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# Koch's postulates: An interventionist perspective

Lauren N. Ross [a,b], James F. Woodward [c,*]

[a] Philosophy, University of Calgary, Canada
[b] Logic and Philosophy of Science, University of California, Irvine, United States
[c] History and Philosophy of Science, University of Pittsburgh, United States

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ABSTRACT

We argue that Koch's postulates are best understood within an interventionist account of causation, in the sense described in Woodward (2003). We show how this treatment helps to resolve interpretive puzzles associated with Koch's work and how it clarifies the different roles the postulates play in providing useful, yet not universal criteria for disease causation. Our paper is an effort at rational reconstruction; we attempt to show how Koch's postulates and reasoning make sense and are normatively justified within an interventionist framework and more difficult to understand within alternative frameworks for thinking about causation.



## 1. Introduction

Koch's criteria for disease causation, commonly referred to as "Koch's postulates," are often considered the first reliable method for establishing that a contagion is the cause of a disease. While Koch developed these criteria in the latter half of the 19th-century, they continue to receive significant attention. Koch's postulates are mentioned in nearly all beginning microbiology textbooks and they continue to be viewed as an important standard for establishing causal relationships in biomedicine.

In the secondary literature, Koch's postulates are commonly represented in the following three-part form[1]:

1. The contagion occurs in every case of the disease.
2. The contagion does not occur in other diseases or non-pathogenically.
3. After being fully isolated and repeatedly grown in pure culture the contagion can induce the disease by being introduced into a healthy animal.

Other formulations split the third postulate into two (Grimes, 2006; Schaffner, 2009) or add a final postulate requiring that the contagion be re-isolated from the diseased animal model and grown again in pure culture (Engelkirk, Duben-Engelkirk, and Wilson Burton 2011; Hogg, 2013).

Formulating a version of Koch's postulates that reflects what he actually says is complicated by the fact that Koch rarely discusses his causal criteria explicitly and, when he does, they are not stated as generally or clearly as the postulates ascribed to him today.[2,3] These features partly explain why there are so many different formulations of his postulates in the secondary literature and why those who analyze his criteria rarely cite his original publications (Carter, 1985, 353). In our view, Koch's criteria are best understood though his detailed discussion of specific laboratory techniques, and experimental results, on which he relies to argue for causality.

* Corresponding author.
*E-mail address:* jfw@pitt.edu (J.F. Woodward).

[1] (Carter, 1987b; Evans, 1976; Falkow, 2004; Fredericks & Relman, 1996, xviii). In correspondence, Carter has drawn our attention to his discussion on p. 136 of his (2003) in which he describes Koch's 1884 paper on tuberculosis as containing the most complete description of Koch's postulates. From this paper Carter extracts five "steps" which he takes Koch to advocate for "proving causation." Four of these steps (labeled Rt1, 2, 4, and 5) largely coincide with the three postulates cited above, but one (Rt3) ("The distribution of organisms must correlate with and explain the disease phenomenon") goes beyond 1–3 above. We focus on 1–3 because these are the most common form in which Koch discusses his criteria and also the most common form in which his postulates are discussed in the secondary literature.

[2] In fact, the designation of these criteria as "postulates" did not originate with Koch himself, but with his student Friedrich Löeffler, (Gradmann 2008, 2009, 3, 238, 219; Brock, 1988, 180–181; Löeffler, 1884). In this paper, we refer to Koch's causal criteria as "Koch's postulates," as is common in discussions of his work, despite the fact that he did not use this terminology.

[3] The fact that Koch rarely provides explicit discussion of his criteria has led some to claim that his publications contain "no original reference" for our modern day understanding of Koch's postulates and even that "Koch himself phrased no such postulates"(Gradmann, 2008, 218).

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Most scholars interpret Koch's postulates within a framework in which causal claims are understood as claims about necessary and sufficient conditions. This is the interpretation favored by K. Codell Carter, considered the "foremost authority" in this area (Gradmann, 2009, 83), and most other historians and philosophers (Broadbent, 2009; Smith, 2001, 2007). Within this approach, the first postulate is equated with the claim that the contagion is necessary for the disease, and the second and third with the claim that the contagion is sufficient for the disease.[4] In Carter's formulation, "[a] phenomenon C is necessary for a phenomenon E if the nonoccurrence of C ensures the nonoccurrence of E" and "a phenomenon C is sufficient for a phenomenon E if the occurrence of C ensures the occurrence of E" (Carter, 1985, 353–4).[5] Carter uses this framework to analyze Koch's causal criteria throughout his publications and to argue that Koch relies on different criteria at different points in his work (Carter, 1985, 354). He claims that Koch's early work begins with a conception according to which causation requires that the contagion is necessary for the disease and only later introduces the requirement that the contagion must also be sufficient. According to Carter, Koch relies on both necessity and sufficiency as criteria for causation in his mid-to-late 1880's publications and this is where we first see the "criteria we now know as Koch's Postulates" (Carter, 2003, 134).

This common interpretation raises a number of puzzles. First, if Koch relies on different causal criteria throughout his work, why does he often state that he has used the same method throughout, which he claims to have introduced in his first publication on disease causation? Second, if Koch's postulates amount to requiring that a contagion is a necessary and sufficient condition for the disease, why does he describe such evidence as only establishing correlation, which he claims can be distinguished from causation with evidence from animal inoculation experiments? Relatedly, why would Koch require that his causal proof involve experiments demonstrating disease in animal models when he knew some contagious diseases lacked such models?

Independent of these interpretive issues, Koch's postulates seem useful for some diseases, but of limited use for others. As often noted, they cannot establish causation for diseases with causes that cannot be isolated in pure culture, that are present in healthy carriers, and that have no known animal model.[6] Furthermore, it is often claimed that the postulates represent a "mono-causal" model that fails to accommodate the causal complexity characteristic of many diseases.[7] While discussions of Koch's postulates often emphasize these limitations, they are also viewed as an important guide and "standard" for establishing causality (Fredericks & Relman, 1996, 18).

They are seen as establishing causality when they can be fulfilled and as a starting point for new and improved causal criteria when they cannot be.[8] These discussions lead to the additional puzzle of how Koch's postulates can be useful, yet not universal.

In this paper, we argue that Koch's postulates are best understood within an interventionist account of causation, in the sense described in Woodward (2003). We describe how this interpretation is supported by Koch's discussions of disease causation, the causal reasoning he employs, and important aspects of the historical context within which he conducted his work. We view our paper as an effort at rational reconstruction; we attempt to show how Koch's postulates and reasoning make sense and are normatively justified within an interventionist framework and more difficult to understand within alternative frameworks for thinking about causation. Our discussion proceeds as follows: in section two, we discuss the historical context surrounding Koch's work and how it influenced his method of establishing disease causation. In section 3 we describe Woodward's (2003) interventionist account of causation and examine its relation to Koch's animal inoculation experiments, which comprise the third postulate. Section 4 discusses the relationship between interventionism and necessary and sufficient conceptions of causation in the context of understanding Koch's work. Section 5 argues that the first and second postulates are best understood as assumptions about *causal specificity*, a notion which plays an important role in Koch's causal reasoning. Section 6 provides more details regarding Koch's reasoning throughout his publications and how this is best understood with an interventionist framework.

## 2. Historical background

### 2.1. 19th century theories of disease and contagia

In the early to mid-19th century, the European medical community remained significantly divided over the nature of disease causation. Some favored a miasmatic theory which maintained that diseases were caused by noxious airs or "miasmata" that emanated from putrid or decaying substances (Smith Hughes, 1977, 1). These miasmata were characterized as undetectable, immaterial, and capable of causing diseases that seemed to be highly contagious and transmitted by air. Explanations for seemingly communicable diseases often appealed to "miasmatic influences" in addition to other long lists of causal factors, including dietary excess, exposure to extremes of temperature, emotional disturbance, and even the transgression of moral or social norms (Carter, 2003; Smith Hughes, 1977). Different diseases were often explained by citing similar lists of causal factors and the diseases themselves were characterized by groups of overlapping symptoms.

The miasmatic view contrasted with a contagionist theory of disease, which held that communicable diseases were caused by small material pathogens. The applicability of the contagionist theory to human disease was supported by evidence that certain plant and animal diseases were caused by microscopic contagia and that similar microscopic particles were present in some human diseases.[9] Jacob Henle, a German anatomist and professor to Koch, was one of the earliest and most well known supporters of the contagionist theory. Although Henle favored this theory, he admitted that there was insufficient evidence to conclusively support it as an account of human disease (Henle, 1961). Like most others at the time, he viewed the observation of an association between microscopic matter and disease as inconclusive evidence

[4] (Carter, 1987b, xviii; Smith, 2007, 95–96; Smith, 2001, 21).

[5] In his (2003) Carter argues that causation is a "theoretical" notion and that " in the absence of an accepted theory no amount of empirical evidence can demonstrate causal relations" (p.196). He takes this to be Koch's view as well. Carter informs us (personal correspondence) that on this basis that he would reject any necessary and sufficient condition conception of causation as philosophically inadequate. He also holds that Koch is not committed to such a conception. We are not sure how to reconcile these remarks with the passages quoted above. In any case, as observed above, a number of other writers do hold interpretations of Koch's postulates in terms of necessary and sufficient conditions. The general issue of whether (apart from what Koch may have thought) causation is a "theoretical" or "non-empirical" notion (or whether this contrast a fruitful one) is beyond the scope of this paper.

[6] (Evans, 1993; Smith Hughes, 1977).

[7] (Broadbent, 2009).

[8] For examples of suggested modifications of Koch's postulates, see: (Evans, 1976; Falkow, 1988; Fredericks & Relman, 1996; Smith, 2001).

[9] For example, in 1835 Augostino Bassi provided evidence that muscardine disease of silkworms was fungal in origin and in 1839 Johann Lucas Schonlein discovered the parasitic fungus thought to be responsible for "Impetigines" (Bulloch, 1938, 395; Smith Hughes, 1977, 2).