Reviewer's report

Title: Notes on causality and susceptibility to disease

Version: 1 Date: 30 January 2006

Reviewer: Jørn Olsen

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General

Diseases have causes; some of these causes are man made, therefore, some diseases are preventable. These axioms underpin the rationale for public health and, as seen, the concept of causation is key.

It may be worth mentioning that Hume’s writing played a role in the “Koch postulates” of sufficient and necessary causes and that necessary causes were made necessary by defining the disease in a way that included the cause. Even for smallpox, the case may not be so simple. Monkey Pox has clinical features that are close to what you see for Small Pox.

Causal models that do not operate with probabilistic outcomes are, however, rare. And, I agree that Rothman’s causal modules have been, and still are, extremely useful in epidemiology. His work also has roots in the philosophical literature, especially in the work by Mackie in the 1960’s and 70’s and his comprehensive book on the topic (The Cement of the Universe) published in 1974.

The term necessary cause makes sense for any event that has taken place for the individuals or for a population. The Mackie/Rothman model says that diseases have causes following the INUS rules. These individual component causes are insufficient but necessary within the causal field. The causal field is unnecessary (if there are more) but sufficient. The model tells us that the strength of association between a given exposure and the disease depends upon the prevalence of the other component’s cause in the relevant causal fields. A statement that is well supported by empirical evidence. The model also tells us that we only occasionally have reasons to believe simple additive or multiplicative effects.

I am not sure that epidemiologists have a tendency to focus upon main effects. They have a tendency to focus upon avoidable causes. They have a tendency to focus upon preventable effects which may be quite reasonable. In philosophy people have debated the action versus the condition as causes like “the cause of fire is lighting of a match, not the presence of wood”. If the fire could have been prevented by removing the wood, we would, however, accept this as a cause.

At the population level, we run into problems if we try to compare attributable fractions with etiologic fractions. Attributable fractions may be zero if the exposure causes the disease for some and prevents it for others. The Mackie/Rothman causal model further illustrates that it makes little sense to talk about causes in relation to single exposures.

The reservation of the twin study to provide evidence for an inherited fraction of the disease is well taken. As an additional example, one could add that schizophrenia may be partly caused by an intrauterine infection. The risk of this infection to cause fetal brain lesions may be a function of brain development and the number of chorions that would often differ for dizogotic and monozygotic twins. And as the author rightly states, many of these concepts become even more complicated when studied over time to capture disease progression from step to step.
Major Compulsory Revisions (that the author must respond to before a decision on publication can be reached)

None

Minor Essential Revisions (such as missing labels on figures, or the wrong use of a term, which the author can be trusted to correct)

None

Discretionary Revisions (which the author can choose to ignore)

For the authors to consider

**What next?:** Accept after discretionary revisions

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Declaration of competing interests:**

I declare that I have no competing interests.