MUSCULOSKELETAL PAIN
IASP Special Interest Group

FACT SHEET

The Use of Quantitative Sensory Testing when Assessing Musculoskeletal Pain

- **Kristian Kjær-Staal Petersen, Dr.Med., PhD, MSc**, Center for Neuroplasticity and Pain (CNAP), SMI, Department of Health Science and Technology, School of Medicine, and Center for Mathematical Modeling of Knee Osteoarthritis (MathKOA), Department of Material and Production, Faculty of Engineering and Science, Aalborg University, Aalborg, Denmark.
- **Tuhina Neogi, MD, PhD**, Section of Rheumatology, Department of Medicine, Boston University Chobanian & Avedisian Schoool of Medicine, Boston, USA.
- **David Rice PhD, BHSc**, Health and Rehabilitation Research Institute, School of Clinical Sciences, Auckland University of Technology and Department of Anaesthesiology and Perioperative Medicine, Te Whatu Ora Waitematā, Auckland, New Zealand.

Assessment of Somatosensory Function using Quantitative Sensory Testing

For most musculoskeletal (MSK) pain disorders, nociception arises in the peripheral nervous system, is transmitted through the peripheral nervous system to the dorsal horn and further transmitted upwards to higher brain centers, where the pain experience is produced. Additionally, the descending pain pathways (neurons projecting from the brain down to the dorsal horn) can be activated and inhibit or facilitate incoming nociceptive signals. Quantitative sensory testing (QST) is a neurological examination of somatosensory function, that provides information about the transmission and modulation of nociception in 1) the peripheral nervous system, 2) the dorsal horn and 3) via the descending pain pathways.

Although many QST modalities exist, people with MSK pain are often most sensitive to pressure stimuli, as these target muscles and joints and MSK pain conditions are often mechanically mediated (such as osteoarthritis). Commonly, pressure pain thresholds (PPTs) are utilized as a proxy assessment of localized pressure hyperalgesia, which is a sign of peripheral pain sensitivity, temporal summation of pain (TSP) is often utilized as a proxy assessment of the excitability of dorsal horn neurons and conditioned pain modulation (CPM) is utilized as a proxy assessment for descending pain inhibitory control[2].

Quantitative Sensory Testing findings in people with MSK pain

Substantial evidence suggests that, on average, people with chronic MSK pain have lower PPTs, higher TSP and impaired CPM when compared to healthy pain-free people[2], which could indicate that the nervous system is sensitized to nociceptive input. Some studies indicate that the extent of pain duration might be associated with a higher degree of pain sensitivity[1]. It is important to understand that there is substantial variation in QST findings among people with chronic MSK pain[10] and therefore it would be incorrect to state that all people with chronic MSK pain are pain sensitive. Instead, it is likely that subsets of more or less pain sensitive people with chronic MSK pain exist, particularly given that the pain experience is influenced by numerous factors.

Potential as predictors of MSK treatment outcomes

Studies have investigated whether the extent of pain sensitivity could be linked to treatment outcomes for common MSK treatments. In general, a higher degree of pre-treatment pain sensitivity has been linked to 1) chronic postoperative pain after total joint replacement surgery, 2) lower analgesic effect to non-steroidal anti-inflammatory drugs (NSAIDs) and paracetamol, and 3) poor

pain relief to exercise-based therapies[5,6]. Although these associations significantly predict treatment outcomes, it is important to highlight that the predictive value of QST is low-to-moderate[6], suggesting that QST cannot be a stand-alone prognostic tool in the clinic. Chronic MSK pain is complex with multifactorial contributors, involving several pain mechanisms, lifestyle factors, and psychosocial influences, among others. As such, it is likely that a broader multimodal approach towards prediction models is needed.

Factor likely to influence QST findings in people with MSK pain

A subset of people with MSK pain are obese, inactive and report poor quality of sleep, and these lifestyle factors are likely to influence pain sensitivity in MSK pain. Obesity is not directly associated with pain sensitivity, but obesity is associated with a higher degree of pro-inflammatory cytokines, and animal studies suggest that these cytokines can sensitize both peripheral and central nociceptive neurons, leading to increased pain sensitivity[8]. Similarly, preclinical studies have demonstrated that inactivity is linked to a higher level of inflammation[4], which can potentially sensitize the nociceptive system, and human studies suggest that improving physical activity might be linked to less pain sensitivity[5]. Finally, poor sleep quality is linked to increased clinical pain in people with chronic MSK pain and data suggest that sleep quality may modulate the extent of pain sensitivity[3,9]. Psychological factors (e.g. depression, anxiety) may influence QST findings in MSK pain, but recent data questions this association[7] and therefore this should be further studied in the future.

Conclusion

Substantial evidence suggests that people with chronic MSK pain frequently have more pain sensitivity than healthy pain-free people, and there are subgroups of people with chronic MSK pain who are more pain sensitive than others. Higher degrees of pain sensitivity have been linked to poor response to standard pain treatments such as joint replacement surgery, NSAIDs and paracetamol, and exercise-based therapy. Lifestyle factors such as obesity, inactivity and poor quality of sleep might be indirectly associated with the degree of pain sensitivity in people with chronic MSK pain.

References

[1] Arendt-Nielsen L, Egsgaard LL, Petersen KK, et al. A mechanism-based pain sensitivity index to characterize knee osteoarthritis patients with different disease stages and pain levels. Eur J Pain;19:1406–1417 2015.

[2] Arendt-Nielsen L, Morlion B, Perrot S, et al. Assessment and manifestation of central sensitisation across different chronic pain conditions. Eur J Pain;22:216–241 2018.

[3] Hertel E, McPhee ME, Petersen KK. Investigation of pain sensitivity following 3 nights of disrupted sleep in healthy individuals. Eur J Pain 2023.

[4] Lesnak JB, Berardi G, Sluka KA. Influence of routine exercise on the peripheral immune system to prevent and alleviate pain. Neurobiol pain (Cambridge, Mass);13:100126 2023.

[5] Lyng KD, Andersen JD, Jensen SL, et al. The influence of exercise on clinical pain and pain mechanisms in patients with subacromial pain syndrome. Eur J Pain:1–14 2022.

[6] Petersen KK-S, Kilic K, Hertel E, et al. Quantitative sensory testing as an assessment tool to predict the response to standard pain treatment in knee osteoarthritis: a systematic review and meta-analysis. PAIN Reports;8:e1079 2023.

[7] Plinsinga ML, Vuvan V, Maclachlan L, et al. Pain-related cognitions and emotional distress are not associated with conditioned pain modulation: an explorative analysis of 1142 participants with acute, subacute, and chronic pain. Pain;164:1593–1599 2023.

[8] Schaible HG. Nociceptive neurons detect cytokines in arthritis. Arthritis Res Ther;16:470 2014.

[9] Staffe AT., Bech MW, Clemmensen SLK, et al. Total sleep deprivation increases pain sensitivity, impairs conditioned pain modulation and facilitates temporal summation of pain in healthy participants. PLoS One;14:e0225849 2019.

[10] Vaegter HB, Graven-Nielsen T. Pain modulatory phenotypes differentiate subgroups with different clinical and experimental pain sensitivity. Pain;157:1480–1488 2016.