Correction: Loss of balance between protective and pro-inflammatory synovial tissue T-cell polyfunctionality predates clinical onset of rheumatoid arthritis


We identified an error occurred in the merging of two data files which led to a mismatch of sample labels and corresponding gene counts for figure 3 in the original manuscript. We have now re-analysed the corrected data file. Outlined below is the ‘revised description of figure 3’ and in the corrected figure 3.

Revised description of figure 3:

Due to the rarity and low immune cell content of IAR synovial tissue biopsies, analysis of previously obtained bulk RNAseq data of synovial tissue biopsies from 132 RA patients 10 IAR and

Figure 3 IAR and RA patient synovial tissue samples are enriched for T cell-related gene pathways. (A) Enrichment plots of Bulk RNAseq data pathway analysis for significantly upregulated gene pathways for IAR versus HC, RA versus HC and RA versus IAR. Dot size represents number of differentially regulated genes per pathway, colour intensity represents significance and x axis is indicative of the pathway’s fold enrichment change. (B) Term plot of the MAPK pathway for IAR versus HC and RA versus HC comparison. Only significantly upregulated (green) or downregulated (red) gene members of the pathway are shown. HC, healthy control; IAR, individuals at risk; RA, rheumatoid arthritis.
28 HC was performed (GEO accession number GSE89408) (figure 3). Comparing IAR to HC synovial tissue biopsies, 11718 differentially expressed genes were identified. Of the 11718 genes, 9771 have known interactions belonging in 223 pathways with the KEGG database TCR activation, Th17 differentiation and Th1 and Th2 differentiation signalling pathways being significantly enriched in IAR compared with HC synovial tissue (figure 3A). Interestingly, pathway enrichment analysis of differentially expressed genes (14353 with 11412 genes with known interactions) in RA compared with HC synovial tissue shows enrichment in T cell activation and differentiation pathways similar to that of IAR (figure 3A,B). Importantly, the progression from IAR to RA is potentially dependent on 4437 differentially expressed genes (3959 of known interactions) belonging to 196 pathways including further enrichment in T cell activation and differentiation, HIF-1 signalling as well as key intracellular signalling pathways (PI3K-AKT signalling pathway) (figure 3A).