Carving the Brain at its Joints

A Commentary on Michael L. Anderson

Axel Kohler

When neuroscientists explain the biological basis of a phenomenon of interest, they usually try to identify the parts of a system that seem do the relevant job, and propose a model of how those parts interact to produce the phenomenon. This mechanistic framework of explanation is widely used and has been investigated from a philosophical point of view by different authors. In his target article, Michael Anderson poses a challenge to the currently dominant version of mechanistic explanation as advocated, e.g., by Carl Craver. Taking empirical results and explanatory models from studies on retinal starburst amacrine cells as a starting point, Anderson suggests that the current framework for mechanistic explanation should be extended to include a differentiation between systems and mechanisms, which would allow more leeway in understanding processing in the nervous system. Mechanisms can then be seen to provide enabling constraints on the functioning of systems, where the mechanisms do not need to be subsumed under the system and do not even have to be on the same organizational level. Although Anderson's proposal is interesting and worth exploring, I am unconvinced that this extension conforms to real-world explanatory practice and/or is necessary for accommodating the understanding of direction-selectivity in the retina. I examine another sample of research on starburst amacrine cells, where the integration of empirical data and computational models shows that, on close inspection, it is distributed networks to which certain characteristics are ascribed—a situation that can be handled with the available tools of mechanistic explanation.

Keywords
Constitution | Direction selectivity | Enabling constraint | Enabling constraints | Mechanism | Mechanistic explanation | Motion processing | MT | Neuroscience | Neuroscientific explanation | Starburst amacrine cells | Top-down causation | V1

Commentator

Axel Kohler
axelkohler@web.de
Universität Osnabrück
Osnabrück, Germany

Target Author

Michael L. Anderson
michael.anderson@fandm.edu
Franklin & Marshall College
Lancaster, PA, U.S.A.

Editors

Thomas Metzinger
metzinger@uni-mainz.de
Johannes Gutenberg-Universität
Mainz, Germany

Jennifer M. Windt
jennifer.windt@monash.edu
Monash University
Melbourne, Australia

1 Introduction

One of the dominant frameworks of explanatory practice in the neurosciences and the biological sciences in general is the model of mechanistic explanation proposed in its modern form by Bechtel & Richardson (1993) and recently extended by Carl Craver (2007). Mechanistic explanations describe entities and activities that together bring about a phenomenon of interest (Machamer et al. 2000). When we are interested in how vision works, for example, we try to localize the relevant parts of the brain, and identify components and their types of interactions in order to understand how we can see things (Bechtel 2008). This model of mechanistic explanation is thought to capture the dominant explanatory practice in the biological sciences (Bechtel & Richardson 1993), but normative claims are also made with respect to the adequacy of explanatory accounts. Craver (2007) proposes a number of constraints on constitutive mechanistic explanation in order to decide whether a mechanistic model is viable or not.

In his target article, Michael Anderson (this collection) takes current models of mechanistic explanation as a starting point for proposing an important extension of the existing accounts. In previous models, the system that

exhibits a phenomenon and the mechanism that explains the phenomenon were not separated. Sometimes parts of the system can be screened off with respect to the phenomenon at hand. The windshields of a car and its radio components are not really important in order to understand how it drives, for example. It's fine to say that the whole car drives, but that only the relevant components (engine, axles, tires) are doing the mechanistic work. Focusing on the essential components of a mechanism within a larger system is unproblematic. But Anderson worries about more complex cases in the neurosciences where the system displaying a phenomenon does not encompass the relevant mechanism producing the phenomenon and might not even be on the same level of description as the mechanistic components.

Anderson wants to demonstrate that componential constitution is not sufficient as a model of mechanistic explanation for the processing of directional selectivity in the retina. Mechanisms computing direction of motion are already available at the earliest stages of the visual hierarchy. The vital components of direction selectivity in the retina could be identified. In particular, in recent discussion starburst amacrine cells (SAC) have been viewed as a mechanistic substrate of motion processing. The SACs receive input from bipolar cells, which are not themselves directionally selective, and provide output to direction-selective ganglion cells (Zhou & Lee 2008). The SACs themselves seem to be the core component for retinal motion selectivity (Park et al. 2014; Yoshida et al. 2001).

Examining the current models of how direction selectivity is created in SACs, Anderson takes note of a discrepancy between how direction selectivity is mechanistically achieved and to which parts it is ascribed. He argues for a distinction between the system S that Ψs (that is, exhibits direction selectivity) and the mechanism M that accounts for S's Ψ-ing. For the case at hand, the SACs themselves or even just single dendritic compartments of SACs Ψ, but a much broader network of neighboring SACs and bipolar cells needs to be considered in order to provide a mechanistic account of SAC direction selectivity. Anderson proposes this distinction as an important extension of Craver and Bechtel's model of mechanistic explanation. This has two major advantages, according to Anderson: (1) there can be entities and actions that play a role for M, but are not necessarily parts of S. This allows a certain flexibility in defining the system that displays Ψ, while at the same time including all relevant components in the mechanistic account of S's Ψ-ing. (2) But if there are parts of M that don't need to be spatially subsumed under S, neither do they need to be at a lower level than S. So even the requirement of componential constitution might be relaxed to allow for higher-level mechanistic components that play an important role in S's Ψ-ing.

As an alternative account of the relationship between mechanisms M and the respective systems S, Anderson proposes that M acts as an enabling constraint on S:

> [A]n enabling constraint is a relationship between entities and/or mechanisms at a particular level of description and a functional system at the same or a different level, such that the entities/mechanisms bias (i.e., change the relative probabilities of) the outcomes of processing by the system. (this collection, p. 12)

In the case of retinal direction selectivity, the mechanistic interaction between neighboring SACs and BCs acts as an enabling constraint for the direction selectivity of a specific SAC dendritic compartment (i.e., the system).

The most straightforward move by proponents of existing models of mechanistic explanation, as Anderson (this collection) also notes, would be to claim that the differentiation of system and mechanism is vacuous. Only the mechanism as a whole can do the work. Even in complex cases, one just has to pick out the right subparts of the network (specific synapses, specific compartments of neurons) that together produce the phenomenon of interest. Anderson provides a number of arguments against this way of extending the concept of mechanism/system, which I would like to briefly summarize:

1. Neuroscientists just don't talk about complex directionally selective networks, but about the direction selectivity of certain dendritic branches.
2. The mechanism as a whole does not display a specific direction selectivity (it is not rightward-selective etc.), it only contributes to the specific selectivity in the respective SAC dendrites. The mechanism contributes to different kinds of selectivities in different dendrites.
3. Making fine-grained distinctions between subparts (synapses, axon branches, dendrites etc.) of the very same neurons that contribute to different directional selectivities is implausible.
4. When the whole network is said to be direction-selective (i.e., it Ψs), what about the dendrite itself? Is it supposed to only signal direction selectivity (signal Ψ-ing)? It is unlikely that a clear distinction between Ψ-ing and signaling Ψ-ing can be made.

The aim of this commentary is twofold. First, I would like to argue that the described cases can be handled by current models of mechanistic explanation when one considers the options of reconstituting the phenomena and top-down causation. Second, using another example of research on SACs, I would like to show that the straightforward ascription of direction selectivity to the SAC dendrites is at least debatable. When looking at how empirical results are often integrated with computational models of direction selectivity, it becomes clear that those phenomena can only be understood by considering the distributed nature of the involved networks.

2 Reconstituting the phenomena and top-down causation

Anderson proposes a separation between systems and mechanisms. No matter whether the system is constrained to be a dendritic compartment or whether it is extended to encompass all mechanistically relevant parts, there are tools available to describe the respective situation. The mechanistic model does not necessarily consider systems in isolation from the environment or surrounding processes. Even if the system is defined as the dendrite only, factors influencing dendritic processing as well as the embedding of the system in the overall economy, its organization, have to be considered in order to arrive at an understanding of the system's functioning (Bechtel 2008, pp. 148–150). On the other hand, I would like to argue that we have good reason to extend the boundaries of the system to encompass all the contributing parts. This is a situation in which the original ascription of a function to a system part has to be revised to accommodate new findings. This process is termed *reconstituting the phenomena* by Bechtel & Richardson (1993). Although direction selectivity was thought to be bound to or even intrinsically generated in SAC dendrites, it turns out that the system can only be understood in combination with other neural elements that vitally contribute to the mechanism in question.

One advantage that Anderson suggests comes with the differentiation of system and mechanism is that mechanistic components can then be set at a different level of organization than the relevant system. The SAC dendrite is at a lower level compared to the input from bipolar cells and the network structure (bipolar cells and neighboring SACs) that enables SAC direction-selectivity. But once the question of how exactly we should carve up the brain into systems and mechanisms has been answered, I don't think that complex inter-level relationships are much of an issue for mechanistic accounts. They can be easily accommodated within the framework of top-down causation proposed by Craver & Bechtel (2007). They suggest that any reference to inter-level interactions can be analyzed in terms of within-level causal relationships between parts of entities, where parts and entities are related in a constitutive fashion and entities can be located on different levels. Emphasizing the fact that complex inter-level interactions often need to be considered in order to offer adequate explanatory accounts in neuroscience is important, but it is not outside the scope of current models of mechanistic explanation.

3 Systems and mechanisms for direction selectivity

Since the processing of direction selectivity in the retina is currently a very active research field, there is substantial controversy concerning the relevant entities and activities that contribute to the mechanism, as Anderson points out in his target article. Some accounts focus on local processes within the SAC dendrites themselves (Hausselt et al. 2007), while others draw a broader picture of a multi-component process, where the exact arrangement of cell types and their compartments is vital for direction selectivity (Lee & Zhou 2006). For our purposes here, I would like to use a most recent update on SAC function offered by the group working with Sebastian Seung. The group uses high-resolution electron-microscopy images of brain tissue to reconstruct complete brain networks on a cellular level. Apart from trained reconstruction experts, the project also makes use of so-called "citizen neuroscientists"—volunteers who contribute to the reconstruction process through an online platform that employs gaming features to guide and motivate the community effort (http://www.eyewire.org).

In their study, Seung and colleagues used images from the mouse retina to analyze SAC circuitry. They took a closer look at the exact wiring between bipolar cells (BCs) and SACs (Kim et al. 2014). BCs provide input to SACs, but do not show any directional selectivity by themselves. The main point of the article is to show that different BC subtypes display different patterns of connectivity with SACs. By analyzing branch depth and contact area, they could show that one subtype (BC2) has mainly connections close to the soma, while another subtype (BC3a) has more connections far from the soma in the outer parts of the dendrites. Importantly, the BC subtypes, in turn, have different intrinsic visual response latencies. BC2 seems to lag BC3a by 50ms and more. It can be shown that the differential connectivity patterns and the divergent latencies add up to produce selectivity for a preferred direction of movement going out from the soma on the respective dendrite in accordance with empirical results.

What is important about the paper is not just the main result itself. Any empirical observation may be overruled in the (near) future. So it is not particularly relevant whether these exact cell types and this exact type of wiring is vital for the phenomenon at hand. What I found intriguing in this study, however, was how the relevant mechanism was described and how the data were integrated with a computational model of direction selectivity, reflecting a recent trend in the neurosciences to combine biological and computational perspectives in explanatory accounts. It shows how neuroscientists pick out the relevant parts of a system that contribute to a specific phenomenon in question. The proposed computational model (Fig. 1a; Kim et al. 2014) maps the biological entities onto specific parts of the computational circuit. The output element at the lower part of the figure is the SAC. The input stems from BC2 (left) and BC3a (right); their respective response properties are captured as delay values and sustained vs. transient response types. The circuit combines elements of classical models of direction selectivity, the Reichardt (Fig. 1b) and the Barlow-Levick detectors (Fig. 1c). Clearly, the direction selectivity cannot be attributed to any one of the system components in isolation. Mechanistic accounts and the corresponding computational models both point to the whole complex of entities as the relevant system that achieves directional selectivity.

In its computational abstraction, the model can be thought of as a canonical system of directional selectivity. Similar models have also been applied to different hierarchical levels of neural processing and different species. For example, mechanisms of directional selectivity have been studied for a long time in the fly visual system. With very different neural elements and wiring, a system of interconnected neurons achieves directional selectivity with response properties closely resembling the Reichardt-type of motion detector (Borst & Euler 2011). Again, only the combination of elements from different processing stages succeeds in delivering direction selectivity as a system. On a cortical level, direction selectivity has been first described for complex cells of the

primary visual cortex (V1) in the seminal work of David Hubel & Torsten Wiesel (1962). Without offering a quantitative computational model, they nevertheless suggest a hypothetical connectivity pattern between different cell types that might underlie the observed responses to moving patterns in complex cells (Hubel & Wiesel 1962, Fig. 20). The model shares features with other motion detectors; a mapping between components is possible.

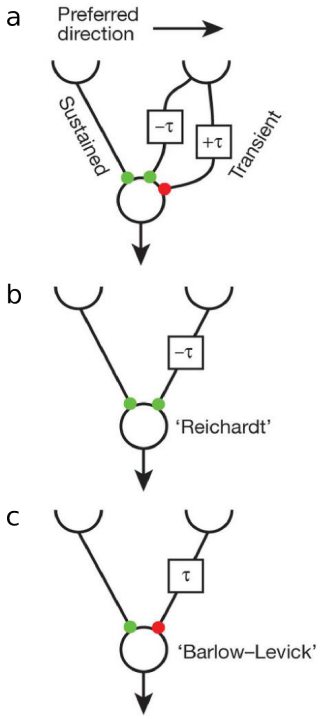

Figure 1: Computational models of direction selectivity (a) The selectivity of SACs described in Kim et al. (2014) can be modeled with a computational framework using a combination of sustained and transient response properties as well as excitatory and inhibitory connections. The displayed wiring would lead to direction selectivity for rightwards motion. The proposed model can be considered to combine previous classical models of direction selectivity, the Reichardt detector (b) and the Barlow-Levick model (c). Green dots indicate excitatory and red dots inhibitory synapses. '-τ' indicates a temporal lead and '+τ' a temporal lag. Reprinted by permission from Macmillan Publishers Ltd: Nature (Kim et al. 2014), copyright (2014).

When it comes to motion selectivity in the brain, one of the most intensively studied cortical areas is the middle temporal (MT) region. The region was first described in the macaque (Dubner & Zeki 1971; Zeki 1974) and owl monkeys (Allman & Kaas 1971). The human homolog, the human MT complex (hMT+; Tootell et al. 1995; Zeki et al. 1991), turned out to be a collection of areas with related response properties (Amano et al. 2009; Kolster et al. 2010). Again, to understand the direction selectivity of MT, it is necessary to consider the cooperation of cells in MT and the input processing stages, mainly from V1. This cooperation and the need for an integrated perspective is emphasized in empirical studies (Saproo & Serences 2014) as well as computational models of MT functioning (Rust et al. 2006). Only the V1-MT system as a whole is understood to deliver motion selectivity as output of the MT stage.

But in terms of the role of MT in motion processing, a case could be made in support of Anderson's suggested distinction between a system that exhibits a certain selectivity and the mechanism that produces this selectivity. The apparent locality and modularity of motion processing in MT is based on very selective deficits in patients with lesions in and around MT (Zeki 1991; Zihl et al. 1983). And stimulation of MT with transcranial magnetic stimulation (TMS) in healthy participants leads to selective deficits in motion perception (Beckers & Hömberg 1992; Beckers & Zeki 1995; Hotson et al. 1994; Sack et al. 2006). In a recent study, patients undergoing brain surgery near MT could be investigated with electrical stimulation (Becker et al. 2013). Only stimulation of MT and a related area nearby, MST, led to an inability to perform a simple motion-detection task, a rather specific result concerning the relevance. Results of that kind drive the intuition that the system that is responsible for motion perception, independent of any cortical areas that might mechanistically contribute to the processing chain leading up to MT (like V1), are localized in MT.

Lesion and other interference studies (e.g., with TMS) are suggestive, but there are also well-known difficulties with interpreting the results. Lesions mostly affect larger parts of the brain and are rarely limited to a single cortical site. As such it is often hard to identify the actual parts of the complex brain networks that

are affected. The advantage of stimulation techniques is that the interference is temporary and can be precisely targeted on a specific location. But, given the rich connectivity structure of neural networks, stimulation effects can be seen even in remote target sites (Bestmann et al. 2004; Sack et al. 2007). In addition, TMS studies have shown that activity of MT might not even be sufficient for conscious motion perception without the involvement of V1 (Pascual-Leone & Walsh 2001; Silvanto et al. 2005). There are also further empirical as well as philosophical reasons for rejecting the claim that motion perception can be attributed to MT in a stringent fashion (Madary 2013), which I won't discuss here.[1]

So while at first glance MT is a very strong candidate for straightforward and very local attribution of function, it seems again that the relevant system is more appropriately described on a network level. The tendency to see system parts as vital for a function may also stem from the limitations of our employed methods. Lesion cases and interference techniques are commonly interpreted as being informative about the relevant gray-matter structures that are affected by the lesion or stimulation. But there is evidence that interference with white-matter connections between network parts can be even more incapacitating than gray-matter damage. It has long been known that frontoparietal areas are implicated in a deficit of visuospatial attention called *neglect*. But very recently Thiebaut de Schotten et al. (2005, 2011) revealed that the properties of fiber connections between frontal and parietal sites are most predictive of visuospatial processing capacities, and that their electrical stimulation leads to severe deficits. Transferring this insight to the case of MT, we simply have most direct access to the cortical gray-matter centers involved in motion processing, and since they are vital components of the system, this also leads to corresponding deficits when they are affected or stimulated. But this might conceal the fact that motion selectivity is a product of a wider network that crucially depends on integrated processing for proper functioning.

In sum, I think that close inspection of how direction selectivity is investigated and treated in neuroscientific research is in disagreement with Anderson's arguments (1) and (3). 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. Even considering the case of conscious motion perception, it is unclear whether the presumed locality of motion representation stands up to stringent tests. Rather, it seems to be a case of localized interference with a distributed system where damage to vital hubs leads to fundamental deficits.

4 Conclusion

In this commentary, I have defended the claim that the current tools of mechanistic explanation are sufficient for accommodating the explanatory goals in current neuroscience, particularly in the special case of direction selectivity in the retina and other neural systems. A closer look at explanatory practice shows that, in representative cases of empirical research, models of direction selectivity have to take a number of components in a distributed network into account in order to provide a full-fledged description of the relevant processes. On the philosophical side, the conceptual tools of "reconstituting the phenomena" (Bechtel & Richardson 1993) and "top-down causation" (Craver & Bechtel 2007), offered by existing models of mechanistic explanation, might be sufficient for capturing the problematic cases to which Anderson (this collection) points.

On the other hand, Anderson's proposal (this collection) to extend existing models of mechanistic explanation with the notion of enabling constraints is very interesting and might offer an avenue to more nuanced mechanistic

1 Madary (2013) uses two sets of empirical results to show that representation of motion cannot be ascribed to MT simpliciter. One is the recent emphasis on spontaneous activity making significant contributions to the state of sensory systems—they add content referring to the attentional or sensorimotor state of the organism to input-derived sensory representations. The other demonstrates that in MT specifically, the response properties of cells can be quite variable and are not consistently related to perceptual content only.

descriptions of systems in their contextual embedding. In almost all relevant cases in neuroscience research, there are various external factors influencing the workings of a system, and it is often difficult to draw clear boundaries between vital and non-vital, but nevertheless highly influential system components. Anderson's framework would offer a viable solution for handling those modulatory constraints. Resolving this debate will also depend on a clear conception of how the entities that display a certain phenomenon are best identified and described.

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