European Journal for Philosophy of Science (2023) 13:27
https://doi.org/10.1007/s13194-023-00530-z

PAPER IN GENERAL PHILOSOPHY OF SCIENCE

Mechanisms and the problem of abstract models

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Received: 8 March 2022 / Accepted: 11 May 2023 / Published online: 20 June 2023

Abstract

New mechanical philosophy posits that explanations in the life sciences involve the decomposition of a system into its entities and their respective activities and organization that are responsible for the explanandum phenomenon. This mechanistic account of explanation has proven problematic in its application to mathematical models, leading the mechanists to suggest different ways of aligning abstract models with the mechanist program. Initially, the discussion centered on whether the Hodgkin-Huxley model is explanatory. Network models provided another complication, as they apply to a wide number of materially diverse systems. In this article, we examine the various attempts to integrate abstract models within the mechanist program, also presenting a further challenge: the Heimburg-Jackson model, which was introduced as an alternative to the Hodgkin-Huxley model. We argue that although the notion of abstraction as the omission of irrelevant mechanistic details appears to give a mechanistic solution for accommodating abstract models, this notion does not suit models whose epistemic strategy is not decompositional. As a result, the mechanist has to choose whether to dilute the mechanistic approach nearly beyond recognition or to claim that many, if not most, abstract theoretical models do not deliver mechanistic explanations, or qualify as explanatory at all.

Keywords Scientific models · Mechanisms · Abstraction · Explanation · Hodgkin and Huxley model · Network models · Heimburg and Jackson model · Thermodynamics

This article belongs to the Topical Collection: EPSA21: Selected papers from the biennial conference in Turin

Guest Editors: A.C. Love, C. Marchionni, M. Redei, J. Williamson

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1 Introduction

The extension of the mechanistic account of explanation to modeling has led to a rift in the previously unified field (e.g. Bechtel, 2011, Levy & Bechtel, 2013). The question has been raised of how to incorporate abstract models into the new mechanistic philosophy, which is based on the decomposition and detailed description of real-world mechanisms. In the earlier discussion, mechanists addressed the question of whether the Hodgkin-Huxley model of the nerve impulse is explanatory. This model consists of a system of equations that does not describe the way that ions cross the neuronal membrane, yet it is able to reproduce a wide number of observed features of the nerve impulse. More recently, mechanists have been concerned with network models and dynamical systems models. These various kinds of models appear to challenge the mechanistic ideal of describing mechanisms in a manner that will "account for all aspects of the phenomenon by describing how the component entities and activities are organized such that the phenomenon occurs" (Craver, 2006, 374).

In the discussion of the explanatory character of the Hodgkin-Huxley model, Craver (2006, 2007) suggested that it is non-explanatory since it is only a sketch of a mechanism, whereas Levy (2014) in turn argued that it is explanatory, as it aggregates the contributions of lower-level entities in the variable "current" that the equations describe. Later on, Levy and Bechtel gave a mechanistic reading of Alon's network motifs, claiming that they abstract by omitting mechanistic details in order to "track those features of the system that make a difference to the behavior being explained" (2013, 256). In this article, we concentrate on the notion of abstraction as omission as it features in the mechanistic program. In particular, we argue that it does not succeed, contrary to several mechanists' claims, in accommodating many theoretical mathematical models within the mechanist framework. The problem is that the epistemic strategy of network models, or dynamical systems models, is decidedly non-compositional, and so, claiming that such models abstract from the mechanistic details is beside the point. Furthermore, we also examine a new challenge to the mechanistic interpretation of models: the Heimburg-Jackson model, recently presented as an alternative to the Hodgkin-Huxley model. This phase transition model of the nerve impulse refrains from considering microscopic details and instead uses a coarse-grained description able to exhibit phase transitions. Yet, it cannot be considered a phenomenal model that merely "describes the behavior of the target system" (Kaplan & Craver, 2011, 608) without any explanatory content.

Given that the notion of abstraction as omission does not apply to the aforementioned prominent kinds of theoretical modeling, mechanists appear to have a couple of choices. First, they can claim that one can recover mechanisms underlying the phenomena that the seemingly non-decompositional models seek to study (e.g. Green et al., 2018). Second, they can admit that there are non-mechanistic explanations (e.g. Glennan, 2017), or claim that models that are not decompositional to their core are not explanatory (e.g. Kaplan & Craver, 2011). In trying to render decompositional modeling strategies into the mechanistic mold, the first option runs the risk of diluting the mechanistic approach beyond recognition. In contrast, the second admits, in effect, that the mechanistic approach does not have the resources to address many of the most prominent styles of mathematical modeling. For instance, Glennan's

explanatory pluralism makes space for different kinds of non-causal explanations, while, as we will show below, many mechanists still struggle with the question of how to accommodate abstract models.

The argument proceeds as follows. In Sect. 2, we study mechanists' efforts to account for the Hodgkin-Huxley model and dynamic and network models in mechanistic terms. We then outline the mechanist notion of abstraction in Sect. 3, arguing that while the notion of omitting irrelevant or non-difference-making factors does seem to fit the mechanist ontology, it does not necessarily suit the modeling strategies the mechanists seek to address with it. In Sect. 4, we discuss the challenge the Heimburg-Jackson model offers to the mechanist account. The shortcomings of the mechanistic notion of abstraction, related to the difficulties in rendering such models as the Heimburg-Jackson model or the dynamic or network models in mechanistic terms, leads us to conclude that the mechanistic account is not well-equipped to deal with many abstract models. It will lose its distinctive decompositional core if it is extended also to cover modeling strategies that do not approach a phenomenon as produced by components, their properties, activities, and their organized interaction.[1] We conclude in Sect. 5 by calling on the mechanistic program, which started with the intention of giving a both normatively and descriptively adequate account of explanation, to pay more attention to the actual modeling practices.

2 The mechanistic accounts of mathematical and computational modeling

Although the new mechanical philosophy was originally presented in the context of scientific explanation in the life sciences, the mechanist philosophers soon also addressed modeling (e.g. Glennan, 2005; Craver, 2006; Darden, 2007). One central question concerned the status of models within the mechanist account, given its commitment to "ideally complete mechanist explanation" (Boone & Piccinini, 2016, 691) and the observation that in many mathematical models "[i]nstead of properties of specific parts or operations, the focus is on the *relational* structures and patterns and their implications for system behavior" (Green et al., 2018, 1773). In the mechanist discussion of modeling, one can discern two partially overlapping phases that address different kinds of models. While much of the earlier discussion centered on the renowned Hodgkin-Huxley model, the focus of the more recent discussion has shifted to network science and its applications to systems biology and neuroscience. Interestingly, as we will explain below, most of these discussions have converged on the question of abstraction in modeling, and how it could be accommodated within the mechanist framework. Several arguments to that effect have been presented: denying the explanatory value of abstract models, or claiming that they make use of aggregation, recomposition, or omit non-difference makers. We will consider each of these proposals in turn.

[1] We limit our analysis to mechanisms in the life sciences that was the original context of the mechanistic framework. We do not consider e.g. mechanisms in the social sciences that appear to employ a different notion of a mechanism (see Kuorikoski, 2009).

2.1 The Hodgkin-Huxley model: a mechanism sketch or an aggregative abstraction?

The Hodgkin-Huxley model consists of a set of dynamical equations that are able to simulate the empirical recordings these scientists obtained from the giant axons of squid in the late nineteen-forties. This model provided a landmark in mathematical physiology and influenced the agenda of neurophysiology for the subsequent decades. The currently accepted explanation of nerve impulse transmission builds upon this model, providing an explanation of the nerve impulse voltage wave transmission in terms of voltage-sensitive ion channels that open when the nerve cell is excited, leading to ion currents that change voltage across the axon membrane.

The Hodgkin-Huxley model is based on the analogy of an electrical circuit with the nervous membrane. Based on this analogy, Hodgkin and Huxley were able to obtain equations that establish relations between current, voltage, and ionic permeability at one point in the membrane. The numerical approximation of the solutions to the equations displayed voltage-time curves that are equivalent to empirical recordings in voltage-clamp experiments. Hodgkin and Huxley later used the cable equation to develop an equation for the transmission of the signal that also considers electrical variables. The cable equation was originally used to model telegraph signal decay in underwater transatlantic cables. The Hodgkin-Huxley model then has two versions, a system of four differential equations for modeling the ionic currents at one point in the membrane, and a partial differential equation which describes the transmission of the voltage variations. As the Hodgkin-Huxley model has been extensively discussed in the existing literature, we will only shortly review what we consider to be the main mechanist accounts of the explanatory value of the Hodgkin-Huxley model.

In the context of his more comprehensive mechanistic account of explanation, Craver (2006, 2007) argued that the Hodgkin-Huxley model should not be understood as being derived from the laws of physics, as Weber (2008) had proposed.[2] For Craver, the Hodgkin-Huxley model only amounts to a how-possibly sketch for a mechanistic explanation of the nerve impulse, because it does not give an account of the mechanism by which ions cross the nervous membrane. Based on this fact – recognized by Hodgkin and Huxley themselves – Craver claimed that the explanation of the nervous impulse was not truly given until the proteins that form ionic channels across the membrane were discovered, thereby completing the explanatory sketch. Discrete ion fluxes were first detected in the patch clamp experiments by Neher and Sackmann in the 1970s, supporting the idea of a passive mechanism of ionic transport. In the late 1990s, MacKinnon and coworkers crystallized the protein making up the potassium ion channel. The combination of these results supports the hypothesis that the voltage-sensitive protein-ion-channels change the permeability of the membrane during a nervous impulse. According to Craver, it was only at this stage that a *complete mechanistic explanation of the nerve impulse* was delivered.

[2] Weber (2008) zoomed in on the role of physical laws in the development of the Hodgkin-Huxley model. In his view, the Hodgkin-Huxley model explains in the same way as many physical explanations do: by entwining the experimental regularities and general physical laws (that are invariant under some interventions). Bogen (2008), in turn, pointed out the importance of analogical reasoning in the derivation of the HH equations.

In sum, for Craver, the Hodgkin-Huxley model was not yet explanatory in 1952, since in order for mechanistic models to explain they would need to "account for all aspects of the phenomenon by describing how the component entities and activities are organized such that the phenomenon occurs" (Craver, 2006, 374). Let us mention here, in anticipation of our discussion below, that many recent accounts of explanation do not suppose that the explanatory value of a model depends on how complete and detailed it is.[3]

If the Hodgkin-Huxley model were only a how-possibly mechanism sketch in view of its incompleteness, the question would be whether any mathematical model could qualify as a how-actually model (e.g. Knuuttila & Loettgers, 2013). After all, mathematical models are typically simplified, idealized, and approximate in character. Glennan (2005) recognized this problem early on and instead followed Giere in suggesting that models are neither true nor false, but rather similar or dissimilar to their target in some respects and to certain degrees. According to Glennan, models need not be complete nor accurate to be a part of mechanistic explanations, it is only required that some parts of the model pick up the elements that will allow scientists to reconstruct the (complete) mechanistic explanation.

Levy (2014) also argued against the completeness requirement by considering the Hodgkin-Huxley model as well. He claimed that the explanatory achievement of the Hodgkin-Huxley model is precisely due to its *abstract* character. Because the Hodgkin-Huxley model abstracts from the individual movement of ions, it could account for the ionic currents even though the mechanism of ion transport was yet unknown. For Levy, the contribution of the model is due to its characterization of regularities at such an *aggregate level*: "the discrete-gating picture relates whole-cell behavior to events at a lower level via aggregation: the system's total behavior is the sum of the behaviors of its parts" (Levy, 2014, 15). He goes on to explain that such "aggregative abstraction" could be "truer to the mechanistic ideal, because it explains the relationship between lower-level mechanisms and higher-level ones" (20).[4]

Indeed, the question of abstraction became central to the subsequent mechanist discussion of modeling, seeking to give a mechanistic interpretation of dynamic and network models in terms of *recomposition* and *omission*. The notion of *aggregation* does not feature in these discussions. While the notion of recomposition, in line with the notion of aggregation, still explicitly invokes the idea of (de)composability as the cornerstone of (any) mechanistic account, abstraction as omission does seem to suggest that the specificities of lower-level components and activities could be abstracted away such that only their organization is left (Levy & Bechtel, 2013). However, as we will show, at the bottom of the idea of abstraction as omission, at least in the hands of mechanists, is still the ontology of parts, their activities, and organization, on different levels that do not suit the epistemic strategies of many modeling practices.

[3] See Bokulich (2017) for an extensive discussion of recent accounts of explanation.

[4] In our view, the model showed that the voltage variation could *in principle* be explained by the ionic currents, but it did not explain the relationship between the level of current and the level of individual ions.

2.2 Dynamic and network models: recomposition and omission

Before the focus on abstraction, another mechanist strategy for accommodating mathematical and computational modeling was tested: the idea of recomposition. In a series of articles discussing the molecular networks underlying the circadian clock, Bechtel and Abrahamsen argued that the mechanistic account should be extended to cover dynamic mechanistic explanations (e.g. Bechtel & Abrahamsen, 2009; Bechtel, 2011). According to them, mathematical and computational models addressing the non-linear dynamic phenomena characteristic of cyclic biological organization deliver such explanations. The "basic mechanistic account", Bechtel observes, is unable to deal with cyclic phenomena due to its focus on the "sequential execution of qualitatively characterized operations" (Bechtel, 2011, 533). Consequently, Bechtel and Abrahamsen suggested that the earlier mechanistic focus on decomposition should be supplemented with that of recomposition. Such recomposition would frequently amount to constructing "systems of differential equations in which variables and other terms correspond to properties of the mechanism's parts and operations" (Bechtel, 2011, 533). As an example, Bechtel and Abrahamsen discussed circadian clock research in which researchers have successfully isolated several clock genes and their protein products, whose complex interactions produce circadian rhythms in various model organisms. However, as Knuuttila and Loettgers (2013) have shown, this decompositional experimental program does not easily translate into mathematical models of genetic circuits. Such models are general, and based on formal templates imported from other disciplines, notably from physics and engineering.

Instead of recomposition, Levy and Bechtel (2013) invoke the notion of abstraction to give a mechanist reading to the research on "network motifs" in systems biology. Here again, the case discussed seems to be amenable for rendering mathematical and computational modeling under the guise of the mechanist agenda. Levy and Bechtel discuss the work of systems biologist Uri Alon, who has used graph theory to extract network motifs from data on transcriptional regulation in the *E. coli* bacterium. Network motifs are "basic interaction patterns that recur throughout biological networks, much more often than in random networks."[5] An example of such a motif is a feedforward loop depicted in Fig. 1, where X and Y represent transcription factors that bind to the receptor Z. The feed-forward loop is for Alon one of a supposedly small set of basic building blocks ("motifs") of which more complicated transcription networks are built (Alon, 2007). Levy and Bechtel consider network motifs as abstractions that represent "the pattern of causal connections among elements of a system" (Levy & Bechtel, 2013, 242) by distinguishing "those underlying factors that matter from those that do not" (256).

Based on this interpretation of network motifs, Levy and Bechtel mount an attack against Machamer et al. (2000), who consider abstractions as mechanism schemas. Such schemas lie between sketches and how-actually explanations and are to be filled in with details of entities and their operations as scientific research progresses. Levy and Bechtel argue that this "is a mistake; abstraction both serves the virtue of identifying the relevant causal organization and facilitates generalization" (2013, 258). The

[5] http://www.weizmann.ac.il/mcb/UriAlon/research/network-motifs.

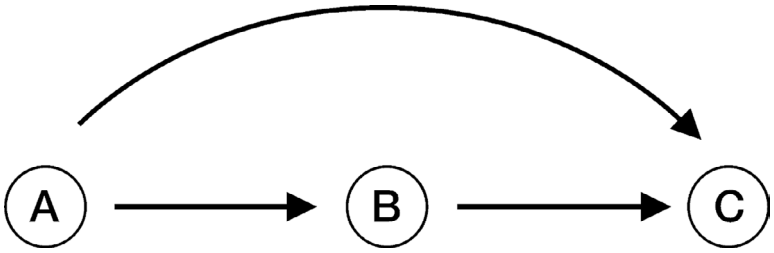

Fig. 1 A feedforward loop motif in which A regulates both B and C, and B regulates C

point they make is that the difference-making feature picked by network motifs is the causal connectivity, i.e. causal organization abstracted from particular components and their behaviors. However, at the same time they believe that "[w]hen a graph has been adequately constructed, *the nodes and edges represent parts and operations in actual mechanisms*" (247, emphasis added).

Given that the origin of the graph theory is in mathematics, and the graphs themselves are highly generic, the crucial question remains: how is one supposed to achieve a graph that would *correspond* to the parts and operations of some actual mechanism?[6] The notion of abstraction as omission may suggest that a representation abstracts away from a more detailed description of a system or mechanism (e.g. Jones, 2005; Godfrey-Smith, 2009; Cartwright, 1989). However, abstraction does not necessarily refer to an actual model-building heuristic of stripping away detail, either mentally or otherwise (Nersessian, 2002; Gallegos Ordorica, 2016; Jones, 2018; Portides, 2021; Carrillo & Martínez, 2023; Levy, 2021) has indeed later argued that the "abstractness of a description has nothing to do with whether it was arrived at via abstraction" (5863-4). Yet, for mechanists, there would still need to be a real-world mechanism including elements and their behaviors underlying the abstract graph. Boone and Piccinini (2016) have spelled out in more detail what they take to be "*legitimate*" kinds of mechanistic abstraction.

3 Mechanistic abstraction

Boone and Piccinini (2016) target the criticisms according to which the ontic conception of mechanistic explanation would require maximal detail.[7] They address what they call "the ideally complete mechanistic explanation" within which "abstraction serves two interrelated roles: (1) to explain a phenomenon at a particular degree of generality, and (2) to identify all and only the components, properties and organizational relations that constitute a mechanism at different levels" (691). As Boone and Piccinini maintain that regularities are explained in the same way as token events, the

[6] It is worthwhile to note that before Alon, network motifs were introduced to study social networks (Holland & Leinhardt, 1976). The motifs' frequency in social networks was contrasted to their frequency in random networks. This origin should make us wary of the idea that network motifs omit underlying mechanistic details of some particular biological systems.

[7] Boone and Piccinini (2016) also discuss the epistemic roles of abstraction, related to scientists' knowledge/ignorance of the target systems, their interests, and the tractability and solubility considerations.

abstraction from the specific to the more general does not pose any particular problem for them. More generic explanations involve the identification of the relevant subset of causal powers that are common to the appropriately organized tokens of relevant types. Since many mathematical models do not denote mechanistic details, Boone and Piccinini's account of abstraction from lower to higher levels of organization seems illuminating concerning the mechanistic assumptions of mathematical modeling (691). They construe it as an ascent from lower to higher levels:

> When we go up a level in our mechanistic explanations (e.g., from neurons to hippocampi), *we abstract away from some of the causal powers of the components in order to single out the specific causal powers that produce the phenomenon at the higher level*. (693, emphasis added)

Consequently, Boone and Piccinini's analysis of mechanistic abstraction relies heavily on isolating relevant causal powers on the one hand, and on the other, the ladder of abstraction that enables ascending and descending between different levels of mechanisms.

How does Boone and Piccinini's account of abstraction fit models whose explanatory strategy appears not to rely on a decompositional strategy of isolating relevant subsets of appropriately organized and causally efficacious components and their properties? They claim, referring to Levy and Bechtel (2013) that in such cases it is the microstructural details that are omitted leaving only the organizational structure.[8] But this seems to us to be a very different kind of abstraction than isolating relevant causal factors at different levels. Furthermore, it is not in line with the actual modeling practices. The application of graph-theoretic tools and concepts does not boil down to isolating difference-making factors for some specific function. The mechanist account of abstraction ignores how an essentially subject-independent mathematical theory can be used to render, in principle, nearly any kind of system in terms of network graphs consisting of nodes and edges. Such nodes and edges are a far cry from the kinds of concrete components, and their properties and interactions that the mechanists were originally interested in, not to mention the important productive dimension of mechanisms that is missing from such graphs! Of course, this fact has not escaped the attention of Levy and Bechtel. In discussing a network motif model of arabinose mechanism, they contend cautiously that "the model *aims* to track those features of the system that make a difference to the behavior being explained" (256, emphasis added).

Due to their being subgraphs of larger networks – of various kinds, and not limited to molecular biology – network motifs may still seem to be susceptible to mechanistic interpretation. However, such a decompositional approach does not apply to the analysis of most large-scale networks. Rathkopf (2018) argues that far from being models of parts and operations, network models do not assume that the systems are

[8] Boone and Piccinini (2016) and Glennan (2017) appear to take Levy and Bechtel's (2013) mechanistic interpretation of Alon's network motifs at face value. We find this somewhat surprising in the case of Glennan, as he is not obliged to give the network motifs a mechanistic reading – in his account, not all explanations that refer to topological properties are mechanistic.

decomposable. It is precisely for this reason that network models are particularly efficient in studying non-decomposable systems, i.e. systems in which "the behavior of any given component part, even over a short period, depends on the behavior of many other individual components" (p. 69). In contrast, the notion of abstraction as omission, according to Levy and Bechtel, appears to suggest that one could in principle have access to the component parts, their properties, and activities, but omit them in favor of concentrating only on organization such that one could nevertheless later on reconstruct or recover the mechanistic componential details.

In networks, however, neither the mechanistic details nor their omission plays an epistemic role, but rather the mathematical properties of the network (e.g. the average path length and clustering coefficient). For instance, Huneman (2015) and Kostić (2018) claim that in network science, the topology of a model and its mathematical consequences are explanatory instead of the entities and their activities at the local level. Topological explanations address global "higher-level" structures, and as a result, they can be realized by a great variety of systems differing from each other in material and micro-structural features. Consequently, network explanations do not abstract from the details of a more complex phenomenon, but rather adopt a strategy that does not engage with those details. The issue of whether those details can later be added would seem to be the question of whether non-mechanistic explanations provided by these models can eventually be complemented with mechanistic information about the components of the nodes or edges. Yet, *whether such information improves or complements the characteristically topological explanations offered by network models is an independent question of how network models explain in the first place.*

The mechanist struggle with non-decompositional modeling strategies is evident in Green et al. (2018), which discusses various examples of network analyses and how they "align with, extend, or depart from more traditional mechanist strategies" (1772). Although most of the models discussed start from the network's overall behavior, Green et al. point out that it is possible to identify "modules as classes of nodes that are highly interconnected", thus alluding to the components of the "traditional mechanist approaches" (1758). However, this reinterpretation of entities does away with their causal pushing and pulling efficacy emphasized by Craver. Green et al. point to the work of Ravasz et al. (2002) as an example of how it is possible to *recover* "classically defined mechanisms" based on network analysis, but this discovery method is nonetheless different from the mechanist one in starting from the whole network and proceeding from there to identify the clusters (Green et al., 2018, 1761). Finally, while Green et al. observe that dynamical systems theory departs from the mechanistic approach in "rejecting the core strategy of decomposition" (1774), they at the same time claim that in order to be predictive, dynamic analysis should be grounded "in details of actual biological systems" (1775). They do not provide any reasons for such a requirement, however.

In our view, referring to the abstraction of mechanistic details, or the need for mechanistic grounding, is beside the point when it comes to the epistemic strategy of such modeling methods, which do not address decomposable systems. In a sense, this should be obvious enough, and so we suspect that the insistence on recovering mechanistic details follows from some normative and/or ontological considerations.

Our overall impression from the literature referred to above, is that mechanists tend to take for granted the requirement of grounding higher-level organizational phenomena in the activities and interactions of (lower-level) entities. They appear to assume that, although network analyses go beyond the traditional mechanist approaches in elaborating some global aspects of biological organization, such approaches should eventually yield to the mechanistic ontology.

Below we present a case study of yet another model, the Heimburg-Jackson model, whose epistemic strategy cannot be rendered under the mechanistic umbrella. This case is interesting in that it touches upon Kaplan and Craver's (2011) claim that models characterizing global patterns, such as phase transitions, are only phenomenal models since they do not describe "mechanisms underlying the phenomena" (602), i.e. "the underlying component parts, their relevant properties and activities, and how they are organized together causally, spatially, temporally, and hierarchically" (605). Moreover, it also addresses the nerve impulse, as does the Hodgkin-Huxley model, though, in contrast to it, its epistemic strategy does not rest on lower-level entities or components that would carry out the explanatory work.

4 The Heimburg-Jackson model

Notwithstanding its status as part of an accepted account of nervous transmission, the Hodgkin-Huxley model has been challenged recently[9] by alternative modeling strategies that seek to make sense of accumulated recalcitrant evidence. The Heimburg-Jackson model is one of the most developed alternatives to the Hodgkin-Huxley model. While the Hodgkin-Huxley model is already ambiguous with respect to a mechanistic interpretation (see also Sect. 5), we will argue below that the epistemic strategy of the Heimburg-Jackson model does not rely on mechanistic decomposition.

The Hodgkin-Huxley model does not account for all the interesting features of the nerve impulse. For instance, mechanical changes accompanying the nerve impulse have been empirically detected, involving a shortening and a swelling of the fibers (Iwasa & Tasaki, 1980). It has also been found that nerve impulses can be generated with mechanical stimulation or cooling (see Heimburg & Jackson, 2005, Heimburg, 2014 and references therein). These mechanical aspects are difficult to accommodate within the electrical approach of the Hodgkin-Huxley model (Drukarch et al., 2018). Moreover, measurements suggest an emission and reuptake of heat in the axon during the nervous impulse that seems to conflict with the predictions of the Hodgkin-Huxley model, which suggests a continuous dispersion of heat due to charges moving across the resistor. Hodgkin himself considered the inability of the model to account for the temperature recordings to be an important challenge (Hodgkin, 1964, 70).

There have been several attempts to approach nerve signaling in a way that accounts for the changes in temperature, volume, density, and length of the axon during nerve signal transmission. Around a decade after the publication of the Hodgkin-Huxley model, Ichiji Tasaki performed several experiments that characterized

[9] There are also earlier studies challenging the electrical view that supports the Hodgkin-Huxley model (see Carrillo & Martínez, 2023; Drukarch et al., 2018).

non-electrical features of the nerve impulse. He eventually suggested that addressing the thermodynamical features of the pulse is crucial for understanding this phenomenon (Tasaki, 1982). There are also recent attempts to either complement the Hodgkin-Huxley model or furnish alternative models that address such non-electrical features. The recently developed Heimburg-Jackson model characterizes the nerve impulse as a phase transition that conforms to a solitonic wave that carries with it density, volume, and permeability changes as well as a heat emission and reuptake.

Heimburg and Jackson based their model on findings that isolated lipids of biomembranes display order-disorder (gel-fluid) phase transitions. The model analyzes in terms of such phase transitions the series of changes that we observe when a signal travels along the axon, including the volume and density changes as well as the voltage variation and temperature emission and reuptake. For such phase transitions to transmit information, they need to constitute discrete signals that travel along the axon. In this thermodynamic model, phase transitions in biomembranes form localized solitonic waves. Solitons are conservative waves that maintain their shape and velocity and do not annihilate or change shape when colliding with other waves, making them good transmitters of information. Heimburg and Jackson found that the occurrence of solitons is possible within the physiological conditions of a sample of biomembranes and synthetic membranes (Heimburg & Jackson, 2005).

The scientists used equations for solitons in hydrodynamic systems to obtain equations that model the transmission of electromechanical pulses. Recall that the transmission of the pulse in the Hodgkin-Huxley models is obtained via the cable equation. On the other hand, in the Heimburg-Jackson model, the transmission of the signal is obtained via a wave equation with nonlinear and dispersive terms that allows it to have localized solitonic solutions.[10] Heimburg and Jackson then showed that solitonic solutions exist for that equation by showing that membranes have dispersive and nonlinear features. Non-linearity entails that if one considers the sound velocity as a function of the density, the speed of sound for the membrane should be such that as the density increases, the sound velocity first decreases and then increases. This can be understood in terms of the membrane being "like a spring that becomes softer when compressed," since compressibility and sound velocity are equivalent (Heimburg & Jackson, 2005, 9791). Dispersion, on the other hand, entails that the sound velocity increases as the frequency of sound increases. The first condition is met as long as the membrane undergoes liquid-gel phase transitions at densities above normal density. The dispersion of the membrane involves testing whether the membrane becomes stiffer as the sound frequency increases.

In their 2005 paper, Heimburg and Jackson report the results of testing the two aforementioned features, nonlinearity, and dispersion, in two kinds of biomembranes (*Escherichia coli* and *Bacillus subtilis*), and two synthetic membranes (DPPC and lung surfactant). Considering these membranes as a representative set, the scientists concluded that there are good reasons to think that nonlinearity and dispersion are generic features of membranes and so, one can assume that solitons occur naturally in all of them.

[10] The wave equation used is close to the Boussinesq approximation equation in hydrodynamics.

Although the model does not account for all the features that the Hodgkin-Huxley model addresses, the model does estimate the speed of the nerve impulse transmission, as well as the heat and thickness changes in the membrane (Heimburg & Jackson, 2005). In addition, the model offers a simpler explanation of general anesthesia. The protein-ion-channel view that has followed the Hodgkin-Huxley model suggests that general anesthetics act by affecting the transmembranal proteins that constitute the voltage-sensitive ionic channels that change the permeability of the membrane during the excited state (e.g. by blocking them). However, this explanation has proven to be at odds with some empirically established patterns. For example, the Meyer-Overton rule establishes that a general anesthetic will be more potent the more liposoluble it is. General anesthetics vary from simple atoms to complex molecules, and there is no obvious structure-activity relationship for general anesthetics.

Moreover, there seems to be a connection between general anesthesia and thermodynamic factors, since the anesthetic effect can be reversed with increased pressure (called the 'pressure reversal effect'). Instead of the protein-specific approach, the Heimburg-Jackson model explains general anesthesia in terms of how anesthetics dissolved in the lipid membrane change its thermodynamical features, suggesting a simple and elegant explanation. According to the Heimburg-Jackson model, by blending into the lipid membrane, anesthetics lower the freezing point of transitions in the biomembranes. As a result, more energy is required to generate a phase change. This accounts for the Meyer-Overton rule and pressure reversal effect by making use of the freezing point depression law that originally established that the melting temperature of water is lowered when salt is added to it.

What interests us in this model is the fact that its explanatory power does not come from decomposing the system into its constitutive entities and reconstructing the phenomenon from their activities. The explanation in terms of freezing point and the notion of phase transition are both at the same scale. Also, the explanation is not translatable into aggregation or omission of microscale phenomena. It is not that entities or activities at a lower scale would be aggregated or abstracted away, the analysis engages with the scale at which the phenomenon is observed without decomposing the system into a set of constitutive elements at a smaller scale (the microscopic scale, in this case).[11] Finally, it is neither the case that this explanation would be tentative until the micro details are obtained.[12] Simply put, there is no epistemic gain in decomposing the system when addressing the phenomenology associated with phase transitions. This is not only our reconstruction of the epistemic strategy of the Heimburg-Jackson model, but it also accords with how the scientists themselves understand their model. For Heimburg, such a macroscale approach offers an advantage when it comes to explaining many issues associated with the nervous impulse:

[11] One might claim that also in this case, the micro level is omitted, but such a claim would trivialize the notion of omission.

[12] Its tentative character is due to the model construction assumptions, and with the fact that it is an *in principle* explanation (see Sect. 5). These are features of the abstract framework and the way it is applied, and not those of a worldly mechanism.

> The accepted model for nerve pulse propagation in biological membranes seems insufficient. It is restricted to dissipative electrical phenomena and considers nerve pulses exclusively a microscopic phenomenon. A simple thermodynamic model that is based on the macroscopic properties of membranes allows for explaining more features of nerve pulse propagation including the phenomenon of anesthesia that has so far remained unexplained. (Heimburg, 2010, 1)

The requirement of constitutive grounding of phenomena on the entities of a lower level provides the core of the mechanistic approach. Such mechanisms, however, are not what the Heimburg-Jackson model is aiming at in modeling phase transitions involved in nerve impulse propagation. Phase transitions such as melting processes are described and explained at the macroscale since these transitions involve "cooperative processes" between the molecules such that there is nothing in the structure of, say, water molecules that explains that ice should melt at zero degrees. (Heimburg, 2014). Heimburg emphasizes that "[t]he melting of membranes is a process that cannot be understood at the level of single molecules. Features of such transitions become apparent only on mesoscopic or macroscopic scales." (2014, 262).

To sum up, while there already are issues in rendering the Hodgkin-Huxley model into the mechanistic guise, the modeling strategy of the Heimburg-Jackson model stands in stark contrast to those mechanistic approaches that seek constitutive explanations, or rely on the notion of decomposition and abstraction as omitting non-difference-making causal factors (see Batterman & Rice, 2014). Of course, mechanists could argue that the Heimburg-Jackson is not an explanatory model, and thus it is not a counterexample to the mechanistic desiderata of explanation (as they have tried to do for the Hodgkin-Huxley model). Such a strategy would only work if there were no alternatives to mechanistic explanation. This is not in accordance with the different accounts of explanation on offer. Bokulich (2017) gives a general account of model explanation that appears to cover multiple accounts of explanation: non-causal, structural, and causal, the latter including the mechanistic one. She characterizes model explanations as having three features. First, the explanans makes an essential reference to a model. Consequently, the model itself does not yet need to provide an explanation, as recognized by Craver, for example. Second, the model "explains the explanandum by showing how the elements of the model correctly capture the patterns of counterfactual dependence in the model" (106). Such counterfactual dependencies can also be delivered by fictional models. Lastly, some justification must be given that specifies what makes the model trustworthy as well as its domain of applicability.

The explanation provided by the Heimburg-Jackson model complies with Bokulich's account of model explanation. The Heimburg-Jackson equations that assume the pulse behaves as a soliton are crucial for the elaboration of their thermodynamical explanation of the nerve signal in terms of phase transitions in the phospholipids. The structure of the model shows counterfactual dependencies in temperature, volume changes, and lateral density, allowing predictions of how the system would change if variables or parameters were to change. And finally, the justification and domain of applicability come from the experiments Heimburg and Jackson performed with *Escherichia coli*, *Bacillus subtilis*, dipalmitoyl phosphatidylcholine (DPPC), and

lung surfactant membranes, showing that the two conditions for solitonical solutions, dispersion, and nonlinearity, characterize with high probability most biomembranes. These results also chart the domain of applicability of the Heimburg-Jackson model, since the transition temperature of the lipids must be close to physiological temperatures for these pulses to travel along the lipids of the membranes.[13]

Not only does the Heimburg-Jackson allow for a model explanation of the nerve impulse, but it also addresses several features of the nerve signal that remained unexplained by the electrical approach of the Hodgkin-Huxley model. Additional features that *can* be explained from the perspective of the Heimburg-Jackson model are temperature emission and reuptake, the volume and density changes, and why the nerve is stimulated when cooling, among others. At the same time, the Heimburg-Jackson model is not compatible with the Hodgkin-Huxley model because the conservative nature of the former model is not easily merged with the dissipative nature of the latter.[14] One important thing to note as well is that the criteria that determine what is relevant to explain the nerve signal are not shared amongst these two groups of scientists (Carrillo & Martínez, 2023).

Even if the Heimburg-Jackson model were to be considered an inadequate explanation of the nerve impulse, it is difficult to think that phase transitions never participate in biological phenomena. It is also not the case that complementing the model with microscale details would somehow render it explanatorily more complete. For these reasons, we think that the Heimburg-Jackson model offers more than a counterexample to the mechanist attempt to appropriate abstract mathematical models, as it is also a representative of an important class of models, whose associated explanations are not amenable to a mechanist (de-compositional) reading. While Green et al. (2018) seem to suggest that network analyses could complement or be at least aligned with the mechanistic account, we do not think that the same is the case with the Heimburg-Jackson model (and do in fact doubt whether many network models can be made to fit under the mechanist umbrella anyway). Models like the Heimburg-Jackson model challenge and offer alternatives to mechanistic strategies. Indeed, identifying mechanisms provides only one way of grounding the counterfactual dependencies that enable scientists to use models to answer *what-if-things-would-have-been-different* questions (Bokulich, 2011, 40).

5 Mechanistic reification

The mechanist attempts to address mathematical modeling have been numerous and divergent. On the one hand, the mechanists have realized that many mathematical models appear not to conform to the decompositional core of the mechanist program, and so they have been considered by mechanists as either non-mechanistic explana-

[13] Hodgkin-Huxley model can also be used to produce a model explanation in Bokulich's sense, depending on what is the why-question that is posed. As we argued above, if the question were that of why ionic currents are enough to account for the nerve signal's shape, the scientists in the past century would have been justified in using the Hodgkin-Huxley equations as an explanation of why this could be the case.

[14] There are attempts at integrating these models, however. See Holland et al. (2019) for discussion.

tions or have been written off as non-explanatory. On the other hand, there have been efforts at giving mathematical models a mechanistic interpretation. It is especially for such efforts that the notion of abstraction has come to the rescue.

The particular notion of abstraction adopted by mechanists, abstraction as omission, seems convenient at first glance, in that it allows mechanists to account for the generic nature of models. Consequently, according to this view, abstract models focus on some causal factors shared by several systems of the same type, or by different mechanistic levels, or they might even abstract away from all other mechanistic features than organization. Yet the notion of abstraction as omission does not stand up to closer scrutiny when it comes to models whose epistemic strategy does not rely on decomposition or even resists it. For instance, network models address the collective behavior of the network without necessarily any knowledge of the underlying biology. To assume that such networks would abstract from the mechanistic details ignores the fact that they address "the *collective* behavior of interest by representing that behavior as a function of a pattern variable" (Rathkopf, 2018, 67, emphasis added). Any possible supplementation of mechanistic details would not be part of the network modeling strategy.

We have presented an additional challenge: the Heimburg-Jackson model. This phase transition model does not obtain its epistemic power from decomposing the target system and reconstructing the role of the different constitutive entities. The point is not whether the system can be decomposed or not – it's no mystery that the configuration of the lipid molecules underlies the phase transitions. However, the model's explanatory power does not stem from such decomposition. Although one could recover such compositional details, the model seeks to address a collective-level phenomenon that cannot be predicted from individual-level behaviors. In a nutshell, the model does not tell us *why* by telling us *how* the phospholipids behave.

We also think that several mechanists' claims on the Hodgkin-Huxley model are not doing justice to it, making their accounts of the model descriptively inadequate. Apart from being a normative project, the new mechanical philosophy has also sought to be descriptively adequate (e.g. Craver, 2007, vii). Consequently, the mechanists have referred to Hodgkin and Huxley's understanding of their own work in order to claim that the equations did not seek to offer an explanation of nerve impulse transmission. Particularly, they refer to the scientists' discussion of the potential ontological implications of the fact that the conductances of the different ionic species could have had different mathematical expressions (Hodgkin & Huxley, 1952, 541):

> The agreement [between the approximation to the solutions of the system of equations and the empirical recordings on giant axons] must not be taken as evidence that our equations are anything more than an empirical description of the time-course of the changes in permeability to sodium and potassium. An equally satisfactory description of the voltage clamp data could no doubt have been achieved with equations of very different form […]

In the above quote, Hodgkin and Huxley are referring to the adjustment they made to the equations with first-order dynamics to reproduce the empirically observed conductances of sodium and potassium. This quote has been used by mechanists

to show that Hodgkin and Huxley *themselves* did not commit to the tentative ontological interpretation of their model. However, such mechanist interpretation leaves open the question of what the model *did* do for the scientists. The (less cited) passage immediately following the previous quote explains the epistemic virtues of the model, *according to the scientists* (ibidem):

> The point that we do consider to be established is that fairly simple permeability changes in response to alterations in membrane potential, of the kind deduced from the voltage clamp results, are a sufficient explanation of the wide range of phenomena that have been fitted by solutions of the equations.

The scientists did not take their model to be a sketch that had to be filled with further (mechanistic) details but rather as an *in principle proof* of the explanatory power of ionic currents.

Instead of focusing on how scientists themselves understood the contribution of their model, some mechanists have preferred to impose their normative framework on their modeling practice. Craver claimed that the Hodgkin-Huxley model is a how-possibly mechanistic sketch that would become an explanation only in the light of the evidence for the existence of protein ion channels responsible for membrane permeability changes in neurons. Craver clearly understands what the model does not seek to accomplish, but chooses not to address its epistemic role, suggesting instead an anachronistic interpretation, according to which the later knowledge would make the model mechanistic. Kaplan (2011) further downplays the epistemic role of the Hodgkin and Huxley model, arguing that the Hodgkin-Huxley model is a phenomenological model. According to Kaplan, phenomenological models are "often constructed via ad hoc fitting of the model to empirical data" (351). In our view, it is implausible that these scientists would have been interested in (merely) reproducing the shape of the nerve impulse, especially given that the equations arrived at had little use at the time when there were no computers that could estimate the solutions to the equations. Instead, the solutions were tiresomely calculated by hand by Huxley: "The propagated action potential took about three weeks to complete and must have been an enormous labour for Andrew [Huxley]" (Hodgkin, 1976, p. 19). What sense would it have made to have a phenomenological model for which it took three weeks to estimate each solution?

We call for the mechanists to pay more attention to what the models do for the scientists lest they not want to impose the mechanist (normative) framework onto the modeling at the expense of interpretative errors. For instance, Kaplan (2011) reifies part of the Hodgkin-Huxley model claiming that although "Hodgkin and Huxley's choice of variables and terms in the original model was governed by purely descriptive and predictive aims, it turns out that the critical rate variables in the model do approximately map onto components in the mechanism […]" (358). In our view, Kaplan makes too much of the phenomenological sections of the model and too little of its epistemically powerful parts. The idea that we can *now* interpret the Hodgkin-Huxley model along a model-to-mechanism-mapping, amounts to a reification of a model that *was never intended to be interpreted in such a way.* Moreover, such an interpretation is most likely not so easily made about the sodium channel as the potas-

sium one, not to mention the fact that alternative explanations of the nerve impulse, such as the Heimburg-Jackson model, can be given. The real power of the Hodgkin-Huxley model is not given enough credit, i.e. showing that the currents of sodium and potassium can in principle account for the excitable behavior of the membrane, even though the scientific community clearly valued it as such, earning Hodgkin and Huxley the Nobel Prize.

To conclude, the mechanistic paradigm that has worked well for many biological explanations may not be extendable to many important kinds of biological models – at least not without significant sacrifices. The attempt to render network and phase transition models mechanistically through the notion of abstraction-as-omission with the consequent possibility of recovering the purported underlying mechanisms is not only a stretch when it comes to accounting for their explanatory role, but it also leads us astray in understanding the epistemic strategies of those models. A similar thing happens with the claim that they are non-explanatory: it simply ignores what those models do, retaining the normative character of the new mechanistic philosophy, but making it descriptively inadequate. Identifying modeling strategies that neither explain (nor predict) in virtue of decomposition as non-mechanistic seems to us a sensible move, as well as a positive one in the sense that it recognizes the richness of both modeling and explanatory practices. It also challenges us to rethink how we understand abstraction.

Acknowledgements The authors would like to thank Thomas Heimburg, Andrew Jackson and Edgar Villagrán for discussing thermodynamical features of the nerve impulse with them. They would also like to thank the anonymous reviewers, whose comments improved the article.

Funding This project received funding from the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation programme (grant agreement No. 818772).

Declarations

Conflict of interest The authors declare no conflict of interest.

Ethical approval and informed consent No animal or human subjects were investigated for this research.

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