Workshop: Causality in complex systems (list of abstracts)

Trey Boone: Degeneracy and Causal Complexity in Neuroscience

Degeneracy is a hallmark of causal complexity in biological systems. Broadly, it refers to circumstances in which functions are performed stably despite significant variation in the structures that perform those functions. In neuroscience, degeneracy can be found across all levels of investigation, from individual cells to small circuits to large-scale anatomical networks. It complicates ordinary methods of causal investigation because, in degenerate systems, manipulation of variables that are causally relevant to some function may nonetheless fail to disrupt the associated function. In this project, I relate the challenges posed by degeneracy to core issues in philosophy of causation involving modularity, causal faithfulness, and the incorporation of timescales into causal models.

David Danks & Maralee Harrell: Causality, Levels, and Complexity

Causality is notoriously difficult to identify — or even understand -- in complex systems for a variety of reasons. In this talk, we focus on one natural response to these challenges: namely, shifting the levels of description or analysis for the system. We first show how level-shifting can improve causal inference, modeling, and understanding, even in highly complex systems. However, we then show how this same level-shifting can create a significant new problem, as multilevel causal models often cannot be interpreted in the same ways as standard single-level causal models. We conclude by proposing a conceptual resolution to this problem that highlights important pragmatic elements of causal models.

Tobias Henschen: Complexity and the Aristotelian distinction of four types of causes

This paper aims to identify some of the conceptual and inferential connections and disconnections between important features of complex systems (numerosity, feedback, nonlinearity, spontaneous order, nested structure, adaptivity etc.) and philosophical accounts of (material, formal, efficient, and final) causality: cases, in which these features and accounts seem conceptually connected (spontaneous order and formal causality, adaptivity and final causality etc.); cases, in which they appear inferentially disconnected (cases of extreme sensitivity to initial conditions, certain cases of downward causation etc.); and cases, in which it seems possible to reconnect them if they initially appear inferentially disconnected.

Carl Hoefer: Generic causation in complex, mind-dependent systems

Often, in medicine and social sciences, we are interested finding generic causal facts: facts of the form *X causes Y*, where *X* and *Y* are event types rather than specific individual ("token") events. Typically, we are interested because *X* is something that is at least partially under our control: e.g., an educational policy that can be implemented, or a public health intervention that can be made. The evidence-based medicine and evidence-based policy movements urge that we base medical and socio-political decisions on high-quality evidence that, ideally, strongly supports statements of this form, *X causes* (or *X prevents*) *Y*. It is a presupposition of these movements, and the forms of research they wish to rely on (including RCTs) that such facts about generic causation exist; our job is just to uncover them. But might this presupposition be mistaken, in some cases which are causally very complex? In recent years I have become convinced that this presupposition is indeed mistaken, in at least some contexts that share these characteristics: complexity, strong dependence on initial conditions, and dependence on human behaviour. Using examples from the recent covid19 pandemic, I will illustrate the possibility that certain generic causal facts may fail to exist: it is not correct to assert that *X causes Y*, yet it is misleading to assert *X does not cause Y*. The discussion will bring together ideas from some of my earlier works on causation and on objective chance.

Marie Kaiser: Dealing with Causal Complexity: The Role of the Social Niche Concept

Individual animals differ – not only in their traits, which temperatures they tolerate, and what they feed on, but also in their social behaviors and in the social interactions they engage in. In other words, individual animals differ in their social individualized niches. However, social interactions between animals and the social organization and structure of animal groups are complex, a phenomenon referred to as social complexity (Kappeler 2019). In this talk, I analyze the epistemic role that the social niche concept plays in dealing with the complexity of causal (social) interactions between individual animals. I show that the social niche concept provides criteria that allow the biologists to focus on certain types of causal factors while ignoring others. First, the social niche concept requires identifying one focal individual whose individualized niche is at stake. Second, it guides the biologists to focus on direct causal interactions with the focal individual, and on causal factors that are relevant to the fitness of the focal individual. Helping to manage causal complexity is just one central role that the social niche concept plays in contemporary biological practice, which shows that the niche concept is far from being superfluous (e.g., Wakil and Justus 2022).

Beate Krickel: Integratable or incompatible? Different explanatory approaches in cognitive neuroscience

In this talk, I will first explore the relationship between topological and mechanistic explanations in cognitive neuroscience. While topological explanations emphasize abstract, mathematically defined relationships between nodes, mechanistic explanations focus on causal interactions between parts. This distinction raises a critical question: Can these explanatory approaches be integrated, or are they fundamentally incompatible? Drawing on joint work with Leon de Bruin and Linda Douw, I will argue that many topological explanations function similarly to mechanistic ones. I will present a framework that clarifies the conditions under which topological explanations can be considered complete mechanistic explanations. To illustrate this, I will discuss a neuroscientific case study using multiplex modeling to investigate cognitive deficits in Alzheimer's disease. Finally, I will consider whether this framework offers a general strategy for integrating different explanatory approaches, including computational and representational explanation, with mechanistic explanations in cognitive neuroscience.

James Ladyman: Causality, Complexity and Closure

This paper applies the analysis of the open/closed distinction in recent work by Ladyman and Thebault to reconsider in what way complex systems are open, and how this bears upon the issue of levels of causation and emergence.

Mariusz Maziarz: Causal inference in randomized field experiments – an argument for simplicity

In my presentation, I analyze two aspects of randomized field experiments (RFEs) that may undermine the internal validity of causal conclusions: (1) testing for baseline imbalances and rerandomizing participants to treatment and control groups and (2) the advancement of analytical models and common control for covariates instead of the straightforward testing for the difference in means. I argue that these two approaches to designing and analyzing RFEs inflate the number of researchers' degrees of freedom and may lead to biased results. This is so because preregistration of RFEs is not mandatory, and registrations do not include sufficiently detailed statistical analysis plans. Based on statistical and metascientific literature, I argue that testing for baseline imbalances and rerandomizing, and using complex econometric models to analyze data can be used to p-hack for statistically significant results and hence such practices reduce the quality of evidence stemming from RFEs.

Alessio Moneta: High-level Causation and Causal Inference

Experimental methods for causal inference (e.g. randomized controlled trials) are believed to conclusively identify causal relations in virtue of realizing ideal conditions (Woodward, 2003) that avoid confounding. We observe that many high-level aggregate variables have

potentially ambiguous effects on other variables due to their heterogeneous causal role in the population of interest (Spirtes and Scheines, 2004). We argue that, when heterogeneity is present and when data on individual units are unavailable, experiments provide a much weaker inferential leverage. The reason is that the ideal conditions on which a conclusive inference would depend are in principle unrealizable. Contrary to the case of variables with homogeneous causal roles, the evidence may not conclusively validate an experiment because confounding may never be ruled out. Granting that causal inference may be warranted in such contexts, the problem arises of how exactly it should be justified. We propose a rationalization based on a form of abductive reasoning.

Angela Potochnik: Causes don't push

Complex systems research has shown that many systems of different types and at different scales exhibit similar features. These include robust behavioral regularities that can be described without referencing system specifics, variability in how systems accomplish these regularities, and interdependence among system elements. In this talk, I will explore implications of these developments for our very concept of causation. Specifically, I will conjecture that the model of causation as isolated direct influence, like billiard balls, is deeply misleading. The association of causation with pushing, inherited from the mechanistic philosophy that reined in Newton's day, is reinforced by contemporary science's experimental practices and causal modeling techniques. Yet, consideration of the uses and limitations of these contemporary techniques supports a different conception of causation, what we might think of as a causal web. The persistence of the conception of causation as pushing obscures the expansiveness of causal relevance and, as a result, is virtually inapplicable to the complex systems that comprise our world.

Lauren Ross: Causal complexity and causal distinctions

Causation is central to many scientific aims, but it does not always show up in a single, uniform manner. In fact, scientists routinely distinguish among causes that are proximal versus distal, deterministic versus probabilistic, structuring versus triggering, and among causes that vary in terms of stability, strength, specificity, and speed. While the philosophical literature contains many different definitions of causation, these alone struggle to capture the multitude of causal distinctions found in science. This talk provides a framework for capturing common causal distinctions in the life sciences and it examines the importance of these distinctions for scientific explanation, causal reasoning, control, and communication about causality.