Mactis Abstracts

Ken Aizawa & Carl Gillett: The Autonomy of Psychology in the Age of Neuroscience

Over fifty years ago, H.M. was treated for chronic epilepsy by a bilateral hippocampectomy. Among the lasting side effects of this treatment was that H.M. could no longer form certain types of long term memories, although he could form others. One of the many morals philosophers and psychologists have sometimes drawn from this sad case (and others) is that information about the brain can be used to guide theorizing about the mind. More specifically, it has been claimed that differences in the way in which psychological properties are realized in the brain can be used in the delimitation of distinct psychological properties. In this paper, we build on the Dimensioned theory of realization and a companion theory of multiple realization to argue that the discovery of differences in neurobiological realization do not by themselves lead to the splitting of psychological properties. Such differences in realizers could constitute unique realizations of distinct psychological types or multiple realizations of one psychological type. Whether one has unique realizations or multiple realizations—whether psychological properties are split or not—is not determined by the neuroscience alone, but by the psychological theory under examination. Thus, one might say that, in the splitting or non-splitting of properties, psychology enjoys a kind of autonomy from neuroscience.

Tudor Baetu: Beyond explanatory reductionism and antireductionism: cross-theoretical schema-filling in genetics

Explanatory antireductionism in genetics is the view that molecular biology explains aspects of inheritance not explained by classical genetics. Two antireductionist positions are identified: an integrationist one, stating that classical genetics and molecular biology complete each other by sharing explanatory strategies and experimental techniques; and a radical one, according to which classical explanations cannot be bettered by taking into account molecular details. The aim of the paper is to refute the latter.

In order to understand the differences between the two positions, I make use of a distinction between general schemata of explanation and individual instantiations of mechanisms originally introduced by Lindley Darden. According to Darden, "[t]he general knowledge in molecular biology is best characterized not in terms of laws or a theory but as a set of mechanism schemas", where a "mechanism schema is a truncated abstract description of a mechanism that can be easily instantiated by filling it with more specific descriptions of component entities and activities" (the Machamer-Darden-Craver analysis of mechanisms). I argue that the details of a general explanatory schema can be completed (i) inter-theoretically in order to generate another general explanatory schema, as illustrated by Darden's 'serially connected mechanisms' account of the transition from classical genetics to molecular biology; (ii) intra-theoretically, by filling in phenomenon-specific details using the theory's own internal theoretical and experimental resources in order to generate individual explanations of particular phenomena; or (iii) cross-theoretically, whereby theoretical and experimental considerations from one theory are used to fill in the specific details of individual explanations derived from another theory without altering the general explanatory schema of the latter. Both antireductionist positions allow for (i) and (ii). However, the integrationist interpretation remains silent on (iii), while radical antireductionism explicitly denies it.

Two arguments are mounted in favor of radical antireductionism: classical explanations cannot accommodate the level of complexity revealed by molecular biology (the theoretical incompatibility charge); alternatively, accommodating such complexity does not yield better explanations (the non-improvability charge). I show that the classical functional unit/cistron gene concept entails the same kind of genetic complexity as molecular gene concepts. This indicates that the unbridgeable gap between the simplicity of classical genetics and the complexity of molecular biology is a pseudo-problem. In respect to the non-improvability charge, I show by means of a variety of examples from clinical genetics that molecular elucidations need to be accommodated by changes in classical explanations, and that these accommodations result in increased empirical adequacy, predictive power and applicability to a wider range of genetic phenomena. At the same time, I note that the general explanatory schema of classical genetics is not derived from, reduced to or replaced by the general explanatory schema of molecular biology. I conclude that molecular elucidations have a positive impact on individual classical explanations in a cross-theoretical manner.

Ben Barros: Negative Causation and Natural Selection

This paper examines the nexus of two controversies in the philosophy of science. First, a dispute exists in the philosophy of biology about whether natural selection is itself causal, or whether it is merely a statistical reflection of other events. Second, philosophers of science have engaged in a long-running dispute about whether instances of what might be called negative causation, such as interferences, failures, and absences, are properly viewed as causes.

The two issues come together because many instances of natural selection appear to involve negative causes. Absence of water, for example, can be seen as the cause of the prevalence of drought-resistant traits within certain species of plants. If negative causes are not real causes, then the larger argument that natural selection is causative is seriously undercut. I argue, however, that worries about negative causation should not lead to worries for a causal view of natural selection. This argument has two parts. First, drawing in part on work by Jonathan Schaffer, I argue that negative causation is causation properly understood. Second, I argue that the inherently comparative nature of natural selection addresses concerns about causal promiscuity raised by critics of negative causation such as Phil Dowe and Helen Beebee.

Having concluded that worries about negative causation do not undercut a causal understanding of natural selection, I proceed to consider a related issue: do instances of negative causation undercut the applicability of mechanistic accounts of explanation to natural selection? The leading account of mechanistic explanation, put forward by Machamer, Darden, and Craver ("MDC"), characterizes mechanisms as: "entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions." I have previously argued that natural selection can be characterized as a mechanism using the MDC account of mechanisms. Some instances of natural selection, however, appear to involve absences, raising the difficult issue of how an absence can be understood to be an activity. I argue that absences can be incorporated into the MDC account of mechanisms, but only at the cost of undercutting MDC's robust understanding of activities and their role in mechanisms.

Lorenzo Casini: Social Mechanisms: What Social Sciences Can Learn From Natural Sciences.

Following a trend in natural sciences, social scientists (Elster 1998; Hedstrøm and Swedberg 1998; Bunge 2004; Little 2005; Sawyer 2004; Steel 2004; Squazzoni 2008) are beginning to advocate mechanistic approaches to analyse social phenomena. However, they also claim that social mechanisms are qualitatively different from natural mechanisms. Is this claim justified? After considering ontological and epistemological issues, I answer negatively: social and natural mechanisms are qualitatively similar.

Ontology.

Some social scientists deny the reality of social mechanisms over and above individual actions and interactions in terms of which these are analysed: social phenomena are non-emergent. In addition, all social scientists talk of downward mechanisms—of social properties/structures on individuals (e.g. 'Democratic institutions allow individual participation'); and stress the "intentional" character of social mechanisms—i.e. agents are capable to represent to themselves social structures and consequences of actions aimed at changing these structures.

Current mechanistic accounts, however, can accommodate these features of social mechanisms, so that apparent ontological differences are explained away. Whether or not there is social emergence, no account of ontological emergence (Kim 2005; O'Connor and Wong 2005; Humphreys 1997; Campbell 2009) supports the thesis that this is absent from the social domain but present at underlying levels. Downward causation can be uncoupled into an intralevel causal relation and an interlevel constitution relation (Craver and Bechtel 2007). Intentionality can be causally explained as the emergence of attractor patters for potential behavioural options out of interactions between system's components (Christensen and Hooker 2001; Atlan 1998).

Epistemology.

Social scientists who endorse a mechanistic perspective claim that causal generalisations and explanations, not just narratives or descriptions, are possible in social as much as natural sciences. For instance, the "price-mechanism" (increase in supply → drop in price → rise in demand → price hike) and 'Smoking causes cancer' are general claims, even if they do not always hold. However, the complexity of underlying mechanisms, it is often said, is responsible for a special difficulty in predicting aggregate social behaviours and outcomes of interventions/policies—which amounts to an epistemological, or "weak", kind of emergence (Bedau 1997). Also, it is claimed, social complexity requires different modelling techniques, for instance, agent-based ones, where only individuals and their relations are modelled, and social phenomena emerge as aggregate behaviours.

All this, however, is compatible with current mechanistic accounts. Weak emergence, which regards also natural phenomena, can be explained in terms of degrees of invariance—broader at the individual level, narrower at the social level (Woodward 2003), without undermining the value of social generalisations—which, due to dynamical systems theory considerations, are largely stable in spite of what happens at the lower level. Stability/invariance of social, as much as natural, claims can be tested *in silico* with the aid of "generative models" (Epstein 2006), i.e. agent-based simulations, which under sound assumptions provide explanations for micro-to-macro mechanisms.

These considerations suggest that social mechanisms are not qualitatively different from natural mechanisms. Current accounts of natural mechanisms can accommodate features of social mechanisms usually considered distinctive mark of the social.

Brendan Clarke: Mechanisms, causation and the Russo-Williamson thesis

Russo and Williamson [2007] suggest that causation in medicine depends on both mechanistic and statistical evidence. From an HPS perspective, their account seems to conform well with the process of determining cause in medicine in most instances. In this paper, however, I will present three cases which seem to offer modest counterexamples.

The first – the discovery of McArdle's syndrome – appears to be a case of strong causal inference arising in the absence of statistical evidence. Is, therefore, evidence of mechanism alone sufficient warrant for causation in some cases? The second example – the discovery of the causal relationship between infection with hepatitis B virus and the development of liver cancer – relies almost entirely on statistical evidence, with a much weaker role played by mechanisms. In fact, the mechanisms found in this case are generally plausible mechanism-sketches, based in analogy, rather than anything more confirmed. This example therefore suggests that perhaps mechanism too may be redundant in some cases. The third example details the apparently causal role played by infection with herpes simplex virus in the pathogenesis of cervical cancer. While this relationship was supported by a good deal of strong mechanistic and statistical evidence, the purported causal relationship turned out to be illusory. Risk of herpes simplex infection is merely correlated with risk of developing cervical cancer. Does this case mean that the Russo-Williamson thesis is sometimes unable to differentiate causation from correlation?

My answer to all three instances is a qualified 'no' – that is, given suitable extension of Russo and Williamson's arguments. In very brief summary, I assert that their evidential pluralism is a necessary part of formulating useful causal claims in medicine. However, the nature of both mechanism and statistical evidence employed requires some finessing in order to accommodate causal situations of these types. The first case is disposed of by slightly broadening the requirement for statistical evidence to a requirement of evidence of difference-making in a more general sense. The second case requires an exploration of the nature of mechanistic dependency, in particular in situations of discovery or confirmation of mechanisms. While this section focuses on the role of intervention in mechanism construction, it also requires some discussion of the role of plausible mechanisms and of analogy in causation. The third case requires strengthening Russo and Williamson's argument by requiring that the two types of evidence used must be strongly integrated if they are to support a judgement of causation. I will argue that subtle differences in the methodology of research programmes may grant us an epistemic warrant to do this differentiation. My paper therefore concludes with some suggestions on the nature of integration between mechanistic and statistical evidence.

Russo, F. and Williamson, J. 2007. Interpreting Causality in the Health Sciences. *International Studies in the Philosophy of Science.* 21(2): 157—170.

Mark Couch: Mechanisms and Explanatory Relevance

The notion of a mechanism has become increasingly important in philosophical analyses of the sciences. Many philosophers now accept that explanations that appeal to mechanisms have a fundamental role to play in our understanding of scientific practice (Glennan 1996; Machamer, Darden and Craver 2000). The notion of a mechanism, however, still remains inadequately understood. As Craver explains in his recent book *Explaining the Brain* (2007), a significant problem is that we have only a poor understanding of how to identify the relevant components of a mechanism. Though we recognize a difference between the relevant and irrelevant components involved in a structure's operation, we still lack a philosophically sophisticated account of this distinction. Craver has called this "the problem of explanatory relevance."

This paper will offer a novel answer to the problem of relevance. Consider the various parts of a mechanism that contribute to its overall makeup. Some of these will be relevant to a particular capacity the mechanism performs, and some of these will not. I suggest that we distinguish between the relevant and irrelevant components of a mechanism by attending to the constraints involved in giving a functional explanation of a capacity. In giving a functional explanation, we begin with the capacity of the mechanism we want to explain, and then look for the components within the structure that are needed for bringing the capacity about. In this view the relevant components are the ones that are needed for the performance of the capacity. To explain the notion of "being needed" here, I further suggest that we appeal to resources provided by certain theories of causation. Specifically, I argue that something similar to J. L. Mackie's account of causation and *inus* conditions can do important work in elucidating the notion of a mechanism, although it has so far been neglected by Craver and others. I defend the view that the relevant components of a mechanism are what I term "inus components." A relevant component can be defined as an *insufficient but necessary part of an unnecessary but sufficient* mechanism that serves as the realizer for some capacity. I explain how this proposal clarifies a number of important issues about mechanisms and capacities (including the notion of realization), and provides us with a better understanding of mechanisms in general. Along the way I show how the account enables us to solve the problem Craver describes.

Rachel Coventry: The Endurance of Economic Mechanism: A corparison of Closed Systems and Nomological Machines

On way to caracterise the literature in the philosophy of economics is in terms of the existence of hidden underlying mechanisms. Tony Lawson, for example, argues that the neoclassical orthodoxy in economics fails because it relies on a deductive methodology which cannot illuminate the real mechanisms which underlie economic phenomena. Lawson's work extends Bhaskars realism to the realm of economics. Bhaskars uses a transcendental arument from the intelligibility of scientific experimentation to demonstrate the central importance of causal mechanisms to the activity of science. Lawsons realist conception for economics can be contrasted with more empirical views, such as causal holism which employs van Fraassen's constuctive empiricism to advocate a more cautious view of of economic phenomena, especially with respect to theoretical terms. This paper will argue that while economics should not be limited to descriptions of obserable objects some degree of caution is required. In particular a comarison of Bhaskar's closed systems with Nancy

Cartwrights ‘nomological’ machines will show that economic mechanisms may not be as enduring as is supposed by Bhaskar and Lawson.

Lindley Darden "Mechanisms in Molecular Biology"

The general knowledge in molecular biology is best characterized as represented in a set of mechanism schemas. Diagrams often represent the mechanisms of DNA replication, protein synthesis (schematized in the Central Dogma of molecular biology) and the myriad mechanisms of gene regulation. There is no general theory of molecular biology or laws of molecular biology; such terminology is not used in the field. Nor does the term "cause" get much usage. "Cause" is best viewed as a very abstract schema term that is specified by particular activities. The most important in molecular biological mechanisms include geometrico-mechanical activities: the stresses and strains that change the shape of molecules when their active sites are occupied are much the same as the activities that Descartes employed in characterizing physiological mechanisms. However, modern chemistry has supplied additional activities, unavailable to seventeenth century mechanists, in the form of different kinds of chemical bonding, from strong covalent bonding to weaker polar bonds. Hydrogen bonds form and break rapidly at various stages as the protein synthesis mechanism operates. Molecular biology is a field that supplies paradigm examples of biological mechanisms and it figures prominently as the field providing evidence for the Machamer, Darden, Craver (MDC) view of mechanisms: "Mechanisms are entities and activities organized such that they are productive of regular changes from start or set up conditions to termination or finish conditions." (*Philosophy of Science* 2000), discussed further in L. Darden, *Reasoning in Biological Discoveries: Mechanisms, Interfield Relations, and Anomaly Resolution* (Cambridge University Press, 2006).

Jonathan F Davies: Systems Biology, Mechanisms and the Integration of Localised and Distributed Causal Explanations

Genome mapping projects have provided scientists with unprecedented quantities of data that are valuable for addressing biological questions. However making sense of these data is not trivial and efforts are underway to develop methods that will translate genomic, proteomic and other molecular data into explanations for biological phenomena. Prominent among these techniques is systems biology (SB). I argue that some strands of SB are usefully understood as attempting to integrate two hitherto antagonistic approaches to explaining biological phenomena: localised and distributed causal explanations (LCEs and DCEs).

This paper consists of two main arguments. The first is that that the categories of LCEs and DCEs are useful in the understanding of explanatory approaches that have featured in biology and can help to illuminate the problems of understanding the processes underlying complex biological systems and explaining co-operative behaviour amongst system components in mechanistic terms. The second is that techniques and theoretical insights from SB suggest the possibility of integrating apparently antagonistic approaches to biology.

Making use of notions developed by recent mechanistic philosophers I outline the distinction between LCEs and DCEs. LCEs in biology are characterised by functional localisation and an emphasis on the intrinsic properties of components while DCEs have tended to be pursued by formulating general principle or system-level laws that generate the phenomenon of interest. I

illustrate this distinction with the use of two examples: Jacob and Monod's operon model of gene regulation and Stuart Kauffman's explanation for system stability in gene regulatory networks.

An important strand of SB makes use of techniques and insights from systems theory and complexity science in an effort to model the behaviours of large numbers of interacting genetic and epigenetic components organised in complex systems. A drawback is that available techniques have tended to treat elements (components) of the system as relatively simple and homogeneous and component interactions as of a few types only. It has been argued that models developed on the basis of these techniques cannot accommodate the heterogeneity of biological systems. Instead the data generated by molecular biology and genomics must be understood from the bottom-up. Higher-level behaviours are comprehensible only through an understanding of the properties of the individual molecular components. This has been characterised as a dispute between bottom-up (pragmatic) and top-down (systems theoretic) SB, reflecting the wider dichotomy of reductionism versus emergentism. Against this, I argue that a more helpful way of understanding explanatory strategies in SB focuses on the extent of spatial and temporal distribution of the explanations and that mechanistic DCEs are possible from the bottom-up. Multiple heterogeneous interacting components can be simulated *in silico* so generating DCEs that do not depend on systems-level principles or laws. LCEs and DCEs do not constitute a dichotomy but rather represent the opposite ends of a continuum of explanatory strategies. This analysis allows us to understand how it is that mechanistic explanations can account for "emergent" phenomena without recourse to "levels of reality".

Leen De Vreese: Evidence-based medicine and causal mechanisms

In 1990, the internist Gordon Guyatt introduced the term "evidence-based medicine". Since that point in time, the importance of this idea(l) has steadily grown. By consequence, and given the evidence hierarchy of the EBM movement, randomized controlled trials (RCT's) have been more and more appreciated as the basis for good practice in medicine. There is nonetheless a lot of controversy about the usefulness of stressing this methodology at the expense of other sources of evidence. Critics have raised several problems for an evidence-based approach to medicine. One of the least discussed, but nonetheless highly important, challenges for EBM is how to relate the reliance on the causal evidence from - primarily - RCT's to the importance of insight into the underlying causal mechanisms. Fortunately, in the medical literature and research agenda's, attention for this topic is now emerging. Medical doctors seem to start to get well aware of the need for knowledge of mechanisms on top of evidence from RCT's. We might stand at the edge of a turning point. "Mechanism-based evidence" is more and more appreciated and sought for to fill up evidence from RCT's.

In my lecture, I will briefly sketch the controversies and evolution in the thinking about EBM as a methodological standard. I will highlight some challenges for evidence-based practices that are especially of concern for medicine. Further, I will present some medical examples that illustrate how important knowledge for clinical practice might stay hidden as a result from relying on the outcomes of RCT's without an insight into the underlying causal mechanisms. These examples will also demonstrate that deciding on whether to give priority to (further) RCT's or rather to research into the underlying causal mechanisms is not only a methodological, but also an ethical matter. To conclude, I will argue that the discussion about the relation between knowledge from RCT's and knowledge of causal mechanisms can best

be situated in the strained relation between searching for pragmatically useful information and striving for insight providing knowledge. The difficulty is how to find the right balance in the search for both.

Isabelle Drouet: Propensities, mechanisms and causation

Dealing with physical singular causation, I will focus on propensities in the context of analyzing the relationship between singular causation and mechanims, as well as between different notions of causation. Following Popper's proposal, I will consider a propensity as a natural force that is attached to a physical set-up, that tends to produce a singular event, and whose degree of reality is measured by the probability of this event.

On the one hand, propensities thus characterized may be considered as, or at least they have to do with, mechanisms and they are the vectors of a relation of causal production of singular events by physical set-ups. First I will make these ideas more precise. I will identify the conception of mechanisms under which mechanisms are related to propensities and determine what this relationship exactly is. I will also specify the sense in which the physical set-ups to which propensities are attached can be considered as causal producers of the events that propensities tend to realize. This will clarify the articulation between the notions of mechanism and of causation as far as they are both related to the very notion of propensity.

On the other hand, propensities as characterized by Popper are intimately linked to probabilities, and probabilistic analyses are the most basic of difference-making theories of causality. More precisely, probabilistic analyses make an essential use of the notion of probability-raising, and this notion is generally understood in terms of conditional probabilities. But it is well-known that the propensity interpretation is in trouble for conditional probabilities – this is Humphreys' paradox.

Second I will maintain that Humphreys' paradox can be solved and that possible solutions have the consequence that analyzing causation as probability-raising leads to a version of the counterfactual analysis of causation. In other words, my contention will be that counterfactual analyses of causation can be seen as versions of the probabilistic analysis for the case of physical singular causation.

In this context, it will appear that propensities are related by definition to a mechanistic notion of causal production as well as involved into difference-making analyses of physical singular causation. Third and finally, I will characterize more precisely the relationship between these different notions of causation as far as they have to do with propensities and, starting from this characterization, I will try to draw more general conclusions concerning the relationship between mechanistic and difference-making notions of causation.

Ruben Flores: Mechanisms and Causality in Sociology

In recent years scholars interested in the philosophy and theory of social science are increasingly paying attention to questions of causal inference and mechanisms (Cartwright 2007; Russo 2009). Arguably, the social sciences are no exception to the Aristotelian observation that “knowledge is the *knowledge of causes*.” (Russo 2009: 6; author’s emphasis). Indeed, there is hardly any subject within these disciplines where causality does not matter. From poverty and inequality to financial crisis, from the workings of global capitalism to

science policy, from epidemiology and public health to development, we often ask the question 'why?' That is, we demand explanations and, in so doing, we invoke causality in some way or another. Nevertheless, social scientists are in far from one mind about the role that causal inference plays within their disciplines. Celebrated by some as essential – harshly criticised by others as inadequate – the concept of causality is a controversial issue here (cf. Abbott 1997; Rhoads 1971).

The purpose of this paper is to explore the way in which mechanisms relate to causality within one of the social sciences, namely, sociology. How is causality understood within sociology? What is the role of mechanisms within causal explanations in sociology? How does the understanding of causality and mechanism within sociology compare with such understanding in other social sciences and indeed in other disciplines? What kind of ontological, metaphysical and epistemological assumptions are routinely made when we talk about causality in mechanistic terms within sociology? What kind of social phenomena are suitable to be explained in mechanistic terms? In search for answers to these questions, I shall review a number of key writings in the areas of historical sociology and social theory. In so doing, I aim to throw light at the connection between mechanistic causality and fundamental categories of sociological thinking such as micro-, meso- and macro- levels of analysis, agency and structure, individuals and society, systems and complexity, and intentionality and contingency.

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Phyllis McKay Illari and Jon Williamson: Function and Organization: comparing the mechanisms of protein synthesis and natural selection

Mechanistic explanation is ubiquitous in many sciences, and particularly important in the biomedical sciences. Many biochemists see their job as investigating the mechanisms responsible for protein synthesis, while many evolutionary biologists studying the adaptation of populations to their environments treat natural selection as the mechanism responsible for adaptation. Our question is whether the kind of explanation is the same in these two apparently quite different fields.

We begin our paper by examining experimental work aimed at identifying the mechanisms of protein synthesis and natural selection. The aim is to use detailed examination of experimental work to put the comparison of the similarities and differences of mechanistic explanation in these two fields on a firm footing. We argue that, on the face of it, the two fields have significant methodological differences. To give just one example, detailed investigation of microstructure is crucial to protein synthesis in a way that cannot be exaggerated, with breakthroughs in understanding often being precipitated by the development of technological advances in techniques for seeing molecular structure. This way of finding a mechanism is quite absent from evolutionary biology. Further, the kinds of explanations given in the two fields are different. Natural selection is quite mathematical, with probability a key concept. Populations are modelled statistically, using approximations

over large numbers of individuals. Graphs, equations and tables of data are common parts of explanations. Protein synthesis is concerned with physical interactions of molecules, binding, interlocking and breaking each other. The phenomena of protein synthesis are most commonly explained using diagrams showing the structures and interactions of these physical things.

We argue that despite these appearances, there are important analogies between the structure of mechanistic explanation in these two fields. We identify three core and related elements of mechanistic explanation: functional individuation, hierarchical nestedness or decomposition, and organization. These are now uncontroversial elements of mechanistic explanation in fields such as protein synthesis, but it is difficult to see how natural selection involves decomposition, or organization. Indeed, Skipper and Millstein (2005) argue that neither notion applies unproblematically to natural selection. We take each in turn and show how a more careful examination of how decomposition and organization actually works for protein synthesis allows an analogous understanding of the kind of explanation offered by natural selection.

George Kampis: Mechanisms as Constrained Systems

Ever since its introduction in the recent literature, the concept of mechanism has often been considered (and heralded as) an alternative to the old-fashioned deductive-nomological scheme of explanation (DNS). The idea is that DNSs construe explanation on a logical (or mathematical) ground, whereas mechanisms use causality as their basis of explanation. Further, in mechanisms, objects play a distinctive role, whereas the DNS uses variables. And so on; there seems to be a gap here, and if that is the case, the research question is how to utilize (and maybe characterize) this gap best – as is elaborated by many authors.

I suggest that there is a natural alternative to this view of mechanisms, one that puts mechanisms back into the DNS order, while retaining their status as being extraordinary; moreover, that on this view, mechanisms can be characterized as a well defined class of natural systems (namely those that permit a mechanism-based explanation due to some operationally identifiable properties).

The idea expounded here rests on the notion of constraint in systems. Constraints are relations that, as the name suggests, constrain the possible motions and processes, in such a way that makes possible outcomes more predictable, even in the absence of initial conditions or calculations based on covering laws. A typical example is rails: on a rail, motion is only possible along certain directions, and not others. A still better example is rotation; it marks an invariant axis that constrains the motion of the rolling wheel in a like fashion. In terms of DNS, this means that besides initial conditions and natural laws, further contingencies play a role, and that these contingencies can have a significant, and often overlooked role. As a limiting case, we get totally constrained systems, where constraints alone can determine outcomes (in the presence of the natural laws but not requiring them for the prediction and/or explanation of the processes).

I am going to suggest that the typical examples for mechanisms, on which the very concept was rehearsed (such as mechanical or molecular systems), pertain to this class, and I offer to characterize them as cases of reduced (contingency-based) DNS forms, or, from a logical point of view, as enthymemes.

This line of ideas originates from works of H.H. Pattee, R. Rosen and others, many of whom were members of the Chicago Committee on Mathematical Biology, at a place and time when W. Wimsatt, one of the forerunners of the mechanism concept developed his own ideas. I am going to also make a historical tour to make the case that the two concepts, those of totally constrained systems and mechanism, may have a common origin, and later developed in different directions.

Peter Fazekas and Gergely Kertesz Mechanisms and the Metaphysics of Causation

A mechanistic explanation accounts for a phenomenon in terms of component parts, their interactions, and spatial-temporal organization. Causation, thus, plays a fundamental part in mechanistic explanations: what component parts do, how they interact with each other is one of the main aspects of the explanation provided.

Moreover, mechanisms come in levels—it is a *higher level* phenomenon what is explained in terms of its *lower level* parts, where the tasks these lower level parts perform are further explainable in terms of the interactions and organization of still lower level parts (etc.). Therefore, when describing a mechanism, this approach *evokes causation on every level of analysis*.

Nonetheless, what proponents of mechanistic explanations evoke *scarcely ever* are metaphysical questions regarding the nature of causation. One might find it strange though, given that at the same time they aspire to such honours as maintaing the autonomy of special sciences, or making downward causation intelligible via mechanistically mediated effects.

Consider e.g. the metaphysical problem of *causal preemption*. It is a serious threat for any approach evoking causation on different levels of organization or composition. The threat comes in the form of a dilemma. On the first horn, the composite entity has no causal power distinct from the relevant causal powers of its component parts. The lower level parts do everything there is to do in order to perform a certain task, which makes the higher level entity causally impotent (preemption happens)—or at best, identical with a certain bundle of lower level entities. This implies that there is genuine causation only at the bottom level, and forces us to interpret higher level causal talk in an *instrumentalist* way. On the second horn of the dilemma, there is genuine causation on higher levels as well (no preemption). That is, higher level entities have certain causal powers which are *over an above* the causal powers of their component parts. In this case, however, we are forced to endorse emergentism, or a similar view claiming that there are entities which are *more* than mere composites of certain parts.

The present paper shows that this dilemma poses a serious problem for the proponents of mechanistic explanations, *if* they want to claim that their approach is effective in accomplishing such goals as maintaing the autonomy of special sciences, or making downward causation intelligible. On the one hand, it seems to be a central tenet of the mechanistic approach that in those cases which are explainable by mechanisms there are no causal powers at higher levels unanalyzable in terms of the causal powers of the entities at the lower level. Since this claim is incompatible with the second horn of the dilemma, it undoubtedly puts the approach onto the first horn. On the other hand, maintaing the

autonomy of special sciences, and making downward causation intelligible, seem to be incompatible with the first horn. This paper argues that the dilemma cannot be resolved without giving up certain bits of what mechanistic explanations would like to achieve.

Meinard Kuhlmann: Mechanisms in Dynamically Complex Systems

In recent debates mechanisms are often discussed in the context of complex systems, with biological examples, for instance concerning neuronal processes, as paradigmatic cases. Complex systems of this kind have a complicated compositional structure. I want to draw the attention to the fact that there is still another, radically different kind of complex system, in fact one that many scientists—in particular in the physical sciences—regard as the only genuine kind of complex system. Instead of being compositionally complex these systems rather have highly non-trivial *dynamical* features, on the basis of structurally simple arrangements of large numbers of non-linearly interacting constituents. To be sure, I want to call this kind 'dynamically complex systems'. One example, which has been studied extensively in statistical physics, is the ferromagnet with a surprising dynamical behaviour despite of the fact that it consists of nothing more than a simply array of numerous identical dipoles. Analyses of dynamically complex systems are by no means limited to physics. For instance, it is common practice to model socio-economic contexts by using dynamical multi-agent systems, which deal with 'microscopic' agents in a very simple arrangement and with a very simple individual behaviour.

Whereas for a compositionally complex system it is usually feasible to predict its behaviour once the compositional structure and the behaviour of its parts is known, this is completely different in the case of dynamically complex systems. Here the knowledge of the compositional structure, e.g. agents with only two possible behaviours arranged on a square lattice, together with the knowledge of the behaviour of its parts in isolation as well as in simple composites, often allows for hardly any straightforward predictions of the dynamical behaviour of a given complex system. Nevertheless, an ensemble of similar complex systems can exhibit statistical characteristics the robustness of which calls for an explanation in terms of underlying generating mechanisms. Thus, not only for compositionally complex systems, but also for dynamically complex systems the identification of generating mechanisms is essential in order to explain their often surprising behaviour.

Unfortunately, I want to argue, dynamically complex systems are not sufficiently covered by the available conceptions of mechanisms. Whereas for mechanisms in compositionally complex systems the decomposition into modules is an essential and non-trivial task, it is usually a non-issue for dynamically complex systems. Instead, the recognition and detailed (statistical) analysis of dynamical patterns becomes one main task, besides the identification of generating mechanisms. The most important novelty, however, is that the material nature of the mechanisms' parts in dynamically complex systems is irrelevant in a far more drastic way than in many classical biological mechanisms, for instance. For this reason, mechanisms in dynamically complex systems must be construed in a more abstract structural fashion. Despite of these and other differences, it is still appropriate to talk about 'mechanisms' in both cases, that is for compositionally as well as for dynamically complex systems, since, among other things, in both cases the interaction of parts and the robustness regarding the resulting behaviour of the composite system are essential, albeit both features need to be filled in a different way.

Jaakko Kuorioski: Varieties of Modularity for Causal and Constitutive Explanations

Many current writers define mechanisms to be causal structures and then use the invariance under interventions –account of causation to characterize what it is to be a causal structure. The intervention–account of causation imposes a modularity constraint on causal structures: a local intervention on a part of the system should not disrupt other causal dependencies (sub-mechanisms) in the system. This constraint has evoked criticism against the account, since many ordinary causal mechanisms seem to break this condition. This paper answers to this criticism by making a distinction between a causal system and a causal structure. A causal system is a spatiotemporally delineated piece of the world or a tangible set of phenomena that has a boundary/interface with its environment that is recognizable without knowing the inner causal workings of the system. A causal structure (a mechanism) is a set of causal relations within a system, definable only against a background of causal properties ignored or held fixed. It makes sense to ask what the modularity properties of a given causal structure are, but not whether a causal system is modular *tout court.* The counter-examples to the invariance-account turn out to be cases of systems in which a particular structure (or the corresponding model of that structure) is *modular in variables*, but not *in parameters*. A failure of parameter-modularity does not by itself threaten the causal interpretation of (the model of) the structure, but it does mean that knowledge of the structure is not sufficient to constitutively explain system-level properties of the embedding system.

Adam La Caze: Basic Science in Evidence Based Medicine

Proponents of Evidence Based Medicine (EBM) argue that therapeutic decisions should be informed by applied clinical research (Evidence-Based Medicine Working Group 1992). Proponents of EBM do not provide an explicit account of the role of basic science in medical decision making|and the little they do say is contradictory. Operationally, however, EBM urges clinicians to base decisions on the outcomes of large randomised studies rather than the mechanistic understanding of pharmacology and physiology provided by basic science. On EBM's account, mechanism should play a minor role in therapeutic decision making.

A focus on applied clinical research for therapeutic decisions is reasonable if understood in a certain way. A randomised interventional study showing that a therapy is e_ective in patients under routine care can provide more compelling evidence for therapeutic decisions than pharmacological evidence that a drug works by a mechanism understood to treat a disease. This is because much is unknown in clinical science; many drugs have promising pharmacological properties that, for one reason or another, do not bring about the expected beneficial outcome in patients. In clinical science, pharmacological mechanisms sometimes predict patient outcomes, and sometimes they don't; in any particular instance, it is often unknown which will be the case until applied clinical studies have been conducted.

The mechanistic understanding provided by the basic sciences may not be causally relevant in the context of clinical care, but EBM's account of medical evidence fails to recognise how interrelated basic science and applied clinical research are. This causes problems for clinicians. Without an account of the role that basic science plays in interpreting clinical research, judging whether the results of a randomised study are relevant to a patient in the clinic is impossible. The problem of judging the relevance of clinical studies to individual patients is the problem of `external validity'. Despite 1 being well recognised, there is precious little in the EBM literature on how the problem of external validity might be overcome.

Philosophy of science can provide assistance. One way the relation between basic medical science and applied clinical research can be understood is by recognising the series of intermediary theories (and models of these theories) which exists between raw observation and higher level mechanistic theories. Patrick Suppes' (1962) `hiearchy of data models' provides an account of experimental inquiry along these lines. I follow aspects of Suppes' work to provide a framework for the role of theory in EBM. The hierarchy of data models illustrates that the mechanistic understanding provided by basic science is central to the design, analysis and interpretation of applied clinical research.

I use the hierarchy of data models, and the account of the relation between theory and data provided, to capture the arguments that are made in analysing data from clinical research. The hierarchy of data models explicates aspects of the analysis that are too often left implicit or neglected by EBM. Indeed, using the hierarchy of data models, I show that the mechanistic understanding provided by basic science often plays a pivotal role in applying the results of randomised studies to individual patients.

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Jan Lemeire: Causality in the Mind versus Causality in the World.

In trying to define causality, it seems that neither of the current approaches is able to explain all cases in which we call something the cause of something else. We will argue that we have to make a difference between the concept used with respect to a mental model and the concept used to denote things in the physical world. We will show that many of the known counter examples to current theories about causality can be explained by making this distinction. In the physical world, causes are closely related to mechanisms. In a mental model, a cause is relative to the knowledge already present in the model. The model consists of facts and production rules by which the mind tries to mimic what is going on in the world, in the way simulations do. A cause of an event consists of the facts and/or laws that should be added to the model such that it makes the occurrence of the event more plausible. The definition relies on a counterfactual statement; would we omit the cause, the effect would not occur according to the model. It explains causation by omission. Furthermore, a mental model allows to reason about the world, it allows to reason with hypothetical ideal interventions.

Bert Leuridan: Causal relations, constitutive relations, and interlevel experiments.

Complex systems mechanisms are commonly defined such that a mechanism's higher-level behaviour is 'realized by' the behaviour of its lower-level parts [1-6]. This general characterization gives rise to two questions. Q1: how should this lower-level behaviour be characterized? Q2: what do we mean by the phrase 'realized by'? I will show that there is a tension in the literature between one of the most popular answers to Q1 and a well-known answer to Q2. This tension is explicitly present in Craver's *Explaining the Brain* [6].

One of the most popular answers to Q1 is that their behaviour is best described by *invariant generalizations* [see 4 and 5]. According to Woodward [7], a generalization is causal if and only if it is invariant (remains stable under ideal interventions). That is, (actively) changing the presumed cause variable results in a change in the presumed effect variable predicted by the generalization. This view is explicitly endorsed by Craver [6, chapter 3].

Craver and Bechtel's [8] answer to Q2 is that the relation between a mechanism's lower level and its higher level is constitutive. They explicitly argue that a mechanism's interlevel relations are *not* causal. At the same time, they acknowledge that interlevel experiments (this term is used in [6]) "involve either intervening to alter a component of the mechanism and observing the behavior of the mechanism as a whole or intervening to alter the behavior of the mechanism as a whole and observing the behaviors of one or more of its parts" [8, p. 554]. This suggests that constitutive relations can be described by invariant generalizations.

Craver [6, chapter 4] considers *mutual manipulability* as characteristic for these non-causal constitutive relations. It is inspired by Woodward's concept of invariance, but makes use of a distinct notion of ideal intervention.

I will critically assess the notion of mutual manipulability and of non-causal constitutive relations. To begin, mutual manipulability implies invariance (in line with what the quotation above suggests). Therefore, it seems it needs to be (non-trivially) modified. Craver [6, chapter 4] cites three reasons why constitutive relations are not causal: they are symmetric, synchronic, and involve logical dependence (due to the componency relation). However, I will argue by means of the ideal gas laws that some causal relations are symmetric and synchronic, at least up to the limits of chronometry. Moreover, componency relations are not always known at the start of (interlevel) experiments, so that one lacks crucial information to rightly distinguish between causal and constitutive relations. In sum, Craver and Bechtel's insistence on the non-causal nature of constitutive relations is partly ill-founded and it unnecessarily adds inferential challenges to interlevel experiments.

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Juan Montana: Break the Toaster: Manipulationist Descriptions of Mechanisms

Manipulationism and the mechanistic account of causation are the two most prominent theories of causal explanation today. Despite their differences, there is a strong consensus that the two approaches can be integrated. Many assume that the integration can be easily carried out by representing mechanisms using structural models (Woodward, 2002; Craver, 2007; Steel, 2007, Glennan, 2002). The basic idea, to which I'll refer as the *Simple Translation View* (STV), is that mechanisms can be translated into structural models by having each variable represent a part of the mechanism and the equations represent their relations. This paper argues against the STV: The variables in structural models cannot be used to represent parts of mechanisms, they can only represent what I call 'attributes'.

First, parts and attributes belong to very different ontological categories. Parts are mereological components of mechanisms. As such, they are spatiotemporal particulars. Attributes, on the other hand, are determinables that can take different determinates. I will give a more detailed characterization of these notions below, but for now the distinction can be appreciated with an example. The wheels of a car are a part of the car. The speed of the car, on the other hand, is not a part. We may take away the wheels, but not the speed: the speed can be zero or change from 40 to 50 mph (different determinates), but the car will always have a speed (a determinable). The wheels cannot take any values.

Second, and more importantly, accepting the STV is self defeating. We want the STV to be able to express mechanistic explanations in the language of structural models used by manipulationists. But if the STV is true, this language would be of no use to us. As I will argue in detail, manipulationists are essentially committed to the idea that all structural models used to make causal claims must be modular. According to this view, a structural model failing the modularity requirement is not a correct structural model of a non modular causal system; on the contrary, it is an incorrect representation of a causal system. However, structural models that result from the application of the STV to a mechanistic description of a causal system (if at all possible) are poised to fail the modularity requirement.

The first section of this paper introduces the manipulationist account and discusses the modularity requirement and its role in this framework in considerable detail. The second section sketches the mechanistic account and presents my first objection to the STV: that parts and attributes belong to very different ontological categories and as such we have prima facie reason to think that there is little hope for the STV. Finally, the third section discusses STV's problems with modularity. Here I will focus of Cartwright's (2001) famous objection to modularity and show that it depends on a tacit endorsement of the STV. Once the STV is rejected, however, modularity is easy to attain.

Gry Oftedal: Mechanisms versus Difference-Making in Molecular Biology

There are at least two central assumptions regarding causality in genetics and molecular biology: (1) a cause makes a difference to the effect, and (2) there is a causal mechanism that links cause and effect.

The first is recognized in how experiments generally are set up. A goal is to keep all relevant factors constant except for the factor(s) we are interested in. This factor is then manipulated so as to vary among the experimental population and the control population. If the variation in the relevant factor correlates with a variation in the investigated effect, we have a strong indication that the factor is a cause of the effect. Thus, a general experimental set-up is based on the view that a cause makes a difference to the effect.

The second assumption is recognized in how much effort is put into the search for causal mechanisms. It is not scientifically satisfying just to find that the variation in a factor x causally influence the variation in a factor y. A description of *how* variation in x causes variation in y is needed in order to explain the effect and prove the causal relationship. In genetics and molecular biology such a causal mechanism is typically given as a description of a continuous and dynamic chain or network of interactions between objects on different levels, connecting the proposed cause and the relevant effect.

These two scientific approaches parallel two lines of inquiry in the philosophical causation debate. On the one hand we have difference-making/counterfactual theories of causation. On the other hand there are production/physical connection views of causation.

Although these views traditionally are seen as competing theories of causation, some harmonizing attempts are made, e.g. by defending causal dualism (Hall 2004) or by arguing that counterfactuals and mechanisms work in tandem to give a better understanding of causation (Psillos 2004). I find the idea of harmonizing counterfactuals and mechanisms interesting based on the observation that both approaches are prominent and seem to build on each other in genetics and molecular biology. However, there are significant problems with the mentioned harmonizing attempts that I will discuss, and I will make a renewed harmonizing effort based on the causal assumptions often made in the relevant biological disciplines.

Psillos holds that counterfactuals are more basic than mechanisms insofar as mechanisms are depend on counterfactuals and not the other way around. I dispute this claim and develop the view that counterfactual approaches and theories are about our *access* to causation, while causal mechanisms approaches are about *explanation* of causal relationships. Additionally, mechanisms are informative of causal structure, but not of causal strength, while difference-making approaches can be informative of causal strength and causal priority, but not of causal structure. Difference-making theories and production theories of causation are not necessarily competing theories of causation, rather, they discuss different types of causal information that well could be about the same “causation in the objects”.

Relevant literature (discussed in the paper):
Craver (2005, 2006), Dowe (2000), Glennan (1996, 2000), Hall (2004), Lewis (1973, 2000), Machamer et al (2000), Menzies (2004), Psillos (2004), Salmon (1998), Waters (2007), Woodward (2002, 2003).

Viorel Pâslaru: Causality and Mechanisms in Ecology

An examination of research by ecologists reveals that they increasingly think of ecological explanations as descriptions of mechanisms that produce phenomena under scrutiny. Philosophers of science have largely ignored this mechanistic approach of ecologists. In this paper, I scrutinize what is arguably one of the most important ecological mechanisms – competition. I offer an account of this ecological mechanism and draw some implications for the mechanism – causality relationship.

Ecologists characterize competition at the individual-level as interaction between individuals and at the population-level as interaction between species for a shared resource in limited supply. Several ecologists (e.g., Gause 1934, Tilman 1987) have argued that the phenomenon of competition must be approached mechanistically, meaning thereby that one has to use information on the physiology, morphology, and/or behavior of individual species or functional groups to predict the outcome of interaction for limited resources.

I contrast ecologists' description of the mechanism of competition with the new mechanistic philosophy (NMP hereafter) consisting of the conceptions of Glennan (1996, 2002), the team of Machamer, Darden and Craver (2000), as well as that of Bechtel (2006; Bechtel and Abrahamsen 2005). I argue that due to its focus on the specific instances of causation, NMP is able to account for componential, organizational and phenomenal aspects of the mechanism of competition at the individual and population levels. However, it fails to characterize that ecological mechanisms operating at these levels are invariant and insensitive to various changes in their constitution and background. To account for this characteristic of mechanisms, I show that one should rely on Woodward's (2006) conception of invariant and insensitive causal relationships. Given these considerations, I offer an account of ecological mechanisms in terms of stable networks of invariant and insensitive causal relationships that are instantiated at the individual as well as at the population level. Additionally, I show that specification of these relationships requires determining the functions and properties of causal components. Furthermore, I suggest that any account of mechanisms is incomplete without a conception of invariant and insensitive causal relationships that make up the underlying structure of mechanisms.

Alexander Reutlinger: A Modified Interventionist Theory of Causality and Mechanisms in the Social Sciences

In the recent debate on causation, the interventionist approaches (see Woodward 2003; Hitchcock 2001; Halpern & Pearl 2005) appear to be an attractive position. Interventionist theories are especially promising with respect to the social sciences (and other special sciences) because, unlike other theories of causation, they do not rely on (universal) laws of nature. This clearly is an advantage, because the existence of such laws can be reasonably doubted in the special sciences. Rather, one may characterize the generalizations used in the social sciences as *ceteris paribus* laws (see, e.g., stability theories by Lange 2000, Woodward 2003).

Although being adequate for the social sciences by not presupposing universal laws, interventionist theories face other difficulties. Let me point out a serious problem: Interventionism is a counterfactual theory (as its proponents themselves claim). According to interventionism X causes Y (here X and Y stand for random variable, $\{x_1, \ldots, x_n\}$ and $\{y_1, \ldots, y_n\}$ stand for their possible values) iff the following two counterfactuals are true:

> (i) If the value of X would change to be x_i (by an intervention), then the value of Y would change to y_i – and all variables that are not on the path in question between X and Y are held fixed.
>
> (ii) If the value of X would be changed from x_i to x_j (for $x_i \neq x_j$) (by an intervention), then the value of Y would change – and all variables that are not on the path in question between X and Y are held fixed.

Surprisingly, the standard account by Woodward (2003) lacks a theory of truth conditions for those decisive counterfactuals. Unfortunately, this problem is usually not noticed (Psillos 2007 is an exception).

The easiest way to solve the problem is to provide proper semantics. Following Psillos (2007) slogan *No Laws In, No Counterfactuals Out* I propose to use either traditional Stalnaker-Lewis-Semantics (which operates with a similarity relation between worlds) or a theory of truth conditions as it was proposed recently in the debate on counterfactual theories of causation (see Pearl 2000, Hüttemann 2004, Maudlin 2007, Loewer 2007). Both kinds of semantics have the same upshot for an interventionist theories of causation: The theory of causation has to rely directly on laws. These laws may be *ceteris paribus* laws. The *counterfactual theory plus semantics* I call a *modified* interventionist theory of causation.

Relying on laws, the step from a modified interventionist theory of causation to a theory of mechanisms is a small one. Glennan (2002: S344) defines mechanisms as follows:

> 'A mechanism for a behavior is a complex system that produces the behavior by the interaction of a number of parts, where *the interaction between parts can be characterized by direct, invariant, change-relating generalizations*.'

From a modified interventionist theory's point of view, mechanisms appear to be entities that are described by a special sort of causal statement (see, similarly, Psillos 2004). Building on Glennan's definition, I want to argue that a *mechanistic* causal statement (a) refers to the state(s) of a complex system and (b) relies on (temporal) laws describing the interaction of the parts of a complex system. Thus, the laws we refer to in the semantics of counterfactuals are laws of interaction. This theory of mechanisms fits the debate in the philosophy of the social sciences (see Bunge 1997; Elster 1998).

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Attilia Ruzzene: The relevance of mechanistic evidence in case-study research

Pluralism about evidence for causal claims has often been defended in the recent philosophical literature. Unfortunately there are few detailed accounts of how evidence from different sources should be integrated and used in a way that is mutually supportive. The overall aim of this paper is to discuss one case in which such an integration has been managed successfully.

Social scientists who engage with case-study research typically explain phenomena of interest by offering historical narratives. A historical narrative spells out the particular sequence of events leading over time to the phenomenon in question, and the causal relationships among them. The outcome of interest is thus accounted for by describing how events in the chain are causally related in such a way that it is finally brought about. Hence, for a historical narrative to be specified knowledge of causal character is first needed. In this paper I argue that the integration of causal evidence of various types might remarkably facilitate this task. In particular, the employment of different methods of causal analysis endows the researcher with evidence that, if combined, jointly structures the causal backbone around which the historical narrative is ultimately articulated. In this respect, knowledge about mechanisms might be relevant in conferring causal order on the morass of factors the researcher has to deal with.

To illustrate my claim I refer to a case-study research by regional planner Annalee Saxenian which explains why at the end of the Eighties, after parallel paths of growth and decline, Silicon Valley and Route 128 experience diverging economic performances. Saxenian offers a historical narrative that rests on causal evidence the small-n method of comparison and process-tracing jointly provide. These two methods detect causal relationships among factors that allow to fully specify the historical narrative: the events leading to stable growth in Silicon Valley and gradual decline in Route 128 are finally recounted as they might have happened had these causal relationships been in place. Jointly employed in finding causes in the case study in question, the small-n method of comparison and process-tracing play well-distinct roles that turn out to be complementary. The former provides the researcher with the factor responsible for the observed outcome. In so doing, it gives the enquiry a start and a possible point of departure for the historical narrative. The latter identifies the causal mechanism connecting the purported cause to the outcome of interest. In this way it gives the morass of variables causal order and fully specifies the historical narrative. In this paper I will devote particular attention to investigate how process-tracing helps the detection of causal mechanisms by identifying patterns, that is, stable associations of type-like events. Furthermore, I will examine how in the case of interest the relevant mechanism relates to the sequence of causally connected events the historical narrative ultimately recounts.

Predrag Sustar: Natural Selection as a Process-Mechanism: A Tip from Cancer Research

In this paper, I will focus on the following question: how should we best characterize Darwinian *natural selection*? In particular, I will examine the prospects of a *mechanistic* answer to that question, such as one defended in Barros' recent paper (Barros 2008). With the account advanced in that paper, the so-called 'new mechanistic philosophy of science' has made its most explicit contribution to the issue of *evolutionary mechanisms* and has joined to the debate between the two leading philosophical interpretations of the nature of Darwinian

selection, i.e., the *force* and *consequence interpretations* (for an overview of these two interpretations, see Brunnander 2007).

In answering the above question, I will proceed as follows: in the first part of the paper, Barros' account of natural selection as a mechanism in a rather strict sense will be more closely analyzed. Accordingly, since Barros' corresponding account works out a more comprehensive version of the so-called MDC account of mechanisms (see Machamer, Darden, and Craver 2000), and rejects the other main mechanistic account (see, e.g., Glennan 2002), relevant underpinnings of putative evolutionary mechanisms will also be addressed to a certain degree.

In the second part of this paper, natural selection – considered as one of the main 'ingredients for human cancer disasters' (see Alberts *et al.* 2007) – will be described. Human cancer as a 'micro-evolutionary process' (see Merlo *et al.* 2006) offers in that regard suitable data resources for at least two related reasons: (i) the onset of human cancers provides a more direct access to evolution by natural selection, which draws on a determined time range of human life; (ii) the proximity of natural selection in tumorigenesis and, more broadly, in cancerogenesis to the biological mechanisms in a more restricted sense. Both reasons will help in making progress toward a satisfying answer on what, after all, is the nature of natural selection. In that regard, I will argue in favor of the view according to which Darwinian natural selection is most adequately understood as a certain kind of *process-mechanism*. The final section of the paper will expand on that particular point given the evidence in human cancer research.

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Elanor Taylor: Systems and Non-Mechanistic Explanation

In this paper, I present a case study from cognitive science, the Haken-Kelso-Bunz model of phase transitions in finger movements, and suggest that the explanatory role of this model is to offer a 'systems explanation'. A systems explanation is a form of scientific explanation that proceeds by profiling the activity of the interactive system that gives rise to the phenomenon of interest. This directly challenges the view, offered by Bechtel and Craver, that explanation in cognitive science, and indeed scientific explanation more generally, proceeds by uncovering mechanisms.

The Case Study[1]

The HKB model describes the following feature of human finger movements: when a person moves their index fingers from left to right across the same plane and at the same frequency, there are two possible 'gaits' or phases of movement, in-phase and anti-phase. Once a certain

[1] Kelso, 1995. *Dynamic Patterns: The Self-Organization of Brain and Behavior*, MIT.

frequency of oscillation is reached, however, the movements converge onto in-phase. The HKB model is a dynamical model, a set of equations that describe this phenomenon as the evolution of a dynamical system. Some dynamical systems have phase spaces with *attractors*; these are states of a system that are simply more popular than others, towards which the system converges. A *bifurcation* occurs when a control parameter reaches a critical point that changes the attractor layout. In the HKB experiment, the system at standard frequency has two attractors – in-phase and anti-phase. When the frequency of oscillation reaches a key value a bifurcation occurs and a system with two attractors becomes a system with one.

Bechtel and Craver on Mechanistic Explanation

Bechtel's account of mechanistic explanation is inherently decompositional[2]. He addresses questions about the place of dynamical explanations in a mechanistic context, and argues that the appropriate place for dynamical explanation is in illustrating features of a mechanistic system[3]. I'll argue that subsuming dynamical explanation under mechanistic explanation in this way misses something important about the explanatory role that systems-level explanations can play in science. I'll argue that Craver's view is also similarly committed to decomposition, though it leaves some room for the systems-level form of explanation I am interested in.

Systems Explanation

I argue that the explanatory role of the HKB model is to provide a systems explanation, where a systems explanation proceeds by describing the activity of an interactive system that gives rise to the phenomenon in question. The target phenomenon of the HKB model is the predictable switch in the pattern of the finger movements. Taking the relevant why-question as: *Why do the finger movements change in this way?*, the answer is as follows: *The finger movements form a system with the state space described in the model. The point at which the finger movements change is a bifurcation point – the system changes from having two attractors to having one.*

Giving systems this central role in explanation also raises ontological questions about the status of systems, which I'll briefly discuss at the end of the paper.

Dingmar van Eck: Elaborating functional decompositions in mechanistic explanations

In this paper I argue that Craver's treatment of functions in his account of mechanistic explanations (2001, 2007) harbours two related problems. Firstly, the distinction between "isolated" role function descriptions and "contextual" role function descriptions separates, rather than integrates, mechanistic explanations at lower and higher levels. Secondly, isolated role function descriptions cannot be expressed in mechanistic explanations for an explanandum activity. I propose a solution for these problems by interpreting an isolated role

[2]This view is developed in a number of places, and the primary texts include Bechtel and Abrahamsen, "Phenomena and Mechanisms", in Stainton, ed, *Contemporary Debates in Cognitive Science*, Oxford 2006; Bechtel, Mandik, Mundale, Stufflebeam, eds. *Philosophy and the Neurosciences: A Reader*, Blackwell 2001; Bechtel "Dynamics and Decomposition: Are They Compatible?", *Proceedings of the Australian Cognitive Science Society,* 1997.

[3] "Dynamics and Decomposition: Are They Compatible?", *Proceedings of the Australian Cognitive Science Society*, 1997.

function as a sub function that temporally ordered with one or more other sub functions compose a contextual role function. This solution is based on functional modelling research in the engineering sciences, a domain typically not the subject of mechanistic analyses[4], and on philosophical analyses of the part-whole relationships for functions that are defined in the engineering domain (Vermaas & Garbacz, 2009). I demonstrate both problems and solution in terms of Craver's multi-level descriptions of the heart's mechanistic role in the circulatory system (2001).

The background of my argument is the following. Isolated role descriptions characterize the function φ of an entity X without reference to how X and φ are organized with other Xs and their φ-ings in instantiating the activity Ψ of a mechanism S. Contextual role descriptions, instead, characterize the function φ of an entity X in terms of how X and φ are organized with other Xs and their φ-ings in instantiating the activity Ψ of a mechanism S (Craver, 2001). Craver gives the following example: the activity Ψ of the circulatory system S is delivering oxygen and calories to body tissues. This Ψ-ing of S is instantiated by the organized φ-ings of Xs, with Xs being components of S. An instance of such an organized φ-ing of X is the pumping of blood (φ) by the heart (X). This is a contextual description, since it describes the contribution of X's φ-ing to Ψ in virtue of how X's φ-ing is organized with other φ-ing Xs. An isolated description given by Craver of the φ-ing of X is the contracting (φ) of the heart (X). This description of φ does not refer to its contribution to Ψ in terms of how φ is organized with other φ-ing Xs. Such isolated descriptions of φ constitute the explanandum activities for which mechanistic explanations at a lower level are developed. Continuing the heart example, X's φ-ing 'in isolation' (contracting) is instantiated by the organized ρ-ings of Ps that compose X's φ-ing 'in isolation', such as the contractions (ρ-ings) of heart muscles (Ps) (Craver, 2001).

I argue that by these isolated descriptions Craver's (2001) aim of integrating mechanistic explanations at lower and higher levels fails, because they, instead, separate levels. Part-whole relationships between (ρ-ing Ps, φ-ing Xs, Ψ-ing Ss) at different mechanistic levels are determined in virtue of how organized activities of entities at a lower level instantiate activities of entities at a higher level (Craver, 2001, 2007). I argue (and assume this is uncontroversial) that isolated role descriptions cannot figure in mechanistic explanations, since they do not refer to the contribution of X's φ-ing, in virtue of its organization with other φ-ing Xs, to S's Ψ-ing. This, however, implies that the introduction of isolated role descriptions as a bridging concept between lower and higher levels backfires: the transitivity relationship between (ρ-ing Ps, φ-ing Xs, Ψ-ing Ss) that is both assumed and necessary to integrate mechanistic explanations at different levels (2001, 2007) is violated. Since isolated role descriptions cannot figure in mechanistic explanations, an X φ-ing 'in isolation' is not a "proper part" (Craver, 2001, p. 63) of S's Ψ-ing. An X φ-ing 'contextually', instead, is. Since, however, a ρ-ing P is a proper part of an X φ-ing 'in isolation', and not of an X φ-ing 'contextually', transitivity relationships between (ρ-ing Ps, φ-ing Xs, Ψ-ing Ss) do not hold.

I solve this problem by using an engineering approach for functional decomposition in which overall artefact functions are modelled as sets of interconnected sub functions (Stone & Wood, 2000). Based on this modelling, I recast isolated role descriptions as descriptions of connected sub functions of a contextual role function and introduce a temporal ordering on these sub functions (cf. Vermaas & Garbacz, 2009). Specifically, a set of two or more sub functions in a certain temporal ordering composes a contextual role function. Applied to the heart example, the temporally ordered sub functions "contracting" (In Craver's account, φ-

[4] A notable exception is De Ridder (2006).

ing of X ‘in isolation’), “importing blood”, “contracting”, and “expelling blood” compose the ‘contextual’ φ-ing of X (pumping blood). These functional decompositions of X’s φ-ing (”pumping blood”) can then be used to develop mechanistic explanations at a lower level. The sub function “contracting”, for instance, can be explained in terms of, amongst other, the organized contractions (ρ-ings) of cardiac muscles (Ps).

By introducing this temporal ordering on sub functions, they are organized with one another and I can capture their organized contribution to a contextual role function. This restores transitivity relationships between functions at different levels: ρ-ings are now proper parts of sub functions, which in turn are proper parts of contextual φ-ings, which are proper parts of Ψ.

It is suggested that incorporating these functional part-whole relations in mechanistic explanations strengthens Craver’s position as a competitor to psycho-neural reduction models.

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Erik Weber: How probabilistic causation can account for the use of mechanistic evidence

In their paper ‘Interpreting Causality in the Health Sciences’ (*International Studies in the Philosophy of Science* 21 (2007), pp. 157-170) Federica Russo and Jon Williamson argue that an analysis of causality in terms of probabilistic relationships does not do justice to the use of mechanistic evidence to support causal claims. I will present Ronald Giere’s theory of probabilistic causation, and show that it can account for the use of mechanistic evidence (both in the health sciences – on which Russo and Williamson focus – and elsewhere). I also review some other probabilistic theories of causation (of Suppes, Eells and Humphreys) and show that they cannot account for the use of mechanistic evidence. I argue that these theories are also inferior to Giere’s theory in other respects.

Friedel Weinert: On Rainbows and Slavery: From Physical to Social Mechanisms

The aim of this paper is to examine the notion of ‘social mechanisms’ by comparison with the notion of physical mechanisms. There is a basic disagreement about the epistemological status of social mechanisms. Some writers see them as akin to physical mechanisms, while others consider them to be no more than patterns of social behaviour. In the light of this disagreement, it seems best to start with an analysis of the notion of physical mechanism – like the one causing the formation of rainbows. It is found that a mechanism in both the physical and the social sciences can reasonably be conceived as a *system,* in which relata are held together by appropriate relations. The analysis then reveals a basic difference between physical and social mechanisms, which is ultimately reducible to a difference in the underlying regularities (relations). The paper argues that physical mechanisms are based on lawful regularities – physical laws, like the laws of reflection and refraction in the formation of rainbows - while social mechanisms are based on mere trends. Unlike physical laws, social

trends are inductive generalizations from initial conditions. Social trends are dependent on initial conditions in the way that physical laws are not. Furthermore, social trends are reversible in the way that physical laws are not. As mechanisms are defined as systems of relata and relations, there exists a logical difference between physical and social mechanisms.

The second part of the paper then applies this insight to a consideration of the abolition of the slave trade (1807) as a case of social causation. It is found that the current understanding of social mechanisms is insufficient to explain a unique social event, like the abolition of the British arm of the slave trade. By contrast Weber's notion of 'adequate causation' is able to explain this unique event, as well as more regular patterns of social causation. Weber's notion of adequate causation is really a form of a conditional model of causation, since it conceives of causes and effects in terms of (necessary and sufficient) antecedent and consequent conditions, which may be held together by statistical or, in the limit, deterministic relations. On this account, mechanisms – if they are available for explanation - are part of the antecedent conditions. These mechanisms can be further specified into physical and social mechanisms, depending on the nature of the relations, which hold between the relata. This means that a conditional model of causation is also able to explain the formation of rainbows and other causal, physical events.