

00744 Characterisation of the gut microbiota in patients with rheumatoid arthritis

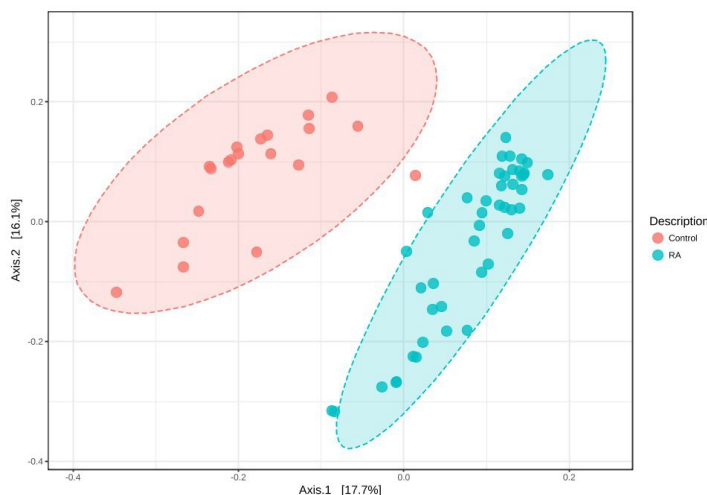
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Background: Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disease of unknown aetiology, characterized by an erosive synovitis that leads to the destruction of cartilage and bone in multiple joints. Recent studies suggest that alterations in the gut microbiota may contribute to the pathogenesis of the disease. Thus, we characterized the gut microbiota of 44 patients in different phases of the disease course (RAs) by comparing their faecal microbial community profiles with those of 20 healthy controls (HCs).

Materials/methods: DNA extracted from subjects' stool samples was amplified and the 16S rRNA gene V3–V4 region was sequenced using an Illumina MiSeq™ platform. For downstream sequence analyses, we used a combination of software packages QIIME (v1.9.1) and VSEARCH (v1.1), and we generated a biological observation matrix (BIOM) at different taxonomic levels (from phylum to genus). The BIOM was analysed using the Web-based program MicrobiomeAnalyst. Statistical analyses were performed using the R phyloseq software package. Differences in the relative abundance of individual bacterial taxa (i.e., genus level) between a priori defined groups were assessed by LEfSe analysis.

Results: RAs displayed a decrease in the gut microbial species richness compared to HCs. The principal coordinate analysis (PCoA) visualization of UniFrac distances and analysis of group similarities (ANOSIM) showed that faecal microbiota composition was significantly different between HCs and RAs ($P < 0.001$), suggesting differences in the relative abundance of specific bacterial taxa (see Figure below). The LEfSe analysis thus detected significant changes in the gut microbiota between RAs and HCs. Taxa overrepresented in RAs were *Bacteroidetes* and *Proteobacteria*, whereas those in HCs were *Verrucomicrobia* and *Firmicutes*. These findings suggest that gut microbiota is altered in RAs, but these changes seem to not relating with a particular RA phase, probably due to the low number of patients in the subgroups.



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Conclusions: This study poses the basis for further investigations about how alterations in the gut microbiota composition play a role in RA disease causation and progression.

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