

Hand Enthesophytes and Knee Enthesopathy: Is Osteoarthritis Related to a Systemic Enthesopathy?

To the Editor:

We read with interest the recent article by Gibson and colleagues¹ and the accompanying editorial² on the role of enthesopathy in osteoarthritis (OA). Gibson, *et al* suggest that OA is not a systemic enthesopathic disease, based on the finding that there is no increase in radiographically determined hand enthesophytes in subjects with bone marrow lesions of the knee¹. We contend that there is insufficient evidence from the study to make such a claim.

It is considered that isolated knee OA and knee bone marrow lesions are relatively more sensitive to mechanical and trauma-related stressors like obesity and injuries^{3,4}, while hand OA has a stronger genetic basis⁵, a case of nurture (or lack of, in the case of the knee) versus nature (genetics). It would be more logical to use a more genetically related model (hand OA) as the basis for the study like Gibson's, and to look for further evidence of generalized enthesopathy in other joints, rather than to use a more environmentally related form of disease such as isolated knee OA.

A further consideration is that knee magnetic resonance imaging (MRI) is a sensitive technique with the ability to detect early changes in both marrow and soft tissue. However, plain radiography of the hands used in the study¹ is a less sensitive imaging technique, and would have been less sensitive at identifying small enthesophytes visible on MRI or even ultrasonography. As mentioned in the accompanying editorial², the uniplanar characteristic of the plain radiograph is unable to visualize possible enthesophytes at the tendon insertions, resulting in potentially lower counts of enthesophytes, in addition to completely overlooking early-stage enthesal abnormalities that are pretty ubiquitous in early hand disease⁶. MRI of the hands is a more sensitive imaging method to study hand OA⁶, and permits identification of enthesopathic processes, giving a much more accurate picture of the actual changes taking place.

Further, Gibson, *et al* looked only at enthesophytes at the mid-shaft of phalanges, therefore missing enthesophytes commonly seen at the distal insertions of collateral ligament in the hand⁶. In addition, enthesopathy includes not only enthesophytes, but other changes due to enthesitis, like bone edema and soft-tissue changes such as structural abnormalities of the tendons, ligaments, and joint capsules. Therefore using only plain radiography of the hands will underestimate the true representation of enthesopathy.

Gibson, *et al* attempted to help define generalized OA in relation to enthesopathy¹. Haugen² noted that generalized OA is a puzzle in that there was no consensus for the definition, and the study by Gibson, *et al* raised the question of whether there is a more systemic process or a more local biomechanical factor to explain the distribution of the enthesopathic changes. We would point out that the role of enthesopathy in OA remains difficult, but not a puzzle, with emerging literature that recognizes the het-

erogeneous expression of OA. For example, our work⁷ considers systemic enthesopathy-related OA as a specific subcategory of disease, which was omitted by Gibson, *et al* and the editorial. There is evidence that some OA is enthesopathic-related but much of it is clearly arising elsewhere — especially for knee disease. When OA is classified according to anatomic changes, we can see that early knee OA includes not only enthesopathic-related disease but also other disease categories, including osteogenic, meniscogenic, and chondrogenic variants⁷. This allowed a logical definition of OA based on structural changes in light of more recent knowledge, hence so-called idiopathic OA is no longer a phenomenon.

We therefore suggest that the study by Gibson, *et al* does not allow for conclusions about systemic enthesopathy in OA. In any event the evidence for such a claim may no longer be sustainable as enthesopathic-related OA represents a specific category of disease.

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REFERENCES

1. Gibson N, Guermazi A, Clancy M, Niu J, Grayson P, Aliabadi P, et al. Relation of hand enthesophytes with knee enthesopathy: Is osteoarthritis related to a systemic enthesopathy? *J Rheumatol* 2012;39:359-64.
2. Haugen IK. The puzzle of generalized osteoarthritis (OA) — Is OA a systemic enthesopathy? [editorial]. *J Rheumatol* 2012;39:203-5.
3. Neogi T, Zhang Y. Osteoarthritis prevention. *Curr Opin Rheumatol* 2011;23:185-91.
4. Koster IM, Oei EH, Hensen JH, Boks SS, Koes BW, Vroegindewij D, et al. Predictive factors for new onset or progression of knee osteoarthritis one year after trauma: MRI follow-up in general practice. *Eur Radiol* 2011;21:1509-16.
5. MacGregor AJ, Li Q, Spector TD, Williams FM. The genetic influence on radiographic osteoarthritis is site specific at the hand, hip and knee. *Rheumatology* 2009;48:277-80.
6. Tan AL, Grainger AJ, Tanner SF, Shelley DM, Pease C, Emery P, et al. High-resolution magnetic resonance imaging for the assessment of hand osteoarthritis. *Arthritis Rheum* 2005;52:2355-65.
7. McGonagle D, Tan AL, Carey J, Benjamin M. The anatomical basis for a novel classification of osteoarthritis and allied disorders. *J Anat* 2010;216:279-91.

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