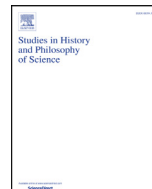




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D. Tulodziecki

Purdue University, Department of Philosophy, 7132 Beering Hall, West Lafayette, IN 47907, United States

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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.

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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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E-mail address: tulodziecki@purdue.edu.

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 \(2008\)](#).

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).

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