No Levels, No Problems: Downward Causation in Neuroscience

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I show that the recent account of levels in neuroscience proposed by Craver and Bechtel is unsatisfactory since it fails to provide a plausible criterion for being at the same level and is incompatible with Craver and Bechtel'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 apparent cases of downward causation can be analyzed away.

1. Introduction. The notion of "levels" 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.

Carl Craver and Bill Bechtel have recently presented a theory of "levels of mechanisms" (Craver 2007; Craver and Bechtel 2007; Bechtel 2008) that has gained broad acceptance and is currently the most promising account

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†I thank Bob Richardson, Bill Bechtel, Carl Craver, Albert Newen, Jani Raerinne, Leon de Bruin, Raphael van Riel, and Laura Bringmann for very helpful comments on earlier drafts of this article or related presentations. I also thank Malte Ahlers for introducing me to the details of retina research. I have presented different versions of this paper at the Trends and Tensions in Intellectual Integration research colloquium at the University of Helsinki (March 2012), the workshop "Metaphysics of Mind and Brain" in Berlin (July 2012), the Eighth International Congress of the Society for Analytic Philosophy in Constance (September 2012), and PSA 2012 in San Diego (November 2012). I thank the audiences of these talks for helpful questions and feedback. The research that led to this publication was financially supported by the Hanse-Wissenschaftskolleg Institute for Advanced Study and the Ruhr University Bochum.

Philosophy of Science, 80 (December 2013) pp. 1042–1052. 0031-8248/2013/8005-0019$10.00
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https://doi.org/10.1086/673898 Published online by Cambridge University Press

of levels in philosophy of science. In levels of mechanisms, the relata are mechanisms at higher levels and their components at lower levels. Importantly, these are not general levels of organization but are 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 apply this theory of levels to 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 article, I will show that the mechanistic account of levels is unsatisfactory (sec. 3), defend an alternative "deflationary" account of levels in which the notion of level is replaced with the more fundamental notions of composition and scale (sec. 4), and explore the consequences this has for the debate on downward causation (sec. 5). My focus is on neuroscience and downward causation, but the general arguments I raise against levels apply more broadly. First of all, I will briefly present the account of levels of mechanisms.

2. Levels of Mechanisms. In most philosophical theories of levels of organization, 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 S's 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, 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. These may be of different sizes, but they are distinguished by the fact that they figure in the functioning of the mechanism. 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).

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Craver's (2007, 165–70) main example is the case of spatial memory and long-term potentiation (LTP), 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, and so forth. Entities at each lower level are components of a higher-level mechanism: the hippocampus is an active component of the spatial memory mechanism, synapses are active components of 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 and Putnam (1958), Churchland and Sejnowski (1992), or Wimsatt (1994). "Levels of mechanisms are not monolithic divides across all of nature. Levels of mechanisms are defined locally, within the context of a given type of mechanism" (Craver and Bechtel 2007, 550). "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).

Craver and Bechtel 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, this also has some radical implications: 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, not all things are 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 (Bechtel 2008, 147). For example, a component C_1 of mechanism M is one level lower than M, and a subcomponent S_1 of C_1 is one level lower than the component C_1. Another component C_2 of M is also one level lower than the mechanism M, and its subcomponent S_2 is one level lower than the component C_2. However, according to the mechanistic account, the question whether subcomponents S_1 and S_2 are at the same or a different level makes no sense since they do

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not stand in a part-whole relation to each other. I return to this issue in the next section.

To summarize: in levels of mechanisms, the relata are behaving mechanisms at higher levels and their components or working parts at lower levels; things that do not stand in part-whole (or component-mechanism) relation are not at higher or lower levels with respect to each other. Consequently, levels of mechanisms are local: they are identified with respect to a certain phenomenon or function and the mechanism that is responsible for it. In the next section, I show that these features lead to problems.

3. Components, Mechanisms, and Problems. Let us consider the mechanism for phototransduction (the conversion of light signals into electrophysiological information) in the retina (see, e.g., Pugh and Lamb 2000). Components of 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 mechanism for light transduction. 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 a 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 the same types of things with the same properties, at the same scale, in the same system, and are 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 sec. 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 (bind-

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ing 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 not only is peculiar but also is 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 then be "mechanistically mediated" upward or downward 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 in which 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]

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 (195).[3]

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 C_1 and C_2 in mechanism M. They are at the same level since C_1 is not a component of C_2 and C_2 is not a component of C_1. Consider then a subcomponent S_1 of C_1. It is also not a component of C_2, and C_2 is not a component of S_1. Then C_2 and S_1 are also at the same level, as well as all the further subcomponents of S_1 and all their subcomponents! This would be a rather strange account of the same-level relation.

The idea must rather be that things that are components of a mechanism but not components of 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 re-

1. Fazekas and Kertész (2011) point out other examples and argue that, quite generally, if the components of a mechanism causally interact, their subcomponents also have to causally interact.

2. The account of causation most naturally fitting the general framework here is the interventionist theory of causation (Woodward 2003), which Craver (2007) also explicitly endorses.

3. In Bechtel's account, things are at the same level if they are working parts of the same mechanism (2008, 146–47). This leads to the same problems as Craver's criterion.

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lation to each other, 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 of the mechanism directly and not in virtue of being components of 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. However, 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.[4] It is too weak because it implies that in many cases things that are causally interacting or are the same types of things in the same system 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) stretches the level metaphor 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 relates only 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 to its logical conclusion and to deflate the notion of mechanistic levels into mechanistic composition. We can simply reinterpret the mechanistic

4. In personal communication, Craver has pointed out that he no longer believes that we need a criterion for being at the same level; what is important is determining when things are at different levels. However, I doubt whether it makes sense to talk of levels if there is no sense in which things can be at the same level.

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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 their discussions of levels, Craver (2007, chap. 5) and Bechtel (2008, 143–45) acknowledge the importance of size scale but argue that it is secondary to composition: components cannot be larger than 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.

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 and 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 that I discussed above is 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 the 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 and McGill 2012). For example, cGMP-gated ion channels in rods are obviously found at the same size (and temporal) scale as

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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, at least not in any sense resembling levels of mechanisms. If scale and composition are sufficient for analyzing explanations in neuroscience, the notion of "level" does not add anything to our conceptual tool kit. 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, chap. 5) suggests, levels talk is not ubiquitous in neuroscience, neither in journal articles nor in standard textbooks (e.g., Kandel, Schwartz, and Jessell 2000; Purves et al. 2008). This applies also to the case of memory consolidation and LTP, which is Craver's paradigmatic example of a multilevel mechanism; see, for example, McGaugh (2000) and Malenka and Bear (2004) for two representative review articles in which there is no mention of levels. When it does appear, the term is most often referring to levels of processing, such as the different stages of visual information processing (the retina, the lateral geniculate nucleus, the visual cortex, and so on), which are something very different from levels of mechanisms, and levels only in a metaphorical sense.[5] This supports my point that the notion of level, at least in the sense of levels of mechanisms, does not pick up anything distinct or explanatorily substantial.[6]

If one prefers using the term "level" to refer to compositional hierarchies 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 hierarchies in terms of levels, one is easily led 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 of mechanisms is thoroughly problematic 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

5. Craver (2007, chap. 5) offers a useful taxonomy of the different ways the term "level" is used in neuroscience.

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

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the framework of levels (e.g., Campbell 1974; Kim 1992, 1999; Emmeche, Køppe, and Stjernfelt 2000; Craver and Bechtel 2007; Kistler 2009). 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 downward in the mechanism: there is no causation from higher to lower levels or the other way around.

From the discussion in the previous two sections, it is clear that the reason why the solution of Craver and Bechtel is problematic 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. 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 (Craver [2007, chap. 5] and Bechtel [2008, 144] agree on this). Therefore, we have to conclude that Craver and Bechtel's approach to 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 noncausal dependency. It does not seem right to say that, for example, 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 is in principle unproblematic (category 2). In this

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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 (because of 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 related) 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] or Bennett [2008] for more).

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.

REFERENCES

Bechtel, William. 2008. *Mental Mechanisms*. London: Routledge.

Bennett, Karen. 2008. "Exclusion Again." In *Being Reduced*, ed. J. Hohwy and J. Kallestrup, 280–305. Oxford: Oxford University Press.

Campbell, Donald T. 1974. "'Downward Causation' in Hierarchically Organised Biological Systems." In *Studies in the Philosophy of Biology*, ed. F. Ayala and T. Dobzhansky, 179–86. Berkeley: University of California Press.

Churchland, Patricia S., and Terence J. Sejnowski. 1992. *The Computational Brain*. Cambridge, MA: MIT Press.

Craver, Carl F. 2007. *Explaining the Brain*. Oxford: Oxford University Press.

Craver, Carl F., and William Bechtel. 2007. "Top-Down Causation without Top-Down Causes." *Biology and Philosophy* 20:715–34.

Emmeche, Claus, Simo Køppe, and Frederik Stjernfelt. 2000. "Levels, Emergence, and Three Versions of Downward Causation." In *Downward Causation: Minds, Bodies and Matter*, ed. Peter B. Andersen, Claus Emmeche, Niels O. Finnemann, and Peder V. Christiansen, 13–34. Aarhus: Aarhus University Press.

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 are so prominent in the article since this makes the account unnecessarily complex and confusing.

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Fazekas, Peter, and Gergely Kertész. 2011. "Causation at Different Levels: Tracking the Commitments of Mechanistic Explanations." *Biology and Philosophy* 26:365–83.
Kandel, Eric R., James H. Schwartz, and Thomas Jessell. 2000. *Principles of Neural Science*. 4th ed. New York: McGraw-Hill.
Kim, Jaegwon. 1992. "'Downward Causation' in Emergentism and Nonreductive Physicalism." In *Emergence or Reduction? Essays on the Prospects of Nonreductive Physicalism*, ed. Ansgar Beckermann, Hans Flohr, and Jaegwon Kim, 119–38. Berlin: de Gruyter.
———. 1999. "Making Sense of Emergence." *Philosophical Studies* 95:3–36.
———. 2002. "Mental Causation and Consciousness: The Two Mind-Body Problems for the Physicalist." In *Physicalism and Its Discontents*, ed. Carl Gillett and Barry Loewer, 271–83. Cambridge: Cambridge University Press.
Kistler, Max. 2009. "Mechanisms and Downward Causation." *Philosophical Psychology* 22: 595–609.
Ladyman, James, and Don Ross. 2007. *Every Thing Must Go: Metaphysics Naturalized*. Oxford: Oxford University Press.
Malenka, Robert C., and Mark F. Bear. 2004. "LTP and LTD: An Embarrassment of Riches." *Neuron* 44:5–21.
McGaugh, James L. 2000. "Memory—a Century of Consolidation." *Science* 287:248–51.
Oppenheim, Paul, and Hilary Putnam. 1958. "Unity of Science as a Working Hypothesis." *Minnesota Studies in the Philosophy of Science* 2:3–36.
Potochnik, Angela, and Brian McGill. 2012. "The Limitations of Hierarchical Organization." *Philosophy of Science* 79:120–40.
Pugh, Edward N., and Trevor D. Lamb. 2000. "Phototransduction in Vertebrate Rods and Cones: Molecular Mechanisms of Amplification, Recovery and Light Adaptation." In *Handbook of Biological Physics*, vol. 3, *Molecular Mechanisms of Visual Transduction*, ed. D. G. Stavenga, W. J. de Grip, and E. N. Pugh, 183–255. Amsterdam: Elsevier.
Purves, Dale, George Augustine, David Fitzpatrick, William Hall, Anthony-Samuel LaMantia, James McNamara, and Leonard E. White, eds. 2008. *Neuroscience*. 4th ed. Sunderland, MA: Sinauer.
Rueger, Alexander, and Patrick McGivern. 2010. "Hierarchies and Levels of Reality." *Synthese* 176:379–97.
Simon, Herbert A. 1962. "The Architecture of Complexity." *Proceedings of the American Philosophical Society* 106:467–82.
Wimsatt, William C. 1994. "The Ontology of Complex Systems: Levels of Organization, Perspectives, and Causal Thickets." *Canadian Journal of Philosophy* 20 (Suppl.): 207–74.
Woodward, James. 2003. *Making Things Happen*. Oxford: Oxford University Press.

https://doi.org/10.1086/673898 Published online by Cambridge University Press