No Levels, No Problems: Downward Causation in Neuroscience

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Abstract

I show that the recent account of levels in neuroscience proposed by Bechtel and Craver is unsatisfactory, since it fails to provide a plausible criterion for being at the same level and is incompatible with Bechtel and Craver's account of downward causation. Furthermore, I argue that no distinct notion of levels is needed for analyzing explanations and causal issues in neuroscience: it is better to rely on more well-defined notions such as composition and scale. One outcome of this is that there is no distinct problem of downward causation.

1. Introduction

The notion of "level" appears in several contexts in philosophy of science. For example, the debates on downward causation, mechanistic explanation, reduction, and emergence are conducted in the framework of levels. However, there is no agreement on the definition of a level, or on the criteria for distinguishing levels.

Craver and Bechtel (2007) have recently presented a theory of "levels of mechanisms", which has gained broad acceptance and is currently the most coherent and promising account of levels. They argue for levels of mechanisms, where the relata are mechanisms at higher levels and their components at lower levels. Importantly, these are not general levels of organization, but identified with regard to a certain mechanism. Craver and Bechtel claim that although levels of mechanisms is certainly not the only sense in which "level" is employed in neuroscience or philosophy, it captures the central sense in which explanations in neuroscience span multiple levels. They also employ this theory of levels to deal with the problem of downward causation, arguing that what appears as downward causation can be explained away as same-level causation that has mechanistically mediated effects.

In this paper, I will (1) show that the mechanistic account of levels is unsatisfying, (2) defend an alternative "deflationary" account of levels, where the notion of level is replaced with the more fundamental notions of composition and scale, and (3) explore the consequences this has for the debate on downward causation. My focus

is on neuroscience and downward causation, but the general arguments I raise against levels apply more broadly.

In the next section, I will briefly present the account of levels of mechanisms. In section 3, I will show that this account fails as a theory of levels, since it does not provide any plausible same-level criterion. In section 4, I argue that we should get rid of the problematic notion of "level" altogether and replace it with notions such as scale and composition, which are far better understood. In section 5, I explore some of the consequences this has for the debate on downward causation.

2. Levels of Mechanisms

In most philosophical theories of levels, the core idea is that levels are *compositional*: wholes are at a higher level than the parts that they are composed of (e.g., Oppenheim and Putnam 1958; Wimsatt 1994; Kim 1999). The mechanistic account of levels retains this basic idea, with one important amendment: the relata are not just wholes and parts; they are *behaving* mechanisms and their *active* components. This means that the higher-level entity is an active mechanism performing some function, and the lower-level entities are components that contribute to the mechanism for this function.

Craver gives the following characterization: "In levels of mechanisms, the relata are behaving mechanisms at higher levels and their components at lower levels. These relata are properly conceived neither as entities nor as activities; rather, they should

be understood as acting entities. The interlevel relationship is as follows: X's Φ-ing is at a lower mechanistic level than Ψ-ing if and only if X's Φ-ing is a component in the mechanism for S's Ψ-ing. Lower-level components are *organized together* to form higher-level components." (Craver 2007, 189)

In a similar vein, albeit in more vague terms, Bechtel writes: "Within a mechanism, the relevant parts are … working parts—the parts that perform the operations that enable the mechanism to realize the phenomenon of interest. … It is the set of working parts that are organized and whose operations are coordinated to realize the phenomenon of interest that constitute a level" (Bechtel 2008, 146).

Craver's (2007, 165-170) main example is the case of spatial memory and LTP (Long Term Potentiation), where he identifies four levels. On the top of the hierarchy, there is the level of *spatial memory*, which involves various types of memory and learning. The level of *spatial map formation* includes the structural and computational properties of various brain regions involved in spatial memory, most importantly the hippocampus. The *cellular-electrophysiological* level includes neurons that depolarize and fire, synapses that undergo LTP, action potentials that propagate, and so on. At the bottom of this hierarchy is the *molecular* level, where we find NMDA and AMPA receptors, Ca^{2+} and Mg^{2+} ions, etc. Entities at each lower level are components in a higher-level mechanism: for example, the hippocampus is an active component in the spatial memory mechanism, synapses are active components in the hippocampal mechanism of memory consolidation, and finally, NMDA receptors are active components of the synaptic mechanism of LTP.

Importantly, Craver and Bechtel emphasize that levels of mechanisms are not general levels of organization in the vein of Oppenheim & Putnam (1958), Churchland & Sejnowski (1992) or Wimsatt (1994). “A consequence of this view is that levels are identified only with respect to a given mechanism; this approach does not support a conception of levels that extend across the natural world” (Bechtel 2007).”How many levels there are, and which levels are included, are questions to be answered on a case-by-case basis by discovering which components at which size scales are explanatorily relevant for a given phenomenon” (Craver 2007, 191).

Bechtel and Craver see this as a point in favor of the mechanistic account of levels, since accounts of general levels of organization are ridden with problems: it makes little sense to compare the “level” of glaciers and pyramidal cells, or black holes and microchips. However, the limitations Bechtel and Craver impose are quite extreme: in the mechanistic framework, it does not make sense to ask whether things that belong to different mechanisms are at the same level or not. We cannot even say that a certain molecule in a hippocampus is at a lower level than the hippocampus, unless the molecule is a component of some hippocampal mechanism (Craver 2007, 191).

Even within one mechanism, things that do not stand in a part-whole relation may not be in a level-relation to each other (see, e.g., Craver 2007, 193). One salient example of this is that there is no sense in which the subcomponents of different components of the mechanism are at the same or different level. For example, a component C1 of mechanism M is at one level lower than M, and a subcomponent S1

of C1 is one level lower than the component C1. Another component C2 of M is also one level lower than the mechanism M, and its subcomponent S2 is one level lower than the component C2. However, according to the mechanistic account, the question whether subcomponents S1 and S2 are at the same or different level makes no sense, since they do not stand in a part-whole relation to each other. I return to this issue in the next section.

To summarize, the key features of this account are the following: (1) Levels are "local" – they are always defined relative to one mechanism and the phenomenon of interest. (2) The relata are mechanisms at higher levels and components or "acting entities" or "working parts" at lower levels. (3) Things are assigned to different levels solely based on the part-whole (or component-mechanism) relation: wholes are at a higher level than their parts; parts are at a lower level than the wholes they belong to. In the next section, I show that these features lead to problems, particularly feature (3).

3. Components, Mechanisms, and Problems

Let us consider the mechanism for phototransduction (the conversion of light signals into electrophysiological information) in the retina. Components in this mechanism include rod and cone cells, which are morphologically and functionally distinct types of cells. However, the phototransduction cascade in both rods and cones involves similar components: G proteins (transducin), cyclic guanosine

monophosphate (cGMP), cGMP-gated ion channels, and so on. The cGMP-gated channels in rods and the same types of channels in cones are subcomponents of *different* components of the mechanisms for light adaptation. They do not stand in a part-whole relation. Hence, according to the mechanistic account, there is no sense in which they are at the same or higher or lower level with regard to each other.

However, this is quite implausible. cGMP-gated ion channels in rods and cGMP-gated ion channels in cones are same types of things with same properties, at the same scale, in the same system, and playing a corresponding role in their respective mechanisms (i.e., they are the same types of "acting entities"). If the mechanistic account implies that there is no sense in which these ion channels are at the same level, something seems to have gone wrong, or at least the levels metaphor is used in a way that is extremely unintuitive (I return to this in Section 4).

Things get even more problematic when we consider subcomponents that are causally interacting with each other. For example, consider synaptic transmission between rod cells and (OFF-type) bipolar cells. In the mechanism for synaptic transmission between these cells, active components of the rod cell include synaptic vesicles, which in turn have glutamate molecules as their subcomponents. The active components of the bipolar cells include (AMPA) glutamate receptors, which have "binding sites" as active components. When the rod cell is firing, the glutamate molecules in the vesicles are released, and they bind to the binding sites of the glutamate receptors.

This means that subcomponents (glutamate molecules) of one component (synaptic vesicles) are causally interacting with subcomponents (binding sites) of a different component (AMPA receptors).[1] Yet, Craver and Bechtel explicitly state that there is no sense in which subcomponents of different components are at the same level. This is not only peculiar, but also in fundamental conflict with Craver and Bechtel's (2007) account of cross-level causation: they explicitly defend the view that there is no cross-level or downward causation – causation is an *intralevel* matter, and effects can be then "mechanistically mediated" upwards or downwards in the mechanism. In other words, being at the same level is a *necessary condition* for causal interaction. However, we have now seen that if we follow Craver and Bechtel's own theory of levels, there are clear cases where there are causal interactions between entities that are *not* at the same level. Thus, there is a fundamental conflict between the mechanistic theory of levels and the mechanistic account of downward causation.[2]

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[1] This is not an isolated example - Fazekas and Kertesz (2011) have recently pointed out other examples and argued that, quite generally, if the components of a mechanism causally interact, also their subcomponents have to causally interact.

[2] I do not want to discuss the nature of causation here, and my main points hold independently of any particular theory of causation. However, the account of causation most naturally fitting the general framework here would be the interventionist theory of causation (e.g., Woodward 2003), which also Craver (2007) explicitly endorses.

These problems are related to the fact that the mechanistic account gives no satisfactory criterion for determining when things are at the *same* level. According to Craver, there is only a partial answer to this question: ”X and S are at the same level of mechanisms only if X and S are components in the same mechanism, X's Φ-ing is not a component in S's Ψ-ing, and S's Ψ-ing is not a component in X's Φ-ing.” (2007, 192). In other words, what places two items at the same mechanistic level is that they are in the same mechanism, and neither is a component of the other (Craver 2007, 195).

One way of interpreting this is that if any two components in the mechanism are not in a part-whole relation with each other, they are at the same level. However, this would have some bizarre consequences. Consider components X and S in mechanism M. They are at the same level, since X is not component of S and S is not a component of X. Consider then a subcomponent S1 of S. It is also not a component of X, and X is not a component of S1. Then X and S1 are also at the same level, as well as all the further subcomponents of S1 and all their subcomponents! This would be a rather strange account of the same-level relation.

Supposedly the idea is rather that things that are components in a mechanism but not components in any *intermediate* component are at the same level. For example, rod A is at the same level as rod B, since they are components of the phototransduction mechanism and do not stand in a part-whole relation, but a cGMP-gated ion channel in rod B is not at the same level as rod A, because the cGMP-gated ion channel is a component of rod B, and not a “direct” component of the

phototransduction mechanism. Let us call such components that are components in the mechanism directly and not in virtue of being components in another component *direct components*.

If no further restrictions are added, direct components can include things of radically different sizes with very different causal properties. For example, direct components in the mechanism for light transduction in rod cells include things such as the outer segment of the cell, which has the function of capturing photons and may contain billions of opsin molecules. On the other hand, direct components in the mechanism also include single photons hitting the cell, or Na^+-ions in the cell - these are also not components in any intermediate component of the mechanism. It follows that rod outer segments are at the same level of mechanism as photons or Na^+-ions, even though they differ in scale with a factor of at least 10^7 .

Thus, it seems that the same-level criterion that Craver proposes is both too weak and too strong. It is too weak because it implies that in many cases things that are causally interacting and have very similar properties are *not* at the same level. It is too strong because it implies that in many cases things that are of radically different size and that interact at completely different force or time scales are at the same level. This (1) makes the criterion ineffective for distinguishing between interlevel and intralevel causation, and (2) streches the metaphor of "level" near the breaking point.

4. Levels: A Deflationary Account

The main source for the problems outlined above is that the account of Craver and Bechtel is too limited as a theory of levels. It is not an undue exaggeration to say that the account of levels of mechanisms is in fact an account of mechanistic *composition*: it relies entirely on the component-mechanism relation and simply labels whole mechanisms as being at higher "levels" and their components as being at lower "levels". For this reason, it is difficult to define any reasonable same-level relation in this framework: composition only relates parts and wholes, and not parts with other parts or wholes with other wholes.

My suggestion is, first of all, to take the approach of Craver and Bechtel into its logical conclusion and to deflate the notion of mechanistic levels into simply mechanistic composition. We can simply reinterpret the mechanistic account of levels as an account of mechanistic composition, as long as we strip away the idea of being at the "same" mechanistic level and the related claims about same-level causation. I fully agree with Craver and Bechtel in that explanations in neuroscience refer to robust properties and generalizations throughout the compositional hierarchy – for example, in the explanation for phototransduction we need to consider the 11-cis-retinal molecule changing shape, the rod photoreceptor cell hyperpolarizing, the retinal network computing, the eye converting light to electrophysiological signals, and so on.

However, it is obvious from section 3 that this will not be sufficient as a framework for dealing with issues such as downward causation. Therefore, the second step of

my solution is to take into account the dimension of *scale*, which is largely independent from composition. In his discussion of levels, Craver (2007, ch. 5) acknowledges the importance of size scale, but argues that it is secondary to composition: components cannot be larger that the wholes they are part of, so in this sense the size dimension partly follows the compositional dimension. However, we have also seen above that composition and size often come apart: the direct components of a mechanism can be of radically different sizes, and similarity or difference of size does not imply that entities are in any way compositionally related. Composition and scale are largely independent dimensions (see also Richardson and Stephan 2007; Rueger & McGivern 2010).

The most commonly discussed scale is size scale, but also other scales such as the temporal scale (the speed of interactions) or the force scale (the strength of interactions) may be just as important in understanding complex systems (see, e.g., Simon 1962; Rueger & McGivern 2010). For example, molecular interactions happen at a much faster time scale than interactions between neurons, which are again faster than interactions between brain areas. The force scale is particularly important when considering physical and chemical interactions: for example, the forces binding subatomic particles (quarks) together are much stronger than the forces binding atoms together, which are again stronger than the forces binding molecules together. For the sake of clarity, I focus here mostly on the size scale.

One problem of the mechanistic account of levels was that its same-level relation leads to results that seem arbitrary and unintuitive: for example, there is no sense in

which subcomponents of components are at the same mechanistic level, even when they are same types of things, while entities of radically different sizes can be at the same level. In my view, it is better to get rid of the idea of being at the "same level" altogether, and just to focus on how things are related on different scales (see also Potochnik & McGill 2012). For example, cGMP-gated ion channel are obviously found at the same size (and temporal) scale than cGMP-gated ion channels in cones, while rod outer segments are found at very different size (and temporal) scales than Na^+ ions.

One outcome of analyzing levels in terms of scale and composition is that we no longer need any distinct notion of level. If scale and composition are sufficient for analyzing explanations in neuroscience, the notion of "level" does not add anything to our conceptual toolkit. Explanations in neuroscience are "multilevel" only in the sense that they refer to robust properties and generalizations at various stages in the compositional hierarchy and at different (size) scales.

This approach is also supported by neuroscientific practice. In contrast to what Craver (2007, ch. 5) suggests, levels talk is not very common in neuroscience, neither in journal articles nor in standard textbooks such as Kandel, Jessell and Schwartz (2000) or Purves et al. (2004). In many articles (see, e.g., Malenka & Bear 2004) the term does not come up at all. When it does appear, it is most often referring to levels of processing, such as the different stages of visual information processing (the retina, the LGN, the visual cortex, and so on), which are something

very different from levels of mechanisms, and "levels" only in a metaphorical sense.[3] This supports my point that the notion of level does not pick up any distinct or important category.[4]

If one insists on using the term "level" to refer to stages of composition or to different size scales (or to various other things – scale and composition are merely the senses most relevant in this context), one has to at least make clear in exactly which sense the term is used. However, the danger in this is that other intuitions about levels may creep in – for example, when talking of compositional stages as "levels", one is easily lead to think that things can be at the "same level" of composition.

5. Downward Causation and Levels

I have argued above that the idea of levels is thoroughly problematic, at least in philosophy of neuroscience, and that we should abandon the project of trying to define levels. Let us now turn to the issue of downward or top-down causation that has been traditionally discussed in the framework of levels (e.g., Campbell 1974;

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[3] Of course, the *word* "level" often comes up in the trivial sense of "luminance level", "level of oxygen", "level of noise", etc.

[4] Ladyman and Ross (2007, 54) reach a similar conclusion in the philosophy of physics.

Emmeche et al. 2000; Kim 1992, 1999; Craver and Bechtel 2007; Kistler 2009).[5] The question is whether higher-level causes can have lower-level effects. In spite of various arguments to the effect that downward causation is not possible, the debate keeps resurfacing, partly because (neuro)scientists often rely on top-down experiments and explanations that seem to imply some kind of downward causation.

As we have seen above, Craver and Bechtel (2007) have proposed a novel solution to the problem of downward causation. They argue that what appears to be downward causation in top-down experiments and elsewhere should be understood as normal same-level causation that has "mechanistically mediated" effects downwards in the mechanism: there is no causation from higher to lower levels or the other way around.

Considering the discussion in the previous two sections, it is clear that the reason why the solution of Craver and Bechtel does not work is that it relies on the distinction between same-level and cross-level causation. We have seen how difficult it is to define the same-level relation, or levels in general, in a coherent and scientifically plausible way. The term "level" does not seem to pick up any distinct

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[5] In a recent article, Love (2012) discusses top-down causation in terms of levels, but in a way that comes closer to my approach: he argues that there are many different kinds of level-hierarchies and correspondingly many different kinds of top-down causation.

category in neuroscience. For this reason, basing the account of downward causation on the distinction between same-*level* causation (which is supposed to be unproblematic) and cross-*level* causation (which is supposed to be unacceptable) necessarily leads to problems.

One possibility would be to try to reformulate Craver and Bechtel's solution in terms of scale and composition. If we could distinguish between same- and different-"level" causation in terms of scale and composition, perhaps the solution could still work. Unfortunately, this does not seem to be the case. As I have already pointed out in the previous section, composition as such does not involve any same-"level" relation. Regarding (size) scale, the problem is that there is absolutely no reason to restrict causation to things of same or similar size: elephants squash flies, the fission of uranium atoms causes cities to disintegrate, and so on. Therefore, we have to conclude that Craver and Bechtel's approach downward causation is unsatisfactory.

If we abandon the framework of levels and focus on scale and composition, what appears to be downward causation reduces to two categories: (1) Causes that act from the mechanism as a whole to the components of the same mechanism, and (2) causation between entities of different (size) scales. In my view, it is fairly clear that there can be no causation between things that are related by composition (category (1)), since composition is a form of non-causal dependency. It does not seem right to say that, e.g., the retina as a whole causes a rod cell in that retina to fire. On the other hand, as the examples in the previous paragraph show, causation between things of

different size[6] is in principle unproblematic (category (2)). In this way, putative cases of top-down or downward causation can be analyzed away in terms of composition and scale.[7]

One remaining problem for "downward" causation of category (2) is Kim's argument against higher-level causes. It might prima facie seem that getting rid of levels dissolves this problem, since it is often formulated in terms of levels: the argument states that a higher-level property cannot be a genuine cause for a lower-level property, since (due to physical causal closure) the lower-level property already has a sufficient lower-level cause (see, e.g., Kim 1992; 1999). However, the idea of "levels" is not essential in Kim's argument: what is at issue there is the tension created by two competing (and non-causally correlated) causes for the same effect. Without the framework of levels, the argument does not disappear, but turns into the general causal exclusion argument (see, e.g., Kim (2002) Bennett (2008) for more).

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[6] Whether the same holds for other scales, such as the temporal or the force scale, is an open question that goes beyond the scope of this paper.

[7] One way of interpreting Craver and Bechtel (2007) is that their main point is quite similar, namely that apparent causation from parts to wholes or wholes to parts can be analyzed away in terms of normal causal relations. If this is the case, it is unfortunate that the theory of levels and the distinction between "same-level" and "different-level" causation is so prominent in the paper, since this makes the account unnecessarily complex and confusing.

What Craver and Bechtel (2007) are considering, and what I have discussed in this section, is the intelligibility of causes acting from higher to lower levels. I have argued that downward causation is not intelligible in the sense of causation from a mechanism as a whole to the parts of that same mechanism, but causation from higher to lower scales is as such unproblematic. There may be real problems related to causation in neuroscience, such as the causal exclusion problem, but there is no distinct problem of downward causation.

6. Conclusions

In this paper, I have argued that the account of "levels of mechanisms" is unsatisfactory as a theory of levels, since it does not include a plausible same-level relation, leads to extremely unintuitive results, and is in conflict with the account of downward causation proposed by Craver and Bechtel. Generally speaking, there seems to be no need for a distinct notion or theory of levels in philosophy of mind or neuroscience; it is better to rely on more familiar and well-defined notions such as scale and composition. With this approach, apparent cases of downward causation can be analyzed away.

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