Response to: 'The role of infrapatellar fat pad resection in total knee arthroplasty' by White *et al*

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We thank White and Melhuish[1] for their interest in our original paper[2] and our response[3] to Bai *et al*.[4]

There is moderate level evidence in a recent systematic review showing that infrapatellar fat pad (IPFP) preservation improves clinical outcomes post total knee arthroplasty (TKA), whereas IPFP resection increases post-TKA knee pain.[5] This is contradictory with a previous systematic review suggesting that there were no differences in function, range of motion and anterior knee pain between preservation and resection groups after TKA in knee osteoarthritis (OA).[6] This new evidence is consistent with our findings that the IPFP maximal area was associated with reduced knee pain, decreased loss of cartilage volume and reduced risks of increases in cartilage defects cross-sectionally in older adults[7] and longitudinally in older women,[2] indicating a beneficial effect of IPFP size.

More epidemiological and clinical studies are emerging to show that IPFP size or preservation in TKA may have protective roles for knee symptoms and structural changes in knee OA. Teichtahl *et al*[8] reported that a larger IPFP at baseline was significantly associated with reduced knee pain at follow-up and decreased lateral tibial cartilage volume loss, and Cai *et al*[9] reported that greater IPFP volume was associated with greater tibial and patellar cartilage volume, and fewer cartilage defects, bone marrow lesions and osteophytes in patients with knee OA. The findings from Gwyn *et al*'s retrospective study[10] showed that complete resection of IPFP was associated with patellar tendon shortening 1 year after TKA, while there was no significant change in tendon length with partial resection of IPFP.

In contrast, IPFP with abnormal quality may play a detrimental role in knee OA. Our recent study demonstrated that IPFP signal intensity alteration, which was negatively associated with maximum area of IPFP, was associated with increased knee cartilage defects, subchondral bone marrow lesions and knee pain, and decreased knee cartilage volume cross-sectionally and longitudinally in older adults.[11] Consistently, we reported that some IPFP signal intensity measures, assessed using a semi-automated quantitative method, were consistently associated with increased structural abnormalities of the knee in patients with knee OA.[12]

All these evidences support our hypothesis that IPFP would have biphasic effects in knee OA: it may have a beneficial role physiologically through increased size, but could be detrimental when pathological changes are observed as signal intensity alteration on MRI. We proposed that IPFP with normal qualities, rather than abnormal qualities, should be preserved or not damaged during knee surgery,[3] which needs to be tested by well-designed randomised controlled trials.

In summary, we cannot totally agree with White's view that 'practice should transition to favour IPFP preservation where possible', but suggest to have IPFP quality evaluated using MRI before an action (IPFP preservation or resection) is taken in TKA. If there are no significant signal intensity alterations within IPFP, IPFP should be preserved during TKA. High quality randomised controlled trials with appropriate sample size and outcome measures are required to determine our hypothesis.

Contributors All authors contributed to this article.

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