No evidence that pain is painful neural process

Commentary on Key on Fish Pain

Riccardo Manzotti
Department of Psychology
IULM University

Abstract: Key (2016) claims that fish do not feel pain because they lack the neural structures that have a contingent causal role in generating and feeling pain in mammals. I counterargue that no conclusive evidence supports the sufficiency of any mammalian neural structure to produce pain. We cannot move from contingent necessity in mammals to necessity in every organism.

Riccardo Manzotti  riccardo.manzotti@gmail.com  is professor of psychology at IULM University (Milan). He is the author of more than fifty papers on the basis of consciousness. His main area of research is the physical basis of consciousness — what is consciousness in the physical world? He endorses a radical externalist framework for consciousness that goes beyond embodied cognition and enactivism. www.consciousness.it

Key's (2016) target article states that pain “arises in the cortex” because the “necessary and sufficient […] minimal neural architecture” underpins “conscious neural processing.” Bluntly, he claims that species that lack the neural substrate with which mammals generate pain cannot feel pain— hence, because they lack this neural substrate, fish do not feel pain. Such a view is questionable.

Just to state the obvious, feathers are necessary for birds to fly. Yet, however important feathers are for birds, their absence does not imply that flying is impossible. In fact, bats fly without feathers. Likewise, certain neural structures can be perfunctory for pain in Homo sapiens but irrelevant in, say, trout. The argument from anatomical similarity has some force in positive cases, but is very weak in negative cases.

To make his case, Key points out that “brain regions,” “conscious neural processing,” “recurrent connections,” “dynamic and fluid activity of the network,” or “strong reciprocal and networked circuitry … uniquely defines the quality of the feeling.” Such evidence would only be persuasive if we had an account of how neural processes generate pain (Adolphs, 2015; Aru & Bachmann, 2015; Koch, 2012). Data based on correlation are valid only insofar as things are the same, which is not the case here. Are fish similar enough to mammals to make such an inference? Unfortunately, this is precisely what is unknown since nobody knows how neural activity generates pain. The more one shows that fish are different from mammals, the less conclusive the lack of homologous neural substrates.
Empirical evidence does not back up any necessary relation between neural structures and consciousness. At best, it shows only that certain neural structures are, in the case of human beings, contingently necessary. By *contingent necessity*, I mean that such structures are necessary given how natural selection singled out human beings. As far as we know, had *Homo sapiens* turned out differently, human beings might be conscious with completely different biological machinery. No known psycho-neural law forbids it.

Once Whitehead noted that some “assumptions appear so obvious that people do not know what they are assuming” (Whitehead, 1925, p. 48). Likewise, Feyerabend warned that implicit assumptions “enter the debate in the guise of observable events” (Feyerabend, 1975, p. 60). Such disguised assumptions are here embedded in the underlying model — incoming stimuli are transduced into neural signals that are eventually-yet-unfathomably “consciously processed,” thereby becoming “conscious (felt) neural processing” and allowing “pain to arise in the cortex.” Such a causal model is posited but never argued for. While Key assures us that in the future neuroscience will discover “how these self-regulatory oscillations may generate the pain sensation,” at present, nobody has a clue.

The very notion that neural substrates generate pain that only the organism can feel is problematic. First, we have no idea what biological mechanism — either at the cellular level or at the macro level — generates pain out of neural firings. Second, we have no idea what pain is. Third, we have no idea what is the entity that feels it. Because of such open issues, it appears difficult to draw any conclusion based on the absence of neural structures that, in mammals, correlate with pain.

It is well known that human beings, in certain situations, feel pain. Likewise, there is a wide consensus that such evidence can be extended to mammals (Coenen, Schlaepfer, Maedler, & Panksepp, 2011; Damasio & Carvalho, 2013; Panksepp, 2011). However, to what extent, can the neural evidence derived from mammals be applied to other vertebrates such as fish? While the gist of the mechanism that generates pain is unknown, neural evidence from mammals cannot be generalized.

Regarding the boundaries of the physical substrate of pain, even in mammals there is no definitive proof that neural activity is sufficient to generate pain. In all known cases, neural structures are involved, but so are bodies, the environment, stimuli, tissue damage, past and future behavior, and social interactions. We have no reason to discard all of that in favor of the neural underpinnings alone. Thus, we have no reason to move on from the fact that human beings feel pain to the much narrower hypothesis that neural structures feel pain. No cases of pain in a vat have ever been reported. The claim that neural processes are conscious would be an instance of the so-called mereological fallacy (Bennett, 2003). The standard approach in neuroscience is to address the physical substrate of feelings at the system and the cellular level (Damasio & Carvalho, 2013). It might be worth adding an ecological level that encompasses a broader network of causal processes, including the body and the environment. The substrate that generates the feeling of pain may be distributed in multiple systems and their interactions.
Of course, in the present context, the key question remains whether fish have the requisite neural substrate. And the reply is that we cannot answer by checking for the presence of the kind of neural substrates that in mammals are associated with pain.

References


