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Levels, Nests, and Branches: Compositional Organization and Downward Causation in Biology

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Overview

The idea of compositional levels of organization is deeply rooted into contemporary biology and its philosophy. I argue that when we take a closer look at the structure of biological systems, it turns out that hierarchical organization in nature is more complex and messy than most accounts of levels assume. More specifically, biological components tend to be very heterogeneous, which results in branching and tangled hierarchies instead of neat levels. Consequently, the traditional idea of levels should be treated as a heuristic abstraction that is only useful in some specific biological contexts. I demonstrate this approach by applying it to the debate on downward causation, where assumptions about levels play an important role and should be made explicit.

Scientists and others tend to be quite fond of neat, clear-cut patterns. Nature is not. Nature is inherently messy.

—Denis Noble (2006, p. 52)

4.1 Introduction: Levels and Compositional Hierarchies

The idea that nature is structured into nested compositional levels is ubiquitous in biology and its philosophy. As early as 1929, Joseph Woodger wrote, “The organism is analysable into organ-systems, organs, tissues, cells and cell-parts. There is a hierarchy of composing parts or relata in a hierarchy of organizing relations. These relations and relata can only be studied at their own levels ... and not simply in terms of the lower levels” (Woodger, 1929, p. 293). In a classic article, Lewontin (1970) argued that the principle of natural selection can apply “at all levels of biological organization” and gave as examples of such levels molecules, cells, gametes, individuals, and populations. In his seminal work on levels of organization, Wimsatt (1976, 1994/2007) argued that compositional levels are a deep and fundamental feature of the ontological architecture of the world. And the list goes on (see, e.g., Bunge, 1977; Eldredge, 1996; Novikoff, 1945; Simon, 1962/1996; Tëmkin & Eldredge, 2015; Umerez, 2016; Zylstra, 1992).

The core idea of nested compositional hierarchies is clear: most things studied in biology consist of parts or components, and those parts themselves also consist of parts. Thus, there

are wholes (e.g., cells) that consists of parts (e.g., cell parts), and these wholes and their parts are “nested” within bigger wholes (e.g., tissues). This kind of organization is particularly salient in multicellular organisms. An organism such as a human is composed of tissues, tissues are composed of cells, cells are composed of cell parts, cell parts are composed of molecules, and so on.

The approaches to these compositional levels fall into two broad categories: global and local (see, e.g., Craver, 2007, 2015; Love, 2012). In global approaches to levels (e.g., Oppenheim & Putnam, 1958), the idea is that levels span the whole of nature, or at least a substantial part of it (i.e., not just one organism or mechanism). Thus, it is thought that there is a global level of molecules, level of cells, individuals, populations, and so on, and that there is some clear and consistent sense in which, for example, cells in fruit flies and cells in humans are “at the same level.” Depictions of such global levels are very common in biology, for example, in textbooks (Eronen & Brooks, 2018).

In local approaches, no general framework of this kind is assumed. Instead, the focus is simply on compositional hierarchies as they appear in a specific (type of) system or mechanism (Bechtel, 2008; Craver, 2007, 2015; Love, 2012; Winther, 2006). For example, there is a certain compositional structure, with certain hierarchical levels, in the spatial memory mechanism of mice and a different hierarchy with different levels in the human respiratory system (Craver, 2007). With such a local approach, there is no need to assume that levels span the whole of nature or even a whole organism: they can be determined on a case-by-case basis and can be extremely local and context specific. The most famous and influential local approach is “levels of mechanism,” proposed by Bill Bechtel and Carl Craver, where the idea is that a mechanism as a whole forms the highest level, its components form a lower level, the subcomponents of those components form the next lower level, and so on (Bechtel, 2008; Craver, 2007, 2015; Craver & Bechtel, 2007).

In this chapter, I will point out certain features of biological compositional hierarchies that make them messier and more tangled than is usually assumed by philosophers and scientists. Although I focus on local levels, my arguments apply *a fortiori* to the more general global levels. However, they are not aimed at levels that are defined by other criteria than composition (e.g., scale-based levels or levels of processing). After critically discussing compositional levels, I provide a more positive viewpoint by outlining various ways in which the notion of levels can still be useful for biological research. Finally, I apply these insights to clarify the debate on downward causation.

4.2 Branching Hierarchies

Biological compositional hierarchies are typically downward-branching. To see what I mean by this, it is useful to contrast biological hierarchies with physical ones. In many physical systems, there is a neat hierarchical organization (see, e.g., Simon, 1962/1996): for example, a volume of gas consists of gas molecules, and when we decompose those gas molecules, we find similar things, even if the gas molecules are different (e.g., nitrogen and oxygen molecules): the gas molecules are composed of atoms, the atoms are composed of electrons and protons, and so on. Thus, even after several rounds of decomposition, we find similar things in the different branches.

In systems like this, we can delineate very neat compositional levels, where each level is occupied by similar kinds of things, and these similar kinds of things compose higher-level things, which are also similar to one another. This idea of levels is very intuitive and widespread and is particularly evident in “layer-cake” conceptions of levels (stemming from Oppenheim & Putnam, 1958; see Brooks, 2017, for more).

In biology, there are also some cases that come close to this picture, most prominently the levels of (multicellular) organisms and cells. Multicellular organisms are arguably similar in important ways: for example, they are capable of reproduction and have a similar basic metabolism. Furthermore, they are composed of cells, which are also similar to each other in important ways (e.g., all eukaryotic cells have a membrane). Thus, we seem to have a level of similar things (cells) that make up higher-level things (organisms) that are also similar to one another, leading to two neat levels of organization.

However, when we take a closer look at this picture, cracks begin to appear (see also Potochnik, this volume). First of all, cells make up organisms only in a very indirect and abstract sense. Rather, it would be more accurate to say that cells make up tissues, which make up organs, which make up organ systems, which (in combination with many other things) make up multicellular organisms (see also the quote from Woodger, 1929, at the beginning of this article). If we adopt any notion of composition that does justice to the complex and nested nature of biological organization, it is a vast oversimplification to say that organisms are composed of cells (see also Brooks, this volume).

To make this more concrete, I will assume here (and in the rest of this chapter) that when we analyze the compositional structure of a biological system, we should look for components that are biologically relevant “working parts”: that is, parts that play a non-redundant role in the functioning of the higher-level whole (Bechtel, 2008; Craver, 2007). In addition, what are relevant for analyzing levels are the *direct* components or working parts—for example, the cell organs are direct components of cells, but the components of cell organs are components of the cell only in an indirect way, as there is another compositional “step” in between.¹ Let us then look at the components of, say, a mouse. This will include things like the reproductive system, the cardiovascular system, and the nervous system. Although these are all organ systems in some broad sense, they are very different kinds of systems. And things get even messier when we look at the next lower level, formed by the components of these systems. One component of the mouse nervous system is the brain. One component of the mouse reproductive systems is an ovary. Ovaries and brains are very different kinds of things regarding their scale, function, and organization. Does it make sense to claim that they are at the same level, just like atoms in the gas example, because they are both two rounds of decomposition away from the mouse as a whole? Probably not.

What this example illustrates is that when we take a biological organism or a system and look at what it is composed of, we are likely to find very heterogeneous components, very much in contrast to the gas example. This becomes even more evident when we consider the stereotypical organizational unit in biology: the cell. In some sense, it is true that the components of cells are molecules, just like it is true in some sense that the components of organisms are cells, but again, this is an unhelpful oversimplification. If we look at the biologically relevant components or “working parts” of a (eukaryotic) cell, we find things such as the cell membrane, the nucleus, mitochondria, liposomes, ribosomes,

and so on.² These are very different kinds of things, with different properties, and on different scales. For example, a mitochondrion is an organelle that has its own DNA and is a relatively large structure, whereas liposomes are much smaller and have a simple container-like structure. When we then look at the subcomponents of these two components, this heterogeneity is amplified. The components of the mitochondrion include things like the inner membrane, the outer membrane, and the matrix that contains the mitochondrial DNA. The components of liposomes do not include any of these things but rather things such as lipid molecules and water molecules. It is evident that water molecules and the mitochondrial matrix are very different kinds of things and at very different scales. Thus, in contrast to the gas example, after just a few rounds of decomposition, we find wildly heterogeneous things in the different “branches” in the compositional structure of the cell.

This kind of downward-branching is a very common feature in nature. Although I have focused on downward-branching, analogous observations can also be made regarding upward-branching: for example, the same kind of protein can be a component in many different kinds of wholes (see also Wimsatt, 1994/2007, especially figure 10.2). Branching is also not an exclusive feature of the biological world but can in principle also occur in nonliving systems, such as complex artifacts. All it requires is sufficiently heterogeneous components, as then this heterogeneity will be amplified when it comes to subcomponents. Branching also occurs in the standard example of mechanistic levels, namely, a mouse navigating a water maze (Craver, 2007). One of the levels in this mechanism is the “cellular-electrophysiological level,” where, according to Craver (2007), we find most importantly the synaptic long-term potentiation (LTP) mechanism. The components of this mechanism include things such as NMDA receptors, synaptic vesicles, cell membranes, magnesium and sodium ions, and so on. Just as in the case of the cell, it is clear that if we decompose these heterogeneous components, we do not find similar things that would form a coherent level but rather many different branches with wildly different components.

Although branching is often briefly mentioned in the literature on levels (e.g., Bechtel, 2008; Craver, 2007; Kim, 2002; Wimsatt, 1976, 1994/2007), its consequences have not been sufficiently acknowledged. Supporters of the mechanistic approach to levels have correctly emphasized the local and case-specific nature of levels, but as the examples above show, even in local and specific contexts, compositional organization can get extremely messy. Downward-branching implies that biological compositional hierarchies rarely form neat levels, *even when we focus on a single mechanism or phenomenon*. Instead, they form branching structures, where usually after just two rounds of decomposition, we find very heterogeneous things in the different branches.

This does not necessarily mean the end for the notion of compositional levels or levels of mechanisms. One can also bite the bullet, accept downward-branching, and stipulate that each set of (direct) components that we find is worthy of the title “level.”³ For example, the (direct) components of a nerve cell (i.e., cell organs, etc.) could be said to form a “level” in this sense. With this approach, compositional levels are in essence reduced or deflated to sets of (direct) components (see also Eronen, 2013, 2015; Krickel, 2018). Thus, it is debatable whether these sets of direct components should be called “levels” or whether the term just invites misleading intuitions here (Eronen, 2013, 2015; Potochnik, 2017 and this volume).

4.3 Scale-Based Levels and Heuristics

One reaction to the problems in defining levels has been to abandon the whole idea of levels in biology as too confused and misleading (e.g., Guttman, 1976; Thalos, 2013; see also Potochnik, 2017 and this volume). However, it is important to keep in mind that conceptions of levels serve various purposes and scientific goals (Brooks & Eronen, 2018; Eronen, 2015), and for different goals we may need different notions of levels. If one notion of levels, or a set of assumptions underlying the use of levels, turns out to be deeply problematic, this does not imply that other notions or uses of levels are problematic as well. In particular, downward-branching and other considerations against levels discussed above only apply to *compositional* levels and not to other kinds of levels. Most important, they have no implications for scales or scale-based levels, which are also central in biology.

The role of scales in understanding biological organization has been extensively discussed by other authors (e.g., DiFrisco, 2017; Noble, 2012; Potochnik & McGill, 2012; see also Simon, 1962/1996). The scale that is probably the most intuitive one is the size scale, which is based on how big things are. The size scale is a key element in Wimsatt's (1994/2007) approach to levels, and different ways of measuring size (e.g., length, mass, or volume) and the associated scales play an important role in fields such as ecology or allometry. Another biologically crucial scale is the time scale, which is based on the rate at which interactions or processes take place. For example, interactions between neurotransmitters and receptors are much faster than interactions between neurons, which again are faster than interactions between organisms (e.g., reproduction). Scales have two key advantages over levels of organization (Eronen, 2015; Potochnik & McGill, 2012). First, scales are continuous, not discrete. Sometimes scales can separate so that interactions at a higher scale become to a large degree independent from interactions at lower scales (e.g., Simon, 1962/1996; Wimsatt, 1994/2007), but in general, scales allow for continuity and do not require cutting organisms or systems into discrete layers (see also Green & Batterman, 2017). Second, scales are relatively well defined: when the relevant quantitative property of an entity or process is measured (e.g., mass, volume, or rate of interactions), it is straightforward to determine where that entity or process falls on the scale.

It is also possible to formulate more substantive (noncompositional) accounts of levels based on scales. For example, Wimsatt (1994/2007) suggests that levels can be seen as local maxima (i.e., peaks) of regularity and predictability when plotted against a (size) scale.⁴ Potochnik and McGill (2012) argue that scale-based quasi-levels can be defined based on causal relationships. DiFrisco (2017) puts forward a dynamical approach, where levels are conceptualized in terms of time scales. These kinds of scale-based levels and their role in research and theory is undoubtedly an important field for future work on levels, but it is important to emphasize that they are different from composition-based levels and should not be conflated with them.

Another, complementary approach to levels is to take a *heuristic* perspective to them (Brooks & Eronen, 2018). Instead of seeing levels as ontological categories of levels of nature, they can be understood heuristically, in much the same way as Bechtel and Richardson (1993/2010) treat decomposition and localization as heuristics that work in some contexts (and for some purposes and goals) but not in others. As an example, consider the intuitive idea of levels as "similar things made up of similar things." As we have seen

above, there are very few if any cases in biology that *exactly* fit this idea. However, there are cases and specific contexts where it can be a good approximation. For instance, consider the protein network underlying the circadian clock mechanisms in mammals, as summarized by Bechtel (2017). The most important components in the cellular circadian clock mechanism are different kinds of proteins (e.g., PER, CRY) and the genes that transcribe these proteins. Both proteins and genes are composed of monomers (amino acids and nucleotides, respectively). In this context, it is perhaps useful (for some purposes) to conceptualize a higher level that is formed by similar things (genes and proteins) and a lower level that is formed by their components (monomers), which are also similar to one another. This two-level framework is obviously an abstraction, as the actual mechanisms are vastly more complex and include many more components (see, e.g., Skillings 2015). Nevertheless, this two-level picture can, for example, provide structure to the scientific debate or help in transporting the basic ideas of the organization of the mechanism to a context where the details matter less. A key question in this case as well as in others is whether the benefits of the levels-abstraction outweigh the drawbacks of distorting and losing details. The answer to this question will depend on the specifics of the case and the purpose or goal of the use of the notion of levels (see also Brooks & Eronen, 2018; Potochnik, this volume).

4.4 Levels and Downward Causation

Above I have argued that the traditional idea of compositional levels of organization is problematic but that there are nevertheless biologically relevant ways of making sense of levels. What is important to keep in mind is that these different notions of levels (e.g., minimalistic compositional levels, scale-based levels, heuristic idealizations) come with different assumptions and implications. In most scientific or philosophical contexts, these differences cannot be overlooked but are crucially important. One example of this is the debate on downward or top-down causation.

The concept of downward or top-down causation is intertwined with the concept of levels: in downward causation, higher-*level* states or processes cause lower-*level* states or processes. Many scientists and philosophers consider downward causation to be an important feature of nature (e.g., Campbell, 1974; Ellis, 2008; Noble, 2012), whereas others approach it with skepticism, for reasons having to do with causal exclusion and the nature of causation (e.g., Craver & Bechtel, 2007; Kim 1999, 2005). In this section, I will argue that there are many distinct forms of downward causation, corresponding to different notions of levels, and that arguments in favor or against one type of downward causation do not automatically apply to others.

First, the term “downward causation” often refers to causation where a change in a higher-level whole results in a change in the components of that whole. This kind of downward causation is the target of the now-classic paper by Craver and Bechtel (2007), who discuss downward causation in the framework of levels of mechanisms. Craver and Bechtel argue that what appears to be downward causation can be explained away as regular same-level causation that then has “mechanistically mediated effects” downward to the components in the mechanism. One of the examples provided by the authors is Hal’s tennis match. Hal is playing tennis, and this results in changes in the glucose metabolism of cells in Hal’s body. This looks like downward causation: the activity of the mechanism

as a whole (i.e., Hal playing tennis) causes changes in the activity of the components of the mechanism (the cell metabolism). However, according to Craver and Bechtel, seeing this as downward causation would violate many of the core ideas that we associate with causation, in particular, that causes must precede their effects and that causes must be distinct from their effects. As an alternative, Craver and Bechtel propose to analyze these cases in terms of the constitutive (noncausal) relationship between the mechanism and its components: when the activity of the mechanism as a whole changes, the activities of some of its components change as well, but simply because these activities of components make up the activity of the mechanism as a whole (see also Romero, 2015). There is no causation going from the higher to the lower level; rather, the higher level is constituted by the lower-level entities and activities.⁵

Craver and Bechtel (2007) are thus targeting *compositional* downward causation, where a system (or mechanism) as a whole exerts causal influence on its *own* parts. It is important to understand that their reasoning against downward causation does *not* carry over to contexts where downward causation is understood in a *noncompositional* way. One prominent example of such a context is the debate on mental causation. In this context, “downward causation” refers to cases where a mental state is a putative cause of physical behavior (Baumgartner 2010; Kim, 1999, 2005; Raatikainen, 2010). For example, John’s desire to drink beer is a putative cause for John walking toward the fridge (and the question then is whether or not this mental cause is excluded by a neural cause). Importantly, the physical behavior (e.g., walking toward the fridge) is *not* a component of the mental state (e.g., desire to drink beer) but is at a lower level in some more general sense.⁶ In Kim’s (1999) terminology, this kind of downward causation is *nonreflexive*, as the higher-level state or process is not influencing its own constituents. The sort of downward causation that Craver and Bechtel discuss, that is, where a whole influences its own parts, is *reflexive*. In non-reflexive downward causation, causes clearly precede their effects and are distinct from them, so the arguments of Craver and Bechtel do not apply.

As a biological example of (putative) nonreflexive downward causation, consider the following case discussed by Love (2012). When explaining protein folding, scientists appeal to chaperone molecules in the cellular environment. Chaperones interact with the primary structure of the folding protein (the amino acids in a sequence), and this causal impact is due to the three-dimensional overall structure of the chaperones. This seems to be a case of downward causation in the sense that the higher-level properties of chaperones affect the lower level of primary amino acid structure. However, as the amino acids in the folding proteins are *not* components of the chaperones, the (putative) downward causation here is not reflexive: the causal relationship is not between a whole (mechanism) and its own parts. Consequently, this kind of downward causation does not face the metaphysical problems raised by Craver and Bechtel.

In the context of this book chapter, a key question is how to understand the “levels” in this kind of downward causation. In my view, the heuristic approach characterized at the end of the previous section is perfectly suited for this purpose. As chaperones are proteins themselves and thus also made up of amino acids, they are similar to the folding proteins in important ways: both are proteins and composed of amino acids. Thus, in this limited context, the entities of interest seem to fit the idea of “similar things made up of similar things” sufficiently well to make the levels notion useful. For this reason, it is justifiable to label this

case as downward causation, as long as it is appreciated that this kind of downward causation is very different from the reflexive downward causation discussed above. Moreover, as the levels here are not conceptualized ontologically but understood heuristically, the sense in which causation goes “downward” in these cases is also heuristic and not ontological. This suggests that in contexts where levels are understood heuristically, also the question of whether there is downward causation or not becomes heuristic in nature.

There are also further types of downward causation where a *scale-based* notion of levels seems to be more appropriate than a compositional or heuristic one.⁷ Consider the following examples from the downward causation literature. Bishop (2008) argues that downward causation occurs when convection cells in a fluid constrain the behavior of the individual molecules in the fluid. According to Green (2018), properties of tissue structure (e.g., resistance) set boundary conditions for the propagation of action potentials and this amounts to downward causation. Based on his seminal work on cardiac rhythm, Noble (2006, 2012) points out that the behavior of ion channels in a cell depends on the cell potential, which is a system-level property of the cell. In all of these cases, the relevant levels do not seem to be compositional but scale-based levels, as is also explicitly stated by Green (2018) and Noble (2012). For example, there is no sense in which an ion channel is a component of the cell potential. Thus, the worries raised by Craver and Bechtel (e.g., that causes need to be distinct from their effects) do not apply, at least not in the same form as with reflexive downward causation (see also Green, this volume; Woodward, this volume, for more on downward causation). On the other hand, nonreflexive forms of downward causation (both scale based and heuristic) potentially face the causal exclusion problem, which has been intensely debated in recent years (see, e.g., Baumgartner, 2010, for a clear statement of the problem and Eronen & Brooks, 2014, and Woodward, 2015, for proposed solutions).

A more detailed analysis of the plausibility of downward causation in its different forms is beyond the scope of this chapter. I hope to have shown that it is crucially important to pay attention to the notion of levels underlying claims of downward causation, because different notions of levels correspond to different forms of downward causation. As arguments in favor or against one form do not automatically transfer to another form, it is important to clearly distinguish between them and to give each form a separate treatment.

4.5 Conclusions

The idea of compositional levels of organization figures prominently in contemporary biology and its philosophy. In this chapter, I have analyzed the complex relationship between compositional organization and levels of organization. The upshot is that compositional organization may result in neat layer-cake style levels when components are sufficiently homogeneous, but the more heterogeneous the components are, the more branching and less layered the organizational structure will be. In biology, components tend to be extremely heterogeneous, which leads to branching structures and highly local compositional levels that do not even span across one mechanism or system. The idea of levels as similar things that are composed of things that are similar to one another can at best be seen as a heuristic abstraction that may be useful in specific biological contexts. Finally, in the debate on downward causation, it is particularly important to make assumptions about levels explicit, as different notions of levels correspond to different types of downward causation.

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Notes

1. More technically, direct components can be defined as the components of a system that are not components of any other component of the system (Eronen, 2015).
2. This is just a simplified “textbook”-style picture of the components of the cells—the actual compositional organization is far more complex and heterogeneous (see, e.g., Skillings, 2015).
3. In some cases, it may also be possible to delineate compositional levels that are more general. The cases that I have in mind are those where *all* (or most) compositional branches in a system eventually involve certain types of components. As an example, let us assume that in all compositional branches in the spatial memory mechanism in a mouse brain, we will at some point end up with synapses as components. In this case, it can be useful and informative (for various purposes) to think of these synapses as being “at the same level.” This would then amount to a synaptic level that is not just specific to a certain branch of the mechanism but cuts across the whole mechanism.
4. Wimsatt (1994/2007), however, argues that these levels are also compositional.
5. See, however, Bechtel (2017), who now argues that downward causation occurs when a mechanism as a whole constrains or controls the behavior of its parts. A similar argument has been made by Kistler (2009). Another way of saving compositional downward causation is to conceptualize mechanisms as temporally extended entities, in which case higher-level causes *can* be distinct from their lower-level effects; see Krickel (2017) for more.
6. It is far from clear in what sense behavior is at a lower level than mental states. In a closer analysis, it may well turn out that levels talk is not very suitable or helpful here.
7. See also Ellis (2008), Emmeche et al. (2000), Moreno and Umerez (2000), and especially Hulswit (2005) for overviews of yet further types of downward causation.

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