Response to: 'Correspondence on 'Which factors are associated with bone marrow oedema suspicious of axial spondyloarthritis as detected by MRI in the sacroiliac joints and the spine in the general population?' by Su *et al*

We would like to thank our colleagues from Taiwan for their interest in our work and their comments. The Study of Health in Pomerania (SHIP) cohort is a population-based epidemiological study which was designed to investigate common risk factors, subclinical disorders and manifest diseases with no interest in one specific disease but rather in data relevant for the prevalence and incidence of common diseases and their risk factors. Importantly, this was the first population-based cohort study worldwide which included whole-body MRI, which raised our interest many years ago.

We appreciate the comments of our colleagues from Taiwan on potentially better methods to assess MRI lesions suggestive of axial spondyloarthritis (axSpA). In general, we agree that the STIR sequence, in combination with a T1-weighted sequence, is currently the best method to identify axSpA-related lesions in sacroiliac joints (SIJs)² and the spine. As a matter of fact, in our study, short tau inversion recovery (STIR) sequences for the evaluation of the SIJs were used. For the evaluation of spinal MRIs, T1 and T2 sequences were simultaneously displayed on the evaluation monitors ('paired reading approach'). This allows for accurate evaluation of inflammatory (hyperintense on T2 and hypointense on T1) and structural (hyperintense on both sequences) MRI lesions. Since discrepancies between readers were rare (2.5% of all variables) and resolved by consensus reading,³ the background noise due to MRI sequences used was small. In addition, it needs to be stressed that we are not dealing with patients in this study but a sample from the general population of less than 45 years of age only some of which had back pain³ and just a few may well have had axSpA as suggested by linkage to claims data.4

The second point addressed by our colleagues is the scoring system used, and the authors seem to prefer the Spondyloar-thritis Research Consortium of Canada (SPARCC) over the Berlin score. However, both well-established scoring systems have been shown to perform similarly well, they have been used in many studies, and they have both strengths and weaknesses—which we can't discuss here in more detail. However, the argument put forward regarding the depth of lesions is as such not correct since the Berlin score even evaluates the depth of lesions more precisely with a grading of 0–3 than SPARCC, which uses a binary approach (0–1). Furthermore, what has been reported in our publication is not only the mere presence or the number of MRI lesions in the SIJ but the extent of lesions. In addition, symmetry of SIJ involvement does not matter so much, especially not in early disease.

The third point is actually most interesting because it deals with the role of human leukocyte antigen (HLA) B27. First, we would like to remind again that, in contrast to the work they cited, the participants of SHIP were not patients but volunteers from the general population. Furthermore, and our argument is not new—colleagues from Leeds already reported in 2008⁷ that the extent of bone marrow oedema in association with HLA B27 was clearly most relevant for the progression to more advanced disease stages, for example, ankylosing spondylitis. Thus, we are convinced, based on the data provided, that some bone marrow edema (BME) in the SIJ or some enthesitis⁸ is neither associated

with HLA B27 nor with a diagnosis of axSpA, but it is rather the severe cases that show such association—actually similar to what we have learnt in reactive arthritis. Therefore, although we agree that the role of HLA B27 has not yet been fully elucidated, we think that this HLA class I molecule is not relevant for mild BME in the SIJ that seems to rather frequently occur in the normal population.

Finally, we can only agree to the statement that the occurrence of BME needs further study. Actually, we have just recently shown that BME in SIJ alone is not very specific for a diagnosis of axSpA but performs much better in combination with chronic structural changes such as erosions.²

Taken together, and in agreement with the colleagues from Taiwan, we think that the most important lesson for rheumatologists from our cohort study is that caution is needed when diagnosing axSpA mainly based on imaging findings such as mild BME in the SIJ. Regarding the pathophysiology of axSpA, we think that the initial event of mechanical stress which seems to be rather prevalent in the population is not sufficient to explain a development to chronic disease. In addition, there are other factors such as a genetic disposition that leads to structural changes in the SIJ and later in the spinal column. It seems likely that the sex of patients also has an influence on this development.

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Handling editor Josef S Smolen

Contributors All coauthors worked on this corresponendence and approved the final version

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not required.

Provenance and peer review Commissioned; internally peer reviewed.

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To cite Baraliakos X, Richter A, Schmidt CO, et al. Ann Rheum Dis 2023;82:e171.

Received 4 May 2021 Accepted 5 May 2021 Published Online First 17 May 2021



► http://dx.doi.org/10.1136/annrheumdis-2021-220567

Ann Rheum Dis 2023;82:e171. doi:10.1136/annrheumdis-2021-220632

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