healthy control individuals matched with ankylosing spondylitis (AS) patients to detect it.

We re-calculated the HWE p value of the SNP rs2284178 in healthy controls. The result is in accordance to Brown's. To confirm our conclusion that the tumour necrosis factor (TNF) gene *TNFα*–850 C→T SNP associates with AS independently of human leucocyte antigen (HLA) gene *HLA-B27*, we re-selected a tag SNP rs1131896 on *MICA* (MHC class I chain-related gene A), which is 47kb closer to *HLA-B27* and further away from *TNFα* than SNP rs2284178. The genotype frequencies of this SNP in both case (HWE p value = 0.212) and control groups (HWE p value = 0.274) did not deviate from HWE. The linkage disequilibrium (LD) test shows no linkage disequilibrium in both case (D′ = 0.256) and control (D′ = 0.057) groups between the SNPs rs1131896 and rs1799724. The results of SNP rs1131896 genotype and allele distribution between AS and control groups are shown in table 1. The LD maps for the two SNPs in the case and control groups are shown in figs 1 and 2.

HLA-B27 typing of AS patients was performed by flow cytometry and typing of healthy controls was carried out by polymerase chain reaction as described previously.[3] The *HLA-B27* positive and negative data we acquired for case and control groups merely represented phenotype rather than exact genotype. Therefore the D' value between the *HLA-B27* and *TNFα* loci could not be calculated using the phenotype results of HLA-B27. Though we cannot directly detect the linkage between *HLA-B* and *TNFα* by the LD mapping method, we can test the LD status within *TNFα* and genes located in the range from *HLA-B* to *TNFα*. The tags SNPs rs1131896 and rs2284178 showed no linkage disequilibrium with *TNFα* as calculated by the software Haploview 3.32 (http://www.broad.mit.edu/mpg/haploview/), although the result from the second SNP was controversial. Our results suggested that recombination between SNP rs1131896 and *TNFα* could have occurred and supported our conclusion that *TNFα*–850 C→T SNP associates with AS independently of *HLA-B27*.

Brown supposed that LD status between two markers is not able to reflect real recombination or linkage events, with an example resulted from the HapMap (http://www.HapMap.org) data for the Chinese population. However, this opinion is not effective just based on the example. The reasons are given below.

Firstly, the subjects used by Brown were collected from Beijing, where the make-up of the population is quite complex and hence some unknown factor(s) of population structure may have affected the power of the assessment.

Secondly, the small group size in the HapMap database also means it is unlikely to reflect real LD status in the whole Beijing population.

Finally, the minor allele frequencies (MAFs) of the rs11752262 and rs11757602 SNPs used by Brown were less than 5%. These low informative markers possibly show improper linkage relation.

When our data were corrected by the Bonferroni method, the SNP rs1799724 failed to show association with AS. But the biggest problem of Bonferroni correction is that it is too rigorous, which might increase the probability of a type II error, and thus makes it likely that genuinely significant results are rejected. For this reason, we also used other methods to correct the significance of the SNP rs1799724, such as the Bonferroni step-down (Holm) correction (p = 0.021),[4] and a 3000-fold permutation in Haploview 3.32 (p = 0.049).

Though our experimental data suggested that *TNFα*–850 C→T SNP associates with AS susceptibility independently of *HLA-B27*, whether *TNFα* was a genuinely susceptible gene to AS is yet be confirmed in other larger populations. Additionally, the mechanism of function of –850T variation on the whole disease process remains to be elucidated further.

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CORRECTION

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The authors of the paper by Song *et al* (Knee osteoarthritis. Efficacy of a new method of contrast-enhanced musculoskeletal ultrasonography in detection of synovitis in patients with knee osteoarthritis in comparison with magnetic resonance imaging. *Ann Rheum Dis* 2008;**67**:19–25) are listed in the wrong order. The correct order should be:

H Song, CE Althoff, KG Hermann, AK Scheel, T Knetsch, M Schoenharting, C Werner, GR Burmester, M Backhaus.

A corrected PDF is available online at http://ard.bmj.com/cgi/content/full/ard.2006.067462/DC1