



Mechanisms, Pathways, and Networks: On Their Description and Explanation

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Abstract

Philosophical discussions of the biomedical sciences have attracted considerable interest over the past few decades, with much of the debate focusing on causation, complex systems, and explanatory levels, each understood in different ways. Although a wide range of views has emerged, a small number of notions have played a prominent and cross-cutting role in understanding how biological systems function. These notions, i.e., mechanism, pathway, and network, are especially central in molecular biology, the field we focus on in this contribution. More specifically, we aim to provide a comprehensive analysis of the relations among these notions as they are employed in molecular biology, shedding light on the relationship between description and explanation, which we treat as two distinct epistemic activities.

Keywords Description · Explanation · Molecular biology · Molecular biomedicine

Introduction

Over the past several decades, the biomedical sciences have become a major focus of philosophical inquiry. Much of this work has centered on issues such as causation, complexity, and different conceptions of explanatory levels. Despite the diversity of perspectives that have emerged, a limited set of concepts has consistently served as a common framework for understanding biological organization and function. In particular, the notions of mechanisms, pathways, and networks have assumed a central role, especially within molecular biology, which is the focus of this article. We examine how these concepts relate to one another in the context of molecular biology, with the goal of clarifying the connections between their descriptive and explanatory roles.

To contextualize this issue, it is worth recalling that the revival of mechanisms in the philosophy of science,

understood within a probabilistic framework, began in the early 1980s with a focus on the notion of process. More recently, this notion has instead been at the center of reflections aimed at drawing a distinction between mechanisms and processes. In Wesley Salmon's work (Salmon 1984, 1989, 1998), processes are spatiotemporally continuous lines that interact with one another and, by doing so, constitute the mechanisms responsible for the phenomena at stake. Although pivotal, Salmon's work was criticized, among other reasons, for being too abstract and too distant from explanatory accounts as they are actually pursued in the sciences, especially in the social and life sciences, where complex systems are investigated. Since the 1980s, the philosophical literature on mechanisms has addressed multilevel complex systems, their characteristics, how their behavior can be causally explained, and how they can be discovered. The debate on the nature, features, and roles of mechanisms promoted by so-called neo-mechanistic approaches is extremely broad and still ongoing. Although different authors have proposed definitions of mechanism that are sometimes slightly different and sometimes closely aligned (see, for example, Machamer et al. 2000; Bechtel and Abrahamsen 2005; Glennan 2017), a comprehensive definition that is often recalled is the following: "A mechanism for a phenomenon consists of entities and activities organized in such a way that they are responsible for the phenomenon" (Illari and Williamson 2012, p. 120).

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Beyond the specific commitments of individual accounts (see, for example, Darden and Craver 2013; Levy 2013; Andersen 2014a, b; Matthews and Tabery 2017; Glennan and Illari 2018; Krickel 2018; Ioannidis and Psillos 2022; Craver et al. 2024; Cordovil et al. 2024), neo-mechanists generally understand a mechanism as an organized set of parts or entities that act and interact to produce the overall behavior of a system. Discovering a genuine causal mechanism involves progressively filling in the gaps between causes and effects, so that a mechanistic causal explanation is expected to show why something is the case by unraveling how certain parts behave when interacting with other parts under given conditions.

The use of mechanisms, mechanistic notions and mechanistic evidence in the biological and biomedical sciences has been widely discussed, both in support of neo-mechanism and in order to highlight its limits and shortcomings. Examples span fields ranging from epidemiology to neuroscience, from evidence-based medicine to pharmacology, and from oncology to psychiatry (for example, Campaner 2010, 2016; Broadbent 2011; Campbell 2013; Aronson et al. 2018; Bechtel 2018, 2019, 2020a; Hartner and Theurer 2018; Canali 2019; Aronson 2020; Kendler 2020). This debate has helped to clarify both the distinctive methodological and conceptual tools of particular disciplines and subdisciplines, and the specific features of the cases addressed within them. Approaching complex systems in mechanistic terms has been regarded as effective in capturing their sensitivity to changing conditions, the importance of internal organization, the possibility of decomposing and recomposing systems, the admissibility of schematic and partial explanations, the explanatory role of relations within and across levels, and the need to distinguish mechanisms themselves from mechanistic models (see, for example, Glennan 2005).

Despite their popularity, mechanisms are not the only available framework to tackle features of complex systems in biology. Various neo-mechanists have acknowledged the importance of processes in their own accounts, in different respects—e.g., pointing out that the phenomena one wants to explain are often processes, or that the activity part of mechanism characterization aims to capture the process dynamic occurring in biological phenomena.¹ In recent years, processes have been brought to the forefront and have been claimed to be conceptually distinct from mechanisms and ontologically prior to them. Even if processual ontology per se is not antagonistic to new mechanistic characterization, a few processualists have stressed contrasting features in the literature, usually by analogizing new mechanistic characterization to machines. According to two major

advocates of the processual approach, John Dupré and Daniel Nicholson, “the world—at least insofar as living beings are concerned—is made up not of substantial particles or things, as philosophers have overwhelmingly supposed, but of processes. It is dynamic through and through” (Dupré and Nicholson 2018, p. 3), since “change is essential to the identity and persistence of any process” (Dupré and Nicholson 2018, p. 12). The occurrence and causal continuity of underlying activities or changes are taken to secure the identity of a given process over time. Process ontology emphasizes that organisms have life cycles, exist in thermodynamic disequilibrium with their environments, and depend on many environmental features. While not opposed to neo-mechanistic views, process ontology adopts a different stance toward organisms. As in mechanistic accounts, organisms are conceived as spatiotemporally extended entities, but their mereological composition is claimed to be fundamentally contingent, and their essence to consist in changeability. Accordingly, “the identity of an organism is grounded not in certain collections of privileged properties, or features which it could not fail to possess, but in the succession of diachronic relations by which it persists, or ‘perdures’ as one entity over time” (Austin 2020, p. 13). Process ontology is therefore presented in opposition to substance-based ontologies, which take entities as fundamental and define their identities independently of change (see also Meincke 2019; Meincke and Dupré 2020).

It is to note, however, that, beyond the ontological primacy, the mechanistic approach and the processual approach sometimes converge since some new-mechanists, as already recalled above, often have outlined the importance of processes in their accounts, underlining that the activity part of mechanism characterization aims to capture also the process dynamic occurring in biological phenomena.

Proponents of the processual view regard processes as the privileged route to understanding biological and biomedical phenomena, with consequences that extend from ontology to epistemic practice. This perspective has recently been discussed in relation to cancer research. The persistence of any organism depends on a precise balance between cell division, cell differentiation, and cell death, and all forms of cancer involve failures in the processes that regulate cell production and destruction. From a processual perspective, health depends on the regulated intertwining of multiple organismic processes, and carcinogenesis is understood as a disruption of processes at multiple levels of organization that normally regulate and stabilize one another (see, for example, Bertolaso and Dupré 2018). In cancer research, the epistemic role of processes has an analog in the notion of pathway (see, for example, Boniolo and Campaner 2018, 2022; Ross 2021; see also Thagard 2003; Schaffner 2016; Brzović et al. 2021; Balorda and Šustar 2025; Glennan

¹ Processes are discussed, for instance, in Machamer et al. (2000); Machamer (2004); Craver and Darden (2013); Glennan (2017).

2025), with molecular pathways constituting a central target of investigation in oncology.² Views on the relation between mechanisms and processes or pathways vary. Rather than defending the priority of one notion over the other, we argue that their usefulness depends on the epistemic context and on the purposes for which they are applied, both in descriptive and explanatory accounts.

A third notion that plays an important role in molecular biology is that of a network. Networks are central to scientific research and have become the subject of growing attention in philosophical analyses of molecular biology. When multiple biological intra- and intercellular pathways interact, they form biological networks, in particular a subgroup of them, that is, interactomes, which we focus on here. Networks represent patterns of interconnectivity and make it possible to approach biological complexity through graphs, whose nodes represent biological molecules, such as proteins, DNA, and RNA, and edges represent interactions among them. It is widely acknowledged that this approach has both advantages and disadvantages. On the one hand, it allows fragmented information to be integrated and presented in an intuitive visual form, highlighting key elements of the system (Qi and Ge 2006; Lima-Mendez and van Helden 2009). On the other hand, it involves strong simplifications of biological complexity, which may lead to limitations. Despite these simplifications, and sometimes because of them, network approaches have yielded informative results and important discoveries (see, for example, Vidal et al. 2011). Many simplifications concern semantic limitations, as network representations can be unspecific with respect to the structure and diversity of the molecules involved. To address this issue, numerous visual additions, such as cartoons, arrows of different colors and shapes, and supplementary tables, have been introduced to provide clearer and more detailed information about specific networks (Kohn 1999). Advocates of network approaches are aware of these limitations and have explored ways to overcome them (see, for example, Boniolo et al. 2010, 2012, 2015; Marchi et al. 2021). However, no consensus has yet been reached on how best to schematically represent networks without losing too much relevant detail (Faeder et al. 2005; Kitano et al. 2005; Bruck et al. 2006; Cho et al. 2012).

Taking all of this into consideration, the aim of our investigation is to offer a comprehensive account of the epistemological use of mechanisms, pathways, and networks (interactomes) in molecular biology, after having clarified in what sense they can be described and explained. Bechtel (2018, 2019, 2020b) has emphasized the relation between

mechanisms and networks, and Ross (2021) has discussed the relation between mechanisms and pathways³. We aim to extend these analyses by examining how such relations are considered from the point of view of explanatory and descriptive practices in science. All in all, the debate between supporters of processes—or, in explicitly molecular contexts, pathways—and of mechanisms is broad and multifaceted. Ross, e.g., has pointed out that “mechanism” and “pathway” both figure in biological explanations, that they have different features and roles, and therefore are not to be used just interchangeably. A whole range of various occurrences of “pathway” (e.g., signaling, metabolic, anatomical, developmental) is used—she has stressed—that can be differently related to what is referred to, on a case-by-case basis, as a mechanism. Ross delves deep into the peculiarities of the concept of pathway and its uses in explanatory contexts. We do not oppose her view, but, rather, focus on different steps in the elaboration of molecular explanations, intertwining discourse on the distinctive meanings of pathway/process and mechanism with that on the distinctive epistemic status of descriptive and explanatory accounts. In a recent article, Glennan (2025) has criticized Ross’s claims, maintaining 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” (p. 18). Without tackling ontological or metaphysical debates, as Glennan does, we agree with him that “any successful attempt to characterize the varieties of causal structures we find in the world must appeal to distinctive features of the world” (Glennan 2025, p. 20). However, we also believe that different concepts can be more fitting than others in doing so, depending, e.g., on the explanandum at stake, the purpose of the investigation, and the precise epistemic processes we are stepping into. In what follows, we will analyze in depth what such processes can be like in molecular biology.

Although no consensus holds regarding theories of scientific explanation, description and explanation are generally regarded as distinct epistemic activities. Once this distinction—which we embrace—is accepted, how should explanation and description be understood in relation to mechanistic, pathway, and network analyses? How do these approaches contribute to accounts of what a system is like, how it behaves, and why it behaves as it does? Foundational work in neo-mechanism explicitly claims that describing a mechanism in terms of entities, interactions, and organization amounts to explaining the behavior it produces: “to give a *description* of a mechanism for a phenomenon is to

² We assume, at least for our focus limited to molecular biology, that “pathway” and “process” can be taken as synonyms in that context. From now on, we will use only the first term even. The term used by the quoted author is kept in quotations.

³ Our interest is here devoted to epistemological issues. For some reflections on the relations between, e.g., a mechanistic ontology and pathways which refer to both neo-mechanism and Ross’s view, see Glennan (2025).

explain that phenomenon, i.e., to explain how it was produced” (Machamer et al. 2000, p. 3; italics added). Nevertheless, mechanisms exist independently, whereas explanations are epistemic constructions, and it is not mechanisms themselves that explain, but mechanistic models, since “models are explanatory when they describe mechanisms” (Craver 2006, p. 367). How, then, should pathways and networks be understood from an explanatory and descriptive perspective? This question is addressed in the following sections by examining more closely the relation between explanation and description (next section), and the relations among mechanisms, pathways, and networks (section “Mechanisms, Pathways, and Interactomes”). By reflecting on these issues, we think that a more detailed, coherent, and comprehensive perspective on epistemic processes in molecular biology can be achieved.

Explanation and Description: Enemies, Accomplices, or Allies?

As recalled above, “to give a *description* of a mechanism for a phenomenon is to *explain* that phenomenon, i.e., to explain how it was produced” (Machamer et al. 2000, p. 3; italics added). Emphasizing the close relation between description and explanation in discussions of mechanisms does not amount to equating them, nor is this what we suggest here. Actually, the relation between description and explanation has been discussed extensively in the literature for a long time. For example, Reese (1999), in an article tellingly titled “Explanation is Not Description”, notes that in the recent history of philosophy some authors have strongly emphasized the distinction between the two, including James (1907), Bergmann (1957), and Toulmin (1953). By contrast, others have defended some form of equivalence between them, such as Mach (1886), Skinner (1931), and Kantor (1953).

A first crucial point is the distinction between *description* and *explanation*, which serve fundamentally different yet complementary roles in inquiry. A description informs us that something is the case: it identifies a phenomenon, event, or object, referred to as the *describendum*, and specifies its observable characteristics. In doing so, it provides what is known as the *describens*, that is, the set of statements that depict what the object of investigation is like. Descriptions, therefore, are primarily concerned with representation: they aim to accurately portray features, structures, or occurrences. By contrast, an *explanation* moves beyond mere representation and seeks to account for *why* a phenomenon is as it is. Here, the focus shifts to the *explanandum*, the event or state of affairs that requires explanation. The explanation itself is

constituted by the *explanans*, which includes the reasons, causes, mechanisms, or principles that justify or produce the *explanandum*. In this sense, explanations are often causal, and they always make clear what relation holds between the *explanans* and the *explanandum*: they aim to uncover the dependencies, laws, or generalizations that make the phenomenon intelligible. The distinction can be further clarified by noting that descriptions answer questions of the form “*What is it like?*” or “*What happens?*”, whereas explanations address questions such as “*Why does it happen?*” or “*What brings it about?*”. While a description may catalog the properties of an object or the sequence of an event, listing features and hence being related to taxonomical activities, it does not, on its own, provide insights into the underlying ways of working. Consider the case of a car engine. A description might state that the engine consists of pistons, cylinders, and a crankshaft, and that the pistons move up and down during operation. This tells us *what* the engine is like and *what* occurs within it. However, an explanation would go further by clarifying *why* the pistons move: namely, because combustion of fuel-air mixtures generates expanding gases that, in turn, drive the mechanical motion. The explanation identifies the causal processes (the *explanans*) that account for the observed behavior (the *explanandum*). Similarly, we can describe Napoleon or an apple, the *describenda*, in many different respects. For example, we can describe Napoleon’s character in general, some of his military strategies, or his behavior in his final years. Analogously, we can describe what apples are like in general, or the features of a particular apple in front of us. The descriptions of properties of entities or events are not explanations of properties of entities or events. Rather, properties of entities or events can be described, and why they hold can be explained. We can explain why Napoleon won at Austerlitz, why an apple fell from a tree, or why it has a given phylogenetic history. More generally, we can explain why something has come to have certain features, why it has occurred, why it behaves in a certain way, or why it changes over time.

Etymology reflects these distinctions. The verb “to describe” derives from the Latin *de-scribere*, where *scribere* means to write and *de* indicates both the act and its completion. Thus, the term *describere* originally referred to the act of depicting a state of affairs by means of language. By contrast, “to explain” derives from *ex-planare*, meaning to make something plain or unfolded, and by extension to make something clear by indicating why it occurs in a certain way or why it changes over time. Surely philosophical analysis is not grounded in etymology, but etymology should be taken into consideration when we use philosophical terms.

Depending on the nature of the *describendum*, descriptions can be conceived from two perspectives, namely as *static descriptions* and as *dynamic descriptions*, which, although both yield a *describens*, differ significantly in their epistemic orientation. A *static description* concerns what a state of affairs *is like* at a given moment. It provides a snapshot of an object's properties, structure, or configuration without reference to temporal development. In this sense, static descriptions are *atemporal*: they abstract away from change and instead focus on identifying and characterizing the components or features that constitute the object of investigation. Differently, a *dynamic description* concerns how a state of affairs *changes over time*. Rather than presenting a fixed snapshot, it traces sequences, transformations, or processes. Dynamic descriptions are therefore *temporal* in nature: they capture motion, development, and succession, often emphasizing stages, interactions, and transitions between states. However, and this is a main methodological point, a dynamic description, despite its attention to processes unfolding in time, does not amount to a genuine explanation. While it tells us *how* something happens (i.e., the sequence of events), it does not necessarily tell us *why* it happens. In other words, it lacks the *explanans*: it does not explicitly identify the causes, underlying mechanisms, reasons, or justifications that account for the occurrence of those temporal changes. Thus, dynamic description remains at the level of *organized observation, rather than genuinely explaining*.

This distinction becomes clearer when applied to an internal combustion engine. As exemplified above, a static description of the engine would focus on its structural composition. It would identify the main components and describe their arrangement. This answers the question: *What is the engine like?* A dynamic description, by contrast, would describe the sequence of operations: the intake of the air-fuel mixture, its compression, ignition, expansion, and exhaust. This answers the question: *How does the engine operate over time?* It provides a temporally ordered account of the engine cycle. Yet, even this dynamic account does not truly explain the process. It tells us that combustion follows compression, and that expansion drives the piston, but it does not necessarily clarify *why* combustion occurs under those conditions,

why expanding gases exert force, or *why* these processes reliably produce mechanical work. A genuine explanation—i.e., an account which can be properly speaking labeled “an *explanation*”—would require reference to underlying physical laws, such as thermodynamics and gas laws, and to causal mechanisms that justify the observed sequence.

We can therefore distinguish three levels, as described in Table 1.

More generally, dynamic descriptions concern variations that involve the history of a state of affairs and its temporal evolution. They depict change over time and, depending on research interests, may refer to more proximal or more distal temporal phases. Dynamic descriptions concern changes in features or behaviors occurring through time, from conditions at an initial time t_i to conditions at a final time $t_f > t_i$. In sum, while dynamic descriptions enrich our understanding by introducing temporality and process, they do not suffice from a genuinely explanatory standpoint and need to be supplemented by causal reasoning. A comprehensive scientific account typically requires the integration of all three levels.

Emphasizing the nature and role of dynamic descriptions brings us very close to the domain of explanation, at least in some contexts. If static descriptions concern what a state of affairs is like, dynamic descriptions often implicitly attribute explanatory relevance to certain elements, such as proximal or distal causes, interactions, or functions. Identifying *that* something is explanatorily relevant does not equate with making explicit what the explanatory account is like. Returning to the falling apple, for example, the dynamic description may include the breaking of the petiole from the branch, which figures as a proximal cause of the apple's fall. More generally, by telling us that something has happened in a certain way, dynamic descriptions set the ground for then identifying some reasons, rather than others, why something happened or is happening over time. In doing so, they orient the subsequent explanation.

While descriptions and explanations remain distinct epistemic activities and should not be conflated, it is nevertheless possible to identify an important intermediate case: a dynamic description can be understood as an *enthymematic explanation*, that is, as a form of discourse that partially fulfills an explanatory role, while leaving some of its essential components implicit. As is well known, the notion of an enthymeme, especially discussed in Aristotle's *Rhetoric* (see 2014, I.2, 1355a), refers to an argument in which one or more premises are left unstated because they are assumed to be obvious, shared, or contextually given. In classical rhetoric and logic, an enthymeme is therefore understood as an incomplete syllogism, in which the audience is expected to supply the missing premise. However, in subsequent Greek and Medieval

Table 1 Description and explanation: The three levels of analysis.

Epistemic level	Question Answered	Epistemic Role
Static Description	What is it like?	Structural characterization
Dynamic Description	How does it change?	Processual/temporal account
Explanation	Why does it happen?	Explanatory account

usage, the term acquired a broader meaning, becoming less strictly tied to syllogistic reasoning and instead denoting a form of discourse in which elements are not always rendered explicit. We adopt the term in this latter sense, recalling that its persuasive force lies precisely in its implicitness: what remains unsaid is treated as contextually nonrelevant. Thus, we may say that a dynamic description often functions like an enthymeme. It presents a temporally ordered sequence of events, effectively narrating the “history” of a state of affairs, while omitting explicit reference to the causal principles, mechanisms, or laws that would render the sequence fully intelligible. In other words, it tells us *how things unfold* but leaves unstated *why they unfold in that way*. This omission is not accidental but context-dependent. In many descriptive settings, especially in everyday or observational discourse, the underlying causes are considered either obvious, already known, or irrelevant to the immediate purpose. As a result, the dynamic description relies on a background of latent knowledge that implicitly supplies the missing *explanans*. The listeners or readers are expected to “fill in”, if they want, the connections without them being explicitly articulated. Consider the case of an internal combustion engine again. A dynamic description might state: fuel enters the cylinder, is compressed, ignited, causing the piston to move, and then exhaust gases are expelled. This sequence presents a coherent temporal progression. However, such an account is enthymematic. It does not explicitly state why ignition leads to expansion, or why expanding gases exert force on the piston. These causal links are left implicit, presupposing a background understanding of physical principles such as combustion and gas expansion. In this sense, the dynamic description is not an explanation properly speaking, but, rather, an anticipation of an explanation, orienting it, where the explanatory premises are omitted because they are taken for granted.

Turning to mechanisms, if we focus only on the entities that compose them and on their spatial configuration, we provide a static description. If we focus only on how mechanisms change over time, we have a dynamic description that carves out the temporal scenario within which we operate. Eventually, if we are interested in “why” there are changes over time, we search for an explanation in which the boundaries of the mechanistic model are drawn by the dynamic description.

In the next section, we will argue, through several examples, first of all, that the claim “to give a description of a mechanism for a phenomenon is to explain that phenomenon” should be reformulated as follows: to give a static description of a mechanism for a phenomenon is to indicate its components and their spatial organization, whereas to give a dynamic description of its behavior is to give a sort of enthymematic explanation, preparing the ground for a genuine explanation.

In elaborating mechanistic explanations, where, as recalled, mechanistic models are designed, and contexts are extremely relevant (see also our “Conclusion”), dynamic descriptions play a very important role. Secondly, we will discuss whether the same analysis applies when the focus shifts from mechanisms to pathways or networks.

Mechanisms, Pathways, and Interactomes

Notwithstanding the widespread use of the term “mechanism” in the biological literature, there is no clear or generally accepted definition of it. It often appears that the precise articulation of this notion is primarily a philosophical concern, whereas biologists are typically content with an intuitive understanding of what a mechanism is, largely derived from ordinary language (see, for example, Moss 2012). The situation is different for the term “pathway”, which is defined with much greater precision in biology. From the philosophy-of-science-in-practice perspective we adopt, this difference is particularly important, as it allows us to investigate how the notion is actually used in scientific contexts. For instance, according to the National Human Genome Research Institute of the NIH, “[a] biological pathway is a series of actions among molecules in a cell that leads to a certain product or a change in the cell. It can trigger the assembly of new molecules, such as a fat or protein, turn genes on and off, or spur a cell to move.”⁴

A molecular pathway can therefore be understood as a temporally continuous series of n causal bonding steps, starting from one molecule and its interactions at an initial time t_i and leading, at a final time t_f , to a molecular outcome that fulfills a specific function. Molecular pathways can occur within a single cell, as in the case of the apoptosis pathway, or between cells that are in close proximity, as in intracellular signaling. They can also involve cells that are distant from one another, as in pathways mediated by hormones traveling through the bloodstream to reach remote targets. There are several types of molecular pathways, including metabolic, gene regulatory, and signaling pathways. Each of them, however, shares two key features. First, it consists of a temporally continuous succession of bonding steps, each corresponding to a causal interaction among molecules. Second, it is involved in the production of a molecule with a particular function. At this point, three issues are worth noting. First, focusing on pathways entails examining the performance of a specific function that is enacted under specific molecular

⁴ <https://www.genome.gov/about-genomics/fact-sheets/Biological-Pathways-Fact-Sheet#:~:text=A%20biological%20pathway%20is%20a,spur%20a%20cell%20to%20move.>

setup conditions, that is, within a particular molecular context (see Boniolo and Campaner 2018; Boniolo et al. 2012). For each pathway, “we can specify the set-up conditions as the molecules that initiate the first reaction in the pathway, and the terminating conditions as the molecules that are produced by the last reaction in the pathway” (Thagard 2003, p. 238). Changes in the molecular context constrain the performance of the function that a given pathway is meant to realize. Different contexts therefore call for different functional analyses of the behavior of the same molecule, as is paradigmatically illustrated by regulatory pathways. In this respect, the context can be said to set the conditions for the analysis’s adequacy. Context dependency thus constitutes a crucial aspect of the behavior of molecular pathways and, thus, a crucial element in molecular biology; elements of the context must hence figure in any description intended as a first step toward an explanatory account.

Second, identifying a molecule M as the beginning of a pathway and a molecule M' as its end involves an abstraction that enables us to focus on a particular succession of molecular steps. In nature, pathways do not have fixed boundaries, and different pathways often operate together to accomplish biological tasks. This, however, does not imply that anything can count as a pathway. Constraints are imposed, for example, by the features of the phenomenon under investigation and by the activities in which the pathway under consideration is involved. A description, therefore, plays a crucial role in setting the boundaries of the pathways to be considered.

Third, as already noted, the term “mechanism” is often used in an intuitive manner, and in many scientific articles the terms mechanism and pathway are employed without any substantial distinction. However, while the biological literature lacks a precise definition of “mechanism”, it does provide, as seen above, a definition of “pathway”. More generally, the relation between mechanisms and pathways can be understood from two complementary perspectives. On the one hand, a pathway can be conceived as a chain of basic mechanisms, each corresponding to a single step in the pathway. On the other hand, an entire pathway can itself be conceived as a mechanism.⁵ Which of these perspectives is preferable depends on the epistemic context.

Let us now turn to networks, and more specifically to interactomes. In the biological literature, interactomes are defined with considerable precision as the complete set of interdependent molecular interactions (see, for example, Plewczyński and Ginalski 2009; Bonetta 2010). In this sense, interactomes can also be conceived as representations of sets of mechanisms, of varying degrees of complexity, and of intersecting

pathways.⁶ It is often emphasized that interactomes capture the complexity of molecular interaction scenarios, and the kinds of interactions they include can vary. Some interactomes focus exclusively on protein–protein interactions, whereas others include all types of molecular interactions. Similarly, some are restricted to interactions within a single cell, while others also encompass interactions among different cells. Interactomes are nonetheless often endowed with explanatory power, and any resulting ambiguity is typically resolved pragmatically within the context of the specific study in which they are employed. To clarify how these notions relate to the epistemological issues discussed here, namely the construction of descriptive and explanatory accounts, we now turn to some concrete examples drawn from molecular biology.

The KEGG Network

Let us consider an exemplar case study of interactomes by focusing on a specific melanoma network available in KEGG, the Kyoto Encyclopedia of Genes and Genomes, developed by Minoru Kanehisa (Kanehisa et al. 2004, 2006; Aoki-Kinoshita and Kanehisa 2007; Kanehisa 2013). This biological database is a freely available web resource that integrates completely sequenced genomes with functional information on the molecules involved. Within KEGG, objects include genes and proteins, small molecules, reactions, pathways, diseases, and drugs (see Fig. 1).⁷

We focus on two specific pathways involved in the proliferation and survival of melanocytic cells through MITF activation following stimulation by stem cell factor (SCF) (Ronnstrand 2004; Lennartsson and Ronnstrand 2012). Let us

⁵ This is clarified in what follows, where we discuss the “p53 degradation mechanism” or “p53 degradation pathway”.

⁶ Bechtel (2020b, p. 1) has recently challenged the idea that “network representations are flat while mechanisms are organized into a hierarchy of levels” suggesting that the lesson to be drawn from the ways in which large-scale networks are analyzed in systems biology is different. Networks can be conceived as “clusters of nodes that operate as modules or mechanisms” (p. 1), stressing connectivity patterns holding between entities, and “bio-ontologies such as gene ontology (GO) [can be used] to annotate nodes with information about where entities appear in cells and the biological functions in which they participate” (p.1). This is taken to both support a mechanistic interpretation of networks and to be heuristically fruitful, providing new mechanistic knowledge on the parts and operations of the systems at stake. Rather than being opposed, mechanistic and network approaches are often used together, with networks too being interpreted as representing hierarchies of levels. Furthermore, processes enter the picture as well and are totally compatible with it: “nodes that cluster in the network and are annotated with the same biological process can be viewed as parts of mechanisms involved in those processes” (Bechtel 2020b, p. 6). A different view is held by Philippe Huneman (2025), who contrasts network analysis with a mechanistic methodology, while arguing that network analyses can also be explanatory.

⁷ This example is taken from Boniolo and Lanfrancione (2016).

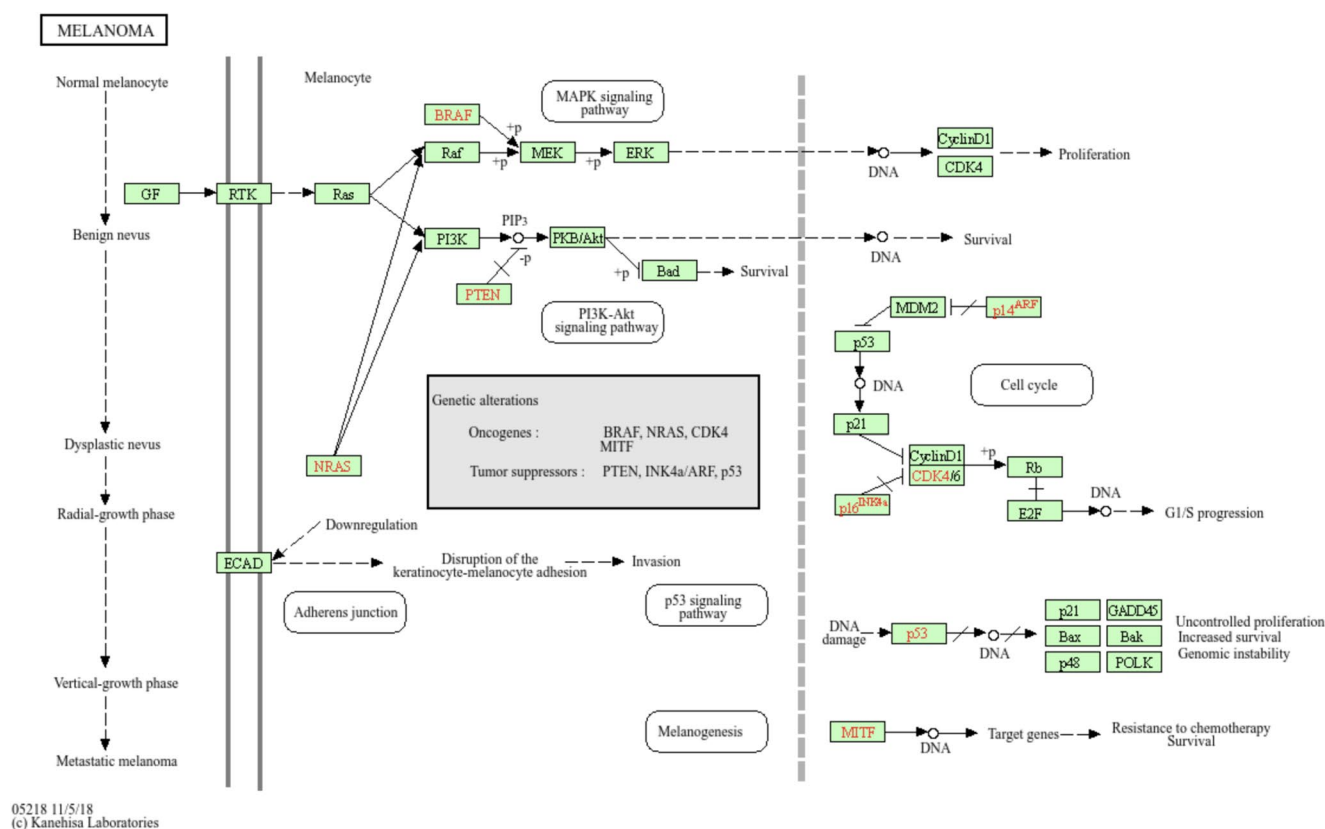


Fig. 1 The melanoma KEGG pathway. (Network from KEGG, © Kanehisa Laboratories. <https://www.kegg.jp/pathway/hsa05218>)

first consider the MAPK signaling pathway.⁸ A *static description* of such a pathway needs to consider the entities involved, that is, all the molecules at play. The set is the following:

< SCF, c-KIT, SRC, SHC1, Grb2, SOS, RAS, GTP, RAF, MEK, MAPK, ATP, ATP, ATP, ATP, ATP, ATP, MAPK >

It appears that ATP is indicated six times, that is, the exact number of times that a token of this molecule occurs in the considered pathways. A static description will be provided by making explicit what each member of the list above is like.

If we want a *dynamic description* of what happens, we can use the molecular biology jargon and express it as follows. The pathway starts from the SCF (a growth

factor—GF—prototype) and finishes with the activation of MAPK. More specifically, we have the binding of SCF to c-KIT (that is, a tyrosine kinase receptor (RTK) prototype in the same scheme), which leads to dimerization and auto-phosphorylation of the receptor. In this way, c-KIT can bind downstream signaling effectors, such as SRC and SHCA adaptor proteins (Roskoski 2005; Masson and Ronnstrand 2009). Then SRC can bind c-KIT and, by doing so, gets activated, phosphorylates SHCA, and suppresses differentiation to favor proliferation (Phung et al. 2011). SHCA provides association sites for the Grb2/SOS complex and activates RAS and RAF binding, thus sustaining MAPK activation⁹.

Considering what was said above on the dynamic description, this could be seen as an *enthymematic explanation*, where the description of what happens in time from SCF to MAPK activation leaves aside why each step is realized and why the final product is obtained; in particular, it leaves aside why the various binds and the various activations happen. We

⁸ In this case, following the nomenclature indicated by the *HUGO Gene Nomenclature Committee*; (www.genenames.org) MITF is the Microphthalmia associated transcription factor; SRC is the sarcoma viral oncogene homolog; SHCA is the SRC homology 2 domain containing, transforming protein A; Grb2 is the growth factor receptor-bound protein 2; SOS is the Son of Sevenless; GTP is the guanosine triphosphate; ATP is the adenosine triphosphate; PI3K is the phosphatidylinositol-4,5-bisphosphate 3-kinase; AKT is the serine/threonine-specific protein kinase; PIP2 is the phosphatidylinositol bisphosphate; PIP3 is the phosphatidylinositol trisphosphate; PDK1 is the phosphoinositide-dependent kinase-1; MAPK is the mitogen-activated protein kinases, originally called ERK (Extracellular signal-regulated kinases); MEK is the mitogen-activated protein kinase kinase; RAS is the Rat Sarcoma protein; RAF is a serine/threonine-protein kinase; GSK is the glycogen synthase kinase.

⁹ Grb2 is an adaptor protein involved in the signal transduction cascade downstream of several receptors, while SOS is a guanine nucleotide exchange factor that activates RAS. Stimulating the RAS/MAPK pathway finally leads to MITF activation by its direct phosphorylation (Samayawardhena and Pallen 2008). MITF is a transcription factor whose transactivation activity is increased when phosphorylated, resulting in increased proliferation of melanocytic cells (Vance and Goding 2004; Phung et al. 2011).

could have a “real” explanation if we were capable of giving an exhaustive *explanans* of the *explanandum* “the MAPK is activated”.

Let us consider the other pathway: the PI3K-AKT signaling pathway. Again, we can have a *static description* of the molecules involved, which are the following:

< SCF, c-KIT, SRC, P85-PI3K, P110-PI3K, PIP2, PDK1, ATP, ATP, ATP, ATP, AKT >

If we move on to a *dynamic description* of this pathway, we can observe that PI3K and AKT play a central role, especially in melanoma, by regulating cell growth, survival, motility, and metabolism (Davies 2012). PI3K is a heterodimer composed of a p110 catalytic subunit and a p85 regulatory subunit. Both subunits are responsible for the production of PIP2 and PIP3, whose function is to recruit signaling proteins to cell membranes and propagate the signal. Upon PI3K activation, both AKT and PDK1 bind PIP2 and PIP3 and translocate to the plasma membrane, where PDK1 phosphorylates AKT. The PI3K and AKT pathway mediates cell survival through MITF phosphorylation induced by MAPK. MITF phosphorylation stimulates both the transactivation of downstream effectors and the degradation of the protein through the ubiquitin-proteasome pathway, depending on cell context (Wellbrock et al. 2008). Moreover, PI3K and AKT signaling regulates MITF protein levels in cells through GSK activity (Terragni et al. 2011).

Again, we have what could be seen as an *enthymematic explanation*, in which the description of what happens over time, from SCF stimulation to PI3K activation, leaves aside why each step is realized and why the final product is obtained. In particular, it leaves aside why the various binding events and activations occur. We could have a genuine explanation if we were capable of providing an actual *explanans* of the *explanandum* “the PI3K is activated”, making the relation holding between the explanans and the explanandum explicit.

It is also worth noting that, at the initial stage, there are some shared bonds, namely those among SCF, c-KIT, and SRC, followed by a bifurcation into two different pathways. This depends on the molecular context, thus exemplifying what was noted above, namely that the pathway under consideration is context-dependent.

Here, we can identify three possible descriptive and explanatory foci: the network level, in this case an interactome; the level of the pathways composing the network; and the level of the single interactions composing each pathway.

From graph theory, we know how many edges, or links, a graph with N nodes has. Let this number be K . Thus, given any interactome with N nodes corresponding to molecules, we have $W < K$ links, since not all logically possible links are biologically possible. Graph theory also tells us that any graph can be decomposed into pathways of a given length. This means that an interactome with N molecule nodes and

W links between molecule nodes can be decomposed into Z pathways of a given length.

In the examples above, we selected two pathways, the first with 18 molecule nodes and 16 links, and the second with 13 molecules and 11 links, from the Z possible pathways of the KEGG melanoma network.

What this exemplifies is that, in general terms, any molecular pathway is part of a molecular interactome. Its static description provides information about the composing molecules, whereas its dynamic description offers an enthymematic explanation of what it produces, starting, in this case, from a proximal cause.

Let us now consider a molecular pathway with L molecule nodes and $L-2$ links. Any pathway composed of L molecules and $L-2$ links can be decomposed into $L-2$ interactions. Again, we can provide both a static and a dynamic description, that is, an enthymematic explanation of the interactions. The latter, by describing what is happening, also offers some guidance toward explaining why it is happening.

At this point, we could propose a minimal meaning of the term “mechanism”, by conceiving it as the basic component of a pathway, namely a single interaction. In this sense, any pathway with L molecules is composed of $L-2$ mechanisms, each producing a molecule that enters the subsequent step, that is, the subsequent mechanism. Beyond this “minimalist” use of “mechanism”, could there be another one that might prove fruitful in the debate on explanation and description in molecular biology? To address this question, let us consider the example concerning the degradation of the protein p53.

The p53 Mechanism/Pathway

Let us start by recalling that p53 is a tumor suppressor, discovered in 1979, encoded by the *p53* gene located on the short arm of the human chromosome 17 (we focus here on human p53). This protein is a central node in a complex signaling pathway that evolved to detect a variety of cytotoxic and genotoxic stresses that could compromise genomic stability and promote neoplastic transformation. Once activated by a stress signal such as DNA damage, hypoxia, unscheduled oncogene expression, viral infection, or ribonucleotide depletion, p53 exerts its role as “guardian of the genome” and mediates a series of cellular outcomes that can vary from cell cycle arrest to DNA repair, senescence, and apoptosis, depending on the cellular context. The p53 pathway is altered to some degree in all human cancers.

Despite the huge body of knowledge available on p53, many aspects of its functions and regulation remain rather obscure. For example, which stimuli lead to activation of p53 tumor suppressive activity? Which modifications and interactions are essential for p53 functions? What are the mechanisms that allow p53 to regulate one particular subset of target

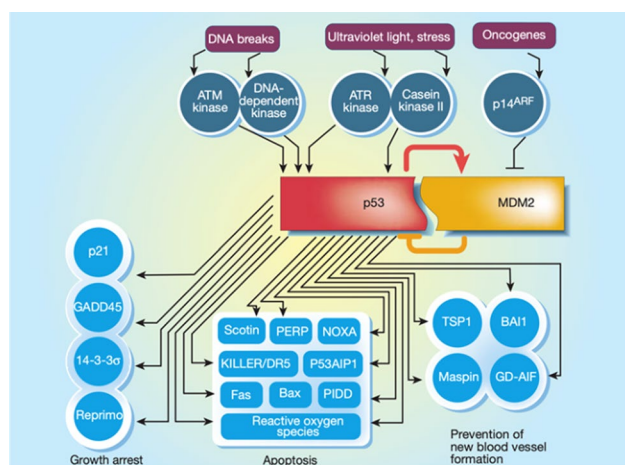


Fig. 2 The P53 network emphasizing its interaction with the ubiquitin ligase MDM2, which drives its degradation. From Vogelstein et al. (2000). Reprinted with permission

genes over another, leading to distinct cellular outcomes? (e.g., Gostissa et al. 2003; Hofseth et al. 2004; Hainaut and Wiman 2005).

Above, we have considered p53 pathways and p53 mechanisms. Moreover, we could also think of the p53 network, since p53 is a hub protein at the center of an intricate network that can be represented in different manners, depending on what we are interested in (see Figs. 2 and 3).

As recalled above, any interactome can be decomposed into pathways, and a pathway can be decomposed into a succession of elementary mechanisms, each one involving a single interaction. Among the many molecular contexts in which p53 is involved, we focus on its degradation, which has been well discussed in the literature. It is worth noting that in the dedicated literature, we sometimes find “p53 degradation pathway” and sometimes “p53 degradation mechanism”. In this case, “mechanism” is not used to indicate an interaction belonging to a pathway, but rather it is meant to indicate the series of successive interactions giving rise to a particular

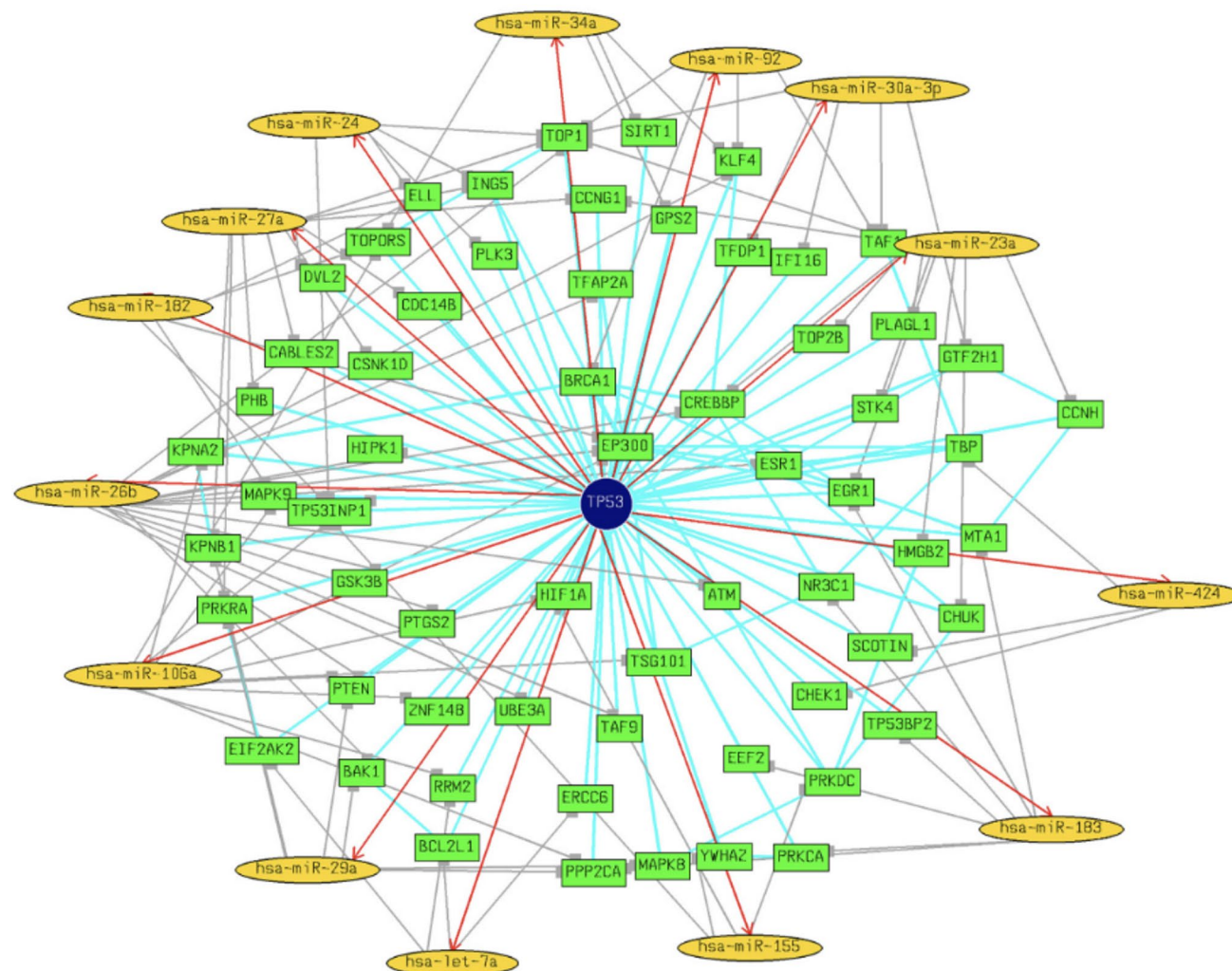


Fig. 3 The P53 network emphasizing the many molecules with which it interacts. From Sinha et al. (2008). Reprinted with permission

phenomenon: the degradation. Thus, in this specific biological case, “mechanism” is used as a synonym for “pathway”.

To wrap up, in the biological jargon, by “mechanism” sometimes we mean a basic interaction among molecules, sometimes a set of successive interactions aimed at producing a certain molecular phenomenon. There is no ambiguity for biologists: the issue is solved by considering the semantic context in which the term is used. The context, in other terms, orients the understanding of the term, which typically can figure both into descriptive and explanatory accounts.

However, returning to a dynamic description of the p53 degradation pathway/mechanism, let us highlight that it indicates that its function involves regulating the quantity of p53 in a cell. This is realized by the ubiquitin ligase MDM2, a protein encoded by the gene MDM2, which binds the N-terminal trans-activation domain of p53, thus inhibiting its transcriptional activity and stimulating its ubiquitination and consequent proteasome-dependent degradation. Ubiquitination is the pathway that causes a protein, in this case p53, to be labeled by the protein ubiquitin. This “labeling” step marks p53 for destruction by proteasomes, very large protein complexes within the cell. In these terms, the p53 degradation pathway/mechanism is composed of mechanisms, each depicting an interaction.

Summing up, we believe that, in order to adequately account for epistemic processes in the design of explanatory accounts, three epistemic foci, and their mutual relations, should be distinguished: static descriptions, dynamic descriptions, and genuine explanations. While being utterly compatible with views such as Machamer’s, Darden’s, Craver’s, Glennan’s, and others, our proposal is meant to draw more precise distinctions, to gain a better grasp of the various steps in understanding a biological system. Static descriptions identify what the components of the system are, dynamic descriptions how they change across time, and explanations why they do so. In this sense, we can hold dynamic descriptions to be what we labeled enthymematic explanations, insofar as their content is already selected and hence driven by some criterion of explanatory relevance. In other words, descriptions are presupposed by explanations, and they orient them. Examples above have shown how these matters then translate into actual analyses of phenomena in molecular biology when explanatory goals are at stake.

Conclusion: The Nest of Steps in Addressing Epistemic Processes in Molecular Biology

We have supported the idea that “describing” and “explaining” are two distinct epistemic activities, providing further articulation of the relation between them, in the light of some examples from molecular biology. Description concerns the

account of the features of a state of affairs, whereas explanation tackles the question of why such a state of affairs occurs. We have also shown that both static and dynamic descriptions can be provided. Static descriptions concern the relevant entities and their features, while dynamic descriptions concern changes over time and can be understood as a form of enthymematic explanation—in a peculiar sense, as implicitly orienting genuine explanations.

Moreover, we have underlined that, in molecular biology, there are usually three main foci of analysis: networks (in particular interactomes), pathways, and mechanisms. These foci are not mutually incompatible and depend primarily on which features of the *describendum* or the *explanandum* are being emphasized.

We have illustrated this perspective by discussing part of the melanoma KEGG network, with particular attention to specific mechanisms and pathways, as well as the degradation mechanism or pathways involving the protein p53. In doing so, we have disentangled in what respects and for what purposes it is appropriate to speak of mechanisms, pathways, or networks, and clarified the relations holding between them. This clarification allows us to better understand how molecular biologists shift descriptive or explanatory focus depending on the question at stake, without assuming that one epistemic approach is more important than the others. Also, through actual examples and related figures, we have tried to make clear what the various epistemic steps in addressing molecular biology can be like.

More generally, our discussion suggests that epistemic practices in molecular biology include different descriptive or explanatory aims, require different kinds of descriptions and explanations, and these descriptions or explanations can be combined to provide an understanding of biological phenomena meant to fit different circumstances. Static descriptions offer an essential framework for identifying the relevant entities and their organization, while dynamic descriptions make explicit that interactions unfold over time and lead to specific outcomes. The latter can be regarded as enthymematic explanations insofar as they implicitly identify the *relevant* change, which the actual explanation then addresses. Eventually, explanations tell us how this occurs and why the outcome has been produced.

We believe these distinctions help adequately address descriptive and explanatory accounts in molecular biology analysis, by providing us with tools fitting the foci in biological scientific practice on, according to the cases, mechanisms, pathways and networks. In sum, we have aimed to provide some elucidation of epistemic processes in molecular biology which take into deep account scientific practice, emphasizing not only that describing biological systems and explaining their behavior are related but distinct epistemic tasks, but how the two can be related: recognizing this distinction, and the

legitimate shifting among different explanatory foci, should help clarify how scientific understanding in molecular biology is constructed and justified in practice.

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