Causal Distinctions: Specificity and Beyond

University Of Cologne

22nd – 23rd October 2020

Day 1: Thursday Oct 22nd

10.30-11.40

Marcel Weber: *Instructive Causes, Embryonic Induction and Causal Specificity*

Some philosophers have argued that the common distinction between permissive and instructive causes in developmental biology can be fleshed out in terms of causal specificity. I argue in this talk that this is a mistake; the notions of permissive and instructive cause refer simply to different kinds of embryonic induction. In the permissive case, a single developmental pathway is caused to proceed or to arrest, while an instructive cause selects between different pathways, one of which is often a default. When there are just two different pathways to choose from, an instructive cause can be just as switch-like as a permissive cause and therefore has the same causal specificity. However, causal specificity can be used in developmental biology to distinguish between different kinds of instructive causes, i.e., different kinds of embryonic induction. Some inductions are more causally specific than others. I use causal specificity in order to conceptually analyse a classic example of embryonic induction, namely the Spemann-Mangold organizer, which is a small piece of embryonic tissue initially found in amphibians capable of inducing a new body axis. The sense in which this tissue has „organizing power" has been controversially discussed ever since it was discovered. I use the notion of causal specificity in order to explicate this notion.

12.00-13.10

Neil McDonnell: *The Privileged Context*

The truth of any given counterfactual statement is widely thought to be in some way a function of the context of that statement. Thus causal statements, interpreted via a counterfactual analysis of causation, may vary in truth value across contexts. Those of us with sufficiently-strong reductive ambitions for a theory of causation are troubled by this. In this paper I will argue that, within the context of a counterfactual approach, positing a Privileged Context for the assessment of causal claims gives us a definitive mind- and contextindependent standard for causation. For many this standard will be overly permissive, and thereby insufficiently specific to account for our intuitive causal assertions. I agree but I will argue that a second layer of considerations – which are free to be neither mind- nor context-independent – can be applied to give us the theoretical and practical tools we want when using the word "cause". This requires a methodological shift in the philosophy of causation, but I will argue that that just such a shift is required in the context of various long-standing impasses in the literature.

15.00-16.10

María Ferreira Ruiz: *Attributing Causal Specificity*

The literature on causal specificity assumes that attributing this property to causal relations is at least in principle a straightforward (if not systematic) task. This is true both for parity advocates and parity opponents in philosophy of biology. In this presentation, I reconsider this assumption and take a step back to reassess the value and limitations of the notion of specificity for the parity dispute. This is not carried out on the basis of further analysis of the overly discussed cases of DNA vs. the polymerase, or vs. alternative splicing. Rather, I identify prior difficulties for attribution of specificity in the concept itself.

In particular, my contribution reveals an irreducible "dual" nature of causal specificity as involving two components that are not mutually entailed. Assuming the relevant relations are causal, these are "repertoire" (many possibilities exist for each relatum) and "connectedness" (the possibilities on both sides are connected in a special way, e.g. bijective). While there is partial recognition that relations can differ in their specificity roughly along these two aspects, the mutual independence of components (and its implications) goes rather unnoticed. I clarify this point, and show how it affects attributions of specificity. Notably, intuitions about switch-like cases hang on the relative weight given to the components. These cases can be deemed minimally specific, as score low on repertoire, or maximally specific, because the function is bijective, which is paradoxical.

I contend these are not minor issues, as they directly affect the very purposes for which specificity is often invoked: in general, to compare/distinguish amongst causal factors; in particular, to settle the causal parity dispute in philosophy of biology.

16.30-17.40

Thomas Blanchard: *The Roles of Specificity (and related notions) in Experimental Causal Inference*

In this talk I will explore some of the roles that specificity and related notions play in experimental causal inference (understood in a broad sense that includes observational research based on "natural experiments"), and argue that experimental research relies in an important way on the ability to identify and exploit specific, stable and proportional causal relationships. Drawing on existing suggestions in the literature, I will examine various desiderata of specificity, stability and proportionality that play a key role in experimental design and the identification of natural experiments. I will also discuss limitations that arise in contexts where those desiderata cannot all be satisfied, or can be satisfied only by compromising on other desirable features of experimental design.

18.00-19.10

Janella Baxter: *What's Synthetic Biology Got to do with It?*

The emerging discipline of synthetic biology is characterized by diverse disciplinary approaches – ranging from systems biology, computer science, synthetic chemistry, engineering, and more. This diversity notwithstanding, synthetic biologists are unified by their interest to construct biological components and systems that not only have not evolved in the actual world but are unlikely to do so without human ingenuity (Baxter 2019). Initially, this focus of synthetic biology appears to be in stark contrast with the aims of more traditional areas of biology that seek to understand life as it has actually evolved in our world (Mitchell 2008, 2009, 2015; Ronai 2017; Waters 2007). This has prompted authors to reflect on what exactly is this discipline's relationship to the rest of the life sciences. As the historian and philosopher of science, Evelyn Fox Keller (Keller 2009), has asked: Do the products of synthetic biology fall within the explanatory scope of more traditional areas of biology or do they merely expand the universe of entities to explain?

While some work has already been done to illustrate how synthetic biology has played a crucial role in systems biology (Knuuttila and Loettgers 2013), in this talk I will argue that synthetic biology is playing an explanatory and investigative role in protein modeling. Synthetic biology methods have been particularly effective for engineering proteins that display artificial types of causal specificity. It is by manipulating engineered types of causal specificity that structural biologists can make observations and inferences about the three-dimensional conformation of a protein.

What this shows is that (at least some) areas of synthetic biology are much more integrated into the study of more traditional areas of biology than philosophers (and synthetic biologists themselves!) have previously appreciated. I wish to argue that integration of synthetic biology methods into structural biology is not

merely a matter of enabling novel observations. I maintain that synthetic biology also informs the explanations biologists formulate. For when structural biologists are called upon to justify the inferences they make about protein structure, they formulate explanations that appeal to the synthetic techniques they employed. So, while some representatives from the community (notably Drew Endy) often claim that synthetic biology is generating knowledge about life by bypassing the study of messy, bespoke natural systems, the reality may be quite the opposite.1 Synthetic biology is helping scientists advance knowledge about life precisely by enabling better study of messy, bespoke natural systems.

Day 2: Friday Oct 23rd

10.30-11.40

Bram Vaassen: *Halfway Proportionality*

According to the so-called 'proportionality principle', causes should not contain too much or too little information relative to their effects. This principle is subject to an ongoing debate. On the one hand, many maintain that it is required to address the problem of causal exclusion and take it to capture a crucial aspect of causation. On the other hand, many object that it render accounts of causation implausibly restrictive and reject the principle wholesale. I argue that both sides exaggerate. While one half of the principle is overly demanding, the other half is unobjectionable. And while the unobjectionable half does not block exclusion arguments, it does shed light on underappreciated issues in higher-level causation and fits with recent developments in philosophy of causation. I conclude that at least half of the proportionality principle is worth taking seriously.

12.00-13.10

Mathias Rolffs: *Interventionism, Causal Exclusion, and Proportionality*

Woodward's (2003) original formulation of interventionism arguably confirms a version of the exclusion principle and accordingly leads to an interventionist exclusion argument (cf. Baumgartner 2009, 2013). Woodward (2015) reacts to the interventionist exclusion argument by modifying the definition of an intervention. More specifically, he builds exemption clauses for supervenience bases into the definition of an intervention variable. This amounts to a rejection of the interventionist exclusion principle and to a compatibilist reply to the interventionist exclusion argument.

In this paper, I explore the prospects of developing an alternative reply to the interventionist exclusion argument: Instead of modifying the definition of an intervention variable and rejecting the interventionist exclusion argument, interventionists could accept the interventionist exclusion principle and argue for an incompatibilist reply to the interventionist exclusion argument. As I will show, this strategy against the interventionist exclusion argument at first looks promising, but has two major problems: First, it has to be based on a strong proportionality requirement on causation, which arguably is highly problematic (cf. McDonnell 2017). Second, it cannot properly account for certain common cause structures and thereby leads to unexplained correlations between certain variables (cf. Hoffmann-Kolss 2014). I conclude that there is no viable incompatibilist reply to the interventionist exclusion argument.

15.00-16.10

Vera Hoffmann-Kolss: *Can Counterpossibiles Solve the Interventionist Exclusion Problem?*

In this paper, I explore a new route to solving the so-called interventionist exclusion problem. According to the standard version of this problem, higher-level variables occurring in an interventionist causal model are causally pre-empted by the lower-level variables upon which they supervene because it is metaphysically

impossible to intervene on the higher-level variables while keeping the values of the lower-level variables fixed. The recent debate on interventionism and causal models has shown, however, that such counterpossible interventions occur quite frequently in scientific reasoning and are also less problematic than is usually assumed. But once counterpossible interventions are permitted, there is no good reason to ban them from causal-exclusion contexts. The upshot of the argument will be that even though higher-level properties are not causally autonomous in the standard sense of the word, they are autonomous in the sense that higher-level and lower-level properties enter into different *counterpossible* relations.

16.30-17.40

Ulrich Stegmann: *Biological Specificity Goes Beyond Causal Specificity*

Many biological processes and entities are specific in some way, such as enzymes catalyzing the synthesis of only one substance. Woodward's (2010) notion of fine-grained causal influence (INF), and its quantitative formulation (SPEC; Griffiths et al. 2015), has dominated the philosophical discussion of biological specificity. Indeed, much of the discussion seems underpinned by the assumption that causal specificity (as INF and/or SPEC) adequately accounts for biological specificity. This is a gross simplification, or so I will argue. Biological specificity involves multiple phenomena, and causal specificity captures only one of them. Some instances of biological specificity are best understood in terms of Woodward's (2010) "one cause-one effect notion". But even this is insufficient. I will argue that certain instances of biological specificity require going beyond a cause and its effect. Some instances exhibit peculiarities at the level of several cause-effect pairs, and others are non-causal.

18.00-19.10

Lauren Ross: *Distinctions Among Causation: Causes with material continuity*

In the philosophical literature on causation recent projects have focused on clarifying distinctions across types of causal relationships. These distinctions include stability, specificity, and proportionality (Woodward 2010; Lombrozo et al. 2018). In this talk I suggest that there is another distinction among causation that has yet to receive attention in this literature. This distinction has to do with whether a causal relationship has material continuity or not. I provide an analysis of causal relationships with this feature and I argue that these relationships (1) figure in distinct explanations, (2) are studied with unique methods, and (3) provide special types of causal control.