Dr. Gibson and Dr. Felson reply

To the Editor:

We appreciate the interest from Tan and McGonagle[1] regarding our article[2], as we recognize their expertise in this subject. To clarify, we did include enthesophytes at the distal interphalangeal (DIP), proximal interphalangeal (PIP), and metacarpophalangeal joints, and did not limit examination to the shafts as suggested in their letter. These results are displayed in our Table 2[2]. They also showed no relation of enthesophytes in hands and knees.

We recognize that the sensitivity of plain radiographs of the hand is less than that of magnetic resonance imaging, especially since radiography does not show bone edema that may be indicative of enthesopathy. However, the use of plain radiographs to detect hand enthesophytes does potentially offer more specificity. Findings that are specific but not sensitive (like those on radiograph) generally reveal associations that are as strong as or stronger than findings that lack specificity. Our findings were not even suggestive of a tendency for there to be a generalized enthesopathy in osteoarthritis (OA). Therefore, while perhaps we did not conclusively reject any association, our findings provide strong evidence against one.

As Tan and McGonagle mention, the precipitants of knee OA and hand OA may differ, with a greater contribution of mechanical forces to development of changes in weight-bearing joints such as the knees. As they state, MacGregor, *et al*[3] found that there may be a genetic influence in the development of OA in the joints of the hands (especially the DIP and PIP) not found in other sites examined. This suggests that since OA is not really a generalized phenomenon in most cases, enthesophytes, if they were consistently related to OA, would also not be generalized as a systemic process. Further, the origin of enthesophytes may diverge from one joint to another, so that generalized enthesopathy may be as uncommon as generalized OA.

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Supported by NIH AR47785.

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J Rheumatol 2012;39;7; doi:10.3899/jrheum.120474