Pain Eliminativism
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Abstract

The philosophical thesis of pain eliminativism can be understood in several ways. As a claim about the inadequacy and replacement of folk explanatory pain constructs and concepts, it is congruent with scientific findings. Eliminativism is controversial in as much as it demands a more or less radical recharacterization of the phenomenon of pain in order to ensure compatibility with physicalism and an identity model of reductive explanation. In this respect, eliminativism is at odds with experimental and explanatory paradigms at work in biomedical research. The latter is concerned with reproducible phenomena, controlled experiments generating evidence for causation and causal-mechanistic explanations. Since nothing here supports conclusive inferences about the identity or non-identity of pain, or aspects of it, with biological activity, it is not clear what, if anything at all needs to be eliminated.

In philosophy of mind, eliminative materialism is the thesis according to which folk psychological explanations of the sort ‘I removed my hand from the stove because I felt pain’ should be replaced by explanations in terms of neural mechanisms. A replacement is justified when an explanation is not merely incomplete, approximately correct, or a special case—in which case it could be reduced to a more complete, accurate or general explanation—, but is false or systematically uncorroborated. Replaced theories are abandoned, which usually entails that explanatory constructs postulated by these theories are eliminated.

Historically, constructs such as ‘phlogiston’ and ‘gravitational forces’ were eliminated once the explanations in which they figure were abandoned. By analogy, it may be argued that pain doesn’t play the explanatory role folk psychological explanations attribute it. For instance, the explanation posting that a conscious pain mental state causally mediates the transition from the hot stove stimulus to hand withdrawal is false, since this nociceptive response is largely reflex. In this case, eliminativism is justified, although it must be emphasized that the elimination doesn’t concern the phenomenological experience of pain as perceived from a first-person perspective or as reported by those experiencing it, but rather the causal role attributed to this experience by folk explanations. Thus, an eliminativist can deny that an explanatory construct of pain refers to a causal structure existing in the world, without denying that people experience pain. This is a position Daniel Dennett (1978) and Patricia and Paul Churchland (1981; 1986) seem to adopt.

Alternatively, Daniel Dennett (1978) and Valerie Hardcastle (1999) argue that pain is an incongruous concept encompassing both subjective experiences and a set of common intuitions about what it means to be in pain, such as the beliefs that one cannot
be mistaken about being in pain and that being in pain is sufficient for having an awful experience. This raises a problem for folk psychological explanations, since a subject may simultaneously hold contradictory 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. The IASP definition of pain\(^\text{25}\) seems to suffer from a similar incongruity. The definition tells us that pain is an unpleasant experience while at the same time endorsing reporting as a valid, reliable and accurate method for measuring pain. Yet patients with frontal lobotomies and cingulotomies sometimes report feeling similarly intense, but not distressing pain in response to noxious stimulation. Since such pain concepts and definitions are logically inconsistent, they should be eliminated.

This variety of eliminativism too is uncontentious. The proposed elimination does not concern the perceived intensity of pain experiences or their negative valence, but the necessary and universal association between pain intensity, unpleasantness and certain cognitive appraisals. This is just another way of saying that the three dimensions along which pain varies—sensory-discriminative, motivational-affective, and cognitive-evaluative—can be dissociated\(^\text{7,8}\). Some authors take this variety of eliminativism to be superfluous, since they doubt that the necessary and universal association targeted for elimination is part of most people's conception of pain in the first place\(^\text{6,10}\). As for the so-called IASP definition of pain, it may be argued that it is not what philosophers understand by a definition—namely, necessary and sufficient conditions for something to count as pain—, but rather the description of a statistically significant phenomenon documented in the general human population. This is consistent with the fact that the studies to which Dennett and Hardcastle allude describe natural or controlled experiments in which certain interventions (strokes, cingulotomy, analgesics, hypnotic suggestion) result in atypical pain experiences.

Since concepts and constructs are revised in light of empirical findings, these varieties of eliminativism are compatible with scientific practice. Moreover, they are at least in part justified. Problems arise when elimination targets pain as a phenomenon to be explained. Elimination is viewed as necessary because, according to an identity model of reductive explanation, aspects of subjective experience and psychological phenomena that cannot be identified with biophysical entities, processes or mechanisms constitute an unexplainable, irreducible and immaterial phenomenological surplus. To restore physicalism and the explanatory closure of science, any surplus should be eliminated.

Some eliminativists treat this potential phenomenological surplus as an artefact created by a misrepresentation or false theory of reality. Larry Hardin (1988)\(^\text{11}\) and Georges Rey (1997)\(^\text{12}\) argue that some aspects of subjective experience must be a sort of error, since they represent physical objects as having phenomenological properties which they don’t really have. For instance, we feel that pain is in the burned hand, but the hand itself does not have the property of being in pain. This is particularly obvious in the case of phantom limb pains. The apparent objectivity of phenomenological properties that seem to be located ‘out there,’ in the external world, is a misrepresentation of reality and should be eliminated. Following a similar line of argumentation, Paul Churchland (2007)\(^\text{13}\) targets for elimination the apparent subjectivity of some conscious experiences. He suggests that we commonly think of and report some experiences as being subjective, and therefore distinct from objective physical reality, simply because we failed to realize that these experiences represent, and ultimately refer to objective states of physical reality.

Daniel Dennett adopts a more mitigating approach, relegating talk of pain experiences to a scientifically naive model of reality. This model remains instrumentally useful for predicting behaviour, but has no place in modern science. At the ‘personal level’ of phenomenological experience, pain is unanalyzable into any kinds of components, thus blocking any further investigation of mechanisms. We may 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)\(^\text{15}\). It must be emphasized that Dennett does not deny the existence and usefulness of phenomenological reports. Notwithstanding, such reports are effectively eliminated from science.

Despite its physicalist motivations, this last variety of eliminativism clashes with current experimental and explanatory paradigms. The life sciences comprise primarily experimental disciplines relying on statistical methods of data analysis for characterizing reproducible correlations and on controlled experiments for discovering causal structures linking correlated variables. Roughly speaking, the former constitutes phenomena in need of an explanation, while the latter are the main building blocks of mechanistic explanations. For instance, the object of study in pain research is not the subjective experience of pain simpliciter, but an experimental model of pain. It could be an experimental setup consisting of human subjects reporting their experiences of pain in response to noxious stimulation. The phenomenon replicated in this model amounts to the fact that subjects are not randomly outputting pain values, but reliably report certain values when presented with certain stimuli and information, in
conjunction with certain behaviours, when administered analgesics, etc. More generally, the phenomenon of pain consists of an extended network of correlations between stimuli, pain as experienced in self-experimentation setups, pain as reported by other subjects, medical conditions, descriptive terms appearing in verbal reports of pain, biological, psychological and social variables, etc.

Can certain aspects of a phenomenon be eliminated? For instance, can the correlation between chest pain and reduced blood flow to the heart be ignored given that a sensation of pain cannot possibly be in the chest? Should we recharacterize the phenomenon of angina or replace the variable ‘pain location’ with something else? I think this kind of elimination is unwarranted. If ‘pain location’ can be reliably measured and, moreover, it systematically correlates with other measured variables, then pain location is part and parcel of empirical reality.

Explanation too, diverges from the identity model assumed by eliminativists. Some of the most ambitious experiments in neuroscience rely on interventions targeting biological factors. If such interventions result in differences in psychological outcomes, as contrasted with comparable controls, it may be inferred that biological factors are causally relevant to these psychological outcomes. Nothing here demonstrates that psychological states are identical with (or, more generally, supervene on) biological states. If anything, according to standard causal metaphysics, controlled experiments can only demonstrate what psychological states are not identical with, namely the biological factors shown to causally impact on these states.

The argument extends to mechanisms as well. Mechanisms are hypothesized or reconstructed by compiling knowledge about various causal determinants. 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; the second, that ibuprofen inhibits the production of prostaglandins, which are known to sensitize spinal neurons to pain.

Likewise, one may argue that the recently discovered pattern of neural activity predicting whether a subject will report a heat stimulus as being painful or not refers to a putative mechanism of pain, not pain itself. It reveals which structures and patterns of brain activity should be monitored to measure pain or targeted by interventions in order to alter pain experience, but it doesn’t demonstrate the identity of pain with a biological activity.

The net result is an abundance of causal explanations and a lack of evidence for psychoneural identities. The gap between the explanatory project in neuroscience and the mind-brain identity assumption led some authors to abandon reductionism altogether and explicitly embrace a thoroughly causal metaphysical picture of reality in which physical and mental states are distinct and equally real causes and effects. In its most familiar form, this amounts to epiphenomenalism about mental states. More generally, it refers to an exclusively causal model that precludes any form of reductive physicalism postulating non-causal relationships (supervenience) between biological and mental states.

Still, controlled experiments do not prove that psychological states are not identical with some biological activity either. Absence of evidence is not evidence for absence. It is mere lack of information, which can only justify agnosticism. Given that many psychological variables lack a clear physical interpretation (we don’t know what exactly we are measuring when we measure these variables), it is possible that two operationally defined variables, one psychological and one biological, refer to the same biological activity—as reductionists have it—, even though controlled experiments show that one variable is causally relevant to the other. It is therefore possible that as a physical interpretation of psychological variables emerges, an extended causal structure involving biological and psychological variables, such as those postulated by biopsychosocial models of pain, will eventually collapse into a set of strictly biological mechanisms.

Interestingly enough, in this scenario, nothing needs to be eliminated. The starting point is that of agnosticism about the physical interpretation of certain psychological variables. This entails that the phenomenon of pain does not come with a built-in physical or metaphysical interpretation that needs to be subsequently eliminated. As for variables, correlations, and phenomena, they are all still there. A psychological and a biological variable may ultimately refer to the same biological factor, but this does not mean that the variables themselves are identical. They are still measured by different techniques, such that distinct observable outputs with distinct characteristics (different units, values, scales, etc.) are produced in virtue of the distinct inner workings of the techniques in question.

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References