Epidemiological Explanations

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Alex Broadbent, ***Philosophy of Epidemiology***. London: Macmillan (2013), 228 pp., $95.00 (cloth).

1. Introduction. Informal polling will readily reveal that epidemiology is rather unfamiliar to your *Zeitgenossen* outside academia. On the other hand, most of us are well aware that some systematic research strategy must be behind common knowledge such as "smoking causes lung cancer," "cholesterol levels should be kept in an acceptable range," and "regular exercise can prolong your life." Such news is brought to you by epidemiologic research. While some epidemiologists have displayed a keen interest in the philosophical underpinnings of their work for decades (Susser 1973; Rothman 1976; Weed 1986), philosophers only began writing about epidemiologic issues a few years ago (Thygesen, Andersen, and Andersen 2005; Russo and Williamson 2007). Now, Alex Broadbent's book *Philosophy of Epidemiology* (henceforth *PoE*) represents the first comprehensive, critical, and constructive look at epidemiology from within philosophy. In this critical review my goal is to discuss *PoE* and its main arguments from the epidemiologist's perspective.

The book "is a survey not of concrete problems, but of themes"; it "is not organized around a unifying argument or position, . . . although it does have a unifying theme" and "sets out to explore and explain rather than argue" (7–8). This exploratory and explanatory approach is helpful because it provides epidemiologists with a critical perspective on their concepts, language, and assumptions; provides philosophers of science with a concise but detailed overview of interesting problems in one of the most important

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health sciences; and provides both with encouragement to contribute to the philosophy of epidemiology.

In the first part (chaps. 1–3), Broadbent describes what he considers "striking features" that render epidemiology worthy of philosophical inquiry (chap. 1) and outlines philosophical and epidemiologic concepts as a basis for what follows (chap. 2). He provides a discussion of the "causal interpretation problem," that is, the absence of causal meaning from the mathematical definition of epidemiologic measures of association (chap. 3). The second part (chaps. 4–7) is the centerpiece of the book. Broadbent suggests replacing the notion of successful translation of epidemiologic research into better health as a criterion for causal inference by the notions of stability (chaps. 4 and 5) and prediction (chaps. 6 and 7). In the third part (chaps. 8–11), Broadbent addresses specific epidemiologic topics related to attributable risk, risk relativism, multivariability, and legal aspects. Throughout the book, he champions a contrastive causal inference view, mainly based on Lipton's (1991) work, and suggests de-emphasizing causation in favor of explanation.

Before I begin my discussion of the central argument put forward in *PoE*, I want to make it eminently clear that Broadbent's is a very important book in multiple ways. First, I truly like the general spirit of the book, deconstructing (and sometimes debunking) traditional assumptions that underlie epidemiologic research. It is written in a lively and highly accessible style that makes it a page-turner for all epidemiologists interested in a fresh look behind the scenes of their trade, in particular for those who consider their work "disease causation research." The book makes for excellent reading material at the intermediate epidemiology course level. Finally, it offers a unique contribution to the currently expanding discussion of epidemiologic issues in philosophy of science, for example, mechanisms.

Although I mostly agree with Broadbent, I would like to offer a few thoughts on some of his basic notions about epidemiology in the next section. In the subsequent section, I turn to his central argument. In the final section, I offer some cursory comments on his discussion of epidemiologic specifics in chapters 8–10 of *PoE*. I devote more space to the central section of the book and less to the first and third sections.

2. Basic Notions

2.1. Definition. Broadbent defines epidemiology as "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). It would be a mistake to interpret Broadbent's definition of epidemiology as excluding veterinary epidemiology, but it would be nice to consider animal health explicitly. It would also be wrong to think of it as including public health, which is the field whose purpose is to im-

prove population health. Strictly speaking, epidemiology is just one of the basic sciences of public health. The short-term goal of epidemiology is not to improve health but to generate data for decision making in medicine and public health. For instance, David Savitz writes that "the goal for epidemiologic research (is) the quantification of the causal relation between exposure and disease" (2003, 9). This more focused definition ascribes epidemiology a realm of study (health- and illness-related phenomena), restricts its activities to the collection and analysis of data in the context of various study designs, and deliberately includes causal inference. This is where philosophy comes in—at the point of transition from observed data to causal inferences needed to justify medical or public health action.

2.2. Epidemiology's Features. I fully agree with four of six "striking features" Broadbent describes to distinguish epidemiology from other forms of research: the virtual absence of experimental wet lab work (although molecular, genetic, and infectious disease epidemiologists might disagree), population thinking, the lack of theory, and the high stakes associated with the health issues studied.

Another feature is "domain insensitivity" of its methods, because the main technique for gathering data is simple counting, which obviously can (and frequently does) include phenomena and characteristics outside the medical realm. In this section, Broadbent seems to restrict epidemiology's realm of influence somewhat artificially to medical science. However, most of observational epidemiology is designed to inform those who work in public health, not medicine, where nonmedical risk factors (e.g., poverty, environment, access to health care) and nonmedical interventions are often of highest importance. Only one epidemiologic study design, the interventional randomized controlled trial (RCT), often has direct influence on medical decision making. Observational studies, on the other hand, generate information about the relationships between exposures and outcome, thereby hinting at issues related to disease causation.

Broadbent also points out that epidemiology and philosophy of science share a vested interest in causation and causal inference. I certainly agree that epidemiologists doing this kind of work will indeed benefit from philosophers' attention; what will be needed is that epidemiologists realize the importance of such interdisciplinary work. The book's emphasis on how useful epidemiologic concepts, techniques, and results are in improving population health will be particularly helpful in achieving this goal.

3. Core Issue. In chapter 3, Broadbent begins to justify his main suggestion in *PoE*, to de-emphasize causation in favor of explanation: "Explanation is a much more useful concept for understanding measures of association (chap. 3) and the nature of causal inference (chaps. 4 and 5) than

causation itself is, because causation is really only part of what we seek to measure and infer, respectively. What epidemiologists really seek to do is explain, and their practices are seen much more clearly when described as such" (8). The point of departure is what Broadbent calls the "causal interpretation problem": the difficulty of finding "the 'extra ingredient' in the meaning of epidemiological causal claims beyond the mathematical definitions of measures of associations" (34).

This is different from "causal inference" based on epidemiological data. Indeed, very few epidemiologists think they can find causation in their data. Most of us (I include Broadbent as an honorary epidemiologist here) will probably agree with David Savitz that "causal inference is just that—an inference by the interpreter of the data, not a product of the study or something that is found within the evidence generated by the study" (2003, 20), and with Douglas Weed, one of the few philosophically versed epidemiologists, who writes that "nowhere in the peer-reviewed literature, in the tables, nor in the graphs and figures displayed in all the studies that have been published, can we find this thing called 'causation.' Not one of these shows us that, in fact, human papillomavirus causes cervical cancer. Not one. To tell you the truth, we have seen causation only in words" (2008, 948). Broadbent offers cogent reasons why neither probabilistic nor counterfactual accounts of causation are viable solutions to the causal interpretation problem, although both have traditionally played a role in epidemiologic causal inference (Parascandola and Weed 2001). Instead, he suggests an explanatory approach toward causal interpretation of a measure of causal strength: "A measure of causal strength is a measure of the net difference in outcome explained by an exposure" (50). Here, Broadbent builds on Peter Lipton's work, who suggests that inference to the best explanation is best arrived at by asking, "Why this *rather than* that?" not just "Why that?" (Lipton 1991), employing the technique known as "contrastive explanation." Broadbent holds that in epidemiology this question can be answered by looking at the population exposure difference condition "to explain a difference in outcome such that the outcome in group A is greater than outcome in group B by degree n, we must cite a difference in exposures between groups A and B, which causes at least degree n of the outcome in group A" (53), where "degree n" stands in for any measure of strength of association. He asserts that this condition is not circular, because "the causal claims being analysed are general causal claims, while the notion employed in analysing them is singular causation" (53).

Coming from the contrastive explanatory perspective, Broadbent turns his attention in chapters 4–7 to stability and prediction as explanatory tools. This "explanatory turn" is interesting and deserves careful consideration by epidemiologists and philosophers alike. In the remainder of this section, I first comment on Broadbent's discussion of "the myth of translation" (as in

"translational research"; Woolf 2008). I then discuss stability and prediction and suggest adding a confirmatory aspect to this proposal: explanation by intervention.

3.1. Translation. In light of his emphasis on the usefulness of epidemiologic results, Broadbent rejects the notion of "translation" as "what happens between a piece of scientific research being *done* and being *used*" as a myth (57). In particular, he suggests that the term is used out of frustration with the fact that the results of epidemiologic research have yet to yield the expected impact on population health, which would be "another way of expressing the results in question" (58). He rejects the idea that "the fact that smoking causes lung cancer can be *translated* into a reduction in smoking prevalence" (58), as if the term "translation" is used by epidemiologists as an indication that the epidemiologic claim (smoking causes lung cancer) is necessary and sufficient to initiate a public health intervention (smoking cessation), which is in turn necessary and sufficient to produce the intended population health effect (reduced incidence of lung cancer).

I think that this is not what most biomedical researchers refer to when using the term "translational research." Indeed, it is not frequently used to describe epidemiologic research at all. In an oft-cited commentary, medical doctor and public health researcher David Woolf suggests that the term "translational research" has two meanings: "harnessing knowledge from basic sciences to produce new drugs, devices, and treatment options for patients," and "ensuring that new treatments and research knowledge actually reach the patients or populations for whom they are intended and are implemented correctly" (2008, 211). The first meaning refers to bench (basic) science results being "translated" into useful medical interventions, mainly by feeding basic science results into drug and device development. As such, the first meaning of "translation" is mainly confined to the medical realm. The second refers to activities that ensure access to health care and a healthy environment, thereby focusing on issues usually covered by public health. Note that neither meaning explicitly includes observational (risk factor) epidemiologic research, although the first can be interpreted as including results produced by RCTs, the main form of interventional epidemiologic research.

Broadbent appears to think of translation as a transformation of the results of a given study into more clinically relevant post hoc interpretation of those very results. I agree with one of my students, who recently wrote,

> I think of translational research as taking a causal claim from basic research to the next level (drug development) by rephrasing the same causal claim in a more clinically relevant way. For example, one might use basic research to investigate the causal claim that a given drug inhibits a re-

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> ceptor of interest. You might then "translate" this claim into the claim that oral administration of the drug will affect a more "patient-oriented" outcome such as mortality, obesity, etc. To investigate this new causal claim, which includes some hypothesized causal ancestors and/or descendants of the basic science causal claim, we have to conduct more studies, mainly RCTs. (A. R. Fiorentino, personal communication)

In other words, it is not the case that results of translational work are the translation, but they can be used to design interventions intended to improve the health of populations, which in turn is the job not of those working in epidemiology but of those working in public health (see Savitz's definition of the goal of epidemiology above). Broadbent correctly interprets "translation" as a metaphor for "the path from the production of knowledge to its use" (57). Some might view this path as a straight, direct, and reliable road, leading safely and somewhat automatically (without further ado) to guaranteed success (improved health) even if you are asleep at the wheel. Thus interpreted, I agree that such "translation" is a myth. However, I am quite certain that most epidemiologists think of "translation" as a winding, rocky road through unknown territory, sometimes leading nowhere, but almost always to new hypotheses, more research, and novel ideas.

3.2. Stability. As an alternative focus "for frustrated scientists hoping to have more impact on population health" (59), Broadbent suggests "stability." The main idea put forward in chapters 4 and 5 is that the stability of an exposure/outcome relationship renders it worthy of consideration for intervention design. Here, stability of an epidemiologic finding can be thought of as its robustness—a finding that is not easily contradicted by subsequent results generated by well-performed research.

This version of "stability" is reminiscent of one of Hill's classic viewpoints on causation in epidemiology: consistency (Hill 1965), which refers to the persistence of an epidemiologic finding across multiple different study designs and populations. One single study can yield one particular result that would be considered stable without being confirmed, for example, because no additional studies on the same subject have ever been conducted or accepted for publication due to a lack of novelty. I think that stability implicitly requires multiple attempts to show that the initial, single finding is due to chance, bias, or confounding, the three traditional ways in which a spurious finding is explained in epidemiology. There is no question, from the epidemiologic perspective, that stability lends explanatory value to the interpretation of an epidemiologic finding.

3.3. Prediction. In chapters 6 and 7, Broadbent outlines a theory of prediction in epidemiology. He states that a causal inference is not a prediction

of successful action: "just because X causes Y, that does not mean that removing X is a sufficient means to removing Y" (83). After surveying the admittedly sparse epidemiological and philosophical literature on prediction and finding that both leave much to be desired, Broadbent answers the question, What is a good prediction? He offers a detailed discussion of why we need "an understanding which shows how good prediction activities lead to good (true) prediction claims" (91) and concludes that epidemiological prediction activities cannot be assessed solely based on comparison with the truth on certain occasions or in the past and that prediction activities need to justify the prediction claims they yield. In chapter 7, Broadbent shows that this can be achieved by requiring prediction activities to yield stable results. He rejects the idea that stable predictions can be arrived at by extrapolating (e.g., measures of association from one population to another), by making inferences from laws of nature, or by referring to knowledge of underlying mechanisms. To make a long and very interesting story short, Broadbent suggests, in keeping with his contrastive approach, that a prediction activity is good only if it explains why the prediction claim is true rather than another scientifically possible outcome (111). As a pithy criterion for a good prediction claim, Broadbent offers, "What could possibly go wrong?"

Apparently, Broadbent takes the distinction between causal and predictive claims much more seriously than most epidemiologists. For example, I have previously argued from a neopragmatic perspective that "it simply does not matter whether you say that 'smoking is associated with lung cancer' or 'smoking predicts lung cancer.' What does matter is that identifying, and then removing or reducing predictors from populations can improve the human condition" (Dammann 2009). The first statement ("it does not matter") reflects the long-standing tradition among epidemiologists of calling risk factors "predictors." Yes, there are measures of association (relative risk estimates) and measures of prediction (positive and negative predictive values), although the latter are used almost exclusively by clinical epidemiologists interested in screening and diagnostic testing. Particularly in the branch of epidemiology that deals with the identification of risk factors, few epidemiologists would have a problem with using the terms "predictor," "antecedent," and "risk factor" synonymously.

The second statement above ("what does matter") suggests that we should de-emphasize causal talk (and maybe even predictive talk) and emphasize what is actually the goal of epidemiology, namely, to help reduce the individual and societal burden associated with illness. This, in turn, motivates us to suggest adding "explanation by intervention" to Broadbent's shortlist of explanatory foci, explanation by stability and prediction.

3.4. Explanation by Intervention. Broadbent holds that "good causal inference in epidemiology must deliver a piece of causal knowledge that

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can be used to improve population health" (56). This notion echoes Nancy Cartwright's focus on usage in causal inference (Cartwright 2007). It is also "a functional approach to causation . . . that takes as its point of departure the idea that causal information and reasoning are sometimes useful or functional in the sense of serving various goals and purposes that we have" (Woodward 2014, 693). Finally, it is a somewhat neopragmatic account in that it shifts the attention away from talk about causation and toward talk about health improvement (Dammann 2009).

In recognition of the fact that epidemiologic results are generated with the goal of designing medical and/or public health interventions, I suggest that stability and prediction should be supplemented by an interventionist account of explanation. In fact, I think that Broadbent is actually doing this implicitly. However, I suggest that the interventionist stance should be considered explicitly in a theory of explanation in epidemiology because it is the main underlying idea of epidemiology in the setting of RCTs and public health interventions.

I think of successful intervention at the clinical or population level as the litmus test of causal inference by explanation in epidemiologic contexts, broadly construed as including both observational and intervention studies. Causal claims in epidemiology (broadly construed) are best supported by explanations that include evidence that interventions on the exposure variable result in the expected change of the outcome variable (Dammann and Leviton 2007). Of course, findings produced by clinical or population trials should themselves be stable and allow for good predictions. Successful intervention should perhaps be thought of as a post hoc confirmation by intervention based on observational data rather than an additional way to support the conclusion that observational results themselves might be useful. On the other hand, even in observational settings, intervention on the exposure (e.g., variable manipulation such as stratification) during data analysis might be associated with changes in the outcome that qualify as useful observational results (quasi-intervention).

4. Attributability, Risk Relativism, Multifactorialism, and the Law. I end with a few comments on the last four chapters of the book. In these last chapters, Broadbent zooms in on four philosophically interesting aspects of epidemiology that are not of primary import for his major argument I summarized in the previous section. Each of these can be read as a freestanding, self-contained paper, and each debunks at least one of the urban myths of epidemiologic practice.

4.1. Attributability. In chapter 8, the limelight is on measures of attributability (as in attributable [aka excess] risk). The attributable/excess risk is

calculated as the difference between risk among the exposed and risk among the unexposed individuals in an epidemiologic study. First, Broadbent describes the attributable risk (AR) as being prone to two fallacies: exclusive cause fallacy, that is, the erroneous belief that the cause of interest is the only and unconfounded cause of the outcome, and the counterfactual fallacy, that is, the erroneous belief that "magicking the exposure away" would reduce the number of cases of the disease in this population by exactly the number indicated by the AR. He holds that the AR does not accurately reflect the amount the risk would drop if you removed the exposure, because in practice other unaccounted-for exposures might replace the exposure of interest and would cause the outcome even in the absence of the original exposure. This is an interesting, possible, and perhaps even likely scenario. However, this caveat does not apply so much to what is measured by the AR as to how we frequently interpret it, that is, as the fraction of total risk we would get rid of if we got rid of the exposure for which the AR was calculated. Broadbent discusses issues related to the causal interpretation of the term "attributable to" as "caused by" and suggests, again, to transition from causation to explanation by stating that "a fraction of a risk is attributable to an exposure if and only if the exposure explains why the corresponding net difference between exposed and unexposed risks arises" (127).

4.2. Risk Relativism. Estimating the relative risk (RR) of developing the outcome among the exposed compared to the risk among the unexposed is at the core of epidemiologic inquiry. Broadbent discusses the mistake of interpreting RR as an expression of causal strength. He reviews the recent epidemiologic literature and reminds us that epidemiology is a context-sensitive toolbox rather than a natural science that is supposed to describe natural phenomena independent of context.

4.3. Multifactorialism. In his tenth chapter, Broadbent posits that epidemiology's lack of domain specificity (v.s.) broadens the scope of medicine by identifying new kinds of causes and new kinds of diseases. Rather provocatively, he states that "merely by studying the common and distinctive elements in the histories of persons who meet an unhappy fate, epidemiologists exert pressure on health professions to countenance both those elements and that unhappy fate as part of their concern" (147). After having offered an interesting discussion of whether those who work in medicine and public health ought to respond to such pressures, Broadbent transitions into multivariability, mainly thought of as multicausality. He contrasts it with monocausality as conceptualized in the Henle/Koch postulates (one bug, one disease), which he calls an unhappy implication. Indeed, as an instructor of introductory epidemiology, I confirm that, unfortunately, this assumption pre-

vails among medical and public health students, although "to call a disease multifactorial . . . hardly says anything at all, because every kind of event we ever encounter is multifactorial (149). He ends his chapter with yet another provocative suggestion, that is, to consider the contrastive model a better way to classify states of ill health than monocausal and multicausal models.

4.4. Epidemiology and the Law. In his final chapter, Broadbent surveys "existing legal positions on the use of epidemiologic evidence" and finds them confused (162). The main point here is the tension between general and specific causation, as well as the related question whether epidemiologic evidence can be applied at all in individual cases. The perception of what qualifies as good epidemiologic evidence in the courtroom in support of specific causal claims appears not to go far beyond the RR > 2/AF > 50% rule that gives rise to the question, Is it more likely than not that the exposure caused the outcome in the particular case at hand? Broadbent debunks such a simplistic notion as fallacious, based on the exclusive cause fallacy offered in his previous discussion of the AF. Still, he holds that epidemiologic evidence should be able to "help prove" under the right circumstances, and he offers a very interesting discussion about what exactly these circumstances might be.

5. Conclusion. Broadbent suggests redirecting our attention away from causation and toward explanation, and he provides a cogent contrastive model of explanation that emphasizes stability and prediction. I suggest that this model should be supplemented by a more explicit additional focus on the interpretation of results of intervention studies. Together, these points add up to the joint proposal that epidemiologic results are most likely to be useful if and only if they (1) are stable, that is, unlikely to be invalidated by subsequent good science, and (2) provide stable predictions. They are definitely most useful if confirmed by successful medical and public health interventions.

Broadbent's book is a superb contribution to both the philosophical and epidemiological literature. I believe that it is no exaggeration to say that Broadbent has almost single-handedly created a new field of philosophical investigation. He provides it all: a concise, lucid, yet comprehensive introduction to the subject, a novel theory of epidemiologic reasoning, and his unique philosophical perspective on various issues usually left unquestioned by epidemiologists. Moreover, he offers very valuable hints about areas that deserve further study (e.g., epidemiologic theory, theory of prediction). Although not all epidemiologists will necessarily agree with all of his definitions and arguments, all will eventually benefit from his work.

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