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CAUSAL PLURALISM IN THE LIFE SCIENCES

Kolja Ehrenstein

Reviewed by
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Causal Pluralism in the Life Sciences: A Journey along the Frontiers of Conceptual Plurality[□]

Kolja Ehrenstein

Cham: Springer, 2022, £89.99

ISBN 9783030879440

Cite as:

Baxter, J. [2023]: 'Kolja Ehrenstein's *Causal Pluralism in the Life Sciences*', *BJPS Review of Books*, 2023

The last century of philosophical work has seen a proliferation of competing causal theories: regularity theory, probabilistic causality, counterfactual analyses, interventionism, process theory. It's common to find authors

expressing the attitude that there is no single, universal theory of causation. Yet, these authors often mean different things by the term 'pluralism'. With this book, Ehrenstein aims to achieve greater clarity and rigor concerning claims of causal pluralism in the philosophy of science literature.

Ehrenstein is sceptical of numerous claims of pluralism about causality in the life sciences. He argues that some causal distinctions, such as Mayr's ultimate and proximate cause, are inherently incoherent and do not amount to a meaningful pluralism. Other claims to pluralism, such as Elliot Sober and Stuart Glennan's analyses of causal relevance and causal production, are not helpful in resolving the disputes they were purported to address. Still other proposed causal concepts, notably the concepts of permissive and instructive causes, are not genuine cases of causal pluralism.

Ehrenstein is not opposed to pluralism in principle, but rather holds that 'the range of reasonable applications for pluralism is smaller than some may have expected' (p. 11). He takes as unproductive the question of whether pluralism or monism is true of the world. Any answer to this question requires substantive decisions about which universally accepted desiderata a theory of causation must satisfy, and then further argument to support the conclusion that such a theory is (or is not) possible—a trajectory he suggests only leads to further disagreement. Instead, he prefers to investigate the limits and strengths of causal pluralist claims for specific problems confronted by the life sciences.

Chapter 1 aims to make the concept of causal pluralism intelligible. Ehrenstein takes inspiration from Leen De Vreese's ([2010]) threefold distinction between epistemological–methodological, metaphysical, and conceptual types of causal pluralism. Ultimately, Ehrenstein concludes that conceptual causal pluralism is the preferred lens through which to make sense of this topic. Conceptual causal pluralism is helpfully contrasted with conceptual causal monism. The latter position states that a theory of causation claims to analyse a master concept of cause into which all other causal concepts are subsumed. Later, in Chapter 2, we learn more about what Ehrenstein has in mind with this distinction. He writes, 'pluralist theories, by contrast, analyze one or more causal concepts and subsume them under a general causal concept but without analyzing the general concept' (p. 68). From the examples and discussion, it becomes clear that a view counts as pluralist so long as there is no developed analysis of a master causal concept. I take it that this could be because authors have simply chosen not to develop an analysis, not that such an analysis is impossible or difficult to achieve. This strikes me as rather counterintuitive. I would expect conceptual causal pluralism to instead be committed to giving up 'the ideal that there is an overarching concept of cause and effect' (p. 153) or, rather, the denial that there is a single master causal concept. This interpretation is distinct from the of conceptual causal pluralism Ehrenstein works with in Chapters 1 and 3. Acknowledgement of this, and a discussion of why the concept used in Chapters 1 and 3 deserve the title of causal pluralism, was warranted.

Chapter 2 is an overview of the major theoretical players in the causation literature. Ehrenstein gives a brief historical overview of each theory—regularity, counterfactual, probabilistic interventionist, process, and normality theories are all discussed—as well as some of their problems and solutions. A noteworthy choice here is to treat normality theories as another theory of causation. The discussion of H. L. A. Hart and Tony Honoré's ([1985]) defence of normal causes characterizes their view as holding that 'conditions are no causes', and a bit later Ehrenstein writes, 'we should avoid being distracted from their thesis that it is *wrong* to claim that 'every factor necessary for the occurrence of an event is equally entitled to be called "the cause"' (p. 50). Normality theories are committed to an egalitarian concept of cause. That is, normality theories hold that not all causes are equal in some sense. Normality theories reject the Millian arbitrary selection principle (also known as Millian parity). This is the well-known thesis that any effect is the result of a conjunction of

numerous antecedent causes and, moreover, any privileging of some subset of causes is arbitrary and unprincipled (p. 20; see also Mill [1973], book 3, chap. 5, sec. 3). Essentially, normality theories reject the thesis that any selection of a subset of causes as the significant or important causes of an effect is arbitrary; instead, they propose a selection method grounded in some 'objective' or reasonable principle. Later, in Chapter 7, Ehrenstein argues in favour of normality theories as a better alternative to other theories of causation, on the grounds that they justify the exclusion of absurd, far-fetched causal claims (more on this below).

Chapter 3 illustrates the role of conceptual causal pluralism two different account. (Plain) pluralism is what many of us think of when we think of causal pluralism. This is the idea that there is no single, master concept under which all causal concepts may be subsumed. Ehrenstein develops the concept of arity pluralism as another dimension along which conceptual causal pluralism can land. Arity pluralism is a feature of causal theories that posits causal concepts of different arity. Ehrenstein uses first-order predicate logic to show how within the same natural language, different causal concepts can require different arity relationships. For example, one causal concept might have ternary relations, while another concept might have quaternary relations. In this case, it is impossible to subsume one under the other. Arity pluralism is conceptually distinct from (plain) pluralism because unlike the latter, the former needn't reject a single, master concept under which all other causal concepts are subsumed. Ehrenstein uses this distinction to offer a modest defence of the pluralism implicit in views like Eells's ([1991]) type-level theory of probabilistic causation. Eells's view has been criticized by Christopher Hitchcock ([2007]) for not satisfying (plain) pluralism. However, Ehrenstein shows that such views can be understood as a type of arity pluralism.

Chapter 4 takes up an extended discussion of Ernst Mayr's ([1982]) proximate–ultimate distinction. Mayr is well known for contending that proximate and ultimate causes provide answers to different types of biological questions. Ehrenstein iterates between examining difficulties in interpreting this distinction and uncovering conceptual flaws that follow from interpretative choices made of the Mayr text. Overall, the author does a good job of illustrating the incoherency of the distinction. He employs first-order predicate logic again to clearly demonstrate that the extension of proximate and ultimate causes needn't be distinct from each other. One debate with which Ehrenstein engages here is whether ultimate causes are reducible to proximate causes, as has been argued by André Ariew ([2003]). Ehrenstein is overall sceptical that Ariew's arguments succeed.

Ariew offers two lines of argument for replacing Mayr's distinction with one between dynamical and statistical explanations. The first is that Mayr's programme account of ultimate and proximate causes is dispensable, and that his concept of proximate cause is too narrow and should be made more capacious to include non-genetic processes and elements that are relevant to development. Ehrenstein argues against this that Mayr would not have disagreed with the claim that his distinction is dispensable, and that Ariew's reasoning falls short of refuting Mayr's programme account. For it may still be shown that Mayr's proximate cause concept and Ariew's more inclusive concept have different extensions. Ariew's second line of argument is that the concept of ultimate cause reduces to one of proximate cause and, consequently, Mayr's framework leaves unexplained evolutionary outcomes. Ariew maintains that Mayr's analysis captures dynamical rather than statistical processes. The former have to do with causal relationships instantiated between individuals and their environments, whereas the latter explain statistical attributes of a population. Ehrenstein explains that this critique need not undermine Mayr's position, at least on some interpretations of this distinction. For example, following A. Landsborough Thomson ([1926]), proximate and ultimate causes might yet turn out to have different extensions.

Chapter 5 takes up the permissive–instructive distinction, first articulated by Howard Holtzer ([1968]), and the question of whether this distinction warrants the status of conceptual causal pluralism. Roughly, the two concepts appear to pick out distinctive things. Permissive causes allow an outcome to occur; instructive causes shape the particular outcome in detail. However, the distinction lends itself to further interpretation and subsequent authors have adapted the meaning of these terms in different ways.

Holtzer formulated the distinction when thinking about how developing cells come to differentiate. On a common picture of cell development at the time, naïve cells differentiate when they receive a signal from an external inducing agent. Inducing agents were thought to carry detailed information that ‘instructs’ the cell to specialize into, say, a chondrocyte. However, the nature of the inducer changed as more empirical evidence suggested that they do not carry as much instructional information; instead, it seemed that naïve cells are poised to differentiate and that inducers merely initiate—or rather, permit—the cell to follow the path for which it is already instructed. Ehrenstein follows the historical work of Marc W. Kirschner and John C. Gerhart ([2005]), who interpret Holtzer’s permissive–instructive distinction to be a matter of degree. The more specific information a biomolecule contains, the more it lies on the side of the instructive spectrum; the less information, the more a cause lies on the permissive side. Yet, the permissive–instructive distinction has taken on different interpretations over the years. Michael J. Barresi and Scott F. Gilbert offer one such interpretation in their textbook, *Developmental Biology* ([2020]), characterizing an instructive interaction as a signal from the inducing cell that is necessary for initiating new gene expression in the responding cell. By contrast, a permissive interaction is an environment that allows the expression of traits in an already specified tissue. Pierrick Bourrat ([2019]) notes that Barresi and Gilbert’s distinction is comparable to the role of background conditions and triggering causes.

Ehrenstein’s discussion next turns to a contemporary debate concerning Woodward’s ([2010]) account of causal specificity. Causal specificity is a property of special causal variables capable of taking a range of alternative values where (for the most part) each value associates with one and only one value in an effect variable. Importantly, specificity is a matter of degree. A causal variable that takes a larger set of alternative values (where each value associates with a unique value in an effect variable) has a greater amount of causal specificity than a variable that takes a subset of alternative values. Woodward suggests that the concept of causal specificity might account for the instructive–permissive distinction. Instructive causes possess high degrees of specificity; whereas permissive causes are non-specific.

While Ehrenstein is amenable to Woodward’s notion of specificity as an analysis of instructive and permissive causes, he strongly objects to the biological examples Woodward employs to illustrate the concept: RNA polymerase as a non-specific cause and DNA as a specific cause. Ehrenstein provides a compelling argument that this is comparing apples to oranges. Ehrenstein attributes Woodward’s mistake to ‘a superficial analysis of protein biosynthesis’ (p. 117) and failing ‘to have a firm grasp of [...] protein biosynthesis’ (p. 129). But I think Ehrenstein has misdiagnosed Woodward’s mistake. It’s not that Woodward doesn’t understand protein biosynthesis. Rather, he seems to be making a hasty comparison that essentially changes the subject and, furthermore, that numerous other authors failed to identify.

Chapter 6 examines a common distinction between two types of causation, namely, production and relevance. This chapter surveys three different accounts, by Ned Hall ([2004]), Elliot Sober ([1984]), and Stuart Glennan ([2009]), that essentially adopt (albeit very) different variations of this distinction. Ehrenstein uses first-order predicate logic to show how Hall’s distinction does indeed count as a form of conceptual causal pluralism, but Hall’s analysis has yet to be applied to cases in the philosophy of biology. Unlike Hall’s account,

those offered by Sober and Glennan are explicitly intended to address philosophical problems that arise in biological reasoning. In this chapter, Ehrenstein accepts that Sober's and Glennan's distinctions also meet the conditions for conceptual causal pluralism. Yet, this is not the end of discussion. His aim is not just to examine the coherence of claims to causal pluralism; it is also to evaluate whether genuine cases of causal pluralism turn out to be useful. Ehrenstein's conclusion is that neither Sober's nor Glennan's accounts actually resolve the philosophical problems they were originally designed to address.

Chapter 7 substantiates an assertion made in Chapter 1 about counterfactual and interventionist theories of causation, that these theories adopt an account of causal explanation 'which is unsuited for biology' (p. 6). Here, Ehrenstein tackles the notorious problem in the causal literature of omissions. The causal systems that characterize life are full of omissions. A gardener who fails to water the flowers is causally relevant to the flowers dying, and the absence of lactose in *E. coli* causes the absence of β -galactosidase. Yet, omissions are philosophically puzzling for various reasons. The puzzle here, first raised by David Lewis ([2004]), concerns how acceptance of omissions into one's ontology permits the acceptance of implausible causes. On the one hand, admitting of omissions as causes seems to compel us to accept an enormous number of omissions. For it is not just the gardener's failure to water the flowers that causes their death, but it is also the failure of the chancellor to do so, as well as the absence of sufficient rain and so on. On the other hand, there seems to be no principled way to accept some omissions as relevant causes and reject other far-fetched, absurd omissions. Absurd, far-fetched omissions include things that 'we are quite certain do not exist or the existence of which we have no reason to suspect' (p. 175). Ehrenstein gives the example of the absence of minuscule aliens that hypothetically participate in the lactose mechanism. According to Ehrenstein, an adequate theory of causation should provide biologists with justification for ruling out such far-fetched, absurd causes.

One move the interventionist might make (with which I am sympathetic) is to question the usefulness of a causal theory that justifies ruling out what Ehrenstein regards to be absurd types of omissions. Few scientific claims are known with a level of certainty on par with the certainty of the non-existence of minuscule aliens. A more common occurrence, it seems to me, is that scientific communities often change what they regard as far-fetched causal hypotheses over time. A desirable feature of a causal theory is that it not rule out *a priori* causal hypotheses that may erroneously be taken to be far-fetched and absurd, and it is a strength of interventionism that it does not do this. Of course, it can be useful to prioritize some types of interventions and backgrounds over others. Some causal hypotheses might be regarded as far-fetched and absurd because at that moment there is no technical or conceptual way to make progress. Indeed, Woodward's interventionist theory is compatible with additional, pragmatic principles that justify ranking some types of causal hypotheses as more important than others. For example, Woodward ([2000]) explains that physics tends to prefer explanations that satisfy symmetry requirements, and a variety of additional principles have been defended elsewhere (see Waters [2007]; Weber [unpublished]). The approach adopted by these authors is to treat the interventionist theory as an analysis of causes and non-causes, and to adopt pragmatic principles for privileging some types of causes as complementary and additional to interventionism. Interventionism treats the explanatory power of a causal relationship as an empirical matter that involves discovery, and it is a strength that it accounts for the role discovery must play in scientific inquiry.

Ehrenstein, however, concludes that normality accounts of causation are best equipped to handle the problem of absurd omissions. Ehrenstein appears to be amenable to a plurality of ways to spell out normality conditions. He first considers the work of Hart and Honoré, whose analysis is based on an intuitive sense of what is normal for a given circumstance, showing how their theory might overcome objections. On Hart and

Honoré's approach, variables that deviate from the norm are the explanatory causes. He also considers Hüttemann's ([2013]) disposition-based process theory, which involves identifying systems and their subsystems. Systems consist of objects with dispositions to behave. Systems have a default behaviour if left undisturbed. The explanatory causes are disturbing factors that interfere with the default behaviour of the system. What this suggests is that philosophical justification is needed for substantiating scientific (and perhaps everyday) choices about which types of possibilities are countenanced and which are not.

Causal Pluralism in the Life Sciences offers a series of rigorous arguments, presented at a relatively high level, that introduces the reader briefly to the historical context and logical clarity of an issue that has widespread currency in philosophy of science. The book doesn't focus much on how causal reasoning and causal investigation work in science; there are no historical case studies from which philosophical reflection is extracted. Instead, the focus is the critical examination of the conceptual foundations of causal distinctions proposed by philosophers, making the book of greatest interest to philosophers of science with metaphysical leanings or those who are invested in the causal distinctions Ehrenstein considers. However, readers might question Ehrenstein's investment in causal theories that rule out causal hypotheses regarded as far-fetched or absurd and, relatedly, whether he has shown interventionism to be truly ill suited to the life sciences. This disagreement may very well be the result of Ehrenstein's previously mentioned methodology in the book. Ehrenstein's approach does not involve examination of how scientific investigation operates, but rather draws on scientific examples to inform the adequacy of philosophical theories. While this is a legitimate methodological approach to philosophy of science, interventionism is more compatible with the dynamic nature of scientific investigation and thus readers may regard the approach taken here as rather limited. For this reason, philosophers of science who value understanding the processes of scientific inquiry are unlikely to be satisfied with Ehrenstein's criticisms.

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References

- Ariew, A. [2003]: 'Ernst Mayr's "Ultimate/Proximate" Distinction Reconsidered and Reconstructed', *Biology and Philosophy*, 18, pp. 553–65.
- Barresi, M. J. and Gilbert, S. F. [2020]: *Developmental Biology*, New York: Oxford University Press.
- Bourrat, P. [2019]: 'On Calcott's Permissive and Instructive Cause Distinction', *Biology and Philosophy*, 32.
- De Vreese, L. [2010]: 'Disentangling Causal Pluralism', in R. Vanderbeeken and B. D'Hooghe (eds), *Worldviews, Science, and Us*, Hackensack, NJ: World Scientific, pp. 207–23.
- Eells, E. [1991]: *Probabilistic Causation*, Cambridge: Cambridge University Press.
- Glennan, S. [2009]: 'Productivity, Relevance, and Natural Selection', *Biology and Philosophy*, 24, pp. 325–39.

- Hall, N. [2004]: 'Two Concepts of Causation', in J. Collins, N. Hall and L. A. Paul (eds), *Causation and Counterfactuals*, Cambridge, MA: MIT Press, pp. 225–76.
- Hart, H. L. A. and Honoré, T. [1985]: *Causation in the Law*, Oxford: Clarendon Press.
- Hitchcock, C. [2007]: 'How to Be a Causal Pluralist', in P. Machamer and G. Wolders (eds), *Thinking about Causes: From Greek Philosophy to Modern Physics*, Pittsburgh, PA: University of Pittsburgh Press, pp. 200–21.
- Holtzer, H. [1968]: 'Induction of Chondrogenesis: A Concept in Quest of Mechanisms', in R. Fleischmajer and R. E. Billingham (eds), *Epithelial-Mesenchymal Interactions: 18th Hahnemann Symposium*, Baltimore, MD: The Williams and Wilkins Company, pp. 152–64.
- Hüttemann, A. [2013]: 'A Disposition-Based Process-Theory of Causation', in S. Mumford and M. Tugby (eds), *Metaphysics and Science*, Oxford: Oxford University Press, pp. 101–22.
- Kirschner, M. W. and Gerhart, J. C. [2005]: *The Plausibility of Life: Resolving Darwin's Dilemma*, New Haven, CT: Yale University Press.
- Lewis, D. [2004]: 'Causation as Influence', in J. Collins, N. Hall and L. A. Paul (eds), *Causation and Counterfactuals*, Cambridge, MA: MIT Press, pp. 75–106.
- Mayr, E. [1982]: *The Growth of Biological Thought: Diversity, Evolution, and Inheritance*, Cambridge, MA: Belknap Press.
- Mill, J. S. [1973]: *A System of Logic Ratiocinative and Inductive: Being a Connected View of the Principles of Evidence and the Methods of Scientific Investigation*, London: Routledge.
- Sober, E. [1984]: 'Two Concepts of Cause', *Proceedings of the Biennial Meeting of the Philosophy of Science Association*, 1984, pp. 405–24.
- Thomson, A. L. [1926]: *Problems of Bird-Migration*, London: H. F. and G. Witherby.
- Waters, C. K. [2007]: 'Causes That Make a Difference', *Journal of Philosophy*, **104**, pp. 551–79.
- Weber, M. [unpublished]: 'Causal Selection vs Causal Parity in Biology: Relevant Counterfactuals and Biologically Normal Interventions', available at .
- Woodward, J. [2000]: 'Explanation and Invariance in the Special Sciences', *British Journal for the Philosophy of Science*, **51**, pp. 197–254.
- Woodward, J. [2010]: 'Causation in Biology: Stability, Specificity, and the Choice of Levels of Explanation', *Biology and Philosophy*, **25**, pp. 287–318.
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