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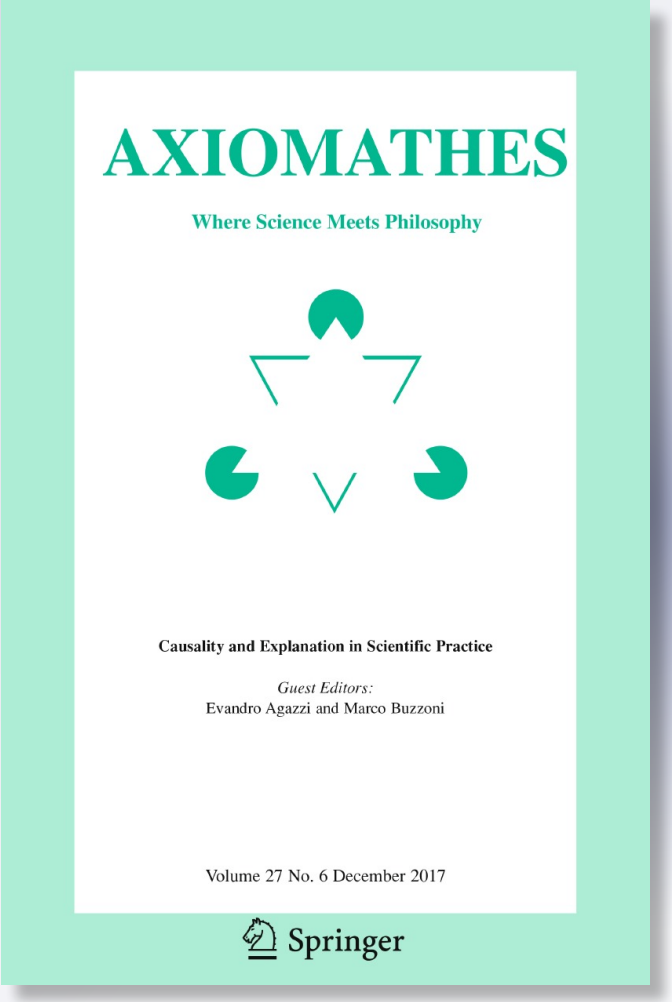

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ORIGINAL PAPER

Comparative Causation at Multiple Levels and Across Scientific Disciplines

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Abstract In this paper, we analyse the fruitfulness of Ronald Giere's *comparative model for causation in populations*. While the original model was primarily developed to capture the meaning of causal claims in the biomedical and health sciences, we want to show that the model is not only useful in these domains, but can also fruitfully be applied to other scientific domains. Specifically, we demonstrate that the model is fruitful for characterizing the meaning of causal claims found in classical genetics, epidemiology and electoral sociology. Additionally, we propose an adapted comparative model which is needed to get a grip on higher level causal claims. We show that such claims are present in population genetics, (environmental) epidemiology and electoral systems research. We conclude with some reflections on the implications of our findings for the issue of causal pluralism.

Keywords Causation in epidemiology · Causation in genetics · Causation in political science · Comparative model of causation · Ronald Giere

1 Introduction

In the last decades of the twentieth century Ronald Giere developed his *comparative model for causation in populations*, the final version of which was published in chapter 7 of Giere (1997). Giere's examples in this chapter and in chapter 8 (which is about evidence for causal claims: randomised experimental design and non-

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experimental studies) come almost exclusively from the biomedical sciences. This is not an accident, because Giere has biomedical claims in mind when he developed the model. For instance, he motivates the model by saying that it is useful for characteristics "studied in the biomedical sciences and those of interest to public health officials" (1997, p. 203).

In this paper we want to show that Giere's model can be fruitfully applied in many scientific domains beyond its original intended scope. It helps us to get a grip on the meaning of causal claims in many scientific disciplines to which Giere himself did not pay much attention (or gave no consideration at all). Before we give more details about what we want to argue, it is important to emphasise that we will distinguish applications of the original comparative model without adaptation from applications of a slightly modified model that we propose. Our two first aims in this paper are the following:

(1) To show that Giere's comparative model in its original version can shed light on the meaning of causal claims in classical genetics.
(2) To show that an adapted comparative model can shed light on the meaning of causal claims in population genetics.

As will become clear in the sections dealing with these aims, population genetics operates at a higher level of reality than classical genetics. This is important for understanding the third and fourth aim of this paper:

(3) To show that biomedical sciences make causal claims at two levels of reality; that many claims at the lower level can be understood by means of Giere's original comparative model, while our adapted model is fruitful for the higher level.
(4) To show that social sciences also make causal claims at two levels of reality; again, many claims at the lower level can be understood by means of Giere's original comparative model, while our adapted model is fruitful for the higher level.

These four aims explain the three key phrases in our title: 'comparative causation' refers to Giere's model, 'multiple levels' to the motivation behind the adaptation of that model that we will propose and 'across scientific disciplines' to our applications beyond the original intended scope.

In Sect. 2 we present Giere's comparative model of causation. Each of the Sects. 3–6 deals with one of the four aims. The last section of the paper contains a brief reflection on causal pluralism: do our findings suggest that causation (in the sciences) has something in common?

2 Ronald Giere's Comparative Model of Causation in Populations

The following definitions constitute the core of Ronald Giere's comparative model of causation in populations:

> 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* $\mathbf{P}_K(E)$. (1997, p. 204)

Giere considers only binary variables. So in his definitions, **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 obtained by changing, for every member of U that exhibits the value Not-C, the value into C. K is the analogous hypothetical population in which all individuals that exhibit C are changed into not-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. causal claims are about finite populations).

An example might clarify this. 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 (like the first two relations). If we claim that "smoking behaviour" (**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 feature of Giere's account is that he defines causation in terms of what would happen in two *hypothetical* populations. In this way the policy relevance of causal claims becomes clear. Why should policy makers want causal knowledge? The hypothetical populations X and K correspond to populations a policy maker may create by means of some direct intervention (e.g. a ban on smoking, a mandatory inoculation,…).

Another important feature of Giere's definitions is that the relevant population is explicitly mentioned. That this is important can be shown by an example that we borrow from Daniel Steel (2008, p. 82). The following causal claims are true:

> Aflatoxin B_1 causes liver cancer in rats.
> Aflatoxin B_1 causes liver cancer in humans.

However, the following claim is false:

> Aflatoxin B_1 causes liver cancer in mice.

Since the population we talk about makes a difference (not only in this case, but in general) it is important that we always explicitly take the intended population into account when analysing the meaning of type level causal claims.

The three relations that Giere defines are jointly exhaustive and mutually exclusive: in a given population, exactly one of them holds for a given **C** and a

given **E**. In our analyses in the next sections we mainly need positive causal factorhood. In line with Giere, we define this as follows:

(PCF) C is a positive causal factor for E in the population U if and only if $\mathbf{P}_X(E) > \mathbf{P}_K(E)$.

This is Giere's definition, but with "if and only if" instead of "whenever". The complementarity of Giere's three definitions (i.e. the fact that the three relations are jointly exhaustive and mutually exclusive) entails that this biconditional formulation is in fact equivalent to the original formulation.[1] Given that we will focus on positive causal factorhood and hardly use the two other relations, it is better for us to use a biconditional formulation (in this way all possible confusion is excluded).

3 Comparative Causation in Classical Genetics

3.1 Introduction

Genetics is usually subdivided into several subfields: classical genetics, population genetics and molecular genetics. Causation in molecular genetics falls outside the scope of this paper. Population genetics is discussed in Sect. 4.

Classical genetics contains many claims about the effect of genes on phenotypes. Here are some examples of the kind of claims, taken from Elrod and Stansfield 2010:

> Black wool of sheep is due to a recessive allele *b* and white wool to its dominant allele *B*. (p. 53)
> Ebony (black) body color in *Drosophila* (fruit fly) is governed by a recessive, mutant allele *e*, while wild-type body color is gray and is governed by a dominant allele e^+. (p. 25)
> Tall tomato plants are produced by the action of a dominant allele *D,* and dwarf plants by its recessive allele *d*. Hairy stems are produced by a dominant gene *H*, and hairless stems by its recessive allele *h.* (p. 48)
> The lack of pigmentation, called albinism, in humans is the result of a recessive allele *a* and normal pigmentation is the result of its dominant allele *A*. (p. 53)

Words indicating a causal relation are underlined.

In what sense are phenotypes the "result of", "due to", "governed by" and "produced by" certain genes? In Sects. 3.2 and 3.3 we show that applying Giere's model gives a plausible answer.

[1] If you believe that C is a positive causal factor for E in a population U, you cannot believe at the same time that C is a negative causal factor for E in U. Then (according to Giere's one-sided definition of negative causal factorhood) you have also to reject that $\mathbf{P}_X(E) < \mathbf{P}_K(E)$. A similar line of reasoning based on the definition of causal irrelevance leads to the rejection of $\mathbf{P}_X(E) = \mathbf{P}_K(E)$. Hence, you are forced to accept that $\mathbf{P}_X(E) > \mathbf{P}_K(E)$: this is the only option left. In this way it can be shown that Giere's definitions that use "whenever" jointly entail that in each case the other direction is also valid.

3.2 Genes as Positive Causal Factors for Phenotypes

Geneticists link specific genes to specific phenotypes. For instance, in the first textbook example above they claim:

Black wool of sheep is due to an allele *b*.
White wool of sheep is due to allele *B*.

We believe that Giere's comparative model captures what is meant by these and similar claims. For instance, we think that the first claim amounts to:

In sheep, having a b gene is a positive causal factor for having black wool.

And the second claim:

In sheep, having a B gene is a positive causal factor for having white wool.

If we apply Giere's comparative model, the meaning of the first claim can be explicated as follows:

In a hypothetical population X where *b* genes are introduced into all sheep that don't have any, more sheep have black wool than in a hypothetical population K where all *b* genes are removed from all sheep.

The meaning of the second claim can be explicated as follows:

In a hypothetical population X where *B* genes are introduced into sheep that don't have any, more sheep have white wool than in a hypothetical population K where all *B* genes are removed from all sheep.

3.3 Plausibility of the Analysis

Why is this a plausible analysis? First, the explications to which Giere's model lead reflect the practical importance of causal knowledge: intervening on genes is a way to change something in phenotypes. As already indicated in Sect. 2, this is a generic property of Giere's model. However, the model has two more interesting features: (1) it allows us to distinguish causation clearly from mere correlation, and (2) it nevertheless forges a link between causation and correlation: causation presupposes correlation. In order to clarify this, we first look at the evidence that geneticists have in favour of the causal claims they put in their textbooks.

We take the PCF claims in Sect. 3.2 as examples. They are supported by empirical generalisations combined with theoretical biological background knowledge. The relevant empirical generalisations are:

Sheep with genotype *BB* or genotype *Bb* are white.
Sheep with genotype *bb* are black.

The relevant biological background knowledge relates to the process of gene expression: DNA determines what RNA is made, this determines protein synthesis

and hence phenotypical traits. This background knowledge supports a belief in causal relations in the direction as it is in the claims in Sect. 3.2: from genes to phenotypes.

More specifically, the two elements of evidence taken together give us a ground to believe that, if we would create a hypothetical population X as characterised above, at least some of the sheep would be black: $\mathbf{P}_X(\text{black}) > 0$. Analogously, we have a ground to believe that if we would create K as defined above, it would contain only white sheep: $\mathbf{P}_K(\text{black}) = 0$. Hence, $\mathbf{P}_X(\text{black}) > \mathbf{P}_K(\text{black})$: having a *b* gene is a positive causal factor for having black wool. For the other PCF claim the situation is similar.

The two empirical generalisations above can be reversed:

White sheep have genotype *BB* or genotype *Bb*.
Black sheep have genotype *bb*.

So there is a correlation between the *B* gene and white wool (and vice versa, because correlations are symmetric) and between the *b* gene and black wool. But there are no causal claims in the opposite direction. Geneticists do *not* claim this:

The presence of recessive alleles *b* is due to black wool.
The presence of dominant alleles *B* is due to white wool.

Definition (PCF) allows us to understand why they reject these claims: they do not believe that by intervening on wool colour (i.e. by painting, bleaching or artificially adding some pigment in the hairs) one can change the genetic constitution of a sheep. More precisely the 'reversed' claims they *don't* believe in are:

In a hypothetical population X where all sheep are made black, more sheep will have a *b* gene than in a hypothetical population K where all sheep are made white.

In a hypothetical population X where all sheep are made white, more sheep will have a *B* gene than in a hypothetical population K where all sheep are made black.

The biological background knowledge mentioned above and our knowledge on how genetic material is (re)produced in DNA replication processes are evidence *against* the 'reversed' causal claims. This is why geneticists reject them.

We have shown that (PCF) allows us to draw a clear distinction between causation and mere correlation. But at the same time causation presupposes a correlation: there has to be a difference between $\mathbf{P}_X(E)$ and $\mathbf{P}_K(E)$, and this difference shows itself in correlations in the real population U. For instance, geneticist will *not* claim the following:

In sheep, foot rot resistance is due to allele B.

That is because they do not believe that the same allele that governs wool colour governs this other hereditary trait. So they do not believe the following:

> In a hypothetical population X where *B* genes are introduced into sheep that don't have any, more sheep are resistant to foot rot than in a hypothetical population K where all *B* genes are removed from all sheep.

They do not believe this because there are no empirical generalisations (like the ones used above) which establish a correlation between the *B/b* locus and foot rot.

3.4 Conclusion

By applying definition (PCF) we have argued that Giere's comparative model of causation in populations can shed light on the meaning of an important type of causal claims in genetics: claims that describe the effect of genes on phenotypes. Our insights are important from an applied philosophy of science perspective: if you want to understand what causation in genetics is, what we have done in Sects. 3.2 and 3.3 is a good start. If our aim in this paper was to get a full grip on causation in classical genetics, we would now continue with an analysis of other types of causal claims one finds in this subdiscipline. In genetics textbooks, we also find claims in which types of mating are the cause variables and the genotypes in the filial population the effect variables. Here are some examples from the book we already used:

> The matings C^RC^W *X* C^RC^R (roan female × red male or roan male × red female) are expected to produce ½ C^RC^R (red) and ½ C^RC^W (roan) progeny. (p. 44)
> The mating C^RC^W *X* C^RC^W (roan x roan) is expected to produce ¼ C^RC^R (red), ½ C^RC^W (roan), and ¼ C^WC^W (white) progeny. (p. 44)

These claims relate to the coat colour of certain breeds of cattle. If you leave out the phrases/words between brackets, these are purely claims about how genotypes in two parental populations that are crossed influence the genotypes in the filial population. The information between brackets tells us what the phenotypic expression of these genotypes is.

We do not analyse causal claims of this type here, because our aim is different. As mentioned in the introduction, we want to show that Giere's model can be fruitfully applied in *many* scientific domains beyond its original intended scope. So rather than staying within the domain of classical genetics, we move on to the next domain: population genetics.

4 Comparative Causation in Population Genetics: Environmental Factors as Causes

4.1 Introduction

Elrod and Stansfield define the subdiscipline of population genetics as follows:

> Population genetics is the study of the patterns of genetic variation at the population level and how changes in these patterns that result from evolutionary forces bring about evolution over time. (2010, p. 248).

A well-known example of such an "evolution over time" that is explained by population genetics is the fact that the melanic form of the peppered moth became dominant in industrial areas in England in the second half of the nineteenth century. This phenomenon is known as industrial melanism. In Sect. 4.2 we give more details about this phenomenon and the explanations that population geneticists offer. In Sect. 4.3 we clarify that, because of some inherent ontological assumptions, Giere's model cannot be applied to population genetics. We reformulate the model in a more abstract way (i.e. with less underlying ontology) so that it can be applied in this domain.

4.2 Industrial Melanism

In their textbook on genetics, Hugh Fletcher and Ivor Hickey summarise the standard explanation adopted by population geneticists as follows:

> *Biston betularia*, the peppered moth, provides a classic example of natural selection. They are nocturnal, and the 'typical' form is grey and speckled, which is camouflaged very well on lichen-covered trees where they rest during daytime. In 1849 the first dark melanic specimen was collected in Manchester, England. Collectors are eager to obtain rarities, so we can assume they were very rare before this. The melanic form is known as 'carbonaria' and is caused by a dominant allele of a single gene (*C*) so typical moths are homozygous *cc*. Carbonaria turned from a collectors' rarity to the common form and by 1895 about 98% of that population of moths was dark. Pollution, principally acid rain from the industrial revolution, had killed the lichens on the trees, exposing the dark bark, which may have been darkened further by soot. The grey typical moths were now not camouflaged as well as the dark Carbonaria. Direct observations and mark-release-recapture experiments supported the hypothesis that predators, in this case birds, were catching a higher proportion of typical than carbonaria in polluted industrial regions. When both types were released in unpolluted rural woods, the situation was reversed and Carbonaria were eaten proportionally faster than typical. (2013, pp. 220–221).

What happened in Manchester also happened in other areas (e.g. around Birmingham) in the same period. Parallel development occurred in other moth species in England, and in other countries (central Europe, northeastern United States) later on (Grant 1985, p. 108).

The unit of analysis of population genetics is often called the "Mendelian population". Elrod and Stansfield define them as follows:

> … a group of sexually reproducing organisms with a relatively close degree of genetic relationship ([…]) residing within defined geographic boundaries wherein interbreeding occurs. (2010, p. 248)

For sake of clarity we prefer to avoid the label 'Mendelian' here (Mendel being one of the founding fathers of classical genetics which was the subject of Sect. 3). We use the term 'local breeding populations' (this term is used by Grant 1985). This is a

goof term because it contains reference to the geographical boundaries as well as the interbreeding mentioned in the definition of Elrod and Stansfield.

One of the questions that population geneticists want to answer in the industrial melanism case, is:

> Why did certain local breeding populations of peppered moths (viz. those in industrial areas) in England became predominantly melanic by the end of the nineteenth century, while the local breeding populations in rural areas remained predominantly speckled grey?

A core ingredient of the answer is:

> Local breeding populations in industrial areas inhabited woods that/which were subject to acid rain; the local breeding populations in rural areas inhabited woods that were not subject to acid rain.

In order for this difference in environment to be relevant for explaining the contrast, we have to accept that there is a causal relation between the presence of acid rain and melanism. The question to be addressed is: what kind of causal relation? Definition (PCF) leads to problems (we show this in Sect. 4.3) so we need an alternative (which is presented in Sect. 4.4).

4.3 Explananda and Causal Relations at Different Levels

Scientific interest in the causes of wool colour in sheep is triggered by a simple observation: some sheep have black wool, others have white wool. This observation may lead to the following question:

> Why are some sheep white while others are black?

The answer is:

> Some sheep carry the *B* gene (i.e. have genotype *BB* or *Bb*), while others do not (i.e. are homozygous *bb*).

This difference is relevant for explaining the white/black wool contrast if the causal relations discussed in Sect. 3 hold.

In general, explanation seeking questions of the following form are important in classical genetics:

> Why do some individuals of a species have phenotypical property A, while others have phenotypical property B?

Wool colour is an example of this. The quotes we gave in Sect. 3.1 provide other examples. The explanandum is a contrast between biological individuals belonging to a given species. To support the answer—which is in terms of other differences between the individuals: genetic differences—we need a positive causal factor claim with the species to which the individuals belong as domain: claims as defined in (PCF).

In population genetics the explananda are at a higher level. The contrast in the melanism case fits into the following general format:

> Why do so some local breeding populations of a given species have property A, while other local breeding populations of the same species have property B?

The answer is given in terms of other differences between the local breeding populations (usually differences in the environment). The explanatory relevance of these differences rests on the presence of a causal relation. Can this be a causal relation as defined in (PCF)? The answer is negative because of inherent ontological assumptions.

In biomedical causal claims, the target population (denoted as U by Giere and in our definition) is often the *whole human population*. This is the case, for instance, if we claim that drinking decaf coffee increases levels of harmful cholesterol or that limb amputation increases the risk of death from heart disease (these are examples from Giere's book). But in many biomedical claims the scope is narrower. For instance, when considering the effect of drinking during pregnancy on the intellectual ability of children at school age, U contains only pregnant women. So biomedical causal claims are often about a *subset of the human species*. What all these biomedical claims have in common is that U is a collection of biological individuals. This is an implicit ontological assumption in Giere's theory. If we apply Giere's ideas to classical genetics—as we have done in Sect. 3—this ontological assumption raises no problems: the causal claims in classical genetics are about populations consisting of all members of a biological species (humans, other animals or plants).

In population genetics the causal claims we need in order to corroborate the explanations are at a higher level. The objects treated as units of analysis in population genetics are populations, not biological individuals. The domains of the causal claims that are made in population genetics are *sets* of such populations (in the same way as the domains of biomedical causal claims and causal claims in classical genetics are sets of biological individuals).

In order to apply the comparative ideas of Giere in population genetics, we have to get rid of the ontological assumption. From a technical point of view, this is possible: all we need to make definition (PCF) work from a formal point of view is a set with designated elements. So we need certain types of "objects" that are treated as units of analysis, and sets of such objects (which constitute the possible domains of causal claims). Because of these considerations we propose an abstract version of PCF:

(PCF-A) C is a positive causal factor for E in the set S if and only if $\mathbf{P}_X(E) > \mathbf{P}_K(E)$.

We deliberately avoid the word 'population' (which we use to refer to a 'collection of biological individuals') in this more abstract definition. Instead we have the set S, which is characterized in an abstract way: a collection of elements. In principle, there is no ontological restriction on what elements S contains. In population genetics, the elements of S are 'local breeding populations' (see Sect. 4.4). In some

of the (biomedical and political science) examples that we discuss further in this paper, the elements of S are 'countries'. In other cases, the elements in S are technical artefacts. In De Bal and Weber (forthcoming) Giere's model is adapted for the application to causal relations in the context of technical problem solving (repairing malfunctioning technical artefacts). In that context, the elements of S are technical artefacts such as car engines, radios, bicycle brakes, etc.

4.4 Positive Causal Factors in Population Genetics

The standard explanation presented in Sect. 4.2 relies on the following causal claim:

> In the set S of all local breeding populations of pepper moths: inhabiting an area subject to acid rain (C) is a positive causal factor for being predominantly melanic (E).

If you do not believe this, you cannot consider acid rain to be a relevant part of the explanation of the contrast (between populations in industrial areas and populations in rural areas) or evolutions over time within one population (from predominantly speckled grey to predominantly melanic). Our definition (PCF-A) allows us to analyse the meaning of this claim. It amounts to the following:

> In a hypothetical set X where all local inbreeding populations of peppered moths are given an acid rain environment, there will be more local breeding populations that are predominantly melanic than in a hypothetical set K where all acid rain environments of local breeding populations are made to disappear.

This analysis is plausible given the evidence (cf. the quote above: mark-release-recapture experiments yield different results in different environments) and the use that is made of the causal claim (it is used as a difference-maker in the explanation).

4.5 Causation in Classical Genetics and Population Genetics: Similarity and Difference

If we compare our results in this section with those of Sect. 3, we see that causation in classical genetics and in population genetics share a formal property: they both involve a *difference* between *two hypothetical sets*. These two hypothetical sets are defined by the cause-variable that is considered, while the difference is related to the effect-variable that is considered. This is possibly a unifying property for causation across (at least some) scientific disciplines. This potential unity will be further discussed in Sect. 7.

Ontologically, causation in classical genetics and causation in population genetics are different. In the first domain, the relevant hypothetical sets are populations of biological individuals. In the second domain, the relevant hypothetical sets are at a higher level: they are sets of local breeding populations.

5 Comparative Causation in the Biomedical Sciences

5.1 Introduction

As we already explained in the introduction of this article, Ronald Giere developed his comparative model for causation in populations with biomedical claims in mind. Therefore, it is no surprise that the model is well-suited for characterizing many causal claims in biomedical sciences such as epidemiology. In Sect. 5.2 we use an epidemiological claim concerning the causes of breast cancer to demonstrate this fruitfulness. However, part of the research into the causes of breast cancer is situated at a higher level. In Sect. 5.3 we argue that we need the adapted model which we proposed in Sect. 4.3 to characterize the causal claims at this higher level.

5.2 Positive Causal Factors in Epidemiology: The Lower Level

It is well known that a woman's age at first full-time pregnancy influences the risk of breast cancer. For instance, Layde et al. (1989) write:

> Compared with women who had their first full-term pregnancy under age 18, women with later ages at first full-term pregnancy had increasing risks of breast cancer. (p. 966).

Translated into our terminology, these authors claim the following:

> In the population of women with at least one child, having the first full-term pregnancy at 18 or older is a positive causal factor for breast cancer.

This is at the level of reality which Giere had in mind: a population of biological individuals. Definition (PCF) can be applied. According to this definition the meaning of this causal claim can be explicated as follows:

> In a hypothetical set were all women with at least one child are ensured to have their first full-time pregnancy at 18 years or older, the incidence of breast cancer is higher than in a hypothetical population K where all women with at least one child are ensured to have their first full-time pregnancy below the age of 18 years.

The meaning of many typical biomedical causal claims can be explicated in the same way. Consider e.g. the examples that we already mentioned in Sects. 2 and 4: smoking and lung cancer, decaf coffee and harmful cholesterol, limb amputation and death from heart disease.

5.3 Positive Causal Factors in Epidemiology: The Higher Level

Epidemiologists also make claims at a higher level. Our example here is taken from Garland et al. (1990). The units they use are counties in the U.S. They claim that the amount of sunlight (average over 365 days) has an effect on the mortality rates from breast cancer in strongly urbanised counties (in which 90% of the population lives in cities; examples are New York, Los Angeles, Chicago, but also smaller ones like

Honolulu and Lexington). In counties that are 'very sunny' (average daily solar radiation more than 445 cal/cm^2) such as Honolulu, Phoenix and Las Vegas the mortality rate from breast cancer (per 100.000 inhabitants, adjusted for age) is 'very low' (below 23) while in less sunny counties the mortality rates are higher. So we have:

> In the set of U.S. counties: being very sunny is a positive causal factor for having a low breast cancer mortality rate.

According to definition (PCF-A) this means:

> In a hypothetical set X where all U.S. counties would be made very sunny, there would be more counties with a very low breast cancer mortality rate, than in a hypothetical set K in which all U.S. counties would be made 'less sunny'.

'Less sunny' roughly means between 250 and 350 cal/cm^2 average daily solar radiation. The claim is not about the characteristics of different individuals within a certain population, but about the characteristics of different geographical places in the US. So we need (PCF-A) to characterize Garland et al.'s causal claim.

6 Causal Claims in Political Science

6.1 Introduction

The first aim of this section is to show that also the social sciences make causal claims at two levels of reality, and that they do have to make these claims at two levels because they deal with explananda at different levels. Our examples relate to political elections.

If a social scientist studies elections one possible aim is to find out "who votes for whom". This may be called 'electoral sociology' and starts from the fact that people vote for different political parties. Explanation-seeking questions of the following type are omnipresent in such investigations:

> Why did (in country X in year Y) some people vote for political party A while others did vote for party B or C or …?

At a higher level, social scientists may study "electoral systems". An important question in this type of research is the following:

> Why do some democratic countries have a multi-party system, while other democratic countries have a two-party system?

Answers for questions of the first type can be given in terms of different properties that *voters* may have. Answers to questions of the second type can be given in terms of differences between *countries*. In both cases the explanatory relevance of the differences pointed at in the explanans has to be corroborated by means of a causal claim. Because of the multiple levels that occur in the explananda (properties of

voters vs. properties of democratic countries) the causal claims are also situated at multiple levels.

In Sect. 6.2 we will show that Giere's original comparative model can capture the meaning of causal claims in what we have labelled 'electoral sociology'. This should be no surprise: the claims are about sets of voters, i.e. about populations (interpreted as 'collections of biological individuals'). In Sect. 6.3 we show that our adapted model of (definition (PCF-A)) is fruitful for the higher level.

6.2 Electoral Sociology

Our example is drawn from Chapter 7 of the book *The Rise of New Labour. Party Policies and Voter Choices* (Heath et al. 2001). This book uses data from the 'British Election Studies' which started after the 1964 general election and are since then held immediately after every general election in the UK (see http://www.britishelectionstudy.com/). In order to understand the example we give, it is important that throughout the book the authors divide the electorate into five social groups: 'petite bourgeoisie' (small employers (less than 25 employees) and own account workers), 'salariat' (managerial and professional occupations), 'routine non-manual class' (ancillary professional and administrative occupations), 'working class' (occupations with routine manual labour and technical occupations).

In the elections between 1974 and 1992, the Conservative Party got between 65 and 77% of the votes of voters belonging to the petite bourgeoisie. Their overall result (all categories) never exceeded 50% (it varied from 35 till 47%). In the 1997 elections the Conservative Party got only 42% of the petite bourgeoisie votes, but that is still much more than their average result in that year (30%). These data support the following causal claim:

> In the U.K. electorate, being petite bourgeoisie member is a positive causal factor for voting for the Conservative Party.

According to our definition (PCF) this means:

> In a hypothetical population X of U.K. voters in which everyone is turned into a petite bourgeoisie member, there are more Conservative Party voters than in a hypothetical population K of U.K. voters in which occupations are intervened upon so that no one belongs to the petite bourgeoisie category.

Similar causal claims can be made with other social categories as cause variable and/or the Labour Party as effect variable. They all belong to electoral sociology. Their domain is the population of U.K. voters, which is a collection of biological individuals. Because of this ontological situation, (PCF) can be applied.

6.3 Maurice Duverger on Electoral Systems

The example we use here is a cluster of three interrelated causal claims, one of which is known in political science as 'Duverger's Law'. Kenneth Benoit introduces it as follows:

> Among students of electoral systems, there is no better-known, more investigated, nor widely cited proposition than the relationship between plurality electoral laws and two-party systems known as Duverger's Law. Since its publication more than a half-century ago in *Political Parties* (1959), hundreds of articles, books, and papers have been written to elaborate the workings of Duverger's propositions. This growing literature has produced numerous empirical studies to explain how electoral systems and changes in electoral rules influence the number of political parties which compete for and win office. (2006, p. 69)

Benoit quotes Duverger literally:

> The simple-majority single-ballot system favours the two-party system. (2006, p. 70)

Note the use of the word "favour", which indicates a positive causal relation (see below). In a simple-majority single-ballot system there is one member of parliament to be elected in each voting district. The candidate who gets more votes than any other candidate is elected (even if there is no majority, i.e. the candidate's score is less than 50%). Duverger considers two other systems: the majority system with a second-round runoff (if no candidate receives more than 50% of the initial votes, there is a second round with the top-two candidates) and proportional representation (multiple members of parliament for each district; seats allocated based on percentage of votes for each political party). Duverger's claims about these systems (cf. Benoit 2006, p. 70) are:

> The majority system with a second-round runoff favours multi-partism.

> Proportional representation favours multi-partism.

These are the other two causal claims in the cluster that we consider.

Duverger performed an extensive comparative study of the relation between electoral systems and number of parties. His evidence supports the claim that there is a correlation:

> In countries with a two-party system, the simple-majority single-ballot system occurs more than on average in all democratic countries. Conversely, the probability that a country with a simple-majority single-ballot system has a two-party-system is higher than the probability of two-party systems in all democratic countries.

Duverger brings in a causal direction by invoking two social mechanisms, which he labels 'the mechanical effect' and 'the psychological factor':

> The mechanical effect of electoral systems describes how the electoral rules constrain the manner in which votes are converted into seats, while the psychological factor deals with the shaping of voter (and party) responses in anticipation of the electoral law's mechanical constraints. (Benoit 2006, p. 72)

The domain that Duverger and his fellow political scientists are talking about in this case is the set of all democratic countries. (PCF) cannot be applied, but (PCF-A) can

be. By means of the latter definition, the meaning of the first causal claim (i.e. "Duverger's law") can be explicated as follows:

> If all democratic countries would be given a simple-majority single-ballot system, there would be more countries with a two party system than if all democratic countries would have proportional representation.

Similarly, the third claim in our cluster can be explicated as follows:

> If all democratic countries would be given a proportional representation system, there would be more countries with a multi-party system than if all democratic countries would have a simple-majority single-ballot system.

7 Causal Pluralism

Do our findings suggest that causation has something in common across scientific disciplines? At an abstract level the same type of causation is present in all the cases we have analysed: causal claims are claims about a difference between two hypothetical sets. So the analyses in the preceding sections indeed suggest that causal claims *in the sciences* always involve some *kind of* positive difference making. The words we have put in italics indicate important disclaimers:

(1) Causal claims in the sciences are type-level causal claims: they are about causal dependencies between types of properties (represented by variables). We have not considered causation at token level (causation as a relation between particular events).
(2) The 'kind of' points at the fact that positive difference-making can be and is instantiated in several ways.

Let us elaborate (2) a bit. In this paper we have proposed two ways in which the idea of positive difference-making can be developed: (PCF) and (PCF-A). Both definitions are applicable only if we have two discrete variables. For other cases we need other definitions (e.g., the potential outcome model as presented in Morgan and Winship 2007 (pp. 31–37) is useful for cases where the effect variable is continuous). In our view, what we need to analyse the meaning of causal claims in the sciences is a *system of* definitions. Our hunch is that these definitions will instantiate a common core idea (viz. a positive difference between two hypothetical sets). But we will need a lot of specific definitions of various types of causation in order to get grip on the meaning of causal claims in all scientific disciplines. Because of these multitude of definitions, we consider ourselves causal pluralists, though we conjecture that there may be something in common in all type-level causation.

8 Conclusion

We have demonstrated the usefulness of Ronald Giere's comparative model for causation in populations in diverging domains of science. However, we have also shown that the model is too restricted to capture the meaning of higher-level causal

claims. We argued that this can be solved by adopting an abstract version of definition (PCF): definition (PCF-A), in which we get rid of Giere's implicit ontological assumption that the domain of a causal claim is a population of biological individuals. By applying (PCF-A), we have characterized higher-level causal claims that are present in various scientific disciplines. Finally, we have explained what our results may mean for the causal monism/causal pluralism debate.

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