Remarkable progresses in the biomedical sciences in the last decades have led to increased awareness that most diseases are complex, and that their onset and course are often difficult to represent and explain. This has prompted two responses in the health science research community. One is a tendency towards pluralism: an increasing tendency to see diseases and other health states as complex, multifactorial, and generally resistant to being subsumed under a single, general, unifying explanation. The other is an increasing interest in developing models in various domains—of diseases, of crucial mechanisms, and of causal structures, for example. The two tendencies are both responses to an increasingly complex information environment, but in some ways they are at odds with one another, since modeling is usually an attempt to achieve useful simplification, while the other trend—towards admitting complexity—tends to deny that simplification is useful.

We label these two trends the pluralism and modeling trends. Between these two very broad trends in the health sciences—the modeling trend and the pluralism trend—we offer four papers that deal with various aspects of this tension. Paper 1, "Representing and explaining: On modeling disorders" focuses on mechanistic models and their explanatory import. The paper points out that the identification of the explanandum is itself contentious in seeking to explain, and employs examples from neuropsychiatry and cancer research to address the co-evolving of descriptive and explanatory processes in modeling disorders. Paper 2, "Questioning the usefulness of mechanistic models for predicting which medical treatments will benefit humans" argues that the usefulness mechanistic models for discovering treatments that benefit patients has been greatly exaggerated. Paper 3, "DAGgers at dawn? Understanding the potential outcomes 'revolution' in epidemiology", asks what recent developments in causal inference techniques in epidemiology really amount to. The paper argues that "causal models" are neither necessary nor sufficient for good causal inference, which remains a messy, informal business, even with the advent of powerful formal modeling tools. Paper 4, "Pluralism in research on PTSD: Implications for clinical practice", explores the use of pluralistic approaches to investigating post-traumatic stress disorder, and their potential implications for clinical practice.

Format: 4 papers, 20 minutes each plus 10 minutes discussion, tot: 120 min.

Paper 1. "Representing and explaining: On modeling disorders"

This contribution focuses on modeling disorders, and, more specifically, on mechanistic models of disorders and their explanatory import. We address some issues arising from mechanistic modeling of disorders that remain still poorly understood and that are investigated from different, and often distant, disciplinary standpoints.

One particularly thorny problem in the elaboration of explanatory models is the exact identification of the explanandum. In the case of many puzzling disorders, we do not start from a single, accurate and complete description of the system under investigation. The elaboration of the explanatory model rather starts from the choice of a set of characterizing features of the target system, which can be regarded as an array of inter-regulatory subsystems. In the process, any progress in mechanistic understanding of some level further constrains the space of possible mechanisms underpinning the disorder, with descriptive and explanatory processes co-evolving, and correcting each other.

This contribution focuses on the mutual interaction of descriptive and explanatory processes as they de facto occur in medical contexts. We shall highlight some distance between the philosophical debate on mechanistic models and how disorders are actually—and always tentatively—modeled, and the need of further conceptual tools to give justice to the dynamics of modeling disorders at the crossroad of known and unknown systems.

The talk addresses such aspects, stressing how the field in which the investigation is pursued and its purpose shape the kind of questions raised, the methods and tools employed to answer them, and the sort of answers accepted. Some specific examples from neuropsychiatry and cancer research are offered to clarify the continuous and iterative processes at play between biomedical research and clinical practice to identify and describe pathogenetic mechanisms, showing some limits of the mechanistic philosophy in effectively capturing such processes.

Paper 2. "Questioning the usefulness of mechanistic models for predicting which medical treatments will benefit humans"
Roughly 70% of money spent on medical research is spent on basic science (animal, in vitro) studies that investigate the mechanisms of health and disease. The justification for spending the majority of research money on more basic science rather than studies that have a more direct impact on human health (such as randomized clinical trials) appears to be that the more basic mechanisms research eventually leads to treatments that benefit humans.

While there are many important and widely celebrated cases where clinically beneficial treatments have been developed on the back of mechanistic models, empirical evidence suggests it happens far less frequently than is generally believed. The reason for the exaggeration is that the theoretical and empirical problems with evidence of mechanisms are persistently ignored. Moreover when it comes to methods for treatment discovery, mechanism research is not the only — or, as I shall argue, most efficient — game in town. The other method is empirical observation.

To anticipate, I begin by describing how basic mechanistic research might, in principle, help generate hypotheses about treatments that will benefit humans. In brief, the argument is that if we have evidence of a mechanism linking an intervention with a clinically relevant outcome, then the intervention is likely to benefit humans. I then point out problems with (a) the reliability of evidence of mechanisms, (b) the stability of mechanisms, and (c) inferences from evidence of one mechanism to claims that an intervention will have a net benefit for patients.


There is an ongoing “methodological revolution” in epidemiology, according to some commentators. The revolution is prompted by the development of a conceptual framework for thinking about causation called the “potential outcomes approach”, and the mathematical apparatus of directed acyclic graphs (DAGs) that accompanies it. These graphs and accompanying “structural equations” allow “causal models” to be constructed. In principle, these enable testable quantitative predictions to be calculated where previously they would not have been calculable.

In epidemiology, the revolutionaries have made remarkable claims, both positive and negative: positively, that stubborn old problems can be solved; and negatively, that stock causes acknowledged by epidemiology—such as race or sex—are not, properly speaking, causes. In this talk, I seek to understand the real significance of this “revolution”. Specifically, I ask whether these methodological developments are truly revolutionary, in the sense of replacing what went before, or whether they are methodological developments that supplement but do not replace older, vaguer heuristics for assessing causality, such as Hill’s famous “nine viewpoints”. Do epidemiologists need causal models for causal inference, and if so, do they need anything besides causal models?

In reaching the conclusion that the “revolution” is not—or ought not to be—successful, I press two main claims. First, I seek to dismantle the strong and unwarranted metaphysical commitments about the nature of causation that the revolutionaries espouse—commitments leading to the startling conclusion that race and sex, for example, are not causes for epidemiological purposes because they cannot be manipulated. I counter that they remain of central importance in epidemiology. Second, I argue that causal models are neither necessary nor sufficient for good causal inference. They offer a useful additional tool, but not a whole new toolkit for causal inference.


Philosophers of science have recently begun to address the implications of pluralism in science. For example, Helen Longino (2013) has recently surveyed the many scientific approaches used to study aspects of human behavior and has concluded that, while each of the approaches is, because they are so radically different from one another, they cannot be integrated to develop a single comprehensive account of the phenomenon of study. She further concludes that most sciences will exhibit this kind of pluralism.

If Longino is correct, the prospects may be dim for using science to inform practical or policy endeavors. This is particularly true for medicine, which now emphasizes the importance of basing clinical practice on high quality research evidence; in the absence of scientific unity, it is not clear which approaches or models should inform practice. And in the case of psychiatry, which struggles to be seen as scientific, it is an even greater challenge. In this talk, I use an extended case study of research on post-traumatic stress disorder (PTSD) to investigate the implications of scientific pluralism for psychiatry. PTSD has been extensively studied in humans and in animals using a variety of distinct models (e.g., the fear conditioning model, the cognitive model, and, most recently, a model positing a dissociative subtype of the disorder). I consider the evidence offered in support of each of these models to determine whether they converge on a plausible mechanism (or mechanisms) underlying the disorder. I then (1) examine whether they exhibit the kind of irreducible pluralism that Longino predicted and (2) consider the implications of using these different models for clinical decision-making with regard to diagnosis, prognosis, and treatment.