• **FACT SHEET No. 15**

Joint Imaging

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Osteoarthritis (OA) is a leading cause of joint pain. Although degeneration of articular cartilage is one of the hallmarks of OA, cartilage is aneural, and we still do not have a clear understanding of how the various structural changes of other joint tissues interact or how they account for the prevalence of pain, especially knee pain. Similarly, the variance of pain explained both by independent structural changes as well as their sum and interactions remain poorly defined.

OA is assessed radiographically either by semi-quantitative scoring systems such as the Kellgren and Lawrence grading scale or the Osteoarthritis Research Society International atlas, which grades tibiofemoral joint space narrowing and osteophytes separately for each compartment. In addition, quantitative joint space width (JSW) measurements can be accomplished either manually or in (semi-) automated fashion. Minimum JSW is the standard metric, but use of location-specific JSW has been reported.

Magnetic resonance imaging (MRI) may be evaluated by semi-quantitative scoring approaches, 3D segmentation, or by using compositional techniques. Several MRI scoring systems are available for the assessment of OA, with each having advantages and disadvantages. For assessment of synovitis, contrast-enhanced MRI enables more accurate evaluation than scoring of non-enhanced MRI data. Cartilage or meniscus quantification requires segmentation and exploits the three-dimensional nature of MRI data sets to evaluate tissue dimensions (such as thickness, areas volume, and others) as continuous variables. Compositional MRI techniques allow visualization of the biochemical properties of different joint tissues. It is therefore very sensitive to early, pre-morphologic changes that cannot be seen in

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conventional MRI. Its role for elucidating associations with pain incidence, progression, or fluctuation needs to be shown.

In population-based studies, a significant discordance between radiographically diagnosed OA and knee pain has been reported. Although radiographic evidence of joint damage predisposes to joint pain, the underlying pathologies leading to pain cannot be readily discerned from radiography alone and may require consideration of other factors.

Novel study designs are one approach to deal with the so-called structure-symptom discordance. For example, when individual differences influencing the pain experience are adequately accounted for, a strong relationship between radiographic OA and knee pain was observed. One study applying direct unanesthetized examination of articular tissues in the human knee joint has provided some insight into particular structures that do and do not elicit pain when probed. Using imaging modalities such as MRI, several structural alterations such as meniscal tears, subchondral bone marrow lesions, subarticular bone attrition, synovitis, and effusion have been related to knee pain.

Furthermore, changes in bone marrow lesions and inflammatory markers on MRI are associated with fluctuations in pain in patients with knee OA. How much of the variance in pain is accounted for by structural change is not fully understood. One reason for this difficulty is that most studies focused on late disease stages when numerous pathologic changes are already commonly present. In fact, abnormalities on MRI are very common even in knees that are considered to be radiographically normal.

A systematic review examined the concurrent relation of MRI findings in OA to symptoms. Of these, just over half demonstrated a statistically significant association, indicating that studies to date have found inconsistent associations of structural features to symptoms. Nonetheless, in general, large bone marrow lesions were strongly associated with knee pain, followed by synovitis and effusion, and cartilage volume and thickness. Interpretation of these relationships is challenging, as it is not clear as to whether all of these associations are truly causal or rather are markers of the severity of other structural pathology that may be contributing to the pain experience.

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