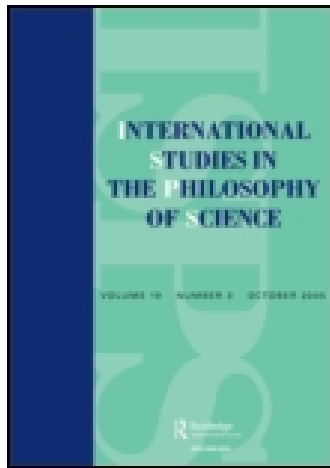


This article was downloaded by: [Macquarie University]

On: 26 January 2015, At: 19:39

Publisher: Routledge

Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



## International Studies in the Philosophy of Science

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/cisp20>

### Genetics and Philosophy: An Introduction

Monika Piotrowska<sup>a</sup>

<sup>a</sup> Department of Philosophy, Florida International University  
Published online: 12 Sep 2014.



CrossMark

[Click for updates](#)

To cite this article: Monika Piotrowska (2014) Genetics and Philosophy: An Introduction, *International Studies in the Philosophy of Science*, 28:2, 223-226, DOI: [10.1080/02698595.2014.932533](https://doi.org/10.1080/02698595.2014.932533)

To link to this article: <http://dx.doi.org/10.1080/02698595.2014.932533>

PLEASE SCROLL DOWN FOR ARTICLE

Taylor & Francis makes every effort to ensure the accuracy of all the information (the "Content") contained in the publications on our platform. However, Taylor & Francis, our agents, and our licensors make no representations or warranties whatsoever as to the accuracy, completeness, or suitability for any purpose of the Content. Any opinions and views expressed in this publication are the opinions and views of the authors, and are not the views of or endorsed by Taylor & Francis. The accuracy of the Content should not be relied upon and should be independently verified with primary sources of information. Taylor and Francis shall not be liable for any losses, actions, claims, proceedings, demands, costs, expenses, damages, and other liabilities whatsoever or howsoever caused arising directly or indirectly in connection with, in relation to or arising out of the use of the Content.

This article may be used for research, teaching, and private study purposes. Any substantial or systematic reproduction, redistribution, reselling, loan, sub-licensing, systematic supply, or distribution in any form to anyone is expressly forbidden. Terms &

Conditions of access and use can be found at <http://www.tandfonline.com/page/terms-and-conditions>

It is specifically on these issues that the volume—especially as a ‘*metaphysician’s* guide’ through the puzzles of causation—can be expected to stimulate further discussion, and possibly further reflections on their relevance for non-fictional situations, and for illuminating actual case studies from the sciences. The volume can also be expected to fuel reflections on how ‘doing metaphysics can be understood as a contribution to our overall understanding of the world’ (Paul 2012, 24), and more work on whether a single account of causation will ever be able to fit all varieties of causal relations.

## References

- Collins, John, Ned Hall, and L. A. Paul, eds. 2004. *Causation and Counterfactuals*. Cambridge, MA: MIT Press.
- Hitchcock, Christopher. 2007. “What’s Wrong with Neuron Diagrams?” In *Causation and Explanation*, edited by Joseph Keim Campbell, Michael O’Rourke, and Harry S. Silverstein, 69–92. Cambridge, MA: MIT Press.
- Paul, L. A. 2012. “Metaphysics as Modeling: The Handmaiden’s Tale.” *Philosophical Studies* 160: 1–29.

RAFFAELLA CAMPANER

*Dipartimento di Filosofia e Comunicazione*  
*Università di Bologna*

© 2014, Raffaella Campaner

<http://dx.doi.org/10.1080/02698595.2014.932532>

## Genetics and Philosophy: An Introduction

PAUL GRIFFITHS and KAROLA STOTZ

Cambridge, Cambridge University Press, 2013

viii + 270 pp., ISBN 9781107002128, £50.00, US\$90.00 (hardback); ISBN 9780521173902, £17.99, US\$29.99 (paperback)

The book has several themes, but the main thesis is that gene products are not solely determined by what most people think of as genes, i.e. the coding sequences of DNA. The transcriptome, for example, contains sequence information not literally encoded in the DNA. Where did this information come from, if not from the DNA sequence from which it was transcribed? To answer this question, Paul Griffiths and Karola Stotz persuasively argue that the information comes from ‘the coding sequences, regulatory sequences and their RNA and protein products, and the environmental signals that act via that regulatory machinery’ (5). They provide numerous examples from molecular biology to demonstrate how gene products are partially determined by both the coding sequences of DNA and by the activation and selection of these sequences. Moreover, they show how additional information created during post-transcriptional processing can also help specify the final gene product.

Their argument moves outward, from ‘intra-cellular’ to ‘inter-cellular’ to ‘extra-cellular environmental signals’ (84), showing the impact on the final sequence of

the gene product at each level. Much of the book is thus aimed at persuading the reader that genes are not ‘the prime movers in all biological processes’ (8) and that ‘postgenomic genes’ are better understood in a functional sense, as ‘things an organism can do with its genome’ (75).

With the main argument in place, the authors examine its impact on a number of philosophical debates. I will discuss three of them: causation, information, and reduction. With respect to the debate on causation, the authors focus on the application of James Woodward’s (2003) widely cited philosophical account of causation to genetics. Kenneth Waters (2007), a major player in this debate, has argued that DNA is the only ‘actual difference maker’ when it comes to the specificity of gene products. According to Waters, those who want to emphasize the role of other factors in the process of development, like Griffiths and Stotz, do so only because they mistakenly believe in the ‘causal parity thesis’, which states that favouring one cause over others cannot be justified on ontological grounds, because all causes are equal.

In response, Griffiths and Stotz reject what they consider to be Waters’s caricature of the parity thesis, explaining that the thesis ‘asserts that the roles of causal factors in development do not fall neatly into two kinds, one role exclusively played by DNA and RNA sequences, and the other role exclusively played by elements other than nucleic acids’ (160). Hence, on their interpretation, the parity thesis does not suggest that all causes are equal. Instead, it makes room for factors other than genes to serve as causes in certain explanatory contexts. Given what scientists know about genome structure and function, to Griffiths and Stotz it seems obvious that ‘factors outside the DNA sequence co-specify the precise sequence of the RNAs and polypeptides that will be derived from the DNA’ (179). If the DNA sequence is not the main source of the specificity of gene products, Waters cannot successfully rescue DNA as the sole bearer of causal specificity.

The two arrive at a similar conclusion with respect to the debate on information. Here, they deny that genes are somehow different from other developmental causes because they carry information. ‘The only really substantial sense in which genes carry information is their role in templating for gene products’ (179), but since other factors are involved in activating, selecting, and creating the gene products, it looks like they, too, carry information. Hence, the concept of information cannot be used to assign a unique causal status to genes.

Griffiths and Stotz also examine the impact of their argument on the reduction debate. They start by explaining that the most active discussion of reduction today no longer focuses on the question of whether one theory can be reduced to another, but on ‘explanatory reduction’, which they define as ‘the idea that higher-level phenomena can be explained by lower-level phenomena and their interactions’ (62). Given that on their account, gene products are contextually determined and are not some intrinsic property of the DNA sequence—‘a set of sequences is a gene because of the way in which it is used by the cell, not because of its intrinsic structure’ (106)—it does not come as a surprise that Griffiths and Stotz argue for an anti-reductionist position. After all, developmental outcomes cannot be explained by looking at the components involved taken out of context. Instead, the system can

only be explained in terms of the organization and interaction of its parts, and this, they argue, does not count as a reductive explanation. When we study parts as an integrated whole, we work towards a holistic explanation.

I must admit that I found myself nodding along with much of what Griffiths and Stotz had to say. Indeed, the themes of the book and its overall thesis seem to bolster earlier arguments of my own. Since I do not have much by way of criticism, let me wrap things up by applying their themes to my own work on extrapolation—a topic not addressed in their book. I will then conclude with one critical point.

In 2009, I published an article criticizing inferences made about the suitability of model organisms that were based on genetic comparisons. I argued that by focusing too much on stretches of DNA when comparing species, we have presupposed an overly simplistic connection between genotype and phenotype. However, my argument is based on merely two regulatory mechanisms that can construct gene products not mirrored in any linear DNA sequence: alternative splicing, which reshuffles the original DNA sequence, and mRNA editing, which modifies the original sequence. Given all the additional factors that Griffiths and Stotz expose as causal contributors to the final ‘postgenomic’ gene product, their argument only strengthens the conclusion of that article—that the connection between genotype and phenotype, ‘even at the narrowest molecular level, is far from straightforward’ (Piotrowska 2009, 848).

Their argument also has implications for inferences scientists make about human diseases from studying ‘humanized’ animals. Since therapies that work on nonhuman model animals can fail to provide similar results in humans, humanized animals are supposed to bridge this inferential gap by simulating a variety of human diseases, e.g. asthma, diabetes, cancer, etc. But it is not clear how to modify these animals in order to make them more human-like, so that the inferences made from humanized animals to humans are justified. In another article (Piotrowska 2013), I explain the difficulties involved with deciding which parts to transfer into these animals, where the boundaries of those parts begin and end, and what to make of the influence of a new context on these parts, post-transfer. Griffiths’s and Stotz’s book only magnifies these difficulties and is likely to leave the reader feeling sceptical about our ability to make any reliable causal generalizations from humanized animals to humans.

Philosophers of biology will certainly find this book interesting, but I suspect non-specialists will find its topics insufficiently motivated and a bit too technical. I find this to be the book’s biggest weakness, although it is worth pointing out that it is likely intentional. Note, for example, the opening sentence: ‘Unlike most books which combine philosophy and genetics in their titles, this is not a discussion of the ethical, legal, and social implications of science’ (1). That said, the book is a gold mine for those interested in such issues, and Griffiths and Stotz could have done more (without a lot of effort) to point that out.

## References

- Piotrowska, M. 2009. “What Does It Mean to Be 75% Pumpkin? The Units of Comparative Genomics.” *Philosophy of Science* 76: 838–850.

- Piotrowska, M. 2013. "From Humanized Mice to Human Disease: Guiding Extrapolation from Model to Target." *Biology and Philosophy* 28: 439–455.
- Waters, C. K. 2007. "Causes that Make a Difference." *Journal of Philosophy* 104: 551–579.
- Woodward, J. 2003. *Making Things Happen: A Theory of Causal Explanation*. New York: Oxford University Press.

MONIKA PIOTROWSKA  
Department of Philosophy  
Florida International University

© 2014, Monika Piotrowska

<http://dx.doi.org/10.1080/02698595.2014.932533>

### **Life, War, Earth: Deleuze and the Sciences**

JOHN PROTEVI

Minneapolis, University of Minnesota Press, 2013

ix + 256 pp., ISBN 9780816681013, US\$75.00 (hardback); ISBN 9780816681020, US\$25.00 (paperback)

In a discussion of the ontology, biology, and history of affect, hence tapping into the Spinozism of the philosophy of Gilles Deleuze (and Félix Guattari), John Protevi in *Life, War, Earth: Deleuze and the Sciences* mentions the case of the racehorse and the plough horse. In spite of our classificatory way of thinking in terms of species and *genera*, these horses have very little in common. Provided that we let go of our habits and think in terms of 'affect', that is, the capacity of a body to act and to be acted on, the plough horse has more in common with the ox, and the racehorse with, for example, a sports car or motorcycle. Protevi explains that one might definitely try to win a race with a plough horse, or plough a field with a racehorse (or a sports car or motorcycle), thus instigating other affects. Such a project can be named an experiment and sheds light on the seemingly oxymoronic term 'transcendental empiricism' from Deleuze's ([1968] 1994) *Difference and Repetition*. Protevi argues: 'Whether the bodies involved could withstand the stresses they undergo in these idiosyncratic assemblages is a matter of (one would hope careful) experimentation' (74). Animals, pieces of transport technology, and also human beings 'individuate . . . as singular patterns of social, neural, and somatic interaction, each actualization structured by and structuring its virtual field' (103). Importantly, Protevi argues that this is not to foster a social constructivism or a genetic determinism. Rather, it is in the nature of nature, technology, and what is human to be 'programmed to be open' (103). However, in the case of a transcendental empiricism, we speak not only of a careful experimentation, but also of a careful balancing act when it comes to ontological claims:

I think it is important to rescue a minimal notion of human nature from extreme social constructivism and to hold that rage episodes [for example] are individuations of a multiplicity encompassing variation in genetic inheritances, environmental input in the form of subjectification practices, and developmental plasticity. (78)