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Causal Concepts in Biology: How Pathways Differ from Mechanisms and Why It Matters

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ABSTRACT

Over the last two decades few topics in philosophy of science have received as much attention as mechanistic explanation. A significant motivation for these accounts is that scientists frequently use the term 'mechanism' in their explanations of biological phenomena. Of course, biologists 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 article analyses two causal concepts in biology—the notions of 'mechanism' and 'pathway'—and how they figure in biological explanation. I argue that these concepts have unique features, that they are associated with distinct strategies of causal investigation, and that they figure in importantly different types of explanation.

1 Introduction

For nearly two decades few topics in philosophy of science have received as much attention as mechanistic explanation. The beginnings of this 'new mechanist' philosophy are often associated with an article by Machamer *et al.* ([2000]), which outlines the general view and remains one of the most cited

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publications in *Philosophy of Science*. While various accounts of mechanistic explanation exist,[1] many of them describe mechanisms as organized sets of entities and activities that underlie and produce some phenomenon of interest. This explanatory pattern involves explaining some outcome by appealing to the mechanism that produces it. While this basic picture is thought to well represent explanation in many domains, it has been most extensively examined and applied to the biological sciences where it has led to a research programme that has recently 'exploded' (Bechtel and Richardson [2010], p. xlviii). Not only does this explanatory framework receive significant attention in the philosophical literature, but many view it as fundamental to any understanding of explanation in biology and as the 'dominant view of explanation in the philosophy of science at present' (Kaplan and Craver [2011], p. 606).

One motivation for these accounts is that scientists frequently use the term 'mechanism' in their explanations of biological phenomena (Wimsatt [1976], p. 671; Machamer *et al.* [2000], p. 2; Bechtel and Richardson [2010], p. xvii). Biologists, of course, 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. For example, Robins and Craver ([2009]) state that although scientists appeal to terms like 'cascades, pathways, systems, and substrates [. . .] We use the term mechanism for all of these' (Robins and Craver [2009], p. 42). Similarly, Craver claims that while scientists 'say that they discover systems and pathways in the flow of information, and molecular cascades, mediators, and modulators [. . .] The term mechanism could do the same work' (Craver [2007], p. 3). These claims receive widespread support in the philosophical literature. This is evidenced by the fact that numerous philosophical projects analyse the appeal to various causal concepts (for example, pathways, cascades, processes, and so on) as instances of mechanistic explanation.[2] Further evidence of this is seen in discussions of non-mechanistic explanation, in which the goal is to find explanations that are non-causal as it is often assumed that all causal explanations (and those causal concepts figuring in them) are mechanistic (Kaplan and Craver [2011]).

This understanding of mechanistic explanation raises a number of puzzles. Although these accounts use the notion of mechanism interchangeably with other causal concepts, it is not clear that this well captures the diversity of

[1] For some of these accounts, see (Glennan [1996]; Craver [2007]; Bechtel and Richardson [2010]). For an excellent overview of different philosophical projects connected with the 'mechanism' concept, see (Andersen [2014a, 2014b]).

[2] For example, the mechanism concept is used to analyse biological phenomena that include: (1) metabolic pathways (Thagard [2003]; Bechtel and Abrahamsen [2005]; Bechtel and Richardson [2010]; Bogen and Machamer [2010]; Bechtel [2011]; Bechtel and Levy [2013]), (2) developmental pathways (Fagan [2013]; Tabery [2014]), (3) gene-expression pathways (Bickle [2006]), and (4) signalling cascades (Brigandt [2013]).

causal structures in biology. Consider the notion of a pathway, which commonly figures in biological explanation. Examples of this concept include gene expression pathways, cell-signalling pathways, metabolic pathways, anatomical pathways, developmental pathways, and ecological pathways. In some cases, these 'pathways' appear to be different from biological phenomena referred to as 'mechanisms'. In particular, it sometimes suggested that a single pathway can be instantiated by different mechanisms, that distinct pathways can have similar mechanisms, and that pathways can be discovered without any knowledge of the mechanisms that underlie them.[3] For example, the same metabolic pathway can be instantiated by different enzymatic reactions or 'reaction mechanisms' across species (Berg *et al.* [2012], pp. 296–7, 313). In other cases, distinct anatomical pathways, such as blood vessels and lymphatic pathways, can share similar 'mechanical properties' including relationships between pressure, volume, and fluid flow (Quick *et al.* [2008]; Venugopal *et al.* [2010]). Finally, in other situations, pathways are identified despite the fact that very little it known about the mechanisms that underlie them. This is the case for many developmental pathways such as stem cell differentiation pathways.[4] How should we understand these causal structures, their features, and their role in biological explanation? Are all causal concepts in biology—including the pathway concept—best understood with the single notion of mechanism?

This article addresses these questions by providing an analysis of the mechanism and pathway concepts in biology and how they figure in biological explanation. I argue that these concepts: (a) have unique features, (b) are associated with distinct strategies of causal investigation, and (c) figure in importantly different types of explanation.[5] To be clear, I agree with the new mechanists's claim that the mechanism concept is important in biology and biological explanation. This article does not offer a criticism of their accounts, unless they make the further claim that all causal concepts in biology are accommodated by their notion of mechanism. What I argue for is simply that the pathway concept is importantly different from the mechanism concept and that it deserves its own analysis. The main goal of this article is to provide a philosophical analysis that develops systematic distinctions between

[3] In some cases, introductory sections of biological textbooks promise to discuss both the 'mechanisms' and 'pathways' relevant to the domain of interest (Berg *et al.* [2012], p. v).

[4] More specifically, consider that the 'traditional model' of the stem cell differentiation process outlines key developmental stages with linear and branching pathways (Fagan [2013], p. 75). These processes are depicted with pathway diagrams, although it is often claimed that their 'mechanisms are still poorly understood' (Dietrich *et al.* [2008], p. 393; Fagan [2013], p. 19).

[5] My interest in this project has been importantly influenced by conversations with Ken Schaffner and his suggestion that the pathway concept is unlikely to be well accommodated by the new mechanist paradigm (Schaffner [2016]). My analysis explores this suggestion by providing a novel characterization of the 'pathway' concept, how it differs from the notion of 'mechanism', and how these differences matter for causal investigation and explanation in biology.

these concepts. This analysis captures methodologically important considerations including scientists' goals, available strategies, and features of the systems under study. I show that scientists sometimes use these concepts in the ways I suggest, while leaving a more detailed analysis of the consistency of their usage for another paper.

One main theme of this analysis is that scientists refer to biological systems as 'mechanisms' and 'pathways' when they share features with structures in ordinary life that we associate with these causal concepts. This involves analogizing biological systems to structures in everyday life that we are familiar with. This strategy can make complicated features of complex biological systems more cognitively accessible. While the presence of such analogies in scientific reasoning is not a new observation, its relevance to the mechanism concept (and other causal concepts in biology) has been surprisingly unexplored.[6] I explore this suggestion and argue for the aforementioned claims (a, b, c) in the rest of this article, which is structured as follows: In Section 2, I briefly discuss the mechanism concept, its features, the strategies of causal investigation that it is associated with, and how it figures in biological explanation. Section 3 introduces the pathway concept, its main features, and a common causal investigative strategy that it figures in. This section compares the pathway concept to the mechanism concept in order to clarify how they differ. In Section 4, I explore one type of pathway explanation in which pathway information is explanatory and mechanistic information is not. The final section returns to the topic of analogy in science and contains some concluding remarks.

2 Mechanisms: The Basics

Biologists frequently appeal to mechanisms in their explanations and descriptions of biological phenomena. They discuss mechanisms of gene regulation, DNA synthesis, nerve firing, muscle contraction, visual processing, and so on. When they use the mechanism concept they often suggest that some biological phenomenon can be understood as a kind of machine or mechanical system—such as a car engine or clock—in the sense of having particular features. This machine analogy encourages thinking of biological phenomena as having component parts that are spatially organized and that causally interact to produce some behaviour of the system. A key feature of this explanatory pattern is that it involves explaining some outcome by appealing to its causal parts. The system-level behaviour serves as the effect or explanatory target, while the interacting mechanical parts are what explain this behaviour.

[6] For discussion of analogy and analogical reasoning in science, see (Hempel [1965], p. 434; Hesse [1966]; Lewis [1986], p. 220; Nersessian [2002]).

Three features of this mechanism concept should be highlighted. First, mechanisms are often characterized as having a constitutive makeup, in the sense of involving particular systems with higher-level behaviours that can be decomposed into lower-level causal parts. This feature is exploited in efforts to discover mechanisms through the common investigative strategies of 'decomposition and localization', which are considered the 'central heuristics' of mechanism discovery (Wimsatt [1974]; Bechtel and Richardson [2010]; Bechtel and Levy [2013]). These strategies involve a process where scientists identify a system and behaviour of interest and then 'drill down' to identify the system's parts, their location, and how they interact to produce the behaviour in question. This process reveals the role of single effects or higher-level explanatory targets in the discovery and individuation of mechanisms. In particular, mechanisms are circumscribed on the basis of which parts causally interact to produce a particular effect.[7] Those causal factors that produce this behaviour make up the mechanism and those that are not involved in this production are not mechanism components. This supports a picture where mechanism boundaries are drawn on the basis of methodological and pragmatic considerations, as opposed to capturing fixed, natural divisions in the world (Craver [2009]; Bechtel [2015]).[8] This contributes to our conception of mechanisms as discrete causal entities in the same way that we talk about particular car engines or clock mechanisms as single, distinct causal systems (Bechtel and Richardson [2010], p. 35). These causal systems have boundaries and they can be discussed as individual units that are distinct from other causal systems in the world (Andersen [2014a], p. 276).

A second feature of the mechanism concept is that it 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. Consider the 'mechanism of enzyme catalysis' where an enzyme catalyses (or speeds up) the chemical conversion of an upstream substrate into a downstream product. Scientists refer to these enzymes as 'molecular machines' because they perform these conversions in multi-subunit complexes that have many causally interacting parts (Spirin [2002], p. 153). These parts and their interactions are represented in 'reaction mechanism' diagrams. These diagrams include components such as the enzyme itself, its substrate, and various cofactors and regulators that alter its functionality. Scientists expect complete descriptions of these mechanisms to contain large amounts of causal information. Consider the following:

> An understanding of the complete mechanism of action of a purified enzyme requires identification of all substrates, cofactors, products, and

[7] This effect-relative individuation is sometimes referred to as 'functional individuation' (Machamer *et al*. [2000]; Bechtel and Richardson [2010]; Williamson and Illari [2012]).

[8] These divisions can change with different contexts, goals, and explanatory targets.

> regulators. Moreover, it requires a knowledge of (1) the temporal sequence in which enzyme-bound reaction intermediates form, (2) the structure of each intermediate and each transition state, (3) the rates of interconversion between intermediates, (4) the structural relationship of the enzymes to each intermediate, and (5) the energy contributed by all reacting and interacting groups to intermediate complexes and transition states. As yet, there is probably no enzyme for which we have an understanding that meets all these requirements. (Lehninger and Cox [2008], p. 205)

As this suggests, scientists expect descriptions of these mechanisms to contain a large degree of causal information—so much information, in fact, that it has not yet been acquired it in our best scientific understanding of these systems. This same sentiment is present in scientists' discussions of the 'mechanism of action' for particular drugs. They claim that these mechanisms must involve 'a complete and detailed understanding of each and every step in the sequence of events that leads to [an] outcome' (Hutchinson [2007], p. 1). Similarly, these mechanisms should provide 'a comprehensive understanding of the entire sequence of events' and 'detailed knowledge of the causal and temporal relationships among all the steps leading to a specific effect' (Hutchinson [2007], p. 7; Ankley *et al.* [2010], p. 731). This mechanistic understanding is contrasted with other approaches that only capture 'selected key events' and that have 'gaps and black boxes in which mechanistic details are either unknown or not needed' (Hutchinson [2007], p. 1; Ankley *et al.* [2010], p. 732). The expectation that mechanisms contain significant causal detail is expressed by many philosophical accounts of mechanism (Machamer *et al.* [2000]; Darden [2006b]; Craver [2007]; Craver and Darden [2013]). This feature of mechanisms is associated with our interest in understanding how they work and our assumption that this often involves identifying more and more information about their causal components, organization, and so on. Furthermore, acquiring such information is useful for various reasons. This information can suggest different potential targets that may change the final outcome, insight on different ways that a mechanism might malfunction, and it can lead to the identification of causal relationships that are more invariant or stable across different contexts.

A third feature of the mechanism concept is that it 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. Machines have parts such as pulleys, levers, hammers, and gears that actively do things. We do not simply say that these parts 'cause' various outcomes in each system, we say that they 'push', 'pull', 'bend', and 'compress' some downstream component. Mechanism descriptions in biology involve a similar emphasis. Scientists say that a cofactor 'activates' an enzyme, which then 'binds'

to a substrate, before 'splicing' off a chemical moiety, and 'attaching' it to another molecule. The fact that the mechanism concept has this feature should be somewhat unsurprising, because the term 'mechanism' literally draws on mechanics or the branch of science and mathematics concerned with 'motion and the forces producing motion' (Soanes [2012], p. 449). What is the significance of this feature? Emphasizing the force or action of causal relationships serves several functions in biological (and other) contexts. First, it helps to satisfy our interest in understanding 'how' a mechanism works—adding force or motion terms adds something more than just saying that X causes Y. Second, these terms also function to fill in space between cause and effect variables, which can suggest closer physical proximity and satisfy our interest in getting more detail about the mechanism of interest. Causal terms involving force and motion appear to fill in black boxes and suggest that we know more about some causal process than merely saying 'that' X causes Y.

In the biological sciences, 'mechanism' is often used to refer to causal systems that have a constitutive character, that are represented in significant, fine-grained detail, and that contain an emphasis on the 'force', 'action', or 'motion' of causal relations. This concept is associated with the causal investigative strategies of decomposition and localization and it is involved in an explanatory pattern where some outcome is explained by appealing to the causal components that produce it.

3 The Pathway Concept

The pathway concept is commonly found in the biological sciences. Biologists refer to gene expression pathways, cell-signalling pathways, metabolic pathways, developmental pathways, circulatory pathways, neural pathways, and ecological pathways, just to name a few. In all of these cases the notion of a pathway refers to a sequence of causal steps that string together an upstream cause to a set of causal intermediates to some downstream outcome. For example, gene expression pathways track causal connections from genes, to their intermediate products to a final phenotype of interest.[9] Signal transduction pathways track causal connections from an upstream signal, through intermediate transduction steps, to some final effect (Figure 1). Metabolic pathways capture sequences of steps in the chemical conversion of an initial metabolic substrate into some final downstream product (Figure 2) (Kaushansky [2006]). Developmental pathways depict a stepwise set of changes in the development of some precursor system (for example, a cell, tissue, or organism) into a later final state (Figure 3). Anatomical

[9] These intermediates are sometimes referred to as 'intermediate phenotypes' or 'endophenotypes'. In the context of disease traits, these are defined as phenotypes 'that form the causal links between genes and overt expression of disorders' (Gottesman and Shields [1972]).

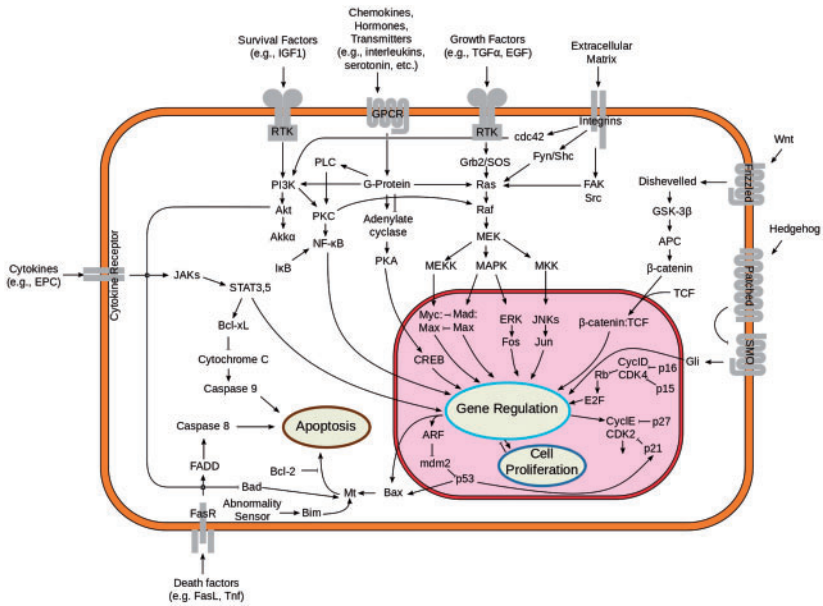

Figure 1. Common signal transduction pathways.

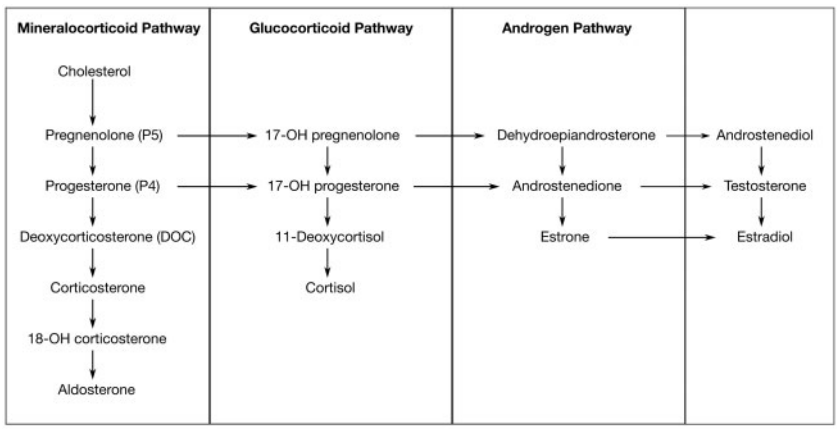

Figure 2. Metabolic (or biochemical) pathways for steroid biosynthesis.

pathways, such as lymphatic pathways, blood vessels, and nerve tracts, capture physical routes that outline causal paths travelled by some fluid, informational signal, or other property of interest (Figure 4). Finally, ecological pathways track causal links of predator-prey relationships that trace the flow of energy through food chain, which make up larger food webs (Figure 5) (Smith and Smith [2012], p. 325).

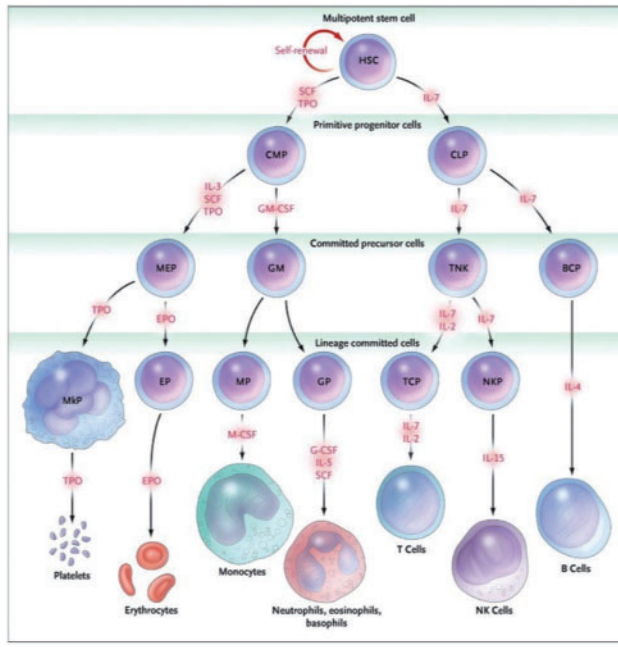

Figure 3. Stem cell developmental (or differentiation) pathways of hematopoiesis (Kaushansky [2006]).

When biologists use the pathway concept they often imply that some system can be understood in terms of causal routes or roadways. These causal routes capture interconnected paths that track the movement of some entity or informational signal through a system. In these cases scientists analogize a biological system to ordinary life conceptions of roadways, highways, and city streets (Bender [1997]; Salway [2004]). What exactly are the features of this pathway concept? How is it used in causal investigation and explanation in biology? Finally, how does it differ from the mechanism concept, if it does at all?

3.1 Main features

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. A first feature of the pathway concept, which the above cases make clear, is that it captures a sequence of causal steps in some process. This sequence captures a fixed order of causal relationships that reflect which outcomes need to occur before and after others in the unfolding of a causal process. For example, consider the first three steps of the mineralocorticoid

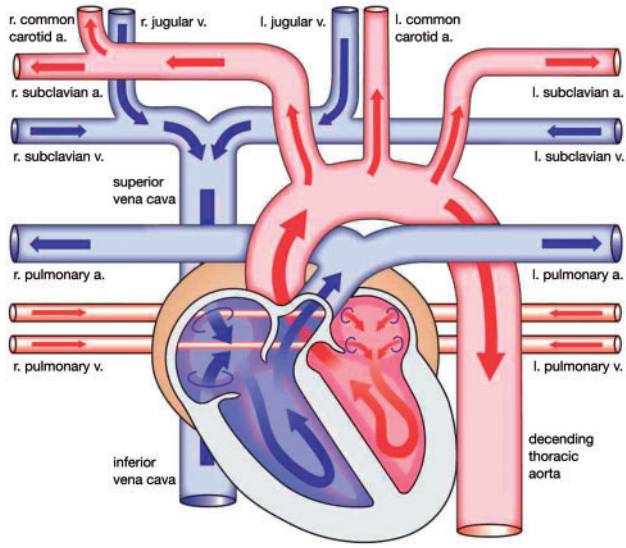

Figure 4. Circulatory pathways of the heart (figure modified from Belleza [2017]).

pathway, shown on the left-hand side of Figure 2. In these steps, cholesterol is first converted into pregnenolone (P5), which is converted into progesterone (P4), which is converted into deoxycorticosterone (DOC). This pathway captures the sense in which these steps need to take place in a particular order. Cholesterol cannot be directly converted into DOC without first forming the P5 and P4 intermediates. Furthermore, it must go through both intermediates without omitting one or reversing their order. This sequence does not just capture a fixed order of entities, but a fixed order of causally related entities. The mineralocorticoid pathway captures a causal chain in the sense that every downstream product depends on an upstream substrate. In other words, the upstream substrate is at least one causally relevant factor in the production of the most immediately downstream product. Other cases of the pathway concept in biology involve this same fixed sequence of causal relations, but they differ in terms of the causal relata along the pathway. Biologists sometimes refer to these causal chains as 'domino causality' because, similar to a sequence of falling dominos, the effect at one step becomes a cause of the next (Grotzer and Basca [2003]).

It might seem that this first feature does not capture a true difference between the pathway and mechanism concepts because both can be understood in terms of sequential causal steps. There is more to say about the causal sequences in pathways that reveal how this concept differs from the notion of mechanism. One difference, and a second key feature of the pathway concept, is that it represents causal sequences that capture the 'flow' of some entity

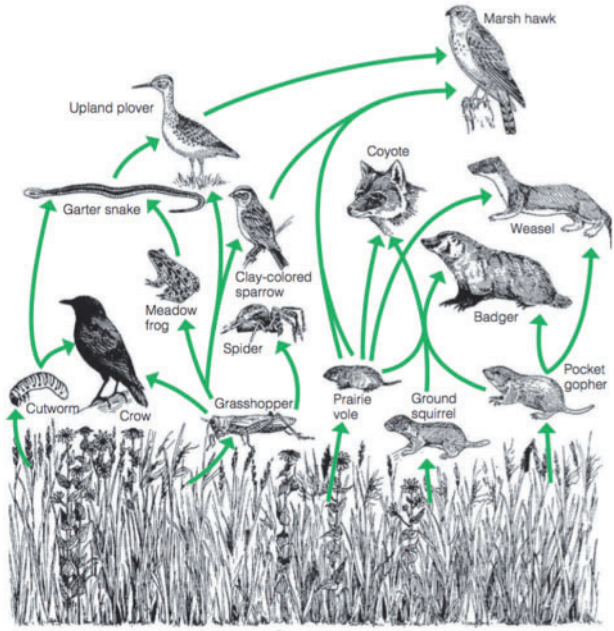

Figure 5. Ecological pathways: Food chains within a food web (Smith and Smith [2012], p. 325).

or signal through a system. For example, cell signalling pathways track the flow of a signal through molecular and cellular systems, metabolic pathways trace the flow of chemical substances through stepwise changes, stem cell pathways capture the flow of cells through developmental sequences, anatomical pathways such as blood vessels trace the flow of blood through the body, and ecological pathways trace the flow of energy through ecosystems. This notion of 'flow' refers to something that is carried over from one causal step to the next—it involves the permanence or continuity of something that travels along causal connections.[10,11] Use of the 'pathway' concept in these cases is not a mere coincidence—this concept is used to refer to these biological systems because they have features that are similar to pathways we encounter in everyday life. One of these shared features is the notion of 'flow'. This notion is common to ordinary life examples of pathways, such as how cars move along freeways and how water courses along pipes in a plumbing system. The fact that scientists explicitly point out this similarity is seen in the language they use

[10] To be clear, I am not suggesting that the pathway concept supports connected process or mark transmission accounts of causation. Instead, pathways represent causal relationships that have the additional feature of capturing the flow of some entity or signal, while not all causal relationships have this feature.

[11] Other examples of biological pathways that involve the flow of information include biosynthetic pathways (such as protein synthesis), neural pathways, and hormone-signalling pathways.

to describe these systems. They refer to the 'flux' of chemicals along 'metabolic roads and byways' (Pardee [1994], p. 375; Lehninger and Cox [2008], p. 528), they claim that lymphatic pathways are 'avenues' and 'routes' along which 'lymph is transported' (Richter and Feyerabend [2004], p. 6; Meyers *et al.* [2005], p. 4), and that food chains are 'energy channels that propagate matter and energy [. . .] linked by predators' (Moore and de Ruiter [2012], p. 225). Additionally, they state that ecological pathways are 'plumbing of sorts—through which matter and energy flow within ecosystems' (Caswell [2005], p. viii). The pathway concept and language of 'flow' are not simply meaningless, colourful metaphors. They highlight objective, physical features of these systems that reveal how they operate in the world and how we can best study, discover, and understand them. For example, flow through these systems is often experimentally studied with tracer and tagging techniques that follow the physical flow through them.[12] Furthermore, problematic 'blockages' in these pathways can be understood and repaired through analogical reasoning with ordinary life traffic-freeway examples. This is seen in the case of inborn errors of metabolism where a blockage incident results in the pathologic build-up of material upstream of the incident. Successful therapeutic measures involve rerouting this material along 'bypass' routes, shunting it to some non-pathologic product, reducing influx into the pathway, and supplying needed material downstream of the blockade. Instead of being a trivial turn-of-phrase, the pathway concept is used to highlight features of these biological systems that matter for how they are scientifically studied and for how they are manipulated to control the outcomes they produce.

When mechanisms are discussed in biological contexts, there is not usually an emphasis on some entity or information 'flowing' through the mechanism.[13] Mechanisms contain parts that interact to produce a final effect, but there is not typically a discussion of something moving across or along these parts. Consider an objection to this claim. One might suggest that 'causal influence' flows through mechanisms, where this refers to the propagation of causal force through a set of intermediates. Of course, there is a sense in which all causal relationships involve the 'flow' of causal influence. However, something more is present in these pathway cases that is not found in all causal relationships, namely the movement of some further entity besides causal influence (for example, metabolites, information, cells, blood, energy, and so on). This is supported by the fact that in biological contexts where 'pathways' are identified there are numerous causal relationships transmitting 'causal

[12] These techniques tag material that is sent into the pathway so that the pathway steps, flow rates, and interconnections can be discovered.

[13] This claim conflicts with some philosophical accounts of mechanism (Darden [2006a]; Bogen and Machamer [2010]).

influence' where only some of these relationships are referred to as pathways. Thus, pathways track more than just 'causal influence'.

A third feature of the pathway concept—and one that clearly differs from the notion of a mechanism—is that pathways represent causal sequences that abstract from significant causal detail. One way that pathways do this is by representing only those causal factors that capture flow through a system as opposed to representing the entirety of factors that support or are causally relevant to this flow. For example, metabolic pathways represent the flow of metabolites and not the many other factors that regulate this flow, such as enzymes, cofactors, temperature, pH, and so on. This is similar to how road maps represent freeways and city streets without also depicting traffic lights, police officers, or road blocks that regulate or alter the flow of traffic along these routes. These pathway diagrams abstract from this type of information. A second way that pathways abstract from detail is by representing complex processes with an economy of causal steps. This is easily seen in the case of developmental pathways that capture the development of living cells, tissues, and organisms in a limited number of stages. For example, the entire life cycle of many organisms is represented in anywhere from four to twelve main steps where these steps could each be further divided into numerous causal links (Mahadeo and Parent [2006], p. 116). Biologists sometimes refer to this difference between mechanisms and pathways. While they emphasize the need for detail in mechanism cases, they explicitly state that pathways are 'not intended [...] to be exhaustive descriptions' (McClanahan and Branch [2008], p. 5). This is also supported by their claims to have identified various 'complete pathways', while 'complete mechanisms' are still beyond their reach. Scientists admit that no 'complete' mechanism of enzyme catalysis has yet to be uncovered due to the immense detail that this requires. However, they claim that the 'whole pathway' of glycolysis was discovered in the 1930s and they identify other 'complete' pathways in the context of ecology (Lehninger and Cox [2008], p. 528; Wentsel *et al.* [2008], p. 217). These cases indicate that the level of causal detail required for a 'complete' pathway is far less than the level of detail required for a 'complete' mechanism.

I am suggesting that one clear difference between these causal concepts is that pathways abstract from significant causal detail, while mechanisms are expected to include it. Consider two objections to this position. First, one might claim that significant causal detail is not a necessary or characteristic feature of mechanisms and that some mechanisms abstract from such information. While some philosophers subscribe to this 'abstract mechanism view' most claim that mechanisms are highly detailed.[14] Philosophers who promote

[14] Proponents of the former position include (Bechtel and Levy [2013]) and the latter (Machamer *et al.* [2000]; Darden [2006b]; Craver [2007]; Kaplan and Craver [2011]).

this former view are likely to claim that pathways are easily accommodated by philosophical accounts of mechanism because there is nothing problematic about the notion of an 'abstract mechanism' and that this is exactly what pathways are. A first problem with this approach is that we see usage of the mechanism concept in biology that does not accord with this 'abstract mechanism' interpretation. These pathway cases and other examples of abstract causal relationships (such as monocausal models of disease) are rarely (if ever) referred to as mechanisms and they are often viewed as devoid of mechanistic information. Second, expanding the mechanism concept to fit these pathway cases fails to make sense of the way these concepts figure in analogical reasoning. In some cases it is useful to analogize a biological system to an ordinary life pathway, as opposed to an ordinary life mechanism. This is because ordinary life pathways and mechanisms have different causal features, and some biological systems are more similar to the former than the latter. These differences can matter for understanding how a biological system operates, how it should be manipulated to control its output, and how its nature and structure should be represented and communicated. A philosophical account that collapses these distinctions, and uses 'mechanism' as an umbrella term for all causal concepts, fails to capture such distinctions and their role in describing and explaining biological phenomena.

A second objection claims that mechanisms involve significant detail and maintains that pathways are early-stage mechanism sketches or schemata that have yet to be filled in with this detail (Craver [2007], pp. 113–4). This is a standard interpretation of these pathway examples in the philosophical literature (Craver and Darden [2013], p. 91). As these interpretations suggest that increases in causal detail provide increases in explanatory power, the lack of detail in causal pathways is said to result in their explanatory deficiency.[15] In fact, these mechanistic accounts associate the pathway concept with the 'vice of chainology', where '[. . .] one becomes fascinated by nodes in a causal chain but loses sight of how the nodes work to produce, underlie, or maintain the phenomenon' (Craver and Darden [2013], p. 91). These accounts claim that pathways are causal structures that are 'incomplete' and reflect a 'shallowness' of understanding (Craver and Darden [2013], pp. 91–2). These views misunderstand the pathway concept in biology. While they suggest that scientists

[15] In a recent article, Craver and Kaplan [forthcoming] argue that their mechanistic account has been misinterpreted as claiming that 'more details are better' or that increasing mechanistic detail provides increases in explanatory power. While I find these new claims hard to reconcile with their published work, my analysis does not hang on this issue. Even if they ultimately view 'abstract mechanisms' or spare causal detail as explanatory, there are other important differences between the mechanism and pathway concepts and the explanations that they figure in. Pathways are still not well understood with the 'abstract mechanism' view, as they have other features (such as flow, emphasis on connection, and so on) that are not characteristic of the mechanism concept.

aim to fill pathways in with increasing amounts of detail, this conflicts with scientists' explicit statements to the contrary. Scientists explicitly claim that such pathways are 'not intended [. . .] to be exhaustive descriptions' (McClanahan and Branch [2008], p. 5). Scientists consider pathways to be 'whole' and 'complete' when they contain far less detail than 'complete mechanisms' (Lehninger and Cox [2008], p. 528; Wentsel *et al.* [2008], p. 217). These points indicate that biological pathways are not properly viewed as precursor, incomplete mechanism sketches, but as 'complete' causal structures that are captured with a distinct causal concept. Furthermore, attention to biological cases indicates that pathways are cited in explanations, without being considered explanatorily deficient. This is discussed further in Section 4, which considers the role of the pathway concept in biological explanation.

A fourth feature of the pathway concept is that it highlights the 'connection' involved in causal relationships as opposed to the 'force', 'action', and 'motion' that are emphasized in causal relationships in mechanisms. Mechanisms involve specifying 'how' X causes Y, while pathways simply capture 'that' X causes Y. Given some set of entities in a system, the goal of the pathway concept is to show what is causally connected to what, as opposed to the fine-grained details of 'how' they are connected. Biologists invoke this feature when they refer to ecological food webs as 'connectance webs' and 'wiring diagrams' that involve 'showing which species are connected to which' (Caswell [2005], pp. vii–viii). The pathway's emphasis on connection is related to the fact that pathways need not be relative to or defined by a particular effect of interest. Recall that mechanisms are defined on the basis of their effects. Isolating a mechanism implies that an outcome of interest has been specified. As pathways emphasize causal connection, they are often used to represent a complex web of causal connections in some domain before any explanatory target, effect, or outcome of interest is identified. Similar to roadways, scientists claim that pathways can have 'arbitrary start and end points' and that it can be hard to determine where a pathway 'starts and ends' (Firn [2010]). These features of the pathway concept are central to understanding the causal investigative strategies that it is associated with, which I turn to now.

3.2 Investigative strategy

Recall that the mechanism concept is associated with the causal investigative strategies of decomposition and localization. These involve 'drilling down' or decomposing a system into its lower-level parts. Before these strategies can be implemented they require a first step that mechanists refer to as identifying a 'locus of control', which involves specifying some (a) system and (b) effect of interest (Bechtel and Richardson [2010], p. 35). All causal components of the

mechanism are identified on the basis of these specifications—they are included or omitted from the mechanism on the basis of whether they are found in (a) and whether they causally contribute to (b). In this sense, mechanisms are circumscribed on the basis of which parts causally interact to produce the effect of interest. These divisions can change with different explanatory questions, goals, and other pragmatic considerations (Bechtel [2015], p. 85). This effect-relative approach leads to the identification of a single, discrete causal system—a set of causal parts that are responsible for the effect and that are represented as distinct from other causal systems in the world.

The pathway concept is often associated with a different causal investigative strategy. A first step in this pathway approach involves identifying causal connections across entities in some domain without specifying either an effect of interest or a causal starting point. In this approach there is an interest in creating a map of available causal connections in some context. This is a kind of road map, or what biologists might call a 'network' or 'landscape' of available causal routes. Unlike the mechanism concept and strategy these maps do not represent a particular, discrete set of causal parts that all interact to produce a specific outcome. Instead they represent available or potential causal connections that are relevant to a variety of explanatory outcomes and causal starting points. These are channels that can be navigated to get from any one point in the map to any other. Instead of identifying a particular explanatory target and 'drilling down', these maps involve identifying a set of entities in some domain and 'expanding out' by tracing their causal connections. These connections are 'available' or potential in a way that differs from the actual causal components in mechanisms—they contain information about various causal possibilities as opposed to a single, circumscribed causal process that leads to an effect. These causal maps are not similar to car engines or watch mechanisms in the sense of depicting parts that are all relative to a single main behaviour of the system. They are more like a set of available freeways that some vehicle can travel along. Examples of these 'pathway maps' are metabolic pathways found in pathway databases,[16] stem cell pathway diagrams, anatomical illustrations of vasculature, lymphatic vessels, neural tracts, and ecological food webs (as shown in Figures 1–5). As these maps are intended to reveal widespread causal connections they are often referred to as 'connectance' diagrams, 'wiring' maps, and connections that represent 'global anatomic continuity' (Caswell [2005], p. vii; Meyers *et al.* [2005], p. viii)).

[16] Examples of these maps include: Reactome, Kyoto Encyclopedia of Genes and Genomes (KEGG), WikiPathways, Nature Pathway Interaction Database (PID), and Pathway Commons.

In all of these cases scientists are first concerned with representing widespread causal connections in some system without being tied to a single explanatory target. Once these causal connections are specified in a map, the map can be consulted to answer a variety of explanatory why-questions. Answers to these questions are provided by pathway information in the map, as opposed to mechanistic information. Consider how scientists discuss this process in the context of using metabolic pathway 'maps' or 'charts' in biochemistry:

> The first important thing to remember is that the chart is no more than a form of map. In many respects it is similar to a map of the London Underground, which is also very complicated. With the latter, however, we have learned to suppress the overwhelming detail in order to concentrate on those aspects relevant to a particular journey [. . .] A similar approach should be used when studying the metabolic chart. The details of individual enzyme reactions are very complex and very important [. . .] However, these details should not be allowed to confuse the mind of the reader when asked the question: 'How is glucose metabolized to fat?' When faced with such a problem, the student should learn to recall sufficient detail relevant to an overall understanding of the pathways involved, while maintaining an awareness of the detailed background information and mechanisms. (Salway [2004], p. 10)

This quote shows how scientists explicitly analogize biological pathways to maps of city-streets and roadways. Part of the function of such maps is to capture pathway information that is relevant to addressing a particular question of interest. These questions often involve specifying a 'particular journey' or how one moves from one location in the map to another—what higher-level causal connections constrain this movement or flow. For these questions, pathway information is explanatory. While lower-level mechanistic information is 'essential for completion of the journey, it is not necessary to an overall understanding of the journey' (Salway [2004], p. 10).

4 Explanation: Pathways and Mechanisms

Scientific explanations are often viewed as answers to 'explanation-seeking' why-questions (Hempel [1965]). Within this framework, a why-question and its answer represent an explanandum and its explanans, respectively. There are some explananda for which pathway information is explanatory and mechanistic information is not. This identifies one way to understand how pathway explanation differs from mechanistic explanation.

I will illustrate three examples of one type of pathway explanation with the diagram shown in Figure 6. In this figure, the letters and nodes represent variables, which are properties that can take on different values.[17] Arrows

[17] This follows an interventionist account of causation (Woodward [2003]).

capture the causal relationships that these variables figure in. As I will soon clarify with some examples, this diagram contains pathway information in the sense of containing information about causal relationships in some area, where these relationships have pathway features (i)–(iv), discussed in Section 3.1. Before I discuss Figure 6 further, it may already appear as though it contains information that answers why-questions that mechanistic information cannot answer. These include questions such as: How many different downstream products can substrate A produce? How many different upstream substrates can lead to the production of D? How many different ways are there to get from B to C? If the uppermost route from A to C in the diagram is blocked, what downstream products will excess substrate A produce? Providing answers to these questions is very natural within the interconnected 'road map' representation of available pathways. However, these answers require a representation of widespread, available causal interconnections and a kind of flexibility of causal starting-point or effect end-point that conflicts with the mechanism concept. In order to explore this further, I use Figure 6 to consider three cases—two from science and one from ordinary life.

Following an interventionist account of causation, the letters and nodes in Figure 6 represent variables, which are properties that can take on different values (Woodward [2003]). In a first example, these variables represent energy in the form of species in an ecosystem, while the causal connections (that is, arrows) between them represent prey-predator relationships.[18] In particular, ecological pathways and the larger food web provide a map of 'difference making' relationships that capture energy changes across species. A predator's energy levels 'depend' on its prey in a counterfactual sense—the presence and absence of the prey controls whether the predator acquires more energy or not.

[18] How should we understand the causal nature of these ecological pathways? Consider the causal arrow between variable A and its most immediately downstream node (at the top of Figure 6), which I call variable 1. A represents energy (in the form of a clam), where this energy can be 'present' or 'absent' depending on whether the clam is present or absent. 1 represents energy (in the form of a fish). 1 can take on the values 'extra energy' or 'no extra energy' depending on whether the fish has eaten a meal or not. The basic idea is that A is a 'difference maker' for 1. Energy changes at A make a difference to and have causal control over energy changes at 1. For similar causal models of food chains and ecosystems, see (Smith and Smith [2012], 325). The presence and absence of clams is 'causally relevant' to whether the fish gains energy or not. Changes in energy levels of prey cause changes in energy levels of their predators. In other words, A 'makes a difference' to 1, and 1 counterfactually depends on A. Counterfactual dependence and causal control are often considered hallmarks of causal relationships (Woodward [2003]). Of course, the clam is just one causal factor related to this outcome—this outcome also depends on other factors that regulate the fish's ability to consume the prey. However, pathways abstract from these other causes—they highlight causal connections that capture the flow of some entity or information. For a similar analysis of the causal character of metabolic pathways, see (Ross [2018]).

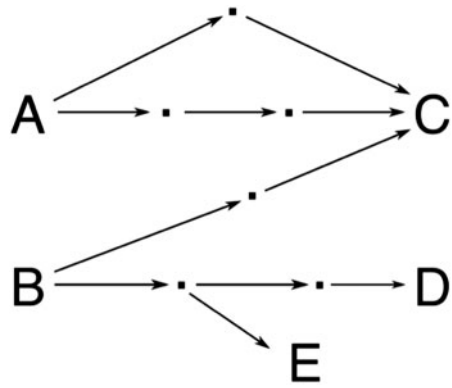

Figure 6. Pathway map: Ecological, anatomical, or city/street pathways.

Consider a scientific case where variables A, B, C, D, and E represent changing energy levels in different species. In this case, A represents these changes in a species of clam, B in a species of crustacean, and C, D, and E each in different species of fish (Stewart *et al.* [2004]). Figure 6 represents relationships between the energy levels of these species where downstream predators consume upstream prey and energy flows downstream, in the direction of the arrows. These species are located in the San Francisco Bay, which contains selenium—an element that is toxic to these organisms in high levels. It has been identified that fish species C contains high levels of selenium, while fish species D and E do not. Scientists want to know why this is the case—they want to know what explains these differences. They explicitly ask: 'Why did concentrations of Se differ so widely among predators in the Bay, and do those differences still occur? Does food web biomagnification of Se occur, and if so, why is it reflected differently in different predator species?' (Stewart *et al.* [2004], p. 4519). They explain this by citing the fact that fish species C is causally connected to species A, which contains high levels of this toxin. Alternatively, species D and E do not have high levels of selenium, because they are causally connected to primary producers with low levels of this compound, namely, species B (crustaceans). In this manner, 'exposures of top predators can be explained by food web relationships' (Stewart *et al.* [2004], p. 4519). More specifically, the differences specified by this why-question are 'explained by food-related variables' (such as those shown in Figure 6), background knowledge about how this toxin bioaccumulates along prey-predator connections, and the fact that in this ecosystem 'predators feed differently' (Stewart *et al.* [2004]).[19] Differences in how predators feed is captured in the

[19] Tracer experiments suggest that these organisms acquire selenium through their diet and that it bioaccumulates (or builds up) along prey-predator connections. Selenium is 'propagated up the

pathway information displayed in Figure 6. This pathway information is explanatorily relevant to the explanandum in this case because it is information that 'makes a difference' to it. If fish species C were no longer causally connected to A (or any primary producer with high selenium) it would no longer have high levels of this compound. If D and E were connected to A, they would have high levels of this compound. These differences are not captured with mechanistic information. All sorts of varying mechanistic details can instantiate these same pathways without these variations 'making a difference' to the explanatory target. So long as there are causal connections in the ecosystem represented by Figure 6, it does not matter 'how' energy and selenium move along these pathways, how they are metabolized by organisms, or further details about how predators consume their prey. It just matters 'that' these materials move through the ecosystem in the particular way captured by the causal pathways in Figure 6. Once these pathways and causal connections are known, particular explanatory why-questions can be addressed, regardless of the mechanistic details that instantiate them.

In a second case, the letters and nodes in Figure 6 represent variables that capture whether material is present (or not) in some spatial location along lymphatic pathways in a human patient. These pathways trace the physical location and movement of lymphatic fluid through these vessels. It is discovered that cancer is present in location C, but not in the nearby locations D and E, and there is an interest in knowing why this is the case. Similar to the previous example, this is explained by the fact that A is the primary site of cancer and that this upstream site is causally connected to C, but not to D and E. The cancerous cells move along lymphatic vessels from A to C, seeding the growth of new cancerous off-shoots along the way. There is no cancer in D and E simply because there is no causal route that connects A to these sites. This method of explaining disease spread by appealing to anatomical pathways is found in other medical contexts with minor variations. Both cancer and infectious material can spread along anatomical pathways such as lymphatic vessels, blood vessels, nerve tracts, and physically connected tissues (such as interconnecting spaces through the abdomen and thorax) (Meyers *et al.* [2005], p. 24). Researchers refer to these anatomical pathways as a 'scaffold' and 'interconnecting space' that disease processes use to navigate through the body. Tracing these pathways helps to identify (1) downstream locations of pathogenesis given an original upstream site, (2) upstream locations of original disease if downstream pathogenesis is known, and (3) expected locations of recurrent disease (Meyers *et al.* [2005], p. 55). Answering these questions requires assessing widespread interconnections within this space, which reveal

respective food webs' as predators acquire it from the prey they consume (Stewart *et al*. [2004], p. 4519).

'potential' routes that a disease entity can travel along.[20] Consider how scientists discuss the pathway concept in the context of this 'cognitive framework':

> Understanding the pathways of extension of intraabdominal disease requires conceptualization of the interrelationship of this network [. . .] as one interconnecting space. This continuity provides avenues for the direct spread of disease [. . .] A disease process, regardless of its site of origin or cause (tumor, inflammation, etc.), upon gaining access to this interconnected space, is provided an anatomic avenue for direct spread [. . .] This unifying concept of direct spread underlies an understanding of the clinical appearance of abdominal disease at a distance from its site of origin solely by direct spread. Knowledge of the possible pathways of spread provides a rational system for a clearer understanding of disease process [. . .] (Meyers *et al.* [1987], p. 601)

Here the emphasis is on depicting 'one interconnecting space' that reveals various potential pathways for disease spread. In particular, the different ways that some locations are connected relative to others figures in explanations of the pattern of spread and location of disease. This should clearly seem different from the discrete, isolable nature of mechanisms that capture individual causal structures, as opposed to a space of interconnected and potential causal routes. It is differences in the way that C is connected up in this space, relative to D and E, that explain why disease is present in the former but not the latter locations. In particular, the fact that C is causally connected to the primary site of disease, via identifiable anatomical pathways, and that D and E are not is what explains disease occurrence in the former, but not latter locations.

Third, consider an ordinary life case that captures a similar explanation to the above two examples. In this situation, the letters and nodes in Figure 6 represent variables that capture whether some entity is present (or not) in a city, where the arrows represent roadways that connect these cities up. Cities A and B have corn factories and this corn is delivered to cities C, D, and E with vehicles that travel along the routes displayed in Figure 6. It is discovered that city C has a supply of corn that is contaminated, while cities D and E do not. We want to explain why this is the case. Why is there this difference in contaminated and uncontaminated corn across these cities? This explanation is provided by the fact that a toxin has infiltrated the corn supply at factory A and that roadways connect the delivery of this supply to city C, but not cities D or E. An explanation of this difference is not provided by mechanistic, or

[20] Consider another example: Why does a clot in the carotid artery cause an infarct in brain vasculature (stroke) as opposed to a heart attack or pulmonary embolism? The reason is because the carotid directly irrigates the vasculature of the brain and not the heart and lungs. The clot gets logged in the small vessels of the brain before it ever reaches the more downstream heart and lungs.

lower-level causal information about this situation. In asking why city C has contaminated corn and why cities D and E do not, notice how unsatisfying it is for someone to tell you the intricate, fine-grained details of how the delivery vehicles were loaded, how they were off-loaded, and even how their engines work. These details do not matter for the difference in question. It does not matter whether this corn was delivered by vehicles that are electric, gas, diesel, front-wheel drive, rear-wheel drive, all-wheel drive, or the particular manner in which they were loaded and off-loaded with corn. The same roadways—with similar or different vehicles and loading and off-loading practices—would still give rise to the same difference in corn supply. Part of what this shows is that changes in these mechanistic details do not 'make a difference' to the explanandum of interest. What does make a difference is the higher-level structure of interconnecting roadways in this system and differences in how cities are connected up. If factory A was connected to C, D, and E, all of these cities would have contaminated corn. If it was not connected to these cities, none of them would have it. The pathway information that captures these higher-level causal features explains the differences in corn supply.[21]

Although a more detailed treatment of these pathway explanations is best left for the subject of a separate paper, it will help to briefly mention a few features of these explanations. To be clear, these cases do not involve an interest in explaining 'how' some entity travels along a pathway or 'why' some pathways have the structure they have. Instead, there is an interest in explaining the difference in some feature across downstream locations. This difference is explained by the causal connections or pathways in some domain, because changes in these connections would create changes in this explanatory outcome.[22] The same cannot be said for the lower-level mechanistic information that instantiates these pathways. In fact, so long as the pathway relations remain fixed, lower-level mechanistic information can vary without making a difference to the explanatory target. In each of these cases, information about the higher-level causal connections in some system explains particular outcomes that lower-level causal information cannot explain.

5 Conclusion: Mechanism and Pathway as Analogy

The world contains different causal systems with different features. These different features lead to distinct causal investigative strategies, explanatory why-questions, and possibilities for how such systems are represented,

[21] This example bears similarities to the concept of 'contact tracing' in 'disease-transmission pathways' in epidemiology and public health (Eames and Keeling [2003], p. 2565).

[22] The explanatorily relevant information in these pathways cases is similar to the 'connectionist' information discussed by (Bechtel and Levy [2013]) and the topological information discussed by (Huneman [2018]), although there are clear differences. Comparing these accounts to the present analysis is an interesting topic for future work.

described, and discussed. Biologists sometimes use particular terms to refer to these distinct causal systems, as seen in their usage of the 'mechanism' and 'pathway' concepts. This practice makes sense in the context of analogizing these systems to structures in ordinary life that we are familiar with.

Consider that analogy is 'a kind of similarity in which the same system of relations holds in two different examples' (Jee *et al.* [2010], p. 2). In this manner, analogy is often characterized as involving a mapping of structural features from a well-known base to a less well-known target (Gentner [1983], p. 157). This mapping can serve a number of purposes. A first main purpose is that it functions to transfer knowledge about some well-known domain to one that is unknown or poorly understood. In fact, analogy is considered one of the most effective strategies for 'convey[ing] an entire system of relations in a new, unfamiliar example' and, because of this, it is often used and examined in educational settings or any situation where a topic is described to a novice (Jee *et al.* [2010], p. 3; Gentner and Smith [2012], 131). It is easy to see this being done in the mechanism and pathway cases—entry-level biology textbooks introduce these causal structures and their features with 'machine' and 'roadway' analogies, respectively (Bender [1997]; Salway [2004] ; Firn [2010]; Berg *et al.* [2012]; Reece *et al.* [2014]).

A second function of analogy is that it is used to highlight key features of a target system to more expert audiences who already have some familiarity with it. In this case, the analogy emphasizes features of the system that are relevant for the context of inquiry. This makes sense of the fact that we see explicit use of these analogies in high-level research publications that focus on key features of some system for a particular purpose. For example, the 'road map' analogy, and associated concepts of 'routes', 'transportation', and 'flux', are used in metabolic research projects that focus on the rate of product formation and flow of metabolic material through biochemical processes. Alternatively, if there were an interest in capturing 'how' an enzyme converted a particular substrate into some particular product, the 'mechanism' concept would more likely be used. In this latter case, 'flow' is not the primary feature of interest, but instead the local, interacting enzyme components that produce a particular outcome. This leads to the characterization of enzymes as 'molecular machines', with constitutive interacting component parts.

A third function of analogy is that it supports problem solving in new contexts. An example of this is seen in cases of metabolic disease that are referred as 'inborn errors of metabolism'. These diseases are understood with a 'freeway' analogy that captures the pathological build-up of material—or 'traffic' caused by a 'roadblock'—and those therapeutic measures that address it.[23] In this case, knowledge about solving problems in the context of

[23] In these cases there is a blockage or 'roadblock' along the metabolic pathway (induced by a gene mutation). This blockage leads to upstream 'traffic' and the 'build-up' of some prior substance and the absence of the required downstream entity. The increasing build-up and inability to

freeway traffic is applicable to solving problems involving 'traffic' on metabolic pathways. In all of these cases, the analogy is a sort of 'psychological aid', which makes complex and potentially foreign features of biological systems more cognitively accessible (Hesse [1966], p. 3). This approach supports reasoning in a new domain by rendering the problem into a familiar context.

As philosophers, it is not just that we have a choice to interpret these causal concepts as relying on analogy—scientists explicitly use these analogies in their work. These analogies pick out similar causal structures that arise in a variety of biological contexts. For example, the pathway concept is used to refer to a particular causal structure (with features i, ii, iii, and v) that is found in molecular biology, biochemistry, stem cell biology, developmental biology, biomedicine, and ecology. Scientists' use of similar analogies in different contexts is consistent with evidence that experts classify systems by shared causal structure even when they arise in different scientific situations (Rottman *et al.* [2012]). Being able to do this—and to have an 'abstract understanding of causality' or ability to identify these 'causal system categories'—has advantages in the sense that these systems have similar implications for prediction, explanation, and control.

What consequences does this analysis have for philosophical accounts of biological explanation? My analysis indicates that biologists use a variety of causal terms to refer to unique causal structures in their field. Moreover, these unique structures motivate distinct causal investigative strategies, varying explanatory why-questions, and different explanatory patterns. We should want a philosophical account of explanation that accommodates this diversity—the diversity of causal structures in the world and our diverse techniques, methods, and strategies for studying them. We should expect a philosophical account of explanation to tell us why some causal details are explanatory and others are not, why some causal concepts are used in some situations, but not in others, and why scientists use a variety of causal concepts, as opposed to always using the notion of 'mechanism'. My analysis outlines an approach for doing this. As causal explanation is frequently understood as involving the explanation of some outcome by citing its causes, in some sense it should be unsurprising that different complex causal structures are likely to lead to different explanatory patterns. Standard accounts of mechanistic explanation can be compatible with this picture, but they should capture a methodologically sound conception of mechanism that leaves room for the explanatory role

produce downstream material can result in severe pathology, which is avoided by therapies that re-route this traffic around the blockage (via a bypass), shunt it into some non-pathologic downstream product, prevent flow into the blocked pathway, or find some alternative way to produce the final products downstream of the blockage. Examples of these diseases include phenylketouria, alcaptonuria, and glycogen storage disease.

of other causal concepts and structures. As many mechanistic philosophers suggest, it makes sense to understand mechanisms in biology as causal structures that involve constitutive relations, significant fine-grained detail, and causal-mechanical interactions expressed in terms of 'force', 'action', and 'motion'. This concept is well representative of many instances where biologists refer to 'mechanisms'. However, if we accept this picture of mechanism, we cannot also maintain—as some suggest—that 'mechanism' captures all causal concepts in biology or that it is representative of all causal explanation in this domain. Clearly, there are causal structures in this area with different features. These claims undercut an honest depiction of the complexity of causal structures in biology and the nuanced and complex reasoning practices, investigative strategies, and explanatory patterns that they generate.

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. What is wrong with this approach? First, if 'mechanism' is synonymous with any causal structure, how is mechanistic explanation different the generic claim that causes (or set of causes) explain their effects? Surely no account of causal explanation would deny this. Relatedly, if 'mechanism' is short for 'any causal structure' why not just say this? Mechanistic accounts have been motivated by the view that 'mechanism' is some type of important or unique causal structure, but this approach distances the account from this motivation. Relatedly, these accounts are typically motivated by our view that mechanisms have unique features that are not found in all causal structures. We see this in the fact that we refrain from calling single causes and causal chains 'mechanisms'. This leads to a second issue, which is that 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. Otherwise, they should stop referring to the scientific use of this term in suggesting the credibility of their accounts. A third disadvantage of this approach, is that by collapsing distinctions between the 'mechanism' and 'pathway' concepts, we loose sight of the role of analogy in causal and explanatory reasoning. Acknowledging the role of this strategy in biological reasoning provides a helpful understanding of the nature of mechanistic explanation and the character of other forms of causal explanation in this

area—such as pathway explanation—that have gone unnoticed. In fact, appreciating the role of analogy in this domain provides a fruitful way to understand the limits and range of a mechanistic explanation. Ever since the introduction of these new mechanist accounts, there have been widespread concerns about their over-extension to various types of causal explanation (Woodward [2013]; Dupré [2013]; Skillings [2015]; Halina [2018]). These concerns have been motivated by the view that mechanistic explanation—while important—does not capture the whole of explanatory practice in the biological sciences. This article gives one way to understand the nature and limits of mechanistic explanation, in a way that makes sense of biological reasoning, and captures a methodologically sound understanding of the causal concepts that commonly figure in this domain.

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