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The final version appeared in Christian A., Hommen D., Retzlaff N., Schurz G. (eds) *Philosophy of Science.* European Studies in Philosophy of Science, vol 9. Springer, Cham, pp. 95-115.]**

[**When Mechanisms Are Not Enough: The Origin of Eukaryotes and Scientific Explanation**](https://scholar.google.com/scholar_url?url=https://link.springer.com/chapter/10.1007/978-3-319-72577-2_6&hl=es&sa=T&oi=gsb&ct=res&cd=0&d=3496029492453128414&ei=97uUXIjgJ4qcmgGxiYSQBw&scisig=AAGBfm2vswP_j-ku-lCPfofBuo2GA6PNEg)**\***

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**Abstract**

The appeal to mechanisms in scientific explanation is commonplace in contemporary philosophy of science. In short, mechanists argue that an explanation of a phenomenon consists of citing the mechanism that brings the phenomenon about. In this paper, we present an argument that challenges the universality of mechanistic explanation: in explanations of the contemporary features of the eukaryotic cell, biologists appeal to its symbiogenetic origin and therefore the notion of symbiogenesis plays the main explanatory role. We defend the notion that symbiogenesis is non-mechanistic in nature and that any attempt to explain some of the contemporary features of the eukaryotic cell mechanistically turns out to be at least insufficient and sometimes fails to address the question that is asked. Finally, we suggest that symbiogenesis is better understood as a pragmatic scientific law and present an alternative non-mechanistic model of scientific explanation. In the model we present, the use of scientific laws is supposed to be a minimal requirement of all scientific explanations, since the purpose of a scientific explanation is to make phenomena expectable. Therefore, this model would help to understand biologists’ appeal to the notion of symbiosis and thus is shown to be better, for the case under examination, than the mechanistic alternative.

**Keywords**

Scientific explanation - Mechanistic explanation - Scientific laws - Eukaryotic cell - Symbiogenesis - Symbiosis

\*This work is fully collaborative; the authors are listed alphabetically.

**6.1 Introduction**

In recent years, mechanistic talk has become very popular among philosophers of science. Particularly, mechanistic talk has displaced the traditional approach to scientific explanation in terms of scientific laws (Nicholson [2012](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR38)). Mechanists claim that scientific explanation consists of looking for a *causal process* –in this sense, the mechanistic movement is just the other side of the coin of traditional causal models of explanation– such that, through connecting the different entities and activities that participate in the process, the phenomenon that we aim to explain simply emerges. This claim is in contrast with the claim made by defenders of nomological expectability models of scientific explanation who generally claim that “to explain a phenomenon is to make it expectable on the basis of non-accidental regularities” (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20), 1414). Mechanists usually put forward biology as their main counterexample against defenders of nomological models: when biologists claim to have explained a phenomenon, they do so on the basis of having found a mechanism that brings that phenomenon about (Machamer et al. [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30)). Biologists do not appeal to laws of nature, logical arguments, or any other kind of logic: they simply appeal to mechanisms. Thus, scientific explanation is, on this view, mechanistic explanation. In this paper, we contend this claim on its own terms, by presenting an example from biological practice. Specifically, we present the case of the origin of the eukaryotic cell and argue that the explanation of the salient features of this peculiar case is more suited to be understood in terms of a nomological expectability model of scientific explanation than in terms of mechanisms. For this purpose, we make explicit a kind of general regularity that biologists seem to be assuming when they provide explanations of the origin of the eukaryotic cell, and which forms the basis of the kind of proposals that they take as explanatory of certain facts that they consider particularly salient and in need of explanation (see Alleva et al. [2017](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR1), for a similar line of reasoning applied to the case of allosterism).

The paper is organised as follows: In Sect. [6.2](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec2), we introduce the symbiosis theory (ST, hereafter) of the origin of the eukaryotic cell, nowadays considered the canonical model for explaining the origin of eukaryotic cells, and we introduce a classification of the questions that ST provides answer to. In Sect. [6.3](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec3), we introduce the mechanistic account of scientific explanation complemented with Woodward’s account of causality and provide evidence that suggests that the appeal to mechanisms is not the most appropriate way to justify the explanatory character of ST of the origin of the eukaryotic cell and why this is so. In Sect. [6.4](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec4), we present a nomological expectability model of scientific explanation that we then use to provide an understanding of the explanatory character of the ST of the origin of eukaryotic cells by considering that ST appeals to scientific laws. Finally, in Sect. [6.5](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec5) we conclude by defending the superiority of the nomological approach over the mechanistic approach in providing an understanding of the explanatory practices of biologists in the context of the theories of the origin of the eukaryotic cell and we propose future lines of research.

**6.2 Symbiosis Theories of the Origin of Eukaryotic Cells**

The biological world is populated by different kinds of entities, ranging from cells, to all kinds of multicellular forms of life. Cells are normally taken to be the basic and most fundamental unit of life, of which all the other entities are made up (Archibald [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR2); Audesirk et al. [2008](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR4); Stearns and Hoekstra [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR45)). There are two main types of cells, classified according to the location of their DNA: prokaryotic cells (subdivided into the domains of Archaea and Bacteria) and eukaryotic cells. The main structural difference between prokaryotic cells and eukaryotic cells is that in the former, the genetic material is dispersed throughout the cytoplasm; whereas in the latter it is encapsulated within a membranoid-structure called the “nucleus”. Apart from this, there are many other structural differences between the two types of cells, concerning aspects such as their size (eukaryotic cells generally being bigger), the types of membranes and the presence or absence of organelles. This last different constitutes a salient feature of eukaryotic cells, since only they host organelles within their bodies. Organelles are structural subunits, analogous to organs in humans, which perform certain functions within the body of the cell they belong to. Two of the organelles within eukaryotic cells are mitochondria (present in all eukaryotic cells) and chloroplasts (present only in plant eukaryotic cells); these two organelles bear their own DNA. Mitochondria are the site of cell respiration. Photosynthesis, in contrast, takes places within chloroplasts. Eukaryotic and prokaryotic cells are quite distinct from each other, and there does not seem to be any record of an intermediate form between the two types of cells, which is why certain biologists have referred to the origin of the eukaryotic cells as “the greatest single evolutionary discontinuity to be found in the present-day living world” (Stainer et al. 1963, quoted in Sagan [1967](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR42), 226). This immediately triggers a serious question for biologists: how did the first eukaryotic cell appear, given that all organisms share a common ancestor, and therefore eukaryotes and prokaryotes must have originated from the same ancestor?

Answering this question about the origin of the eukaryotic cell consists, among other things, of explaining the origin of cellular organelles, as the most salient subunits that allow for the distinction between eukaryotes and prokaryotes, and particularly of answering questions about the origin of mitochondria and chloroplasts. Mitochondria and chloroplasts are, then, one of the hallmarks of “eukaryocity” and, as Martin and his collaborators have put it, “the invention of eukaryotic specific traits required more metabolic energy per gene than prokaryotes have at their disposal, and (…) mitochondria afforded eukaryotic cells an order of magnitude increase in the amount of energy per gene, which (finally) explains *why* the origin of eukaryotes corresponds to the origin of mitochondria” (Martin et al. [2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR33), 2; also Williams and Embley [2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR47), Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1)).[1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn1) Furthermore, it consists of justifying the lack of continuity in the fossil record between eukaryotes and prokaryotes, the biochemical differences between the two types of cells, the different capabilities of one type of cells with respect to the other, etc. Explaining the origin of eukaryotic cells consists, therefore, of providing satisfactory answers to a series of why-questions (facts) about the particular features of the two kinds of cells and especially answering certain questions about the particular nature of each type. The family of surprising facts that a theory of the origin of the eukaryotic cell has to provide explanations of can be roughly classified as:

* *Physiological and biochemical questions*. The model of the origin of the eukaryotic cells has to explain, for instance, why the membrane of mitochondria is biochemically quite distinct from the membrane of the eukaryotic cell, but biochemically closely related to the nature of the membranes of certain prokaryotes; it also has to explain why the genetic material of eukaryotes has a mosaic nature, i.e. it is composed of phylogenetically distinct classes of DNA.
* *Phylogenetic questions*. Mitochondria and chloroplasts are not phylogenetically close to eukaryotes, but they are phylogenetically close to certain prokaryotes. This fact is surprising, since mitochondria are organelle in the eukaryotic cell, so one important question to answer would be why their genetic material is distinct in nature from the one present in the eukaryotic nucleus.
* *Historical questions*. The most important question to be answered is why there is a gap in the fossil record between prokaryotes and eukaryotes, if we take evolution to be continuous with no sudden evolutionary jumps.

So, a theory of the origin of the eukaryotes (i.e. a theory that answers the question: “How did eukaryotic cells originate?”) should provide satisfactory answers to a list of why-questions of different natures, and evaluating its success at doing so is fundamental for the acceptance of one theory over another.

To answer the set of question outlined above, two families of theories have been proposed: on the one hand, *self-genetic or autogenous theories*, according to which the organelles within eukaryotes appeared as a consequence of invaginations within the original pre-eukaryotic cell (Raff and Mahler [1972](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR40); Uzzel and Spolsky [1974](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR46); all reviewed in Sapp [2010](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR43), 130–131; O’Malley [2010](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR39); Archibald [2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR3), R912); and on the other, *symbiosis or exogenous theories*, whose main claim is that eukaryotic cells originated through the symbiotic merger of two previously extant prokaryotic cells (Margulis [1970](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR31); Martin et al. [2012](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR32); Cavalier-Smith [2013](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR11); Dolan [2013](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR21)). In short, the proponents of ST argue that the eukaryotic cell evolved as a consequence of a phagocytic process in which prokaryotes “were swallowed but not digested” (Margulis [1970](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR31), 60). The difference between the two families of theories is radical, and so are the conclusions that one can derive from them. For instance, if one defends an autogenous theory, one has difficulties explaining the genetic affinities between mitochondrial DNA and the DNA of free-living prokaryotes, since one has to explain how this foreign DNA arrived in the mitochondria of present-day eukaryotes. However, if one defends a ST, this fact becomes easily explainable: the fact that in the origin of eukaryotes two different prokaryotic lineages merged makes it more likely that the primitive lineage associated with mitochondria still preserves part of its original DNA. The same logic can be applied to all kinds of questions that might be raised about the difference between prokaryotes and eukaryotes. So, the capacity to play a more effective role in scientific explanation proves to be a good feature for preferring one theory to another.

Nowadays, the ST family predominates among biologists, although the versions of it come in many different forms, with at least 20 different models that explain the origin of the eukaryotic cells appealing to symbiosis (Archibald[2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR3)). What matters for the purposes of this paper is the general structure of the arguments that appeal to endosymbiosis to explain the origin of eukaryotes and to explain the set of why-questions that we have selected as relevant, more than the peculiarities of the different models.

In general, ST appeals to the notion of *symbiogenesis* as the process by which the eukaryotic cell originally appeared.[2](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn2) This symbiogenetic process is supposed to have given rise to an endosymbiotic relationship between the different interacting organisms. The initial organisms involved in the origin of the first eukaryote are hypothesized to have been an archaeon (although there is no definite consensus on this question), with the capacity to phagocytize other microorganisms, and an alpha-proteobacteria, which would have given rise to mitochondria as we know it today (Spang et al. [2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR44)). The peculiar nature of *symbiogenesis* qualifies it as the reason that biologists offer to explain the surprising features that are observed in eukaryotic cells. For instance:

* Why is the membrane of mitochondria biochemically more similar to free-living proteobacteria than to its host, i.e. the eukaryotic cell itself?

Because it originated through symbiogenesis, which means that a free-living microorganism was engulfed but not digested and therefore it is very likely that the lineage this previously free-living microorganism gave rise to still preserves some of its original biochemical properties, such as the composition of the membrane.

* Why does the eukaryotic genome have a mosaic nature?

Because it originated by symbiogenesis. This entails two free-living organisms suddenly stopping their free-living mode to live together as a unit. As a consequence of a long-term relationship after symbiogenesis, it is very likely that there will be genetic exchange between the partners, thereby creating the mosaic structure of the eukaryotic genome.

* Why are mitochondria phylogenetically closer to free-living alpha-proteobacteria than to their host?

Because if mitochondria were once free-living microorganisms that, via a process of symbiogenesis, became organelles within the eukaryotic cell, it seems natural that their DNA would be phylogenetically closer to the DNA of the free-living forms from which they originated than to eukaryotic DNA.

* Why is there a gap in the fossil record between prokaryotes and eukaryotes?

Because if eukaryotic cells appeared through symbiogenesis, it is very unlikely that intermediate forms would be found in the fossil record. Symbiogenesis is a discontinuous process.

The appeal to symbiogenesis is therefore used as a general strategy to answer a different set of why-questions concerning particular features of the eukaryotic cell, providing answers that trace these features back to their evolutionary origin. In the following sections, we analyse whether this general strategy used by biologists is more in accordance with a mechanistic theory of explanation or with a nomological expectability approach, and we argue that what biologists actually do seems closer to the latter.

**6.3 Mechanistic Explanation**

Mechanistic explanation is the most influential approach to explanation in biology. The view was originally presented in direct opposition to the (previously) dominant nomological models of scientific explanation. Mechanists argue that in order to explain a biological phenomenon it is necessary to describe the mechanism that brings the phenomenon about (Glennan [1996](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR23), [2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR24); Bechtel [2011](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR6); Bechtel and Richardson [1993](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR8); Bechtel and Abrahamsen [2005](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR7); Machamer et al. [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30); Craver [2006](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR12), [2007](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR13); Darden and Craver [2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR16)). Describing a mechanism, they claim, is not the same as presenting a scientific law that underlies a phenomenon. In fact, they deny the possibility of explaining a phenomenon by subsuming it under laws. In other words, the explanatory character of a mechanism does not lie on its supposedly underlying regularities, but in the identification of causal relations: “while it is sometimes the case that description of the inner parts of the mechanism will entail a description of the mechanism’s outward behaviour, the explanation lies not in the logical relation between these descriptions but in the causal relations between the parts of the mechanism that produce the behaviour described” (Glennan [2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR24), S348; see also Machamer et al. [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30) for a similar argument).[3](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn3)

There are several ways of describing what a mechanism is. For instance, Machamer et al. ([2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30), 3, our emphasis) claim that a mechanism is a set of “*entities* and *activities organized* such that they are productive of regular changes from starting or set-up conditions to finish or termination conditions”; Glennan ([2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR24), S344, our emphasis) defines a mechanism by saying that it is a “complex system that produces the behavior by the *interaction* of a number of*parts*”; Bechtel ([2006](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR550), 26, our emphasis) says that it is “a structure performing a function in virtue of its component *parts*, component *operations*, and their *organizations*”.

It seems clear from the above definitions that all of them presuppose that a mechanism consists of a set of entities and activities (or parts and operations/interactions) plus their corresponding organization.[4](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn4) To identify a mechanism, therefore, one has to disentangle its parts (the entities), individuated by their properties, and the activities it is involved in, “the producers of change”. Allegedly, the properties of the entities plus their organization are responsible for the way in which the activities come about. In the words of Machamer et al.: “Mechanisms are identified and individuated by the activities and entities that constitute them, by their start and finish conditions and by their functional roles” ([2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30), 6). This dualist reading of mechanisms in terms of entities and activities generates a new framework that should, in principle, be fruitful when it comes to clarifying notions such as causation, lawhood, function and explanation. In particular, the notion of activity is supposed to play the role of causes, laws and functions. For instance, if a law is supposed to be a regularity of something that acts in the same way under the same conditions, philosophers of a mechanistic bent can provide a similar reading of a mechanism: “a mechanism is the series of activities of entities that bring about the finish or termination conditions in a regular way” (Machamer et al. [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30), 7). According to such authors, these regular mechanisms are not accidental and can give support to counterfactual reasoning. Therefore, there is no need to talk of laws in biology, for their role is already played by the identification of activities within mechanisms. In the same vein, Glennan refers to the interactions within a mechanism as “*invariant change-relating generalizations*” which can support counterfactual claims (Glennan [2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR24), S344).

Given this characterization of a mechanism, we can now say that to give a mechanistic explanation of a given phenomenon consists of giving a description of the mechanism that brings the phenomenon about, such that the explanans includes the set-up conditions (arbitrarily taken as the beginning of the mechanism) plus the intermediate entities and activities together with their organization.

Nonetheless, there still remains the problem of providing criteria for identifying the different parts that compose a mechanism and that should be taken as relevant for the purposes of explanation. One possible way out of this problem, adopted among others by Craver ([2007](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR13), 144), is to make use of Woodward’s manipulability criteria for identifying causes (Woodward [1997](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR48), [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR49), [2003](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR50)). Woodward’s strategy is to look for a “difference-making” clause in the explanans that, if we were to change it in various possible ways, would result in the final phenomenon being different. This strategy is mainly interventionist: if we want to identify the relevant factors for the production of a particular phenomenon, we must block certain parts allegedly involved in the causal path that terminates in the phenomenon to see whether this intervention has any consequence on the final output. Following this line of reasoning, one can say that “a part is causally relevant to the phenomenon produced by a causal mechanism if one can modify the production of this phenomenon by manipulating the behavior of the part, and one can modify the behavior of the part by manipulating the production of the phenomenon by the causal mechanism” (Nicholson [2012](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR38), 160).

Woodward is conscious that the interventions he requires to uncover the causes of phenomena might not always be available (think, for example, of historical phenomena). In order to resolve this difficulty, he argues that in those contexts where such manipulation is not feasible, the manipulability strategy takes the form of a counterfactual claim: “The notion of information that is relevant to manipulation thus needs to be understood modally or counterfactually: the information that is relevant to causally explaining an outcome involves the identification of factors and relationships such that if (perhaps contrary to fact) manipulation of these factors were possible, this would be a way of manipulating or altering the phenomenon in question” (Woodward [2003](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR50), 10). In other words, even in contexts where manipulation is not possible, it is “heuristically useful” to pursue or think of causes in the same way as we do when the relevant manipulation is available.

The task now is to try to apply the mechanistic schema plus Woodward’s account of causes to the explanation of the origin of the eukaryotic cell in order to test its usefulness. We will advance question by question, following the schema presented in Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1):

* Why is the membrane of mitochondria biochemically more similar to free-living proteobacteria than to its host, i.e. the eukaryotic cell itself?

This question is about similarity, i.e. it is about why certain biomarkers are similar in an organelle and an organism that is phylogenetically distant from the eukaryotic cell that bears the organelle, whereas those biomarkers are different between the eukaryotic cell and its organelle. The mechanist would want to look for the different entities and activities, and their organization that would allow the phenomenon under investigation (the nature of the membrane) to occur. If we were to do that, the entities would be the membranes and their biochemical nature; the activities would be those of membrane synthesis and membrane destruction; and the organization would depend on the way in which the aforementioned parts are spatiotemporally located in standard mitochondria. Let us suppose we follow this strategy. It is highly likely that we will discover many details about membrane synthesis, the biochemical products that are produced, the way in which they relate to each other, how they become arranged within mitochondria to give rise to a new membrane, etc. However, valuable as this information might be, it does not provide us with the answer we are looking for. This line of research would isolate the causes, allow interventions and provide a better understanding of membrane composition and membrane synthesis. But this is not what we were looking for in the first place. Our question concerned the similarities between mitochondria and a free-living microorganism, and the best answer to the question lies in symbiogenesis, as we mentioned in Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1), and nothing in the strategy that the mechanist might elaborate mentions symbiogenesis.

Nevertheless, the mechanicist might still try to argue that the explanation lies in symbiogenesis because symbiogenesis is, in this particular circumstance, a mechanism. The problem is that we are looking for a historical explanation and thus we can only apply Woodward’s counterfactual strategy. But this does not seem to do the trick either. First, the notion of symbiogenesis does not look like a mechanism at all: it is a very formal and general notion which does not make any reference to entities (it is supposed to cover a wide range of them, from the eukaryotic cell to most insect microbiota), activities (also very wide and diverse, from oxidation of glucose to synthesis of essential amino acids) or organization (which can be very variable). Second, because of the complexity of symbiogenesis, one cannot even imagine a set of factors whose alteration would block the phenomenon from appearing. If the factor we blocked was the symbiotic merger itself, then the result is not that we do not have a biochemical similarity between mitochondria and certain free-living bacteria: the result is that we do not even have either mitochondria or eukaryotic cells in the first place.

* Why does the eukaryotic genome have a mosaic nature?

The argument in this case is very similar to the previous one. The mechanist philosopher might try to isolate certain biochemical elements of the eukaryotic genome whose presence is responsible for the mosaicism. However, these different elements are merely biochemical and do not respond to the question asked in the first place; at most, the mechanist might provide us with a very good mechanistic explanation (in terms of parts, activities and arrangements) of why it is that the compounds of a eukaryotic genome admit mosaicism. But this does not explain why in fact all eukaryotes present this kind of genome and, more specifically, why they present the kind of additional genes they do with the particular functions they have. Again, we need a historical explanation to satisfy our queries and to ask “why” the genome in fact has a mosaic nature. As we said in Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1), symbiogenesis can provide a reply to this: the fact that distinct organisms came together to form the eukaryotic cell and have been living together for 1.5 billion years (with all the “arms races” that exist when distinct organisms live together) would explain this feature and would even explain the specific functions of the genes involved in such mosaicism (namely, these related to avoiding cheating on the part of mitochondria).

Once again, the mechanist philosopher might claim that, if, as biologists assume, the appeal to symbiogenesis provides the right answer, this is because symbiogenesis is a mechanism. But then the mechanist philosopher would have to acknowledge that the concept of a symbiogenetic process is so formal that no entities, activities or organization can be properly recognized and isolated, so as to identify a mechanism. Then the mechanist philosopher would have two options: either to relax the notion of mechanism, which would mean that the concept is made either empty or equivalent to the notion of regularity, or to accept that this fact is not explainable in mechanistic terms.

* Why are mitochondria phylogenetically closer to free-living alpha-proteobacteria than to their host?

Here, the argument against mechanists precisely mimics that presented for question one, merely changing all the details concerning membranes for details concerning phylogenetic relations; so to save space, we will not repeat it.

* Why is there a gap in the fossil record between prokaryotes and eukaryotes?

In this case, a defender of the mechanistic model of explanation might claim that we can always imagine a particular set-up with certain initial conditions and reason counterfactually (as Woodward proposes for historical explanations in general). Let us fix those set-up conditions. It would be a set-up where archaea and bacteria merge symbiotically. If we want to provide a reason why there is a gap, we have to isolate a factor such that, if we block it, the result would be different. Suppose for the sake of argument that symbiosis is such a factor and imagine that archaea evolve progressively until they give rise to eukaryotes.[5](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn5) Would this entail that there is no gap in the fossil record? Not necessarily. We have cases of gaps in the fossil record that are not due to symbiosis. For instance, nobody believes that the famous missing link between dinosaurs and birds is a consequence of symbiosis, despite this missing link creating a gap in the fossil record. Furthermore, there are examples of symbiotic mergers where no gap is present. Paracatenula is known to be the result of ancient endosymbiosis, but its existence does not entail that there is a gap in the fossil record between Paracatenula and other catenulid flatworms (Gruber-Vodicka et al. [2011](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR25)). Therefore, reasoning counterfactually in the strict manner Woodward suggests does not help to explain this particular phenomenon. It seems that what is required is the assumption of a very particular pattern that follows a unique event (namely, a symbiotic merger). This pattern, due to the complexity in determining its parts, activities and organization, cannot be interpreted mechanistically. In addition, it is difficult to see what a mechanistic reading in terms of parts, activities and organization can offer to explain the actual gap.

From this reasoning, three consequences follow. First, taking for granted that the appeal to symbiogenesis explains many of the issues about the origin of the eukaryotic cell, symbiogenesis is not, and it is very far from being, a mechanism.[6](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn6) Second, symbiogenesis seems to be more a general pattern which biologists appeal to in order to explain the features they find in the eukaryotic cell. Finally, even if the reference to mechanisms might complement explanations of some of the questions asked, and it might add some precision, the real explanatory role, as biologists accept, is played by the appeal to symbiogenesis.[7](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn7) Therefore, if symbiogenesis is not a mechanism but a general pattern, then it seems that the appeal to regularities might be explanatory after all.[8](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn8) In the next section, we further explore the possibility of considering symbiogenesis as a regularity.

**6.4 Symbiogenesis as a Nomological-Expectable Explanation of the Origin of the Eukaryotic Cell**

Biologists’ appeal to the notion of symbiogenesis, as we have argued, has the form of a general pattern: the biologists look for a general principle, which may be quite vague (in the sense that it might be applicable to a large number of entities, irrespective of their particular biological properties), that allows them to say not only how the first eukaryotic cell came about, but also why it has the properties it has (which are the answers to the four why-questions we have presented, plus other relevant questions that might be asked). It is convenient to specify at this point why we consider symbiogenesis to work as a regularity that might be used to account for certain facts (Archibald [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR2); Douglas[2010](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR22)).

First of all, symbiogenesis mere implies that the process by which an actual living organism has come about is a consequence of a symbiotic merger. Furthermore, in the case of the eukaryotic cell, it is always specified that this symbiogenesis gave rise to a case of endosymbiosis, whereby one organism lives inside the other. However, nothing about the particular nature of the organisms that interact endosymbiotically is specified, nor does it require to be specified in a general definition of symbiogenesis. Symbiogenesis just says something about how the mode of life of the organisms came about. Second, and related to the vagueness of the term, symbiogenesis is supposed to cover all the different cases of structures (and species) that emerge as a consequence of symbiosis between two different organisms. This entails that the entities that can interact symbiotically and give rise to a case of symbiogenesis are very different with respect to each other: bacteria, fungi, arthropods, mammals, etc.; they can all bear endosymbionts and/or enter endosymbiotic relationships with others. Third, by its very nature and its connection with the appearance of new biological structures, when it occurs through the acquisition of endosymbionts, symbiogenesis tends to trigger certain changes in the organisms involved: genomic decay, genetic assimilation, free exchange of genes between partners, vertical transmission, the appearance of particular bodily structures to bear the symbionts, etc. The evolution of these particular traits will differ depending on the particular relationship between the organisms and their necessities, and is normally what causes endosymbiotic states to be irreversible. Fourth and finally, symbiogenesis normally leaves some traces of the previously independent life of the partners. However, these traces vary quite a lot if we consider them on a case-by-case basis. Sometimes the traces will be biochemical pathways; others, molecular properties or chromosome structure, etc.

We believe that these four characteristics of symbiogenesis justify consideration of the phenomenon as a general pattern that biologists use in order to guide their research and to explain certain features that would not be explained otherwise. Indeed, the key aspect of symbiogenesis, in relation to accounting for the features of the eukaryotic cell as mentioned above, is that it makes these “expectable on the basis of [a] non-accidental regularit[y]” (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20), 1414). Nonetheless, this pattern, though general, is not empirically empty: it says something about the past and the future of the organisms which interact, and this can be studied further (and proved to be true or false). We believe that symbiogenesis, understood as we have specified above, is a kind of scientific law in Mitchell’s sense ([1997](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR34), [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR35), [2003](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR36)). In Mitchell’s account, laws are understood pragmatically, according to the role they play in scientific practice. In other words, laws are not interpreted in terms of necessary and sufficient conditions, as traditional normative approaches suppose, but in terms of what they allow scientists to do. In this vein, Mitchell argues that a scientific statement must be understood as a scientific law if it allows good predictions to be made, good explanations to be provided and feasible interventions to be designed. This flexible conception of scientific laws allows her to provide a new multidimensional framework to represent a whole set of scientific generalizations (Mitchell [2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR35), 259). Furthermore, scientific laws in this sense provide a certain non-zero degree of nomic necessity,[9](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn9) which is established in terms of the stability of the conditions upon which the regularity is contingently dependent.[10](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn10) Therefore, the degree of nomic necessity of regularities in physics is higher than that of regularities in biology, because the stability of the conditions upon which a regularity is contingent in physics and in biology are significantly different. However, both regularities in physic and in biology involve a certain degree of nomic necessity; which is what matters here and is relevant for considering these generalizations as legitimate scientific laws.

In the context of the symbiosis models of the origin of eukaryotes, the appeal to the concept of symbiogenesis seems to play the role of a scientific law in this sense. First, it is often supposed that endosymbiotic association between two different organisms will give rise to a tendency for a series of adaptations to evolve that will increase the tightness of the fit between the partners. These adaptations will tend to evolve due to the possible presence of “cheaters”, i.e. organisms that benefit from the association without providing any benefit to its partner. This is a consequence of the fact that endosymbiotic associations that are capable of evolving adaptations that prevent the possible presence of cheaters outrun those that are not. Second, it is also assumed that the partners in an endosymbiotic association will still preserve some traces of their previous free-living state, as a consequence of the special features of the symbiogenetic process. Indeed, symbiogenesis sometimes entails (and it definitely does so in the eukaryote case) a transition in biological individuality. But, as is well known, the framework of transitions in individuality assumes the existence of individuals whose evolutionary fates align and form a higher-level entity. It is precisely the existence of independent individuals whose individualities become combined into a higher-level unit what makes it reasonable to expect that certain features of their previously independent existence will be preserved. In addition, the features that are preserved could be studied in a lab, making certain predictions possible. It is in at least these senses that we believe symbiogenesis plays the role of a nomic pattern (a pragmatic law): it allows for certain predictions, makes a set of phenomena that can be empirically tested expectable and supports counterfactuals. This nomic character seems to be the aspect of the notion of symbiogenesis that biologists have in mind when they use it for explanatory purposes.

Of course, defenders of mechanistic explanation might still question the alternative that we offer to mechanistic models of scientific explanation. As is well-known, the models that have traditionally appealed to scientific laws as the main explanatory “weapon” are conceptually flawed –they have to face numerous problems: flag-pole cases, contraception pills and male pregnancy, syphilis-paresis cases, vitamin C and flu recovery, etc.– and are not very popular among contemporary philosophers of science (Woodward [2017](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR51)). Maybe, after all, we have to admit that, although not perfect, as our case illustrates, mechanistic explanation is the best theory of scientific explanation that we have for the moment. Nonetheless, Díez very recently proposed a new neo-Hempelian account that solves most of the conceptual problems that have been raised against nomological expectability models and –allegedly– would include mechanistic explanations as a specific subcase satisfying additional conditions (Díez [2002](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR18), [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20)). As this is the only nomological alternative we know of that has these features, we now proceed to evaluate whether Díez’s model can accommodate the case of the origin of the eukaryotic cell.[11](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn11)

Díez’s model takes as a point of departure Hempel’s thesis that “to explain a phenomenon is to *make it expectable* on the basis of non-accidental regularities” (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20), 1414). This expectability, however, is not as strict as it has traditionally been in deductive/inductive nomological models (one of the possible forms that nomological expectability models can take), where the cases in which the explanation is based on a low-probability relationship between the explanandum and the explanans were excluded. The reason for this exclusion was that explanations were taken as logical inferences; thus, in the case of inductive inferences, they demanded high probability (Hempel and Oppenheim[1948](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR27); Hempel [1965](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR26)). In contrast, Díez substitutes the notion of logical inference for the less demanding notion of “embedding”: according to Díez, to explain a phenomenon is to embed it “into some branch of a net of theoretical constraints” (the explanans) such that they make the phenomenon expectable (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20): 1419). The idea of embedding is the structuralist counterpart to the positivist notion of *implication* and it presupposes a distinction in scientific models/structures between *data models* and *theoretical models* (Balzer et al. [2012](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR5)). A data model is a structure that describes the phenomenon to be explained; whereas theoretical models are the structures defined by their satisfying a theory’s laws. A data model is embeddable in a theoretical model when the former “fits” the latter, i.e. the relevant values of the phenomenon square with those of the theoretical model. Embedding is thus a relation between models, not a relation between sentences, which allows for a weakening of the positivist demand for logical inference (for instance, making room for embedding in increasing yet law probability cases) but still preserves the core intuition behind Hempelian *expectability*. To put it in Díez’s words “[e]xplanations are (at least) certain kinds of predictions” (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20), 1420).

We will now provide an example of embedding. Suppose we want to explain the movement of the Moon using Newtonian mechanics. Our data model would include the Earth, the Moon, and the space and time functions that describe the kinematic trajectory of the Moon around the Earth, [DME,M = <{Earth, Moon}, space, time>]. The theoretical model would include, apart from the aforementioned components, the functions of *mass* and *force*, [TME,M = <{Earth, Moon}, space, time, mass, force>] defined by their satisfying Newtonian laws. The idea of the embedding of the data model within the theoretical model would be the following: by using the “machinery” of classical mechanics (laws of motion) plus the relative positions of the Moon and the Earth at a particular time, the theoretical model includes the relevant positions at other times; if such values fit the measured values of the data model, the former successfully embeds the latter, otherwise the embedding fails (and the theory has a Kuhnian anomaly). In this sense, model-theoretical embedding expresses the core intuition of nomological expectability.

However, as Díez explains and the case of the Moon’s trajectory exemplifies, nomological embedding, though necessary, is not sufficient for explanation, since we may still fail to have explanatory embedding in two kinds of cases. First, one may have embedding by merely descriptive/phenomenological theories that systematize data with laws describing general phenomena without explaining them (e.g. Galilean kinematics or Kepler’s laws). Second, in theories with what Kuhn calls “general schematic principles” such as Newton’s Second Law (Kuhn [1970](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR28)), one can always construct *ad hoc* trivial “successful” embedding that cannot count as explanatory. To exclude these cases, Díez adds two further conditions: the embedding has to be *ampliative* and *specialized*. Its *ampliative* character is based on the notion of T-theoreticity (Balzer et al. [2012](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR5); related to Hempel’s distinction between “characteristic” and “antecedently understood”, and Lewis’s distinction between old and new vocabulary). T-theoretical concepts are those introduced by a theory such that, in order to determine their extension, one has to use/accept some T-law (e.g. *mass* and *force* in classical mechanics); whereas T-non-theoretical concepts are those which are already available and that can be determined (at least on some occasions) without the help of T-laws (e.g. *space* and *time* in classical mechanics). Explanatory embedding is ampliative, as in the case of classical mechanics: classical mechanics explains why the Moon is in location *X* at time *t* through embedding the phenomenon and introducing new T-theoretical concepts/entities (masses and forces) that do not appear in the data model DME,M. Thus, for embedding to be explanatory, it must make use of laws that (as in classical mechanics and not in Galilean kinematics or Keplerian astronomy) appeal to new concepts/entities. *Specialization*, on the other hand, requires that we introduce non-*ad hoc* “special laws” in order to account for the phenomena.[12](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn12) As Díez points out, we always require that our explanations include something more than merely schematic, very general principles such as *∑f = ma*. In the case of the Moon–Earth system, for example, we need to introduce the law of universal gravitation, *f = G\*mm’/r**2*, if we aim to explain the positions of the Moon over time.

In short, we might now say that a theory explains a phenomenon if: (1) we can embed the phenomenon in the theory, in such a way that the theory makes the phenomenon expectable; (2) the theory includes and makes use of at least one T-theoretical term; and (3) the theory incorporates and makes use of at least one special law in order to account for the phenomenon (Díez [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR20), 1425). We will show that the appeal to symbiogenesis that biological theory makes to explain the origin of eukaryotes and the different phenomena laid out in Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1), which does not fit the mechanistic account, is nevertheless perfectly legitimate and can be explicated by applying Díez’s re-elaborated model of explanation as nomological expectability.

First, the appeal to symbiogenesis provides a theoretical model that allows the embedding of the phenomena that constitute our data model. In the case of the origin of the eukaryotic cell, the data model would include objects such as membranes –of both cells and mitochondria– or genomes –again, both cell and mitochondrial genomes– and their respective biochemical properties –those of the lipid components of the mitochondrial membrane versus those of the lipid components of the cell membrane; circular, single-strand DNA versus linear, complex DNA, etc.– (DMG,M = <{genome, membrane}, biochemical properties of both>). The theoretical model would include these objects plus entities/functions that correspond to the notions of *fitness* and *symbiogenesis*, which are purely theoretical and associated with particular features of symbiosis relationships and the theory of natural selection (TMG,M = <{genome, membrane}, biochemical properties of both, fitness, symbiogenesis>).[13](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn13) The embedding is possible in this case because DMC,M happens to actually be a submodel that squares with TMG,M, and TMG,M makes the phenomena we aim to explain expectable (as reviewed in Sect. [6.1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec1) in response to questions 1–4).

Furthermore, TMG,M includes a couple of T-theoretical entities/functions, fitness and symbiogenesis, that play an *ampliative* role. Biologists do not explain the features of the mitochondrial genome by appealing to features of free-living bacteria. They explain them by appealing to the idea of symbiogenesis (and its specific endosymbiotic form): certain formerly free-living bacteria (that we can indicate through phylogenetic analysis) were at some point endosymbiotically acquired by an archaeon and, *symbiogenetically*, gave rise to the organelles that nowadays we call mitochondria. The preservation of the genetic and biochemical features of the mitochondrial membrane is explained by appealing to its symbiogenetic origin plus the fact that they confer fitness advantages. In this sense, it seems clear that the embedding is ampliative in the sense Díez’s account requires.

Finally, the explanation in terms of symbiogenesis includes an element of specialization in relation to ST (or the concept of symbiosis): an appeal to a special law which plays a non-trivial role in the explanation of the particular features of mitochondria. Symbiogenesis is a particular form of integration that two symbiotically associated organisms could enact, if the circumstances were favourable. It is well established that there are different types of symbiotic relationship (mutualism, commensalism and parasitism); some might be long-term evolutionarily relationships that are not conducive to integration, whereas others are. If they are conducive to integration and they have the desired fitness effects (i.e. they do not lead to the extinction of the integrated lineages), then they would trigger certain changes in the lineages that evolve symbiogenetically (mosaicism, genomic decay, loss of independent modes of life, etc.), giving rise to the appearance of new biological structures (they would fall down an evolutionary “rabbit hole”, as some biologists describe it, e.g. Moran and Sloan [2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR37)). In contrast, if the symbiosis relationship does not lead to integration, even if it is a long-term relationship, it would lead to a different kind of changes that would affect to both organisms independently, such as certain phenotype changes, changes in behaviour, etc. In this sense, symbiogenesis plays the role of a special law concerning a more general principle of the expected outcomes of long-term symbiotic associations.

We believe this appeal to a special law is the crucial step in ST, it is what provides the main explanatory power and as we argued, it does not have the form of a mechanism. The special symbiosis law certainly is such in Mitchell’s pragmatic sense: it provides a certain degree of nomic necessity, therefore providing biologists with a guide to what they might find. For instance, appealing to a symbiogenetic origin makes it expectable that organelles, i.e. mitochondria, still preserve a certain degree of biological individuality that might be manifested, for example, by the possibility of in vivo replication. It is important to bear in mind that this would not be expected if the origin was self-genetic: in this latter scenario, we would never expect mitochondria to have any degree of biological individuality. Furthermore, if the origin of mitochondria is symbiotic, we will not expect to find intermediate forms in the fossil record, since symbiosis gives rise to saltational evolutionary events, which would not be the case if the origin was self-genetic. This same line of reasoning might be applied to all the features that ST makes nomically expectable and, in this sense, we have something similar to a pragmatic law that provides the research field with some order.

We should still note something about the special character of the law. As we said before, the condition is introduced in order to avoid counting as explanatory cases in which we merely apply general principles to *trivially* justify why certain phenomena occur (using *ad hoc* mathematical functions in ∑*f = ma* to explain intentional movement, for instance). One might argue that the appeal to symbiogenesis is still trivial in this last sense: it is just one general principle we could use to justify every feature we find in an organism. Nonetheless, this is not the case: the appeal to symbiogenesis rules out certain possibilities and it makes a difference (as does the appeal to *f = G\*mm’/r**2*, in the case of planetary movement). It specifies the manner in which evolutionary innovation can arise, and this is in contrast to other possibilities, such as mutation, recombination, methylation, changes in the developmental matrix, or even other types of long-term non-integrative symbiotic relationships. It specifies a very particular pattern followed by the organisms that experience this mode of generation of evolutionary novelties and precludes triviality by ruling out the appearance of certain features that other evolutionary pathways would make expectable.

In conclusion, we have provided a (partially weakened, partially strengthened) nomological expectability framework as a possible alternative to a mechanistic framework of scientific explanation that explicates why biologists consider ST a legitimate explanatory theory of the origin of the eukaryotic cell by appealing to the notion of (pragmatic) scientific laws. In this sense, we have provided reasons to justify why an account of scientific explanation in terms of laws (in the restricted sense we have given) might be appealing to gain an understanding of the explanatory practices of biologists in certain contexts; an understanding that –we have claimed (Sect. [6.3](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec3))– mechanist philosophers cannot provide.

**6.5 Concluding Remarks**

In this paper we have presented the symbiosis model of the origin of the eukaryotic cell together with a set of questions (phylogenetic, biochemical, etc.) that any theory of the origin of the eukaryotic cell must provide answers to. We argue that the notion of symbiogenesis, understood as the process by which a new biological structure (organ, metabolic pathway, etc.) originates as a consequence of a long-term symbiotic relationship, plays the entire explanatory role when biologists aim to provide an answer to the different questions we mention (Sect. [6.2](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec2)). This said, we defend the idea that the mechanistic account of scientific explanation is not well-suited to understanding why the notion of symbiogenesis plays the entire explanatory role in these cases. First, we argue that every attempt to offer a mechanistic explanation to the questions previously mentioned turns out to be unsatisfactory, since they move to a level of detail which turns out to be unnecessary for the matters discussed; moreover, many of the causes that should be mentioned in a mechanistic account seem orthogonal to the type of phenomena that demands an explanation. Second, we show that the notion of symbiogenesis is far from being a mechanism as they are conventionally understood in the literature (in terms of parts, activities and organization): symbiogenesis is a regularity or general pattern that cannot be suitably captured in mechanistic terms (Sect. [6.3](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec3)). Finally, we present Díez’s nomological expectability model of scientific explanation as an alternative to mechanistic models of explanation and defend the notion that Díez’s model helps in understanding the explanatory character of symbiogenesis, despite its not being a mechanism but a general pattern (Sect. [6.4](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Sec4)). If our argument is sound, it shows how and why the appeal to general patterns –that might well be considered scientific laws in Mitchell’s sense, as we argue– might be explanatory in some contexts, thus challenging the universality of mechanistic explanations. It remains to be explored, however, whether the nomological expectability approach to scientific explanation we have defended here could also be applied to other biological contexts, either as a complement to (e.g. Alleva et al. [2017](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR1)) or as a substitute for mechanistic accounts.

**Footnotes**

[1](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn1_source).

This point is however controversial, as some people have also defended the idea that other processes such as a phagocytosis might also be considered as the starting point of eukaryocity (e.g. Cavalier-Smith [1989](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR10)). However, that would not remove the need to explain the origin of mitochondria and chloroplasts in a satisfactory manner, which would lead to the same kind of questions that we mention later. For the purposes of this paper and for simplicity, we will follow Martin’s proposal that equates the origin of eukaryotes with the origin of mitochondria. Thanks to Thomas Bonnin for pointing this out to us.

[2](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn2_source).

Symbiogenesis is the process of generation of a new biological structure (organ, metabolic pathway, etc.) as a consequence of a long-term symbiotic association. In the case of the eukaryotic cell, symbiogenesis refers to the origin of a complete new biological domain as a consequence of symbiosis. Symbiotic organisms can interact in two different ways: endosymbiotically, if one organism lives within the cell(s) of the other, and ectosymbiotically, when one organism lives on the surface of the cell(s) of the other, but not within them (Archibald [2014](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR2)). Symbiogenesis is thus a process, whereas endosymbiosis is a state. This distinction has to be kept in mind for the rest of the paper. Thanks to an anonymous reviewer for encouraging us to clarify this point.

[3](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn3_source).

Leuridan ([2010](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR29)) argues that for every mechanism we can find an underlying regularity. His conclusion is that the explanatory character of mechanisms lies precisely in these hidden regularities, which actually is conceding too much to the nomological expectability models mechanists were criticizing in the first place.

[4](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn4_source).

In fact, the notions of entities and activities come from a modification of the previous description of a mechanism in terms of parts and operations/interactions. Bechtel and Glennan still define mechanisms by appealing to the notions of parts and operations/interactions. The motives for their choice can be found in Bechtel and Abrahamsen ([2005](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR7), fn. 5). Machamer et al. ([2000](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR30), §3) introduced the new notions of entities and activities, mainly for ontological reasons. We take this not to be a substantive distinction for the purposes of this paper.

[5](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn5_source).

Earlier in this section we argued, as part of the response to question 1, that it was quite hard to conceive of symbiosis as a factor. We still believe this, for the reasons discussed there, but we are going to assume here that it might serve as one, just for the sake of the argument.

[6](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn6_source).

In fact it would not even be a mechanism in the sense of “expanded mechanism” as defended by Roe and Baumgaertner ([2016](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR41)), since the problem here is not related to incorporating “pieces” of the environment, as they suggest: the problem is related to the fact that what plays the explanatory role is a regularity.

[7](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn7_source).

One might still wonder about the exact relationship between mechanisms and regularities in certain explanatory contexts. It is not the aim of this paper to elucidate the nature of that relationship. Nonetheless, some mechanist philosophers have already recognized the use of non-accidental regularities in mechanistic explanations (e.g. Craver and Kaiser [2013](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR15); Glennan [1996](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR23)) and we believe that, in most cases of mechanistic explanation, what does the real explanatory work is the presence of background non-accidental regularities. We plan to develop this line of thought further in a future paper.

[8](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn8_source).

Of course, the defender of mechanistic explanation might still argue that the appeal to symbiogenesis is not, after all, explanatory. A similar strategy has been pursued by Craver ([2008](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR14)) concerning the explanatory character of the Hodgin-Huxley model of action potential in neurons. However, we believe that pursuing that strategy would violate some basic commitments common to biologists concerning explanation.

[9](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn9_source).

See also Brandon ([1997](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR9)) for more about biological generalizations having a limited range of nomic necessity and explanatory power.

[10](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn10_source).

Mitchel also includes other parameters: ontological ones (strength, plus the aforementioned stability) and representational ones (degree of abstraction, simplicity and cognitive manageability), which we take not to be relevant for our purposes in this paper. See Mitchell ([2003](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR36), chapter 5) for more details.

[11](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn11_source).

What follows is mainly based on the analysis we already presented in Deulofeu and Suárez ([2015](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#CR17)).

[12](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn12_source).

As we said before, the notion of law that we use is Mitchell’s idea of pragmatic law.

[13](http://philsci-archive.pitt.edu/15839/1/978-3-319-72577-2_6#Fn13_source).

This reconstruction is merely informal and, due to particular complexities of biological theory, it cannot be made as precise as it could be in the case of classical mechanics. In any case, it has all the elements that are supposed to provide a general idea concerning embedding.

**Acknowledgments**

Different versions of this paper were presented at the VIII Meeting of the Spanish Society for Logic, Methodology and Philosophy of Science (University of Barcelona, 2015) and the III Conference of the German Society for the Philosophy of Science (University of Düsseldorf, 2016). We would like to thank all the participants for their helpful comments and suggestions. We would also like to thank Thomas Bonnin, Mark Canciani, José Díez, John Dupré, Çağlar Karaca, Adrian Stencel and an anonymous referee, who read previous versions of this paper and made helpful comments and suggestions. Finally, the Spanish Ministry of Economy and Competitiveness (FFI2016-767999-P) and the Fundación Bancaria la Caixa are formally acknowledged for their economic support.

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