THE FUNCTION OF PAIN

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ABSTRACT

Various prominent theories of pain assume that it is pain's biological function to inform organisms about damage to their bodies. I argue that this is a mistake. First, there is no biological evidence to support the notion that pain was originally selected for its informative capacities, nor that it currently contributes to the fitness of organisms in this specific capacity. Second, neurological evidence indicates that modulating mechanisms in the nociceptive system systematically prevent pain from serving a primarily informative role. These considerations threaten to undermine standard perceptual and representational accounts of pain.

KEYWORDS Pain, Biological Function, Modulation, Perceptualism, Representationalism

1. Introduction

Miss C. led a short, pain-free life. She didn't feel pain when she accidentally bit off the tip of her tongue, when she suffered third-degree burns from kneeling on a hot radiator, or when she was subjected to strong electric shock, hot water, and prolonged ice-baths. Moreover, she didn't feel pain when she put extreme pressure on her joints, when she sat or stood with bad posture, or when she slept in unnatural positions. As a result, her knees, hip, and spine were affected by a condition called *neuropathic arthropathy*, or 'Charcot joint', which led to the progressive

degeneration and infection of her skin and bones. Miss C. died of the effects of this condition at the age of 29 [Melzack and Wall 2008].

The sad cases of those affected by *congenital analgesia*, or pain insensitivity, are commonly interpreted as compelling evidence for the adaptive value of pain's biological role [Nagasako et al. 2003]. Many have taken this role to be that of a standard sensory system, dedicated to inform organisms about their (bodily) environment [Armstrong 1968; Pitcher 1970; Tye 1995; Hardcastle 1997]. On this view, pain's primary function is to inform organisms about damage to their bodies. Call this the *information-gathering story* of pain (henceforth IGS).

IGS is a core commitment of various philosophical theories of pain, most notably perceptual and representational theories. For perceptualists, IGS constitutes the core parallel between pain and perception, supporting their view that pain is a perceptual phenomenon. For representationalists, IGS provides the teleological foundation of their view that pain states represent bodily damage. (See section 2 for a more detailed account of how perceptualists and representationalists rely on IGS). As such, the success of perceptualism and representationalism about pain relies heavily on the assumption that pain has the biological function to inform.

I argue that this is a mistake. First, there is no biological evidence to support the notion that pain was originally selected for its informative capacities, nor that it currently contributes to the fitness of organisms in this specific capacity. Hence, there is no evidence to suggest that pain's adaptive value lies primarily in its informative nature. Second, neurological evidence indicates that pain is systematically prevented from serving a primarily informative role by modulating mechanisms in the nociceptive system. Such mechanisms purposefully inhibit the transmission of system-relevant information and thereby prevent pain experiences from informing organisms about damage to their bodies. Hence, IGS is at best empirically unwarranted and at worst outright false. I conclude that these considerations threaten to undermine standard perceptual and representational theories of pain, and end by suggesting some alternatives.

The idea that IGS is not an adequate characterisation of pain has previously been motivated by two imperativists about pain, Colin Klein and Manolo Martínez. On their view, pain is analogous to homeostatic sensations, such as hunger or thirst, and is best interpreted as an action-guiding, imperative signal that serves the biological function of bringing about appropriate protective behaviours [Martínez 2011; Klein 2015; Martínez and Klein 2016]. As such, pain is to be understood as a *command* rather than as an *informant*. The present paper acknowledges the important contributions of Klein and Martínez, but aims to provide novel and (even more) compelling reasons for rejecting IGS.

This paper is divided into five sections. Section 2 introduces perceptual and representational theories of pain and traces their commitment to IGS; section 3 argues against IGS by drawing attention to the nature of pain modulation and sensory inhibition; section 4 argues against IGS by drawing attention to the lack of biological evidence to support it.

2. Key Assumptions

Various philosophical accounts of pain assume that it is pain's biological function to inform organisms about damage to their bodies. Such accounts are typically perceptual or representational accounts of pain.

2.1. Perceptualism and Representationalism

According to perceptualists, the feeling of pain is the perception of something [Armstrong 1968; McKenzie 1968; Pitcher 1970; Fleming 1976; Hill 2005, 2009; Ivanov 2011]. On their view, feeling a pain in one's toe is to perceive a physical feature or condition, such as tissue damage, in one's toe. As such, pain is considered akin to paradigmatic forms of perception, such as vision, hearing, smell, taste, or touch, in the sense that it relates a subject to its (bodily) environment.

Importantly, proponents of perceptualism commonly endorse the widely-accepted thesis that perceptual modalities *essentially* serve the biological function of gathering information (see Fodor [1983]; Burge [2005, 2010]; Block [2014]):

> It is clear that the biological function of perception is to give the organism information about the current state of its own body and its physical environment, information that will assist the organism in the conduct of life. [...] This is a most important clue to the nature of perception. [Armstrong 1968: 209]

This endorsement, in addition to perceptualists' core claim, viz. pain being a perceptual phenomenon, yields their commitment to IGS. Perceptualists rely on the assumption that it is pain's biological function to inform since, by their lights, pain is a perceptual phenomenon, and perceptual phenomena *essentially* serve an informative function.

According to representationalists, the feeling of pain is a contentful state representing bodily damage in parts of one's body [Tye 1995, 1997, 2005; Dretske 1997, 1999; Byrne 2001; Bain 2003, 2007, 2013, 2017; Cutter and Tye 2011; Cutter 2017]. On their view, feeling a pain in one's toe is to represent tissue damage in one's toe.[1] While representationalism is logically independent from perceptualism, representationalists about pain typically endorse perceptualism. Hence, representationalists agree with perceptualists that pain is a perceptual phenomenon, but insist that perception is to be understood in representational terms. On this view, the feeling of pain in one's toe is the perception of bodily damage in one's toe, where the perception of bodily damage is a contentful, representational state.

Representationalists with a commitment to perceptualism are commonly wedded to IGS in the same way perceptualists are. That is, they consider pain perceptual, and perceptual

[1] Imperatival accounts of pain are exceptions to this generalisation. Unlike other representational theories, imperativism considers pain experiences to have imperatival (rather than indicative) content, and is therefore assumed not to rely on an information-gathering function of pain. See Martínez [2011], Klein [2015], and Barlassina and Hayward [2019].

phenomena as *essentially* serving an informative function. However, various forms of representationalism are committed to IGS in a further sense, independently of their stance on perceptualism. Such commitment arises due to representationalists' engagement with theories of content determination, dedicated to explain how a mental representation gets to be *about* an extramental feature of one's environment. In this particular case, the explanation to be given is how the content of a pain experience gets to be about bodily damage.

One popular approach to this question is by means of teleological considerations. According to teleological theories of content determination, a mental representation gets to represent a particular feature in virtue of a representation-producing mechanism which has the selected *function* of informing about that feature [Millikan 1984; Papineau 1987; Dretske 1988, 1997; Fodor 1990]. On this view, the contents of pain experiences get to be about bodily damage in virtue of a representation-producing mechanism, the 'pain system', having the function of informing about bodily damage (esp. Dretske [1997]). This view of content determination is explicitly committed to IGS. Pain gets to represent bodily damage (or any other environmental feature) *only* if it has the selected function of informingabout bodily damage.

An alternative approach to content determination consists in invoking causal covariation or 'tracking'. According to tracking theories of content determination, 'S represents that P = df If optimal conditions obtain, S is tokened in x if and only if P and because P' [Tye 1995: 101; Cutter and Tye 2011]. On this view, what matters for representation is causal covariation with what is represented. While this account of content determination does not explicitly rely on IGS, it is not easy to see how it could work for pain were IGS not true. For suppose it was not pain's function to inform about bodily damage. In that case, it would be surprising to find that pain still causally covaries with bodily damage in the relevant sense, even though pain is not in the business of tracking it. Hence, even though tracking theories may not be formally committed to IGS, they owe an alternative explanation of what grounds the causal covariation, if not IGS.

2.2. Biological Functions and Information

The above considerations illustrate the importance of IGS to the success of prominent accounts of pain. Both perceptual and representational theories rely heavily on the assumption that it is pain's biological function to inform organisms about damage to their bodies. Now, there is a question of what this assumption amounts to. What does it mean to claim that pain has the *biological function* to *inform*?

There are roughly two ways in which one can interpret the notion of a biological function. One is as an *aetiological* (or backward-looking) notion, the other is as a *consequentialist* (or forward-looking) notion [Garson 2011, 2016, 2019].[2] On the aetiological view, statements such as 'it is the function of the heart to pump blood' are claims about the origin of the heart. They explain why the heart exists by going back in time to specify in virtue of what capacity hearts were originally selected. In the case of IGS, an aetiological interpretation amounts to:

> **AET:** Pain was selected for (in the past) because of its capacity to provide organisms with information about bodily damage.

On the consequentialist view, on the other hand, statements such as 'it is the function of the heart to pump blood' are claims about the heart's current contribution to an organism. Such claims leave history aside and look at how a trait is furthering the interests of organisms at the time. For the consequentialist, it is the heart's function to pump blood not because of issues to do with natural selection, but because the heart is currently contributing to the survival of organisms by pumping blood through their bodies and supplying oxygen to their organs. In the case of IGS, a consequentialist interpretation amounts to:

[2] Some have argued that there are more ways in which function statements can be understood (e.g. Wouters [2003]). However, the two-fold distinction I am relying on here is fully sufficient for the issue at hand.

CON: Pain currently contributes to the interest of organisms primarily by informing them about bodily damage.[3]

The reason CON specifies that pain *primarily* contributes by informing is because it needs to preserve considerations of salience in order to be of use to perceptualists and representationalists. Recall that such theorists think that is a 'most important clue' to the nature of perception that it serves an informative function, and that this clue is also applicable to pain. So if it is meant to be a deep fact about pain, as it is about perception, that it serves an informative function, then the claim cannot simply be that pain somehow tangentially serves such a function. That is to say, it cannot be that pain's informative capacities are a side-event while it's primary contribution lies elsewhere. Rather, the claim has to be that pain's *primary* contribution lies in its capacity as an informant about bodily damage.

AET and CON are not mutually exclusive. One can, for instance, endorse both claims and think that pain was originally selected for its capacity as an informant *and* that it continues to contribute to the interest of organisms in this capacity. In other words, one can think of pain as an *adaptation*, a trait that was selected for its current role [Gould and Vrba 1982]. However, one can also endorse either AET or CON and deny the other. One could think, for instance, that pain was originally selected for its informative capacity, but that it now contributes to the interest of organisms in a different way. In other words, one could think of pain as an *exaptation*, a trait that was co-opted for a role that it was not originally selected for [ibid.].

So what do proponents of IGS think? Do they endorse AET, do they endorse CON, or do they endorse a combination of these claims? Unfortunately, proponents of IGS do not provide the details necessary to give a definite answer to these questions. Moreover, I suspect that we are unlikely to discover a consensus among them. Teleological theorists of mental content are often read as favouring an aetiological interpretation of functions [Neander 2018], but it is far

[3] I speak of the 'interest of organisms' in a very broad sense, allowing for more specific notions, such as e.g. survival or fitness.

from clear that this applies to proponents of IGS more generally. In fact, the general lack of engagement with historical considerations that would support AET makes it plausible to think that proponents of IGS think of functions in a looser sense, more compatible with CON. However, since all of this is rather speculative, I will be thorough by examining the plausibility of both AET and CON independently, as well as in combination. The most efficient way to do so, I believe, is by first considering CON. I will argue that both CON and AET fail, which rules out any of the possible combinatory claims as well.

Before moving on, a quick word on information. Information has proven to be a very difficult notion to make precise, and the term is used in a variety of ways within and across disciplines. Armstrong is explicit that he understands information in terms of belief. For him, the function to inform is the function to acquire true or false beliefs about the world [1968: 209]. However, this notion of information may be regarded as overly restrictive in the present context, seeing that it discourages the attribution of information-gathering functions to traits of creatures that do not entertain belief-states. Insects, for instance, may not entertain beliefs, but their visual systems may well have the function to inform.

Alternatively, we may turn to Dretske for a more permissive, 'commonsense' notion, which defines information as characterised by three essential properties: (i) intentionality, (ii) factivness, and (iii) transmissibility [2008: 274]. On this picture, 'the function to inform' may be interpreted as 'the function to acquire (iii) *transmissible*, (ii) *factive* data (i) *about* features of the world' (whether such data ends up being entertained in belief-states or not). This definition isn't unproblematic, but it's generality is promising for operational purposes. In the case of pain, then, we may interpret 'the function to inform about bodily damage' as 'the function to acquire transmissible, factive data about the organism's physical integrity'.

3. Contra CON

According to CON, IGS is to be understood as the claim that pain currently contributes to the interest of organisms (primarily) by informing them about bodily damage. I will argue that this claim is false. To this end, I will examine the nature of modulating mechanisms in the nociceptive system which, as I argue, prevent pain from having a primarily informative function.

3.1. Pain Modulation

According to the International Association for the Study of Pain, pain is an 'unpleasant sensory and emotional experience associated with actual or potential tissue damage'.[4] The system in charge of such pain experiences is the so-called nociceptive system. Nociception involves the response of specialised receptors, so-called nociceptors, to noxious stimuli, such as heat, pressure, or chemical signals, to areas of the body. The energy of such signals is converted into electrical nerve impulses which are then transmitted to the spinal cord, and finally to the brain, where they pass through the reticular formation, following a series of pathways to the limbic system and cortex. This processing of nervous signals typically leads to a subject's experience of pain.

The above description lends itself to a simplified view of pain as a reliable tracker of injury. However, the link between pain and injury is less cemented than might be expected. Henry Beecher [1959] famously reported the behaviour of severely wounded soldiers admitted to the U.S. field hospital on the Anzio beachhead during the Second World War. He observed that upon arrival to the hospital most wounded soldiers either denied having pain at all, or reported having so little that they didn't feel they needed any painkillers. In fact, only one out of three incoming soldiers asked for morphine to relieve their pain. Similar cases have been reported by Carlen and colleagues [1978], who studied Israeli soldiers with traumatic amputations after the Yom Kippur War. In line with Beecher's observations, most of these soldiers reported their initial injuries as painless, and the eventual onset of pain as delayed for up to nine hours. As is insisted by both

[4] https://www.iasp-pain.org/terminology?navItemNumber=576#Pain

Beecher and Carlen, the soldiers in question were not in shock, but manifested awareness of their situation and its consequences. Some men expressed sadness or anger at incurring their injuries, others guilt towards letting down their comrades, and others still would act surprised about the fact that they didn't feel pain.

If you think cases of wounded soldiers are extreme outliers, note that 'pain free injury is not the monopoly of the battlefield' [Wall 1979: 259]. Melzack and colleagues [1982] conducted a study in which they interviewed patients about their pain experiences upon arrival at the emergency clinic of the Montreal General Hospital. The injuries of considered patients ranged from small cuts and bruises to fractured bones and amputated fingers, and were incurred in various settings, such as at home or the workplace. Melzack and colleagues found that 51 (37%) out of 182 patients stated that they did not feel pain at the time of injury. As in the cases of soldiers, such effects regularly occur despite the patient's full awareness of the injury. Feeling no pain, a machine shop foreman reacted to the accidental amputation of his foot with: 'Well, there goes my holiday' [Melzack and Wall 2008: 8].

The above examples of *episodic analgesia*, or temporary insensitivity to pain, illustrate just how commonly noxious stimuli fail to elicit pain sensations. The reverse is at least as common. Many pains, including headaches and back pain, can cause tremendous suffering without any apparent injury [ibid.: 9]. In fact, in a majority of cases of lower back pain no responsible damage can be identified despite extensive examination [van Tulder and Koes 2013]. Other examples of pain without injury include the phenomenon of so-called 'pain after healing', where former injuries continue to hurt despite full physical recovery of the wound [Livingston 1943].

Pain without injury and injury without pain illustrate the variable link between damage to the body and the feeling of pain. What underlies this dissociation are various modulating mechanisms which are able to exercise inhibitory as well as excitatory influences on the transmission of nerve impulses signalling bodily damage. Some of these responsible mechanisms were identified in the first half of the 20th century, but their full significance has only come to

light with Melzack and Wall's [1965] formulation of the Gate Control Theory of pain, which put modulation at centre stage. According to this theory, sensory inputs are modulated by a 'gate control system' before evoking pain sensation and response [ibid.]. Subsequent pain science has confirmed the essential role of modulating mechanisms, and recognises that the relationship between stimulus and pain experience is defined by a 'dynamic balance between inhibition and facilitation' [Heinricher and Fields 2013: 129].

Pain modulation appears at various pre and postsynaptic stages of nociceptive processing, most notably at the level of spinal cord and brainstem. Particularly extensive involvement has been attributed to the PAG-RVM system, which is primarily constituted of the periaqueductal grey (PAG) and rostral ventromedial medulla (RVM). The PAG-RVM system exerts bidirectional control over nociceptive transmission, integrating both bottom-up influences through the dorsal horn and top-down influences from higher cerebral structures, such as the hypothalamus and the limbic forebrain [Heinricher and Fields 2013: 130]. Research has shown that this modulating system is implicated in both inhibitory as well as excitatory effects on pain [ibid.].

The modulation of nociceptive signals by mechanisms such as the PAG-RVM system provides a mechanistic explanation for the dissociation of pain and bodily damage. On the one hand, nociceptive signals caused by noxious stimuli may be prevented from reaching relevant brain regions, and therefore fail to elicit pain experiences in the organism. On the other hand, nociceptive signals may be enhanced, even in the absence of a noxious stimulus, and thereby cause intensified pain experiences without corresponding injury.

A common assumption among pain scientists is that the ability to suppress or enhance nociceptive signals, and thereby control pain experiences, is beneficial for the organism's survival [ibid.: 120]. The inhibition of a pain experience may, for instance, facilitate an organism's escape by avoiding distraction in the face of immediate danger, whereas the enhancement of a pain experience may promote recuperative behaviour and healing. Whether a nociceptive signal is transmitted, inhibited, or enhanced, will depend on the central nervous system's analysis of the

entire situation at the time [Melzack and Wall 2008: 182]. As such, the modulation of nociceptive signals is context-sensitive.

3.2. The Challenge

The nature of pain modulation, as outlined above, constitutes a serious challenge to CON. To avoid ambiguity, note first what this challenge is not: The challenge is not simply that pain informs about injury too infrequently or too unreliably for pain to really be an informant. If this was the challenge, then I think it would face a ready response:[5] For even if it was agreed that pain is unreliable at informing, this wouldn't yet rule out the possibility that pain has the function to inform. After all, a trait might be adaptive without being perfectly adapted. So an objection from lack of correlation does not get us very far.

However, lack of correlation should be regarded as merely symptomatic of a deeper problem. This deeper problem is the challenge of explaining why a system would purposefully prevent system-relevant information from transmission if it is the system's function to perform such very transmissions. If pain is meant to be an informant in the same way that paradigmatic perceptual modalities are, then why does the pain system frequently hold on to the very information it is meant to transmit? So the problem for advocates of CON isn't simply that pain and injury are weakly correlated, but that the pain system isn't primarily interested in informing, which naturally results in weak correlation between pain and injury. As such, lack of correlation is really only the tip of the iceberg.

Advocates of perceptual and representational accounts of pain have shown surprisingly little interest in pain modulation and the challenge it poses (a fact much lamented by Hardcastle [1997]). This fact can hardly be explained by reference to 'novelty' or 'marginality' of the data. Work on pain modulation has played a significant role in pain science since the second half of the 20th century, and virtually any textbook on pain will include a chapter dedicated to the subject.

[5] See Pitcher [1970] and Corns [2014] for someone who considers a challenge to perceptualism of this sort.

Moreover, at least three major books published on the philosophy of pain over the last 20 years include discussion of the relevant data [Hardcastle 1999; Grahek 2001; Klein 2015]. Instead, then, I suspect that lack of engagement results from the assumption that pain modulation is not particularly problematic for perceptualists or representationalists. Indeed, the few advocates of these views who have engaged with the subject have said as much [Pitcher 1970; Tye 1995]. I argue that this is a mistake. Proponents of IGS have failed to acknowledge the challenge pain modulation poses for them.

An illustrative case study is Michael Tye's defense of representationalism about pain. In developing his account, Tye [1995] considers the potential difficulty of accommodating cases of top-down modulation. Examples include joggers who appear to be feeling less pain when running in a picturesque environment as opposed to on track, Scottish terriers who appear to be insensitive to pain when in great excitement, and cases of people feeling more pain when in states of anxiety [Tye 1995: 114]. The challenge Tye envisages concerns pain's susceptibility to modulation effects of this kind. If pain is correctly thought of as a tracker of bodily damage, then how is the feeling of it susceptible to influences of cognitive states? Tye's response is that such influences are simply a form of quantitative information control. Pain serves as an informant of bodily damage while modulating mechanisms in the nociceptive system exercise control over how much information gets transmitted [ibid.: 115].

Tye's point can be strengthened. For what one might want to say is that modulation, as a form of information-control, *subserves* an information-gathering function. On this view, modulation is a way of regulating the amount of information passed on, and of filtering out relevant from irrelevant news, based on the needs of the organism as a whole. As such, what explains how the tracking function of a system could be compatible with modulating and (especially) inhibitory effects, is that such effects facilitate tracking.

In support of this point, consider that inhibition is widespread among the senses. Inhibitory effects include the fact that we generally don't taste our own saliva [Von Békésy 1967:

11], don't hear the blood flow of our inner ear [ibid.: 9], don't see the white blood cells on our retina [Scheerer 1924], and much else. Yet, despite the prevalence of such effects, we don't seem tempted to dismiss taste, hearing, vision, or any other sense for that matter, as not genuinely serving an information-gathering function – nor should we. Why? Because sensory modulation of this kind is plausibly interpreted as a form of information control, just as Tye suggests. In fact, it is plausible to think that 'without inhibition, there would be no way of handling the great quantity of information we receive' [von Békésy 1967: 215].

Unfortunately, this defense of CON will not do. For while the above interpretation of the functional significance of sensory inhibition may well be correct, it does not extend to the forms of inhibition we encountered in the nociceptive system. The crucial point of contrast between pain inhibition and ordinary sensory inhibition is the following: sensory inhibition acts as a *filter*, whereas pain inhibition acts as a *gate*. As a filter, sensory inhibition reduces and selects from the flow of information when the quantity of information available exceeds what is relevant for the organism. Consequently, only a select and system-relevant amount of information is passed on to higher centres and affects conscious awareness.

Illuminating examples here are so-called adaptation effects, such as the Troxler effect [Troxler 1804]: If one rigidly fixates one's gaze at a point in one's visual field, one can come to experience the surrounding stationary images as fading away. Fixating on a cross in the middle of a drawing, for instance, may lead one to see nothing of the drawing but the cross one is fixating on. The effect results from a suppression of 'saccading', a form rapid eye movement, which ordinarily prevents adaptation of rods, cones, and ganglion cells on one's retina to a constant stimulus. When one's gaze remains rigidly fixated, natural saccading is counteracted, leading to the desensitisation of neurons and thereby affecting one's visual experience.

Adaptation effects are typically regarded as support for the notion that our senses are interested in what is new. The information that matters to an information-gathering system is information it does not already have. Where the available information remains unchanged, the

transmission of information may gradually diminish, since old news is no news at all [Block 2014]. Importantly, however, our visual system tends to keep us informed despite inhibitory effects of this kind. Inhibition through adaptation reduces and selects information, but it does not prevent the registration of information relevant to the system.

The same is *not* true in the case of pain. 'Gating' in the nociceptive system does not (always) simply reduce or select information. Instead, it temporarily prevents the registration of news completely. Reconsider the cases of soldiers who suffer severe injuries in battle, and whose nociceptive transmission is gated. In these instances, information is not simply reduced or selected, but often blocked entirely. No nociceptive signal has yet reached the brain from the injured body part, and no pain has yet been felt. Indeed, soldiers frequently continue to fight on injured limbs because the absence of pain leaves them ignorant of their injury. Crucially, this is not a consequence of the irrelevance of the information. If anything, the information is extremely relevant, both in the sense that it is new, and in the sense that it is the kind of information which nociceptors are specialised to transmit. Moreover, note that in contrast to the Troxler effect, subjects do not first feel pain, and that the pain then gradually wears off because the injury is becoming old news. Rather, subjects do not feel pain and may know nothing of their injury. Unlike perceptual inhibition, then, pain inhibition can hardly be described as a filter selecting relevant from irrelevant inputs. Rather, it is a gate which shuts off higher centres from any flow of information, relevant or irrelevant. No other form of sensory inhibition I am aware of operates in this manner.

The difference between filtering and gating is crucial. That's because the filtering of sensory inhibition is plausibly interpreted as subserving the gathering of information, whereas the gating of pain inhibition is not. It makes sense to think that a system which aims at information-gathering should select from the information available to it, and choose not to process information it already has. However, it makes little sense to think that such a system should completely block the flow of information that is new and relevant to itself as in the case

of pain inhibition. If in doubt, consider what it would be like if gating mechanisms were present in, say, the visual system: sometimes we just wouldn't see.[6]

These considerations reveal that even a strengthened version of Tye's response does not meet the challenge of pain modulation. The notion that modulation is a form of information control, compatible with or even subserving IGS, does not explain why a pure information-gathering system would suppress new and system-relevant information. Unlike other sensory modalities affected by modulating effects, pain systematically fails to keep us informed. I take this fact to be a compelling argument against CON. Note that the point is not that pain never informs us, or that we cannot learn anything from our pain experiences. Rather, the point is that pain does not act in a way proponents of CON would need it to act. If pain is meant to contribute to the interest of organisms primarily by informing them, *in an analogous way* to paradigmatic perceptual modalities, then we shouldn't find that pain counteracts this contribution in a way unlike any perceptual system. Hence, if perceptual and representational theories of pain rely on CON, then such theories are misguided.

4. Contra AET

According to AET, IGS is to be understood as the claim that pain has been selected for its capacity to provide organisms with information about bodily damage. Crucially, AET may still be true, even if we think CON is false. That's because even if pain's *current* role isn't that of an informant, it may still be true that pain was *originally* selected for this role. On this view, pain is not an *adaptation*, a trait that was selected for its current role, but an *exaptation*, a trait that was co-opted for a role it was not originally selected for [Gould and Vrba 1982]. As I argue, however, AET is not a tenable claim.

[6] Of course one has ways of preventing the registration of information of one's senses by other means. One can close one's eyes to stop seeing, or put one's fingers in one's ears to stop hearing. However, such ways of controlling the flow of information are importantly different since they are external to the system aiming to inform.

An important take-away from biology is that aetiological claims about functions cannot be supported from the armchair alone. That is because in the study of evolution, plausibility of an adaptationist story should not count as evidence. Take any organism, carve it up into traits of your choosing, and try to come up with a plausible story for why this trait was selected for and what role it serves – no problem. Unfortunately, doing so is a presumptuous practice which takes for granted that any chosen trait is in fact (perfectly) adaptive, and which ignores non-adaptive influences on evolution, such as genetic drift, allometry, and physical constraints. As a consequence, you are most likely to have carved up traits unnaturally, assigned functions to traits which aren't adaptations at all, and misassigned functions to traits which serve roles not evident to you. Hence, even though it is tempting to use plausibility to assign functions to traits, it is nothing but a game (see Gould and Lewontin [1979]).

The point is that aetiological claims about functions can only gain acceptance if they are supported with hard-earned empirical evidence. In the case of AET, that is evidence which shows that the selective pressures of the time favoured organisms which had the capacity to feel pain *because* it informed them about damage to their bodies, and that, as a consequence, pain was retained as a trait and spread through and across populations. Or, granting that CON is false, evidence which shows that the selective pressures of the time favoured organisms which had the capacity to feel pain *because* it informed them about damage to their bodies, etc., *and* that selective pressures changed such that pain was co-opted to serve a different role. However, *there is no proponent of IGS to my knowledge who has provided evidence of this kind.* The worry here is less that AET or a version thereof couldn't be true. The worry is that AET is vacuous as long as it stands without evidence, and that proponents of IGS base their theories on an entirely unsupported assumption. No serious evolutionary biologist, I dare say, would blindly indulge in speculations of this kind.

There's more. It's not just that proponents of AET haven't provided any evidence to support their claim, but it's not even clear how they *could* go about gathering such evidence. One

issue is that one would have to consider the evolutionary pressures at the time pain originated – but when did pain originate? What is the phylogenetic tree one could draw which maps back to the first organisms to feel pain? How would we find out? The other issue is a non-trivial problem of defining pain as a trait we could trace. Is pain simply a phenomenological episode, a hurting experience? Is pain the nociceptive system and relevant brain regions including all relevant subsystems? Is it both? How would we decide what the evolutionary relevant unit is? If one wants to champion AET, one better have some good responses to these questions. Without any evidence to support it, and plausible suggestions as to how such evidence could be gathered, AET remains a just-so story as vacuous as any other.

But now, one can imagine a proponent of AET to respond in the following way: 'Look, you are not appreciating the fact that perceptualists and representationalists about pain never motivated their view on evolutionary grounds. In fact, our arguments have never rested on the premise that pain has the function to inform. Rather, we developed a metaphysical theory of experience which is so promising as a unifying theory of mind that we extended it to the phenomenon of pain, willing to rely on the assumption that pain has an information-gathering function. But as such, it isn't really a criticism of our view that we don't have any evidence to support AET, since AET isn't what motivates us to be perceptualists and representationalists. AET might turn out to be true or false, but as long as no one has any evidence to the contrary, we don't have to go home because of evolutionary scepticism'.

Fair enough. To those who require more persuading I will offer the following consideration: In section 3 I argued that, contra CON, modulating mechanisms in the nociceptive system systematically prevent pain from serving a primarily informative role. This, however, left open the possibility that pain *originated* as serving such a role. Now, one kind of biological evidence that would speak against this possibility would be that the kind of modulating mechanisms which are present in *current* nociceptive systems were also present in the *original* nociceptive systems. In other words, one way of showing that pain wasn't selected for it's

information-gathering capacities is by showing that pain never had such capacities (because of modulation). This, admittedly, is not an easy thing to show, but it is one I think I can motivate.

The existence of nociceptors and pain behaviours has been shown in a diverse multitude of animal taxa, including *Mammalia*, *Aves*, *Amphibia*, *Teleostei*, *Arthropoda*, and many others [Sneddon 2017; Hearn and Williams 2019]. Importantly, research has shown that the nociceptive systems in many taxa, *including all of the above*, have modulating mechanisms that can inhibit nociceptive processing [Sneddon 2004, 2017; Heinricher and Fields 2013]. In fact, there is no animal taxon with nociceptors for which it has been shown that it *doesn't* have modulating mechanisms (see Sneddon [2017]). Considering the diversity of phyla that instantiate the relevant mechanisms, it has been suggested that modulation of nociceptive responses was present at an early evolutionary stage [Kavaliers 1988: 927]. Walters and Williams [2019] suggest that some forms of modulation, such as endocannabinoid modulation, have been conserved in animals for half a billion years. This puts pressure on proponents of AET. If modulation of nociceptive signals systematically prevents an information-gathering function of pain, and such modulation is found throughout the animal kingdom, dating back towards the origin of the nociceptive system, then there is reason to think that pain was never primarily an informant.

Are these considerations conclusive? Of course not. However, they do shift the supposed burden of evidence from the critic of AET to its proponent. If the proponent of AET is convinced that they are entitled to evolutionary speculation as long as there is no evidence to the contrary, then the above evidence to the contrary should move them to stop speculating and gather evidence that supports their claims. Why should we believe that pain was selected as an informant if it doesn't act like one and, as far as we can tell, never has? In the absence of evidence, proponents of AET have taken on a serious epistemic debt.

5. Conclusion

This paper has argued that standard perceptual and representational theories of pain fail. If it is an essential feature of perceptual modalities that they serve the primary function of informing organisms about their (bodily) environment, then, on either an aetiological or consequentialist interpretation of biological functions, pain is not a perceptual modality. Consequently, perceptualism about pain cannot be true even by perceptualists' own lights. Of course, this leaves open the possibility that pain may still be counted as perceptual by some other standard. It may, for instance, turn out to be cognitively impenetrable [Pylyshyn 1999] or modular [Fodor 1983]. However, by the lights of those who think of perception as essentially conforming to IGS, pain cannot be perceptual.

Moreover, if we are thinking of pain as a representational phenomenon, and if pain gets to represent bodily damage only if it has the selected function to inform about bodily damage, then bodily damage cannot be what pain is representing. Hence, standard representational accounts of pain cannot be true, even if such accounts dissociate from perceptualism. Again, this doesn't mean that we can't be representationalists about pain. One might think, for instance, that what pain is representing isn't tissue damage but a form of protective command [Martínez 2011; Klein 2015]. Such imperatival views are usually based on the understanding that pain's function is not to inform but to encourage protective behaviours. However, while such views may very well be an improvement on standard representational theories of pain, they too need to worry about the adequacy of their assumptions concerning pain's biological function. In any case, standard accounts of perceptualism and representationalism about pain which rely on IGS cannot succeed in their current form.

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