

On Mechanisms, Pathways, and their Models

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Abstract

Lauren Ross has recently argued that the current philosophical enthusiasm for mechanisms poses a threat to a proper understanding of the diversity of causal structures found in biology, and of the diversity of ways in which biologists explain biological phenomena. Ross argues that new mechanists have collapsed a variety of distinct causal structures within the confining analytical strictures of mechanism, and in so doing have failed to appreciate the diversity of concepts and strategies needed to describe and explain biological phenomena. Ross grants that mechanisms are important in biology, but argues that there are other causal structures, like pathways and cascades, that are distinct from mechanisms, and that require distinctive treatments. In this paper I'll argue that Ross's worries arise from a failure to distinguish ontological questions about causal structure from methodological questions about modeling and explanation. I'll argue that a mechanistic ontology is compatible with conceptual and explanatory pluralism, and along the way I will offer a new analysis of pathways and pathway models that draws on some of Ross's insights.

Keywords: Mechanism, Pathway, Model, Causal Structure, Causal Explanation, Lauren Ross

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1. Introduction

In a pair of recent papers, Lauren Ross (2021, 2022) claims that the current philosophical enthusiasm for mechanisms poses a threat to a proper understanding of the diversity of causal structures found in biology, and of the diversity of ways in which biologists explain biological phenomena. Ross argues that advocates of so-called new mechanism have collapsed a variety of distinct causal structures within the confining analytical strictures of mechanism, and, in so doing, have failed to appreciate the diversity of concepts and strategies needed to describe and explain biological phenomena. Ross grants that mechanisms are important in biology, but argues that there are other causal structures, like pathways and cascades, that are distinct from mechanisms, and that require distinctive treatments.

My primary aim in this paper is to show that new mechanists have a response to this criticism, and that many of Ross's insights about the diversity of causal structures and explanatory approaches found in biology can be fruitfully integrated within a mechanistic framework. I will argue that her concerns can be alleviated by attending to distinctions between ontological and conceptual/epistemic issues: specifically, distinctions between mechanisms and pathways as ontological structures, as opposed to mechanistic and pathway models as representations and vehicles for explanations. In making this case, I will also offer a novel analysis of what pathways are as ontological structures, suggesting that Ross's treatment overlooks an ambiguity in the term "pathway." One sense of "pathway" refers to causal processes that are appropriately thought of as kinds of mechanisms, while a second sense refers to persistent structures that clearly are not mechanisms.

I begin in Section 2 with a sketch of some central claims of new mechanists, focusing on how they have tried to accommodate the diversity of causal structure under an expansive conception of mechanism. In Section 3, I describe Ross's analysis of the relationship between mechanisms and pathways, showing how it fails to distinguish between questions about ontology and questions about models. In Section 4, I offer an alternate account of what pathways are as ontological structures, and discuss Ross's examples of biological pathways in light of this analysis. In Section 5, I turn to pathway models and explanations, making the point that these models and explanations can complement rather than compete with other forms of explanation, including mechanistic explanation. Finally, in Section 6, I address some more general objections

that Ross has raised to the expansive conception of mechanism, arguing that this conception is wholly consistent with Ross's goals of exploring the diversity of causal structures and explanatory strategies found in biology.

Before turning to my argument, I want to acknowledge that Ross is hardly alone in raising questions about new mechanist overreach. Chemero and Silberstein (2008) for instance have argued that dynamical explanations are often to be preferred to mechanistic explanations in cognitive and neural science. Chirimuuta (2014) and Batterman and Rice (2014) have argued that minimal model explanations used in a variety of disciplines succeed by abstracting away from irrelevant details. Similarly, Skillings (2015) has argued that what he calls "the basic mechanistic account" fails to provide adequate explanations of biological processes that are, for example, stochastic or non-linear. I have chosen to focus on Ross because her work contains some novel criticisms that have not been discussed in the literature. I believe, however, that the strategy adopted here may fruitfully be applied to these other critiques, suggesting that adopting a broadly mechanistic ontological picture is compatible with recognizing a diversity of causal structures and explanatory approaches.

2. An expansive conception of mechanism

In the opening paragraph of *The New Mechanical Philosophy* (2017), I set out two key claims of new mechanism:

[It] ... says of nature that most or all the phenomena found in nature depend on mechanisms—collections of entities whose activities and interactions, suitably organized, are responsible for these phenomena. It says of science that its chief business is the construction of models that describe, predict, and explain these mechanism-dependent phenomena (Glennan 2017, 1)

Ross (2022) cites this passage, along with quotations from Bechtel, Craver, Darden, and others as evidence of the overarching ambitions of the new mechanists. These ambitions do indeed seem bold, but we cannot begin to assess them without some account of what mechanisms are. My preferred account is **minimal mechanism**:

A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon (Glennan 2017, p. 17)

This is a slightly modified version of a mechanism definition proposed by Illari and Williamson (2012) as a consensus account meant to capture the commonalities between earlier definitions offered by Glennan (1996), Machamer, Darden and Craver (2000), and Bechtel and Abrahamsen (2005). The characterization is deliberately abstract and permissive, allowing most things that scientists in various disciplines have called mechanisms to fall within its scope. If mechanisms were not construed in this broad way, the claim that most or all natural phenomena depend upon mechanisms would not be plausible.¹

Given this account of mechanisms, the new mechanist approach embodies a set of ontological commitments about the world's causal structure. Among them are these:

1. Relationships between causes and effects are mediated by mechanisms.
2. These mechanisms consist of entities (parts) engaging in activities and interactions.
3. The behavior of mechanisms depends upon how these entities and activities are organized (e.g., timing, sequence, spatial proximity, causal dependency).
4. These entities and activities/interactions are typically composites whose properties and behaviors depend upon further mechanisms.

We can illustrate these commitments by applying them to a specific case discussed by Machamer, Darden and Craver (2000) -- chemical signaling across a synapse:

1. The causal relationship between an action potential in the pre-synaptic neuron and the depolarization or hyperpolarization of the post-synaptic neuron is mediated by a mechanism.

¹ Glennan, Illari and Weber (2021) argue that minimal mechanism is an appropriate organizing principle for thinking about mechanisms in the sciences, and it is prominently featured in the *Stanford Encyclopedia of Philosophy* entry on mechanisms (Craver, Tabery and Illari 2024), which provides some useful insights on the historical context of the mechanism literature. Critics of the approach who have offered alternative views of what mechanisms are. For instance, Woodward (2011) has attempted to reduce claims about mechanisms to claims about difference-making, and Ioannidis & Psillos (2022) have argued that the minimal mechanist approach is too metaphysically freighted, and that mechanisms can be understood as “theoretically described causal pathways.”

2. This mechanism consists of entities within or around the pre-synaptic and post-synaptic neurons (e.g., neurotransmitters, vesicles, sodium and potassium ions, receptor sites) and their environment (e.g., the synaptic cleft), and the various activities and interactions that these entities engage in (e.g., binding, diffusing, reuptake).
3. The behavior of this mechanism depends upon how these activities are organized – the sequencing of stages within the transmission process, locations of binding sites, rates of diffusion and reuptake, etc.
4. Components within this mechanism can be broken down into further components and mechanisms. For instance, the opening and closing of ion channels depends upon the activities and interactions of their molecular constituents.

The chief argument for accepting this mechanistic view of the world's causal structure is what Machamer *et al* refer to as descriptive adequacy. Simply put, scientific attempts to uncover various bits of the world's causal structure typically involve looking for mechanisms in this sense –characterizing the various entities and the ways they act and interact so as to discover how they mediate between causes and effects. This is a claim about how scientists characterize the domains they investigate, and it is easily substantiated by looking at language commonly found in scientific journal articles and textbooks.

While I hope readers will take this descriptive point as obvious, there are at least two reasons why critics might still reject these mechanistic ontological commitments. The first is that the claim that ALL causal relationships are mediated by mechanisms cannot be established merely by pointing to the fact that the language of mechanism is widespread in many disciplines. One problem may be that there aren't things like entities and activities when we get to quantum scales (Kuhlmann and Glennan 2014), and indeed there are some who might follow Russell in arguing that there is no causation in physics at all. On the other end of the spectrum, one might argue that the influence of so-called structural causes cannot be explained within this sort of framework. How, for example, would one describe the mechanism by which structural racism impacts the lifespan of marginalized populations?

A second concern is that the ontological commitments of the new mechanists may be based on metaphysical assumptions that are not warranted by the practice or findings of science. This is the crux of Ioannidis and Psillos' (2017, 2022) defense of what they call "truly minimal

mechanism” or “causal mechanism.” Woodward (2011) expresses similar skepticism. In a different kind of critique, Dupré and Nicholson (2018) have taken aim at the new mechanists’ dualism of entities and activities, attempting to reduce objects (at least in biology) to processes. And in yet another recent critique, Jiang (2024) has argued that implicit in new mechanistic metaphysics is a kind of unargued atomism, and that a better and more metaphysically minimal foundation can be given using the resources of ontic structural realism.

Whatever the legitimacy of these concerns, they are not relevant to the argument of this paper. First, even if it turns out that mechanistic characterizations of the world’s causal structure are not appropriate in some domains like quantum mechanics, they are widespread in the domains discussed by Ross in her exploration of the relations between mechanisms, pathways and cascades. Second, the ontological assumptions required for my argument do not go beyond a very modest kind of realism about entities, activities and causal relationships that Ross and the new mechanists clearly share, and which are firmly grounded in the practice of science. All that is needed is the assumption there are in fact entities and activities of different sizes and scales (for entities, for instance, proteins, cells, tissues, organisms, populations, and for activities, for instance, binding, folding, transporting, moving, eating, predating...), and that organization of these things into mechanisms or processes are what make things happen in the world. These modest ontological commitments are enough to allow us to distinguish between causal structure and our representations of that structure, and it is on that distinction that my argument will turn.

With the minimal account of mechanism in mind, we can now turn to Ross’s critique of the new mechanist approach. In the abstract to her (2021) pathways paper, Ross puts the matter this way:

[B]iologists use a variety of causal concepts in their explanations, including concepts like pathways, cascades, triggers, and processes. Despite this variety, mainstream philosophical views interpret all of these concepts with the single notion of mechanism. In using the mechanism concept interchangeably with other causal concepts, it is not clear that these accounts well capture the diversity of causal structures in biology.

This characterization of “mainstream philosophical views” does not seem to me a fair characterization of the mechanist position. Proponents of mechanistic approaches see mechanisms as a broad genus, and they understand that an essential part of the research program

is to explore the different species of mechanisms which are responsible for the various kinds of phenomena biologists (and other scientists) study. Different kinds of mechanisms may demand different approaches to discovery, representation, and explanation.

Some of those working on the variety of mechanisms have attempted to provide general taxonomic principles for characterizing kinds of mechanisms, (Andersen 2014; Glennan and Illari 2018). Others have explored experimental strategies for identifying different features of mechanisms and their organization, and have considered the kinds of mechanisms for which these different strategies will succeed or fail (Bechtel and Richardson 2010; Craver and Darden 2013). Still others have developed accounts of particular kinds of mechanisms in order to spell out their special features (Piccinini 2007; Bechtel and Abrahamsen 2010; Glennan 2010; Ylikoski 2018). The new mechanist's research program, far from papering over differences among kinds of causal structure in the world, provides tools to explore these differences.

While I cannot describe these efforts in full, I will close this section with a discussion of a few ways of distinguishing different kinds of mechanisms that will prove useful in exploring the relationship between mechanisms, pathways, and cascades. A first important distinction is between mechanisms understood as structures or systems and mechanisms understood as processes. In common parlance, the word "mechanism" is used in both ways. The first way takes a mechanism to be a stable structure analogous in some respects to a machine -- a many-parted system/object whose behavior can be explained by the way its parts interact. Watches or cars are mechanistic systems in this sense, as are biological systems like organisms or ecosystems – though these biological systems differ in many ways from human-built machines. The second way takes mechanisms to be processes by which some output is produced from some input, or by which some activity or interaction is accomplished. This processual sense of mechanism is extremely common in biology, as when one speaks of mechanisms of protein synthesis, or cellular respiration, or speciation.² The system/process distinction can be missed because mechanistic processes often occur within mechanistic systems. A clock is a mechanistic

² The early new mechanist literature does not always distinguish between systems and processes. The processual sense of mechanism is evident in the original mechanism definition of Machamer, Darden and Craver (2000), especially in its reference to startup and termination conditions, while definitions provided by Glennan (1996) and Bechtel and Abrahamsen (2005) refer respectively to systems or structures – suggesting a commitment to the systemic sense of mechanism. But in these early works authors discussed examples of processual and systemic mechanisms as the occasion suited them.

system, but its ticking is a mechanistic process going on within it, and indeed it is the fact that this process can and does occur that makes the clock a clock. Still, one should not equate systems with processes. From a metaphysical point of view, systems are continuants – enduring complex objects – while processes are occurrents, things defined by their starting points and end points (Kaiser and Krickel 2017). While I grant that scientists use the term “mechanism” to refer both to systems and processes, I will follow a number of recent discussions in taking the processual sense of mechanism to be what is captured by minimal mechanism.³

One way of distinguishing different species of mechanisms is by identifying different ways in which a mechanism may be responsible for its phenomenon. Craver and Darden (2013, pp. 65–66) pursue this strategy, arguing that biologists search for mechanisms characterized by three different kinds of mechanism-phenomenon relationships. Some mechanisms *produce* phenomena. For instance, aerobic respiration produces ATP from glucose via a mechanistic process. Other mechanisms *underlie* phenomena. For instance, the sliding of filaments within myofibrils underlies the contraction of muscle tissues. Here, the activity of a muscle contracting is constituted by the activities and interactions of its parts. In a third kind of relationship, mechanisms *maintain* some phenomenon. These are homeostatic mechanisms in which some property of a system (e.g., temperature, insulin levels, ...) is maintained by the activities of components within the system in the face of interactions between the system and its environment.

Craver and Darden’s distinction between producing, underlying and sustaining fits within the broader taxonomic approach pursued by Glennan and Illari (Glennan 2017; Glennan and Illari 2018, p. 201). This approach suggests that varieties of mechanisms can be sorted into kinds along five different dimensions: 1) the kind of phenomenon the mechanism is responsible for, 2) the kinds of entities/parts they contain, 3) the kinds of activities and interactions that these entities/parts engage in, 4) the way in which the entities, activities and interactions are organized, and 5) the kind of etiology, i.e., the way in which the mechanism comes to exist. Craver’s and

³ Recent work from new mechanists discussing the relation between the processual and system senses and arguing for the primacy of the professional sense include (Glennan 2017; Krickel 2018; Craver et al. 2021). There is a long history of processual approaches to causation which are discussed in both the Glennan and Krickel books. For a useful discussion of the relation between the new mechanist approach to processes and those of Salmon and Dowe see (Campaner 2013).

Darden's distinction fits within the first dimension, but other ways of characterizing categories of mechanisms use other dimensions. For instance, to call something a neurological mechanism is to classify it by a type of entity it contains, to call it a developmental mechanism is to identify it by the phenomenon produced, to call something a negative feedback or control mechanism is to say something about its organization, and so on. Because these dimensions are largely independent, varieties of mechanisms will not be well ordered into a hierarchy of taxa. The dimensions can cross-classify, so that, for instance, two mechanisms constituted by very different entities and activities may have similar forms of organization, or mechanisms that produce or underlie similar phenomena may have very different etiologies.⁴

This approach to classifying mechanisms allows for exploration of the diversity of causal structure in the way Ross advises. For instance, a cascade certainly meets the requirements of minimal mechanism – there are entities and activities organized in a way that produces a phenomenon. What makes cascades distinctive as a kind of mechanism is their organization. If we accept Ross's analysis, cascades have three key features: "(i) an initial trigger, (ii) sequential amplification, and (iii) stable progression from start to finish." (Ross 2022, p. 6). As Ross notes, there are further subtypes of cascades, depending for instance on whether what is amplified in a cascade is a single product or multiple products. In distinguishing cascades by their organizational features, we are acknowledging that cascades are not limited to a particular class of entities or activities. That is why we can have cascades in immune responses, electrical circuits, bank runs, and genocides.

Ross (2021) was certainly aware that defenders of the mechanistic approach could treat pathways or cascades as species of mechanisms, and in the conclusion of her paper she offers some arguments against this strategy. I will provide an explicit response to these arguments in the final section of this paper, but I want to begin with a serious look at Ross's approach to distinguishing mechanisms from pathways and then propose an alternate account.

⁴ For further examples, see Craver and Darden (2013, Table 5.1) who provide an extensive hierarchically organized list of types of biological mechanisms that organizes these types along several of these dimensions – kinds of phenomena/functions, types of entities and activities, and kinds of organization.

3. Ross on mechanisms and pathways

Ross claims that mechanisms and pathways are distinct species of *causal structure*, but her account starts with an analysis of mechanisms and pathways as *causal concepts*. Her analysis of the mechanism concept identifies three key features, which I label M1 – M3:

M1: [M]echanisms are often characterized as having a constitutive makeup, in the sense of involving particular systems with higher-level behaviors that can be decomposed into lower-level causal parts. (Ross 2021, p. 134)

M2: [T]he mechanism concept ... is used to refer to causal systems that are described in significant amounts of causal detail as opposed to systems that abstract from such information (Ross 2021, p. 135)

M3: [T]he mechanism concept ... often involves an emphasis on the “force,” “action,” and “motion” involved in causal relationships. This emphasis is evident in how we discuss machines in ordinary life. (Ross 2021, p. 136)

If this characterization is meant to say something about mechanisms as an ontological category, it is problematic; M2 and M3 do not identify features of mechanisms as such, but features of models. The abstraction referred to in M2 is a feature of representations, not of mechanisms or causal systems. It may be reasonable to say that mechanistic models provide significant causal detail, but detail or its absence is not a property we ascribe to things in the world. Protein synthesis is not more or less detailed than a circulatory system, though we might have more or less detailed descriptions of either. Similarly, the “emphasis” highlighted in M3 is something we provide in a description of something; it is not a feature of the world.

M1 refers to mechanisms rather than the mechanism concept, so it alone among the three criteria seeks to identify a distinctive feature of mechanisms as parts of the world’s causal structure. According to M1, mechanisms are causal systems like car engines or clocks (Ross’s examples). Behaviors of these systems are the targets of mechanistic explanations, with these behaviors being explained by activities and interactions of the system’s parts. To use an example Ross mentions, a muscle is a system that acts by contracting, and this contracting is constituted by the activities of the muscle’s parts.

The description of mechanisms in M1 does align with a common way new mechanists have talked about mechanisms, and, in particular, with Craver's framing of the problem of constitutive relevance. Craver (2007) introduced the now familiar formalism that denotes the target of a mechanistic explanation as "*S* ψ -ing" -- an entity or system acting, and argued that the problem of constitutive relevance was a problem of identifying working parts of *S* with respect to its ψ -ing, i.e., those entities (X_i) whose activities (ϕ_i -ings) underlie *S*'s ψ -ing.

The problem with characterizing mechanisms as M1 does is that not all mechanisms are mechanisms that underlie the behavior of a system. Sometimes mechanistic processes cross system boundaries. As Craver, Glennan and Povich have recently argued:

[T]he boundaries of mechanisms and the boundaries of entities like bodies, cells, and nuclei often do not coincide. Many mechanisms are mechanisms by which two or more entities interact, as in the mechanisms of synaptic transmission. And some mechanisms, like erosion on a riverbank or the Rayleigh scattering that makes the sky blue, are not embodied in entities; they are not mechanisms by which an entity acts, or by which a collection of entities interact. In such cases there is no determinate entity *S* that ψ s or an *S* and a *T* that ψ with each other; there is only ψ -ing (Craver et al. 2021, p. 8811).

Craver *et al* go on to argue the common feature of all mechanisms is that they have a "processual core." If their argument is correct, for something to be constitutively relevant to a mechanism's activity, it need not be a component of a system; it must instead be a part of a process – an activity of or interaction between some of the entities that constitute the process. Given this processual understanding of mechanisms, minimal mechanism should really be understood as providing an analysis of what causal processes are.⁵ This fact will prove important, because Ross holds (correctly I think) that pathway concepts describe "sequences of causal steps" in an "unfolding of a causal process."

⁵ Ross might object that in offering this processual characterization of mechanisms, Craver *et al* have changed the rules about what count as mechanisms. But, as noted in footnote 2, new mechanists have often analysed biological mechanisms – protein synthesis, synaptic transmission, natural selection, etc – which aren't aptly described with the formalism of *S*'s ψ -ing. What Craver *et al* are acknowledging is that in certain respects the *S*'s ψ -ing formalism, as well as certain diagrams used to abstractly represent mechanisms, have not been perspicuous representations of certain kinds of mechanisms. Regardless of whether we think Craver *et al* have shifted positions, what is most germane to the present discussion is that biologists very frequently use the term "mechanism" to describe processes that are not activities or behaviors of systems.

Ross begins her analysis of the pathway concept by identifying several examples of things biologists call pathways -- cell-signaling pathways, metabolic pathways, developmental pathways, circulatory pathways, and ecological pathways. Generalizing from these examples, she argues:

The pathway concept, as it is commonly used in biology, has at least four main features. This concept captures a (i) sequence of causal steps, where these steps (ii) track the flow of some entity or signal through a system, (iii) abstract from significant causal detail, and (iv) emphasize the “connection” aspect of causal relationships. (Ross 2021, p. 139)

As with the analysis of the mechanism concept, this analysis mixes ontological features of the world’s causal structure with epistemic features of representations. “A sequence of causal steps” refers to a causal chain or process, which is indeed a feature of the world’s causal structure. The expansive mechanist conception can recognize pathways in this sense as a type of mechanism. Specifically, it is the type of mechanism, discussed in the previous section, that Craver and Darden (2013) call a “producing mechanism” – a mechanistic process beginning with some input or trigger, passing through a set of intermediate stages, and terminating in some product or end state. But the other three features Ross identifies -- “tracking,” “abstracting” and “emphasizing” --are things WE do when we describe these pathways. They are features of our models and explanations.

As I show in part 5, the relationship between pathways and pathway models is crucial for understanding what is truly distinctive about pathway explanations. But before turning to this discussion, I offer an alternative to Ross’s analysis of pathways that clarifies their place in our taxonomy of causal structures.

4. Two kinds of pathways

What then are pathways as an ontological category? One possibility is that pathways are a species of mechanism in just the way that I’ve argued that cascades are a species of mechanism. However, in the case of pathways, I’ll argue that the relationship to mechanisms is not that simple. In what follows I’ll suggest that the term “pathway” is ambiguous. Sometimes it is used to refer to certain kinds of mechanisms, but other times it is used to refer to something that is not a mechanism at all.

To understand this ambiguity, it will be useful to begin with an analysis of the ordinary language use of the words “path” and “pathway” (which I’ll treat as synonyms). In its first sense, a path is a geographical feature – a trail or track along which humans or other creatures can travel. I’ll call pathways of this kind **g-paths**, to emphasize their geographic nature. G-paths are affordances for travelling between places, allowing travelers to move more quickly between points *A* and *B*, sometimes providing the only way through otherwise impassible terrain. Some g-paths, like mountain passes, may simply be accidental features of the terrain, while others, like a cattle track, may develop over time as the result of the movements of animals; still others, like our garden paths and roadways, are designed and built by humans.

A crucial feature of g-paths is that they form networks. We can represent a g-path network with a graph – a collection of nodes and edges. The nodes represent geographic locations, while the edges represent paths between these locations. A network of train tracks is a good example of a g-path network. The different stations along the tracks are nodes, and at some of these stations the line may branch, allowing a choice of tracks along which to continue a journey. G-path networks can be characterized by their topologies – some nodes will be directly connected, while some will be accessible only by intermediaries, or not at all; some networks will have loops, some will be unidirectional, while others will be bidirectional, and so on.

In its second sense, a path is a **route** that a traveler takes, or at least could take, to get from one place to another. For instance, when I went to work today, I travelled along a route. First, I turned right out my door and headed north up Delaware Street, then I turned left when I reached 40th Street, continued on, making various turns, until I reached my office. In cases of physical travel, there is an intimate connection between routes and g-paths, because the affordances of the g-paths are what allows for travel along a route. I cannot, for instance, travel by a straight line from my house to my office, because to do so would require me to walk through walls. Of course, not all routes follow a pre-existing set of paths; we sometimes travel “off the beaten path.”

Routes, like g-paths, can be represented using graphs with nodes and edges, and, supposing a network of g-paths characterize all the possible ways to get from one place to another, a g-path network will provide a representation of all the possible routes one can take. A map of the London Underground, for instance, will show all the possible ways I can travel by Underground

to get between any two stations in the system. But despite similarities in the way they are represented, there is a crucial difference between g-paths and routes. The nodes in a route are temporally and causally related, while the nodes in g-paths are spatially and non-causally related. To see the difference, consider route planning using a system like Google Maps. Google Maps knows the network of g-paths lying between your location and your destination, and it uses an algorithm to suggest possible routes. The routes proposed are characterized as steps or stages in a **journey** through this g-path network. The start of your journey along the chosen route begins at some point in time, and each stage of the journey has temporal duration. From an ontological point of view, journeys, and the routes they take, are occurrents –with beginnings, ends, and temporal parts – while g-paths and networks are continuants -- enduring things without temporal parts (Simons and Melia 2000).

What exactly is the relationship between journeys, routes, and travelers? A journey is a concrete causal process, a continuant, but a route is something abstract. It represents a property of an actual or possible journey. Journeys also require something to take the journey. Call these things travelers. In its original non-scientific sense, the travelers on journeys are humans or other animals that travel along routes within some geographic region. But the notion of traveler is easily extended to other things that are less material – trade goods, gossip, disease, and so on – and, in scientific contexts, to things like energy, nutrients, or information.

In non-scientific contexts we frequently extend pathway metaphors beyond geography and physical movement to describe routes through other kinds of spaces. Consider, for instance, pathways to citizenship in the United States. There are two routes – by birth or by naturalization. The birth route simply requires one to be born in the United States – regardless of the citizenship status of the parents (except in strange cases like children of diplomats). The naturalization route has more steps, requiring first that a person become a legal permanent resident – and this status can be gained through several routes, including marrying a US citizen, being the child of a US citizen, being a member of the military, and so on. Once one has permanent residence status (a green card), one needs to take other steps – applying, passing a test, and so on. The crucial indicator that immigration pathways describe something analogous to routes rather than to g-pathways is that the nodes in these pathways are steps or stages that are temporally ordered.

To see the variety of things that can be routes, it is helpful to compare immigration paths with migratory paths. Both migration and immigration are kinds of journeys, and both have routes, but the routes are of different kinds. Migration routes involve physical movement and follow actual g-paths, while immigration paths are routes through a different kind of space, not geographical, but legal, regulatory and causal.

Now that I have laid out these distinctions, I can state the key claims I wish to defend about the relation between pathways and mechanisms as entities within the world's causal structure:

1. G-paths are not mechanisms, because they are not processes and do not involve activity. They are continuants -- persistent features of some landscape that afford and constrain journeys through a space.
2. Journeys on the other hand are mechanistic processes. They involve organized sequences of activities and interactions by which a traveler travels from a start point to an end point.
3. Routes are also not mechanisms because they are abstracta, but they are properties of actual or possible journeys, which are (actual or possible) mechanistic processes.

To this, I want to add one point about pathway models:

4. Pathway models are models that can be described using the tools of graph theory, with nodes and edges, and with characteristic topologies. Pathway models can be used to represent either g-paths, routes, or journeys.

To illustrate and substantiate these claims, let's use them to analyze three pathways discussed in Ross's paper: pathways for steroid synthesis, a food web, and circulatory pathways through the heart. Ross's discussion is accompanied by figures depicting these pathways that I have reprinted as figures 1, 2 and 3.

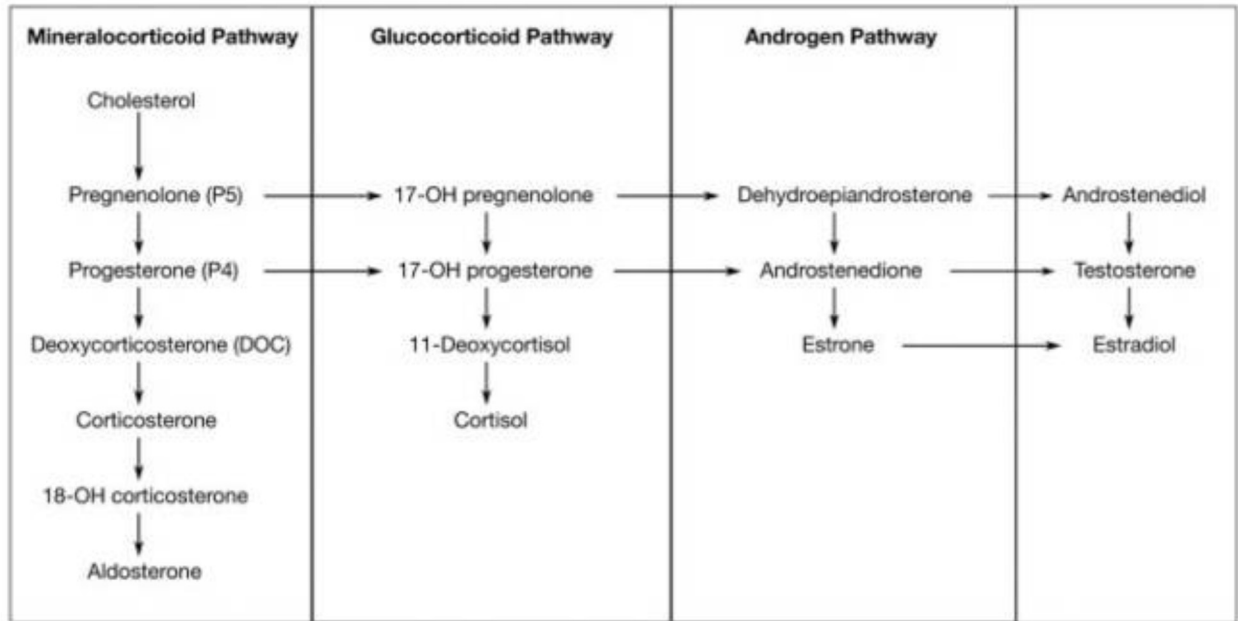


FIGURE 1 Metabolic (or biochemical) pathways for steroid biosynthesis. (reprinted from Ross 2021 figure 2)

The metabolic pathways depicted in figure 1 show the steps by which cholesterol is transformed, via a sequence of molecular products, into various steroids. The diagram is an example of a pathway model. It necessarily leaves many things out, including many of the enzymes and other reactants that trigger steps along these various routes. Still, the diagram captures the order of essential steps in the transformation of one molecule into another.

These metabolic pathways are not g-paths but are routes -- routes by which cholesterol can be transformed into other products. The fact that this diagram is a route diagram is revealed by the fact that the nodes do not refer to geographical places, but to intermediate products in the mechanistic process of synthesizing the various steroids. The edges in the diagram denote temporal and causal relationships rather than spatial or geographic ones.

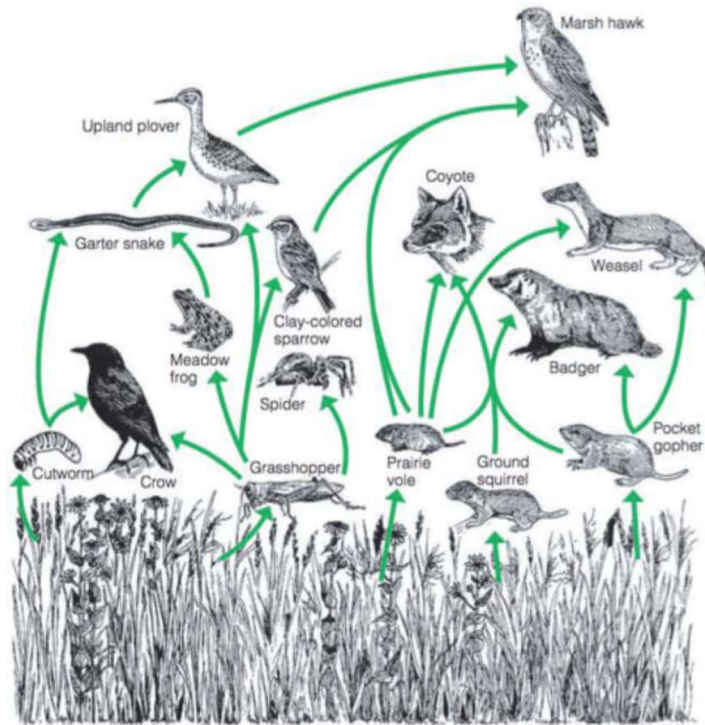


Figure 2: A food web – reprinted from Ross figure 5, originally in (Smith and Smith 2015)

The food web diagram in figure 2 is a similar case, depicting pathways in the sense of routes. A food web diagram represents predator-prey relationships between various species within an ecological community. This figure shows, for instance, that grasshoppers eat grasses, and are eaten by spiders, frogs, and sparrows; and sparrows in turn are eaten by hawks. Predation is clearly a causal relationship, and we can understand the links between species as representing a sequence of causal steps whereby energy and nutrients travel from basal species to apex predators. A food web clearly does not represent a network of g-paths, because it says nothing about **where** these predation relations occur within the community’s habitat. The organization of the graph is temporal/causal rather than spatial.⁶

⁶ While I think Ross is right to claim that links in food chains form pathways (in the sense of routes), it is interesting that ecologists seldom speak of food chains or webs as pathways, and indeed “pathway” is a term seldom used in ecology. The word does not appear in the explication of food webs in the textbook from which Figure 2 is drawn, or anywhere in that textbook’s index or glossary, nor in another ecology textbook I checked. A quick Google Scholar search reveals only around two thousand hits for “ecological pathway” (mostly in the context of environmental science), in contrast to, for instance, two million hits for “metabolic pathway.” The term “trophic pathway” does have limited use in the literature. I asked a few ecologists about this paucity of pathway talk. Their response was that “pathway” is a molecular biologist’s word. They speculated that this may be because so many ecological interactions are weak, multiple, and stochastic, making the metaphor of branching paths less apt.

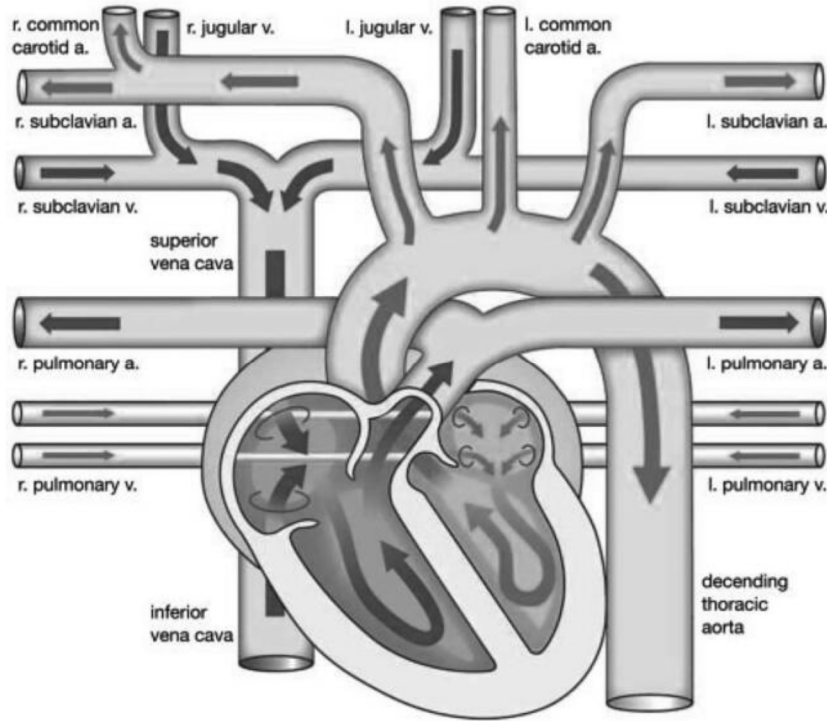


Figure 3. Circulatory pathways of the heart – reprinted from Ross figure 4

If we understand Ross’s analysis of the pathway concept as an explication of what a pathway model is, the key feature of pathway models is that they identify sequences of steps within causal processes. But the third example I’ve drawn from Ross’s paper, circulatory pathways of the heart, really can’t be understood in this way. In contrast to the pathways represented in diagrams 1 and 2, the veins, arteries, chambers, and their intersections represented in Figure 3 do form a network of g-paths. Although there are some arrows representing direction of blood flow, a diagram representing the circulatory system is fundamentally a representation of anatomy, and anatomy is the body’s geography. The nodes and edges represent *places* within the body. Because they are places, we can locate them within the body, noting for instance that a certain junction in the arterial system is located above the heart, or that another is in the arm.⁷ This network of g-paths provides the infrastructure which allow travelers (blood, oxygen, nutrients, etc.) to find routes to different parts of the body. The circulatory system is like a road network – a continuant with spatial rather than temporal parts. It is a system that enables journeys, which are causal processes (occurents), to occur within it.

⁷ Note that the relevant notion of place in g-paths is topological. Like subway maps, g-path diagrams tell you how to get from here to there, and what are the intersections and stops, but they don’t measure absolute distance.

An important lesson here is that structurally similar network diagrams can represent two different kinds of information. Some, like the steroid synthesis and food web diagrams represent causal sequences (journeys and the routes they take), while others, like the circulatory diagram represent g-path networks – spatially or topologically arranged paths through some kind of landscape. Diagrams used in the first way do represent mechanisms, while diagrams used in the second way do not.

The circulatory system is the only example Ross discusses that represents a g-path network. Her other examples are route-focused, showing sequences of steps in journeys that are temporally and causally ordered. These other examples seem to me to capture the most common uses of pathway talk and pathway diagrams in biology. What we call metabolic pathways and developmental pathways, for instance, all are characterized by sequences of causal steps or stages. But the g-path sense of pathway can also be important in biology. Maps of the lymphatic system or a connectome for a nervous system are representations of systems of g-paths. Similarly, migratory pathways are networks with nodes representing locations on our planet, and edges representing g-paths between them. These g-path networks are not mechanistic processes or journeys, but they are biologically (and mechanistically) significant because they permit or expedite journeys.

The upshot of this analysis is that the term ‘pathway’ is ambiguous, and that biologists on some occasions use it to refer to a persisting structure, a network of g-paths, and on other occasions to refer to a mechanistic process, a journey in which something travels along such a network. Pathways in the first sense are not mechanisms, but pathways in the second sense are. Using the taxonomic principles discussed in section 2, we can see journeys on pathways are a particular kind of mechanistic process, with a certain kind of organization. Applying Craver and Darden’s account of mechanism-phenomenon relations, they are mechanisms that produce something (rather than underlying or maintaining something), and the activities in these journeys have linear (though sometimes branching) organization into stages.

5. Pathway Models and Explanations

Although I have been critical of some aspects of her analysis, Ross is certainly right to counsel us to attend to the diversity of causal concepts and forms of explanation. My aim in this

section is to reframe Ross's insights in a way that complements rather than competes with new mechanistic approaches to understanding the world's causal structure. The key will be to carefully distinguish between causal structures and the models we use to describe and explain them. Minding this distinction will allow us to affirm pathway explanation as a distinctive form of explanation while allowing that journeys on pathways are a kind of mechanistic process.

Let's start with a concrete example of pathway explanation. Suppose, for example, we are considering the outbreak of an infectious virus, and we want to explain why some patient, call them patient *X*, has gotten the virus. A common representation of virus transmission will be a contact-tracing tree with its root an original infection – patient 0. The tree will extend out from this root in multiple generations, beginning with patient 0's contacts, and then extending to those contact's contacts, and so on. An explanation of why patient *X* was infected will locate them on this tree and trace a path of infection back to patient 0. This pathway shows just how patient *X* got the virus.

Ross contends that mechanistic detail is explanatorily irrelevant to pathway explanations like this one, because the details do not make a difference to the occurrence of the explanandum. Often this is the case. In this example, identifying the path from patient 0 to patient *X* explains why patient *X* got the disease, and understanding the mechanisms by which contacts transmit the disease doesn't add to that explanation.

Whether a pathway explanation provides sufficient information about a mechanistic process will depend upon the question an explanation is seeking to answer. For instance, so long as transmission rates between close contacts is less than 100%, the contact-tracing tree does not explain why one person along a path of exposure contracts the virus, while another on the same path does not. Additional details about the infectious agent, the exposed individuals, and the mechanisms of transmission will be needed to answer this question. Regardless of whether mechanistic details are explanatorily relevant to a particular question, we can grant that the information in pathway diagrams can be explanatory, and often sufficient in an explanatory context to answer an explanation-seeking why-question.

Let us step back from this particular example to situate pathway explanation within the broader landscape of scientific explanation. To do so I'll make a few assumptions about explanation that are widely even if not universally accepted. First of all, I take it that scientific

explanations work by identifying features upon which some explanandum depends. Second, I assume that the activity of explaining relies upon models. Models are constructs of some kind (material, mathematical, computational) that can be used to represent aspects of the explanatory target.⁸ Models are inevitably abstract and idealized. They represent only certain features of their targets, and they simplify or distort others.

It is very common in discussions of modeling to try to distinguish between different categories of models – material models, mechanistic models, network models, dynamical models, mechanistic models, and so on. But the properties we use to distinguish these classes are of very different kinds. **Referential classifications** identify models by the things in the world that are their targets. When we speak of population models or atomic models, we are saying that these are models *of* these targets. **Formal classifications** identify models by formal or syntactic properties of the model itself. If we take a dynamical model to be one that uses differential equations, we are identifying dynamical models by formal features. **Semantic classifications** identify models by the kind of information they contain about the target. A connectivity model in a cell phone network, for example, will provide information about strength of connections across geographic regions. Attending to these different kinds of classifications will help us situate pathway models in the broader landscape of scientific models.

In the previous section I indicated that pathway models can be represented as graphs consisting of nodes and edges. To identify pathway models in this way is to use a formal classification. Another way we can understand the category of pathway models is by a referential classification. Pathway models are models *of* pathways. But, given my analysis suggesting that the term “pathway” can refer either to g-path networks (which are persistent structures) or journeys (which are mechanistic processes) what it is to be a model of a pathway is ambiguous. To avoid the ambiguity, we could call these different sorts of pathway models g-path models and journey models. Depending upon the referential target, the formal features of pathway models will be used to represent different kinds of information. If the model is a journey model, routes along the pathways provide information about causal and temporal

⁸ Note that while explanations use models, we shouldn't equate models and explanations. For one thing, models have other uses besides explanation, and for another, the activity of explanation often relies upon more than one model.

ordering of events, but if it is a g-path model, the routes provide information about spatial location and connectivity of static structures.

Given this analysis of what pathway models are, how should we think of their relation to mechanisms and mechanistic explanation and to causal explanation generally? On the one hand, if a pathway model, like the model of steroid biosynthesis has a journey as its target, then it is a model *of* a mechanistic process. But even so, the formal properties of the pathway model allow it to represent certain kinds of information of the sort Ross emphasizes, tracking the flow of a signal or information through a sequence of causal steps, while abstracting from many other details. On the other hand, if the pathway model has as its target a g-path network, it is not a model *of* a mechanism, since g-path networks aren't mechanisms. Mechanisms require activity, and g-path networks are static things.⁹

The larger lesson to be learned from this analysis of pathway models and explanations is there is not a one-one mapping between kinds of models or explanations and kinds of causal structure. What kinds of models are appropriate will depend upon what we are modeling, but different models and explanations may complement each other, and we may need a family of models to gain anything like a full causal/mechanistic understanding of the phenomena we seek to explain.¹⁰

6. Mechanisms and conceptual/explanatory pluralism

Let me return in closing to revisit Ross's central objection to the new mechanists' approach – her claim that it papers over the diversity of causal structures found in the world. Mechanisms, she claims, may be one type of causal structure, but there are others, like pathways and cascades, that are distinctive and are important in understanding biological (and other) phenomena.

⁹ Notice that while g-path networks aren't mechanisms, they are entities, and as such can be parts of mechanisms. Railroad tracks, for instance, are parts of the mechanisms by which trains travel from one station to the next.

¹⁰Hochstein (2016) provides an extended defense of the view that mechanistic explanations may require many models. I have focused here on the use of multiple models to give causal and mechanistic explanations, but it is plausible to think that the kind of information represented in pathway models are relevant to non-causal explanations, which may complement causal/mechanistic ones. As Ross herself notes (2021, 151), pathway explanations have affinities with topological and network explanations (Levy and Bechtel 2013; Huneman 2018). On the complementarity of causal and non-causal explanations, see (Andersen 2018). The basic strategy of appealing to multiple models to triangulate on an explanation has been around a long time (Levins 1966).

My reply in this paper has been that mechanists can make room for varieties of causal structure by taking mechanisms to be a broad genus under which many species of causal structure may fall. Additionally, I have shown that there are different kinds of models that may be used to describe and explain aspects of this causal structure. I have tried to make this reply plausible by showing that many of Ross’s insights about pathway explanations can fit within this approach.

Ross anticipated a strategy of this kind, and offers a number of reasons why she thinks the strategy should be rejected. Let’s consider her case:

There may be a ready reply to these claims. Perhaps “mechanism” should be understood as a “catch-all” or “one-size-fits-all” concept that is intended to distinguish any generic causal structure from those that are non-causal. Consider further, that there may be different “types” of mechanisms within this broad category. Perhaps the “mechanism” examples I discuss fall under some “mechanism_a” category, while the “pathway” cases fall under some “mechanism_b” category. This might be used to indicate how a mechanistic account could be flexible enough to capture all interesting causal structures and types of causal explanation in science (Ross 2021, p. 154)

Ross offers three replies to this approach:

First, if “mechanism” is synonymous with any causal structure, how is mechanistic explanation different [from] the generic claim that causes (or set of causes) explain their effects? ...

[Second] this “catch-all” approach simply does not seem to accommodate all instances in which “mechanism” is used in biology ... If the new mechanists want to base an account of biological explanation on a definition of “mechanism” that is divorced from biological use, they should have a good reason for doing so.

[Third] by collapsing distinctions between the “mechanism” and “pathway” concepts, we lose [*sic*] sight of the role of analogy in causal and explanatory reasoning (*ibid*).¹¹

¹¹ While I’ve reprinted the exact text of Ross’s objection here, I expect the second reply doesn’t capture her intended meaning, since the second clause of the sentence implies that the “catch-all” approach is too narrow. Other parts of the text imply that her concern instead is that the catch-all concept is too broad, identifying things as mechanisms which biologists don’t describe as mechanisms.

What Ross has missed in framing this strategy and in her replies to it is that causal structure is one thing, causal concepts are another, and causal models and explanations are yet something else. Failing to attend to these distinctions is arguably the major reason she thinks her argument for conceptual and explanatory pluralism is at odds with the new mechanists' enterprise.

Ross's first objection to the "catch-all" approach is that for mechanists to adopt this view would be inconsistent, since mechanist accounts "are typically motivated by the view that mechanisms have unique features that are not found in all causal structures" (*ibid*). But this is not, I think, what new mechanists typically say. What they typically say is that mechanistic *explanations* have unique features that distinguish them from other kinds of explanations. Mechanistic explanations, for instance, are different than covering law explanations (Bechtel and Abrahamsen 2005; Craver 2007). They also differ from "bare causal" explanations (Glennan 2017) that describe causal dependencies between events or properties without spelling out the mechanism responsible for the dependence. They also emphasize that mechanistic explanations differ from various kinds of structural and non-causal explanations.

It is also not reasonable to suggest that even an expansive conception of mechanism simply equates mechanisms with causal structure. If we take causal structure to include anything in the world that is relevant to giving causal explanations, lots beside mechanisms would have to be included. A g-path network is one such example. A network of roads is not a mechanism, since it is a static structure. But surely the explanations of success of people moving from here to there will depend upon roads.

Those new mechanists who accept my claim that "most or all the phenomena found in nature depend on mechanisms" are advancing a view about the ubiquity of mechanistic causal structure, but such a view is best understood as an alternative to other contemporary views in the metaphysics of science. It may, for instance, be at odds with some recent approaches to process metaphysics, or with metaphysical views about causation that understand causal connections to always be mediated by laws, or that seek to reduce causal dependence to counterfactual dependence, or with views of the world's ontology which deny the existence of causes at all. But this ontological position is not contrary to, and indeed can make sense of, the methodological and explanatory pluralism Ross recommends.

Ross's second reply accuses the expansive conception of mechanism of diverging from biological usage. I'm skeptical though that there is anything like a specific and univocal usage of the term "mechanism" in biology. Ross cites a few cases from the biological literature to substantiate her own "narrow" analysis of the mechanism concept, and no doubt many explanations of mechanisms have the features Ross points to. Nonetheless, the term "mechanism" has a very wide usage in biology and beyond. (My latest Google Scholar search on mechanisms in biology yields 6.5 million hits.) Only a very broad conception of mechanism could conceivably capture all of it. Indeed, Ross and Bassett (2024) have recently acknowledged and descried the highly variable use of the term "mechanism" in neuroscience. They argue that neuroscientists should employ a more diverse and well-defined set of causal concepts to more clearly capture the diversity of causal structures in their domain. As a normative position, Ross and Bassett likely have a point, but the very fact that they think the usage of the term "mechanism" needs to be policed seems to undermine the claim that "catch-all" conceptions of mechanism are inconsistent with biological usage.

Ross's third reply is the most compelling, since it is certainly the case that the concepts of "mechanism" and "pathway" have different metaphorical associations which can give rise to different ways of describing and explaining causal structures. But crucially, the mechanism as genus approach doesn't really "collapse" the notions of mechanism and pathway, and is quite consistent with the view that there are many distinct kinds of causal structures. Even if pathways or cascades are kinds of mechanisms, this does not mean that there are not valuable things to say about what distinguishes them from each other and from other kinds of mechanisms; and the mechanism as genus approach is entirely consistent with the view that pathway models provide different kinds of explanatory information than mechanistic models.

In the end, though, it would be a mistake to see the criticisms I have raised of Ross's argument as being chiefly aimed at defending one taxonomic approach (mechanisms as genus) over another (mechanisms and pathways as species). The larger point of this paper is that any successful attempt to characterize the varieties of causal structures we find in the world must appeal to distinctive features of the world, rather than distinctive features of our representations of the world. We cannot, for instance, argue that one kind of causal structure is abstract, while

another is detailed, because abstraction and detail are features of representations, not of the world itself.

The models we use to represent bits of the world do tell us things about its causal structure. That is their point. Moreover, whether a model is suitable to represent a specific kind of causal structure will depend upon what sort of model it is. A pathway model is, for example, only suitable for describing causal processes that have certain kinds of organization. The mistake occurs when we equate the model with the world. If we make that mistake, we will fail to see that different models can represent different features of the same causal nexus, and that the task of fully explaining the causes of a given phenomenon may require many models.

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