

that we all of us know that there is such a law, though he admits that it cannot be demonstrated or otherwise justified. But, he maintains, such justification is not necessary. "I do not know *how* WE know that things are as they are because they were as they were. But WE do know it."

The last five authors quoted are at one in asserting an alleged law of causation which turns out to be simply Kant's mule. None of them has troubled to ask, any more than Kant asked, whether the two propositions of which this hybrid is composed are compatible. No wonder that opinions differ as to "how we know" such a piece of nonsense, or whether "we" know it at all.

The last four have agreed in using a locution to which I have ventured to call attention by printing the word WE in capitals. This "indeterminate we" is so common among philosophers that a grammarian might call it *σχῆμα φιλοσοφικόν*, and Bacon might have classed it among *the idola theatri*. The word "we" refers to a group or society of

persons among whom the speaker includes himself. It is not used by a man who is thinking clearly unless he is prepared to answer the question "what group is this of which you are speaking, and what are its limits?" Philosophers have got into the habit of using it when in fact they are quoting beliefs of their own sect, but when they imagine themselves to be quoting beliefs common to the entire human race, or at least to all such members of it as have the use of reason. Where such language is found, an alert reader will ask "who are YOU?" It would perhaps have surprised any of the four writers I have quoted, to learn that the answer is "Kantianians." So recent is the "idea of causation" which modern philosophical dogmatism takes for granted. So necessary is it, before swallowing whole the traditions which this dogmatism would force down our throats, to inquire not only into their logic but also into their history.

Commentary: How norms make causes

Maria Kronfeldner

Bielefeld University, Abteilung Philosophie, Universitätsstrasse 25, 33615 Bielefeld, Germany.
E-mail: mkronfeldner@uni-bielefeld.de

International Journal of Epidemiology, 2014, 1707–1713
doi: 10.1093/ije/dyu130

Advance Access Publication Date: 30 July 2014



Within the philosophy of science, Robin G. Collingwood's paper on 'The so-called idea of causation',¹ republished (slightly changed) as 'Three senses of the word "cause"' in his *Essay on Metaphysics*,² is not only regarded as an early defence of a manipulationist account of causation, but also as a classic reference for a debate about causal selection. The former discusses what a cause is and the latter why and how we select a subset of the causes of a phenomenon to be included in the explanation of the respective phenomenon. With respect to causal selection, Collingwood has (at least since H.L.A. Hart and Tony Honoré's seminal book *Causation in the Law*),³ been regarded as the defender of controllability as the principle that guides causal selection in contexts that Collingwood named 'practical sciences' (e.g. law or medicine).

This commentary discusses the relevance of Collingwood's control principle in contemporary life sciences and defends the claim that it is *not* the *ability* to control, but the *willingness* to control that often biases us towards some rather than other causes of a phenomenon.

Willingness to control is certainly only one principle that influences causal selection, but it is an important one. It shows how norms make causes.

The problem of causal selection

When we say, for instance, that 'The cause of malaria is the bite of a mosquito', an example Collingwood used, then we know that the bite of a mosquito is only one of the many causal factors involved in an incidence of malaria. In the once fashionable parlance of necessary and sufficient conditions, the mosquito bite is merely what Mackie⁴ called an INUS-condition, i.e. an *insufficient* but *necessary* part of an *unnecessary* but sufficient condition for malaria. Be it malaria or anything else in our world, there will always be more than one cause for a phenomenon. Why then can the bite of a mosquito be termed *the* cause, i.e. the one cause (out of the many) included in an explanation that is partial but explanatory nonetheless? The problem of causal selection is the problem of understanding how

scientists single out some causes for inclusion in an explanation (foregrounding) and relegate others to the background (backgrounding).

Collingwood's control principle

Practical sciences such as medicine are, for Collingwood, research domains that are defined by the aim to produce and to prevent things. They are areas in which *things going wrong* are studied. In such research domains, Collingwood claimed, it is the ability to control a causal factor that guides scientists in selecting between causes. If I am the explainer, the cause that is included in the explanation is 'the thing that I can put right' (p. 303)². For me (or us) the explanatorily relevant cause is the thing that I (or we) can control, in the sense of *putting it right*.

In his example of a car failing to climb the hill,² there is a hill and there is a loose high-tension cable in a car. They are causes for the stoppage of the car. The hill is a cause since 'more power is needed to take a car uphill than to take it along the level'. Whether there is a hill, or not, makes a difference and is thus causally relevant. Yet, as he added, since we usually cannot intervene on hills (e.g. by stamping on them), we usually ignore the hill as a cause when we give an answer to the question 'Why does the car fail to climb the hill?'—and rightfully so, he believed. It would be pointless to quote the hill as a cause of the stoppage of the car and right to call the loose high-tension cable *the* cause, simply because we can intervene in the latter but usually not the former. He concluded that in everyday life and practical sciences, manipulability or controllability (pick your preferred term) is a principle of causal selection that justifiably guides our biased way of dealing with causes. What Collingwood called the principle of 'relativity of causes' (p. 304)² follows: people differ with respect to what they can control and hence differ with respect to their causal explanations.

Unmanipulable causes and (ab)normality

There is a recurring critique that Collingwood's approach has to face (be it as a classic reference of a manipulability account of causation or with respect to the control principle of causal selection): first, we do regard something as a cause and foreground it even though we cannot control it (for a critique along these lines see e.g.^{3,5-7}; on the problem of the (im)possibility of interventions more generally see⁸); second, there are causes we can control and background nonetheless.⁹

In reaction to Collingwood, Hart and Honoré suggested (ab)normality as the more inclusive alternative to controllability: we select as the explanatorily relevant cause(s)

what is—in a given context of occurrence—abnormal (i.e. what actually differs) and put normal factors (potential difference makers) in the background. Abnormal in this context simply means 'being different', given a respective context of occurrence or reference class (pick your preferred approach: see Menzies¹⁰ on the context of occurrence, a term he takes from Gorovitz;¹¹ see Hesslow¹² for reference classes). If the barn on the next street burned down the other night, a standard toy-example for the debate on causal selection, we do not include oxygen in the explanation of the fire because oxygen is normally present. The presence of oxygen is not an explanatorily relevant cause of the fire because, in the context at issue in the example, it did not differ: oxygen was present when the house burned (unusual situation) and when it did not burn (things going on as usual). The normality approach of Hart and Honoré is still influential: Waters¹³ has a contemporary elaboration on the distinction between actual vs mere potential difference makers; Hitchcock and Knobe¹⁴ backed up Hart and Honoré's approach, which relied on ordinary language philosophy, with evidence from experimental philosophy. However, the basic point, as they admit (p. 601),¹⁴ is still the one from Hart and Honoré: we can solve the selection problem by distinguishing between what is normal and what is not.

On the basis of their normality approach, Hart and Honoré wrote the following on controllability: 'What very often brings 'controllability' and cause together is the fact that our motive in looking for the abnormality which "makes the difference" is most often the wish to control it and through it, its sequel' (p. 37)³.

Even though Hart and Honoré were not convinced by Collingwood's control principle and suggested abnormality as alternative, their account is compatible with the idea that we can use the control principle to *select among* the abnormal factors, i.e. to select among a set of actual difference makers. In the example of the hill, the hill and the loose high-tension cable are actual difference makers. In all non-laboratory contexts it will seldom be that there is only one actual difference maker, except in cases in which one partitions one's explanandum so that there is only one difference maker.¹⁵ Furthermore, Hart and Honoré suggested that the wish to control plays a role in causal selection, despite the fact that some of the causes we foreground are beyond the respective ability to control.

I will develop Collingwood's approach in the direction Hart and Honoré suggested: it is the willingness to control (rather than the ability to, or the explicit wish to) that can often account for why certain causal factors are picked out and others relegated to the status of mere conditions, lingering around in the background, fixed and normal.

What is control?

On the one hand, controlling things is making sure that something keeps running as it normally does (by preventing change or reproducing the normal state), because it is supposed to run like this. This is a keep-things-right or set-things-back-to-right kind of control, it is a *conservative* kind of control: you try to prevent something from changing. Yet, controlling things can also mean that we are able to change something in a way we want it to change, we can manipulate it. I call this *forward* control.

If somebody does not care or want to prevent something from changing (conservative control), they will not pay attention to, or even prevent attention to that something, and will background it in causal explanations; if people want to forward control something, they will pay attention to it and include it in causal explanations. This connection between interests, attention and control is the foundation for how norms make causes.

How norms make causes

The thesis that I want to put forward on the basis of the previously made distinctions is the following: norms based on social conventions, technical possibilities and interest can *make* causes in two senses: norms make causes *visible* by defining what we (usually those in majority or power) are willing to control, i.e. willing to set or keep right, which influences whether the cause is foregrounded (in case of forward control) or backgrounded (no care or conservative control); this in turn makes causes *real* by creating or stabilizing what is normal or not, i.e. by creating or stabilizing what will be an actual difference maker. The first sense refers to causal selection itself and the second to a consequent looping effect.

This approach not only allows the answering of the above-mentioned critique of Collingwood, but also the subsuming of Hart and Honoré's normality principle as one kind of control, namely keeping things normal.

In addition to the hill and fire examples, I will use evidence from the history of explaining cancer and generalize the approach developed to the overall history of genetic explanations. The suggested revision of Collingwood's control principle is thus also meant as a contribution to the so-called parity debate, a discussion within the philosophy of biology about why biomedical research so often singles out genes as causes, despite their ontological parity with other causal factors, i.e. despite the fact that we all know that it is always nature and nurture interacting when a trait develops in whatever organism we look at.

Whether the willingness-to-control principle is generalizable to purely theoretical sciences, such as physics, is an

issue not addressed here. Collingwood himself doubted it. I tend to agree. The goals of these sciences are just so different, so that it is unlikely that they work the same way. And even within the practical sciences, which aim at producing as well as preventing, we can insert a wedge between theoretical contexts and practical contexts, since the control principle might be applied in opposite ways: Weber¹⁶ claimed that in theoretical contexts it is the normal that is foregrounded, because what one wants to explain in theoretical contexts of practical sciences is not malfunctioning, i.e. things going wrong, but regular functioning, i.e. things going right.

A new perspective on the fire example

If we go back to the fire example and the question 'Why did the barn on the next street burn the other night?', the answer might be: 'Because kids were playing with matches'. As mentioned above, the fact that oxygen was present is ontologically equally causally relevant (i.e. a cause), but we usually do not include oxygen in our explanations of why a fire occurs. Oxygen is a causal factor that is backgrounded, set to be lingering around in the background as a (so-called) standing condition, analogous to the hill in Collingwood's example. Why? The ability-to-control principle would state: because those who do the explaining cannot control the presence of oxygen. The normality principle would state: because oxygen is ubiquitous, i.e. normally there. It is the abnormal, the thing that differs (e.g. whether there was a lighted match or not), that 'makes the difference' in phenomena such as accidents, diseases, deviant behaviours, i.e. in things not 'going on as usual'.³ In the fire case, the abnormal behaviour comes from the kids, not from the oxygen.

Do we need to decide between the two accounts? No, because we can unite them: we background oxygen neither because it is normal (because we could change that), nor because we cannot control it (because we could change that as well), but because we want to keep oxygen normal (conservative control) or do not care about it. We foreground other causal factors (e.g. the kids' playing with matches) because we do not approve of it and consequently want to intervene (forward control) in it or would at least appreciate the respective intervention.

The general claim is then: even if we cannot practically change something (i.e. control in Collingwood's own terms), as long as we hope—one day—to be able to intervene in it (which includes that we believe in that possibility and would like to intervene), we foreground it and call it a cause, if not *the* cause. A belief in the non-ability to control thus would (or at least should) constrain the willingness to control: if there is no hope whatsoever that we can one day

gain control over a causal factor, it is rather unreasonable to be willing to control it.

If control enters causal selection at all, then it is the willingness to control, not controllability, that biases us towards some causes as the difference maker of our choice. The thing that we are prepared to change goes to the foreground, the thing that we want to stay normal or do not care to intervene on goes to the background. Thus, even if we cannot practically intervene on a causal factor, we might still single it out as the cause, simply because it is what we wish to intervene on (or would accept being intervened on), irrespective of whether somebody already can or not. Factors we are not prepared to intervene on, matters of fact we want (or accept) to stay as normal as they are, will be backgrounded; they are taken for granted or are wilfully ignored.

As said, the claim is: we ignore oxygen as a cause of fire (in the standard contexts of occurrence), i.e. do not care for the presence of oxygen explanatorily, because it is something that we care about in life; we want it to stay normal. Causal ignoring (backgrounding) or causal selection (foregrounding) is therefore not just a matter of facts, but a matter of policy, a matter about what ought to be done or not done, e.g. intervening on the children (or whoever caused the fire) so that they do not do the things they did. The deviant is the devil. This is how norms make causes visible, i.e. included in an explanation, whether the example is children and oxygen, parts of a car and hills in daily life, or genes and environments in biomedical sciences, an example to which I shall turn next.

How is the looping effect working in the fire example? We foreground the kids' behaviour in order to manipulate it—in order to discipline them; if successful, the kids' behaviour will not be (or will less likely be) an actual difference maker for future fires occurring in the neighbourhood. This is how causal selection is part and parcel of us creating the patterns of differences that build the ontological basis from which we can at all select causes to be included in an explanation.

An example from the history of science: shifts in regularity regimes of cancer prevention

According to the historians Robert Proctor¹⁷ and Alexander von Schwerin,¹⁸ the norms about what should stay as it is (and thus be in the background of causal explanations) historically changed with respect to regulatory regimes of cancer prevention in interesting manners. If they are right with their historical claims, we have a less toy-like example that illustrates the just-made points with respect to contemporary life sciences.

During the 'Atomic Age' after World War II, awareness about the carcinogenic effects of nuclear radiation, new chemical substances and air pollutants increased. Well into the 1980s, a dominant political answer (for dealing with the risks the technological progress brought with it) was a regulatory regime that controlled emissions of radiation and substances by setting limit values. This regulatory regime treated individuals as vulnerable, passive objects of irreversible, harmful, mutational effects of carcinogenetic factors penetrating individual bodies. The general regulatory rationale in policy was to *protect the citizen*. What caused cancer? 'The industry!' was the dominant reply. Proctor¹⁷ called this regime 'body victimology'.

The situation slowly changed during the 1980s when, despite new political regulations, two developments came together: (i) more and more scientific evidence became available that there are individual differences in cancer-relevant cell repair mechanisms (pp. 143–9);¹⁸ and (ii) control for the environmental and chemical hazards turned out to be very hard: there were too many of them, predictive value of mutagenic tests were unreliable and 'many of the artificial risk factors were so tightly connected with the demands and benefits of modern life that their removal was impracticable from economic and social standpoints', as Schwerin put it (p. 150).¹⁸ We got used to and dependent on what caused the higher risk of cancer.

As a consequence of (i) and (ii), the vulnerable citizen over time became an active agent to be governed. The new regulatory rationale was 'body machismo', as Proctor¹⁷ called it. The imperative slowly became *to govern the body*: citizens were asked to boost their repair mechanisms, e.g. by eating fresh vegetables and buying products from the health industry, e.g. artificially produced micronutrients. If people got cancer, it was because they did not boost their self-regulation-machinery properly. The cause of cancer was now increasingly regarded to be found in individuals who act in correct or wrong ways; increasingly in the background of the causal attributions was the environment, containing radiation and substances emitted by a growing industry of energy, food and pharmaceutical production that served a consumer culture which people became used to and dependent on.

In the language used by toxicogenetics, as Schwerin reported, the detrimental environmental influences that had once been conceived as 'bullets' hitting passive citizens, became conceived as mere 'stimulus', relegated to the background of political regulation regimes, a mere 'biochemical signal triggering a cascade of molecular reactions' in abnormal human beings (p. 147).¹⁸ A normal human, with her or his evolved repair mechanisms, who took care of herself or himself, would not get cancer. That was the new regulatory slogan. From then onwards, the 'organism was not a ready-

made victim of fateful radioactive or genotoxic disturbances; instead, here was an organism which was built to handle those environmental factors' (p. 148).¹⁸ This organism finally became the 'entrepreneurial self' of the 'new bourgeois lifestyle' of health and sustainability (LoHaS), a 'flexible self that works around the clock to compensate for exposure to altered environments [...] busy handling risk factors' (p. 156),¹⁸ whereas others make big business producing the environmental hazards in the background.

What is the cause of cancer? The environmental hazards? Or individual differences in repair mechanisms? It depends on what you are prepared to politically intervene on: the toxin-emitting industry, or those individuals who mainly have to face those toxins. And as we all know, some people have to face much more of these toxins, which is how people of colour, for instance, end up having higher rates of cancer: a gene for a certain skin colour (rather than a cancer-specific gene in this group of people) leads to social discrimination (racism), which increases the probability of ending up in harmful environments, which in turn increases the probability of cancer.

Generalization: research on genetic explanation in contemporary life sciences

As many in industrialized countries have for a long time now not been prepared to change their consumerism, many in these countries are not prepared to change social inequalities, or other aspects of our shared and non-shared environment. If so, these people will tend to put the causal factor social inequality to the background and rather investigate what difference genes (and other internal-to-the-individual-body causal factors) make. That way *they* (those in power) will not have to change anything about the structure of the respective society, whereas *others* (individuals who exhibit individual differences, i.e. who are not 'normal') will have to.

Geneticization of traits (i.e. explaining something as being due to genes) involves medicalization, because genes are causal factors internal to bodies. When medicalization is involved, financial interests are too. As Gannett (p. 359)⁹ reminded us: 'The United States government has been motivated to fund the Human Genome Project for the sake of the health not only of the American people but of its developing biotechnology industry'. 'Blaming genes', as she states, 'draws society's attention away from unhealthy environments and weakens its commitment to address factors such as poverty, cigarette smoking (and tobacco advertisements), exposure to pollutants, and racism, which all contribute to these diseases' (p. 455)¹⁹ (racism, not race!).

Kitcher made a point similar to that of Gannett, even though it is the risks of the family members of those in power and wealth that define the context:

Those of us who live comfortably worry less that our sons and daughters will suffer from neglect or abuse—we, after all, intend to provide them with safe and nurturing environments. But we are not immune to disaster. The genes may strike, and if they do, all our effort will be in vain. Accordingly, we are very interested in one kind of cause of the diminution in quality of people's lives. Other kinds of causes, environmental factors that wreak havoc with lives, are not (perceived as) our problem (p. 311).²⁰ (cf. (pp. 131–2)²¹)

It is the same 'logic' of willingness to control: there are social reasons for backgrounding environment (inequality as not important to change, or even as important to stay) and there might be further reasons for foregrounding genes (since some people want to have forward control over them).

In addition to these more social aspects, there is also an aspect of our biased way of dealing with genetic causes that is more pertinent to intrinsic scientific interests, and this aspect should be mentioned as well, in order to illustrate the relevance of the willingness-to-control principle for understanding contemporary life sciences. There is a tradition in the parity debate on genetic causation that I call instrumentalist. The instrumentalist claim is that genes are prioritized in explanations of diseases or other traits because in experimental contexts they are better instrumental handles, i.e. technologically more tractable than environmental factors.^{9,20,22–24} The assumption is that we can screen and experimentally intervene in precise ways in genes, but we cannot screen or intervene in the environment in a similar technologically easy way. Genes are tractable and the environment is not. In other words, instrumentalists assume that geneticists have better control (forward and backward) over genes than over the environment, and therefore focus on genes as explainers.

Although this instrumentalist approach to prioritizing genes can be directly derived from Collingwood's control principle (and Collingwood is often mentioned with respect to this), it ignores two important points: first, geneticists focused on genes long before they had such incredible means of experimental control on genes; second, as Gannett (p. 358)⁹ said, there are 'nongenetic factors both internal and external to the organism [that] are amenable to experimental manipulation', and they are backgrounded nonetheless.

The list of environmental factors (relevant for the development of an organism) is certainly indefinitely large; furthermore, there are indeed many environmental factors

that still are (and may always be) beyond our experimental control, partly because the environment is so incredibly more complex than the genome. Thus, the representation of environmental causal factors will often be, as Kitcher (p. 402)²¹ says, characterized by ‘fragility’ rather than tractability. Yet, some environmental factors (e.g. water or oxygen supply in plants) have been easy to control at a time when people did not know anything about genes, a time when genes were purely hypothetical entities. These environmental factors were, however, often simply used to standardize the experiment in order to have a controlled setting for testing the effect of differences in genetic factors. They were controlled in the conservative sense, i.e. used as mere standing conditions, fixed and controlled in the background of the ‘genetic theatre’, stabilizing what’s going on. If you have the means to control the environmental factor, you might still background it; if you don’t have the means to control genetic factors, you might still select them as *the* cause for a disease. It all depends on what you care about and in which sense. And geneticists simply care about genes. It is their job.

The ability to manipulate can therefore not explain the prioritizing that we saw for roughly the first three-quarters of the 20th century, and it cannot explain the prioritizing between equally controllable factors, i.e. in cases where having the means to control something still leaves room for a choice. It is the preparedness to intervene and not the ability to control that guides causal selection. If you hope to get in the future (or have already) the technological means to study (and thus control) a causal factor in the laboratory, or if the other factors (those relegated to the background) should (or could, given your interests) stay as they are, then you will focus on that causal factor that you are willing to intervene on.

What you are prepared to control, as well as what is easy or convenient to intervene on, depends on who you are and what you care about. Geneticists and the pharmaceutical industry care about handles they can (or hope to be able to) intervene on, because they get refutation from that, or money or both, or because they are driven by some theoretical or personal belief in the importance of this or that. Gannett⁹ mentioned the example of the scientist Jerome Lejeune, who not only discovered the genetic cause of Down syndrome (caused by an extra chromosome 21), but went on trying to find other causes via studying the mechanisms by which the disease comes about, given the extra chromosome. His search for nongenetic causal factors was motivated by his opposition to abortion. Whether a baby will be born or not, was not what he was prepared to intervene in. His goal was consequently ‘to find some other “handle” by means of which to intervene in the treat-

ment or prevention of the symptoms associated with Down syndrome’ (p. 367).⁹

Other people might have different interests, be prepared to change different things, and therefore explain things differently. The willingness-to-control principle of causal selection can thus explain relevant agreement in causal explanations as well as disagreements, by reference to agreement or disagreement in what we care about or appreciate being changed (foreground) or care about or accept staying normal (background). Collingwood’s ‘principle of the relativity of causes’ holds, even though on the basis of willingness to control rather than on the basis of controllability itself.

How about the looping effect in these cases from the life sciences? I have space only for the last case, the Down syndrome example: if we foreground the genetic factors and intervene accordingly, then this will change the genetic pattern of difference in the world. This is how norms make causes real. In the case at issue, the change in the pattern of difference is towards an eugenically homogenized world. (It will certainly never be a completely homogenized world since genes are hard to discipline, maybe even harder to discipline than kids.)

Conclusion

Normality and control are two important principles guiding us in our biased way of dealing with causes in practical sciences.

As mentioned, control has a Janus-faced nature: it can mean that you can change something at will or that you are able to keep something on track, i.e. normal. Collingwood referred to control in the first, forward sense only. Hart and Honoré rightly argued that he ignored normality, but without seeing that what they call normality is just one way of controlling things: keeping things normal, a conservative kind of control. The willingness-to-control principle shows that control is the more inclusive principle.

Collingwood’s control principle, which states that people go for the by-you-actually-controllable cause, can be replaced with a willingness-to-control principle that states that people go for causes if these are (or believed to become) manipulable and if it is not against their respective interest to have them manipulated.

The willingness-to-control principle captures how norms make causes. First, norms make causes *visible* in the causal field (i.e. our representation of a causal situation). Representing something is the first step towards a causal explanation. The norms, so to say, act as a frame for the causal picture that we wish to draw. By drawing a line, a frame, we causally select from an ontic causal structure (causal dependence relations in the world) certain parts of the structure we would be willing to change, and vice versa: we put causal factors to the background, not just because what they refer to is statistically normal, but because we want to keep it normal. By keeping it normal, we, secondly, create a looping effect and thus make causes *real*: we stabilize the statistical as well as the normative norm and thus make what is there to select (the ontic causal structure, i.e. the pattern of differences) for later causal explanations in later instances

of causal selection. Collingwood's control principle is thus highly relevant to understanding how norms make causes.

Acknowledgements

This paper would not exist without the generosity and abundance of ideas of Lisa Gannett and Alexander Reutlinger. I owe them many thanks. I am also grateful to the following people and institutions: to Marta Bertolaso, Evelyn Fox Keller, Lauren Ross, Alexander von Schwerin and Ken Waters, who sent, as did Lisa and Alex, interesting and very helpful written comments on a longer version of this paper; to the Center for Philosophy of Science at the University of Pittsburgh (whose Fellow I was during the academic year 2012-13 when this paper started) for being such a stimulating intellectual habitat; to Philip Kitcher, Sandra Mitchell, Ken Schaffner, Jim Woodward, my co-fellows at the Center, and the audiences at the Dalhousie Philosophy Department Colloquium and the Duesseldorf Philosophy of Science Colloquium for the inspiring discussions on matters of causal explanation.

Conflict of interest: None declared.

References

- Collingwood RG. On the so-called idea of causation. *Proc Aristot Soc* 1938;38:85-112.
- Collingwood RG. *An Essay on Metaphysics*. Oxford, UK: Clarendon Press, 1940.
- Hart HLA, Honoré T. *Causation in the Law*. 2nd edn. Oxford, UK: Clarendon Press, 1985.
- Mackie JL. *The Cement of the Universe: A Study of Causation*. Oxford, UK: Clarendon Press, 1974.
- Hausman DM. Causation and experimentation. *Am Philos Q* 1986;23:143-54.
- Hausman DM. Causation, agency, and independence. *Philos Sci* 1997;64:S15-S25.
- Menzies P, Price H. Causation as a secondary quality. *Br Je Philos Sci* 1993;44:187-203.
- Reutlinger A. Getting rid of interventions. *Stud Hist Philos Biol Biomed Sci* 2012;43:787-95.
- Gannett L. What's in a cause? The pragmatic dimensions of genetic explanations. *Biol Philos* 1999;14:349-73.
- Menzies P. *Difference Making in Context. Causation and Counterfactuals*. Cambridge, MA: MIT Press, 2004.
- Gorovitz S. Causal judgements and causal explanations. *J Philos* 1965;62:695-711.
- Hesslow G. The problem of causal selection. In: Hilton DJ (ed). *Contemporary Science and Natural Explanation. Common Sense Conceptions of Causality*. Hemel Hempstead, UK: Harvester Press, 1988.
- Waters KC. Causes that make a difference. *J Philos*. 2007;104: 551-79.
- Hitchcock C, Knobe J. Cause and norm. *J Philos* 2009;106: 587-612.
- Kronfeldner, M. Die epistemische Fragmentierung des Menschen: Wie der Mensch zwischen Natur und Kultur verschwindet. In: Hügli, Anton et al (eds.) *Die anthropologische Wende. Studia philosophica* 72 (pp. 287-313). Basel: Schwabe. 2013.
- Weber M. Causal selection vs causal parity in biology: relevant counterfactuals and biologically normal interventions. In: Waters CK, Travisano M, Woodward J (eds). *Philosophical Perspectives on Causal Reasoning in Biology*. Minneapolis, MN: University of Minnesota Press, forthcoming.
- Proctor R. *Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer*. New York: Basic Books, 1995.
- von Schwerin A. From the Atomic Age to anti-aging: Shaping the surface between the environment and the organism. In: Grote M, Stadler M (eds). *Membranes Surfaces Boundaries: Interstices in the History of Science, Technology and Culture*. Berlin: MPIWG, 2011.
- Gannett L. Genes and society. In: Ruse M (ed). *The Oxford Handbook of Philosophy of Biology*. Oxford, UK: Oxford University Press, 2008.
- Kitcher P. *The Lives to Come: The Genetic Revolution and Human Possibilities*. New York: Simon & Schuster, 1996.
- Kitcher P. *Science, Truth and Democracy*. New York: Oxford University Press, 2001.
- Kitcher P. Battling the undead: how (and how not) to resist genetic determinism. In: Singh R, Krimbas C, Paul D, Beatty J (eds). *Thinking About Evolution: Historical, Philosophical and Political Perspectives*. Cambridge, UK: Cambridge University Press, 2001.
- Schaffner KF. Genes, behavior, and developmental emergentism: One process, indivisible? *Philos Sci* 1998;65:209-52.
- Keller EF. *The Century of the Gene*. Cambridge, MA: Harvard University Press, 2000.