

Mechanisms, causation and the Russo-Williamson thesis

Abstract for Mechanism and Causality, 2009
University of Kent, Canterbury
9-11th September, 2009

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Russo and Williamson [2007] suggest that causation in medicine depends on both mechanistic and statistical evidence. From an HPS perspective, their account seems to conform well with the process of determining cause in medicine in most instances. In this paper, however, I will present three cases which seem to offer modest counterexamples.

The first – the discovery of McArdle’s syndrome – appears to be a case of strong causal inference arising in the absence of statistical evidence. Is, therefore, evidence of mechanism alone sufficient warrant for causation in some cases? The second example – the discovery of the causal relationship between infection with hepatitis B virus and the development of liver cancer – relies almost entirely on statistical evidence, with a much weaker role played by mechanisms. In fact, the mechanisms found in this case are generally plausible mechanism-sketches, based in analogy, rather than anything more confirmed. This example therefore suggests that perhaps mechanism too may be redundant in some cases. The third example details the apparently causal role played by infection with herpes simplex virus in the pathogenesis of cervical cancer. While this relationship was supported by a good deal of strong mechanistic and statistical evidence, the purported causal relationship turned out to be illusory. Risk of herpes simplex infection is merely correlated with risk of developing cervical cancer. Does this case mean that the Russo-Williamson thesis is sometimes unable to differentiate causation from correlation?

My answer to all three instances is a qualified ‘no’ – that is, given suitable extension of Russo and Williamson’s arguments. In very brief summary, I assert that their evidential pluralism is a necessary part of formulating useful causal claims in medicine. However, the nature of both mechanism and statistical evidence employed requires some finessing in order to accommodate causal situations of these types. The first case is disposed of by slightly broadening the requirement for statistical evidence to a requirement of evidence of difference-making in a more general sense. The second case requires an exploration of the nature of mechanistic dependency, in particular in situations of discovery or confirmation of mechanisms. While this section focuses on the role of intervention in mechanism construction, it also requires some discussion of the role of plausible mechanisms and of analogy in causation. The third case requires strengthening Russo and Williamson’s argument by requiring that the two types of evidence used must be strongly integrated if they are to support a judgement of causation. I will argue that subtle differences in the methodology of research programmes may grant us an epistemic warrant to do this differentiation. My paper therefore concludes with some suggestions on the nature of integration between mechanistic and statistical evidence

Russo, F. and Williamson, J. 2007. Interpreting Causality in the Health Sciences. *International Studies in the Philosophy of Science*. 21(2): 157–170.