Musculoskeletal Pain: With focus on osteoarthritis

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Today's management of osteoarthritis (OA) consists mainly of symptomatic treatment of pain. New safe and more efficient treatment paradigms are needed for OA but such a development requires a solid fundamental understanding of the central pain mechanisms involved in OA.

In the individual patient little association is found between joint damage, local inflammation, and pain as the central nervous system due to sensitization amplify the peripheral nociceptive input. Recently degree of sensitization (spreading extra-segmental sensitization), descending pain control, and central temporal and spatial integration are some of the individual central nervous system factors playing a role for pain amplification, pain severity and hence the disconnect between extend of damage and pain intensity perceived by the patient. Although the peripheral sources of pain in OA are not well understood we know that continuous nociceptive barrage from joint and extraarticular nociceptors will eventually activate the central sensitization processes with pain amplification and expansion of the receptive fields for the dorsal horn neurons (perceived pain areas will increase). It has consistently been shown that pain intensity, pain durations, and number of OA locations are important drivers for such central sensitization in OA.

Better understanding of the individual fundamental central pain mechanisms may improve patient profiling, help individualizing management, suggest new treatment options and thereby advance development of new therapies. Techniques for assessing the central pain sensitization mechanisms in OA patients have been developed and provide the opportunity to quantify pain mechanisms such as temporal summation, descending inhibition, and spreading sensitization. Such pain biomarkers can help to phenotype OA patients based on the role of the various central pain sensitization mechanisms involved. We have recently shown that such biomarkers can predict which patients are most vulnerable to develop chronic post-operative pain after knee replacement.