arising from decaying organic matter or 'miasmata', a mist or vapour that was exuded from decaying material. The pre-theoretical reasoning for the miasmatic theory was intuitive: areas that were purportedly miasmatic had a foul smell, and the smell was either thought to be the poison or an indicator of the presence of poison.

Dr William Farr's version of miasmatic theory was one of the most mature versions of miasmatic theory, involving a number of auxiliary hypotheses that surrounded a central core thesis that the primary vector of transmission of infectious diseases such as cholera was through the air.

One of Farr's major additions to miasmatic theory was the addition of a detailed explanation for the catalysis, structure and development of contagious disease within the body. Farr borrowed heavily from the work of Justus von Liebig's early explanatory model of chemical interaction between living and non-living molecules (Pelling 2002, p. 27), which had previous success in explaining and predicting certain agricultural practices in Europe (Tulodziecki 2017, p. 3). The

causal mechanism Farr first proposed for infection of cholera was as follows: Farr reasoned infection occurred when a nonliving organic substance was introduced into the blood in the human body 'with the lungs as the point of entry to the animal system' (Eyler 1973, p. 84). Upon entry, this organic substance acted as a poison, multiplying in healthy blood and developing the symptoms of disease (Eyler 1973, p. 82). Farr called this process 'zymosis', which Farr believed resembled fermentation (Farr 1855, p. 48). This zymotic theory could 'explain the process of increase of morbid matter, either in the body or outside it' (Pelling 2002, p. 27).

As Ackerknecht 2009 and Eyler 1973 note, this molecular theory of zymosis could explain a number of phenomena that earlier theories that attempted to describe the structure of disease with the body could not. Consequently, Liebig's theory produced a number of novel molecular and biological predictions that stood for some decades as a highly predictive causal model of the growth of disease within the body.

Furthermore, Farr presented an explanation for disease-transmission with immense predictive content: the zymotic material, being heavier than air, would hang over cities like a fog, and while suspended in the air, would gradually disperse over an area after it had exited a body through its lungs or object left to putrefy. The degree and magnitude of dispersal was contingent on a number of environmental factors, such as '[t]emperature, humidity, wind, precipitation, and barometric pressure', which determined 'an epidemic's behavior' (Eyler 1973, p. 84).

In addition to the zymotic theory of disease and Farr's more predictive version of miasmatic theory, Farr further posited that there was a 'natural law' that the mortality of cholera was inversely correlated with elevation: 'The elevation of the soil in London has a more constant relation with mortality from cholera than any other known element' (Farr 1852); 'the poisonous consequences of filth must be inverse to the elevation of soil' (Farr 1855, p. 49); the conditions 'which are so constantly found in alluvial soils, lying on a level with or below the tidal waters' were prime candidate sources for producing miasmata (Farr 1852, p. 163).

Consequently, Farr's three interrelated theories predicted that concentrations of miasmata would be closer to rotting and putrid sources, such as London's long-neglected open sewer system and cesspits, and lower as one moved away from

these sources of contagion. Since mortality was thought to be directly linked to exposure to miasmata, Farr's miasmatic theory predicted that more people would fall ill and die closer to open sources of miasmata, and fewer at a farther distance.

On the basis of these three theories—zymotic theory, miasmatic theory and Farr's law of elevation—Farr extensively corroborated his findings through (for the time) rigorous statistical analysis. After collecting a large data set of the frequency of infection and morbidity, Farr extensively corroborated these predictions in the report by examining the available evidence on mean temperature correlated to number of reported cases, altitude and location of cases of cholera and the reported degree of air pollution in London's different districts (Langmuir 1961, p. 173).

2.2 Further novel predictions of Farr's disease theory

The predictive accuracy of Farr's theoretical system is impressive even today. For example, Farr's theory and subsequent statistical analyses was not limited to cholera epidemics within London, but spanned the entirety of England. He collected data for cholera epidemics outside London and corroborated the novel predictions of his version of the miasmatic theory. As Farr predicted, '[t]he cholera was three times more fatal on the coast than on the interior of the country' and was most fatal in areas 'lying lowest down the river', where the soil was thought to be most miasmatic (Farr 1852, p. 156).

Furthermore, Farr did not limit himself to explaining and predicting the location of outbreaks of cholera in England, but also the duration and course of a number of other various epidemics and diseases outside England:

"The zymotic theory could explain all of these phenomena through its claims that decomposing material produced miasmas...this was the reason why certain diseases were particularly bad during periods of high temperature and in certain geographical regions (for example, the many fevers in Africa), why urban centres were much more affected than rural areas, and why even specific locations in otherwise more or less healthy areas could be struck' (Tulodziecki 2017, p. 5).

The predictive power of Farr's theory extended to number of precise predictions that focused on an individual's response to infection. The predictive accuracy of Farr's zymotic theory of bodily disease extended to the ability 'for the first time—to figure out the effectiveness of different treatments' of disease (Tulodziecki 2016, p. 112). For example, Farr predicted how long an individual would likely suffer from smallpox and the odds of recovery or death given their age and environmental factors (Tulodziecki 2016, p. 110). As Tulodziecki further notes,

all of these were predictions that were impossible before Farr' and extended to, based on his theory of miasmata, producing mathematical models 'true both for the nation as a whole and small regions and districts within (Tulodziecki 2016, p. 112; cf. Eyler 1979, p. 11).

In fact, Farr made a number of startling predictions in a number of diverse areas. This included the course of the rinderpest outbreak of 1865. Rinderpest was a viral disease of cattle with a high infection and mortality rate that spread through England over the course of two years. Hundreds of thousands of cattle were infected with the disease and soon began to die. A member of Parliament, Robert Lowe, warned of 'an epizootic of tremendous size' (as quoted in Brownlee 1915, p. 250) and suggested preparations 'for a calamity beyond all calculation' (251). Farr replied that his 'law of epidemics' implied the reverse of what Lowe, the *British Medical Journal* and other established medical experts anticipated (250-1), specifically that there would be a quick surge in the infection and mortality rates due to rinderpest, which would suddenly decline.

Farr's prediction was correct: the epidemic 'reached its peak a fortnight later than [Farr] had predicted' (Wilkinson 1992, p. 142). Brownlee, using Farr's available data set, calls Farr's results 'a prophecy which approximates which remarkable closeness to the actual facts' (Wilkinson 1992, p. 251). Due to the repeated accuracy of his predictions, the early twentieth century public health expert Arthur Newsholme said Farr 'must be ranked with William Harvey in Physiology or with Lavoisier in Chemistry' (Newsholme 1926, p. 203).

In summation, 'Farr suggested an intimate connection between miasmata and zymotic material; both were, after all, nonliving organic particles capable of suspension in the air. ...for all practical purposes squalor might be said to cause

disease' (Eyler 1973, p. 85). It explained 'why certain localities were particularly prone to (certain kinds of) diseases, while others were spared, even if they were sometimes close by' (Tulodziecki 2016, p. 107).

Furthermore, Farr's theory explained in great detail why diseases like cholera were seasonal, why certain regions were affected by outbreaks of cholera greater than others, and why certain locations, specifically crowded urban areas, had a higher mortality rate than others. The theory also explained how epidemics would propagate, how past attempts at quarantine had failed and why there were different versions of cholera and other epidemic diseases (Eyler 1973). The explanatory power of the theory was such that it was predictively successful in a number of areas: '...it would be difficult to improve on the 1849 prediction of the cholera rates that were based on [miasmatic] theory' (Jekel 1996, p. 68).

2.3 Miasmatic theory provided concrete solutions to the cholera epidemic

After Farr and Chadwick's report of the cholera epidemic was released, Farr communicated with Florence Nightingale and Chadwick on a specific plan to prevent the spread of cholera in London by ending the formation of heavy mists or clouds of miasmata: this involved increased hygiene to remove miasmata clinging to the body, increasing proper ventilation in hospitals to prevent the spread of miasmata, regulating the burial and disposal of the dead, widening city streets, and prohibiting dense living arrangements in slums. Farr's control measures extended to enforcing regulations concerning the assembly of 'large masses of men in pilgrimages' and 'strict sanitary regulation' of boats engaged in international trade (Wilkinson 1992, p. 142). Most importantly, the miasmatic theorists developed a closed sewer system that disposed faecal matter away from residential areas and potable water (Baly 2010; Eyler 1973, p. 85).

What is of interest is not merely the novelty of predictions derived from Farr's theory and other mature miasmatic theories to combat cholera, but the *success* of all these plans in combating the spread of cholera.

Between 1831 and 1866, approximately forty thousand people died from cholera in London. After the completion of the London sewer system, there were no

more cholera epidemics in London.

These reforms that were instigated by miasmatic theorists cannot be understated: as the editors of the *British Medical Journal* concluded after a vote of medical experts, members of the general public and academic researchers, ‘the sanitary revolution’ directly inspired by miasmatic theory was rated the greatest medical innovation since 1840 (Ferriman 2007).

3 The failure of Farr’s theoretical system

In retrospect, however, *all the key members of Farr’s theoretical system were false*: Farr, Nightingale and Chadwick’s work countering cholera and other infectious diseases ‘succeeded despite [their] defective theory of disease transmission’, not because of it (Mackenbach 2007, emphasis added). Later developments in statistical analysis, specifically developments in logistic regression, show that Farr’s statistical analysis did not corroborate either Farr’s ‘natural law’ of elevation or miasmatic theory (Bingham, Verlander, and Cheal 2004, p. 393). Furthermore, these developments in statistical analysis show that Farr’s available data instead corroborated the rival germ theory of disease transmission, indicating that the relevant vector was water, not the air.

Furthermore, while Farr’s law of elevation worked exceedingly well in predicting the course of a number of cholera epidemics in England between the years of 1820 and 1860, *later* epidemics did not conform to his law. Farr’s law of elevation, ‘despite being responsible for some of Farr’s most important novel predictions’ (Tulodziecki 2016, p. 112), was only reliable within a limited range. Lastly, Farr’s zymotic theory was eventually superseded by the germ theory of disease—germs are living rather than nonliving, and their growth bears little resemblance to fermentation within the body.

Thus we can examine a number of potential candidate cases of Dr Farr inferentially knowing a large number of propositions based on a large body of false basis beliefs. Consider the following set of basis beliefs:

P1: Cholera, as well as a number of other infectious diseases, such as smallpox, rinderpest, measles, scarlatina, and typhus are caused

by the inhalation of miasmata primarily transmitted through the air. The direct cause of these diseases is due to organic matter given off putrefying material, i.e. 'miasmata'.

P2: The spread of diseases in the body are a type of or analogous to decomposition. Zymotic material cause the promulgation of disease within the human body, transmitted from the lungs to the bloodstream, and the zymotic material engages in a process similar or analogous to fermentation or decomposition.

P3: The law of elevation, as well as a number of environmental causes such as barometric pressure, wind direction and humidity, predict the location of cholera outbreaks, as well as outbreaks of a number of infectious diseases.

P4: The available statistics on morbidity by infectious diseases corroborate *P1 – P3*. Furthermore, rival theories, such as Snow's germ theory of disease, could be discarded in favour of Farr's theory on the basis of this evidence (Farr 1855).

And consider the following set of true beliefs that were derived from *P1 – P4*:

Q1: '[cholera has] infinite preference for localities that are foetid with organic impurity' (Farr 1855, p. 47).

Q2: Localities closer to water during the periods of a cholera outbreak in England during the decades between 1820 and 1860 would have a greater incidence and morbidity rate than localities farther away from water and at higher elevations.

Q3: The construction of a closed sewer system in London and other large cities that dispose waste far from cities will decrease the frequency of infection by cholera and other infectious diseases.

Q4: The 'law of increase and decline' and law of density will model the overall increase and decline and relative frequency of highly contagious and deadly diseases.

Q5: Cholera and other infectious diseases can be transmitted through fomites, necessitating the removal and destruction of objects that may have fomites found in the living and sleeping quarters of people carrying cholera and other highly infectious diseases.

It is not in dispute that Farr genuinely believed both $P1 - P4$ and $Q1 - Q5$. Furthermore, it may be the case that Farr *knew* $Q1 - Q5$. However, it is difficult to ascribe to Farr any relevant true basis beliefs that entailed $Q1 - Q5$, for Farr rejected the germ theory of disease transmission until 1866, long after his miasmatic theory had made these successful predictions.

Furthermore, $P1 - P4$ are simply not approximate to the truth. A counterfactual or modal account in which Farr would have had true basis beliefs is even more implausible. Neither truth proximity or modal proximity are applicable. The miasmatic theory was not merely partially wrong or approximate to the truth, but almost in its entirety *false*, and false on numerous grounds, such as the causes of cholera, how cholera was transmitted, and how cholera acted in the body. There is no nearby possible world in which cholera is caused by miasma, for miasma does not exist.

Two questions remain: (1) did Farr *believe* $Q1 - Q5$ solely based on $P1 - P4$? (2) If 1, did Farr *know* $Q1 - Q5$?

There are at least two different ways to cash out belief-swamping: the first approach locates the appropriate basis beliefs *outside* the theoretical system that Farr purportedly believed. The second approach locates the appropriate true basis beliefs in the *predictive accuracy* of miasmatic theory. Both positions would deny (1). After addressing these two approaches, I address (2).

The first approach would diagnose the problem as follows: rather than believing solely that miasmata existed, it would be appropriate to ascribe to Farr a number of basis beliefs that produced an inductive inference wholly separate from miasmatic theory. For example, Farr may have believed $P1^*$: 'There is cholera at localities $T^1 \dots T^n$ and these locations are foetid with organic impurity'. Farr consulted the available statistical evidence, believed $P1^*$, and consequently arrived at $Q1$ without depending solely on $P1 - P4$. Since Farr knew $P1^*$, Farr inferentially knew $Q1$, but not based on $P1 - P4$. A similar explanation is avail-

able for $Q2 - Q6$: in each case, Farr relied on an inductive inference Pn^* that entailed Qn .

This approach, however, falls short for two reasons: Farr's statistical analyses

... were not simply instances of straightforward observations; if they had been, people would have discovered them long before Farr. Rather, a lot of theoretical assumptions went into how to construct even the raw data (such as assumptions about what kind of mortality rate ought to be used), and without Farr's appropriate relating of different data sets and his ensuing interpretation, there would have been just a bunch of numbers' (Tulodziecki 2016, p. 111).

The question is whether Farr *did in fact* believe P or P^* . A rational reconstruction of Farr's reasoning process is certainly difficult, but not impossible: Farr approached his statistical analyses of the accumulated data in the 1820s and 1840s having previously accepted miasmatic theory in his published writings, and interpreted his data in light of miasmatic theory; Farr could not have derived the inductive inference that $Q1$, much less arrived at the inductive inference $P1^*$, without believing $P1 - P4$.

This rational reconstruction, however, can be countered by pointing out that while $P1$ may have been indispensable in the formation of $Q1$, that does not rule out the possibility of $P1^*$, although not indispensable, playing some other role. However, appealing to $P1^*$ and not $P1$ proves too much: if it is appropriate to ascribe to Farr $P1^*$ that Farr may have possibly believed, it is also appropriate to ascribe to any epistemic agent that apparently relied on a false basis belief a large number of relevant true basis beliefs the epistemic agent may have possibly believed, but are equally dubious. This approach would open the epistemic floodgates: any 'just so' story can be constructed for cases in which an epistemic agent has false basis beliefs but may have believed truly.

What of the second approach? Assume that in addition to miasmatic theory, Farr also relied on the predictive accuracy of miasmatic theory. Other basis beliefs about the reliability of a model did the epistemic 'heavy lifting' behind the scenes, such as P' : ' $P1 - P4$ is an empirically adequate and predictively successful model of disease-transmission'.

This approach, however, has similar defects: it doesn't reflect Farr's doxastic commitment to $P1 - P4$. It is trivial that for any belief X an epistemic agent S believes is true and provides some prediction Y , S also believes X will be predictively successful in predicting Y . In other words, the statement 'is predictively successful' may be appended to any false basis belief X that is predictively successful in order to produce a true basis belief X' : ' X is predictively successful'. However, what is the basis belief for S to believe X' ? In Farr's case, the basis belief cannot be an inductive inference. Nor are there any true basis beliefs that Farr may have believed. The most plausible explanation for why Farr believed X' is that Farr believed X' on the basis that Farr believed X . Through disquotation, in these similar cases that preclude inductive inferences, if S believes X , S believes X is true. And yet X is stipulated to be false. The epistemic floodgates open once more.

One could conceivably deny that Farr knew $Q1 - Q5$, just as one could conceivably deny any cases that conflict with the counter-closure thesis. But why maintain the counter-closure thesis when faced with so many edge cases that cast doubt on it? Furthermore, the case of Dr Farr may have analogous cases today in medicine: should we conclude that today's medical practitioners, if they were operating with a large set of similarly false basis beliefs, do not know that a drug will be effective once introduced to the population at large? And lastly, no obvious or debilitating consequences for a philosophical programme hinges on rejecting counter-closure.

4 A brief summary

The example of miasmatic theory illustrates a neglected feature of the puzzle surrounding counter-closure: it is *prima facie* plausible that throughout history of science many scientists did indeed have inferential knowledge based solely on theoretical systems in which the most relevant parts of predictive models were not approximately true. The example of zymotic theory violates truth proximity, modal proximity and belief-swamping. This problem for epistemic counter-closure, however, does not lead to epistemic anarchy, since there exists a mitigating factor, namely the reliability of methods that employed the false theoretical

system.

References

- Ackerknecht, E.H. (2009). "Anticontagionism between 1821 and 1867. The Fielding H. Garrison lecture". In: *International Journal of Epidemiology* 38.1, pp. 7–21.
- Armstrong, D. (1973). *Belief, Truth, and Knowledge*. Cambridge: Cambridge University Press.
- Audi, R. (2003). *Epistemology: A Contemporary Introduction to the Theory of Knowledge*. 3rd edition. New York: Routledge.
- Baly, M.E. (2010). "Florence nightingale and the development of public health nursing". In: *Humane Medical Care* 10.
- Bingham, P., N.Q. Verlander, and M.J. Cheal (2004). "John Snow, William Farr and the 1849 outbreak of cholera that affected London: a reworking of the data highlights the importance of the water supply". In: *Public Health* 118, pp. 387–394.
- Brownlee, J. (1915). "Historical note on Farr's theory of the epidemic". In: *British Medical Journal*, ii, pp. 250–252.
- Coffman, E. (2008). "Warrant without truth?" In: *Synthese* 162.2, pp. 173–194.
- Eyler, J.M. (1973). "William Farr on the Cholera: The Sanitarians Disease Theory and the Statistician's Method". In: *Journal of the History of Medicine*.
- (1979). *Victorian Social Medicine—the Ideals and Methods of William Farr*. Baltimore, MD: Johns Hopkins University press.
- Farr, W. (1852). "Influence of elevation on the fatality of cholera". In: *Journal of the Statistical Society of London* 15.2, pp. 155–83.
- (1855). *Report of the Committee for Scientific Inquiry In Relation To The Cholera-Epidemic of 1854*. Eyre and Spottiswoode.
- Ferriman, A. (2007). "BMI choose the sanitary revolution as greatest medical advance since 1840". In: *British Medical Journal* 334, p. 111.
- Hilpinen, R. (1988). "Knowledge and conditionals". In: *Philosophical Perspectives* 2 (*Epistemology*).
- Jekel, J.F. (1996). *Epidemiology, Biostatistics, and Preventative Medicine*. Elsevier Health Sciences.

- Langmuir, A.D. (1961). "Epidemiology of airborne infection". In: *Bacteriological Review* 25, p. 174.
- Luzzi, F. (2012). "Interest-relative invariantism and knowledge from ignorance". In: *Pacific Philosophical Quarterly* 93, pp. 31–42.
- (88). "Counter-closure". In: *Australasian Journal of Philosophy* 4.673–683.
- Mackenbach, J.P. (2007). "Sanitation: pragmatism works". In: *British Medical Journal* 334.s17.
- Newsholme, A. (1926). "William Farr, father of English vital statistics". In: *De Lamar Lectures 1925-1926 of the school of Hygiene and Public Health. Baltimore, 1927*. Ed. by W.H. Howell. Williams Wilkins Co.
- Nozick, R. (1981). *Philosophical Explanations*. Oxford: Oxford University Press.
- Pelling, M. (2002). "The meaning of contagion: Reproduction, medicine and metaphor". In: *Contagion: Historical and cultural studies*. Ed. by A. Bashford and C. Hooker. Routledge.
- Saunders, J. and N. Champawat (1964). "Mr. Clarck's definition of 'knowledge'". In: *Analysis* 25.1.
- Stanley, J. (2005). *Knowledge and Practical Interests*. Oxford: Oxford University Press.
- Tulodziecki, D. (2016). "Structural realism beyond physics". In: *Studies in History and Philosophy of Science* 59, pp. 106–114.
- (2017). "Against selective realism(s)". In: *Philosophy of Science* 84.5.
- Warfield, T. (2005). "Knowledge from falsehood". In: *Philosophical Perspectives* 19, pp. 405–416.
- Wilkinson, L. (1992). *Animals and Disease: An Introduction to the History of Comparative Medicine*. Cambridge: University of Cambridge.