Pain: Modularity and Cognitive Constitution

Abstract

Discussions concerning the modularity of the pain system have been focused on questions regarding the cognitive penetrability of pain mechanisms. It has been claimed that phenomena such as placebo analgesia demonstrate that the pain system is cognitively penetrated; therefore, it is not encapsulated from central cognition. However, important arguments have been formulated which aim to show that cognitive penetrability does not in fact entail a lack of modularity of the pain system. This paper offers an alternative way to reject the modularity of the pain system, which is independent from, but consistent with, the presence of cognitive penetration. It is proposed that, given the current knowledge regarding the functioning and the structure of the pain system, there are good reasons to accept that certain central cognitive mechanisms are part of the pain system. It is argued that such a 'cognitive constitution' of the pain system entails that the pain system is not modular.

Both scientists and philosophers investigating painful experiences commonly assume that cognitive factors are able to influence how the pain is experienced[1]. For instance, the way we experience pain may be modified by beliefs and expectations regarding the painful stimulus (Benedetti et al., 2020; Koyama et al., 2005; Shih et al., 2019), the perceived control over a situation (Wiech et al., 2008), emotional states such as anxiety (Vlaeyen and Linton, 2000), and general traits like a tendency to catastrophize (Sullivan et al., 2011; Turner and Aaron, 2001). In

[1] Throughout the paper, I avoid using phrases such as 'pain perception' or 'the way in which pain is perceived' because, as argued in section 4, I do not believe that the pain system is a typical perceptual system.

fact, many current clinical approaches to treating chronic pain are aimed at changing the cognitive and emotional attitude of patients to their painful experiences (Coninx and Stilwell, 2001; Kerns et al., 2011). Relying on the importance of cognitive factors for the way we feel pain, it has been proposed that at least some cognitive modifications of painful experiences are examples of genuine cognitive penetration (Gligorov, 2017; Jacobson, 2017; Shevlin and Friesen, 2021). In particular, such arguments have been developed in reference to the phenomenon of placebo analgesia, i.e., a reduction in the experienced intensity of pain caused by a mere belief that an analgesic substance has been administered.

The cognitive penetrability of painful experiences has been treated as evidence that the pain system is not a modular, cognitively encapsulated system (Gligorov, 2017; Shevlin and Friesen, 2021). It seems that if cognitive states can modify the way pain is experienced, then the functioning of the pain system is not separated from the information provided by the mechanisms of central cognition. Nevertheless, such a move has been met with opposition. It has been argued that if we consider genuine modularity—as characterized in the classic works by Fodor (1983, 1985)— then even if placebo analgesia is an actual example of cognitive penetration, what is not obvious (see Casser and Clarke, 2022), the presence of such penetration does not imply the lack of modularity of the pain system (Clarke, 2021). Cognitive penetrability can be accounted for in terms of merely modifying inputs and outputs between modules within the pain system, while to reject modularity it has to be demonstrated that central cognitive mechanisms influence modules' proprietary databases, i.e. rules and data, according to which the modules process information.

This paper is an attempt to develop an alternative way to reject the modularity of the pain system, which is independent from, but consistent with, the presence of cognitive penetration. More precisely, I claim that given the current knowledge regarding the functioning of the pain

system, there are good reasons to accept that certain central cognitive mechanisms are part of the pain system. I argue that such a 'cognitive constitution' of the pain system entails that the pain system is not modular.

The paper starts by describing the connections between the phenomenon of placebo analgesia and the notions of cognitive penetrability and modularity. First, the reasons why placebo analgesia may be interpreted as a case of cognitive penetration are characterized (Section 1). In Section 2, the argumentation formulated by Clarke (2021) in order to show that cognitive penetration does not entail a lack of modularity is presented. Further, in Section 3, I introduce the notion of cognitive constitution in order to present an alternative approach to denying the modularity of the pain system. Subsequently, in Sections 4–6, I argue that it is plausible to accept that the pain system is cognitively constituted. More specifically, I claim that gathering information about the bodily state is one of the main functions of the pain system, that this function of the pain system is partially realized by mechanisms of central cognition, and because of that it is plausible to consider certain central cognitive mechanisms as part of the pain system.

1. Cognitive penetration and placebo analgesia

While it is commonly agreed that not every influence of cognitive mechanisms on the functioning of the pain system constitutes genuine cognitive penetration, in philosophical literature there is no unanimously accepted set of factors which distinguishes cognitive penetration from other cognitive influences. One idea, which often appears in philosophical works, is that cognitive penetration should cause a change of an experiential phenomenal character (Deroy, 2015; Firestone and Scholl, 2016; Jacobson, 2017; Shevlin and Friesen, 2021). For instance, due to cognitive penetration, a

pain may be felt as sharper or as more intense. According to this proposal, if cognitive influences only modify the functioning of the pain system without causing any phenomenal changes, or these changes are not related to the way in which pain is experienced but rather to a cognitive attitude to pain, then cognitive influences are not an example of cognitive penetration. Nevertheless, it is not the case that all approaches to cognitive penetration treat the presence of phenomenal changes as a necessary condition. In particular, this is not the case in the classic Pylyshyn (1999) paper which introduced the notion of cognitive penetration in the context of the division between perception and cognition.

Second, it is often claimed that the influence of cognitive penetration should be direct, in the sense that cognitive penetration should not consist merely in modifying input to the pain system. If an influence consists merely in modifying input, then such an influence does not, in fact, affect how the system processes the provided information (Firestone and Scholl, 2016; Jacobson, 2017; Macpherson, 2012). Hence, cognitive influences which cause experiential changes by modifying the attribution of attention are usually not treated as examples of cognitive penetration. In such cases, phenomenal changes are caused by a modification of input which is due to a new distribution of attentional resources (however, as noted by Macpherson, 2012, it is controversial as to whether it applies to all types of attention).

Third, according to many authors, the occurrence of cognitive penetration requires the presence of semantic coherence between the content of a cognitive state and a change in the sensory experience (Green, 2020; Jacobson, 2017; Macpherson, 2012). In other words, there should be a rational link between the content of a cognitive state and the character of the experiential change. For instance, if one fears that a stimulus will be very painful, and in virtue of this state the pain is more intense, then a semantic coherence is present. This is because the

cognitive state concerns the high intensity of pain and the experiential change consists of increasing intensity. On the other hand, if it were discovered that thinking about tomatoes makes pain less intense, such a curious phenomenon would not exhibit the semantic coherence required for cognitive penetration. The presence of such rational link is treated as an important component of cognitive penetration as if it occurs, then the role of propositional states in perception may be similar to the role of such states in reasoning. This may suggest that there is no strict division between perception and cognition, and the models of cognitive architecture should be formulated in a way that does not postulate such a division (Firestone and Scholl, 2016).

It has been argued that the phenomenon of placebo analgesia can plausibly serve as an example of a cognitive penetration (Gligorov, 2017; Jacobson, 2017; Shevlin and Friesen, 2021). Placebo analgesia is a well-documented phenomenon in which a person reports that pain weakens when she believes that an analgesic agent has been administered, even if only a placebo was given (e.g., Amanzio et al., 2001; Bingel et al., 2011; Waber et al., 2008)[2]. It is proposed that the analgesic effect occurs due to the function of descending pathways, which modulate spinal activity by release of endogenous opioids (see Atlas and Wager, 2012; Heinricher and Fields, 2013, and Wiech, 2016 for a review). Because placebo analgesia involves the modification of early levels of pain processing, it seems plausible to accept that the resulting experiential change actually concerns the phenomenal character of pain experience and not merely one's cognitive or emotional attitude toward felt pain. In addition, the influence of beliefs and expectations in placebo analgesia seems to be semantically coherent, as the expectation that a given substance will have an analgesic

[2] More specifically, using Green's (2020) terminology, if placebo analgesia is an example of cognitive penetration, then it modifies experiential content by changing a higher intensity into a lower one, but it does not 'enrich' content as it does not introduce any content which could not be present without cognitive influences.

effect is associated with experiential change consisting of a decrease of felt pain[3]. Furthermore, it is plausible that the effects of placebo analgesia cannot be fully explained merely in terms of a modification of the input to the pain system. In particular, while placebo analgesia is associated with some attentional modulation, it is not likely to fully explain the analgesic effect (see Wiech et al., 2008).

It should be noted that my goal is not to prove that placebo analgesia is undoubtedly an example of cognitive penetration. For instance, it is debated as to what degree it can be explained by conditioning based on previous experiences in medical settings, rather than by the effects of beliefs and expectations. (see Atlas and Wager, 2012; Colloca and Miller, 2011). In addition, the fact that placebo analgesia involves an influence on low-level mechanisms may, in fact, serve as evidence that it is not an example of cognitive penetration. In particular, it has been argued that placebo analgesia may be interpreted as a phenomenon which consists merely in modifying inputs received by the pain system (see Casser and Clarke, 2022). Furthermore, in the subsequent section, I present an argument by Clarke (2021) aimed at showing that the presence of cognitive penetration does not entail a lack of modularity. Against this background, I develop an alternative approach to denying the modularity of the pain system based on the notion of 'cognitive constitution.'

[3] However, if semantic coherence is characterized in terms of an inferential relation between the content of a cognitive state and the content of a sensory state (see Gross, 2017), one may doubt whether there is such a link in the case of placebo analgesia. For instance, an expectation that pain intensity will be lower due to a painkiller does not entail that the occurring bodily disturbance is less bad and should be associated with less intense pain.

2. Cognitive penetration and modularity

Modularity is a relative notion, in the sense that a system is modular if it is encapsulated from influences of some other system. Because of this, the lack of cognitive penetrability of a given system does not imply that this system is fully encapsulated (see Zeimbekis and Raftopoulos, 2015). For instance, a sensory system *S* may be cognitively impenetrable, but its functioning may be influenced by some other sensory system *P*. In practice, philosophers and cognitive scientists are mainly interested in the encapsulation of certain systems from influences of mechanisms that constitute central cognition. While it may initially seem that cognitive penetration implies a lack of encapsulation from central cognition, an important argumentation has been developed in order to show that this is not the case. Below, I focus on the argument presented by Clarke (2021), but similar theses have been proposed by Quilty-Dunn (2020), who believes that attentional influences, which may be involved in certain forms of cognitive penetration, do not lead to a violation of the encapsulation of sensory systems, and Mylopoulos (2021), who argues that the motor system is modular despite being cognitively penetrated.

This argument relies on distinguishing between a module's input, output, and its proprietary database. Input is the information that comes to a system's module from outside the system or from its other modules; output is the information generated by a module and passed to other modules or passed outside the system, and the proprietary database is a set of rules and data used by a module in order to transform input into output. The situations in which a cognitive influence merely modifies inputs received by a system or outputs generated by a system are not usually treated as examples of cognitive penetration. On the other hand, penetration occurs when

cognition influences what happens at 'joints' between the system's modules by modifying the output of one module received as input by another, or influences modules' proprietary databases.

Relying on an interpretation of Fodor's classic works on modularity (Fodor, 1983, 1985), it has been proposed that not all forms of cognitive penetration entail a lack of modularity. More specifically, cognitive penetration is consistent with the modularity of a given system if cognition influences joints between the system's modules, but not their proprietary databases (Clarke, 2021). In other words, a system is encapsulated from central cognition, as long as the proprietary databases of all of its modules are not determined, at least partially, by the functioning of some central cognitive mechanisms. The mere fact that such central mechanisms modify information which is passed by one system's module to another does not pose a threat to modularity. For instance, a system *S* may include modules *M1* and *M2*, such that *M1* as its output generates information *A*, which serves as input for *M2*. If the cognitive influence consists in merely changing *A* (the output of *M1*) into *B*, which is then input to *M2*, the modularity of the system *S* is preserved. However, if the module *M1* processes information according to the rule *R1* when receiving information *I1* from central cognition, but uses a distinct rule *R2* when information from the central cognition is *I2*, then the system *S* is not encapsulated from central cognition.

These distinctions demonstrate that the presence of placebo analgesia, even if it is a genuine example of cognitive penetration, does not have to be problematic for the modularity of the pain system. This is because cognitive mechanisms responsible for placebo analgesia may influence the way in which pain is experienced by affecting 'joints' between some of the modules of the pain system without determining their proprietary databases. For instance, it may be the case that the cognitive influence of placebo analgesia on early spinal processing consists only in modifying the input generated by some early modules of the pain system which is received by some further

modules of this system. Nevertheless, the rules according to which modules of the pain system process their input into output remain unaffected. In this case, placebo analgesia is an example of cognitive penetration which is compatible with the modular character of the pain system.

The above problem is distinct from a common criticism formulated in relation to putative cases of cognitive penetration; namely, that cognitive influences only affect a post-perceptual interpretation of a sensory state and not the perceptual processing itself (Deroy, 2015; Firestone and Scholl, 2016; Zeimbekis and Raftopoulos, 2015). It may be the case that a cognitive mechanism genuinely penetrates the functioning of a sensory system *S*; for instance, by changing the output of a sensory module of *S* before it is received as an input by another sensory module of *S*. However, according to Clarke (2021), such an influence is coherent with encapsulation of *S* as the influence of the cognitive mechanism does not modify the proprietary databases of modules composing *S*, but only inputs and outputs between modules of *S*. For an analogous reason, the argument by Clarke (2021) is not the same as proposing that a certain cognitive influence is not a cognitive penetration because it only affects the input received by a sensory system and not its internal functioning.

In consequence, even if placebo analgesia is a form of cognitive penetration, it may be the case that it is consistent with the modularity of the pain system. In the next section, I introduce the notion of cognitive constitution, which offers a way of denying the modularity of the pain system that is independent from cognitive penetration.

3. Modularity and cognitive constitution

Let us once again consider the situation in which a system *S* has modules, *M1* and *M2*, such that the module *M1* produces output *A* which serves as an input for the module *M2*. In addition, there is a central cognitive mechanism *CCM* which may change the output *A* into *B* before it reaches the module *M2*. As argued earlier, such an influence does not automatically threaten the encapsulation of the system *S*.

Nevertheless, it is easy to observe that the considered central cognitive mechanism *CCM* also functions as a module which takes *A* as its input and produces *B* as its output (see Clarke, 2021 for this observation). In this context, we may ask whether *CCM* is separate from the system *S*, or perhaps, in fact, it is a part of *S*? If *CCM* is a part of the system *S*, then the system *S* is not modular, because it is partially constituted by a mechanism *CCM* which is not encapsulated from the central cognition as it itself is a mechanism of central cognition. Generally, central cognitive mechanisms are not treated as being encapsulated from central cognition. It is rather assumed that they heavily influence each other, and the question regarding encapsulation from central cognition is usually asked in regard to sensory mechanisms rather than mechanisms composing the central cognition. In fact, minimally, if *CCM* is a mechanism of central cognition, then there is at least one mechanism of central cognition whose functioning determines the proprietary database of *CCM*; this central mechanism is *CCM* itself. For instance, the way in which *CCM* functions may determine that if *CCM* receives input *A*, it processes this input in accordance with a rule *R1*; but when input *B* is received, a distinct rule *R2* is adopted. Further, I use the term, 'cognitive constitution,' to name a situation in which a system is not modular due to having a central cognitive mechanism as its part, and I argue that the pain system is likely to be cognitively constituted.

One may oppose the above observation by proposing that if a cognitive mechanism *CCM* belongs to the pain system, then the proprietary database of *CCM* becomes a part of the fixed proprietary database of the whole pain system. Therefore, the pain system is still modular as its proprietary database is stable and is not modified by some cognitive influence. Nevertheless, I believe that there are two problems with this idea. First, if the cognitive constitution occurs, then the proprietary database of the whole system is partially determined by the functioning of a central cognitive mechanism *CCM*. Consequently, it is difficult to maintain that the pain system is fully informationally encapsulated from central cognition because the way in which it functions is shaped by the information processed by a central cognitive mechanism. Second, central cognitive mechanisms are often not encapsulated from other central cognitive mechanisms. Because of that, it is likely that if a mechanism such as *CCM* is part of the pain system, then its proprietary database can be modified by activities of other central cognitive mechanisms which do not belong to the pain system.

When considering complex systems, cognitive constitution and modularity may be interpreted as gradual notions. From such a perspective, a system is minimally non-modular if at least one of its parts is such that its proprietary database is determined by activities of a central cognitive system; it is maximally non-modular if it is also true about all of its parts. Similarly, a system is minimally cognitively constituted if at least one of its parts is a mechanism of central cognition, and it is maximally cognitively constituted if all of its parts are mechanisms of central cognition. In this paper, I aim only to argue for the minimal cognitive constitution of the pain system and so for its minimal non-modularity. I believe this is the usual approach in philosophical debates regarding the modularity of the pain system. In particular, the authors who claim that the

pain system in not modular in virtue of being cognitively penetrated do not claim that all parts of the pain system are cognitively penetrated, but only that such parts exist.

More specifically, I claim that cognitive mechanisms, processing beliefs and expectations relevant for placebo analgesia, are part of the pain system. It should be noted that this claim is largely independent from the question regarding whether placebo analgesia is an example of cognitive penetration.

First, cognitive penetration does not entail cognitive constitutions. For instance, if a cognitive mechanism *CCM* in a direct and semantically coherent way modifies the phenomenal character of an experience by influencing the output of a module *M1* belonging to the system *S*, then *CCM* cognitively penetrates the system, *S*, even if *CCM* is not a part of *S*.

Second, cognitive constitution does not entail cognitive penetration. It is possible that a central cognitive mechanism *CCM* is a part of the system *S*, but its influence on the functioning of *S* does not meet the criteria of cognitive penetration. In particular, such a mechanism may influence *S* in a way that is not appropriately semantically coherent or direct. On the other hand, there is no incompatibility between cognitive constitution and cognitive penetration such that a mechanism which cognitively constitutes a system *S* may also influence its functioning in a way that satisfies the requirements of cognitive penetration.

Furthermore, my considerations regarding modularity and cognitive constitution should be distinguished from related but distinct debates in neuroscience regarding the specialization of the elements of the cognitive system. In particular, it has been discussed whether the same brain region can participate in various functions and whether its contribution to various functions is the same or not (see Anderson, 2007). Similarly, it is asked if a neural network is only specialized if all elements of such a network are specialized, or is it sufficient that the particular combination of

elements composing the network is specialized (see Fedorenko and Thompson-Schill, 2014). However, specialization and modularity are logically independent notions. An element of the system may be specialized in the sense of participating only in one function, but may not be modular due to the fact that a central cognitive mechanism influences its proprietary database. Similarly, an element may be general in the sense of contributing to various functions, but nevertheless, it can still be modular if, in the context of each performed function, its proprietary database is not influenced by a mechanism of central cognition. Analogously, there is also no entailment between specialization and cognitive constitution: it is possible for a specialized system to have a part which is a mechanism of central cognition and for a non-specialized system to be disjoint from mechanisms of central cognition.

In the subsequent sections, I focus on arguing that the pain system is cognitively constituted by mechanisms involved in placebo analgesia, and I leave open the question of whether placebo analgesia is an example of cognitive penetration. I start by discussing the functions of the pain system and argue that its function requires that we include descending modulatory pathways as part of the pain system. Furthermore, focusing on the descending pain pathway known as the PAG-RVM pathway, I show that it is plausible to assume that this pathway is partially constituted by mechanisms of central cognition relevant for placebo analgesia. If this is the case, then the pain system is cognitively constituted and is not modular.

4. Function of the pain system

One influential way of thinking about pain is to treat the pain system as a perceptual system analogous to the visual or auditory system. In this perspective, the main function of the pain system

is to gather information about the state of bodily parts and show that there is a disturbance or threat concerning them. Such a view naturally combines with the indicative representational approach to pain experiences (e.g., Bain, 2013, Cutter & Tye, 2011; Gray, 2014). According to this approach, painful experiences may accurately or inaccurately represent the state of a bodily part. The unpleasant phenomenal character of pain experiences is determined by the representational content concerning the state of a bodily part.

Nevertheless, there are serious empirical and philosophical reasons which suggest that the perceptual and indicative representational view of pain is inaccurate, or at least incomplete. First, the presence of pain and the presence of bodily disturbances is not so strongly correlated as it may initially seem (see Casser, 2020; Corns, 2014; Hardcastle, 1997; Melzack et al., 1982; Wall, 1979). There are many common pains, like tension headaches, which are not associated with any disturbance or threat. On the other hand, even very serious injuries sometimes do not cause felt pain, and the onset of pain is often substantially delayed. Similarly, the severity of pain is not strongly associated with the severity of injury, and in many cases a similar bodily disturbance leads to pain in some individuals but not in others. If the pain system is a perceptual representational system, then it seems that it serves its role poorly by frequently misrepresenting the state of the body.

Second, the perceptual approach to the pain system has problems explaining what is probably the most striking aspects of pain phenomenology, i.e., that pains are unpleasant and motivating. In particular, such an approach has problems in addressing the so-called 'messenger-shooting' objection (see Bain, 2019; Boswell, 2016; Brady, 2015; Cutter and Tye, 2014; Jacobson, 2013 for a discussion). Intuitively, it seems that painful experiences do not merely represent that there is something wrong with a bodily part; they themselves are unpleasant; they provide *prima*

facie reasons for their own removal and not only for removal of an underlying disturbance. However, from the perceptual, indicative, representational perspective, a painful experience is like a messenger who brings bad news, i.e., that there is some threat or disturbance, but one that is not bad in itself. In consequence, it is unclear in virtue of what a pain provides reasons for its own removal.

Third, a significant part of the pain system is not devoted to recognizing whether there is a disturbance in some bodily parts, but to modulating the incoming nociceptive information and further modulating the conscious painful experience (see Casser, 2020; Gligorov, 2017; Hardcastle, 1997). Such modulatory activities are already present at an early stage in the pain system, as spinal pain gates open or close, not merely relying on input from nociceptive receptors and fibers, but also by taking into account information provided by other afferent fibers (see Melzack and Wall 1965; Mendell 2014; Peláez and Taniguchi 2016). The modulatory function of the pain system is even more apparent when one considers its top-down aspects, as there exist descending pathways which, by using cognitive and emotional data, influence the processing of nociceptive information and whose functioning leads to phenomena such as placebo analgesia (see Heinricher and Fields, 2013; Shih et al., 2019; Wiech, 2016).

The above considerations suggest a perspective on the functioning of the pain system that is distinct from the perceptual perspective. The major function of the pain system does not seem to consist in representing the state of the bodily parts, but rather in gathering of a variety of sensory and non-sensory data, evaluating the state of the body and generating states which motivate conducting or stopping certain bodily actions (see Casser, 2020). Such an approach is often combined with interpreting painful experiences as commands with imperative content, proscribing, for instance, that a protective action should be conducted (see Barlassina & Hayward,

2019; Klein, 2007; Martínez, 2015 for variants). In this perspective, it is understandable that pain is not strongly correlated with bodily disorder. Sometimes an actual disorder may not cause the pain because the combination of nociceptive data with other information processed by the pain system leads to a conclusion that no motivational state should occur. For instance, a pain may be felt as less intense if there is also information that we are in control of the painful stimulus, or when a larger threat is present, resulting in some nociceptive input being ignored. On the other hand, the holistic assessment of bodily information may also result in an increase in felt pain, even if no serious disorder is present. For example, the same stimulus may be felt as more painful if a person is anxious about the expected pain (e.g., Benedetti et al., 2020).

The above approach to pain also takes into account the unpleasant and motivational character of pain by characterizing painful experiences as states whose main role is not to present the properties of bodily parts, but as states whose function is to motivate behaviors. It should be noted that while this approach naturally combines with imperativism regarding the content of pain, it does not presuppose any particular version of imperativism. It can also be expressed in terms of other theories of pain; for instance, functional theories, according to which, the unpleasantness of pain consists of relations between pain and other mental states (Aydede, 2017).

Furthermore, if the main function of the pain system is to evaluate the state of the body by relying on gathered information to issue motivational commands, it is clear why various mechanisms of the pain system serve a modulatory function. Such mechanisms play a role in establishing—by utilizing the obtained information about the state of the body and attenuating or strengthening the nociceptive input—whether a motivational state should occur. By attributing the evaluative and motivational function to the pain system, I do not want to deny that it may also have some representational aspects which inform about the state of bodily parts. For my further

consideration, it is only relevant that gathering information, evaluating the state of the body, and issuing motivational commands are the major functions of the pain system.

5. Structure of the pain system

The major functions of the pain system are realized by ascending and descending pathways. The ascending pathways process the nociceptive information concerning various bodily parts as well as other information; for instance, tactile, which modulates nociceptive input (Melzack and Wall 1965; Mendell 2014; Peláez and Taniguchi 2016). In virtue of their functioning, a decision can be reached regarding whether a state of some bodily fragment is such that a painful, motivational command should be issued. The descending pathways allow processing of the ascending nociceptive information in the context of a holistic view of the current situation of the organism by relying, *inter alia*, on data regarding beliefs, expectations, emotional states, and memories (Atlas and Wager, 2012; Heinricher and Fields, 2013; Shih et al., 2019; Wiech, 2016). Due to the functioning of descending pathways, the information being processed in the ascending pathways can be further modulated. In virtue of this modulation, the decision on whether a painful command should be issued can be made by taking into consideration the state of the whole organism.

While the many details regarding the function and composition of ascending and descending pain pathways are still unknown, we currently have a general picture regarding the physiology of the pain system. The ascending pathways are composed of receptors and afferent fibers carrying information to the spinal pain gates, which subsequently pass the information to the brain structures. The pain gates integrate information from various afferent fibers and modulate nociceptive information, which allows for an initial evaluation of the state of the bodily part in

order to decide whether it should lead to a painful experience. The information processed by pain gates is projected to the thalamus and the homeostatic control regions in the medulla and the brain stem. Furthermore, through the brain stem it reaches the hypothalamus and the ventral forebrain.

One of the major and best-known descending pathways is the PAG-RVM pathway, named after two brain regions: the midbrain periaqueductal gray (PAG) and the rostral ventromedial medulla (RVM), whose activity modulates the experiences of pain and are also related to the occurrence of placebo analgesia (see Heinricher and Fields, 2013; Wiech, 2016 for detailed descriptions). In particular, the PAG structure receives information from mechanisms located in regions, such as the frontal lobe and amygdala, which process cognitive information relevant for reasoning and emotional factors. Due to the involvement of these regions, it is likely that the PAG-RVM pathway contains mechanisms which provide cognitive information—e.g., regarding beliefs and expectations—to structures such as the PAG. The PAG integrates this data, and further, through the RVM, it influences the lower-level, spinal pain structures through the release of endogenous opioids (see Fields, 2018 for a detailed empirical model). It is also believed that the placebo analgesia involves the functioning of the PAG-RVM pathway. More specifically, this phenomenon occurs in part due to the fact that cognitive mechanisms process relevant beliefs and expectations and provide input to the PAG structure, which finally results in a modification of spinal processing of the nociceptive information.

It may be observed that in both ascending and descending pain pathways, the same general types of mechanism can be distinguished. First, there are mechanisms which integrate the various relevant input. Such a function is served by both pain gates in ascending pathways and the PAG in the PAG-RVM descending pathway. Second, there are mechanisms that provide information to integrating structures. For instance, afferent fibers in the ascending pathways and mechanisms in

the amygdala and frontal lobe, which provide emotional and cognitive data to the PAG. Third, there are mechanisms which further process integrated output provided by the integrating structures, such as pain gates and the PAG, whose activity influences the characteristics of pain experiences. For example, in the case of ascending pathways, such mechanisms are located in the thalamus and medulla, and in the case of the descending PAG-RVM pathway, are located in the spinal dorsal horn.

Inclusion of these three types of elements which compose ascending and descending pathways is well justified given the major functions of the pain system. Due to the presence of integrating structures, the pain system is able to holistically evaluate the state of the body. In virtue of mechanisms providing input to integrating structures, it can gather information relevant for bodily evaluation. Finally, because of the functioning of mechanisms which further process the output provided by the integrating structures, motivational commands can be issued.

The above observations allow the formulation of an argument in favor of the cognitive constitution of the pain system. As argued above, the pain system gathers and integrates information in order to provide a holistic evaluation of the bodily state and issue motivational commands. These functions of the pain system are plausibly realized by mechanisms belonging to this system as it is generally plausible that the main functions of a given system are realized by some of its mechanisms. The information gathering is partially realized in ascending pathways by mechanisms providing input to the pain gates. These mechanisms are commonly treated as part of the pain system. However, the information allowing for the evaluation of the bodily state is also gathered by the descending pathways such as the PAG-RVM pathway. Within this pathway, the function of information gathering is realized by mechanisms which provide input to the integrating PAG structure. Some of these mechanisms are mechanisms of central cognition, which process

beliefs and expectations relevant, *inter alia*, for the occurrence of placebo analgesia. Further, I refer to them in short by the name 'EBMs' ('expectation and belief mechanisms').

In consequence, one of the main functions of the pain system—i.e., information gathering—is partially realized by EBMs included within the PAG-RVM pathway. Because of this it is plausible to include them as parts of the pain system. If EBMs are part of the pain system and are mechanisms of central cognition, the pain system is cognitively constituted. In fact, the role of EBMs in the pain system is analogous to the role of early mechanisms in the ascending pathways which provide input to integrating structures such as pain gates. EBMs are mechanisms which constitute the early part of the PAG-RVM pathway and provide cognitive input to the integrating mechanisms in the PAG structure. Both mechanisms which provide input to the pain gates in the ascending pathways, and EBMs, which provide inputs to the PAG in the descending PAG-RVM pathway, jointly contribute to one of the major functions of the pain system. It would seem *ad hoc* to accept that mechanisms which provide input to the pain gates belong to the pain system, while accepting that the same is not true about EBMs.

There are several ways in which one may attempt to reject this reasoning. First, it may be rejected that gathering information about the bodily state is an important function of the pain system. Nevertheless, as argued in Section 4, the pain system provides a holistic evaluation of the bodily situation, and gathering various types of information is an integral component of achieving this goal. Furthermore, mainstream characterizations of the pain system ascribe to it such function as they include within the pain system's bottom-up nociceptive mechanism whose role is to provide information to spinal structures.

Second, one may postulate that EBMs do not contribute to the gathering of bodily information by the pain system by providing input regarding certain beliefs and expectations.

However, this is unlikely for two reasons. First, the integrating PAG structure receives information from regions such as the central lobe, which is plausibly involved in processing propositional states such as beliefs. In consequence, it is likely that there are mechanisms—here named EBMs—which provide information concerning such states. Second, the PAG-RVM pathway is involved in occurrences of placebo analgesia and this phenomenon happens, at least partially, due to the influence of beliefs and expectations on the elements of the pain system. Because of this, it is plausible that there are mechanisms which provide information about beliefs and expectations to structures such as the PAG.

Third, it may be proposed that EMBs are parts of the descending PAG-RVM pathway, but this pathway is not a part of the pain system. However, such a move is not plausible given the function of the pain system described in Section 4. To evaluate the bodily state and establish whether motivational commands should be issued, the pain system requires descending pathways which modulate processing of nociceptive information by relying on a variety of data. Stating that the PAG-RVM pathway is not a part of the pain system would lead to a dubious theoretical claim that some main functions of the pain system are realized by mechanisms which do not belong to the pain system.

Fourth, one may argue that while the PAG-RVM pathway is a part of the pain system, its EBMs components processing beliefs and expectations relevant for placebo analgesia are not mechanisms of central cognition. Nevertheless, I believe that this approach is also problematic, particularly because these mechanisms process information of a type that is characteristic of central cognition. Expectations and beliefs leading to placebo analgesia are mental representations which have conceptual, propositional content characterizing the represented entities in an amodal way, i.e., without the phenomenal character typical of particular sensory modalities such as vision or

audition. In addition, the concerned beliefs and expectations have a syntactic structure which allows them to be expressed in language and enter into logical relations with other representations processed by central cognition. The fact that EBMs process such mental representations suggest that they are mechanisms of central cognition.

This claim is also strengthened by the fact that the way in which beliefs and expectations relevant for placebo analgesia are processed is heavily influenced by background propositional knowledge and other cognitive information. For instance, the occurrence and strength of placebo analgesia is influenced by observing another person receiving similar treatment (Bieniek and Bąbel, 2022; Colloca and Benedetti, 2009), the presence of a medical setting (Bingel et al., 2011), and the perceived quality of analgesic substance (Waber et al., 2008). While the presence of a cognitive penetration of sensory systems is controversial, it is common for mechanisms of central cognition to influence each other. The presence of such cognitive influences on the considered beliefs and expectations additionally suggests that the mechanisms processing them belong to central cognition.

There is also a fifth way to refute the cognitive constitution of the pain system. It may be claimed that the PAG-RVM pathway is a part of the pain system; the EBMs which process beliefs and expectations relevant for placebo analgesia are mechanisms of central cognition, but EBMs are not parts of the PAG-RVM pathway. In particular, the PAG-RVM pathway may start with the PAG structure integrating various inputs, but EBMs providing cognitive input to the PAG are outside the PAG-RVM pathway and therefore, also outside the pain system. In other words, it would mean that the analogy between mechanisms providing input to the pain gates and EBMs is weaker that it may initially seem. While the mechanisms providing input to the pain gates are part of the pain system, EBMs have some features which does not allow their inclusion in the pain

system, even if they provide input to integrating structures of the PAG-RVM pathway. I believe that this approach poses the most serious threat to the cognitive constitution of the pain system, and I discuss it in detail in the subsequent section.

6. Scope of the descending pathway

There are three general directions in which one may develop an argument that EBMs are outside the borders of the PAG-RVM pain pathway. First, the problem may concern the way in which EBMs are related to other mechanisms. Second, it may be something intrinsic to EBMs—for instance, the format of information they process or the way in which they process information—that justifies positioning them outside of the PAG-RVM pathway. Third, one may propose that including EBMs in the PAG-RVM pathway leads to unacceptable theoretical consequences for our understating of sensory and cognitive systems.

6.1 Relations to other mechanisms

Regarding the first direction, it may be observed that output generated by EBMs via processing beliefs and expectations is likely to be utilized not only by the PAG-RVM pathway but also by various other cognitive mechanisms. For instance, an expectation that pain will become less intense may be used as a premise for various reasonings concerning further plans and actions. In consequence, it may be proposed that EBMs should not be included as a part of the PAG-RVM pathway as they are not uniquely related to this structure but are equally strongly connected to various other mechanisms and systems.

I believe that the presence of such a situation does not justify the exclusion of EBMs from the PAG-RVM pathway because it is not uncommon for mechanisms to simultaneously belong to more than one system. A good example is provided by the sensory flavor system which, to a significant extent, comprises mechanisms which are also part of other sensory systems. In particular, the flavor system shares mechanisms with orthonasal olfaction, the tactile system, the thermal system, the trigeminal system, and possibly even audition (see Auvray and Spence, 2008; Spence et al., 2014). In fact, even within the pain system one may find examples of mechanisms which also belong to other systems. For example, many mechanisms in ascending pathways providing information to pain gates also play a role in touch, thermal perception, or proprioception (see Melzack and Wall 1965; Mendell 2014; Peláez and Taniguchi 2016).

Nevertheless, it may be the case that the problem with EBMs lies not in their relations to other mechanisms of central cognition, but in relation to the structure of the PAG-RVM pathway. In particular, the pain system is commonly treated as one which processes information relevant to generating painful experiences (Casser and Clarke, 2021). However, one may doubt whether EBMs do in fact process such information; they may merely passively transmit it to the structures such as the PAG. If that is the case, then the mechanisms in the PAG structure should be considered as a starting point of the PAG-RVM pathway, and EBMs should be considered as external elements. Nevertheless, it is unlikely that EBMs play only a passive role. The beliefs and expectations relevant for placebo analgesia are not somehow passively transmitted, but are generated relying on a variety of information regarding other beliefs about the current situation and a background knowledge about painkillers and the medical setting (e.g., Bieniek and Bąbel, 2022; Colloca and Benedetti, 2009; Waber et al., 2008). In fact, the role of EBMs is likely to be far less passive than that of nociceptive mechanisms providing input to the spinal pain gates.

Alternatively, one may propose that the whole processing work regarding the relevant beliefs and expectations occur in mechanisms which provide input to EBMs, and EBMs in fact merely transmit information to the PGA. While such an option is not internally inconsistent, it is also not particularly plausible. First, the empirical state of the art does not provide data suggesting the presence of separate mechanisms that do not process information relevant for placebo analgesia, but merely transmit it to the PGA (see Heinricher and Fields, 2013; Wiech, 2016). Second, proposing such mechanisms is problematic from the theoretical perspective as their presence seems redundant because their role would consist merely in receiving input *A* and providing output *A* without processing it in any relevant way.

Another doubt may be formulated by regarding the contribution of EBMs to the functioning of the pain system. Without the presence of EBMs, the functioning of the pain system would be diminished because the pain system would not be able to gather certain relevant information and integrate it with other data. However, even without EBMs we would be able to experience common types of painful experiences, and many modulatory mechanisms of the pain system would still be able to function. In consequence, one may propose that the contribution of EBMs to the functioning of the pain system is too small to plausibly interpret EBMs as part of the pain system. Nevertheless, the same reasoning also applies to other modulatory mechanisms of the pain system which are commonly treated as its parts. For instance, the ascending pain pathways are partially constituted by mechanisms which modulate the functioning of spinal pain gates through the activities of certain non-nociceptive fibers (so-called 'large fibers' in the classic model of pain gates, see Peláez and Taniguchi, 2016; Wall, 1996). However, even without these mechanisms, various typical painful experiences could still occur, and other modulatory mechanisms could still serve their function. In general, painful experiences are commonly interpreted as complex states having

sensory, affective, emotional, and motivational components which may be affected separately by various dysfunctions of the pain system while other components remain intact (see Hardcastle, 1997). Because of this, the pain system seems to be largely composed of mechanisms where each of them contributes to the character of painful experiences, but the majority of functions of the pain system can be still realized even if one of those mechanisms is no longer functioning. From this perspective, EBMs are typically part of the pain system; they contribute to the top-down modulatory function of the pain system, but even without them many functions of the pain system would still be present. In consequence, the functional contribution of EBMs does not seem to be less than many other mechanisms that are treated as part of the pain system.

6.2 Intrinsic features of EBMs

If the problem with treating EBMs as a part of the PAG-RVM pathway does not consist of the relation of EBMs to other mechanisms, then maybe it is related to the intrinsic features of EBMs. First, it may be proposed that the cognitive information regarding propositional representations processed by EBMs is not the type of information that can be processed by mechanisms belonging to the pain system. However, that cannot be the case, because the PAG—the integrating structure of the PAG-RVM pathway—has to process cognitive information provided by EBMs in order to combine it with other types of data.

Second, one may argue that the problem is not related to the processed information but to the way in which it is processed. In particular, it may be the case that parts of the pain system process information according to a specific set of rules, which differs from the set of rules employed by the EBMs. Such an argument might be plausible if the pain system were a single

module with its own proprietary database, or it were composed of similar modules with highly similar proprietary databases. Nevertheless, this is unlikely given the complexity and the heterogenous character of the pain system, which comprises many ascending and descending pathways that process a variety of sensory and nonsensory information. Furthermore, this information is used to generate distinct, sensory, evaluative, and motivational aspects of painful experiences. In such a situation, it is implausible to postulate that all mechanisms of the pain system possess very similar proprietary databases, because they process distinct types of information and use it for distinct purposes. In consequence, it seems *ad hoc* to exclude EBMs from the pain system due to their rules of information processing.

6.3 Theoretical consequences

However, even if including EBMs as a part of the pain system is not problematic due to its internal characteristics or relations to other mechanisms, it is still possible that such a cognitive extension of the pain system has some negative theoretical consequences. First, it may be believed that the postulation that the pain system is cognitively constituted is unlikely, given the fact that in usual situations we are hardly able to influence our pain by cognitive states. For instance, when having an ordinary tension headache, one may be aware that pain is not associated with any serious disorder, and can expect the painful sensation to cease after some time. However, having such mental states does not seem to lessen the pain. Nevertheless, the input provided by cognitive mechanisms is only one of many types of input processed by the pain system, and the general thesis regarding the cognitive constitution of the pain system does not entail the impact of the cognitive input in various specific situations of being in pain. In consequence, one may

consistently claim that the pain system is cognitively constituted, and thus maintain that in many common situations of being in pain, the phenomenal character of pain is mainly determined by the ascending sensory input, and that the influence of top-down cognitive factors is limited (see Casser and Clarke, 2022 for this observation).

A second worry is that if we include central cognitive mechanisms such as EBMs as part of the pain system, there is no reason why we should not treat the whole central cognition as constituting the pain system. For instance, if EBMs are part of the pain system, then perhaps mechanisms which provide input to EBMs are also part of the pain system. However, it seems implausible that the pain system encompasses the whole of central cognition. Nevertheless, the proposal developed in this paper explicitly blocks such extensions. The pathways constituting the pain system, such as ascending pathways and the descending PAG-RVM pathway, are organized around integrating structures such as pain gates or the PAG. Pain pathways are composed of mechanisms of integrating structures, further mechanisms which process the integrated output, and mechanisms which provide input to the integrating structures. Mechanisms such as EBMs are part of the pain system because they provide input to the mechanisms in the integrating PAG structure, but EBMs themselves are not mechanisms of any integrating structure of the PAG-RVM pathway. In consequence, mechanisms providing input to EBMs are not part of the pain system. Such a proposal is motivated by the main function of the pain system as characterized in Section 4. The pain system (a) gathers information about the state of the body due to mechanisms providing input to integrating structures, (b) evaluates the state of the body in virtue of the functioning of the integrating structure processing the gathered information, and (c) issues conscious, motivational commands due to further mechanisms which process output provided by the integrating structures.

Finally, it may be claimed that if we agree that the pain system is cognitively constituted, we will also have to agree that various sensory systems, like vision or audition, are also cognitively constituted. I believe that the unacceptability of such an idea is not obvious. The division between perception and cognition is notoriously difficult to establish, and it may be the case that there is an overlap between, for instance, the visual system and central cognition. However, there are also reasons which make the cognitive constitution of the pain system more plausible than the cognitive constitution of the visual or auditory system. As argued in Section 4, the function of the pain system is not merely to provide information about the properties of bodily parts, but to make a holistic evaluation of bodily states and issue motivational commands. Because of this, it is plausible to include within the pain system, not only mechanisms processing nociceptive information, but also mechanisms that process cognitive information regarding the state of the body. The situation is less obvious in the case of perceptual modalities such as vision or audition because in contrast to the pain system their main function is to provide information about the entities in the environment. Hence, it is more plausible that they are constituted by mechanisms which process visual or auditory sensory information and not by mechanisms processing information that is characteristic of central cognition. While there may be cognitive mechanisms which do influence the processing of visual or auditory information, it is more likely to interpret such influences as modifications introduced by mechanisms external to the visual or auditory system.

7. Conclusions

The discussion regarding the modularity of the pain system has been focused on the question concerning the cognitive penetration of pain mechanisms. However, while there are reasons to believe that phenomena such as placebo analgesia demonstrate cognitive penetration of the pain system, important arguments have been formulated in order to show that the pain system can be modular despite the presence of cognitive penetration. Nevertheless, there is another way to demonstrate that the pain system is not modular, i.e., by showing that the pain system is cognitively constituted. I have argued that due to the function of the pain system, it is plausible to postulate that the pain system is partially constituted by descending pathways, which include some mechanisms of central cognition. In consequence, the pain system is not modular because some of its mechanisms are also mechanisms of central cognition.

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