Book review

Philosophy of Epidemiology by A. Broadbent.

The philosophy of medicine is a young discipline that can lay claim to fewer monographs than would fill a single shelf on a narrow bookcase. It thus sounds unimpressive to suggest that Alex Broadbent's *Philosophy of Epidemiology* belongs on any philosopher of medicine's shelf, as there is plenty of room. Instead, I will recommend this superb study to any philosopher of medicine, philosopher of science, reflective epidemiologist and intrepid doctor.

Philosophy of Epidemiology inaugurates a new specialty in both the philosophy of medicine and the philosophy of science. It is the first book-length philosophical treatment of epidemiology, a special science as deserving of the philosopher's attention as physics, math or biology. (For the benefit of the philosophers of physics, math or biology, epidemiology, as defined in the book, is 'the study of the distribution and determinants of disease and other health states in human populations by means of group comparisons for the purpose of improving population health' [1]).

As its author notes, the book is not a comprehensive treatment of every philosophically interesting topic in epidemiology. Rather, it explores a selective range of important philosophical topics thrown up by epidemiology, most of which are conceptual or epistemic. Some of the topics, such as those dealing with measures of association, are peculiar to epidemiology, which is itself a peculiar science. Other topics, including those concerned with causation, have already received sustained attention from philosophy of science, but benefit here from attention to epidemiology.

Where exactly does the philosophy of epidemiology fit in a philosophy department course catalogue? Though Broadbent does not define the philosophy of epidemiology explicitly, he compares it with the philosophies of the special sciences like the philosophy of physics. Broadbent avoids 'intellectual territorialism' in his introduction [1], but I will risk it for a moment to help visualize the landscape. The philosophy of epidemiology is nested within the philosophy of science. The philosophy of medicine – I conjecture – cuts across several sub-disciplines of the philosophy of science [philosophy of epidemiology, philosophy of evidence-based medicine (EBM), parts of philosophy of biology] as well as disciplines outside of the philosophy of science (ethics, phenomenology, and others).

The philosophical study of epidemiology overlaps somewhat with the study of EBM but should be considered autonomous from the study of EBM, just as the science of epidemiology is autonomous from (but overlapping with) the practice of medicine. (The first monograph on the philosophy of EBM has already been written [2]). In fact, Broadbent writes that a philosophical treatment of epidemiology, which is a new science, ought 'to reinforce the sense of epidemiology as a discipline in its own right' [1]. Autonomy has its advantages. While EBM and the study of EBM have focused most of their attention on issues around treatment efficacy/effectiveness (due to their preoccupation with randomized trials), epidemiologists more often study harms through observational study designs. There is thus ample opportunity for the philosophy of epidemiology to attend to this woefully neglected area.

In the remainder of the introductory chapter, Broadbent lists plenty of other reasons why epidemiology is philosophically interesting: it is foremost concerned with hunting causes; it is characterized by its methodology rather than by any domain-specific theory; its methods of counting and comparison are relatively domain-insensitive; it concerns populations and their properties; and the stakes are high with epidemiological judgement, lives hang in the balance [1].

Broadbent states that his purpose is to 'explore and explain rather than argue' [1]. But, throughout the book, in addition to a clear and fair reconstruction of various positions and arguments, he provides reasonable criticism and thoughtful defence of his own (often novel) positions. While his intention is not primarily to defend a central thesis, the book does have a 'central theme': that 'explanation deserves more epidemiological attention and causation less' [1]; many conceptual and epistemic issues surrounding causation in epidemiology could be clarified by focusing on explanation (more on this idea later). The book returns to this theme often, especially in chapters 3–7.

The rest of the book is structured into 11 chapters, most of which explore a particular problem or related set of problems. Chapter 2 provides some introductions to the problem of induction, causation, the history of epidemiology and some basic epidemiological study designs. Chapter 3 sets out and explores the causal interpretation problem: how the causal import of measures of association is to be interpreted. Chapters 4–5 develop principles of good causal inference in epidemiology. Chapters 6–7 develop principles of good prediction making. Chapter 8 attempts to understand the causal meaning of one particular measure of public health concern: the attributable fraction. Chapter 9 asks whether there is any epistemic basis for 'risk relativism', a preference for the relative risk (RR) measure. Chapter 10 examines the monocausal and multifactorial models of disease aetiology, and suggests an alternate contrastive model of disease. Chapter 11 wonders what epidemiological evidence can say about the cause of specific cases of disease, and thus about the relevance of epidemiological evidence in the courts. Finally, Chapter 12 is a brief section that restates the central theme.

At the outset, Broadbent sets for himself 'the ideal of a thoroughly philosophical yet thoroughly engaged treatment', which he admits may be elusive [1]. The book teeters at times towards the 'thoroughly philosophical' – chapter 3 on the causal interpretation of epidemiological measures is perhaps a philosopher's chapter – and at other times towards the 'thoroughly engaged' – chapters 9 and 11 engage largely with arguments in the epidemiological literature and the legal literature, respectively. Epidemiologist Sander Greenland is listed as many times in the References as philosopher Nancy Cartwright. Part of Broadbent's heavy reliance on sources outside of philosophy is explained by the lack of attention that the (important) questions he tackles have received from philosophy. Philosophically interesting questions are often first noticed because they vex scientists. However, even after a philosophical literature is built up around a question, engagement

with practice is a desideratum rather than a detriment to philosophy. Indeed, such engagement is characteristic of the philosophy of medicine, including some of its best work.

Perhaps the greatest virtue of Broadbent's monograph is the clarity and perspective it brings to questions that are not just problems *in* epidemiology or medicine, but *for* epidemiology and medicine – problems of great practical import. For instance, chapter 11 questions whether there are any grounds, epistemic or otherwise, for the privileging of 'relative measures' in modern epidemiology and medicine. Commentators often complain that the preference for reporting the RR or relative risk reduction (RRR) is groundless (unless one counts less savory private motivations), and has led to serious distortions in the evidence base that could harm patients[1] [3,4]. Engaging with the concerns of epidemiologists is profitable for philosophy of science as well. Broadbent speculates that the preference for the RR might be due to the long shadow that physics casts over other sciences; the RR of a given exposure is usually thought to be population invariant, or universal, analogous to the laws of physics. But epidemiologists should doubt the universality of any measure of association, and philosophers should likewise resist the urge to conform population-level phenomena to a scientific image based on the physics worldview.

Even if one sets aside the practical significance of the book, it still stands as an important contribution to the philosophy of science. Among its merits, it sheds light on the topic of prediction in science, which has received scant attention from philosophers. It develops theories of causal inference and prediction that are suitably abstract to be broadly relevant, and that connect with existing work in the philosophy of science on mechanisms and on extrapolation.

Broadbent succeeds at meeting the demands of philosophical rigour and practical engagement, even if certain problems he tackles are more recognizably 'philosophical problems' than others. In fact, that some problems are unfamiliar to the philosopher is evidence of the book's success. It shows a willingness to address novel issues, and also shows that the book takes seriously their novelty rather than attempting to force them into existing models as merely tokens of old, familiar problems. Perhaps some of the problems Broadbent tackles are scientific problems, but it does not follow that they are therefore not philosophical problems. Some questions are not the exclusive domain of philosophy or science, and deserve attention from both philosophers and scientists. Epidemiologists should care about their concepts and epistemological devices just as much as philosophers.

My criticisms of the book are few and minor. Broadbent develops his central theme (more explanation, less causation) in most detail at the end of chapter 3. He proposes that the model of contrastive causal explanation developed by Peter Lipton [5] holds the answers to many problems that the book sets out to address: the

causal interpretation problem, causal inference, prediction making and models of disease aetiology [1]. Given its importance for Broadbent's own solutions to these problems, the topic of explanation probably deserved a chapter of its own. In particular, the contrastive model could have been contrasted with other explanatory models, and its adaptation to group comparisons (from its origin in single event comparisons) could have been developed more substantially.

The book persuaded me that an explanatory approach to problems related to causation in epidemiology can be useful. Thus, I accepted the argument that explanation deserves more attention. However, I was not completely convinced that causation deserves less attention, as the rest of Broadbent's central theme states. For one thing, the explanations he advocates are causal explanations. But, more importantly, rather than fully resolving the ambiguities of population causal thinking in epidemiology, the explanatory approach seems to push the ambiguities down onto the level of individual cases (individual causation). For example, part of his explanatory solution to the problem of assessing the causal meaning of population measures (chapter 3) is to say that measures of association like the RR quantify over individual cases [1]. But, we are left wondering as to the nature of these singular causal facts. That is not to say that Broadbent's solution is not successful; having started with two problems (general causation and individual causation), we are now left with only one. Yet one could argue that causation at the individual level is deserving of more attention in epidemiology, not less. Epidemiology is after all a discipline that – as the book mentions – counts the case-control study among its methods.[2]

This objection should not be seen as highlighting any weakness of the text; I think it shows that the book succeeds in unpacking very interesting questions about the scope of epidemiology and the relationship between population-level and individual-level causal claims. *Philosophy of Epidemiology* asks fruitful questions and provides insightful diagnoses for many of epidemiology's philosophical woes – and does it with eloquence and personality. It brilliantly opens up an entire sub-discipline in the philosophy of medicine that – like the book – I hope will receive the serious attention from philosophers and epidemiologists that it deserves.

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[1] The RR and RRR represent the average treatment effect as a ratio, with the base rate (control rate, untreated rate) as the divisor. By comparison, the absolute risk reduction (ARR) represents the treatment effect as a difference in outcome. If the base rate of an undesired outcome in a population is 2% and the treatment lowers the rate to 1%, then the ARR is 1%. Since this reduction of 1% represents half of the base rate, the corresponding RRR is 50%. The RRR can be misleading; it might lead the patient (and doctor) to think that it is a coin toss whether the patient will benefit or not, when really the net benefit from treatment in the population is only 1%. Patients with a distorted view of the effectiveness of treatment might make the wrong decisions, which can in turn be harmful.

[2] In a comparative group study (e.g. a clinical trial), we can determine whether an exposure caused the outcome in some patients, but not which patients (some exposed patients would have gotten the outcome anyway regardless of the exposure). In a case-control study, the investigator attempts to determine whether the exposure caused the outcome in a particular patient (the case) by comparing them to an unexposed but otherwise similar patient that lacked the outcome (the control). Thus, it is false that epidemiology deals only with causation in populations and not in individuals, as might be mistakenly thought.

References

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