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Structural realism beyond physics☆

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ABSTRACT

The main purpose of this paper is to test structural realism against (one example from) the historical record. I begin by laying out an existing challenge to structural realism – that of providing an example of a theory exhibiting successful structures that were abandoned – and show that this challenge can be met by the miasma theory of disease. However, rather than concluding that this is an outright counterexample to structural realism, I use this case to show why it is that structural realism, in its current form, has trouble dealing with theories outside physics. I end by making some concrete suggestions for structural realists to pursue if, indeed, they are serious about extending structural realism to other domains.

1. Introduction

Virtually all discussions of structural realism, no matter whether of the epistemic or ontic kind, have focused on examples from physics.[1] My goal in this paper is to test structural realism against a theory outside physics, the so-called miasma theory of disease. I will examine this case in some detail, determining whether the miasma theory's successor – the germ theory of disease – can be viewed as having retained any of the structural elements of the miasma theory, as ought to be the case if structural realism is true.

Structural realists take themselves to be generally less vulnerable to anti-realist arguments than standard realists, because they take themselves to be immune to some of the most prominent arguments in the realism-debate – those from the history of science. In particular, structural realists think they are immune to historical arguments showing that a theory's theoretical terms need not refer in order for that theory to enjoy novel predictive success: according to structural realists, it is not entities but only structural elements that are retained, and continuity of reference is not necessary for retention at the structural level. Because of this, structural realists think they can avoid counterexamples to standard realism (such as those on Laudan's (1981) famous list) that purport to show that there are mature and genuinely successful past theories that made novel predictions, yet, turned out to fail to exhibit any kind of referential or ontological continuity. Instead, what structural realists are committed to is there being no cases of genuinely successful past theories whose structural elements are abandoned. Thus, structural realists hold that the historical examples against realism don't affect structural realism, since, even if there is failure of continuity among theories and their successors, this failure is not of the right kind: what is required in order for a case to be a genuine threat to structural realism is an example of a theory that (i) was genuinely successful and made novel predictions, (ii) was abandoned as false, and (iii) whose structural elements, in particular those tied to the theory's success, were not retained by later theories. It is only failure of such structural continuity, structural realists contend, that would pose a real problem for structural realists. This, for instance, is the position held by French and Ladyman (2011), who believe there are no examples of such theories, but explicitly acknowledge that providing a case meeting the above criteria would constitute a counterexample to structural realism (32). After all, if structural realism is true, there ought to be structural retention in the progression from successful yet abandoned theories to their successors. It is exactly this prediction of structural realism – that there is retention at the structural level among successive theories – that I will test in this paper.

Since I am interested in this prediction in general and don't want to pre-judge the case against any particular version of structural realism, I will, for the purposes of this paper, take structural realism

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[1] Two notable exceptions are French (2011), who discusses structural realism in the context of biology, and Kincaid (2008), who is concerned with the social sciences. However, neither French nor Kincaid engage in detailed case studies, and so it remains difficult to see what concrete structuralist accounts in these domains would look like. For further structuralist discussions of the social sciences, see also Ladyman & Ross (2007) and Ross (2008).

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in the most general sense only, understanding by it the minimal position according to which the most we are justified in being realists about, at least as far as unobservables are concerned, is the structural content of our scientific theories.[2] I take this assumption to be unproblematic, since both French & Ladyman's challenge and also my own arguments apply equally to epistemic and ontic versions of structural realism (and their varieties). Similarly, I want to remain neutral about the notion of structure, since nothing in my argument depends on what understanding of 'structure' one subscribes to, be it set-theoretic, category-theoretic, or group-theoretic.[3]

After explaining the salient details of the miasma theory (Section 2), I will show that it made a number of novel predictions, based on structural elements that were abandoned in its successor (Section 3). I then explain how the miasma theory violates the predictions of structural realism, despite its initially seeming a good candidate for it (Section 4). However, rather than concluding outright that structural realism fails the historical test, I go on to identify a number of problems that make clear why it is that structural realists have such trouble dealing with cases outside physics. Based on this analysis, I then argue that structural realists have some work to do before structural realism can be regarded as properly testable (Section 5). I end by making some concrete suggestions for structural realists to pursue if, indeed, they are serious about extending structural realism to domains besides physics and about making it properly testable in those domains (Section 6).

2. The miasma theory of disease

The miasma theory of disease is best described as a cluster of related views, all of which shared assumptions about the nature of so-called 'miasma'.[4] According to this cluster, diseases were brought about and passed on through decomposing organic material that would disperse into the air as noxious and disease-causing odours, the miasmas. This noxious air in turn would affect potential victims, causing a variety of diseases of differing strengths. The type of disease, as well as its severity were thought to depend on the complex interplay between a number of factors, some related to the miasmas themselves (such as climate and weather, which were thought to affect miasmatic natures), some related to the potential sufferers of diseases (such as factors relating to the sturdiness of their constitutions or their values, which were thought to affect their susceptibility to various diseases), and some related to the local circumstances in which miasmas existed (such as overcrowding or bad ventilation, which were thought to compound whatever problems were already present).

While some version or other of the miasma theory had been around since the 1600s, its mid-19th century version was no longer the vague and general theory of, for example, people like Sydenham. Whereas Sydenham and his contemporaries talked about the so-called 'epidemic constitution' — literally a particular sort of atmosphere that would waft around and cause diseases — this naive view was supplanted by the miasma theory's later incarnations: by the mid-1800s, people were embracing highly complex and often specific accounts of how various materials and conditions gave rise to miasmas. In this vein, for example, it was debated what sorts of materials were particularly good for producing miasmas and there were also detailed theories about the role of caloric in putrefaction (cf., for example, Aiton 1832). In addition, people such as Farr (cf. Section 3) were drawing in quite some detail on Liebig's chemical explanations, viewing disease processes as analogous to fermentation in various ways.

Farr, for example, posited what he called 'zymotic material' and thought that this played a crucial link in the causal chain of diseases. Different zymotic materials would cause different diseases, and, through interacting with miasma from decomposition, become airborne. While zymotic material could affect different regions by travelling through air, miasmas were local, thus explaining why certain localities were particularly prone to (certain kinds of) diseases, while others were spared, even if they were sometimes close by. This account was also a way of combining disease specificity with atmospheric conditions: factors such as temperature, barometric pressure, and others were all thought to influence the interaction of zymotic materials with miasmas. When miasma, which was itself decomposing, was too highly concentrated in the atmosphere, zymotic material would become more virulent; these conditions would weaken people's constitution and, thus, make them more susceptible to falling ill in the first place. Lastly, the role of individual predispositions was retained in this account: since diseases were still thought to act primarily on the blood, people's blood would determine how they reacted to the zymotic materials.[5]

As I already mentioned, people drew on Liebig's theories in particular, not just because they already enjoyed a high degree of success (in agriculture, for example), but also because they explained the interaction between living and non-living things on a molecular basis (Pelling, 2002: 27). Such accounts were highly suited to explaining diseases, because they could explain, at least in principle, the interaction between human bodies and the environment in its various guises (ibid.). Indeed, Farr's zymotic theory was rooted in Liebig, who wrote widely on putrefaction, central to the miasma theory, and on the process of catalysis, which "could also explain the process of increase of morbid matter, either in the body or outside it" (ibid.).[6] Lastly, it is worth mentioning that not only was it the case that Farr cited Liebig, but also that Liebig had an interest in diseases, writing about miasmas and zymotic matter frequently and in much detail (see Liebig 1842, 1843; and Tulodziecki 2016).

3. The success of the miasma theory

The version of the miasma theory just outlined was highly successful. Besides providing some explanation of disease processes in the body, it could also explain a number of phenomena that the contagionism that was so popular earlier in century had trouble with.[7] For example, it could account for the seasonality of those diseases that were seasonal, for the fact that certain geographical regions were consistently affected much worse than others, why certain diseases were tied to particular regions, why certain locations suffered from higher mortality than others, including prisons, workhouses, and poor, crowded, urban areas. It could also explain how epidemic diseases could move around, even when no route of infection could be traced, why cholera-quarantines had failed, why there were such great differences in

[2] For some of the different positions, see Worrall (1989), Ladyman (1998), Chakravartty (1998), French and Ladyman (2011), Frigg and Votsis (2011), and Ladyman (2014).

[3] For some discussions of the different senses of structure, see Brading and Landry (2006), French (2011), and Ladyman (2014).

[4] For a history of the relationship between these views, see, for example, Baldwin (1999), Eyler (2001), Hamlin (2009), Pelling (1978), and Worboys (2000). Since I cannot do justice to the historical complexities of the view(s) here, I will restrict myself to focusing on those shared claims that matter for my purposes.

[5] I rely heavily on Eyler (1973) here.

[6] See also Worboys (2000: 34), Brock (1998), and Pelling (1978).

[7] In fact, the miasma theory might be said to have arisen partly as a response to those problems; see Ackerknecht (2009) and Eyler (1973).

the mortality of epidemic diseases at different times, and why there were endemic versions of many diseases, in addition to epidemic ones (cf. Eyler 1973).

Impressive as this degree of explanatory power may be, however, it is not good enough for many realists. Psillos, for example, stresses that what matters is "the generation of novel predictions which are in principle testable" (1999: 100), emphasising, in particular, use-novel predictions, i.e. predictions knowledge of which was not used in the construction of the theory in question (cf. Psillos, 1999: 105–7). What I will show in the next section is that the miasma theory was highly successful even according to this stricter standard: it made a number of successful use-novel predictions that were borne out.

3.1. Elevation

William Farr, often regarded as the founder of medical statistics, was not a physician, but widely viewed as an authority on infectious diseases.[8] Among the positions he held were Statistical Superintendent of the General Register Office and member of the Committee of Scientific Inquiries. He also published widely, especially on the different British cholera epidemics. We already saw that, according to the miasma theory, rotting organic matter would give rise to miasmas. In addition, it was thought that the conditions "which are so constantly found in alluvial soils, lying on a level with or below the tidal waters" were particularly good sources for producing miasma (1852a: 163). It also clearly followed from miasmatic views that concentrations of miasma ought to be higher closer to rotting sources and lower as one moved away. Since sickness and mortality were thought to be directly related to (concentrations of) miasma, it was also a consequence of the theory that more people would be sick and die closer to sources of miasma, and fewer as the distance from such sources increased.

Farr, through a number of results, confirmed these claims. He noted the striking fact that

> 46,592 of the 53,293 deaths from cholera in the year 1849 occurred in 134 of 623 districts; in less than a seventh part of the area of England and Wales, among four parts in ten of the population. Only 6,701 deaths took place out of 10 millions of people on 49,228 square miles of territory (1852a: 155).

Based on this, he showed that "[t]he cholera was three times more fatal on the coast than in the interior of the country" and that it was most fatal in regions "lying lowest down the river", where the soil was thought to be particularly conducive to producing miasmas (156). More generally, he found just what the miasma theory predicted: that wherever high concentrations of miasma were predicted, the mortality rate from cholera was high, and wherever the miasma theory predicted that the concentrations of miasma were lower, so was the mortality rate.[9] Farr thought that "[t]he amount of organic matter, then, in the atmosphere we breathe, and in the waters, will differ at different elevations", and that "[t]he emanations, mixing with the super-incumbent atmosphere, ascend like smoke; but at the same time become less and less dense by dilution and by the gradual destructive decomposition" (1852a: 163).

Out of this grew Farr's 'elevation law', based on which he made some of his most striking predictions. This law, well-known in Farr's time, related deaths from cholera to the elevation of the soil. Farr both predicted a relationship between these two variables and, also, managed to capture this relationship mathematically: the "mortality of cholera is inversely as the elevation of the people assailed above the sea-level" (161). Analysing the Registrar's table of cholera deaths in London, Farr found the following:

> [T]he mortality from cholera in London bore a certain constant relation to the elevation of the soil, as is evident when the districts are arranged by groups in the order of their altitude. We place the districts together which are not on an average 20 feet above the Thames, and find that on this bottom of the London basin the mortality was the at the average rate of 102 in 10,000: in the second group, at 20 and under 40 feet of elevation, or on the second terrace, the mortality from cholera was the rate of 65 in 10,000; in the third group, or on the third terrace 40–60 feet high, the mortality from cholera was at the rate of 34 in 10,000; in the fourth group, 60–80 feet high, the mortality from cholera was at the rate of 27 in 10,000; in the fifth group, 80–100 feet high, the mortality was at the rate of 22 in 10,000; in a district 100 feet high, the mortality was 17 in 10,000; in Hampstead, about 350 high, the mortality was 8, or deducting a stranger infected at Wandsworth, but who died there, 7 in 10,000. (1852b: lxii).

Further, Farr points out that

> [b]y ascending from the bottom to the third terrace, the mortality is reduced from 102 to 34; by ascending to the sixth terrace it is reduced to 17 (x) [included as Fig. 1]. It will be observed, that the number representing the mortality on the third terrace is one-third of the number 102, representing the mortality on the first, and that the mortality on the sixth terrace is one-sixth part of the mortality on the first. And a series approximating nearly to the numbers representing the mortality from cholera, is obtained by dividing 102 successively by 2, 3, 4, 5, 6. (Farr, 1852b: lxii–iii).[10]

The table (Fig. 1) shows how good Farr's calculations were, but, nevertheless, Farr thought that "[t]he relation discovered between the elevation of the soil and the mortality from cholera is so important, that it was thought right, after the above calculations were made, to submit the principle to another test, by comparing the elevation and the mortality from cholera of *each sub-district*" (161). Farr found, as before, that this "entirely confirms the announced law" (lxvi) and his illustration of the results (Fig. 2) shows just how convincing his law must have seemed.[11] Lastly, Farr cites a letter from W.H. Duncan, the Health Officer of Liverpool, that also confirmed his findings, this time in an entirely different part of the country (Farr, 1852a, p. 183).

3.2. Smallpox, fever, and rinderpest

Farr also, with spectacular success, predicted the progression of various diseases, the mortality and recovery rates of diseases during different phases of an epidemic and for people of different ages. As before, Farr's data is extensive, and I can only provide some examples, but they suffice to convey a sense of the success of the

[8] For an intellectual biography of Farr, see Eyler (1979).

[9] Moreover, exceptions to this general rule all had an explanation that also followed from the miasma theory. For an example, see Farr (1852b: liv–vi).

[10] Farr's actual equation can be found in his (1852a: 160). A small number of anomalies is due to the fact that some numbers are "deranged by the deaths in the hospitals and workhouses" (Farr, 1852a: 161).

[11] Farr's tables for the sub-districts are on pp. clxvi-ix of his (1852b). Langmuir (1961) graphs these results and concludes that Farr's results "would be impressive to any scientist at any time" (173).

(x)

Elevation of Districts, in feet.	Number of Terrace from bottom.	Deaths from Cholera in 10000 Inhabitants.	Calculated Series (1.)
Feet.			
20—	1	102	$\frac{102}{1} = 102$
20—40	2	65	$\frac{102}{2} = 51$
40—60	3	34	$\frac{102}{3} = 34$
60—80	4	27	$\frac{102}{4} = 26$
80—100	5	22	$\frac{102}{5} = 20$
100—120	6	17	$\frac{102}{6} = 17$
340—360	18	7	$\frac{102}{18} = 6$

Fig. 1. From Farr, W. (1852b: lxii).

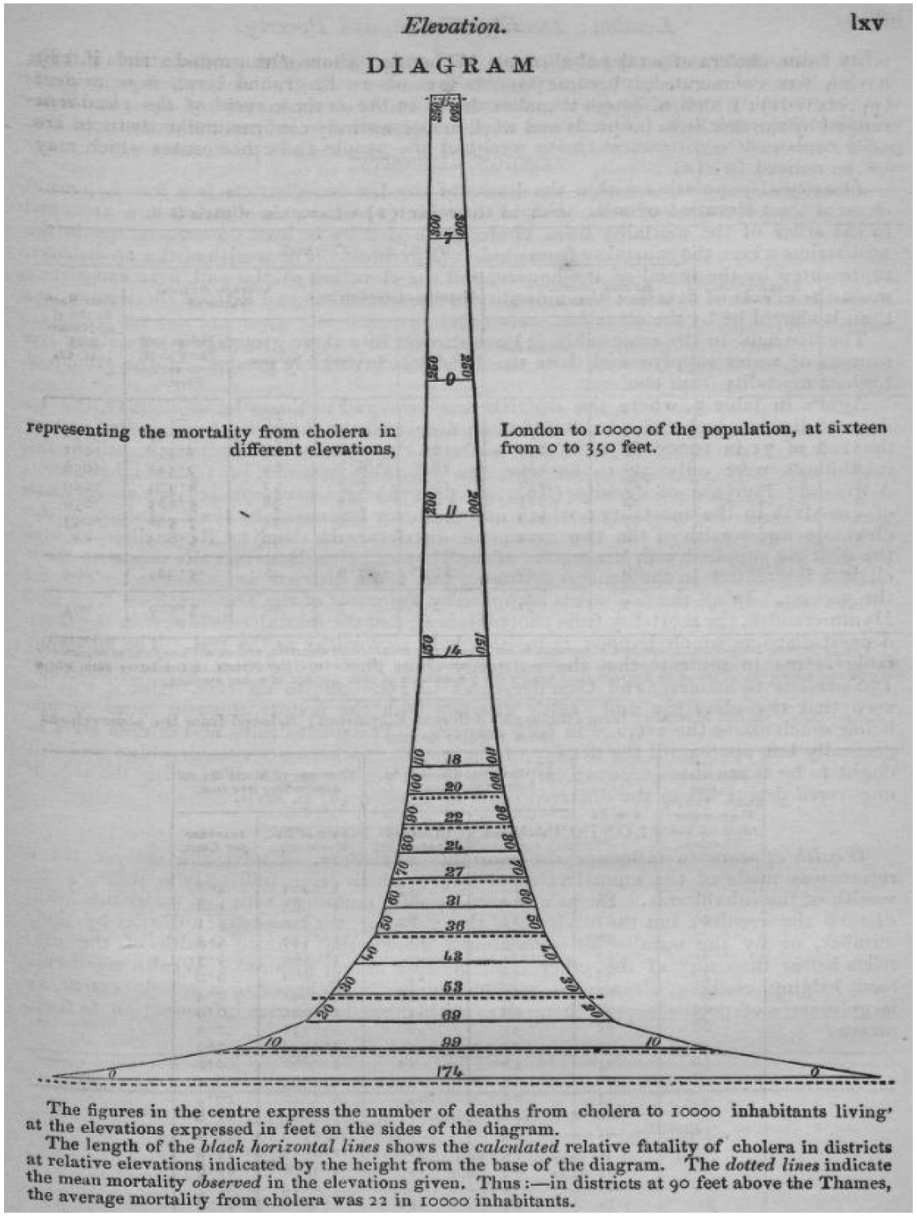
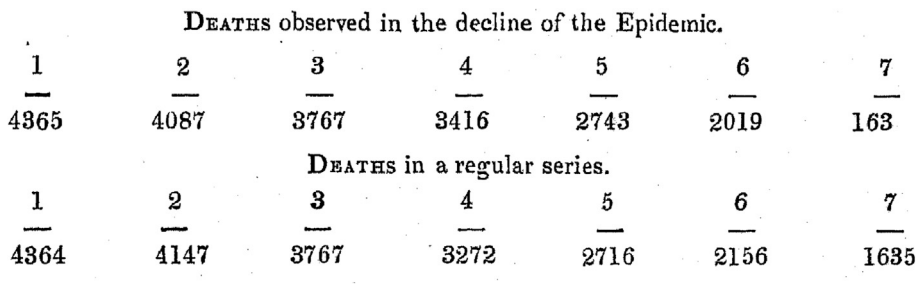


Fig. 2. From Farr, W. (1852b: lxv).



DEATHS observed in the decline of the Epidemic.

1	2	3	4	5	6	7
4365	4087	3767	3416	2743	2019	163

DEATHS in a regular series.

1	2	3	4	5	6	7
4364	4147	3767	3272	2716	2156	1635

Fig. 3. From Farr, W. (1840: 19).

enterprise. For example, Farr constructed a so-called sickness table, based on the records of 5000 smallpox patients 10–34 years of age, all of whom had received treatment at the London smallpox hospital (Eyler 1980: 8–9). In this table, he divided smallpox into 5-day periods, from day 5 to day 150, and showed the numbers of those who died, recovered, and stayed sick. Next, he calculated the patients' chances of each during the different stages of the disease. In doing so, Farr discovered two statistical laws: first, what he referred to as the law of mortality, second, the law of recovery (ibid.). These laws then allowed Farr to make a number of predictions, among them how long smallpox would tend to run in a victim, and, of course, the odds of recovery and death. This was a huge step forward for prognosis, since all of these were predictions that were impossible before Farr.[12] However, Farr did not just for the first time make the above predictions, he also made it possible – also for the first time – to figure out the effectiveness of different treatments. This, in turn, allowed him to understand and predict what would improve a patient's recovery odds and how different treatments would affect the course of a given disease. Similar calculations were made for the observed and calculated mortality rates of fever, which were found to increase by age (cf. also Edmonds 1835: 856).

A different type of prediction Farr attempted was concerned with the duration and course of epidemics. Here, what Farr showed was that the number of deaths from smallpox fell in such a way that it could be described by a geometric series, and that this was true both for the nation as a whole and smaller regions and districts within (Eyler 1980: 11). Fig. 3 shows the observed and calculated series of smallpox deaths and describes the way smallpox epidemics fall.[13] Moreover, Farr realised that, if it was possible to describe epidemics in this way, one could also predict their future behaviour.

That is exactly what Farr did with the rinderpest outbreak that hit Britain in 1865. Rinderpest was a highly communicable viral infectious disease with an extremely high mortality rate, affecting cattle.[14] When this struck Britain, there was worry it would wipe out the entire cattle population. Robert Lowe, an influential parliamentarian, warned of "an epizootic of tremendous size" (quoted in Brownlee, 1915: 250) and asked people to "prepare [themselves] for a calamity beyond all calculation" (Brownlee, 1915: 251). Lowe, and virtually everybody else, thought the epidemic would keep growing and growing, but Farr took a very different line. Replying that "[n]o one can express a proposition more clearly than Mr Lowe; but [that] the clearness of a proposition is no evidence of its truth", Farr went on to argue that his 'law of epidemics' implied the reverse of what Lowe and others anticipated (Brownlee, 1915: 251). Specifically, Farr predicted that there would be an early maximum to the epidemic, which would then decline, not rise; once again, he turned out to be right.[15] The early 20th century statistician Brownlee, who analysed Farr's data using more advanced tools, calls Farr's results "a prophecy which approximates with remarkable closeness to the actual facts" (Brownlee, 1915: 251). Farr's claims about rinderpest were surprising and unexpected, and were, just like his results about smallpox and elevation, clearly novel predictions, since none of the evidence involved in Farr's predictions was used in the construction of the miasma theory.

Results such as these give a sense of Farr's success, and it is worth stressing that they were not exceptions. Farr also discovered a relation between population density and mortality and morbidity rates: he noted that more populous towns experienced a higher mortality than rural regions and, once again, found a way to express this relationship in a mathematical way.[16] He also showed that different occupations, age, and a number of environmental factors affected disease patterns and again many of his predictions were borne out. In short: Farr predicted a number of laws and outcomes that were (i) clearly use-novel, and (ii) (rightly) regarded as impressive successes for the miasma theory. This is not an isolated judgement: the public health expert Arthur Newsholme (1857–1943), for example, said of Farr that he "must be ranked with William Harvey in Physiology or with Lavoisier in Chemistry" (1927: 203).[17]

Crucially, Farr's predictions were also closely tied to his disease theory (cf. Eyler 1973). It simply would not have made sense for people who did not subscribe to a miasmatic view to generate and analyse the kind of data that Farr was using (an enormous endeavour): there simply is no point in producing data on, say, various environmental influences and diseases unless one holds a view according to which they are connected. Farr himself is also explicit about the connection to his theory of disease. About the course of epidemics, for example, he states that "subsidence is a property of all zymotic diseases", and that the

> [d]iminished activity of the zymotic matter and augmented powers of resistance in the survivors are the factors to which the

[12] Farr also notes a positive by-product: "Great credit is often gained by predicting the death of a patient where the bystanders perceive no danger" (1838/2003: 219–220).

[13] For more details on smallpox and its associated series, see also Farr (1838/2003: 281–283).

[14] The World Organisation for Animal Health and the UN declared rinderpest eradicated in 2011.

[15] The *British Medical Journal* also rejected Farr's conclusion, remarking that "Dr. Farr will not find a single historical fact to back his conclusion that in nine or ten months the disease may quietly die out–may run through its natural curve" (quoted in Brownlee 1915: 250).

[16] The basic idea, of course, is that, as density increases, so do morbidity and mortality. Farr was initially unsuccessful in his endeavour to show this; for details, see Eyler (1980: 13–15).

[17] The comparison to Lavoisier is particularly interesting, since the case of Lavoisier is frequently discussed in the realism-literature as a definite case of mature and successful science.

subsidence of epidemics of smallpox, cholera, measles, scarlatina, and typhus are in themselves referable. The matter (cholerine) inducing epidemic cholera was apparently diffused all over England in 1849; and there were two or three deaths by cholera or diarrhoea in nearly every district; everybody more or less felt its power; but the mortality was only great in the low parts of the kingdom, and where the stuff entered the system through water as well as through air and other media (252).[18]

4. The miasma theory from a structuralist point of view

How exactly does this case bear on structural realism? First, it ought to be noted that the miasma theory is potentially a very good example for structural realists, since standard realist accounts, such as those of Kitcher (1993) and Psillos (1999) fail here. These accounts all require, in one way or another, that elements that fail to be retained in successor theories play only a non-crucial role in the original theory: they have to be dispensable to the theory's predictions. However, as we have seen, miasma, despite the fact that it was not retained (and, indeed, failed to exist), was crucially involved in all of the miasma theory's (use-novel) predictions, and so cannot be regarded as a merely idle component, the way Kitcher's account requires, or as non-essential, the way Psillos's does.[19] Thus, being able to give an account of the miasma case would not just be an obvious success for the structural realist view itself, but also, at least in this instance, put structural realists ahead of ordinary realists.

As we have already seen, in order for this case to support structural realism, it needs to fulfil what structural realism predicts: that there are structural elements of the miasma theory, crucially involved in its success, that were retained by its successors. The obvious candidates for such structures in the miasma case are the mathematical relations described by Farr, such as his claims about mortality rates, the progression of epidemics over time, claims about population density and age-structures, and, of course, the elevation law. All of these relations were indispensably involved in Farr's making novel predictions, and, thus, if structural realism is right, we ought to expect to see these relations retained in later views. Moreover, because all of these relations are mathematical, it is easy to determine whether and to what extent they were retained in the miasma theory's successor. It is worth noting here that Farr himself explicitly states that he takes himself to be discovering true relations in the world – (lawlike) facts about diseases – and that that is what he is capturing mathematically through his equations.[20]

How does structural realism fare? Farr did indeed capture relations that we still think are true. Take, for instance, Farr's law of epidemics, which describes the course of epidemics over time and that we saw at work with respect to smallpox and rinderpest: despite the fact that we have come a long way from the days of Farr and now have differential equations describing results more general than Farr's, many of Farr's results can be recovered as limiting cases. For example, Farr was the first to describe what we now call a 'normal' epidemic curve, a standard tool in modern-day epidemiology.[21] And this was not the only one of Farr's successes that survived: his equations describing the vulnerability of different populations, at different ages, to different diseases, and at different times during an epidemic are still with us, as are his equations about what their chances were of death, recovery, or contracting a certain disease, and his description of the relation between population density and mortality also survives.

All of these are excellent candidates for structures, but, what is more, so are their relata: if we think about what it is that these relations are relating, we find that there are no good candidates for standard realists to fall back on. Population density, mortality rates, age-structures, recovery rates, incidence rates, epidemics, and even diseases are not entities in the usual realist sense. It is for this reason that the following two central structural realist claims look very attractive at this point: first, the claim that the relata of structures are themselves relations; second, the claim that the relevant structures are in some sense (ontologically) prior to their relata (see Ladyman, 2014). Of course, one might argue that the relata of the above relations are themselves composed of traditional realist entities: individuals. But, while that is true, it is still impossible to relate these individuals in the way that the relevant relations require: there simply is no way in which individuals can instantiate relations between population density and mortality rates, and it is in exactly this sense in which the structures themselves are what matters. Or, to put this into structural realist jargon: the only properties of individuals that are relevant at this point are extrinsic, not intrinsic properties (cf. Chakravartty, 2012). It is only as structure that the relata have the required properties in the first place. One cannot talk about population density without talking about relations.

So far, things look good for the structural realist. However, one might wonder whether it is really appropriate to regard these structures – the relations described by Farr – as going beyond the empirical. I think we do have good reasons for thinking of them in this way and they are as follows: first, one can make a good case for there being a strong theoretical component to Farr's claims: they were all heavily dependent on a specific disease theory, without which they would have never been discovered, and also have been completely meaningless. They 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. Second, all of Farr's relations have the character of laws, and, indeed, that is what Farr himself called them. In fact, Farr often compared these relations to laws in the physical and chemical sciences and thought they were the same in character. Thus, structural realists ought to say about Farr's relations exactly what they say about other laws.[22]

[18] Farr is not here talking about water-borne transmission in the current sense, but about water, polluted with zymotic material, evaporating into and contaminating the atmosphere. He still thought that diseases were contracted through air and entered the body through the lungs.

[19] I will not argue this point at length here. For further details, see Tulodziecki (forthcoming).

[20] See, for example, Farr (1838/2003): "The reduction of the phenomena of disease to simple laws, susceptible of calculation, offers the same attraction as other fields of investigation; but its intimate connexion with human interests and sufferings gives it stronger claims on attention" (220).

[21] There are, by now, many other epidemic curves. To my knowledge, Farr did not construct any of these other curves, but what he did construct, he got right.

[22] Of course, laws are, in general, based on empirical data, and so someone might object that other laws do not go beyond the empirical, either. For such a person, however, there are more pressing problems than this particular case. I take it that this is not at issue here.

5. Problems for structural realists

All of the above points speak in favour of structural realism, and, thus, one might think it passes the test: according to structural realism, structural elements ought to get retained in the transition from the miasma to the germ theory, and, judging from the previous section, it looks like they are. However, while a structuralist account seems to work well for the relations I have been discussing, there is, of course, the question of Farr's elevation law which, as we have seen, was not retained. Despite the fact that this law worked not just for one, but several cholera epidemics – in London at large, in the London sub-districts, and cities such as Liverpool – later epidemics did not conform to it. And, while we can explain some of the law's success through relating elevation and water supply, this constitutes only a fraction of what the original law was seemingly able to do, and we are not in a position to explain its success at large. The elevation law – despite being responsible for some of Farr's most important novel predictions – was abandoned.

At the same time, however, the elevation law is just as good a candidate for the structuralist as Farr's other structures; in fact, it was one of the most successful parts of Farr's theory, and his paradigm example of a law in medicine. The elevation law, in kind, is no different from the other laws we have seen, except that, possibly, it was more successful and precise than some of the other contenders. This leaves structural realists with a problem: they now have a number of relations, all of which seem to be of the same, predictively successful type, yet some were retained while others were not. Since the elevation law failed to be retained and since the initial structuralist endeavour was to make a connection between success and retention, it seems that the test did not come out in favour of structural realism after all.[23]

How might structural realists respond? Their initial reaction might be to think that the numbers still favour structural realists overall: after all, only one relation was discarded and many retained, and, so, perhaps what the case is giving us is simply a likely connection between success and retention (and approximate truth). However, besides the elevation law, there are plenty of other relations (mostly concerning environmental factors and personal characteristics) that also involved novel predictions and that were rejected. Another possible strategy is to move the focus away from novel prediction, to try to identify other features of the relations, equations, or their theoretical role, and to argue that only the elements tied to *those* features are retained. However, it is very hard to see how such an argument might be made, both in this case and others. Besides the fact that there are no contenders for this in the miasma case, there is also good reason to be sceptical about how widely applicable such a criterion would be, even if one could be found. This is true especially in view of the extant (structural) realist analyses we already have available, most of which rely on (use-)novel prediction. However, unless we have such a criterion, we find ourselves in exactly the position that Farr was in (in retrospect): we have no idea which of our theoretical structures we should put credence in. And, if we don't know what structures to put credence in, we don't know what structures to be realist about, and structural realism fails to do the job it set out to do.

One might conclude at this point that the challenge set by French & Ladyman has been met – a counterexample to structural realism has been found – and, thus, that structural realism fails the test and ought to be regarded with suspicion or even abandoned, at least in domains other than physics. Instead of drawing this gloomy conclusion, however, I want to pursue a more fruitful course of discussion and try to diagnose where exactly the problem lies. So far, we have, in typical structural realist fashion, only focused on the miasma theory's mathematical structure. However, if we look closely enough, it becomes clear that this is only one among many kinds of structure that we might have picked. Examining the theory's mathematical parts seems natural, especially in virtue of other structural realist discussions, but it is certainly not the only option.

The question "What is structure?" has, of course, received extensive structural realist treatment (see note 3). However, while the answer to this question is controversial, the extant discussions focus not on the kinds of different structures one might be able to identify in a given theory, but, rather, on questions about how one ought to understand the notion of structure itself. It is debated, for example, whether a group-theoretic, set-theoretic, or category-theoretic account is most appropriate. But, even if this question could be settled, there is still an important sense in which doing so is not helpful: even if one has an answer to the question of what is meant by 'structure' (say, one endorses the set-theoretic definition), this still does not help one identify specific structures in medical or other scientific theories, and, thus, even if one knows the definition of structure, this does not tell one what counts as a bona fide structure in the miasma case. Here, for example, are some other candidates one might consider:

1. The structure of the disease-process. (This is about how disease happens in the body and, for Farr, would have been Liebig's account of chemical interactions between miasma, blood, and zymotic material.)
2. The structure of the transmission mechanism for disease. (Is the disease transmitted through air, water, fleas, rats, touch, etc.?)
3. The pathological structure of the disease. (What body parts does a given disease affect? How does it enter the body? What is its progression of symptoms? And so on.)
4. The etiological structure of how the disease is spread in (different) populations. (Here, we are talking about the causal claims involved in thinking about how a given disease is spread epidemically and non-epidemically; this question is population- as opposed to individual-centred.)
5. The etiological structure of the disease itself. (This is about the causal disease chain inside the body: how are later stages and symptoms effected by earlier ones?)
6. The structure of the various contributing factors to a given disease. (In what ways do weather phenomena, personality characteristics, exposure to certain circumstances and materials, etc., interact with each other to affect how the disease manifests itself, both at an individual and a population level?).
7. The structure of the overall disease model. (This provides an overall picture of how and why a given disease occurs the way it does.)

All of these structures are quite different from each other: some are mathematical, some are causal, some are explanatory, some are merely descriptive, some are a combination, and some are none of the above. The question is: are all of these good or usable structures for structural realists or not? Structural realists have two options at this point: they could hold, either, that all of the above are good structures, or else that only some of them are.[24] Let's start with the first option, that they all are. At first glance, this seems a plausible

[23] It ought to be noted that all other types of realists also face a problem at this point (many thanks to Anjan Chakravartty for pressing this point); however, since the main focus of this paper is structural realism, I will not pursue these problems at greater length here.

[24] Of course, one could also hold that none of them are, but since this is not exactly in the spirit of structural realism, I will not discuss this option here.

view, since all of the above are clearly structures we are interested in, that we want a true theory to give an account of, and that a disease theory ought to address. The problem, however, is that some of the above structures – those that Farr and others got right – were retained, whereas some other structures – those that Farr and others got wrong – were discarded. For example, Farr got (1) right for some diseases but not others, he got (2) wrong (although for some diseases he was close), (3) he got wrong, (4) he got mostly right, (5) he got about half right (he was right about population density and age, but wrong about elevation), and (6) and (7) pose a special problem, so I will postpone their discussion until later.

Regardless of the exact details, however, it is clear that some of these structures were identified correctly whereas others were not. Moreover, once one has this many different kinds of structure, one might begin to worry that, for any theory, it will turn out that some structures were retained and others abandoned. We are now back to the previous problem: how do we know which of the structures we identify will turn out to be (approximately) right? Certainly, Farr had no way of distinguishing those that would be retained from those that would not. What is so special about those that get carried over? As before, there is no obvious answer to this question, but the situation is now significantly worse: with the proliferation of many different types of structure, it is much harder to identify what, if anything, they might share. In particular, it is worth noting that some of the structures on our list are not essentially predictive. (1) and (3), disease process and pathology, are examples of this; yet both were retained. This fact alone is enough to suggest that any account that focuses solely on (novel) prediction as the one required common element will run into trouble here.

Further, speaking in more general terms, even if there were such things as indicators of (approximate) truth, there is no reason to think that, ultimately, what is an indicator of (approximate) truth with respect to one of these structures ought also to be an indicator of (approximate) truth with respect to some or all of the others: why assume that what is a good indicator of (approximate) truth with respect to pathological structures, say, is the same as what is a good indicator of (approximate) truth with respect to epidemiological ones? Why think at all that there is (only) one criterion that these very different structures would share?[25]

Such is the problem that one runs into if one takes the view that any of the above structures count as good, bona fide structures and admits many different kinds. It is this line of thought that might lead one to think that only certain types of structures ought to be admitted. But, if so: which ones? The problem is that structural realism simply provides no guidance whatsoever about how to identify what counts as a (relevant) structure. One might be tempted here to take clues from some of the domains and examples for which we already have working structural realist accounts. But, the problem with this approach is that there is absolutely no reason to think that relevant structures in completely different domains would be identical or even similar. Physics and medicine, for instance, are very different fields, and even if we could identify some structures in medicine, based on what we know from examples in physics, the real worry that arises is that we would either misidentify some structures, and/or leave out some that turn out to be important. And, of course, we still have our previous problem, that out of a number of similar structures, some were retained and some were not. Thus, even if the second route is pursued, there is still no account of how to identify which is which from within the theory itself. This is clearly necessary, however, since, if we cannot do so, we cannot say which parts of our current theories we ought to be structural realists about, and this was the whole point.

Lastly, there is the worry about structure-types (6) and (7) – overall contributing factors and disease model – that I shelved earlier. For these structures, it is not clear how to answer the question of whether they were retained or not, due to what we might think of as a specificity problem. It is this: if we apply the notion of structure loosely, the miasma theory and the germ theory end up as isomorphic; if we apply it narrowly, they don't. But how do we identify the right level? How do we know, for example, what degree of specificity we need in order to classify and individuate different causal factors involved in bringing about diseases? Ought we to separate out, say, barometric pressure and humidity, or ought we to talk about atmospheric and climate-related conditions jointly and in the broadest sense? It is hard to see how the conclusion at this point could be anything other than: on some level, the miasmatists got it right; on another, they did not. As before, however, this is not helpful, if we are trying to determine which parts of our current theories we ought to be structural realists about.

One response to the specificity problem is to reject it as genuine and to claim that we can clearly identify the right level once we properly examine the succession of theories at stake. Here, the idea is that once we compare the miasma and the germ theory, we see what is retained, and it is this comparison that enables us to know when we've hit just the right spot. The problem with this response, however, is that it is circular: we cannot say that we now know what the right structural elements are because they were retained. If we do so, we no longer have an independent way of picking out those parts that survived from those that did not, but that is exactly what is required. What we want to know is whether certain, independently specified, elements were retained, but, if we can only identify these in retrospect, after they were carried over, then structural realism simply isn't testable against the historical record.

6. Upshot

What is the upshot of all this? To sum up: given that structural realism is about the continuity of structures as a guide to (approximate) truth, and given that structural realists think structures pick out real relations, we need, first, some guidance about how to identify these structures in actual, live theories. If we do not want just anything to count as a structure, we need, concretely, to know what types of structures are and are not legitimate candidates for structuralist relations. Second, even if this matter could be settled, we still need to know what the desiderata of such eligible (successful?) structures are. What criteria does a given structure need to have in order to make it onto the (approximately) true list? What criteria do we need successful structures to exhibit? Unless we have workable answers to these questions, we cannot test whether retained structures possess the relevant criteria, and, if we cannot test this, we cannot test structural realism. However, as we have seen, this solution faces problems: once we allow different types of structure, it is implausible to think that there will be exactly one criterion that fits them all. Worse, even if such a criterion turned out to exist, say novel predictive success, structural realists still face the earlier problem, that some predictively successful structures were retained while others were discarded.

Thus, either we accept structural realism more or less as it stands, in which case we now have exactly the counterexample we were looking for and can conclude it failed at least this test, or else a whole host of questions needs to be answered before structural realism can be regarded as properly testable in the first place. Specifically, we need an account of what features, other than novel predictive success, structures need to exhibit and share in order to count as support for a structuralist account. Of course, the miasma

[25] Once more I should stress that this is not just a problem for structural realists. Any realist account that focuses on a single element tied to retention faces the same issue.

theory is only one case in one domain. However, there is no reason to think that this case or this domain is special in any way. Moreover, even if it is, we still – or especially – want true medical theories; thus, if structural realism does not apply here, we need to know why not and what does.

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