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On Explanation in Neuroscience: The Mechanistic Framework

It is a truism to say that some concepts are extremely popular in science and philosophy. "Mechanism" is undoubtedly one of them and the roots of its popularity can be associated with the rise of modern science and rejection of the Aristotelian conceptual framework in the explanation of nature (Kuhn, 1957). Although Rene Descartes and Isaac Newton presented different conceptions of cosmology, in the field of philosophy of science they were both supporters of mechanicism (Heller, 2011; Heller & Życiński, 2014). "The universe is a mechanical clock" metaphor was tempting, because it promised that the laws of mechanics could be applied both to the universe as a whole and to its elements, including the human body. This may be exemplified by William Harvey's model of blood circulation, in which the heart is understood as a mechanical pump (Craver & Darden, 2013). The seventeenth century scholars agreed in principle that the physiology of living organisms can be explained mechanistically. Descartes put it this way:

> I do not recognize any difference between artifacts and natural bodies except that the operations of artifacts are for the most part performed

> by mechanisms which are large enough to be easily perceivable by the senses – as indeed must be the case if they are to be capable of being manufactured by human beings. The effects produced by nature, by contrast, almost always depend on structures which are so minute that they completely elude our senses (Descartes 1983/1644, IV, § 203).

However, early modern mechanicists differed in their approach to the human mind. According to Descartes, mind was the mental substance (*res cogitans*) and – in contrast to the physical body (*res extensa*) – remained beyond the reach of mechanicism. In turn, another eminent mechanicist Julien Offray de La Mettrie argued that Descartes "had counted one substance too many" (Draaisma, 2000, p. 71). Thus, La Mettrie decided that nothing stood in the way of exploring the mind with mechanistic methods. Contemporary mechanicism in the biological sciences has sided with La Mettrie, extending its reach to brain functions and human cognition. William Bechtel, one of the most important theorists of mechanicism in neuroscience, puts it very aptly:

> Here modern science has taken a different path, outdoing Descartes at his own endeavor by finding mechanistic explanations for mental as well as bodily phenomena. Cognitive scientists, and their predecessors and colleagues in such fields as psychology and neuroscience, assume that the mind is a complex of mechanisms that produce those phenomena we call "mental" or "psychological" (Bechtel, 2008, p. 2).

Thanks to Thomas Kuhn we know that scientific revolutions are associated with conceptual incommensurabilities – after the revolution, a given concept changes its meaning in relation to the state before the revolution (Kuhn, 1962). This relates not only to physical concepts, such as "simultaneity of events," but also to metascientific concepts – those which are the subject of research in the philosophy of science. Even contemporary philosophy of science itself seems to be a child of the metascientific revolution, an event perpetrated on the one hand by philosophers (primarily members of the Vienna Circle and their main

critic, Karl Popper), and on the other by scientists, especially physicists (Życiński, 1988). As a result of the metascientific revolution, the concept of mechanicism changed its modern meaning. However, a contemporary "new mechanicism," typical primarily for the philosophy of biological sciences, differs from the seventeenth century "clockwork universe." Our investigations will focus on the latest installment of mechanicism and the role of the mechanisms in neuroscientific explanation (cf. e.g. Bechtel, 2008; Craver 2007, Craver & Darden, 2013).

In this article, we set ourselves the following objectives. In the first part of the work we characterize the paradigm of mechanistic explanation in contemporary life sciences, narrowing the investigation to neuroscience. We indicate the general characteristics of the mechanistic framework and differences that occur in the various versions of mechanism. We present examples of scientific explanations which have successfully managed to apply the mechanistic framework. In the second part, we present norms of mechanistic explanation which allow us to distinguish explanatory models from non-explanatory models, which serve as heuristics. In the third part, we present a multilevel model of spatial navigation in accordance with the norms of mechanistic explanation – a model emerging from the latest discoveries in neuroscience. In the last, fourth part of the paper we discuss some philosophical and methodological issues of mechanicism.

1. Towards a mechanistic explanation

Explanation of phenomena is one of the main tasks of science. However, there is no consent among methodologists of science regarding what scientific explanation is. The starting point for discussion is usually a neo-positivist conception of explanation, referred to as the deductive-nomological model or covering law model, formulated by Carl Hempel and Paul Oppenheim (1948; cf. Hempel, 1966). In this sense, explanation of the phenomenon involves performing reasoning having the following features:

(1) *explanans* are the premises of reasoning
(2) *explanandum* is a conclusion
(3) there is at least one law in the set of premises (*explanans*)
(4) the presmises have empirical content.

If all the above conditions ((1) – (4)) are met, we are facing a full-blooded explanation of the phenomenon. Explanations that do not meet the condition (4) are not incorrect, but are referred to as potential. In practice, they are often valuable because of their heuristic function. Explanatory reasoning in deductive-nomological model takes the following form:

$L_1, L_2, \ldots, L_r$
$C_1, C_2, \ldots, C_k$ *Explanans* sentences

E *Explanandum* sentence

$L_1, L_2, \ldots, L_r$ represent laws; proper explanation requires obligatory reference to at least one law. $C_1, C_2, \ldots, C_k$, represent sentences related to the initial conditions. Sentence E refers to the phenomenon being explained. Sentences referred to as the laws must relate to general regularities, and thus they take the following logical form: $(\forall x)\,[F(x) \rightarrow G(x)]$, which should be read: for all x, when x is subjected to F-conditions, x acts in a G-way. Hempel writes:

> The laws required for deductive-nomological explanations share a basic characteristic: they are, as we shall say, statements of universal form. Broadly speaking, a statement of this kind asserts a uniform connection between different empirical phenomena or between different aspects of an empirical phenomenon. It is a statement to the effect that whenever and wherever conditions of a specified kind F occur, then so will, always and without exception, certain conditions of another kind, G. (Hempel, 1966, p. 54).

However, this scheme works well only in case of nomothetic sciences – scientific disciplines formulating laws. The prototype of nomothetic science is physics although in biological sciences, especially in neuroscience and medicine (Marcum, 2008; Juś 2014) one can find laws, as a rule they are not nomothetic. Universal, deterministic and mathematicised laws rarely occur in these sciences. Neuroscientists sometimes refer to the general laws of evolution and genetics in their articles (e.g. Hardy-Weinberg principle); however, they cannot be regarded as *prima facie* neuroscientific. This means that the deductive-nomological model of scientific explanation can only be used occasionally in the case of these disciplines. Admittedly, "weaker," statistical variations of this approach have been developed, according to which explanation involves deductive (deductive-statistical model) or inductive reasoning (inductive-statistical model), but they also have not found widespread use within the framework of biological sciences, particularly neuroscience (Craver, 2007; Bechtel & Richardson, 2010). Moreover, it appears that these models of explanation lead to paradoxes (cf. Bromberger, 1966). Problems with the nomological-deductive model of scientific explanation and its weaker variations have led to new insights on the nature of explanation (Salmon, 2006).

Supporters of the deductive-nomological model have also been accused of the fact that they ignore the role of causality in scientific explanation – a role pointed out by Aristotle. Michael Scriven (1962) also noted that the reference to the law in explaining the phenomena may be helpful, but not mandatory. For example, to explain the phenomenon of a blue stain on the carpet, it is sufficient to note that it was caused by spilled ink. In this case, the identification of the cause equals the explanation of the phenomenon. In practice, scientific explanations generally relate to more complex phenomena in which the identification of the cause is not as easy. In addition, at least since David Hume (1975/1748) "causality" is one of the most problematic concepts in philosophy. Although intuitively causality is related to contact in space and time in the sense that causes occur prior to their effects, it is difficult to distinguish causality from correlation.

The theory of causality, which is often used in contemporary analyses of scientific explanation (cf. Craver, 2006; Craver & Darden, 2013; Juś, 2014; Miłkowski, 2013a), was formulated by James Woodward (2003). The starting point of this theory is the requirement of connection of explanations, which are to be provided by science, with experiments. According to Woodward: "We are in a position to explain when we have information that is relevant to manipulating, controlling, or changing nature (…). We have at least the beginnings of an explanation when we have identified factors or conditions such that manipulations or changes in those factors or conditions will produce changes in the outcome being explained" (*ibidem*, pp. 9–10). Woodward argues that mere observation does not say anything about causality. However, knowledge of causality is possible and can be acquired through experimental manipulations (cf. Miłkowski, 2013b; Pearl, 2000). Causal explanations demonstrate how modification of the value of one variable modifies the value of another variable or a set of variables:

> the claim that X causes Y means that for at least some individuals, there is a possible manipulation of some value of X that they possess, which, given other appropriate conditions (perhaps including manipulations that fix other variables distinct from X at certain values), will change the value of Y or the probability distribution of Y for those individuals. In this sense, changing the value of X is a means to or strategy for changing the value of Y (Woodward, 2003, p. 40).

Woodward's theory of causal explanation is applicable in case of basic neuroscience and cognitive neuroscience, as these are *par excellence* experimental disciplines in which, depending on the level of complexity, various research methods are used: single-units recording, bioelectric brain-activity measurements (electroencephalography, EEG) or magnetic brain-activity measurements (magnetoencephalography, MEG), indirect neuroimaging (e.g. functional magnetic resonance imaging, fMRI), or temporary deactivation of brain structures (transcranial magnetic stimulation, TMS). The conception of causal explanation

can also be applied to computational neuroscience and neurorobotics. In computational neuroscience, models are created and tested in computer simulations, in which the manipulated variables influence other variables. In the other one, physical models are constructed and then tested in the environment (Miłkowski 2013a, 2013b). What is more, the theory of causal explanation can easily be extended to include contextual aspects (for contextual account of scientific explanation, see Van Fraassen, 1980). This is because neuroscience discoveries often find their application in neurology, neuropsychology and clinical psychiatry. It must also be noted that the above-outlined approach to causality was clarified mathematically, and its practical application is facilitated by a special programming language (cf. Pearl, 2000).

William Wimsatt (1972, p. 67) states that "at least in biology, most scientists see their work as explaining types of phenomena by discovering mechanisms." This is indicated by frequent appearance of the term "mechanism" in the titles and abstracts of scientific publications in the field of biological sciences – especially neuroscience. Furthermore, a specific form of causal explanation called the "mechanicism" – or to distinguish it from proposals of early modern philosophers (such as Descartes), the "new mechanicism" – is adopted in many monographs (cf. e.g. Bechtel, 2008; Craver 2007; Craver & Darden, 2013; Miłkowski, 2013a) devoted to explanation in neuroscience and cognitive science. Therefore mechanicism is not an artificial framework developed by methodologists of science, but rather a result of the coevolution of philosophical inquiries and practice of scientists. To date, thanks to the mechanical framework, scientists have managed to formulate explanations of biological phenomena such as: cellular respiration and Krebs cycle (Bechtel, 2005, Bechtel & Abrahamsen, 2007), circadian rhythms (Bechtel, 2010), fertilization (Craver & Darden, 2013), genetics and heredity (Darden, 1991; Darden & Maull, 1977), protein synthesis (Machamer, Darden & Craver, 2000). As for neuroscience, *inter alia* the following mechanistic explanations have been formulated: action potential (Craver, 2006, 2008), chemical transmission at synapses (Machamer, Darden

& Craver, 2000), memory and learning (Bechtel, 2009, Craver, 2002, 2003, Craver & Darden, 2001).

According to the mechanistic strategy, explanation of the phenomenon does not depend on its logical inference from the initial conditions and laws of nature, but on the identification of the causal structure generating it. Generally speaking, the mechanism is a system composed of multiple parts (Bechtel & Richardson, 2010), which as a whole manifests a pattern of action (disposition), e.g.:

> The mechanism of chemical neurotransmission, a pre-synaptic neuron transmits a signal to a post-synaptic neuron by releasing neurotransmitter molecules that diffuse across the synaptic cleft, bind to receptors, and so depolarize the post-synaptic cell (…). Descriptions of mechanisms show how the termination conditions are produced by the set-up conditions and intermediate stages. To give a description of a mechanism for a phenomenon is to explain that phenomenon, i.e., to explain how it was produced (Machamer, Darden & Craver, 2000, p. 3).

According to supporters of mechanism, the disposition of a complex system should be explained as the result of the interaction of the individual parts of the mechanism or the structure of the processes taking place in it (Miłkowski, 2013b). Although the majority of supporters of mechanism would agree with this general characteristic, individual theorists propose solutions which differ in the details. Let us examine three alternative interpretations of the mechanism formulated by Stuart S. Glennan (**G**), William Bechtel (**B**) and Peter Machamer, Lindley Darden and Carl F. Craver (**MDC**):

> **G**: A mechanism underlying a behavior is a complex system which produces that behavior by the interaction of a number of parts according to direct causal laws (Glennan, 1966, p. 52).

> **B**: A mechanism is a structure performing a function in virtue of its component parts, component operations, and their organization. The

> orchestrated functioning of the mechanism is responsible for one or more phenomena (Bechtel, 2008, p. 13).
>
> **MDC**: Mechanisms are entities and activities organized such that they are productive of regular changes from start or set-up to finish or termination conditions (Machamer, Darden & Craver, 2000, p. 3).

Although at first glance these definitions appear similar, they emphasize different elements. The first two – **G** and **B** – emphasize that the mechanisms are identified on the basis of phenomena/behavior for which they are responsible. However, there are differences between them. **G** emphasizes the primary role of the interaction between the parts of the mechanism. It also puts particular emphasis on the role of direct causal laws. Glennan's approach is of a reductionist nature – laws functioning at a given level are explained by reducing them to lower level laws, until the fundamental laws of physics are reached. On the other hand, the **B** approach – as emphasized by Bechtel himself – "focused on the 'functions' (operations) that parts perform" (p. 13, footnote 4). With this approach, the need for reference to direct causal laws disappears – causality occurs at the level of interaction between the parts. Last approach, **MDC**, seems to take into account the claims of both **G** and **B**. **MDC** emphasizes the role of activities in the mechanisms, while these activities are not necessarily scientific laws in the strict sense. We operationally choose the definition of mechanism proposed in **MDC** approach. It should be emphasized that in this approach mechanisms should not to be understood in reference to mechanical systems based on the principle of action and reaction. Machamer, Darden and Craver argue that the mechanisms are complex *structures* constituted by both entities and activities.

According to **MDC** approach it is assumed that entities and activities exist in reality and can be identified by empirical research. What's more, it is a pluralistic form of scientific realism: you cannot reduce activities to entities and *vice versa*. **MDC** is therefore inconsistent with both substantialism (e.g. Aristotle) and processual-

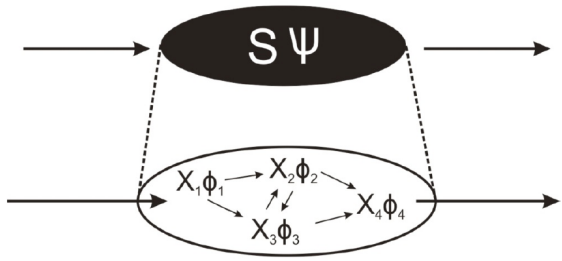
ism (e.g. Whitehead), because each of these approaches proclaims the fundamental nature of only one of these categories: entities *or* activities. For this reason mechanicism according to **MDC** approach is defined as dualistic. Machamer et al. (2000, p. 3) characterizes their approach more precisely as follows:

> Activities are the producers of change. Entities are the things that engage in activities. Activities usually require that entities have specific types of properties. The neurotransmitter and receptor, two entities, bind, an activity, by virtue of their structural properties and charge distributions (…). The organization of these entities and activities determines the ways in which they produce the phenomenon. Entities often must be appropriately located, structured, and oriented, and the activities in which they engage must have a temporal order, rate, and duration. For example, two neurons must be spatially proximate for diffusion of the neurotransmitter. Mechanisms are regular in that they work always or for the most part in the same way under the same conditions. The regularity is exhibited in the typical way that the mechanism runs from beginning to end; what makes it regular is the productive continuity between stages. Complete descriptions of mechanisms exhibit *productive continuity* without gaps from the set up to termination conditions. Productive continuities are what make the connections be- tween stages intelligible. If a mechanism is represented schematically by A → B → C, then the continuity lies in the arrows and their explication is in terms of the activities that the arrows represent. A missing arrow, namely, the inability to specify an activity, leaves an explanatory gap in the productive continuity of the mechanism.

As can be seen in the above passage, in the **MDC** approach the concept of the laws of science is replaced by slightly looser notion of *regularity*. Of course, this does not mean that the laws do not exist or take part in the explanation. Their presence is simply not obligatory. It should also be emphasized that the identification of entities can be made due to the spatial position, structure and hierarchy, while the ac-

tivities can be identified due to the time organization, pace and duration. The diagram below represents mechanistic explanation:

phenomenon
(explanandum)



mechanism
(explanans)

This diagram illustrates the mechanistic explanation (adopted, see Craver, 2007, p. 7).

The top part of the diagram represents the explained phenomenon (*explanandum*). The ψ symbol indicates phenomenon, property or action explained by the mechanism. The S symbol denotes the mechanism as a whole. The lower part of the diagram represents entities (circles) and activities (arrows coming in and coming out of circles). They serve as *explanans* (what explains) in the process of explanation. X is a component (part) of the object, and ϕ symbolises action of the object in the mechanism. The explanation of Sψ phenomenon consists in the discovery and demonstration of structure of the objects $\{X_1, X_2, X_3, ..., X_m\}$ and actions $\{\phi_1, \phi_2, \phi_3, ..., \phi_n\}$.

2. Norms of mechanistic explanation

Comprehensive explanation requires a complete description of the causal structure thanks to which the explained phenomenon occurs.

This calls for the identification of a constitutive level of the mechanism and for its decomposition, thus revealing entities and activities. The constitutive level is determined in scientific practice. For example, specific neural networks, specific subcortical and cortical structures or single neurons establish a constitutive level for the mechanisms examined within the framework of cognitive neuroscience, which studies the brain substrates of cognitive processes (Piccinini & Craver, 2011). Mechanisms considered in the context of cognitive neuroscience are usually hierarchical and multi-level. In case of basic neuroscience, single neurons or biological and chemical entities, constituting the neuronal level itself, become the constitutive level. It should be emphasized that in principle the constitutive level is not defined arbitrarily, but is identified pragmatically for each mechanism (Machamer et. al., 2000). Therefore, although mechanicists apply decomposition, which is *de facto* a form of reduction, they avoid highly reductionist statements about the fundamentality of the physical level for biological mechanisms (cf. Craver, 2007; Miłkowski, 2013b). However, the pragmatic nature of explanation in neuroscience does not mean arbitrariness. On the contrary, the process of clarifying demands compliance with certain standards.

Craver (2006) notes that not every model constructed by scientists is an explanation. He distinguishes between: (i) the phenomenal models and explanations, (ii) sketches and complete explanations, as well as (iii) possibly models and actually models. Regarding the first distinction, it should be noted that phenomenal adequacy does not necessarily involve the explanatory force. The Ptolemaic model of the solar system, which properly predicts the positions of celestial bodies (and therefore is empirically appropriate, cf. Kuhn, 1957), but does not provide an explanation of their movements utilizing the causal mechanism (Craver, 2006), is an often cited example from the history of science. According to a mechanistic norm of explanation (N1), models explaining the phenomena are not only phenomenal models, as they allow for interventions and experimental manipulations, enabling control over the phenomenon. This allows for e.g. the

determination of the causal relationships between objects described by the model (Woodward, 2003).

The second distinction concerns the level of the detail of the description. Any explanatory model can be located on the continuum a mechanism sketch and a fully complete description of the mechanism (Craver, 2006). Although the mechanism sketches describe the structure of entities and activities, they are not complete explanations because they contain gaps identified in the diagrams by e.g. black boxes. Sometimes sketches also contain not defined precisely *filler terms*, to which Craver (2006) includes e.g.: "activate," "encode," "inhibit" or "represent." While gaps in most cases are filled thanks to new discoveries, filler terms often pose an obstacle to the advancement of knowledge. A full description of the mechanism "includes all of the entities, properties, activities, and organizational features that are relevant to every aspect of the phenomenon to be explained" (Craver, 2006, p. 360). In fact, it is an ideal which very rarely, if ever, occurs in scientific practice. According to the mechanistic norm (N2), explanations *should* approximate fully complete descriptions of the mechanisms. However, the progress of science is often a result of simplifying and removing irrelevant factors from the model, and therefore explanations should be *pragmatically complete*. Models that are on the continuum between sketches and fully complete descriptions are referred to as *mechanism schemata*. Diagrams are simplified and abstract descriptions of mechanisms open to future descriptions of entities and activities (Machamer et al., 2000).

The third distinction concerns the *how-possibly* models and *how-actually* models. It refers to another norm of mechanicism (N3): in case of how-actually models "components described in the model should correspond to components in the mechanism (...). How-actually models describe real components, activities, and organizational features of the mechanism, that in fact produces the phenomenon" (Craver, 2006, p. 362). This means that the model constructed by scientists in order to explain the operation of the system should not only be adequate during the stages of input and output, but should also

simulate actual behavior of internal sub-systems. A model that does not meet this requirement is the how-possibly model. Mechanistic models should not only be correct instrumentally, but also adequate in terms of scientific realism (Craver, 2007; Machamer et al., 2000). In summary, explanation (N1) cannot be a purely phenomenal model, (N2) should contain as few gaps and filler terms as possible, and (N3) should specify actual entities and activities, constituting operations of the system. Of course, norms (N1), (N2), and (N3) are not being fulfilled in accordance with the all-or-nothing principle.

Craver (2006) applies these norms of mechanistic explanation to the Hodgkin-Huxley model (1952). This model mathematically describes how action potentials in neurons are initiated and propagated. Developing this model was a milestone in the development of neuroscience, and its creators were honored with the Nobel Prize in Physiology or Medicine in 1963. It characterizes the time-course of the action potential in following equation (cf. Craver, 2006, p. 363):

$$I = C_M \mathrm{d}V/\mathrm{d}t + G_K n^4(V - V_K) + G_{Na} m^3 h(V - V_{Na}) + G_l(V - V_l)$$

I refers to the total current crossing the membrane. It is constituted by four factors: the capacitative current $C_M \mathrm{d}V/\mathrm{d}t$, the potassium current $G_K n^4(V - V_K)$, the sodium current $G_{Na} m^3 h(V - V_{Na})$, as well as the so-called "leakage current" $G_l(V - V_l)$, which is a sum of smaller currents for other ions. Maximum values of conductivity for respective currents are symbolized by G_K, G_N and G_l. V is displacement of V_m from V_{rest}. Differences between V_m and diverse ionic equilibrium potentials are maped by V_K, V_{Na}, and V_l. Membrane's capacity to store opposite charges on the outside and inside of a neuron is represented by C_M (capacitance). Symbols h, m, and n, represent factors with different values in relation to voltage and time.

Craver (2006) states that although the Hodgkin-Huxley model has many practical applications – e.g. it can be used in simulations of neuronal activity – it does not meet the norms of mechanistic expla-

nation. He underlines that, "the authors insist that their model is not an explanation" (Craver, 2006, p. 356). He demonstrates that according to the Hodgkin and Huxley equations are merely empirically adequate description of the time course of permeability changes. Craver (2006, p. 364) also quotes one of Hodgkin's and Huxley's associates who thinks that this model "summarized in one neat tidy little package the many thousands of experiments done previous to 1952, and most subsequent ones" (Cole, 1992, p. 51). Equations do not contain a causal description of the mechanism of changing the membrane conductance. Hodgkin and Huxley model is therefore purely phenomenal, and thus does not meet the (N1) norm of a mechanistic explanation (just as the Ptolemaic model does). It is worth noting that this standard corresponds with the view of Aristotle, who claimed that describing *via* mathematical structures does not provide explanation – causal explanation is necessary (cf. Hankinson, 1998).

Huxley's and Hodgkin's knowledge about action potential went far beyond the components of a basic equation. In other words, a mathematical model was supported by background knowledge of the facts that action potential is a result of the alteration of the permeability of a membrane; that ions move along the surface of the membrane in the direction of the equilibrium potentials; and that their movement results in a transmembrane current (Craver, 2006). Some entities and activities of the mechanism are distinguished. Inclusion of this knowledge leads to a partial explanation of how nerve cells generate action potentials.

Nevertheless, it is not a complete explanation, but only a sketch. Thus, it does not satisfy the norm (N2), although Craver believes that this model may provide a basis for explanations:

> The equations include variables that represent important components in the explanation. And they provide powerful evidence that a mechanism built from those components could possibly explain the action potential. And the equations, supplemented with a diagram of the electrical circuit in a membrane, and supplemented with details about

> how membranes and ion channels work, carry considerable explanatory weight (2006, p. 365).

This example draws attention to an important practical consequence: mechanistic explanations in biological sciences – especially in neuroscience – often use diagrams. They not only help in understanding a particular phenomenon, but reveal its causal structure, actually taking part in scientific explanation (Abrahamsen & Bechtel, 2015; Sheredos, Burnston, Abrahamsen, & Bechtel, 2013).

The last standard of mechanistic explanation (N3) is also not completely satisfied. The mathematical equation of Hodgkin and Huxley leads to a *how-possibly* sketch of the action potential (Craver, 2006). Multiple equations, other than the one proposed by Hodgkin and Huxley, can be used to predict the action potential's time-course. Background knowledge behind these equations can be very different, as well as their empirical interpretation. In other words, different explanatory mechanisms may result in different mathematical models of phenomenon.

According to the standards of mechanicism: (N1), (N2) and (N3), explanation of how action potentials in neurons are initiated and propagated, has become possible thanks to decades of scientific discoveries, among others concerning the structure and function of ion channels. Craver presents the transition from the phenomenal model of Hodgkin and Huxley to a mechanistic explanation *consistent* with this model:

> (…) it is now well-known that conductance changes across the membrane are effected by conformation changes in ion-specific channels through the cell membrane. Biochemists have isolated these proteinaceous channels, they have sequenced their constituents, and they have learned a great deal about how they activate and inactivate during an action potential. It is in this wealth of detail (some of which is discussed below) about how these channels regulate the timing of the conductance changes, as described by the H[odgkin]-H[uxley's] equations, that explain the temporal course of the action potential (Craver, 2006, p. 367).

In this section we presented standards of mechanistic explanation using action potential (a phenomenon fundamental to neuroscience) as an example. In the next section we will present a practical application of mechanistic framework using the example of the more complex phenomenon of spatial navigation.

3. Case study: spatial navigation

Let us consider one of the examples presented by supporters of mechanistic explanation – spatial navigation in humans and animals (Craver & Darden, 2001; Craver, 2003). The issue of the mental representation of space has been present in philosophy at least since Immanuel Kant. At present it has become the subject of psychological and neuroscientific research and is a perfect test area for the mechanistic framework.

At the end of the first half of the twentieth century, the results of experiments conducted on rats led Tolman (1948) to believe that animals store relations between experienced objects and events in form of maps in their memory. This view was not consistent with the stance of the behaviorist who explained spatial navigation by referring to the sensory-motor response relationships. Although Tolman showed that cognitive representations of space explored by the animal *may* resemble maps, he did not indicate their location in the brain. A year later, Donald Hebb (1949) suggested that to increase the strength of synapses occurs when the presynaptic and postsynaptic neuron are simultaneously stimulated. Hebb's rule is often illustrated by the maxim: "What fires together, wires together."

It is assumed that the spatial representation is associated with learning processes that occur by an increase of the weight of synapses (as described by Hebb), which is referred to as long-term potentiation (LTP). It is known that this phenomenon can be divided into three phases: the induction, expression and maintenance. The induction of LTP depends on the activation of NMDA receptors, the increased re-

lease of glutamic acid in axon terminals in CA3 part of the hippocampus, and the depolarization of membrane of postsynaptic neurons in the CA1 part of hippocampus (symbols of the hippocampal parts are derived from a different name of hippocampus: cornus ammonis). This means that the LTP is induced when the simultaneous activation of pre- and postsynaptic cells occurs, something which is consistent with Hebb's rule. The opening of ion channels allows the transport of NMDA receptor-bound calcium ions inside the neuron, playing a crucial role in the induction of LTP. AMPA receptors are activated, resulting in the opening of the sodium channel – it brings an influx of Na+ ions and membrane depolarization (see e.g. Longstaff, 2007, pp. 387–420, for details).

Cells associated with spatial navigation have been found in the mouse hippocampus thanks to research using implanted micro wires (O'Keefe & Dostrovsky, 1971). This conclusion has been confirmed in studies using pharmacological agents and gene knockout technique – changes in the production of LTP in the hippocampus are associated with deficits in spatial learning, but not necessarily learning in general (cf. Tsien, Huerta & Tonegawa, 1996). In contemporary neuroscience it is generally accepted that spatial maps (the existence of which was suggested by Tolman) are stored in the hippocampus (Derdikman & Moser, 2011).

Honoring three neuroscientists – John O'Keefe, May-Britt Moser and Edward Moser with the Nobel Prize in Physiology or Medicine "for their discoveries of cells that constitute a positioning system in the brain" has been the crowning achievement of research on spatial navigation. This system – commonly known as cerebral GPS – consists of several subsystems: space cells located in the hippocampus and grid cells, border cells and head-direction cells activated in the parahippocampal cortex and the medial entorhinal cortex (see Derdikman & Moser, 2011, for a literature review). Derdikman and Moser (2011) claim that "space is represented in these structures by a manifold of rapidly interacting maps generated in conjunction by functionally specific cell types such as place cells and grid cells" (p. 42).

Place cells are pyramidal neurons in the hippocampus which are activated when the animal is in a specific location. In other words, the specified location is a reception area for a given place cell. Quirk, Muller and Kubie (1990) showed that these cells are not specific only for the visual modality, as they are activated both in the light and in the dark. Although place cells are highly specific, apart from the location they also encode rules of learning with regard to scents. This is evidenced by the results of an experiment conducted by Wood, Dudchenko, and Eichenbaum (1999), in which rats were tasked with recognizing odors other than presented before, demanding the use of episodic memory.

Studies in rats show that the brain navigation system is innate. Langston, Ainge, Couey, Canto, Bjerknes, Witter, Moser & Moser (2010) have identified place cells in the brains of rats, when these animals left the nest few days after their birth and began to explore the environment. In the same work, they demonstrated that the movement from one location to another, or a change in environmental conditions is accompanied by the update of the spatial map encoded by the same populations of neurons in the hippocampus. Langston et al. (2010) called this process *remapping*. Grid cells, which like place cells are used in animal spatial navigation in the environment, are also subject to it. Network cells are named after the fact that the open space around the animal is coded as discharges, which are visualized by a hexagonal network. These neurons have been located in medial entorhinal cortex (Fyhn, Molden, Witter, Moser & Moser, 2004) and in part of the hippocampus called the subiculum (Boccara, Sargolini, Thoresen, Solstad, Witter, Moser & Moser, 2010). These structures include border cells and head-direction cells as well.

In summary, the contemporary approach to the hypothesis of cognitive maps is as follows (cf. Longstaff, 2007): the hippocampus is the key structure of the brain for spatial navigation. Optimal navigation of an animal in the environment is the function of cognitive maps. Cognitive maps are created thanks to episodic memory – through the process of association of sensory and motor guidelines representation of

the animal's location in space is generated. Maps are dynamic – they can be modified in the course of space exploration by the animal. Finally, mapping does not require reinforcements, which runs counter to the key assumption of behaviorism. Even such a brief description of discoveries in the field of neuroscience allows for reconstruction within the mechanical framework (Craver & Darden, 2001).

Our approach to mechanism – adopted from **MDC** – requires first of all the distinction of entities and activities in spatial navigation. We distinguish the following entities: pyramidal neurons (e.g. space cells), molecules (e.g. glutamate molecules), receptors (e.g. NMDA receptors), brain structures (e.g. hippocampus), and whole organisms (e.g. rat). Activities include e.g. firing of pyramidal cells, increased glutamate release, depolarization of the cells, coactivation of pre- and postsynaptic cells, or exploration of the environment by the rat.

Craver and Darden (2001) distinguish three aspects of the mechanism: spatial, temporal and hierarchical. The spatial layout of mechanism is associated with the location of its elements. According to most researchers, the hippocampus is an area of the brain which consists of part of the elements of the spatial memory mechanism. With this assumption, we can study the construction of the hippocampus in order to discover how it translates into its activity and how this is related to the functioning of a given mechanism.

The temporal aspect refers to the direction and order of occurrence of activities within mechanisms. It carries information about how activities responsible for productive continuity are temporarily positioned with respect to each other. This helps to avoid the paradox of the asymmetry characterizing deductive-nomological model which hinders a correct indication of causal relations of explained phenomena.

As far as the hierarchical aspect of the mechanism are concerned, four levels of biological complexity can be abstracted the above description, listed from the highest to the lowest:

(L1) level of the whole organism;
(L2) level of brain structures;

(L3) level of synapses;
(L4) level of neuroreceptors.

The mechanistic explanation of the phenomenon takes place in reverse order to the above hierarchy. Activation of the NMDA receptors (L4) can result in LTP, which relates to the increase in synaptic conduction (L3); LTP enables the consolidation of spatial map in the hippocampus – the structure of the midbrain responsible for the consolidation of information (L2). It is due to the spatial map that an organism (L1) – e.g. a rat – can carry out the task given by the researchers, e.g. find its way in the maze. These levels are not completely independent of each other and their hierarchy is a nested one. The mechanistic explanation is therefore of an integrating nature:

> The elaboration and refinement these hierarchical descriptions typically proceeds piecemeal with the goal of *integrating* the entities and activities at different levels. Integrating a component of a mechanism into such a hierarchy involves, first, contextualizing the item within the mechanism of the phenomenon to be explained. This *involves* "looking up" a *level* and finding a functional role for the item in that higher-level mechanism" (Darden & Craver, 2001, pp. 118–119).

We encounter such a nested hierarchy only if the mechanism manifests a productive continuity between the stages at different levels. According to **MDC** it is this objectively existing productive continuity which enables explanation and understanding of the mechanisms. The discovery of a productive continuity between levels in the hierarchy is possible thanks to experimental interventions.

Bottom-up and top-down experiments are distinguished within the framework of the mechanicism (Bechtel, 2002; Craver & Darden, 2001). In the first case, the influence of lower-level interventions on the functions of the higher levels of the system is studied. In the case of lesion studies, researchers are interested in the ways in which damage to parts of the system affect the functioning of the whole system.

It is known that damage to the hippocampus results in spatial memory deficits, confirming the role of this structure in spatial navigation. One of the most famous neurological cases – of a patient with the initials H.M. – provides an example of bottom-up experiments. As a result of the surgical removal of the hippocampus, which aimed at the alleviation of seizures, the patient lost the ability to memorize new facts (anterograde amnesia). On the other hand, in the case of top-down experiments, intervention is performed at a higher level and its impact on the lower level of the system is examined. Studies using techniques of single-units recording, conducted by Nobel Prize winners – O'Keffe and Moosers – are cases of top-down experiments. It is similar in the case of research using neuroimaging techniques, i.e. fMRI. The consistency of the results of top-down and bottom-up recording provides arguments for the existence of relations between different levels of the mechanism. However, productive continuity is revealed by a third type of neuroscientific research – multilevel experiments.

Studies on the influence of so-called gene knockouts on spatial memory elements located at different levels of the studied mechanism are an example of such experiments (McHugh et al., 1996; Rottenberg et al., 1996; Tsien et al., 1996). This bottom-up research is considered the first one to simultaneously test the functioning of the mechanism of spatial memory at all levels. It involved damaging the gene responsible for specific NMDA receptors in mice. During the study it has been found that these mice cope with the tasks far worse than animals in the control group. This phenomenon is explained by the fact that the failure of NMDA receptors in cells prevented LTP in hippocampal CA1 region (Tsien et al., 1996). What is important from the methodological point of view is the fact that intervention at the bottom level led to the detection of phenomena on multiple upper levels, an essential feature of a multilevel experiment.

In line with what has been said in the previous section, explanation (N1) cannot be a purely phenomenal model, (N2) it should contain as few gaps and filler terms as possible, and (N3) it should specify the actual entities and activities constituting the system. As far as the norm

(N1) is concerned, the abovementioned theory of spatial navigation is certainly not only a phenomenal model, but an explanatory one – it is not only a summary of empirical data. Norm (N2) is partially satisfied. First of all, the mechanism outlined above has gaps. The area of LTP expression is the subject of controversy among scholars (cf. Longstaff, 2007). Most scholars believe that the area of expression in the synapses of CA1 and CA3 hippocampal neurons is located in the postsynaptic membrane. However, some researchers claim that the expression of LTP occurs in the presynaptic membrane. Secondly, one can argue that terms such as "encode" or "represent," appearing in the papers on spatial navigation, belong to filler-terms. Thirdly, with regard to the above-described experiments using gene knockout technique, Craver and Darden (2001) argue that their results lead to a possible mechanism. According to them "it is a sketch because we are not remotely in a position to trace out all of the mechanisms at all of the different levels" (p. 132). This results in a lack of the complete fulfillment of a norm (N3): although we know that, for example, space cells or hippocampus are entities of the mechanism, gaps result in the classification of this mechanism as possible, though very probable.

4. Summary and further remarks

In this article we have presented a mechanistic framework of scientific explanation. We argued that within biological sciences – especially neuroscience – it is more adequate than the neopositivist deductive-nomological model. We showed how different methodologists understand the key term to this approach – "mechanism," focusing on **MDC** proposal. In view of the fact that the mechanistic framework is a special case of causal explanation, we explicated the concept of "causality," referring to the approach of Woodward (2003). We presented the norms of mechanistic explanation, showing (on the basis of Hodgkin-Huxley's model (1952)) that not every model constructed by neuroscientists has an explanatory character. Using spatial navigation

as an example we showed that contemporary theories (Derdikman & Moser, 2011) approach the standards of mechanistic explanation. An example of spatial navigation also showed that the mechanistic framework is of a unifying nature – it integrates multiple levels of biological complexity (Craver & Darden, 2001).

At this point we have to say that mechanicism demands a rethink of some issues raised traditionally in the general philosophy of science. Firstly, we wrote that mechanicism is more natural than the deductive-nomological model due to the fact that neuroscience is not a typical nomothetic science (its task is not to formulate laws). Although in principle it is true, it must be said that the very concept of "law of nature" is not self-evident (cf. Armstrong, 1983; Cartwright, 1983), and thus classifying strategies of scientific explanation on the basis of their use of laws of nature may raise objections. Moreover, the very concept of causality that appears in the mechanistic explanation refers to regularity (Craver & Kaiser, 2013; Miłkowski, 2013b). This means that with the right formulation of the concept of "law of nature" mechanistic explanation and deductive-nomological explanation may prove to be uncompetitive (Anderson, 2011; cf. Brożek, 2015).

Secondly, there is a dispute within the philosophy of science about whether this discipline should rather be of a prescriptive or descriptive nature. In other words, the question is whether philosophy of science should only explicate actual scientific practice or rather determine the way for scientists. It seems that the mechanistic view incorporates arguments from both sides of the dispute. The very notion of "mechanism" is not artificial, but is derived directly from scientific practice. However, the mechanistic view formulates norms which distinguish explanatory models from non-explanatory ones. On the other hand, it is difficult to imagine a case of a completely accurate model of a phenomenon examined by neuroscience. To paraphrase a long discussion on the role of the concept of "truth" in science, perhaps we are doomed only to possibly models. This point is related to the last issue we would like to briefly comment upon.

Machamer, Darden and Craver's (2000) approach to mechanicism, which we have adopted in this paper, is marked by scientific realism. While within basic neuroscience it is possible to maintain such an approach, in the case of cognitive neuroscience the issue becomes more complicated (cf. Revonsuo, 2001; Brożek, 2011). Many explanations formulated by cognitive neuroscientists are very difficult to interpret in the spirit of scientific realism. This is because such theories operate not only at the level of observable brain structures, but they also refer to cognitive structures, which as a rule are treated as objects postulated by theory.

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