We do not need a third mechanistic descriptor for chronic pain states! Not yet

Letter To Editor:

Kosek et al¹ should be commended for attacking the difficult task of improving terminology of persistent pain conditions with no clear medical explanation and for their invitation to debate their proposal. Unfortunately, their proposal will not move the field forward. The main reason is that the new terminology is so imprecise and vague that it can aid neither clinical thinking nor research. By the end of their topical review, Kosek et al state, "as our knowledge of pain mechanisms advances, so should pain terminology change." While this statement may seem self-evident, even "pain terminology" lacks precision, and the authors do little to specify what exactly they mean. For example, does the phrase pain terminology refer to the phenomenal aspects of pain, to physiologic mechanisms that may lead to pain, to various challenges that can provoke pain, or to the meaning of pain in an evolutionary or personal context?

It may be pertinent to remind ourselves of what pain is and what it is not. Pain is a feeling experienced by a person, and although a functioning brain is necessary, the pain is not "in the person's brain," nor in his or her peripheral nerves or spinal cord. Pain may be evoked by a multitude of bodily and mental challenges. Although often temporarily associated with the feeling of pain, nociceptor activity or activity in pathways and cortical networks are not pain. Thus, nociceptor activation does not necessarily evoke pain, whereas paradoxically loss of signals from low-threshold mechanoreceptors may cause pain. Finally, mere expectations may evoke pain, without any nociceptor activation involved. When new clinical terminology is introduced, one expects it to serve a purpose, either in a clinical setting or in research. The proposed descriptor is, however, nothing more than a rather trivial clinical observation (ie, chronic pain patients with pain of unknown etiology displaying findings on a neurological examination regarding sensibility).

At the core of the proposal is the concept "altered nociceptive function," which, the authors do not describe more precisely. This is a critical weakness of the article because the whole argument for a new terminology rests on this concept. Which specific aspect of nociceptive function is altered in the patients the authors use as examples? Evidence of altered peripheral mechanisms is lacking or circumstantial regarding the pain conditions targeted by the new terminology (eg, fibromyalgia), and it seems that the authors favor that the altered nociception is of central origin. It is not obvious, however, that this helps to distinguish one case of chronic pain from another, and it certainly does not help to explain the pain experienced in medically unexplained pain conditions. In virtually all cases of persistent pain, regardless of cause, central changes (networks, gray matter reduction) have been reported. The problem is that these changes help very little to explain why the pain arises; they are so

far just structural correlates of persistent pain. Indeed, Kosek et al claim in their discussion that the terms dysfunctional pain and pathological pain "not only give no insight into possible mechanisms but also carry implications that may stigmatize patients." Yet, why should an ill-defined concept of altered nociceptive function be any better? (The authors also claim that, without any references, "patients suffering from altered nociceptive function typically respond better to centrally than peripherally targeted therapies".)

Because the central concept on which the new terminology rests is so unspecific that it can be applied to virtually all persistent pain conditions, "the candidate adjectives for this third descriptor" cannot help either. Is it indeed possible that any state of persistent pain does not have a correlate of altered nervous activity at any level of the peripheral or central nervous system? "Nociplastic," "algopathic," and "nocipathic" are just words with no explanatory power. Any persistent pain condition involves plastic changes and altered sensation, and the term nocipathic is equally vague and self-evident without further specification (does it help to describe fibromyalgia as a condition with a "pathological [ie, not "normal"] state of nociception"?).

In essence, although we have a structural (ie, nerve injury/disease or increased nociceptor activation) correlation to the pain, we do not have a proven causality. What we observe is a pain (localized, regional, or widespread) that seems correlated with nociceptive activation or nerve damage (or disturbed sensibility), nothing more. Because the proposition of Kosek et al neither serves a defined clinical or scientific purpose, it ought to be rejected until its necessity is justified. Words (eg, terminology) should never replace understanding; one might argue that is what happened to placebo.

Conflict of interest statement

The authors have no conflicts of interest to declare.

Acknowledgements

The author is indebted to professor emeritus Per Brodal for his invaluable feedback and comments.

Reference

- [1] Kosek E, Cohen M, Baron R, Gebhart GF, Mico JA, Rice ASC, Rief W, Sluka AK. Do we need a third mechanistic descriptor for chronic pain states? PAIN 2016;157:1382–6.

Lars-Petter Granan^{a,b}

^aDepartment of pain management and research, Oslo university hospital, Oslo, Norway

^bFaculty of Health and Social Sciences, University College of Southeast Norway, Drammen, Norway

E-mail address: lpgranan@gmail.com (L.P. Granan)

<http://dx.doi.org/10.1097/j.pain.0000000000000735>

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.