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Editorial comment

Editorial comment on Karlsson et al. "Cognitive behavior therapy in women with fibromyalgia. A randomized clinical trial"

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In this edition of *Scandinavian Journal of Pain*, Karlsson et al. test the effect of a manual based cognitive behavioral intervention for female patients with fibromyalgia [1]. The randomized, controlled study focuses on maladaptive cognitions and behavior thought to maintain and exacerbate pain conditions. The bio-psycho-social model of pain, a definition of stress presented by Lazarus and Folkman [2], as well as the fear-avoidance model by Vlayen and Linton [3] provide the overarching theoretical framework. The authors use a dimension of the West Haven-Yale Multidimensional Pain Inventory as a primary outcome, hypothesizing that a cognitive behavioral therapy (CBT) developed for pain and stress would influence perceived life control. They also hypothesized secondary effects on interference from pain, emotional distress and social support, as well as stress, depression and pain severity. Their results showed a significant increase in life control, reduction in affective distress and depression, as well as a reduction of stress. The study tells a story about CBT changing the perception of pain in participants, giving a higher quality of life, even though they actually report more pain during the follow-up period.

Their results on stress reduction through CBT are enticing considering the different lines of research indicating the maintaining and exacerbating role of stress in chronic widespread pain [4]. Some even claim that poor stress management alongside cognitive-emotional sensitization, central sensitization and sustained arousal provides us with a causal model for such idiopathic conditions [5,6].

Hypotheses on how the stress construct can affect pain conditions stem in part from studies on childhood traumatic experiences. One study detailed how increased sensitivity to glucocorticoids in the hypothalamic–pituitary–adrenal (HPA)-axis and/or up regulated corticotrophin-releasing factor came as a result of early-life changes in the HPA-axis [7]. Sustained or increased glucocorticoid exposure can have adverse effects on the hippocampus, which causes decreases of synapses and decreased production of new neurons. This damage might progressively reduce the control of the HPA axis and lead to increased stress responses [6]. This would again increase central sensitization, a claim that has been demonstrated in animal experiments with stressed versus non-stressed mice [8]. What is more, overexposure to cortisol also negatively affects activation patterns in the prefrontal cortex. Administration of cortico-releasing hormones into the brains of animals produced integrated endocrine, autonomic and behavioral responses in line with depression and anxiety [9], both common comorbidities in fibromyalgia and chronic widespread pain.

Even though it is often the physiological stress mechanisms and antecedents that are targeted in studies on chronic stress, scientific consensus exists on initiation as well as termination of the stress response being susceptible to cortical dysregulation [10]. And, that this response can be delayed, excessive, flattened, or prolonged by such prefrontal regulation [11].

Normally, these physiological stress processes are regulated by precise feedback mechanisms. But it has been demonstrated several times how a delayed feedback circle includes cognitive modulation of the prefrontal cortex, and hippocampus. This prefrontal cortex modulation involves learning, specifically learning through expectancies about our ability to influence the outcome of stress situations [5]. This means that humans, as opposed to other animals like mice, actually have the capacity to overrule feedback loops through maladaptive cognitions. Hence, Karlsson et al.'s study underlines and supports the importance of targeting stress regulation in chronic pain, and shows that this can be done successfully through existing CBT manuals. Interestingly, this stress reduction is achieved even though the participants subjectively rate more pain, which would entail more stressful situations.

It is specifically the finding of participants reporting more pain, but more life control and less stress that touches on the broader development of psychological treatments of pain these recent years. While there is no denying of the contribution of CBT as a treatment for pain the last 30 years, there is also no denying the potential for improvement when it comes to its effectiveness [12]. Cognitive behavioural therapy has repeatedly been criticized for the lack of process studies in treatments of health complaints – meaning that we do not fully understand what causes the changes we observe [13]. As an example, the emergence and success of acceptance and commitment therapy (ACT) for chronic pain has challenged the more traditional processes of change thought to be salient in CBT. Willingness to experience pain and commitment to life values with present moment awareness has been shown highly

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relevant in chronic pain [14]. Particularly relevant here is that the theoretical model behind ACT would pose that a reduction in pain is not necessary for a higher quality of life [14]. Actually, in the ACT model one would *expect* an increase in subjective reporting of pain. As the willingness to experience pain to experience more value-based actions would in fact be an indication of treatment success [14].

Now, it is important to note that this theoretical model is not at odds with or opposed to the CBT model for chronic pain. The two main facets of the CBT model are that pain and its consequences are both related and separated from behaviour and functioning in our life domains, and the other being that cognition can affect pain experience [13]. Hence, it is not the intention to argue pro-et-contra for ACT or CBT when developing testing manuals for chronic pain patients. Rather, the point is that when testing and studying change processes, we as pain psychologists need to clarify, differentiate and progress cognitive behavioural therapies. And we need to do so through precise delineation and formulation of target cognitions, while at the same time recognizing all past successes of the CBT perspective. In this regard, the study from Karlsson et al. gives a good example of how a known process (stress regulation) still can be further delineated and operationalized to help provide us with even more effective CBT treatments in the future.

Conflict of interest

The author declares that they have no conflict of interest.

References

[1] Karlsson B, Burell G, Anderberg UM, Svärdsudd K. Cognitive behavior therapy in women with fibromyalgia. A randomized clinical trial. Scand J Pain 2015;9:11–21.
[2] Lazarus RS, Folkman S. Stress, appraisal and coping. Springer Publishing Company; 1984.
[3] Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 2000;85:317–32.
[4] Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011;152:S2–15.
[5] Ursin H, Eriksen HR. Cognitive activation theory of stress (CATS). Neurosci Biobehav Rev 2010;34:877–81.
[6] Ursin H. Brain sensitization to external and internal stimuli. Psychoneuroendocrinology 2014.
[7] Yehuda R. Post-traumatic stress disorder. N Engl J Med 2002;346:108–14.
[8] Alexander JK, DeVries AC, Kigerl KA, Dahlman JM, Popovich PG. Stress exacerbates neuropathic pain via glucocorticoid and NMDA receptor activation. Brain Behav Immun 2009;23:851–60.
[9] Koob GF. Corticotropin-releasing factor, norepinephrine, and stress. Biol Psychiatry 1999;46:1167–80.
[10] Sapolsky RM. Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. Arch Gen Psychiatry 2000;57:925.
[11] Oitzl MS, Champagne DL, van der Veen R, de Kloet ER. Brain development under stress: hypotheses of glucocorticoid actions revisited. Neurosci Biobehav Rev 2010;34:853–66.
[12] Eccleston C, Williams ACdC, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults. Cochrane Database Syst Rev 2009;2 [the cochrane library].
[13] McCracken LM, Vowles KE. Acceptance and commitment therapy and mindfulness for chronic pain: model, process, and progress. Am Psychol 2014;69:178.
[14] Vowles KE, Sowden G, Ashworth J. A comprehensive examination of the model underlying acceptance and commitment therapy for chronic pain. Behav Ther 2014;45:390–401.