Causation in Biology  
and related topics

Program for the first CRedS workshop 27.-28. April, 2015

A work-in-progress forum organized by the project «Causation and Reduction in Systems Biology», Department of Philosophy, Classics, History of Art and Ideas, University of Oslo

Workshop location: The Niels Bohr Institute, Blegdamsvej 17, Copenhagen. Room: Aud. C

Monday, April 27

12-13  Lunch in the canteen at the Niels Bohr Institute (building F)

13-13.10  Introduction
Gry Oftedaal

13.10-14.40  Are model organisms concrete theoretical models?
Veli-Pekka Parkkinen, University of Kent

10 min break

14.50-16.20  Establishing Constitutional Relations, in Theory and in Practice
Lorenzo Casini, University of Geneva

10 min break

16.30-18  Mechanistic Explanations in Psychiatry - An Interventionist Perspective
Lise Marie Andersen, Aarhus University

19.30  Dinner at the restaurant Pluto (Borgergade 16)
**Tuesday, April 28**

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Are model organisms concrete theoretical models?

Veli-Pekka Parkkinen

How does studying a model organism create understanding of its target? Is the animal model just a concrete version of a theoretical model: a surrogate system for study, from which results are transferred to the target based on known analogies between the model and the target. Recently, Levy and Currie (2014) have argued that model organism research in biology differs from theoretical modelling in the crucial point of justifying model-to-target inferences. According to Levy and Currie, instead of serving as a surrogate for its target in virtue of deliberately constructed (partial) analogy, an animal model serves as a basis for empirical extrapolations via phylogenetic inferences. I extend the discussion to model organism research in biomedicine, and argue that Levy and Currie’s distinction fails to apply in some important cases.

I present two case-studies – animal models in atherosclerosis research and humanized mice as models of human cancer – to show that model organism research employs reasoning strategies analogous to theoretical modelling, and does not always rely on phylogenetic inference. These strategies include deliberate construction of analogies between the model and the target, and the evaluation of robustness of results derived from a family of models. I intend this not as an argument against the basic point made by Levy and Currie, however. Instead I argue that there is a further difference that distinguishes animal models from theoretical models, having to do with explanation.

I argue that theoretical models explain by describing causal and constitutive dependencies between phenomena. In the model, these dependencies are explicitly expressed, often as mathematical equations, which allows derivations of outcomes of various manipulations of the model. These results are then transferred to the target based on analogies that the modeller has deliberately coded into the model. For example, using the Lotka-Volterra model to answer what happens when both predator and prey populations decline in proportion to their abundance consists of calculating new trajectories for the population variables according to the functions explicitly described in the model. The model thus explains by licensing inferences about the target’s behaviour in various conditions.

By contrast, result obtained in an animal experiment is evidence that some causal or constitutive dependency obtains, but these dependencies are not in any obvious way explicitly expressed in the model. Transferring the result from the model to the target requires a hypothesis about the exact nature of the dependence, and an assumption that the same dependence is exhibited by the target, such as assuming that a similar mechanism is operating in both. The reasoning strategies of phylogenetic inference, analogy, and robustness, serve as criteria for grading the quality of the evidence, and justifying the extrapolation between the model and the target. In conclusion, theoretical and animal models have different epistemic roles, even though broadly similar reasoning strategies may be employed for model-to-target inferences in both cases.
Establishing Constitutional Relations, in Theory and in Practice

Michael Baumgartner and Lorenzo Casini

In this paper, we argue that Craver [2007]'s mutual manipulability criterion for the identification of the (micro) constituents of a (macro) phenomenon is inadequate, both normatively and descriptively, and we offer an 'abductivist' alternative. According to our proposal, constitutional relations are established by finding a decomposition of the phenomenon into causally interacting parts that provides a maximally explanatory account of that phenomenon. Selecting a best explanation, in turn, involves satisfying a number of constraints, which jointly guarantee redundancy-free empirical adequacy and decompositional robustness. Our proposal is distilled from recent research in neuroscience concerned with the identification of constitutional relations in the brain [Nelson et al. 2010, Meunier et al. 2009]. We claim that our account not only provides a faithful reconstruction of the scientific reasoning involved in these examples but also lays the foundation for a normatively adequate methodology of constitutional reasoning.

References


Mechanistic Explanations in Psychiatry - An Interventionist Perspective

Lise Marie Andersen

The view that psychiatry should be elucidating the mechanisms behind mental phenomena is gaining momentum. This view, coupled with an intuition that such mechanisms must, by nature, be biological, has inspired the field to look to cognitive neuroscience for classification of mental illnesses. One example of this kind of reorientation can be seen in the Research Domain Criteria project (RDoC) introduced by the U.S National Institute of Mental Health. The RDoC project is an attempt to introduce a new classification system based on brain mechanisms. It is a central idea of the project that mental disorders can be understood in terms of brain disorders. Kendler (2005) observes a consequence linked with such a reorientation. The consequence being that multilevel models citing mental and social factors as part of the causal structures are often rejected as non-scientific or accepted “only with the caveat that all the “real” causal effects occur at the level of basic biology” (p.435). The problem with this kind of whole-scale reductionism is that it is precisely such multilevel models, which are necessary for progress in this fundamentally interdisciplinary science. As a response to the reductionist reorientation, some
philosophers and scientists have argued that the preference for reductionist approaches is grounded in a misconception of causation in terms of processes. Instead they suggest that an interventionist account of causation, such as developed by Woodward (2003), provides a rigorous framework for understanding the causal structures revealed in multilevel explanations (Campbell 2007; Kendler & Campbell 2009). They argue that it is a clear advantage of the interventionist approach that it “clearly separates the issues of causation from the issue of mechanisms” (Campbell and Kendler 2009). In contrast, this paper argues that mechanisms are essential to psychiatric explanations, but also that the intuitions that lie behind the call for mechanistic explanations can be accommodated in the interventionist model. This claim is not uncontroversial. It has recently been argued that it is the a fundamental issue for interventionist accounts is that they are not able to represent mechanistic information adequately (Dowe 2011; Illari & Williamson 2011). This paper is a work in progress on a reply to such worries.

Causal asymmetry and Specificity

Gry Oftedal

I investigate the question of causal strength and causal importance. What does it mean that one cause is stronger or more important than another? My starting point is an account of causal asymmetries by Sober, Wright and Levine from 1992, which concerns causality in the social sciences. On this basis I suggest how to understand causal asymmetries in relation to biological examples and what may be important differences between biology and social sciences in this respect. I then relate my analysis to an on-going discussion on causal specificity in biology where specific causes are attributed a particular causal status in relation to non-specific causes (Waters 2007, Woodward 2010, Griffiths and Stotz 2013, Griffiths et al 2015).

On the Limits of Causal Modeling: Spatially-Structurally Complex Phenomena

Marie I. Kaiser

This paper examines the adequacy of causal graph theory as a tool for modeling biological phenomena and formalizing biological explanations. I point out that the causal graph approach reaches it limits when it comes to modeling biological phenomena that involve complex spatial and structural relations. Using a case study from molecular biology, DNA-binding and -recognition of proteins, I argue that causal graph models fail to adequately represent and explain causal phenomena in this field. The inadequacy of these models is due to their failure to include relevant spatial and structural information in a way that does not render the model non-explanatory, unmanageable, or inconsistent with basic assumptions of causal graph theory.
Causal analysis, truth conditions, and metaphysics

Anders Strand

Causation has been a topic of interest throughout the history of philosophy. Still, there are unsettled disputes among contemporary defenders of views such as counterfactual dependence analyses, interventionism, dispositionalism, Humean views, and transmission theories. What explains this state of controversy and unsettlement, and the seeming lack of philosophical progress?

Part of the explanation is a lack of explicitness and agreement when it comes to the purposes of - and methods employed in - philosophy of causation. In this talk I address these metaphilosophical issues. I argue that a philosophical account should (1) provide a truth-conditional analysis of key causal claims. Such a semantic analysis can be partly revisionary, and should be guided by how actual causal concepts and other representations work, the role of causal information in human agency and in science, a quest for conceptual unity, and the need for conceptual development in light of various shortcomings and defects. Furthermore, a philosophical account should (2) shed light on epistemological and inferential aspects of causal knowledge, including assumptions needed for valid extrapolation across contexts. Finally, the account should (3) integrate with metaphysical theory, and I discuss one way in which it can do so.