Functional Attributions and Functional Architecture

A Reply to Axel Kohler

Michael L. Anderson

In his commentary (Kohler this collection) on my target article (Anderson this collection), Axel Kohler suggests that componential mechanism (Craver 2008) in fact suffices as a framework for understanding function-structure relationships, even in complex cases such as direction selectivity in Starburst Amacrine Cells. Here I'll argue that while Kohler is correct that the framework *can* accommodate such cases, this approach misses an opportunity to draw important distinctions between what appear to be different sorts of relationships between functioning systems and the mechanisms in virtue of which they function. I tentatively suggest further that the avenue that one prefers may turn on whether one expects the functional architecture of the brain to be primarily componential and hierarchical (Craver 2008; this collection) or typically more complex than that (Pessoa 2014).

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1 Introduction

In my target article (Anderson this collection), I argued that the complexity of the function-structure relationships that give rise to direction selectivity in Direction-Selective Ganglion Cells Direction-Selective Ganglion Cells (DSGCs) and in the dendrites of Starburst Amacrine Cells Starburst Amacrine Cells (SACs) represent a challenge to componential mechanism as currently formulated (Craver 2008). First, I argued that distinguishing between the system S that exhibits the target phenomenon ψ, and the mechanism M in virtue of which it ψs allows one to paint a more nuanced picture of the various ways entities can be organized so as to give rise to observed function. Second, I suggested that the function-structure relationships in these particular cases appeared to violate the bottom-up hierarchical assumptions at the center of the componential mechanistic framework, which requires that the components of M in virtue of which a system exhibits ψ are at a lower level of organization than S. In the cases under

discussion, I argued that some parts of the mechanism in virtue of which SAC dendrites function are at a *higher* level of organization than the dendrite, and that parts of the mechanism in virtue of which DSGCs function are at the *same* level. Moreover, I noted that in neither of these cases were all the entities that constituted M constitutive parts (components) of S.

To accommodate such cases, I recommended extending the notion of mechanistic *constitution* with the notion of an *enabling constraint*: mechanisms, we should say, enable function in systems by changing the relative probabilities of functional outcomes of activity in S. I suggested that this change would allow us to more accurately characterize the variety of structure–function relationships in the brain (and in other complex systems). However, in his commentary on my article (Kohler this collection), Axel Kohler argues that such an extension is unnecessary, for in fact the componential mechanistic framework of Craver and Bechtel (Craver 2008; Craver & Bechtel 2007) can accommodate these cases.

Kohler is correct. The extension is strictly speaking unnecessary, and componential mechanistic explanation can offer one plausible characterization of function-structure relationships in these cases. In fact, it is probably the case that *no* example or set of examples *ever* forces one to give up on an explanatory framework (certainly not one as well-motivated and useful as componential mechanism). What examples such as these *can* do, however, is illuminate the potential *advantages* of a new approach, and I would like to use the opportunity offered by this reply to reiterate what I take some of those advantages to be.

2 Three possible system-mechanism relationships

In my target article (Anderson this collection) I suggested that once one distinguishes between the system S that ψs and the mechanism M in virtue of which it does so, it is easy to see that there are three possible relationships between M and S. First, the components of M can all also be components of S, such that M is a relevant sub-component of S. Let's call this relationship R1. A relationship of type R1 obtains between the drive-train of an automobile and the automobile as a whole. Second, (R2), M and S can be identical. I can't think of an uncontroversial example of this relationship, and imagine that such a case is relatively rare. Third and finally, (R3), M and S can cross-cut in various ways, sharing some but not all of their parts. In my view, for instance, it is the neuron the fires an action potential, but not all of the entities that comprise the mechanism for generating action potentials are also part of the neuron. For example, the ions in the extracellular fluid that are crucial for establishing the membrane potential are not part of the neuron, although they are clearly part of the mechanism. Similarly, I argued in my target article that in the case of direction-selectivity in SAC dendrites, although it is the dendrite itself that is directionally selective, many of the parts of the relevant mechanism are not in fact parts of the dendrite. Moreover, in the case of DSGCs, the cell and the mechanism in virtue of which it is direction-selective share at most *one* part: the synapse between the SAC dendrite and the DSGC.

One advantage of making these distinctions, I believe, is that it allows one to see quite clearly when top-down constraints are responsible for function, as I argued is the case for direction selectivity in SAC dendrites. But Kohler suggests that appearances may be misleading here. In fact, he argues, we should "reconstitute the phenomenon" by recognizing that the relevant direction-selective system is *not* the SAC dendrite, but is rather the dendrite + the non-dendritic elements of the mechanism, including other SACs. This larger system can be then be treated within the standard framework of componential mechanism. We can call this approach to addressing these sorts of cases "the Kohler strategy".

As I noted in my target article, the Kohler strategy is certainly open to the mechanist. It does, however, have the following effects. First, it tends to make the systems of the brain to which functions are attributed relatively *larger* and more diffuse, which arguably reduces preci-

sion. Second, it would in effect turn all apparent instances of R3 into instances of R2.[1] I noted above that I thought the class of R2 would be small. If I am right about the prevalence of R3 functional relationships in the brain, then this strategy would make R2 very large. But it would do so essentially by legislation, as a way of preserving the universal applicability of the componential mechanist framework. How forced this appears will depend on how closely one believes the guiding assumptions of that framework match the architectural facts of the brain. We will return to this last point after reviewing some of the considerations that appear to favor the Kohler strategy.

3 Motivations for the Kohler strategy

Kohler maintains that actual scientific practice in fact supports the Kohler strategy. Exhibit A in his argument is a recent article (Kim et al. 2014) detailing part of the mechanism for visual motion detection. Kohler reproduces a figure depicting their model, and argues that the inclusion of the distributed network in the model suggests that the authors are strictly speaking attributing function to the whole system as depicted:

> Although it is true that investigators sometimes refer loosely to local elements as displaying a certain characteristic, the corresponding detailed and extended accounts of direction selectivity give credit to the distributed nature of the relevant systems that figure in explanations. (Kohler this collection, p. 6)

I agree that this is one possible interpretation of the practice. But here is another: these scientists are distinguishing between the system that exhibits the phenomenon and the mechanism that produces it, and are open to different sorts of relationships between them. Consider the following from the paper Kohler discusses:

> Research on [the visual detection of motion] has converged upon the SAC. An SAC dendrite is more strongly activated by motion outward from the cell body to the tip of the dendrite, than by motion in the opposite direction. *Therefore an SAC dendrite exhibits DS*, and outward motion is said to be its 'preferred direction'. Note that it is incorrect to assign a single such direction to a SAC, because each of the cell's dendrites has its own preferred direction. DS persists after blocking inhibitory synaptic transmission, when the only remaining inputs to SACs are BCs, which are excitatory. As the SAC exhibits DS but its BC inputs exhibit little or none, *DS appears to emerge from the BC–SAC circuit*. (Kim et al. 2014, p. 331; emphases added)

Far from seeming loose, the attribution of direction-selectivity to the dendrite appears to me clear and precise. Moreover, note that in the final sentence quoted above, the attribution of direction-selectivity to the cell is reinforced, even in the context of a reference to the mechanism as the "BC-SAC circuit". Indeed, I would argue it is natural and permissible to gloss the last clause in the following way: "DS *in the dendrite* appears to emerge from the BC-SAC circuit." On this reading, of course, the authors of this article would be proposing an R3 functional relationship such that parts of the mechanism are on a higher level of organization than the system exhibiting the phenomenon.

That these authors are open to R3 relationships of various sorts appears to be reinforced by a line later in the paper:

> Previous research suggests that On–Off direction-selective ganglion cells *inherit their DS from SAC inputs* owing to a strong violation of Peters' rule. (Kim et al. 2014, p. 335; emphasis added)

Here again we see the same pattern: a clear attribution of direction-selectivity *to the DSGC* in

1 Actually, there are some questions here, for there seem to be *obvious* instances of R3 with which the mechanist is and should be entirely comfortable, e.g., the neuron and the mechanism of the action potential. So presumably this strategy would be employed *only* when the relationship appeared to violate the "lower-level entity" constraint. I've not the space to pursue this further here, so will note only that *selectively* pursuing the Kohler strategy would need separate justification.

the same sentence as a reference to the distal mechanism (the SACs), in the context of what is obviously an R3 relationship between system and mechanism. Thus, while I agree that the Kohler strategy is viable, I don't see that consideration of scientific practice forces us to adopt it, or even necessarily favors it.

So what might be other reasons for adopting the Kohler strategy over extending mechanism to include enabling constraints? As I mentioned at the end of the previous section, the matter might come down to how closely one thinks the architectural facts about the brain match the guiding assumptions of the componential mechanist framework. If one expects that the brain is at root a decomposable or nearly-decomposable system of well-defined interacting components, then componential mechanism does indeed seem like a very appropriate framework for capturing at least the majority of its functional relationships (with the few exceptions to be dealt with perhaps as secondary elaborations or special cases). If, however, one takes seriously the notion that the brain is a massive network marked by multiple, nested, cross-cutting, dynamic hierarchies interacting in bottom-up, top-down, feed-forward and feedback fashions (Pessoa 2014), then one might wish for some of the explanatory flexibility that the notion of enabling constraints appears to offer. I, of course, am in this latter camp (Anderson 2015).

4 Conclusion

As Kohler correctly points out, it is possible to accommodate these complex cases of function-structure relationships within the componential mechanistic framework, by reconstituting the phenomenon and ascribing function to the whole mechanism that produces it. I have tried to indicate what I think some of the costs are to the Kohler strategy, including an apparent conflation of R2 and R3 functional relationships and a potential loss of grain in our ascriptions of function to structure. For some, paying these costs will be preferable to the proposed alternative, which might appear to require the admission of spooky top-down causes into our ontology.

For those who instead want to maintain the greater attributional specificity that appears to conform to scientific discourse, and in the current case to explain direction selectivity *in the SAC dendrite*, then I would argue that the most promising strategy is to recognize the ways in which functional parts (including networks) can impose constraints on other functional parts, at whatever relative level of organization. Adopting this strategy will of course focus attention on the nature of these constraints, whether bottom-up, top-down, or synpedionic. I would hope that the careful study of such R3 relationships as those showcased here would result in a better understanding of the varieties of causal interactions in complex systems.

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