PAIN®

Towards a dynamic account of chronic pain

Letter to the Editor:

In their thoughtful perspectives article, Finnerup et al.[4] challenge the idea that there is a transition or shift from an acute nociceptive pain to another chronic pain type that is driven by central sensitization, spinal amplification, and altered descending pain modulation. As an alternative idea, they argue that after resolution of the original physiological cause, new types of pain can be

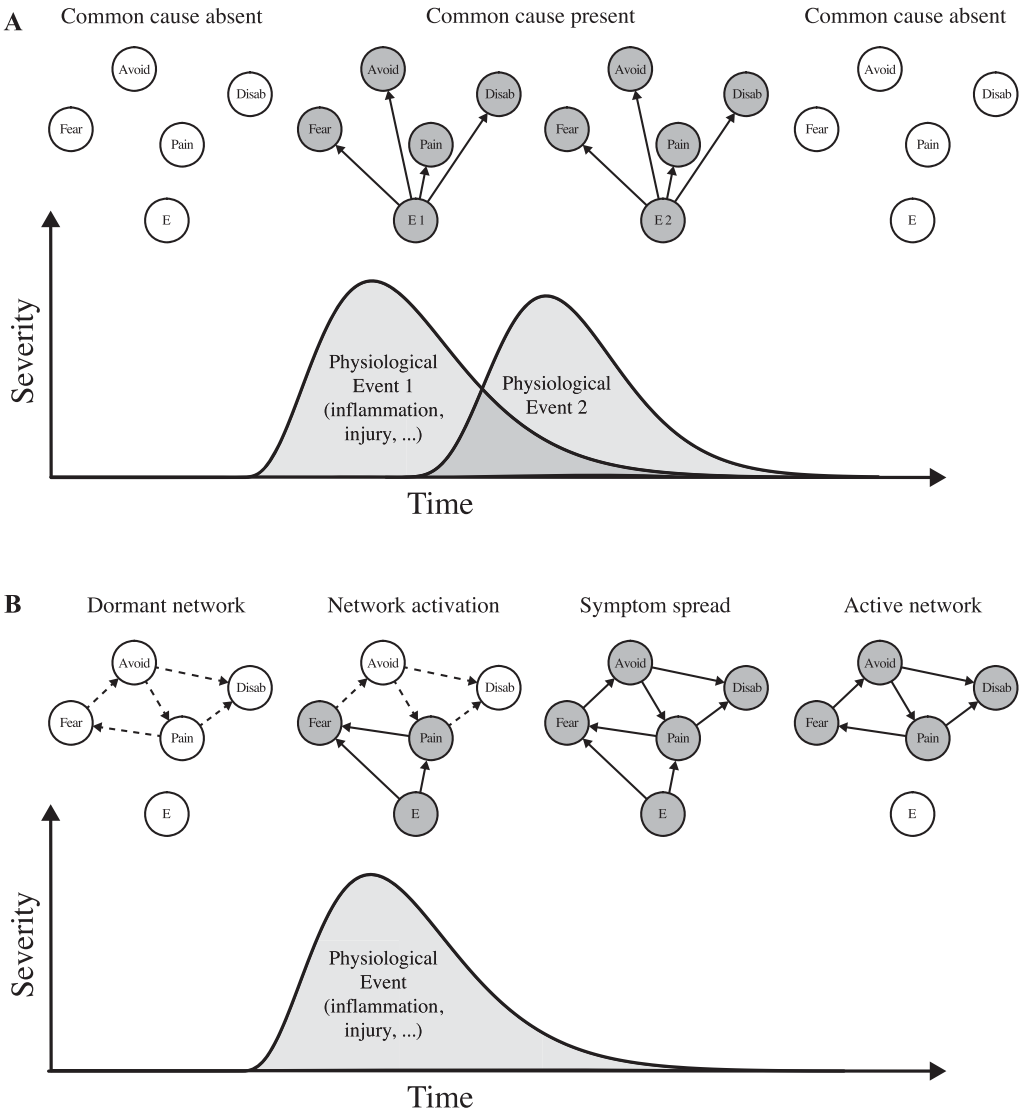

Figure 1. Development of chronic pain after injury (based on ref. 1). (A) A simplified presentation of the common cause model: symptoms (fear, pain, avoidance, disability…) are assumed to be caused by a common cause (E1 and E2, eg, a physiological event such as injury or inflammation). If the common cause resolves, the symptoms also disappear. (B) A simplified presentation of dynamics of the network model: symptoms influence each other and can be maintained even when the nociceptive or neuropathic contributions are resolved. For example, a physiological event (E) that is external to the network activates a dormant network of observable and connected symptoms such as pain and fear, which may spread to other symptoms, such as avoidance and disability. In a strongly connected network, removal of the physiological event does not lead to recovery as the network is self-sustaining.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

PAIN 163 (2022) e1038–1040

triggered by surgery or presurgical or postsurgical events, and that these pain types may develop in parallel with the original cause. The authors recommend identifying separate mechanistic pain types early as they may require different interventions.

We believe that the authors' ideas are interesting and deserve further thought. We also agree that we need a better understanding of the link between acute and chronic pain, and that current constructs such as central sensitization have clear limitations.[8] On the other hand, we also believe that the pain type idea put forward by Finnerup et al.,[4] albeit more diversified, may suffer from the same conceptual limitations as central sensitization: both share the common assumption that the behavioral manifestations of pain (the symptoms) are caused by a physiological event either at the central or at the peripheral nervous system (such as injury or inflammation). The consequence of this line of thought is a focus on the identification of the common cause, and when treated appropriately, the observable symptoms should disappear (**Fig. 1A**).

To the exasperation of many, a common cause for (chronic) pain cannot always be identified, which seriously compromises the search for a successful treatment. A different approach is needed. We propose to extend some of Finnerup et al.'s ideas[4] towards a more dynamic network approach of chronic pain. Specifically, what Finnerup et al.[4] seem to be ignoring is that pain is not only a response to damaged tissue but also part of a larger plexus. Pain is an emotional experience[6] and considered part of a motivational system that alarms, directs, and energizes behavior to minimize bodily harm.[3,10,11] Learning to predict, avoid, and control harmful events is crucial in this endeavor. Typically, escape and avoidance behavior are forms of protective control that promote healing in the short term and may become dysfunctional in the long term.[9] Consequently, rather than assuming that the symptoms are the effect of a latent (underlying) cause, complex pain disorders may better be conceptualized as systems of causally connected symptoms.[2,5] In the acute phase, physiological events can activate symptoms, which are usually observable and expressed through verbal–cognitive, psychophysiological, and behavioral–motoric responses. These symptoms however are not independent from each other, they also can interact, and even cause each other.[1] For example, pain may be mutually maintained by fear, and both can easily spread to avoidance and disability, which in turn increases pain sensitivity (**Fig. 1B**).

Such a network model has the advantage that it unifies both the idea that pain is triggered by physiological events, such as neural or nonneural damage, inflammation, as well as the idea that symptoms may interact and reinforce each other. The factors that trigger acute pain may not be the same as those that maintain pain, and in that sense, acute pain can dynamically transition into chronic pain. In addition, individual differences can be understood from network characteristics. Resilient networks quickly return to the original state, and vulnerable networks transit towards a state of dysfunction even after the disappearance of the perturbating event.[1] A particular advantage of the network approach is that it offers ways to intervene on clearly defined symptoms, and the specific intervention strategy can be informed by the characteristics of the network structure. For example, one may target the symptom that is most central in the network.[2] The network approach aligns well with learning theory and is currently the focus of research in the field of psychopathology.[7] We believe that its application in pain science is promising and hope that the ideas expressed in this letter will inspire pain research that contributes to a better understanding of complex chronic pain disorders.

Conflict of interest statement

The authors have no conflicts of interest to declare.

Acknowledgements

J.W.S. Vlaeyen is supported by the research program "From Acute Aversive Sensations to Chronic Bodily Symptoms," a long-term structural Methusalem funding (METH/15/011) by the Flemish government, Belgium, and J.W.S. Vlaeyen and M.L. Peters are supported by the NWO gravitation program "New Science of Mental Disorders": www.nsmd.eu (grant number 024.004.016) by the Dutch Research Council and the Dutch Ministry of Education, Culture and Science.
J.M.B. Haslbeck is supported by NWO Vici grant nr. 181.029.
R. Sjouwerman is supported by NWO Open Competition grant nr. 406.20.GO.032.

References

[1] Borsboom D. A network theory of mental disorders. World Psychiatry 2017;16:5–13.
[2] Borsboom D, Cramer AOJ. Network analysis: an integrative approach to the structure of psychopathology. Annu Rev Clin Psychol 2013;9:91–121.
[3] Craig AD. How do you feel? Interoception: the sense of the physiological condition of the body. Nat Rev Neurosci 2002;3:655–66.
[4] Finnerup NB, Nikolajsen L, Rice ASC. Transition from acute to chronic pain: a misleading concept? PAIN 2022;163:e985–88.
[5] McNally RJ. Network analysis of psychopathology: controversies and challenges. Annu Rev Clin Psychol 2021;17:31–53.
[6] Raja SN, Carr DB, Cohen M, Finnerup NB, Flor H, Gibson S, Keefe FJ, Mogil JS, Ringkamp M, Sluka KA, Song XJ, Stevens B, Sullivan MD, Tutelman PR, Ushida T, Vader K. The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. PAIN 2020;161:1976–82.
[7] Roefs A, Fried EI, Kindt M, Martijn C, Elzinga B, Evers AWM, Wiers RW, Borsboom D, Jansen A. A new science of mental disorders: using personalised, transdiagnostic, dynamical systems to understand, model, diagnose and treat psychopathology. Behav Res Therapy104096 2022; 153:104096.
[8] van den Broeke EN, Van den Bergh O. Central sensitization in humans: popular phrase or useful concept? J Psychosom Res 2019;119:51–2.
[9] van Vliet CM, Meulders A, Vancleef LMG, Vlaeyen JWS. The opportunity to avoid pain may paradoxically increase fear. J Pain 2018;19:1222–30.
[10] Vlaeyen JWS, Crombez G. Behavioral conceptualization and treatment of chronic pain. Annu Rev Clin Psychol 2020;16:187–212.
[11] Wall PD. On the relation of injury to pain. The John J. Bonica lecture. PAIN 1979;6:253–64.

Johan W.S. Vlaeyen[a,b]
Jonas M.B. Haslbeck[c]
Rachel Sjouwerman[a]
Madelon L. Peters[a]
[a]*Experimental Health Psychology, Maastricht University, Maastricht, Netherlands*
[b]*Research Group Health Psychology, KU Leuven, Leuven, Belgium*
[c]*Department of Psychological Methods, University of Amsterdam, Amsterdam, Netherlands*
E-mail address: j.vlaeyen@maastrichtuniversity.nl (J.W.S. Vlaeyen)
http://dx.doi.org/10.1097/j.pain.0000000000002706