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SCIENTIFIC CONTRIBUTION

Epidemiology and causation

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Abstract Epidemiologists' discussions on causation are not always very enlightening with regard to the notion of 'cause' in epidemiology. Epidemiologists rightly work from a science-based approach to causation in epidemiology, but largely disagree about the matter. Disagreement may be partly due to confusion of the question of useful concepts for causal inference in epidemiological practice with the question of the metaphysical presuppositions of causal concepts used in epidemiology. In other words, epidemiologists seem to confuse the practical results of epidemiological research at the population level with the metaphysical views about the reality of disease causation at the individual level in their writings on causation.

Keywords Causality · Causal concepts underlying scientific practice · Epidemiology · Epidemiologists' debate on causation · Metaphysical presuppositions of causal concepts · Science-based approach to causation

Introduction

The notion of 'cause' is of central importance in the goals and practice of epidemiology. Look, for example, at the following description that should inform future students of the School of Public Health in New Orleans about their future field of activity (http://publichealth.lsuhsc.edu/EPID-Intro.html):

> Epidemiology is **the scientific study of factors affecting the health and illness of populations**, and it serves as the foundation and logic of **interventions made in the interest of public health and preventive medicine**. It is considered a cornerstone methodology of public health research, and is highly regarded in evidence-based medicine for identifying risk factors for disease and determining optimal treatment approaches to clinical practice.
>
> […] epidemiologists employ a range of study designs from the observational to experimental, with **the purpose of revealing unbiased relationships between exposures such as tobacco, nutrition, biological agents, stress, or chemicals to outcomes such as disease, wellness and other health indicators**. Defining the diseases, drawing disease **causal chains**, and formulation of health strategy are important aspects of epidemiology. (my bold)

As one can see, this quote contains a lot of causal terminology: 'affecting', 'preventive', 'interventions', 'risk factors', 'exposures', 'causal chains', etc. Given the central role of the notion of 'cause' in this branch of science, one should not be surprised that the epidemiological literature has seen an ever growing number of theoretical contributions of epidemiologists on 'causation' itself. Epidemiologists wonder in these articles about, e.g.

- The meaning of 'causation', given that our everyday concepts of causation are too rudimentary. (Rothman and Greenland 2005, p. S144).
- "a useful concept of causation in epidemiology." (Olsen 2003, p. 86).
- "some common thinking among epidemiologists about what is meant in saying 'X causes Y.'" (Parascandola and Weed 2001, p. 905)

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Moreover, epidemiologists writing on disease causation show a growing awareness of the complicated relation between the causal factors and the disease outcome. The period that epidemiologists call "the era of chronic disease epidemiology" (second half of the twentieth century) has given rise to theoretical writings on disease causation in which the need for 'multicausal' and 'multilevel' approaches to causation came to the foreground (see e.g., Anderson and Scott 1999; Cacioppo et al. 2000; Krantz and McCeney 2002; Pearce 1996). All these concerns resulted in a considerably large meta-level literature in epidemiology on the topic of causation.

The epidemiological literature on causation is not only of interest for epidemiologists, but also for philosophers of science because it exemplifies a science-based approach to causation.[1] Traditional philosophical analyses of the notion of 'cause' are most often of a general nature, and hence hardly ever focus on the meaning of the concept from the point of view of a specific scientific domain. But although a science-based approach is hardly debated in philosophy, it is clearly of concern for scientists. On the other hand, philosophical reflection on this debate can help epidemiologists in developing a clear science-based view on the notion of 'cause' in epidemiology.

In this paper, I will mainly focus on the difference between searching for (a) useful causal concept(s) for epidemiological practice and (b) inquiring the metaphysical presuppositions of the causal concepts and related methods in epidemiology—a distinction which is often not clearly made in epidemiologists' writings on causation.

My aim in reflecting on epidemiologists' discussions on causation in this paper is twofold. Firstly, I want to illustrate the difference between the two kinds of science-based questions about the concept of 'cause' that I distinguish in this paper by offering a possible answer to both questions. On the one hand, I answer the question for a useful notion of cause for epidemiological practice by defending a probabilistic account in terms of average effects as an appropriate one.[2] On the other hand, I answer the question of the metaphysical presuppositions of causal concepts in epidemiology by defending Kenneth Rothman's view on causation as one providing an insightful answer to that question. The last and central aim of this paper is to point to problems in epidemiologists' theoretical analyses of disease causation that lead to a confusing debate. These problems result precisely from epidemiologists' failure to distinguish in their science-based approach to causation between the two kinds of questions discerned in this paper.

In general, my aim in writing this paper is not to argue pro or contra the content of epidemiologists' views on causation. The discussion concerning content is only indirectly connected with what I primarily want to demonstrate, namely that there is confusion in the way some epidemiologists argue for their own point of view. Further, it is even less my aim to contest the achievements of epidemiology. To the contrary, my aim is to help epidemiologists in thinking clearer about causation, which I hope can further their achievements and the good interpretation of their findings.

The upset of the paper is as follows. In Sect. 2, I further circumscribe what a science-based analysis of the notion of 'cause' involves. I discern two kinds of questions within such a science-based approach. The first is concerned with the usefulness of causal concepts for a certain scientific discipline; the second is concerned with the metaphysical presuppositions of causal concepts underlying a certain scientific practice. In Sect. 3, I will defend a concept of cause in terms of average effects as an appropriate concept for epidemiological practice. I further point to the limited applicability of this approach to individualized prediction and prevention. In Sect. 4, I focus on the question of metaphysical presuppositions underlying the notion of cause in epidemiology and argue that Kenneth Rothman's view provides an insightful answer. In Sect. 5, I show how the confusion over both science-based questions and their possible answers leads to problematic views and arguments in theoretical epidemiological papers on disease causation. In Sect. 6, I come to some final conclusions.

Scientific methods and metaphysical presuppositions

What does it mean to search for 'a concept of cause for a certain scientific discipline'; in this case for epidemiology? Such an approach from the point of view of scientific practice is clearly different from traditional philosophical approaches to causation (see also De Vreese 2006). In traditional philosophical analyses, the question of the usefulness of a given concept of 'cause' for certain

[1] In an earlier paper, I labeled such an approach an 'epistemological-methodological approach' to the notion of 'cause' (De Vreese 2006).

[2] It is not my aim to give a complete overview of causal concepts and related epidemiological methods for causal inference in this paper. Hence, I do not weigh all the pros and cons of these concepts and methods against each other in arguing for an average effect approach, neither will I focus on the Bradford Hill criteria (Bradford Hill 1965). Although elaborating a thorough comparison might be a worthwhile undertaking, it is not necessary in view of, and would lead me too far away from, the central aim of this paper. Hence, although I am convinced of the importance of the average effect approach for epidemiological practice, my defence should primarily be seen as illustrating the kind of ideas and problems one should tackle when answering the question for a useful concept of cause for epidemiological practice.

scientific practices is not under discussion.[3] Consequently, traditional analyses do not care about the prerequisites of a useful notion of 'cause' for scientific practice. For example, although scientists try to find some truth about the causal reality when searching for scientific causal knowledge, they do not search for an interpretation-free, precise and detailed picture of causation in the world, but rather search for that kind of causal information that is useful given the specific goals and interests of the discipline. In traditional analyses, philosophers to the contrary search for *the* single, justified and totally objective interpretation of 'cause', and herein try to free their analyses from the influence of goals or interests.

That a certain concept of cause is useful for a certain scientific discipline means that it is useful from a certain point of view, namely a point of view that reflects the interests and goals of the discipline. Given that the interests and goals will differ from discipline to discipline, the aspect(s) of causation given attention to will differ from scientific discipline to scientific discipline. The search for an appropriate notion of 'cause' for a certain discipline will consequently be influenced by the search for a certain kind of causal information which is of specific interest for the scientific discipline involved. Additionally, the choice for certain methods of causal inference will in turn be influenced by the preferred approach(es) to causation. The case of epidemiology nicely illustrates all this.

Although one starts thinking about causation from the point of view of the practice of science in a science-based approach, metaphysical presuppositions will inevitably underlie the concepts and methods that are used. The fact that goals, interests, and available methods guide the search for causal knowledge in the sciences does not imply that the causal concepts used in scientific practice are free from underlying ideas about what a 'cause' actually is, i.e., apart from our scientific interpretation of it. Insofar as it is of importance for the development of methods and the appropriate interpretation of results, investigating these presuppositions is of relevance to the scientist and does make part of a science-based analysis of causation. Hence, a science-based approach to causation should consider two kinds of questions with respect to causation: not only questions with respect to an appropriate conceptualization of 'cause' given the goals, interests and methods of given scientific disciplines; but also questions with respect to the metaphysical presuppositions of the different methods of causal inference used in the different scientific disciplines. In this article, I will argue that it is indeed useful to be aware of these metaphysical presuppositions and to dare to question them. However, 'inappropriate' metaphysical presuppositions do not form a straightforward reason to reject a scientific method for causal inference. Pragmatic reasons often form a justified basis for applying certain scientific methods which seem nonetheless misguided from the point of view of a general and purely philosophical analysis of what causation is. Precisely such pragmatic considerations make science-based analyses of causation differ from traditional philosophical analyses and demonstrate the necessity of science-based approaches next to traditional ones.

A concept of cause for epidemiology

Individual versus population level

As is clear from the description of the School for Public Health cited in the introduction, the primary interests of epidemiologists lie in the domain of public health, and hence at the population level:

> The true subject matter of epidemiologic practice and of textbooks of epidemiology is research design and methods for disentangling causes and effects. The definition [of epidemiology] may have evolved into the study of diseases in populations mainly because identifying and determining the importance of specific risk factors necessarily involves studying people in groups. Only by comparing people with and without the disease in terms of a history of exposure to a given factor, or by comparing disease rates among those who either have or have not been exposed to a factor of interest, can causes be elucidated. Epidemiology deals with groups of individuals because the methods for determining causality require it. (Mawson 2002, p. 2)

Beverly Rockhill (Rockhill et al. 1998, 2000; Rockhill 2001, 2005) has strongly emphasized this point. She argues against a late tendency in epidemiology to talk more and more in terms of 'individual effects'. Rockhill relates her critique directly to the main goal of epidemiology:

> I start with the assumption that the goal of epidemiologic research is, ultimately, disease prevention. This leads to a key question: "To what uses are

[3] Traditional philosophical approaches to causation are most often purely conceptual or purely metaphysical approaches. A conceptual analysis of causation tries to reveal the meaning of our everyday concept of causation, while a metaphysical approach is bound to shed light on what causation is in the world, i.e. apart from our (scientific) interpretation and description of it. Of course, nor our everyday notion of 'cause' and neither 'causation as it is in the world', is totally unrelated to the meaning accorded to 'cause' in the sciences. More about the differences and relations between different lines of approach to the notion of 'cause', and about the importance of science-based approaches next to purely conceptual and purely metaphysical ones, can be found in (De Vreese 2006).

> quantitative findings from epidemiology most appropriately applied, given a prime concern with prevention?" For the noninfectious diseases, most risk factors (including genetic ones) are associated with very low positive predictive values. This means that strategies based in individualism (e.g., individual risk communication, or "individualized prevention") are questionable scientifically and as public health policy. (Rockhill 2005, p. 124)

Additionally, Rockhill emphasizes that epidemiologists should mainly be concerned with causal factors that can be manipulated at the population level, in such a way that the goal of prevention and intervention is attainable. This leads Rockhill to criticizing epidemiologists' search for what she refers to as "universally necessary component causes of disease" that appear in each individual case of the disease, and without which most people do not develop the disease. She argues that there is no necessary link between the discovery of such causes and prevention and focuses with her criticism mainly on the recent trend of searching for genetic causes of diseases.

In sum, given epidemiologists' goals and methods, the focus lies at the population level. Causation in epidemiology should be interpreted in terms of average influences at the population level, and in terms of the possible manipulation of the presence of responsible factors at this level.

Of course, disease causation ultimately happens at the individual level. If one asks for disease causation as it is in the world, one should refer to the level of the individual. This seeming contradiction between the study of disease causation at the population level and the development of disease at the individual level, leads to confusion in epidemiologists' talk on disease causation. This is precisely due to the fact that epidemiologists are not always clear about the distinction between useful causal concepts for scientific practice, and metaphysical presuppositions underlying these causal concepts.

In the following section, I will go deeper into the causal concept underlying epidemiologists' methods to show why the knowledge gained most often does not bear to individual prediction or prevention.

The limits of epidemiological findings

Epidemiologists study general overall causal patterns of disease causation recurring in the population. These causal patterns are not necessarily exceptionless. The precise causal history leading up to one particular effect in singular cases is often very complicated and intractable and even not interesting for the purpose of prevention. As Giere (1997, pp. 121–122) argues, we often do not have the knowledge at our disposal to get a grip on every individual mechanism leading up to disease. Studying disease causation in large groups makes us nevertheless able to answer the question of what causes diseases without knowing much about the precise biological and chemical mechanisms involved. These considerations led Giere (1997) and Dupré (1993), amongst others, to the following view on causation:

> causes should be assessed in terms of average effect not only across different causal routes, but also across varying causal contexts. (Dupré 1993, p. 199)

Giere (1997) gives the following more detailed definitions:

> C is a positive causal factor for E in the population U whenever $\mathbf{P}_X(E)$ is greater than $\mathbf{P}_K(E)$.
>
> C is a negative causal factor for E in the population U whenever $\mathbf{P}_X(E)$ is less than $\mathbf{P}_K(E)$.
>
> **C** is causally irrelevant for **E** in the population U whenever $\mathbf{P}_X(E)$ is equal to $\mathbf{P}_K(E)$.
>
> (Giere 1997, p. 204)

Giere considers only binary variables. So, **C** is a variable with two values (C and Not-C); the same for **E** (values E and Not-E). X is the hypothetical population which is identical to U, except that each individual exhibits the value C of the causal variable **C**. K is the analogous hypothetical population in which all individuals exhibit C. $\mathbf{P}_X(E)$ and $\mathbf{P}_K(E)$ are the probability of E in, respectively, X and K. Probabilities are defined as relative frequencies (Giere takes U to be finite, i.e. his probabilistic causal claims are about finite populations).

For example, if we claim that smoking (**C**) is a positive causal factor for lung cancer (**E**) in the Belgian population (U), this amounts to claiming that if every inhabitant of Belgium were forced to smoke there would be more lung cancers in Belgium than if everyone were forbidden to smoke. Conversely for the claim that smoking is a negative causal factor. Causal irrelevance is a relation between variables (represented in bold) rather than a relation between values of a variable. If we claim that 'smoking behavior' (**C**) is causally irrelevant for 'the occurrence or absence of lung cancer' (**E**) this means that we believe that in the two hypothetical populations the incidence of lung cancer is equally high.

An important consequence of this kind of approach is the following:

> […] it could turn out that C is causally irrelevant for E in the population U even though C is not causally irrelevant for E in all individuals in U. […] Population models always average over individuals and, therefore, ignore what might be important differences among individuals. (Giere 1997, pp. 204–205).

It is clear that information on average causal influences is not intended to be directly applicable at the individual level. It only describes general causal tendencies within populations, not the individual paths of disease causation. As Rockhill emphasizes:

> Any average quantitative measure of association discovered by epidemiologists can be consistent with myriad biologic mechanisms in different individuals. An average estimate of causal effect does not suggest that mechanisms or causal paths are homogeneous across individuals or even that the average represents reality for any individual. A relative risk of 1.0, for instance, obviously does not mean that all individuals experience no effect on their risk of disease from exposure. Precisely the opposite could be true; the exposure may increase risk in some persons and reduce the risk in others. (Rockhill 2005, p. 125)

Rockhill (Rockhill et al. (2000); Rockhill (2001)) refers in this context also to what G. Rose (1981) calls "the prevention paradox": the fact that many people have to take precautions to prevent diseases in only a few of them. A large benefit of a preventive measure at the level of a group, often offers very little to each of the individuals in this group. The usefulness of some preventive measures might rightly be questioned on this basis, as is demonstrated by the following example of Rockhill et al. (2000). Women at high individual risk of breast cancer were, on the basis of a study published in 1998, advised to consider taking the chemo-preventive agent tamoxifen. Researchers found out that tamoxifen reduced the risk of breast cancer by approximately 50%. Although this is a large benefit at the group level, recklessly relying on this knowledge in a general preventive measure would offer very little at the individual level. To make things precise: if 100 women with an estimated 5-year risk of 0.04 take tamoxifen, then two instead of four of these women will develop breast cancer. Meanwhile, for this effect to occur, 96 women who would also remain free of breast cancer without tamoxifen, should expose themselves to increased risk of the adverse outcomes associated with tamoxifen. Hence, although all these women would see their high individual risk for breast cancer halved, only two individuals would profit from this preventive measure, and 96 others will have taken the risks of the adverse effects while it will turn out to have been needless.

As I said earlier, epidemiologists are themselves not always clear about the limited applicability of the knowledge they gain. Epidemiological findings are communicated more and more as if they are directly and easily translatable to the individual level. According to Rockhill (2005), the hope to find the ultimate (genetic) "universally necessary component causes" of chronic diseases grows within epidemiology and leads to the aspiration of individualized prevention. Epidemiological results are also via the media often communicated as if they form a reliable basis for individual prevention of diseases. All this does not tally with the methods used in epidemiology and denies the particularity of epidemiological research. The discussion on 'black box epidemiology' (or 'risk factor epidemiology') is also related to this problem. Skrabanek (1994) asserted that risk factor epidemiology does not contribute to science and public health, precisely because it concludes to causal relations without linking these findings to underlying biological mechanisms that fully explain the development of the disease. Others (Savitz 1994; Greenland et al. 2004) have, rightly, defended risk factor epidemiology:

> The ability of epidemiologists to conduct true "black box" studies relating some aspect of the external environment to disease patterns in human populations is a unique virtue of the discipline. Even without a clear understanding of mechanism, such observations may provide the basis to modify exposures in order to prevent disease. (Savitz 1994, p. 550)

Skrabanek's problems with black box epidemiology in fact result from his too high expectations of epidemiology. As Savitz (1994) and Greenland et al. (2004) argue, it is precisely the specificity of epidemiology to study the relations between exposures and diseases in this 'black box' way. The results of such studies can be very important: to help us decide on public health matters when detailed knowledge from other biomedical disciplines is missing; to open our eyes for possible explanations or shortcomings in the existing biological explanations; to help in formulating causal hypotheses that are further testable by other biomedical disciplines; etcetera. Further, epidemiological research results should not be treated in isolation. For deciding on public health measures, the evidence from multiple disciplines can be brought together. It is indeed necessary to be aware of the limitations of epidemiologic methods and to take epidemiologic results for what they are: indicators of causal relations at the population level. Indeed, the vast number of results on risk factors for chronic diseases is only important from a population point of view and cannot be used as a basis for predictions about individual disease courses and even less as a worthwhile screening test for disease development (Rockhill 2005; Wald et al. 1999):

> For a risk factor or risk marker to serve as a useful discriminatory tool at the individual level (in terms of accurately segregating individuals into those who will and those who will not get a disease), we need relative risks or odds ratios much greater than usually seen in epidemiology, greater than 50 or so. Genetic

> mutations known to be associated with very large disease risks are found in only a small proportion of the population and account for a relatively small proportion of cases. Furthermore, it appears likely that even some of the high estimates of risks with genetic mutations are overestimated. (Rockhill 2005, pp. 125–126)

There are a few exceptions in which individual discriminatory accuracy is quite high and individualized prevention is appropriate. This is, for example, the case with Human Papillomavirus as a risk factor for cervical cancer. Epidemiologists nonetheless rarely find such associations with extremely high relative risks that can guarantee successful predictions and interventions at the individual level. Further, when epidemiologists are confronted with such strong associations, it will be clear enough that individualized prevention might be appropriate in these cases.

To sum up, apart from some exceptions in which relative risks are very high, the importance of epidemiological findings lies at the population level. As Rockhill (2005, p. 126) states: "The focus on individual risk and individualized prevention is inappropriately strong in our discipline, given the more common situation of poor risk discrimination." It does not lie within the goals and skills of epidemiology to predict individual disease courses. Nonetheless, good epidemiological research delivers causal information that is useful given the public health goals. And of course, epidemiology is only one of the biomedical disciplines, and other disciplines can complement, clarify, reject or correct epidemiological findings.

Metaphysical presuppositions

In Sect. 3, I joined in Beverly Rockhill's critical assessment of individualized prevention on the basis of risk factor calculations (Rockhill et al. 1998, 2000; Rockhill 2001, 2005). Rockhill (2005) refers to the foundational models of causation in epidemiology as the wrongdoers. Specifically, she refers to the model of Kenneth Rothman as such a model of disease causation that induces epidemiologists' "obsession with individual risk and individual susceptibility" (Rockhill 2005, p. 127). Let us take a closer look at this model.

Although Kenneth Rothman's view on disease causation is not based on Mackie's (1974) philosophical analysis of causation, their views are surprisingly similar. Rothman's definition of 'a cause of a disease' (this is, what we call 'a cause') is, for example, analogous to Mackie's definition of an INUS-condition:

> We can define a cause of a specific disease event as an antecedent event, condition, or characteristic that was necessary for the occurrence of the disease at the moment it occurred, given that other conditions are fixed. In other words, a cause of a disease event is an event, condition, or characteristic that preceded the disease event and without which the disease event either would not have occurred at all or would not have occurred until some later time. (Rothman and Greenland 2005, p. S144)

Further, Rothman defines a 'sufficient cause' (cf. Mackie's minimal sufficient condition):

> A "sufficient cause" which means a complete causal mechanism, can be defined as a set of minimal conditions and events that inevitably produce disease; "minimal" implies that all of the conditions or events are necessary to that occurrence. In disease etiology, the completion of a sufficient cause may be considered equivalent to the onset of disease (… onset of the earliest stage of the disease process, rather than the onset of signs or symptoms.) For biological effects, most and sometimes all of the components of a sufficient cause are unknown. (Rothman and Greenland 2005, p. S144)

Further, from Rothman's quote that follows below, it is clear that Rothman interprets what we generally call 'the cause' of a disease in the same way as Mackie interprets an INUS-condition, namely as a condition which is not necessary in general, but a necessary factor in view of a given set of circumstances.

By consequence, Rothman's description of disease causation can account for the multicausality of disease causation: a given disease can be caused by more than one sufficient cause, each of them involving the joint action of a multitude of component causes. Maybe every individual disease results from a (slightly) different constellation of component causes. It is impossible to describe for every individual case the precise combination of component causes which have led to the disease. Consequently, as Rothman emphasized, "most identified causes are neither necessary nor sufficient to produce disease" (Rothman and Greenland 2005, p. S145). But from a pragmatic point of view this does not matter:

> a cause need not be either necessary or sufficient for its removal to result in disease prevention. If a component cause that is neither necessary nor sufficient is blocked, a substantial amount of disease may be prevented. That the cause is not necessary implies that some disease may still occur after the cause is blocked, but a component cause will nevertheless be a necessary cause for some of the cases that occur. That the component cause is not sufficient implies that other component causes must interact with it to

> produce the disease, and that blocking any of them would result in prevention of some cases of disease. (Rothman and Greenland 2005, p. S145)

Mackie's (1974) aim in developing his theory of causation was to clarify the relation between the way we talk about causes and causation as it is in the world. Kenneth Rothman had a similar aim in trying to clarify how we (should) speak about disease causation by "bridg[ing] the gap between metaphysical notions of cause and basic epidemiological parameters" (Rothman 1976, p. 90). In other words, his model tried to show how metaphysical presuppositions about the causation of disease (hence, as it happens in the world, in casu, at the individual level) are related to epidemiological parameters. Rockhill's criticism that this causal model forms the basis for the tendency to individualized prevention in epidemiology is thus based on confusion. Rothman was not primarily concerned with 'causation' in epidemiological practice. Epidemiologists should learn to see the difference which Rothman precisely tried to clarify, namely between metaphysical presuppositions about causation as it happens at the individual level and the population approach in epidemiological practice.

Rothman was further criticized for giving a deterministic account of disease causation (Parascandola and Weed 2001), while biological processes might behave probabilistic. However, Parascandola and Weed (2001) argue themselves that a simple fix allows for a probabilistic interpretation of Rothman's model. One should then think about the component causes as contributing together to the probability of the effect instead of being together sufficient for the effect. The absence of a component will then decrease the probability of the effect to occur.

Confusion in epidemiological papers on 'causation'

The distinction between the question of a useful concept of 'cause' for epidemiological practice and the question of the metaphysical presuppositions of causal concepts in epidemiology is not often clearly made in epidemiologists' theoretical writings on 'causation'. Clearly setting the former question apart from the latter would nonetheless greatly improve the lucidity of the debate. Before showing in this section how the confusion is present in some epidemiological papers, I first give a general overview of different kinds of papers on causation appearing in epidemiological journals:

- Papers purely describing the methods used in epidemiological practice, most often without questioning the concept of cause involved (e.g. Maldonado and Greenland (2002) on the counterfactual model). These papers are so clearly related to epidemiological practice that the confusion does not play.
- Papers discussing or defending certain specific methods of causal inference or concepts of cause for epidemiology. Different causal concepts are often questioned in this kind of papers and weighed against one another. This kind of papers seems most liable for confusing the question of useful concepts with the question of metaphysical presuppositions in their criticisms (e.g. Parascandola and Weed 2001; Olsen 2003).
- Papers describing a general metaphysical point of view on disease causation of which the author thinks it should underlie epidemiologists' reasonings (e.g. Rothman 1976; Rothman and Greenland 2005). The biggest danger for confusion in this case comes from authors commenting on this kind of papers and mistaking them as papers describing a method for epidemiological practice.
- Papers investigating the metaphysical presuppositions of one specific method of causal inference (e.g. Dawid 2000). Although these papers clearly concern metaphysical presuppositions of a certain method, they are prone to confusing both questions by taking problems in these metaphysical presuppositions as a straightforward reason to reject the method, although the latter might be very useful in practice.

Kenneth J. Rothman

As explained earlier, Rothman (Rothman 1976; Rothman and Greenland 2005) is explicit about his goal in writing his papers on disease causation. He wants to give a metaphysical description of disease causation that can clarify how the reality of disease causation is linked to the results of the statistical methods used in epidemiological practice. In other words, he clearly discerns the two questions in order to be able to shed light on the link between their answers. As argued earlier, Rothman's aims are not always interpreted as such. Not only Rockhill (2005) overlooks the fact that Rothman is not intending to propose new methods for epidemiological practice. Also Olsen (2003), for example, overlooks Rothman's proper aims in his defence of Rothman's view. Olsen explicitly states to be searching for a "*useful* concept of causation in epidemiology" (2003, p. 86, my emphasis) and defends Rothman's deterministic view as the appropriate basis for gaining epidemiological knowledge. Doing this, Olsen confuses the two questions. He defends Rothman's view as if it is one answering the question of a useful concept, which it is not. He therefore also wrongly maintains that epidemiologists arguing for a probabilistic approach to causation in scientific practice should reject Rothman's view. From my analysis it should nonetheless be clear that one can hold on to Rothman's deterministic view as an answer to the question for

metaphysical presuppositions, but meanwhile hold on to a probabilistic approach for scientific practice. This comment on the confusion in Olsen's argumentation nonetheless does not imply that Olsen should anyhow be wrong in arguing for a deterministic approach in scientific practice. He might have good (pragmatic) reasons for that, but these will differ from Rothman's arguments for a deterministic description of disease causation as it is in the world.

Parascandola and Weed

Parascandola and Weed (2001) are very unclear on their goals in their popular paper on disease causation. They seem to intend to answer both the question of a useful concept and the question of the metaphysical presuppositions. On the one hand, they say to "make a recommendation about what type of causal definition best meets the goals of the discipline of epidemiology" (Parascandola and Weed 2001, p. 905). On the other hand, they are stating that the focus will be on the ontological nature of causation. However, the answers to both questions intertangle in an unjustified way. Most importantly, they use their central metaphysical argument—namely that biological processes are probably indeterministic and hence a concept of cause for epidemiology cannot presuppose determinism—not only as an assertion relating to metaphysical presuppositions, but also as an argument for stating that a probabilistic concept is the most useful for epidemiological practice. Although a probabilistic concept of cause might indeed be the most useful in practice (cf. Sect. 3), the argument used by Parascandola and Weed confuses the need for a probabilistic approach at the individual level (because of the *metaphysical* argument that biological processes are indeterministic) with the need for a probabilistic approach at the aggregate level (because of the *practical* argument that one should average over the population to get useful causal information in epidemiology). The metaphysical presupposition of determinism is also the argument on the basis of which they reject alternative concepts of 'cause'. That some of these causal concepts and the methods of causal inference based on them may also be useful in practice for pragmatic reasons, is not at all considered by Parascandola and Weed (2001).

Parascandola and Weed formulate their answers to both science-based questions on the basis of their views on the right metaphysical presuppositions of the causal concepts and hence give no attention to the pragmatic aspects of a useful concept of cause for epidemiological practice. This would not form a problem if they were clearly and only writing about the metaphysical presuppositions of different possible causal concepts for epidemiology. However, given that they mix up both questions and their answers in support of their own favoured view, their arguments lack cogency.

A. P. Dawid

Dawid (2000) carried out thorough research into the metaphysical presuppositions of the counterfactual model. His criticisms, although not only directed to the field of epidemiology, have also influenced the debate on causation in epidemiology. Dawid argued that the counterfactual definition of causation does not form a good basis for causal inference in epidemiology because of the unobservability of counterfactuals. An example of this unobservability is simply the following: the same individual cannot be observed in the same circumstances as a smoker and a non-smoker. This means one should work with substitutions, for example by comparing a group of individuals who smoke with another group of individuals of comparable age, sex, health, etc. who do not smoke. As Dawid argues, the problem with this strategy is that the outcome of the causal relations are presupposed to be deterministic at the individual level because one does not take into account unidentified parameters which can influence the outcome in the counterfactual situation for which one makes use of substitutions. Dawid maintains that one should be able to observe the relevant aspects of the actual world ànd the relevant aspects of the counterfactual world to make an acceptable comparison between the outcomes. Since this is not possible and the counterfactual model therefore leans on unobservable and untestable suppositions regarding the counterfactual situation, it should be rejected according to Dawid. With his analysis, Dawid performed good work in making explicit the metaphysical presuppositions of the counterfactual model often used by epidemiologists. It is indeed important for researchers using this model to be aware of shortcomings of its metaphysical presuppositions. In fact, Dawid's criticism on the counterfactual model makes it very clear why epidemiological results on averages cannot be translated to the level of the individual. However, if one uses this model as a basis for research at the population level, the results might be very useful. Hence, the shortcomings in the metaphysical presuppositions of the counterfactual model do not form a thorough reason to reject the model as a useful tool for scientific practice. Dawid nonetheless does reject the model in the whole because of its metaphysical presuppositions. Doing this, he does not recognize the role of pragmatics in scientific practice. Since it is useful to be aware of problems in the metaphysical presuppositions, Dawid's analysis forms nonetheless an important contribution to the debate.

Conclusion

In this paper I have argued for the importance of a science-based approach in the philosophy of causation, and for the

distinction within this approach between the question of useful causal concepts given the specific goals and methods of a specific scientific discipline, and the question of the metaphysical presuppositions underlying reasoning and research on causal relations in the discipline. I have been looking at the discussion on causation in the field of epidemiology to argue this. I demonstrated that epidemiologists writing on causation could indeed add to the lucidity of their debate by making the distinction. Not only is it important to think about which causal concepts are useful for epidemiological practice, one should also gain knowledge on the metaphysical presuppositions of the causal concepts used for causal inference in epidemiology to become aware of the limitations of methods used to gather causal information.

Given that scientific research is typified by pragmatics, the central question is probably not "what are the useful causal concepts for the discipline and are the underlying metaphysical presuppositions correct?", but rather "are the results of our favoured scientific practices to gain causal information interpreted in the right way, given the possible shortcomings in their metaphysical presuppositions?" As Rockhill (2005) mentioned, it is important to communicate our scientific results in a justified way to the public, also via the media. This means that we should teach the public about, for example, the values and the shortcomings of average statistical results and their relation to the development of disease in individuals. An important precondition for being able to do this is, of course, that epidemiologists themselves are very well aware of, and very clear about, the strengths and limits of epidemiological findings.

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