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**Explaining the behaviour of random ecological networks: The stability of the microbiome as a case of integrative pluralism[[5]](#footnote-5)**

**Abstract**

Explaining the behaviour of ecosystems is one of the key challenges for the biological sciences. Since 2000, new-mechanicism has been the main model to account for the nature of scientific explanation in biology. The universality of the new-mechanist view in biology has been however put into question due to the existence of explanations that account for some biological phenomena in terms of their mathematical properties (mathematical explanations). Supporters of mathematical explanation have argued that the explanation of the behaviour of ecosystems is usually provided in terms of their mathematical properties, and not in mechanistic terms. They have intensively studied the explanation of the properties of ecosystems that behave following the rules of a non-random network. However, no attention has been devoted to the study of the nature of the explanation in those that form a random network. In this paper, we cover that gap by analysing the explanation of the stability behaviour of the microbiome recently elaborated by Coyte and colleagues, to determine whether it fits with the model of explanation suggested by the new-mechanist or by the defenders of mathematical explanation. Our analysis of this case study supports three theses: (1) that the explanation is not given solely in terms of mechanisms, as the new-mechanists understand the concept; (2) that the mathematical properties that describe the system play an essential explanatory role, but they do not exhaust the explanation; (3) that a non-previously identified appeal to the type of interactions that the entities in the network can exhibit, as well as their abundance, is also necessary for Coyte and colleagues’ account to be fully explanatory. From the combination of these three theses we argue for the necessity of an integrative pluralist view of the nature of behaviour explanation.

**Keywords**: scientific explanation; mechanisms; mathematical explanation; behaviour explanation; integrative pluralism; network

**1. Introduction**

Explaining the behaviour of ecosystems is one of the key challenges for biologists: why ecosystems have the properties they have, which conditions make them exhibit a stable behaviour, how they react to perturbations, etc. have been some of the most debated questions among behavioral ecologists. Almost since the original publication of the “new-mechanist” *manifesto* (Machamer et al. 2000), the idea that explanation in biology proceeds by discovering mechanisms has been extensively accepted among philosophers of science (Glennan 2002; Bechtel & Abrahamsen 2005; Craver & Darden 2005, 2013; Craver & Bechtel 2007; Craver 2007). Drawing upon the findings in neuroscience and molecular biology, new-mechanists reelaborate causalism (Salmon 1984; Woodward 2003) and argue that to explain a phenomenon (*explanandum*) consists in describing its causes by providing a mechanism (*explanans*) responsible for the phenomenon coming about. According to the new-mechanist interpretation of explanation, a phenomenon can be considered fully explained only once a mechanism is provided and the way its components causally interact to produce the phenomenon is specified.

The universality of mechanistic explanations in biology and neuroscience as originally formulated in the new-mechanist *manifesto* has however been questioned on different grounds. A very popular “non”-mechanist view holds that some explanations in biology are at least *partially* given by appeal to the mathematical properties of the systems under investigation. Some of those who defend the necessity of introducing mathematical modelling in some biological explanations have emphasized the continuity of these explanation with mechanistic explanations, developing the concept of *dynamic mechanistic explanations* (Bechtel & Abrahamsen 2010; Bechtel 2011; Brigandt 2013, 2015), whereas others have highlighted the differences between both accounts, arguing that dynamic mechanistic explanation are indeed no-mechanistic (Issad & Malaterre 2015). A still more radical interpretation of this “non”-mechanist approach holds that some explanations in biology are given exclusively in mathematical terms, with no reference to any type of causal-mechanistic information in the *explanans* (Sober 1983; Huneman 2010; Lange 2013; Jones 2014). This last kind of explanation might be called *mathematical explanation*, and it is usually presented in complete opposition to new-mechanist models of explanation.

One of the most recent topics of attention of defenders of the last approach has been the study of the explanation of the behaviour of ecosystems. Some of the most salient behavioural properties of ecosystems (robustness, resilience, stability), they argue, are explained using the tools provided by network analysis. The use of these mathematical tools to explain how some biological systems behave in the long-term is very abundant in ecology (e.g. the diversity-stability debate) due to the complexity of ecological systems. It has recently been argued that explanations of the behaviour of ecosystems are given exclusively on the basis of the mathematical properties of the networks that are used to represent them (Huneman 2010, 2018a, 2018b, 2018c). The study of behaviour explanations that appeal to network analysis in ecology has focused exclusively on the study of ecosystems that form *non-random networks*, though. However, as far as we know, the explanatory features of behaviour explanations in ecosystems that organize forming *random networks* have received no attention among scholars. A network is said to be non-random when the aggregation of the elements that interact in the network lead to a concrete topological realization (small world, scale-free, etc.) with some *a priori* known properties. A random network, on the contrary, is characterized for lacking a known topological realization, and thus the properties of a random network cannot be known *a priori*, but have to be mathematically discovered. We suspect that the difference between systems that organise according to a random network and systems that organise according to a non-random network get their explanatory force from different sources. In this paper, we aim to test that intuition by studying the explanatory features of the models that explain the ecological behaviour of a random network the human microbiome.

A microbiome is a collection of microorganisms (bacteria, viruses, fungi, etc.) that reside in a concrete environment (Marchesi & Ravel 2015). In case of humans, our microbiome is believed to contain hundreds of species (Human Microbiome Consortium 2014). Furthermore, human’s gut microbiome is known for its ecological stability behaviour: even if it varies quite a lot from one person to another, the gut microbiome is believed to be very stable for one single individual, who tends to carry the same species of microbes for a long period (Dethlefsen & Relman 2011; Faith et al. 2013). Recently, Coyte et al. (2015) and Foster et al. (2017) have elaborated a model to explain why the human gut microbiome exhibits a stable behaviour over long periods of time. They found out that, contrary to an usual assumption in evolutionary biology, competition, and not cooperation, is the key factor explaining this stable behaviour. Here, we analyse how Coyte and colleagues explain the stability of the microbiome by appealing to linear stability analysis. This case shares several elements with other cases of behaviour explanation in terms of networks, although it also differs in some features that led to some differences regarding the nature of behaviour explanation and that we aim at clarifying.

In §2 we introduce the notions of mechanistic and mathematical explanation, and frame the choice of our case study in the context of the mechanistic vs. mathematical debate. In §3 we present Coyte and colleagues’ explanation of the stability behaviour of the human microbiome. In §4 and §5 we discuss the consequences of the case study for the understanding of scientific explanation. In §4 we argue that even if the explanation in our case study displays a model of mechanism, it does not specify a proper causal story to account for the *explanandum*, thus suggesting that mechanisms, as understood by new-mechanists, do not play the explanatory role. In §5 we argue that the explanatory force in Coyte and colleagues’ account comes from the display of a mathematical model of the behaviour of the microbiome, as it is provided by their linear stability analysis of the network that the microbiome instantiates. However, in contrast with some recent analysis of behaviour explanations of ecosystems in terms of networks, we argue that insofar as an essential component of the explanatory force of Coyte et al.’s model is their appeal to the different interaction types within the ecological communities (cooperative, exploitative, competitive) that might exist in the microbiome, and thus to the establishment of a range of topologies, and not exclusively to a concrete topology, their explanations differs from purely mathematical explanations. We further argue that the appeal to this element is exclusive of random networks. In §6 we explore the pluralistic consequences of our case study for the analysis of behavior explanation, suggesting that our case study supports the general appeal to integrative pluralism. Finally, we present our concluding remarks.

**2. Two models of explanation in biology: mechanistic vs mathematical**

In its most basic meaning, to explain a phenomenon consists in giving the reasons why the phenomenon obtains in a concrete system due to its behaviour. Those reasons may be specified by appealing to laws of nature –deductive-nomological theories of explanation– or by appealing to the causes that are responsible for the phenomenon. In the first case, it is usually assumed that to explain a phenomenon is to provide an argument so that the phenomenon (*explanandum*) logically follows from the laws of nature that regulate the behaviour of the system where it obtains, given a concrete set of initial conditions (*explanans*) (Díez 2014; Alleva et al. 2017). In the second case, the phenomenon is embedded in a causal network in the world, in a way such that the phenomenon (*explanandum*) is a causal consequence of the behaviour of the system (*explanans*) (Woodward 2017).

New-mechanist theories of scientific explanation are of this last kind. They assume that to explain a phenomenon consist in citing its causes by providing a mechanism that specifies how those causes produce the phenomenon. The definition of “mechanism” is different in differing accounts (Nicholson 2012; Deulofeu & Suárez 2018), although most new-mechanists share a similar conception: a mechanism consists in a set of *entities* with a concrete spatial *organization* plus a set of *activities* governing the behaviour of those entities (*model of the mechanism*). The phenomenon to explain, new-mechanists argue, *causally* obtains as a consequence of the activities of the entities (*causal story*) (e.g. Machamer et al. 2000; Bechtel & Abrahamsen 2005; Craver & Darden 2005, 2013; Craver 2007; etc.). For new-mechanists, the presence of a *model of the mechanism* and of a *causal story* is necessary and sufficient for having a mechanistic explanation (Issad & Malaterre 2015: 270).

New-mechanists generally accept a hierarchical view of mechanisms, thus not confining themselves to the narrow approaches of previous causalists (Salmon 1984). Furthermore, they usually neglect the capacity of mathematical models *alone* to explain any phenomenon. In a well-known paper, Kaplan & Craver argued that “the [mathematical] generalizations are explanatory because they describe the causal relationships that produce, underlie, or maintain the *explanandum* phenomenon” (2011: 612) and insisted that:

“In successful explanatory models in cognitive and systems neuroscience (a) the variables in the model correspond to components, activities, properties, and organizational features of the target mechanism that produces, maintains, or underlies the phenomenon, and (b) the (perhaps mathematical) dependencies posited among these variables in the model correspond to the (perhaps quantifiable) causal relations among the components of the target mechanism.” (2011: 611).

Mathematics, thus, can play an explanatory role for new-mechanists only if it captures *a causal relationship* among the entities that are posited in the mechanism. Otherwise, they are merely “phenomenological models”, which represent the reality and allow predictions without really explaining why those predictions obtain (e.g. Kepler’s laws, Snell’s laws, etc.) (Díez 2014).

Radically contrasting with this last view, some people have vindicated a more substantial role for mathematics in biological explanation by highlighting the importance of mathematical properties for explaining the features of some biological systems. The explanations that rely on the mathematical properties of the system to explain a phenomenon have been called “mathematical explanations” (Baker 2015), or “structural explanations” (Huneman 2018a).[[6]](#footnote-6) They have been defined as follows:

“Family of explanations for which the mathematical tools used in the description of an explanandum system belong to a mathematical structure whose properties are directly explanatory of some aspects of the system (such as equilibria, behaviour, limit regime, asymptotic behaviour, etc.) (…) They explain by accounting for the explananda through pinpointing structural relations that are mathematical relations of some sort.” (Huneman 2018a: 695)

The mathematical properties that appear in a mathematical explanation might be of different types, and they could be used to explain different kinds biological questions. They might consist in: the application of an arithmetic theorem to explain the life cycle of some species (Baker 2005, 2009, 2015); the establishment of one or more points of equilibrium to explain a tendency in a population (Sober 1983; Kuorikoski 2007; Rice 2012, 2015; Suárez & Deulofeu, under review); the application of statistics to explain certain evolutionary patterns in a population (Walsh 2015); the discovery of a concrete topology that explains the behaviour of a complex system (Huneman 2010; Jones 2014); the use of matrix calculus to explain the processes that regulate some physiological states (Issad & Malaterre 2015); etc.[[7]](#footnote-7)

Not every substantial use of mathematics in biological explanation needs to be in principle completely opposed to every element of the new-mechanistic account of explanation, though. The explanation of some cyclical biological processes such as the circadian rhythms has been argued to constitute an extension of mechanistic explanation, namely a *dynamic mechanistic explanation* (Bechtel & Abrahamsen 2010; Bechtel 2011; Brigandt 2013, 2015). In general, an explanation is considered a dynamic mechanistic explanation in virtue of making use of some mathematical model in its *explanans* that: (1) is essential to account for the *explanandum*, (2) replaces the role that new-mechanists attribute to the causal story, without being itself a causal story *stricto sensu*, (3) is combined with a model of mechanism, i.e. a set of entities and activities plus their organization. Nonetheless, it is precisely because of the lack of a causal story that some of these explanations entail that they have been argued to be “anti”-mechanistic, or at least far away from the core elements that new-mechanists consider necessary and sufficient to formulate an adequate scientific explanation (Issad & Malaterre 2015).

In still more extreme cases, however, some explanations in biology have been argued to be even more substantially mathematical, abstracting away also from the model of the mechanism, and being explanatory of the biological phenomenon *exclusively* in virtue of the mathematical properties of the *explanans*, whatever their type (Sober 1983; Baker 2005; Huneman 2010; Jones 2014; cf. Kuorikoski 2007; Potochnik 2015). The analysis of behaviour explanations in biological systems that present a network structure (e.g. ecosystems, immunological systems, etc.) has been argued to follow this pattern of mathematical explanation. The behaviour of this type of systems is usually explained in two steps: (1) the system is attributed a concrete network topology, which provides the mathematical properties of the system; (2) the properties of this topology are studied and then its behaviour is attributed to the biological system, explaining why the system behaves how it does. Because of the reliance of the last type of mathematical explanation on the topological properties of the networks, it has been called *topological explanation* (Huneman 2010, 2018b; Jones 2014; Brigandt et al. 2017).

Those who have studied topological explanation more attentively have made two points: First, that the appeal to the topological properties of the mathematical structure alone (its graph structure, or its network motifs, for instance) is *sufficient* to explain some of the properties of the biological system that the structure is believed to represent, irrespectively of the entities and the activities of the entities that realize those systems (see also Huneman 2018c); second, that the addition of any mechanistic details, instead of making the explanation of the properties more precise, obfuscates the question and turns out to be deeply irrelevant for the embedding of the *explanandum*. This second point might be explicated as follows: in sharp contrast with dynamic mechanistic explanations, in topological explanations, neither the model of mechanism (nature of the entities, nature of the activities), nor the causal story are explanatory relevant. All that matters in the explanation is that the topology is provided and that it gives information about the organization of the system.

One of the fields where the appeal to topological properties to explain biological phenomena has proven more fruitful is in the diversity-stability debate in ecology (McCann 2000; Nikisianis & Stamou 2016). In that context, the aim of ecologists is to elaborate network models that represent the relationships among the biotic members of an ecological community with the aim of inferring some general features about its behavioural patterns. To do so, once the network model is elaborated and linked to behavioural properties of the ecological community which is being studied, ecologists analyse the global properties of the network –e.g. how it will respond to a perturbation, to an increasing/decreasing number of nodes, to an increase in the number of connections, etc.–, and then attribute the exact same properties to the ecological community that the network is believed to capture. The driving question in the diversity-stability debate is whether increasing the number of species in the community would make the community ecologically stable[[8]](#footnote-8) and, if so, under which conditions. Some recent discussion surrounding the diversity-stability hypothesis have tried to unravel how some ecological communities will react to the loss of some species for communities where the connections between the nodes are non-random (Solé & Montoya 2001). In the context of network theory, a network is non-random if it instantiates a particular topology, e.g. scale-free networks, small worlds (Figure 1). Because the topological properties of theses types of networks are known, and both small worlds and scale-free networks are known to be highly stable to the elimination of some of their nodes (Montoya & Solé 2002), it is *enough* for ecologists to prove that a concrete ecosystem instantiates one of these networks to explain why the ecosystem exhibits a stable behaviour. The explanation in these cases would work as follows:

‘Ecosystem E instantiates a network N which, in virtue of being of type X has the topological property P. Therefore, E also has P’ (adapted from Huneman 2010).

Interestingly, these types of explanations: (1) do not mention either the entities or the activities that might be going on in the ecosystem, insofar as network analysis only represents relations in terms on the number of nodes and the strength of their interactions –thus being applicable to multiple kinds of systems just replacing “node” for the objects that are studied in the field (Internet, metabolic networks, social networks, etc.)–; (2) do not elaborate any kind of causal story that is responsible of producing the phenomenon under investigation. The *explanandum* (E having P) is accounted for simply because the network is of type X, and thus necessarily must instantiate P. Thus, topological properties alone would explain P obtaining, and there is no role left for mechanisms (Huneman 2010).

The study of behaviour explanations provided in terms of networks analysis has been centred in the study of explanatory patterns in systems that instantiate non-random networks (e.g. scale-free networks, or small worlds). However, no attention has been devoted to study the explanatory patterns that underlie behaviour explanations for systems that instantiate purely random networks. We suspect that because random networks do not have a particular topology associated, nor a exclusive type of network motifs, the type of explanatory patterns that underlies behaviour explanations for the systems that instantiate them might be slightly different from the types of explanatory patterns provided in terms of non-random networks. The rest of the paper is thus devoted to analyse the explanatory features of a behaviour explanation given for a system that instantiates a random network.

**3. Case study: The stability of the human microbiome**

The human gut is an ecosystem consisting in a large community of microbes (≈1000 species), whose stability behaviour is crucial to maintain human’s health.[[9]](#footnote-9) Recent empirical research suggests that the human microbiome exhibits a stable behaviour: even if different individuals might bear different microorganisms in their microbiome, the species that compose the microbiome of an individual, and their relative densities, tend to remain largely stable during her lifetime (Dethlefsen & Relman 2011; Faith et al. 2013). The reasons that make such essential community to behave stably despite the existence of constant perturbations are yet unknown, though. One possible way to explain why the microbiome behaves stably would be to argue that it does so as a consequence of the great number of species that compose it. The explanation in this case would work as follows: insofar as the microbiome is an ecosystem which is composed by a great number of species, and ecosystem biodiversity is believed to foster ecological stability under certain circumstances, then it will be expected that the human microbiome exhibits a stable behaviour (McCann 2000; Ives & Carpenter 2007). This way of accounting for the *explanandum* poses a serious challenge, though: the positive correlation between diversity and stability only works for non-random ecological communities; however, the opposite has been demonstrated to be true for random communities, in which an increase in biodiversity fosters *in*stability (May 1972). Because the microbiome is a random ecological community that due to its biological nature is expected to be suffering constant perturbations, then it will tend to be unstable. Therefore, what ecological theory predicts (instability) and what is empirically observed (stability) are at odds. The question that arises is then the following: what type of dynamics are instantiated in the microbiome so that its interactions result in a stable behaviour?

Fairly recently, Mougi & Kondoh (2012) have elaborated a model that overcomes the difficulty that May’s results pose to explain the stability behaviour of random communities. In their view, the problem with May’s model is that he only analysed communities with one interaction type (i.e. where all the members were either mutualistic, or antagonistic, etc.), and they thought that different results might be obtained if the communities were studied taking into account the fact that there might be different interaction types interacting simultaneously. They observed that, in fact, the existence of different combinations of interaction types in a community might be a solution to May’s results, and thus allows showing that an increase in biodiversity *alone* (i.e. irrespectively of the interacting types) does not trigger instability. Mougi & Kondoh applied their reasoning to macroscopic communities, showing that in communities with different interaction types, an increase in biodiversity does not necessarily foster instability, if the proportion of cooperative types is high, thus explaining stability in terms of cooperation.

Coyte et. al’s explanation of the stability of the microbiome follows the same logic as Mougi & Kondoh’s research. They agree with them that a key element to explain the stability behaviour of the microbiome is the appeal to the existence of different interacting types. However, contrary to the claim by Mougi & Kondoh that cooperation fosters stability in macroscopic communities, Coyte et al. argue that it is *competition*, *not cooperation*, what explains the stability behaviour of microscopic communities. To prove their claim Coyte et al. develop a mathematical model and a series of computational simulations of the behaviour of the microbiome, and showed that the same result (i.e. that competition stabilizes and cooperation destabilizes communities) was observed irrespectively of the size of the community. Based on their analysis, they hypothesize that the destabilizing effect of cooperation is due to the strong dependencies among species that it generates, which would lead to the appearance of feedback loops in the community, whose destruction would lead the community to collapse. These feedback loops, on the contrary, would not appear when the species in the microbiome compete, thus making the community behaviour more stable. In this section, we analyse the mathematical model that Coyte et al. use to explain the stability of the microbiome.

Coyte et al.model the microbiome as a network of interconnected species to abstractly study its dynamics by applying a combination of linear stability analysis with computational simulations.[[10]](#footnote-10) Their model describes the dynamics of density change of one species given the interaction with another species , and it is written as a modification of the Holling type 1 functional response:

System of equations (1) expresses how the density of species *i* will change over time given its own intrinsic growth rate its interaction with other members of the same species, i.e. its self-interaction rate , which it is assumed to be the same for all species (i.e. = *s*), and its interactions with the members of every other species, or interaction strength , such that *j* *i* ). Finally, S expresses the total number of interacting species of a given community.

In Coyte et al.’s model, the interaction of a given species within the network will be determined by two parameters: first, the connectivity of *i* in the network, *C* = [0, 1], defined as “the fraction of all *S* species that a single species *i* interacts with” (Coyte et al. 2015: Supplementary 4). Second, the nature of the interaction types between microbial species. They can take up to five possible forms, based on the signs of / : (cooperation +/+), (competition -/-), (exploitation +/-), (commensalism -/0) and (amensalism +/0). Accordingly, the proportion of interaction types between species must be established for a given community, being the total proportion of interaction types equal to one:

(2)

Given this, Coyte et al. elaborate a phase portrait of their model to study its dynamical behaviour. To do so, they start by determining its equilibrium points, i.e. the points for which the variables of the system remain constant over time. Second, they determine the stability of each equilibrium point. An equilibrium point will be stable if after a small perturbation in the system the variables return to the same values that they had before the perturbation, and it will be unstable otherwise (Figure 2). To determine the stability of each equilibrium point they perform a linear stability analysis. The linear stability analysis for an equilibrium point *y* is performed in three steps: (i) constructing the Jacobian matrix *M* of the system. The Jacobian matrix of a *N* dimensional system is a square matrix whose elements will be given by the partial derivatives of the system. (ii) evaluating *M* at the equilibrium point *M|y* (iii) computing the eigenvalues of *M|y*. Once the eigenvalues are computed, the stability of such equilibrium is determined by the following criterion: the equilibrium point *y* will be stable if and only if the real part of all the *N* eigenvalues of *M|y* is negative.[[11]](#footnote-11)

When working with large dimensional systems the eigenvalues of the Jacobian matrix *M* tend to follow a concrete distribution. In the case of Coyte et al.’s model, the eigenvalues that they compute for each equilibrium fall into an ellipse of horizontal radius in the complex plane and centre at (,0), being *s* the “average self-interaction”, except for a single eigenvalue which can lie outside (Figure 3). Therefore, because an equilibrium requires all its eigenvalues to have a real negative part to be stable, an equilibrium in Coyte et al.’s model will be stable if and only if

(3)

Once we have briefly introduced a basis to study the stability of a given large dynamical system we can focus on analyse our case study. If we denote by *y* a given equilibria for Coyte’s model, the Jacobian Matrix evaluated at that equilibria has the following entries (Coyte et al. 2015: Supplementary 3),

(4)

so that its eigenvalues —and therefore its stability— depend on the values of the self-interactions *s* and the terms, i.e. on the type of interactions between the species (cooperation, competition, etc.). Moreover, the connectivity also plays a role in the entries of the Jacobian matrix, so that the lower the connectivity of the network, the more terms will be equal to zero.

Coyte et al. are interested in computing the eigenvalues for each equilibrium point because the ecological stability behaviour of the system is characterized in terms of magnitudes directly related to the eigenvalues distribution of such equilibrium. First, if the equilibrium point is mathematically stable, then it will be ecologically stable too, i.e. the species density before the perturbation took place will eventually be recovered afterwards. In their context, a perturbation is produced when the densities of the species of community changes. Second, the behaviour of the community will be classified as more or less stable depending on how quickly it recovers its initial density distribution after the perturbation. Mathematically, this can be studied by analysing the eigenvalue distribution, such that the more negative the values of the distribution, the more attracting the equilibrium point will be, i.e. the faster the densities will go back to their initial states, and thus the system will be classified as more stable.

To study how the degree of ecological stability of each equilibrium point depends on the parameters , *s*, , in system of equations (1), Coyte et al. run a series of simulations with different community types (exploitative, random and competitive). To do so, they define a measure of stability *U*, such that:

(5)

(5) gives the rightmost eigenvalue, i.e. the eigenvalue that represents the lower level of ecological stability. Right after, they study the behaviour of the following derivative , that measures how *U* varies with respect to the proportion of cooperative interactions . The sign of this derivative describes whether the community behaviour becomes more stable (if it is positive) or less stable (in case it is negative) in function of the proportion of interaction types of the species in the community. With this method, they show that for a given value of *C* and any given community type, gradually increasing cooperative interactions nearly always increases the overall return rate (the time it takes for the community to recover its initial densities after the perturbation), and the likelihood of the community being unstable (Figure 4). This method also serves to prove the key role that *C* plays in the community behaviour: for a constant value of , gradually increasing the value of *C* always has a destabilizing effect on the community. The overall result of their different simulations is that the higher the values of and the higher the values of *C*, the less stable the community will be (Figure 5).

A final step in Coyte et al.’s analysis is the ecological interpretation of their results, i.e. the explanation of what happens in the ecological community when the values of and *C* increase so as to make the community less stable. In their view, communities with high values of *C* and might generate strong dependencies among some of its members, which could be transformed into the existence of feedback loops in the community. The existence of feedback loops makes the community sensitive to small perturbations, insofar as a small change in the density of one of the species might trigger a cascade effect in the community that will in the end move it towards a different equilibrium point than the one it had before the perturbation. A low value of *C* and avoids the creation of feedback loops and, therefore, has a stabilizing effect.

Coyte et al. rely on the empirical work done by Stein et al. (2013) to claim that their model is supported by empirical data. After the linear stability analysis and the different simulations they run, Coyte et al. make the following prediction: the proportion of destabilizing cooperative interactions in a stable microbiome has to be low in comparison with competitive and exploitative links, which will be predominant. The data presented by Stein et al. provides empirical validation for such hypothesis, thus suggesting that their model captures the right explanation of the stability of the microbiome.

**4. Coyte’s explanation as non-mechanist: Explaining without providing a causal story**

The case study presented above illustrates a type of explanation that we argue does not strictly follow the standard conception of scientific explanation as presented by the new-mechanists. To recapitulate, new-mechanicists argue that to explain a phenomenon consist in: first, identifying a model of mechanism (individualized by its entities, its activities and their organization); second, identifying a causal story by means of which the model of mechanism produces the phenomenon to be explained. The question is now to determine in which sense (and to which extend) Coyte et al.’s explanation of the behaviour of the microbiome does not fulfil these two requirements. Particularly, we argue that even if a model of mechanism can be identified in Coyte et al.’s explanation, it is not the case that the explanatory force of the explanation they provide comes from a causal story being told.

Let us start by considering whether Coyte et al. individuate a model of mechanism. As we explained above, Coyte et al. model the microbiome as a network of interacting microbial species that behaves following the dynamics dictated by the system of equations (1). In their model, the entities are the different *S* microbial species that are part of the network, i.e. that compose the microbiome, and whose densities are being studied. The activities of each of the entities are determined by the type of interactions they engage in (competition, cooperation, etc.), and would be given by the sign of . Finally, the organization of the community is given by the random network that describes the interactions among the species, including its number of nodes and its connectivity. Because the elements of the model of mechanism seem to be present in the case of the explanation given by Coyte et al., it seems that the first requirement to have a mechanistic explanation is satisfied by their model.

Once the model of mechanism has been specified it becomes necessary to show how the interactions among the different parts that compose it can produce the *explanandum*. In the case of Coyte et al. the *explanandum* isthe stability behaviour of the microbiome, that is, how the species densities remain constant over time despite the existence of perturbations (i.e. some species that vary their densities). The *explanans* says that the key element that makes the microbiome stable is the existence of a high degree of competitive interactions among the species that compose it (so that competition *explains* stability). To get the appropriate connection between the *explanans* and the *explanandum* Coyte et al. proceed as follows: (i) they stipulate a community type (e.g. exploitative community); (ii) they determine its equilibrium points; (iii) they analyse the behaviour of the rightmost eigenvalue in function of the variation of the value of two key parameters of the topology (the connectivity *C*, and the proportion of interacting behaviours, as defined in (2)) in order to determine the degree of stability of each equilibrium point. In their analysis they observe that, for a fixed value of *C*, proportionally increasing tends to make the system less stable (ecologically: less resilient). In other words, the time that the system will take to return to its initial equilibrium state after a perturbation will be larger, until a critical value of is reached, such that the return time is equal to *∞*, that is, the system becomes unstable.[[12]](#footnote-12)

Now, the question for the new-mechanist is: does Coyte et al.’s mathematical model for the explanation of the stability behavior of the microbiome actually captures *a causal story* of what happens in the microbiome so that it is ecologically stable? We suspect that the answer to this question is negative. First of all, because the way how Coyte et al. determine its stability behaviour is by means of a linear stability analysis of the system of ODEs specified in (1). And second, because even once the linear stability analysis is performed, what needs to be modelled to produce the *explanandum* is how the variations in will affect the stability behaviour of the system.

Concerning the first point, the new-mechanist might argue that once the system is modelled, each ODE tells us a different causal story, so that the system as a whole is just an abstraction of the sum of all the individual causal stories modelled by each different equation. But, even when all these causal stories are put all together, they do not lead to the production of the phenomenon. At most, the ODEs give us information about how the variations in the densities of some species will respond to the variations in the densities of others. They do not give any information about how these variations are produced, nor how the variation of the densities of one species will affect the variation of the density of the rest of the species that compose the microbiome. By themselves, they say nothing about how the dependencies among the entities that compose the microbiome produce its stability. The only way in which the ODEs might say something about it is by numerically computing its evolution with respect to time (i.e. the trajectories of the system) when a minimal perturbation occurs.

However, for their explanation, Coyte et al. do not even consider the particular trajectories of the system. Their explanation only requires to study the stability of the equilibria by performing the mathematical steps described before (creating the Jacobian matrix, evaluating it, studying its eigenvalues, etc.), a procedure which does not require to specify the intermediate values of any of the variables of the system. It is enough to study its long term behaviour, no matter which intermediate processes generate it. For this reason, it is difficult (if not impossible) to see how any of the steps followed by Coyte et al. describe a causal story that relates the specific interactions of the entity with the phenomenon to be produced.[[13]](#footnote-13)

In relation to our second point, it seems to us that the explanation by Coyte et al. includes an additional step that makes it even harder to see how the causes can produce the stability behaviour. Concretely, once the ordinary differential equations (ODEs) generated by the system of equations (1) are solved, Coyte et al. still need to run simulations to determine how the variations in the proportions of the interacting species will affect the stability of the community. This step is crucial, because their *explanans* is precisely that a community will be stable if and only if it has the right proportion of interacting types. This step, as we said, is performed by analyzing the variations of the rightmost eigenvalue to an increase in the proportion of . But it is not specified which of the concrete species interactions will become cooperative, nor is that necessary to highlight the negative impact of cooperation on stability. A knowledge of the proportion of species that interact cooperatively is enough to establish their claim. Thus, again, the rehearsal of a causal story seems unnecessary to produce the phenomenon that Coyte et al. are explaining and therefore their explanation is not mechanical.

**5. Explaining with mathematics: combining topology with interaction types to explain stability behaviour**

Coyte et al.’s model explains the stability behaviour of the microbiome but it does so in non-mechanistic terms. The question now is to determine how their model gains its explanatory force. In this section, we argue that what makes Coyte et al.’s model explanatory is the combination of the topological properties of the network instantiated by the microbiome with the knowledge about the dynamics that each of these topologies acquires in virtue of the combination of interacting types in the community.

Let us start by discussing the role of the topology in their *explanans*. In Coyte et al.’s model, the knowledge of the parameters and allows determining the value of *C* and thus provides the topology of the system. Notice that if our network were non-random, i.e. if it realized a concrete topology, at this point we would have all the necessary information to decide whether the network is stable or unstable. Take the example of a scale-free network: were the microbiome a scale-free network, we could already identify its hubs (these nodes whose alteration would destabilize the network), as well as how it will behave if we increase the number of nodes, if we destroy some of the edges between nodes, etc. If this were the case, Coyte et al.’s explanation would be much simpler: the microbiome behaves stably *because* it realizes a scale-free network. This type of explanation would have the same nature as other topological explanations, gaining its explanatory force simply from the nature of the network that the system instantiates (Huneman 2010, 2018b; Jones 2014).

However, contrary to what happens in non-random networks, the microbiome instantiates a random network, which means that none of its topological properties will be known *a priori*. For that reason, Coyte et al. need to study the dynamics that the topology instantiates in order to determine which are the conditions that will make it stable. Furthermore, in their case, what ultimately explains whether the dynamics instantiated will be stable is the proportion of interacting types in the microbiome. That is, not every community with the same topology will be equally stable. Their explanation, therefore, needs to combine the determination of the topology of the system with the study of the dynamics that the variables that instantiate that topology will have under different conditions, i.e. for different interaction types (Figure 6).

Therefore, Coyte et al.’s explanation of the stability behaviour of the microbiome would consists in something like:

‘Ecosystem *E* instantiates a network *N* which (i) *N* corresponds to one of a set of topologies *φ*, and (ii) *φ* has the right proportion of interaction types. Thus, *N* has property *P*. Therefore, *E* also has *P*’

Let us now develop what this model of explanation consists in. First, concerning (i), it is possible to mathematically compute which networks will make the microbiome stable, but this knowledge alone will render the *explanandum* unexplained, because there is still an element missing: which is the property in virtue of which *N*’s topology is stable? Or, in other words, why *N* has a dynamics such that the system will tend to keep its stability? Appealing to its topology, by itself, will undermine the answer, because the system is purely random and there are multiple states that will make it stable, as well as multiple states that will make it unstable. But this option is not possible in the case of purely random networks, and therefore the explanatory force of Coyte et al.’s model, even if requiring the appeal to the topological structure of the microbiome, needs to be acquired from somewhere else.

Second, about (ii), our proposal is that the key additional element that makes Coyte et al.’s model fully explanatory of the stability behaviour of the microbiome is their appeal to the different interaction types that might appear in the network. The reason is that for the microbiome to be stable it is necessary that the *dynamics* instantiated by the topology are conducive to a stable equilibrium state. The only way of showing what are the conditions under which the dynamics instantiated by a random topology are conducive to a stable state is by studying their response to slight modifications in the parameters that define the system, namely . The way of computationally studying this is by evaluating the response(s) of the rightmost eigenvalue to changes in the values of in relation to changes in the proportions of interacting types in the community. In other words, it is necessary to study how the changes in the proportions of cooperative, competitive, exploitative, etc. interactions will affect the stability of the microbiome. Only once this response is studied, and the results are analyzed, will the model be fully explanatory of the stability behaviour of the microbiome. In this sense, even if knowing the topology is necessary, since the dynamics of the system directly depends on the topology it instantiates, its knowledge is not sufficient to explain its behaviour. It is necessary to additionally understand how the community will respond to different proportions of interaction types. Only after this is done will the model will gain its explanatory force, and Coyte et al. can assert that competition *explains* the stability behaviour of the microbiome.

Even if the points we have made here are relative to the case study we have analyzed, we suspect that our conclusions about the nature of explanation can be extended to all the cases of behaviour explanations when they are given in terms of random networks. One key feature that distinguishes random from non-random networks is that the latter, but not the former, have specific properties derived from their network motifs and that can be unequivocally ascribed to every system that instantiates them. This does not mean however that random networks do not have network motifs: it means that their network motifs will highly depend on the way in which the network is constructed, and it will not be constant among all the systems that instantiate a random network. Because of this, we suspect that explanations of the behaviour of systems that instantiate a random network can only be given by studying an alternative mathematical property of the network (e.g. in Coyte et al.’s, the dynamics of the network), because: first, their network motifs will only be known once its mathematical properties have been studied; second, they will be highly variable depending on the mathematical properties that the network instantiates. This sharp contrast between random and non-random networks is thus fundamental to understand why explanations in terms of the latter can be exclusively topological, whereas in the case of the former the topology has to be complemented with an appeal to dynamics.

**6. A case for integrative pluralism in behaviour explanation**

Until now we have argued that Coyte et al.’s explanation of the stability behaviour of the microbiome is neither purely mechanistic, since it lacks a causal story, nor exclusively topological, since the topology needs to be combined with a dynamics to be fully explanatory. We now argue that our case study supports an integrative pluralistic picture of behaviour explanation in biology. Following Mitchel (2003) and Brigandt (2010, 2013b; Brigandt et al. 2017), we take an explanation to be integrative when it requires the combination of concepts from different fields and of different types in order to gain its explanatory force. Such integration is normally driven by pragmatic considerations about the question asked: that is, as some scientific questions are about very complex phenomena, their responses usually require the integration of the knowledge of different fields to be fully satisfactory (Brigandt 2013). In this sense, integrative pluralism is both beyond simple pluralism and against reductionism. Like simple pluralism, integrative pluralism accept that some phenomena in ecology might be explained by simply telling a causal story (i.e. describing a mechanism), whereas others might be exclusively given in mathematical terms. However, integrative pluralism tries to go beyond this simple idea by showing how some complex phenomena are explained by integrating knowledge from different fields (ecology, population genetics, molecular biology, etc.), using both, causal mechanistic strategies and mathematical modelling. Against reductionism, integrative pluralism embraces the idea that even if the explanation of a complex phenomenon requires the appeal to knowledge from different fields, all of them are indispensable for the *explanans* to account for the *explanandum*. In other words, that the explanation of the phenomenon is only possible by integrating all the knowledge provided by the different fields, which would not be obtained if the knowledge of one of the fields were reduced to the knowledge of some of the others.

Following the tenets of integrative pluralism, we will make the point that the explanatory model of the stability behaviour of the microbiome presented by Coyte et al. exemplifies an integrative explanation. Concretely, their explanation combines a model of a mechanism with a mathematical model plus a series of computational simulations, integrating mathematical knowledge (network modelling, linear stability analysis), with knowledge about the patterns of interactions in ecological communities. We further argue that the necessity to integrate knowledge about the model of the mechanism with knowledge about the mathematical properties of the community (including its topology) is common for every scientific explanation that accounts for the phenomenon in terms of random networks.

As we argued in §4, Coyte et al.’s *explanans*, despite not telling any causal story of what happens in the microbiome, describes a model of the mechanisms enumerating which are the interacting entities, what type of activities they engage in, and the type of organization that the microbiome has. Also, as we argued in §5, their explanatory model relies on the dynamics of the microbiome, which is acquired in virtue of the topology that it instantiates. The study of the behaviour of this dynamics determines a set of possible topologies, any of which will be stable, and thus one of the possible states the microbiome could be at. Importantly, both the model of the mechanism and the mathematical model are necessary and none of them is by itself sufficient without the other for Coyte et al.’s *explanans* to account for the *explanandum* in terms of “competition”, as the authors say it does.

First of all, the model mechanism without the dynamics is completely unspecific about the stabilizing role of competition in the microbiome. Since they are explaining a behaviour (stability behaviour), and the behaviour is the result of the set of interactions between the entities (the species that compose the microbiome), it is necessary to study the way in which the interactions result in the behaviour that is observed. Or, in other words, detailing the entities that interact and the activities they engage in is insufficient to explain the stability behaviour if the way how these interactions make the system change is not studied adequately. For sure, the model of the mechanisms specifies that competition is one of the activities that the entities of the microbiome engage in and that can affect its stability. However, because the model of the mechanism in itself does not describe how the system changes in time, its specification is insufficient to make competition explanatory.

Secondly, the topology of the system, without the knowledge of the model of the mechanism, is also insufficient to explain why the microbiome exhibits a stable behaviour. As the community instantiates a *random* network, its network motifs have to be studied by analyzing its dynamics. In the case of Coyte et al. they perform a linear stability analysis to study the reaction of the community to small perturbations. This analysis, however, does not make competition explanatory of the stability behaviour of the microbiome. As we explained extensively in §5, once Coyte et al. have determined the distribution of that make the microbiome stable, they have to study how the variations in the proportions of interaction types will affect the stability of the community. In other words, what makes competition explanatory in Coyte et al.’s model is not the strength of the interactions between the members of the microbiome, but the character (competitive, cooperative, exploitative, etc.) that those interactions have.

The previous observation entails that it is possible to have two communities with the same topology (i.e. with the same nodes, edges), but where only one of them is stable, whereas the other is not, due to the role that the interaction types have on the stability behaviour of the microbiome. Importantly, the knowledge of the influence of the interaction types on the dynamics that the topology instantiates is only possible once the model of the mechanism has been specified. Before this happens, and cooperation, competition, exploitation, etc. have been defined as possible activities of the entities, it is impossible to know whether the interaction types will have any influence on the dynamics of the network, since the latter is not the case for many other networks that might instantiate a random topology (e.g. the internet). Therefore, the knowledge of the dynamics is, by itself, insufficient to make competition explanatory, since the knowledge that competition will influence stability derives from the knowledge of the model of the mechanism of the microbiome.

From the two previous observations we derive that the explanation of the stability behaviour of the microbiome is a case of integrative pluralism, where a model of the mechanism needs to be combined with a mathematical analysis in order to be explanatory. As we argued in §5, the stability behaviour of the microbiome cannot be explained exclusively in terms of the topological properties of the network. Since it is a random network, it needs to be complemented with the study of the dynamics that the topology instantiates. As we argued there, this is a consequence of the fact that the network motifs of a random network can only be discovered *a posteriori*, by determining the effects that the interaction types have on the stability of the network. However, this claim about the study of the dynamics cannot be extended to every random network, since the reasons that make each random network stable will be different depending on their nature, and thus on the type of mathematical analysis that has to be done to determine its properties. The type of mathematical analysis will nevertheless depend on the model of the mechanism that is instantiated in each case, which will determine the nature of the entities that interact, as well as how their activities will be produced. The point we are making is thus that every scientific explanation that relies on the realization of a topology will be a case of integrative pluralism if the network that is realized is random.

**7. Conclusion**

The case study analysed in this paper fits well with the recent tendency in philosophy of science to emphasize the important role that mathematics play in some scientific explanations. Particularly, our case study, even if focused on behaviour explanations, shares many similarities with some of the cases of topological explanation analysed by Huneman (2010, 2018a, 2018b). As in the cases he studies, the stability of the microbiome cannot be explained purely in mechanistic terms, due to the impossibility of telling a causal story that explains how the system behaves. Additionally, the explanation we analyse in this paper also gains its explanatory force from the specification of the topological properties of the system. However, and in contrast with the cases of explanations of the behaviour of non-random networks, in the case presented by Coyte et al. (which analyses the behaviour of a random network) the specification of the topology of the community is not enough to account for the *explanandum* (the stability behaviour of the microbiome). The authors are also required to study the dynamics that the topology instantiates, as well as to discover which set of topologies will make the system stable in virtue of its dynamics. Such set of topologies can only be discovered by studying the response of the rightmost eigenvalue to different proportions of . Because the knowledge that the interaction types will influence the stability behaviour of the microbiome is only acquired after the model of the mechanism for the phenomenon is expelled out, Coyte et al.’s explanation constitutes a case of integrative pluralism. In other words, the explanatory force of their model is only gained from the combination of mathematical and mechanistic knowledge.

As a consequence, the analysis of our case study proves two main points: a) the explanation of the behaviour of non-random ecological networks is different form the explanation of the behaviour of random networks. That is to say, because the network is random, there is not any network type whose specification would automatically explain the behaviour of the system, and thus the network motifs have to be discovered; b) behaviour explanations of random ecological networks are cases of integrative pluralism, in which knowledge from mathematics and from ecology are integrated to solve a complex problem. Concretely, the model of the mechanisms determines how to construct and study the mathematical model so that the *explanans* that accounts for the *explanandum* can be provided.

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1. Logos, BIAP / University of Barcelona (Spain). [↑](#footnote-ref-1)
2. Logos, BIAP / University of Barcelona (Spain). [↑](#footnote-ref-2)
3. Egenis: The Centre for the Study of Life Sciences, University of Exeter (UK). [↑](#footnote-ref-3)
4. Department of mathematics, Universitat Politècnica de Catalunya (Spain) [↑](#footnote-ref-4)
5. The paper is the result of the discussion among the three authors, who actively collaborated in the development of all the ideas. JS conceived and structured it. RD and APC wrote §3. RD and JS wrote the philosophical analysis. [↑](#footnote-ref-5)
6. To refer to mathematical explanations as “structural explanations” might be confusing, since the later could be interpreted as special cases of the former, as one reviewer has correctly suggested. However, the way in which Huneman (2018a) describes them, as well as the family of explanations that he includes under the umbrella of “structural explanations” makes clear that the two are synonymous. For purposes of clarity, however, we will refer to this family of explanations as “mathematical explanations”. [↑](#footnote-ref-6)
7. We take all the aforementioned properties to be different types of mathematical properties. [↑](#footnote-ref-7)
8. The exact definition of stability is an agitated topic in ecology, and different diversity-stability hypotheses are formulated accordingly (McCann 2000: 230, Table 1; Nikisianis & Stamou 2016: 35–36; Gonze et al. 2018: 42, Box 1). In most cases, though, a system is qualified as stable if and only if it is able to return to its initial state after a perturbation (resilience), or also the capacity of a population to resist invasions by external species. We will specify later what “stability”means in our case study. [↑](#footnote-ref-8)
9. # In ecology, the concept of “stability” can be used to mean both that the number of species of the microbiome remains constant (i.e. that no species gets extinguished, also called *persistence*), and that the species density in the community recovers quickly after the community has been perturbed (i.e. once the density of one of the species in the community has slightly changed, also called *resilience*). A community whose species density remains constant is said to be in equilibrium. Obviously, if a community is stable in the second sense, it will also be stable in the first sense, but the opposite is not necessarily the case. In the case study that we present here, “stability” refers to the ability of the microbiome to recover its initial species density after a perturbation, i.e. it is a model to study resilience.

   [↑](#footnote-ref-9)
10. Their research consists in three different mathematical methods. In method 1 (linear stability analysis), they

    only consider communities that are close to equilibrium, while in methods 2 and 3 (permanence analysis,

    individual-based model) they investigate the behaviour of communities that are far from their equilibrium.

    Those two later methods yield the same results as the former (cooperation destabilizes communities). For

    reasons of space, we only consider method 1 for our analysis of the nature of explanation. [↑](#footnote-ref-10)
11. We will use “stable points” to refer to what mathematically are defined as “asymptotically stable points”. [↑](#footnote-ref-11)
12. In their model, Coyte et al. do not exactly determine at which point the system will become unstable. It is enough for their explanation to work to show the general tendency of the community to an increasing value of *Pm*. [↑](#footnote-ref-12)
13. Our argument in this section is inspired by a similar argument presented in Issad and Malaterre (2015: 284). [↑](#footnote-ref-13)