Response to: 'Role of nerve growth factor (NGF) and tropomyosin receptor kinase A (TrkA) in the pathogenesis of osteoarthritis. Might NGF be the link interwinding obesity and OA?' by Iannone *et al*

We thank Dr Iannone[1] for his interest in our paper.[2] We share his interest in possible non-neuronal effects of nerve growth factor (NGF). In addition to possible effects on inflammation, chondrocytes and adipocytes as highlighted by Iannone *et al*,[1] NGF might also contribute to angiogenesis[3] and bone turnover.[4] Our data[2] suggest primary analgesic actions through reduction of sensitisation rather than through effects on inflammation, cartilage or bone turnover. However, although we did not observe structural disease-modifying effects of tropomyosin receptor kinase A (TrkA) inhibition, we cannot exclude small but clinically important effects. Non-neuronal effects of NGF or TrkA inhibition might be either beneficial or might contribute to adverse events, highlighting the importance not only of analgesic mechanisms, but also other possible roles of NGF–TrkA pathways. We agree that further investigation of the complex interactions between sensitisation, osteoarthritis structural damage, obesity and pain deserve further study.

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