Response to: 'Correspondence to 'Normal human enthesis harbours conventional CD4+ and CD8+ T cells with regulatory features and inducible IL-17A and TNF expression" by Wang and Ma

We thank Wang and Ma[1] for their comments on our description of T-cells and their cytokines profile at the normal human spinal enthesis.[2] Wang and Ma[1] report on synovial T-cells in psoriatic arthritis (PsA) obtained from synovial biopsies, and amongst other things, describes that infliximab therapy leads to a reduction in interleukin (IL)-23 related pathway transcripts indicating a potential pathogenic interplay between tumour necrosis factor (TNF)-α and IL-23/IL-17 axis at the synovium.[1]

The enthesis and synovium form what is known as the synovio-entheseal structure complex.[3] A major unresolved issue in the immunopathology of PsA, is the link between synovial and entheseal immune cells. Animal models suggest that disease either TNF or IL-23 originating enthesitis may drive synovitis,[4 5] but it is unclear if this is the case humans. It is possible that the findings of Wang and Ma[1] could be extended to the enthesis and bone but formal studies are needed since the precise link between these immune compartments is unclear. The authors' results suggest a 'dampening' of the IL-23/IL-17 axis following infliximab therapy by acting on TGF-β and aryl hydrocarbon receptor signalling, that are involved in the regulation of the 23/17 axis. These findings are of interest towards the further understanding of the link between the enthesis and synovium in PsA and spondyloarthritis.

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