**Pain in Psychology, Biology and Medicine: Some Implications for Pain Eliminativism**

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*(forthcoming in Studies in History and Philosophy of Biological and Biomedical Sciences)*

## Abstract

An analysis of arguments for pain eliminativism reveals two significant points of divergence between assumptions underlying biomedical research on pain and assumptions typically endorsed by eliminativist accounts. The first concerns the status of the term ‘pain,’ which is a description of a phenomenon, rather than an explanatory construct. The second concerns reductive explanation: pain is explained causally, in terms of mechanisms or factors that produce or determine it, rather than by identifying it with a physical structure, process or mechanism. These discrepancies undermine several arguments for pain eliminativism.

## 1. Introduction

According to eliminative materialists, folk psychology will eventually be replaced by, rather than be reduced to, neuroscience. Note, however, that eliminativism is not a unitary thesis that stands in sharp contrast to reductionism. What most contemporary eliminativists argue is that some, but not all, psychological terms are irreducible to neuroscientific terms and therefore will be eliminated. More specifically, psychological concepts, constructs and phenomena that do not refer to or can be identified with neurobiological mechanisms, structures or activities will be abandoned from science. This characterization accounts for three peculiarities of eliminativism: eliminativists disagree about what exactly is being eliminated; most eliminativists with respect to some aspects of psychology are also reductivists in respect to other aspects; and eliminativism is closely linked to an identity model of reductive explanation.

This paper assesses the validity of arguments for pain eliminativism from the perspective of contemporary pain research in medicine, biology and psychology. Although not always explicitly distinguished, three distinct claims are associated with pain eliminativism:

1. pain as measured by self-report tests should be eliminated[[1]](#footnote-1);
2. pain explanatory concepts postulated by folk psychology should be eliminated;
3. pain as a natural kind should be eliminated.

Given the dissatisfaction of eliminative materialists with folk psychology, it is safe to assume that their arguments are primarily meant to support (ii). However, since some eliminativists argue that phenomenological descriptions are incoherent or incompatible with neuroscientific descriptions and explanations, (i) is implied as well. Claim (iii) is entailed by eliminativist arguments stating that what we commonly call ‘pain’ is in fact a complex phenomenon that dissociates into distinct dimensions underpinned by distinct biological mechanisms.

The aim of the paper is to show that arguments in favour of claims (i) and (iii) rest on assumptions incompatible with the methodological principles governing biomedical research, as well as the primary model of explanation at work in the life sciences, neuroscience included.

The paper is organized as follows. In Section 2, I discuss arguments for elimination drawing on analogies with cases from the history of science. These target claim (ii). The main objective of this section is to draw a preliminary distinction between pain as an explanatory (theoretical) construct and pain as a phenomenon to be explained. In Sections 3, 4 and 5, I discuss arguments suggesting that the phenomenon of pain is ill-defined and needs to be recharacterized in biological terms. In these arguments, claim (i) is implied. In Section 3, I defend the validity of current characterizations of a pain, as described by reports of pain experiences. In Section 4, I evaluate arguments for elimination assuming an identity model of explanation. My overall response is to challenge the assumption that biomedical research assumes an identity model of explanation. Instead, I argue, scientific inquiry aims to explain the phenomenon of pain by elucidating its causal mechanisms. If mechanism and pain stand in a relationship of cause and effect, then it is not clear how pain could be identified with or replaced by its causal mechanisms. Section 5 covers arguments from conceptual inconsistencies of pain concepts and definitions. I argue that these inconsistencies can be resolved if the concepts and definitions in question are construed as a revisable characterization of a phenomenon against which hypotheses are tested and the clinical effectiveness of treatments is established. In Section 6, I provide a methodological rationale for resisting extreme versions of claim (iii) stating that pain mechanisms are unique to each individual. My objection hinges on the notion that empirical research requires a minimal degree of generality in order to describe phenomena and interpret the results of experimental interventions. Finally, in Section 7, I summarize the main claims defended in the paper.

## 2. The argument from the falsity of folk psychological explanations

One of the most influential arguments in favour of the elimination of pain concepts (claim ii) hinges on the claim that folk psychological explanations are false, unfruitful or likely to be replaced by neurobiological explanations (Bickle 1998; Churchland 1981; Churchland 1986; Dennett 1988; Ramsey et al. 1990). Thus construed, pain eliminativism is analogous to well-documented examples from the history of science where terms such as ‘phlogiston’ and ‘gravitational forces’ were eliminated once the explanations in which they figure were abandoned.

A good case for pain eliminativism can be made in relation to folk psychological explanations of nociceptive behaviours, such as withdrawing one’s hand from a hot stove. The phenomenon explained here is a withdrawal behaviour induced by a noxious stimulus. Given my strong intuition that what caused me to withdraw my hand from the stove is the fact that I felt pain, I may be inclined to accept a folk psychological explanation postulating a pain mental state causally mediating the transition from stimulus to response. Yet, despite what introspection tells me about the causes of my behaviour, the explanation is false. Although nociceptive reflexes are modulated by psychological variables such as emotion, anticipation and expectation, they remain involuntary and can occur in decerebrated animals. These findings suggest that pain experience plays no causal role in determining these behaviours and that a strictly mechanistic explanation in terms of a reflex arc is sufficient. It may therefore seem plausible to hypothesize that just as nociceptive reflexes are nothing else but the activation of certain neural circuits, other folk psychological explanations will also be replaced by biological explanations, ultimately leading to the elimination of the notion of ‘pain mental states causally responsible for pain-related behaviours and beliefs.’

Hypothetical extrapolations aside, the above example demonstrates two things. First, introspection is not an infallible method granting direct and irrefutable knowledge of the inner workings of our minds. Since many folk psychological explanations rely on introspection, this provides a strong rationale for subjecting these explanations to more rigorous scrutiny. Second, the experience of pain and its reporting can, at least in some cases, be dissociated from pain as a cause and explanation of behaviours and beliefs. The former is something that can be measured, and therefore studied, irrespective of its putative causal role in the generation of behaviours and beliefs. Thus, one can deny that a causal-explanatory concept of pain refers to a causal structure existing in the world, without necessarily denying that subjects experience pain. This is a position Daniel Dennett (1988), as well as Patricia and Paul Churchland (1981; 1986) seem to adopt in their assessments of folk psychology.

## 3. The argument from the inadequacy of phenomenological characterizations of pain

While eliminative materialists don’t deny that we experience subjective states, they often insist that ordinary talk about subjective experiences, such as phenomenological pain reports, will eventually be replaced by talk about neural representations and mechanisms. Eliminativists point out that the way in which the brain represents the external world and internal states of the body is radically different from, and potentially incommensurable with the way in which subjects verbally describe the world and body states (Churchland 1989; Churchland 1986). Thus, they conclude, neural representations will most likely be part of future science, while phenomenological reports are bound to remain a sort of impasse leading nowhere. Alternatively, some eliminativists argue that subjective experiences are ‘illusions’ or ‘errors,’ since they represent physical objects as having properties which they don’t have. Since qualia (affects) neither are, nor represent properties of physical objects, we shouldn’t commit ourselves to a sense-datum ontology extending the physical ontology of the natural sciences (Hardin 1988).

In the above lines of argumentation, eliminativism goes beyond the mere rejection of the explanatory value of folk pain concepts (claim ii) and further suggests that pain as measured by self-report tests will be eliminated in favour of something else (claim i), either because such measures are not scientifically useful or because they commit us to a problematic ontology which should be avoided. This raises some interesting questions about the possibility of eliminating observational terms and the ontological commitments associated with measurements.

 As illustrated in Section 2, the fate of a term hypothesized for the specific needs of an explanation is largely determined by the fate of the explanations in which it appears. In contrast, the legitimacy of an explanandum as a bona fide object of investigation doesn’t depend on the success of its explanations, but rather on experimental considerations such as the validity, reliability and accuracy of measurement techniques, the reproducibility of measurements and the adequacy of experimental design (Baetu 2019b). In principle, none of these are dependent on assumptions specific to the explanations being tested and are instead grounded in general methodological desiderata governing experimental practice across all sciences. Thus, the elimination of observational terms must be justified in light of entirely different criteria than the elimination of theoretical terms.

It is not clear that psychological characterizations of pain affects are in any way a problematic or ill-defined phenomenon that needs to be recharacterized in terms of underlying biological mechanisms or neural representations. In biomedical research, pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (IASP Task Force on Taxonomy 1994, updated online 2014). There are three elements to this definition (Eccleston 2010, 59-60). First, pain is a psychological phenomenon. Second, causal determinants and modulators of pain include actual tissue damage (a biological component) as well as learned associations and cognitive appraisals involved in the evaluation of potential damage (a psychological component). Third, pain is measured by reporting the experience. This third element points to an operationalized definition of pain widely used in clinical practice: “Pain is whatever the experiencing person says it is, existing whenever he says it does” (McCaffery 1968, 95).

Defining pain as what subjects report on the occasion of a pain assessment test may seem like an attempt to measure a phenomenon by using a fickle instrument. Yet, contrary to this expectation, documenting when and how subjects report pain led to the development of valid and reliable tests for assessing the presence, intensity and other features of the subjective experience of pain.[[2]](#footnote-2) Not only these tests are routinely used for diagnosis, they also provide a characterization and measure of a phenomenon of pain against which explanatory hypotheses are tested and the clinical effectiveness of treatments is established (Melzack and Katz 2013). More importantly, in as much as psychological measures of pain satisfy the requirements of validity and reliability, there are no methodologically legitimate rationales for the elimination of pain as measured and described in verbal and written reports.

It is also not clear that measurements of affect-phenomena–in this case, pain–commit us to a sense-datum ontology. In the absence of a robust physical interpretation, pain and its attributes are nothing else but measured variables. When a subject is instructed to rate the intensity of her pain on a scale of 0 to 10 and she answers ‘5,’ the variable ‘pain intensity’ takes the value ‘5.’ Nothing here demonstrates that ‘pain intensity’ and ‘5’ refer to psychic entities and properties existing alongside with neurons and action potentials. The worry that phenomenological measures and descriptions commit us to some form of dualism or psychophysicalism is premature.

## 4. The argument from psycho-neural identities

 The reduction of folk psychology to neuroscience is typically construed along the lines of an identity model of reductive explanation. That is, psychological terms will come to be identified with terms appearing in neurobiological explanations (Churchland and Churchland 2001). In the case of theoretical terms, this may mean that hypothetical constructs are shown to refer to something in physical reality or that two explanatory constructs have the same referent. An example of the former is the identification of classical genes with DNA sequences. The latter is illustrated by Benzer’s experimental proof that the unit of recombination coincides with the unit of mutation. In the case of phenomena and observational terms, identities may concern macro-properties related to the characterization of phenomena and microstructures postulated by explanations or theories about these phenomena. For instance, the statistical-mechanical formulation of the ideal gas law is often interpreted as the claim that temperature is the average molecular kinetic energy of the molecules composing a gas.[[3]](#footnote-3)

By itself, identity does not entail elimination. However, in as much as the identified terms have radically different properties, it is difficult to avoid the need for an additional explanation accounting for the emergence of ‘macro-properties,’ such as temperature, from ‘microstates,’ such as molecules bumping into one another. Some eliminativists solve the problem by claiming that scientific inquiry ultimately reveals that reality does not consist of psychological phenomena such as pains and the perceived solidity of a table, but rather of firing neurons (Rorty 1965, 38) and tiny atomic nuclei surrounded by huge electron clouds essentially devoid of matter (Eddington 1929, x).

Daniel Dennett (1981; 1996) adopts a more subtle strategy, arguing that talk of pain affects pertains to a scientifically naïve model of reality, which nevertheless remains instrumentally useful for predicting human and animal behaviour. We are told that “no events or processes could be discovered in the brain that would exhibit the characteristics of the putative ‘mental phenomena’ of pain, because talk of pains is essentially non-mechanical, and the events and processes of the brain are essentially mechanical” (1996, 91). At the ‘personal level’ of phenomenological experience, pain is unanalyzable into any kinds of components, thus blocking any further investigation of mechanisms. We may choose to switch to the ‘sub-personal level’ of neurophysiological mechanisms, but then we change the subject matter from pain experience to “the motions of human bodies or the organization of the nervous system […] abandoning the pains and not bringing them along to identify with some physical event” (1996, 94). Note that even though Dennett does not deny the existence and usefulness of phenomenological descriptions of pain, such descriptions are effectively evacuated from neuroscience.

According to a third line of argumentation, studies such as representational similarity analyses reveal homomorphisms between the structure of stimuli, the structure of neural representations and perceived similarities between conscious experiences. These homomorphisms are typically taken as evidence that conscious experiences represent objective features of physical reality. Paul Churchland goes a step further, and argues that conscious experiences not only represent, but are terms that refer to objective features of reality. For instance, he argues that

we might try to identify each external color with a specific electromagnetic reflectance profile had by any object that displays that color. The objective reality of colors would then emerge as being no more problematic than is the objective reality of the temperature of an object (which is identical to the mean kinetic energy of its molecules), or of the pitch of a sound (which is identical to the dominant oscillatory frequency of an atmospheric compression wave), or of the sourness of a spoonful of lemon juice (which is identical with the relative concentration of hydrogen ions in that liquid). These parallel properties also fail the ‘first-order resemblance’ test imposed by Locke […]. Nonetheless, their successful reduction to objective properties of material objects is an accomplished fact, both of science and of settled history. Locke’s criterion for objective reality–a first-order resemblance to the qualities of our sensations–was simply ill-conceived. (2007, 199)

Although Churchland defends reductionism, something is eliminated: the apparent subjectivity of affects. Churchland argues that, just like Locke, we commonly think of descriptions of affects as being subjective, and therefore distinct from objective physical reality, simply because we failed to realize that affects represent, and ultimately refer to objective states of physical reality.

It is also interesting to note that the homomorphisms to which Churchland alludes are statistical correlations subject to some degree of variation. Hence, there is always something about conscious experiences which doesn’t mirror features of physical stimuli. Churchland concedes this caveat but does not discuss its implications. For instance, Tor Wager and colleagues (2013) showed that the temperature of a stimulus, the brain activation produced, and the subjective ratings of pain given by the subjects are well correlated, to the point that researchers could predict subjective ratings given readings of brain activation patterns with a high degree of accuracy. However, the correlation is not perfect. In a subsequent study (Woo et al. 2017), the group showed that the residual variability (the pattern of activation that did not correlate with stimulus intensity) still correlated with the pain ratings. This “stimulus intensity independent pain signature” may be interpreted as “a measure of the conscious experience of the pain, independent of its discriminative nociceptive properties” (Adolphs and Anderson 2018, 276). In contrast, Churchland dismisses any residual variability as ‘noise’ (e.g., measurement uncertainty), tacitly eliminating it from the description of the phenomenon to be explained.[[4]](#footnote-4)

In all of the above accounts, elimination is tightly linked to an identity model of reductive explanation. Under this model, aspects of conscious experience that cannot be identified with biophysical structures, processes or mechanisms constitute by necessity an unexplainable ‘phenomenological surplus.’ The only way to restore the explanatory closure of science is to eliminate the surplus. Thus, some physicalists outright deny the existence of a surplus, some relegate it to a non-scientific worldview, while others treat it as noise.

It is interesting to note, however, that identity is neither the only, nor the most common model of reductive explanation in science. It is true, physics textbooks teach us that temperature is the mean kinetic energy of molecules. Yet physics textbooks are also in the habit of defining temperature as a measure of the average translational kinetic energy of the molecules of a gas. Under a causal-realist interpretation of measurement of the sort typically endorsed in experimental science (Trout 1998), this definition brings back temperature as something distinct from kinetic energy, namely the measurable manifestation of this form of energy in a particular experimental setup. Moreover, this is just one example of physical interpretation. Strictly speaking, there is nothing inherent to the mathematical formalism of physics favouring identity rather than some other interpretation. Mathematical equations expressing laws are just as often interpreted as causal relationships. Take Newtonian dynamics, for instance, which is often interpreted as a causal explanation of Kepler’s kinematics of planetary motion.

 The problem of the physical interpretation of mathematical relationships is particularly acute in the case of homomorphism. Evidence for homomorphisms between the similarity space of stimuli, patterns of neural activation and the similarity space of phenomenological descriptions does not prove that these items are identical with one another. In the absence of further evidence, each item is nothing else but a predictor, marker or correlate of the other two. In fact, stimulus-response experiments strongly suggest that such homomorphisms reflect causal dependencies between stimuli, neural activation and conscious experience.

More generally, discovery in the life sciences is largely driven by controlled experiments explicitly designed to demonstrate the causal relevance of variables to an outcome of interest (Bickle 1998; Kendler and Campbell 2009; Woodward 2008). Research on pain is not any different. If pain intensity is a variable that can be measured by documenting pain reports, it is possible to identify causal determinants impacting on this variable by comparing test and control conditions. For example, when exposed to noxious stimuli, C-fibers display an increase in action potential frequency correlating with reported pain. Both C-fiber activity and pain intensity decrease in the presence of a local anesthetic, such as lidocaine, which is known to block action potentials.

These results neither rule out, nor support the possibility that pain is identical with a neural structure. The experimental design is simply aimed at demonstrating something else, namely that C-fibers are causally relevant to measured pain as mediators in the transmission of information about noxious stimuli (Handwerker 1996; Puccetti 1977). Identity is an entirely different conclusion which is not tested by the experiment and cannot be logically inferred from evidence for causal relevance. Thus, as two pain researchers, Mathieu Roy and Tor Wager (2017, 87) put it, “although pain cannot be ontologically reduced to any one state of neuronal activity, it may be causally reduced to neuronal activity.”

In as much as the vast majority of experiments in pain research in humans are explicitly geared towards the discovery of causal determinants of reported pain, it should not be particularly surprising that proposed biological explanations of psychological phenomena have a lot to do with causal mechanisms and virtually nothing with non-causal supervenience. After all, mechanisms are hypothesized and reconstructed by compiling knowledge of causally relevant factors gained by means of experimental interventions (Bechtel 2006; Bechtel and Richardson 2010; Craver 2007; Craver and Darden 2013; Darden 2006). For instance, according to biopsychosocial models, pain “is determined by the interaction among biological, psychological (which include cognition, affect, behaviour), and social factors (which include the social and cultural contexts that influence a person’s perception of and response to physical signs and symptoms)” (Asmundson and Wright 2004, 42). These models aggregate various causal factors, arranging them whenever possible along putative causal pathways. Likewise, neuroscientists are interested in elucidating “the mechanisms that produce pain” and identifying “novel targets for the treatment of pain” (Cavanaugh and Basbaum 2011, 21). For example, the gate control neural circuitry explains why pain intensity subsides when we rub a smack, while the mechanism of action of ibuprofen explains its analgesic effects. Both explanations are unambiguously causal. The first mechanism posits that the simultaneous activation of nociceptors and thermomechanical sensors blocks the transmission of noxious signals to the brain (Melzack and Wall 1965); the second, that ibuprofen inhibits the production of prostaglandins, which are known to sensitize spinal neurons to pain (Collier 1971).

In as much as pain research, and the life sciences in general, explicitly and systematically adopt an experimental methodology and a causal model of explanation, it is less obvious that there is a ‘phenomenological surplus’ to be eliminated. The key requirement for a causal model of reductive explanation is that conscious experience has biological causes. Or there is ample experimental evidence demonstrating that biological variables and mechanisms are causally relevant to psychological outcomes and phenomena.[[5]](#footnote-5)

I can think of three responses to my rejection of eliminativism. One empirical argument for psycho-neural identities may exploit well-documented cases of psychological measurement techniques (self-report, cognitive tasks, behavioural descriptions) being replaced by biological ones (neurological and physiological correlates, molecular markers). Nevertheless, replacement of one technique by another does not demonstrate the identity of the variables targeted by these techniques. Replacements are invariably justified by validation procedures whereby biological tests are shown to consistently give the same results as psychological tests. Thus, biological tests are in fact extensions of earlier psychological tests motivated by the discovery of biological correlates and determinants of psychological outcomes. The use of cortisol as a biomarker of psychological stress (Smyth et al. 1997) illustrates this point.

In the same vein, an effect or symptom-based characterization of a disease is not replaced by a strictly causal-mechanistic or physiopathology-based one. Rather, the latter extends the former, by specifying the causes or mechanism responsible for a clustering of symptoms (Baetu 2019a, Ch. 2). For instance, Huntington’s disease is still defined and diagnosed in terms of its symptoms, most notably chorea. It is only that modern nosology further distinguishes this form of chorea from other symptomatically similar diseases based on a pattern of inheritance and mutations in the huntingtin gene. In other words, Huntingon’s is never recharacterized as ‘autosomal dominance’ or ‘huntingtin mutations,’ but rather as ‘autosomal dominant chorea’ or as ‘huntingtin mutation-induced chorea.’ The same goes for pain. We may characterize pain as an unpleasant sensation, then recharacterize it as an unpleasant sensation induced by tissue damage or as an unpleasant sensation underpinned by certain neural pathways or structures. ‘Unpleasant sensation’ is still there.

A second reply would be to appeal to the notion of mechanistic constitution. Craver and Bechtel (2007) argue that the phenomenon of light transduction in the eye consists of, as opposed to being caused by, a hierarchical structure of mechanisms involving parts behaving in certain ways, much in the same way the temperature of a gas consists of, and is not caused by, the mean kinetic energy of gas molecules. The immediate difficulty with this proposal is that it is not clear how constitution relationships can be inferred given that experiments in the life sciences can only provide evidence for causal relevance. Craver proposes the following solution:

a component is relevant to the behaviour of a mechanism as a whole when [… the] two are related as part to whole and they are mutually manipulable […]: (i) X is part of S; (ii) in the conditions relevant to the request for explanation there is some change to X’s φ-ing that changes S’s ψ-ing; and (iii) in the conditions relevant to the request for explanation there is some change to S’s ψ-ing that changes X’s φ-ing. (2007, 153)

For instance, the whole S could be a guinea pig organism; S’s behaviour ψ, an inflammatory response; part X, prostaglandins; and X’s behaviour φ, the binding of prostaglandin receptors, which triggers a signalling cascade leading to the expression of several gene products involved in inflammatory responses. The fact that prostaglandin injections result in inflammatory responses may be construed as a ‘bottom-up’ intervention on a part having an effect on the whole, while ultraviolet exposure could be a ‘top-down’ intervention on the guinea pig-whole affecting its prostaglandin-parts.

Since mutual manipulability relies on causal relevance tests (controlled experiments) it may seem natural to conclude that the relationship between mechanism and phenomenon is one of reciprocal causal dependency (Leuridan 2012). Nevertheless, Craver and Bechtel insist that the requirement for part-whole relationships has undesirable consequences for a causal interpretation, chief among which is the fact that cause and effect are no longer distinct events (Craver 2007, 153-54; Craver and Bechtel 2007, 552-54).

Be that as it may, it is difficult to make sense of the claim that experimental interventions target parts and wholes. If I push a cup of coffee over the edge of the desk, do I intervene on the cup, the things of which the cup is a part or the parts of the cup? It is by no means clear. What is clear is that measurements and interventions target variables. For instance, what varies between test and control conditions in the ‘top-down’ experiment sketched above are not the guinea pigs or erythema, but rather the intensity of ultraviolet radiation, which is the input condition of the phenomenon of ultraviolet-induced inflammation. Despite Craver’s suggestion that “[o]ne intervenes on S’s ψ-ing by intervening to provide the conditions under which S regularly ψs” (2007, 146), it is not clear why an intervention on a variable referring to an aspect (the intensity) of a stimulus (ultraviolet radiation) should be equated with an intervention on an organism-whole (guinea pig) or its behaviour (erythema).

Finally, it may be counterargued that only an identity model of reductive explanation can successfully address the mind-brain explanatory gap (Polger and Sufka 2005, 326-28). The concern here might be that causal difference-making of the sort demonstrated in biomedical research is contingent and therefore doesn’t really explain the nature of pain. In contrast, identity is a relation holding with a necessity of a much stronger variety. The only answer I can offer is that perhaps the universe is such that the depolarization of C-fibers is a causal difference-maker to pain reporting behaviours and not such that there are psychic pains identical with neural activities. My point here is that there is good evidence satisfying clearly defined and universally accepted experimental standards to justify scientific knowledge about causal structures in both biology and psychology. In contrast, proponents of an identity model of explanation struggle to specify the methodological desiderata to be satisfied in order to test identity hypotheses, explain in what sense an identity relationship is more necessary than a causal one and, last but certainly not least, they don’t tell us what we are to do with the causal knowledge accumulated so far, the practices and technologies it supports and the experimental methodology that made it all possible.

## 5. The argument from the inconsistency of pain concepts and definitions

One last family of arguments aiming to undermine the existence of a pain phenomenon (claim i) target inconsistencies associated with pain concepts and definitions. Daniel Dennett (1978; 1988) and Valerie Hardcastle (1999, Ch. 7) argue that the folk concept of pain encompasses a set of common intuitions about pain, including the beliefs that one cannot be mistaken about being in pain and that being in pain is sufficient for having an awful experience. Thus construed, the folk pain concept fails to capture reported instances of dissociation of pain experience into a sensory-discriminative and an affective-motivational component. Dennett argues that this failure entails that a subject may simultaneously hold incompatible mental states, such as experiencing a non-awful pain under the influence of an analgesic while remaining convinced that being in pain is sufficient for having an awful experience. Alternatively, Hardcastle concludes that the folk concept fails to accommodate the complexity of scientific findings. Given these shortcomings, the term should be replaced by more adequate alternatives.

In response, some authors deny that folk pain concepts comprise the kind of beliefs eliminativists attribute them (Conee 1984; Corns 2016). Still, it would seem that the IASP definition of pain at work in biomedical research suffers from an internal inconsistency. As outlined in Section 3, the definition tells us that pain is an unpleasant experience while at the same time endorsing self-reporting as a valid, reliable and accurate method for measuring pain. Yet in extreme cases of pain asymbolia, subjects are reporting both being in pain while attributing pain intensity a value consistent with the absence of pain.[[6]](#footnote-6) Doesn’t this undermine the validity of pain assessment methods relying on self-reports? Or, if the tests are valid, shouldn’t the IASP definition abandon the ‘unpleasant experience’ component in favour of the strictly operational definition typically adopted in clinical contexts?

There is a relatively simple solution to this apparent contradiction. The so-called ‘IASP definition of pain’ is not a bona fide intensional definition, but rather the description of a phenomenon.[[7]](#footnote-7) The description of a phenomenon is sometimes used as a substitute for a definition, for instance in order to make a diagnosis or classify an item. Nevertheless, its main epistemic role in both clinical and basic research is to provide a characterization of an explanandum against which hypotheses are tested and the clinical effectiveness of treatments is established. This is consistent with the fact that the scientific articles to which Dennett and Hardcastle allude do not propose a recharacterization of pain. Rather, they describe natural or controlled experiments documenting the effects of interventions (prefrontal lobotomy, cingulotomy, opioid administration and hypnotic suggestion) on pain. These results provided some of the evidence supporting the view that pain has three dimensions–sensory-discriminative, motivational-affective, and cognitive-evaluative–each subserved by distinct neural mechanisms (Melzack and Casey 1968).

## 6. The argument for the elimination of pain as a natural kind

The argument from the inconsistency of pain concepts, especially under Hardcastle’s formulation, further suggests a different sort of elimination, namely of pain as a natural kind. Edouard Machery (2009, Ch. 8) draws a distinction between traditional eliminativism, advocating the elimination of certain terms, and scientific eliminativism, which disputes that a class of objects constitutes a natural kind, as understood under Richard Boyd’s (1991) homeostatic property cluster account. The latter targets the objectivity and scientific usefulness of classificatory terms. Similarity-based classifications, such as those proposed in nosology and taxonomy, are objective in the sense that they rely on consistently documented correlations between measurable symptoms or morphological traits. Since these correlations are shown to be statistically significant, it is unlikely that they are chance occurrences. Nevertheless, this minimal notion of objectivity can be contrasted with a more robust one on the grounds that members of classes that display a similar clustering of properties in virtue of similar mechanisms are more likely to behave in similar ways in response to experimental interventions and natural sources of interference (Boyd 1991; Schaffner 2001; Steel 2007). This is less likely to be the case for members of classes that don’t share common mechanisms. For instance, patients with highly similar symptoms usually respond differently to identical treatments when the underlying physiopathology is different.

Scientific eliminativism states that only classes whose members share properties in virtue of common causal mechanisms have objective reality (Machery 2009, 241). Applied to pain, the question is whether pain, as measured by a specified technique or set of techniques, constitutes a genuine natural kind or merely a convenient way of grouping superficially similar phenomena. The key issue here is the generality of mechanistic explanations of pain. Is pain, for instance as reported by subjects, generated by the same or similar mechanisms? If the answer is ‘No,’ then pain is not a natural kind. In this case, elimination is analogous to causal-mechanistic reclassifications of diseases, such as the splitting of diabetes, initially defined as a cluster of symptoms, into several types reflecting differences in the mechanisms of disease. Likewise, pain would be replaced by several pain types, each corresponding to a natural kind underpinned by a common mechanism. For the sake of convenience, we may continue to talk about pain in general in the same way we colloquially talk about seafood, but scientific research and its clinical applications are concerned solely with the natural kinds encompassed under the generic term.

 Is pain, then, a natural kind? It is highly unlikely that pain is underpinned by a single mechanism. Even though peripheral nervous structures dedicated to the processing of nociceptive inputs have been identified in humans and other animals, phantom limb and chronic pains, as well as the discovery of psychosocial determinants of pain indicate that extensive brain processing is also involved. Furthermore, the high degree of variability in the neural correlates of pain supports the conclusion that there are no discrete regions dedicated to pain processing (although this does not preclude that existence of specialized neural circuits). These considerations led Ronald Melzack (2001, 1378) to hypothesize that pain “is produced by the output of a widely distributed neural network in the brain,” dubbed the ‘neuromatrix,’ which “is the primary mechanism that generates the neural pattern that produces pain. Its output pattern is determined by multiple influences, of which the somatic sensory input is only a part, that converge on the neuromatrix.” The proposal here is that a variety of inputs activate and modulate a general-purpose neuromatrix mechanism capable of representing several body states, including pain. In turn, this central mechanism generates a complex sequence of pain-related behaviours, including pain attribution and reporting. The inputs of the neuromatrix are presumably processed by distinct distal mechanisms, some of which involve the better understood peripheral pathways involved in nociception and the central mechanisms modulating it (Apkarian et al. 2005; Melzack and Wall 1965), as well as the cellular and molecular mechanisms responsible for inflammatory processes (Basbaum et al. 2009). The proximal neurological signature of pain is yet to be discovered, but preliminary research is encouraging (Wager et al. 2013).

If correct, the neuromatrix model may support both the view that pain is a natural kind, since all instances of reported pain share a common proximal mechanism, as well as the splitting of pain into several types reflecting differences in the distal mechanisms that may trigger the neuromatrix signature of pain. This may seem contradictory, yet both pain and types of pain qualify as natural kinds as defined by Boyd and Machery. In fact, such situations are extremely common in medicine. Diabetes can be said to be a natural kind in the sense that its symptoms are underpinned by a common proximal mechanism involving a dysregulation of blood glucose levels; for this reason, a regimented diet is part of the treatment for all forms of diabetes. At the same time, distinct types of diabetes can be differentiated in terms of distal mechanisms, which accounts for differences in responses to treatments such as insulin injections.

In contrast to the above proposal, Jennifer Corns(2012, Ch. 4)argues that the neuromatrix model and the current state of pain research support the conclusion that different instances of pain are underpinned by different combinations of mechanisms determined by the unique circumstances and experiences of the individual. Given that there might be as many pain mechanisms as there are individuals in pain, Corns (2012, Ch. 5; 2016) endorses scientific eliminativism in respect to both pain and types of pain.

 I think it is extremely unlikely that a radical position according to which distinct mechanisms are at work in each individual can ever receive conclusive experimental support. Unique mechanisms amount to instances of singular causation. While metaphysically possible, singular causation is incompatible with the methodological assumptions underlying empirical research as we know it. For example, it is impossible to say whether the occurrence of a singular event is the effect of an experimental intervention or mere coincidence. Evidence for causation requires evidence for correlation, that is, of a constant, rather singular, conjunction of two events. Moreover, the kind of extreme individual variability assumed by Corns entails that it is impossible to assess the causal impact of an intervention, say, on a neural pathway, by comparing test and control subjects since no two subjects are comparable in respect to the neural pathways under investigation. If Corns is right, then studies comparing populations of test and control subjects are either bound to remain irreproducible, or, if reproduced, amount to spectacular coincidences leading to mistaken conclusions about the existence of inexistent neural pathways conserved across subjects.

The very nature of empirical research presupposes some degree of generality of both phenomena and the mechanisms producing them (Baetu 2013). This provides reasonable grounds to believe that either future research will succeed in identifying one or more natural pain kinds, or evidence for or against pain kinds will remain inconclusive. Recent explanatory breakthroughs in pain research suggest the existence of a set of common mechanisms of pain widely shared in the general population (Roy and Wager 2017). I therefore strongly incline towards the view that different dimensions of pain experience, as well as distinct types of pathological pain are underpinned by mechanisms conserved across all members of the species, or even across species, and in this sense qualify as natural kinds.

## 7. Concluding remarks

I have argued that pain eliminativism is incompatible with what biomedical research has to say about pain. This is in good part due to the fact that, in science and medicine, pain is not a folk psychological explanans competing with biological alternatives, but rather a well-defined phenomenon. The only way to get rid of a phenomenon is either to show that it is an irreproducible empirical finding attributable to chance, or radically rethink the experimental methodology underpinning scientific research. The first option is denied by experimental findings. The second is something which no eliminativist account is prepared to offer (Chomsky 2000, Ch. 4).

Perhaps the most troubling and far-reaching challenge flagged in the paper is the absence of an experimental design capable of supporting inferences about identity or some other form of non-causal determination. The immediate consequence explored in Section 4 is that pain research assumes a causal model of reductive explanation, which, contrary to an identity model, does not leave much room for elimination. However, the absence of experimental evidence for mind-brain identities in biomedical research and the failure to bridge the gap between evidence for causal relevance and identity claims have implications that go beyond eliminativism. These shortcomings further entail that any physicalist model postulating type or token identity (Lewis 1994), supervenience (Kim 2005) and mechanistic-constitutive (Craver and Bechtel 2007) relationships between psychological and biological states is poorly supported by experimental results. I think these discrepancies ultimately entail that either adjustments are made in order to align eliminativist and physicalist accounts with the causal picture emerging from scientific research or a link bridging the gap between evidence for causation and non-causal models of the mind-brain relationships must be provided.

## Acknowledgments

This work was supported by a start-up research budget from the Université du Québec à Trois-Rivières (Québec, Canada).

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1. Verbal/written pain reports constitute the best public evidence for the ‘feeling,’ ‘affect,’ ‘experience’ or ‘qualia’ of pain. Crucially important, pain reports are a type of observable behaviours and therefore something that can be measured and studied from a 3rd-person perspective. [↑](#footnote-ref-1)
2. Pain tests should be valid, reliable and clinically useful. Validity encompasses evidence that the test is measuring what is supposed to measure (construct validity)–e.g., agreement with psychophysical measurements, correlation with the administration of analgesics, as compared with spontaneous variation in pain intensity (Hewer et al. 1949); and that it addresses all the aspects of pain experience (content validity)–e.g., statistically significant clustering of pain descriptors, correlations between intensity ratings and pain descriptors irrespective of the cultural, socio-economic, and educational background of the subjects (Melzack 1975). Reliability refers to statistical measures of the reproducibility of pain intensity ratings, clustering of pain descriptors and correlations between intensity ratings and pain descriptors, administration of analgesics, symptoms and medical conditions. Clinical usefulness encompasses sensitivity and specificity, namely the ability to detect all and only cases of pain. The validity and sensitivity (rate of detection of true positives) of commonly used pain assessment tools is well demonstrated across multiple testing methods; reliability and specificity (ability to discriminate false positives) are sometimes disputed (Noble et al. 2005). [↑](#footnote-ref-2)
3. Such macro-phenomena are often construed as aggregative behaviours. For instance, the pressure of a gas can be intuitively construed as the total effect of myriad molecules colliding with the walls of the gas container, while an electroencephalogram reflects the overall electric activity of billions of neurons. [↑](#footnote-ref-3)
4. I am grateful to an anonymous reviewer for suggesting this line of argumentation. [↑](#footnote-ref-4)
5. More stringent accounts require ‘complete’ mechanistic explanations (Baetu 2015; Craver and Darden 2013), which current explanations of pain don’t satisfy. However, since pain research relies on the same methodology and discovery strategies adopted in other, very successful, fields of investigation, there are no preliminary reasons to believe that causal explanations of pain are bound to remain incomplete. [↑](#footnote-ref-5)
6. For a review of the scientific literature on pain asymbolia, see (Aydede 2013, Section 6.1). [↑](#footnote-ref-6)
7. A definition is expected to specify necessary and sufficient conditions for the applicability of a term. Any given item either satisfies or not these conditions, falling ‘as a matter of definition’ within or outside a certain category. In contrast, a phenomenon is a web of correlated variables susceptible to recharacterization in light of new empirical findings. For instance, at the heart of the phenomenon of acute pain there is a consistently reproducible causal difference-making relationship linking a certain category of stimuli to the joint occurrence of certain avoidance and reporting behaviours. The list of noxious stimuli is augmented as new forms of stimulation resulting in similar responses and reports are discovered. Conversely, new behavioural, physiological, cellular and molecular responses are appended to the description of the phenomenon and new forms of assessment are developed based on correlations with known pain behaviours, dependency on noxious stimulation, and agreement with previously validated tests. Satellite phenomena are also eventually characterized, such as chronic pain, which displays the avoidance and reporting behaviours typically associated with acute pain, but in the absence of identifiable noxious stimulation. [↑](#footnote-ref-7)