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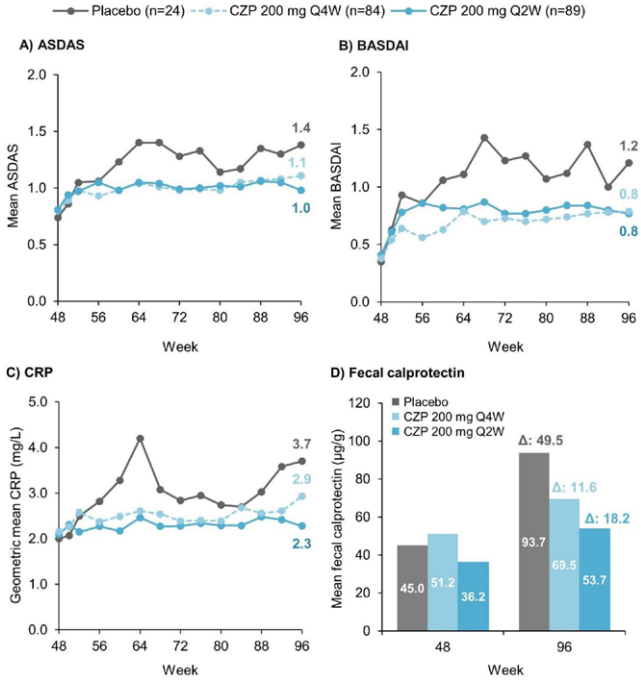
Figure. (A) ASDAS, (B) BASDAI, (C) CRP and (D) fecal calprotectin in patients who did not experience a flare during the C-OPTIMISE maintenance period (Weeks 48–96)



Missing data were imputed using last observation carried forward. Δ values for fecal calprotectin show change from Week 48. ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; CRP: C-reactive protein; CZP: certolizumab pegol; Q2W/Q4W: every 2/4 weeks.

Disclosure of Interests: Lianne S. Gensler Consultant of: AbbVie, Eli Lilly, Gilead, GSK, Novartis, Pfizer, UCB Pharma, Grant/research support from: Pfizer, Xenofon Baraliakos Speakers bureau: AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, Novartis, Merck, Pfizer, UCB Pharma, Paid instructor for: AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, Novartis, Merck, Pfizer, UCB Pharma, Consultant of: AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, Novartis, Merck, Pfizer, UCB Pharma, Grant/research support from: AbbVie, Merck, Novartis, Lars Bauer Shareholder of: UCB Pharma, Employee of: UCB Pharma, Bengt Hoepken Shareholder of: UCB Pharma, Employee of: UCB Pharma, Thomas Kumke Shareholder of: UCB Pharma, Employee of: UCB Pharma, Mindy Kim Shareholder of: UCB Pharma, Employee of: UCB Pharma, Robert B.M. Landewé Speakers bureau: Abbott, Amgen, BMS, Centocor, Merck, Pfizer, Roche, Schering-Plough, UCB Pharma, Wyeth, Consultant of: Abbott, Ablynx, Amgen, AstraZeneca, BMS, Centocor, GSK, Merck, Novartis, Pfizer, Roche, Schering-Plough, UCB Pharma, Wyeth, Grant/research support from: Abbott, Amgen, Centocor, Novartis, Pfizer, Roche, Schering-Plough, UCB Pharma, Wyeth

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POS0230 INTESTINAL MICROBIOTA CHANGES TNF-INHIBITORS INDUCED IN IBD-RELATED SPONDYLOARTHRITIS

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Background: the close relationship between joints and gut inflammation has long been known and several data suggest that the dysbiosis could represents the link between Spondyloarthritis (SpA) and Inflammatory Bowel Diseases (IBD). To date, the manipulation of the intestinal microbiota is considered the key to the cure or control of the natural history of several pathologies sustained or favored by dysbiosis. The introduction of biologic drugs, in particular Tumor Necrosis Factor inhibitors (TNFi), revolutionized the management of both these diseases, thanks to the strong inhibition of inflammation and partially indirectly with mechanisms not yet fully clarified. While the impact of conventional drugs on gut microbiota is well known poor data are available about TNFi.
Objectives: to investigate the impact of TNFi on gut microbiota.
Methods: we included CD or UC patients fulfilling criteria for axial or peripheral SpA (ASAS 2009) on a typical Mediterranean diet, naïve to biologics needing TNFi. Clinical history, physical examination, instrumental examinations, biochemical examination including C-reactive protein (CRP), erhytrocyte sedimentation rate (ESR), HLA-B27 and fecal calprotectin at the baseline and after 6 months were performed. TNFi included infliximab and adalimumab. Fecal samples were collected by patients themselves by 24 hours before the start of the therapy. The processing was performed through metagenomic NGS (next generation sequencing) including the amplification of the V3 and V4 regions of the 16S (V3 and V4) and sequencing on the Illumina MiSeq platform. All patients received the treatment at least until week 24. Clinical disease indices included Visual Analogue Scale (VAS)-pain and Visual Analgue Scale(VAS)-disease activity for all patients, Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) for axial involvement, clinical disease activity index (CDAI) and Health Assessment Questionnaire-Disability Index (HAQ-DI) for peripheral involvement, Harvey-Bradshaw Index for CD (HBI), or partial Mayo (pMAYO) score for UC, were assessed at baseline and at the end of the study. The study was approved by the ethics committee (approval code 0056924).
Results: we evaluated 20 patients affected by enteropatic arthritis, naïve for biologic drugs, treated with TNFi. After six months of therapy we observed a significant increase in Lachnospiracae family (Δ +10.3, p 0.04) and in Coprococcus genus (Δ +2.8, p 0.003). We also observed a decrease trend in Proteobacteria (Δ -8.0 p 0.095) and Gammaproteobacteria (Δ -9, p 0.093) and an increase trend in Clostridia (Δ +8.2 p 0.083). We didn't find differences between TNFi responders (SpA improvement or IBD remission achieved) and not responders in terms of alpha and beta-diversity.
Conclusion: the decrease of Proteobacteria and the increase of Lachnospiraceae and Coprococcus is consistent with the hypothesis that TNFi therapy, by decreasing inflammation, tends to restore the intestinal eubiosis. However further studies on larger cohort incuding the evaluation of gut virota and micobiota will be necessary to definitively clarify the effects of TNFi on the composition and function of the gut microbiota.

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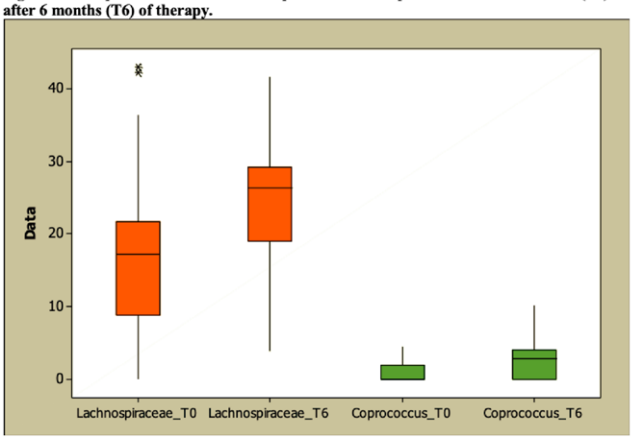
Table 1. Comparison between clinical variables between baseline and T6 (six months)

Clinical variables	T0	T6	P_value
Fecal Calprotectin(µg/g)- median (IQR)	207.5(125.5-446.2)	81(50-197.2)	0.004
CRP(mg/L)- median(IQR)	8.2(4.8-20.8)	2.9(1-4)	0.001
ESR(mm/h)- median(IQR)	21.5(10.8-34)	11(7.8-21)	0.003
VAS_pain- median(IQR)	50(38.8-60)	35(10-42.5)	0.001
VAS_disease- median(IQR)	50(38.8-50)	37.5(25-42.5)	0.006
HAQ- mediana(IQR)	0.6(0.1-0.8)	0.2(0.1-0.6)	0.004
BASDAI_score- median(IQR)	5.2(4.1-5.6)	2.8(2.5-4.3)	0.013
CDAI_activity- median(IQR)	13(10.5-16)	7(5.2-11)	0.004
IBD activity n(%) - 0	11 (55%)	20 (100%)	0.174
IBD activity n(%) - 1	6 (30%)	0 (0%)	
IBD activity n(%) - 2	2 (10%)	0 (0%)	
IBD activity n(%) - 3	1 (5%)	0 (0%)	

Disclosure of Interests: None declared.

Figure 1. Comparison between Lachnospiraceae and Coprococcus at the baseline (T0) and after 6 months (T6) of therapy.



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POS0231 IMPACT OF GENDER AND COMORBIDITIES IN ENTEROPHATIC SPONDYLOARTHRITIS: A CROSS-SECTIONAL STUDY

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Background: Enteropathic Spondyloarthritis (ESpA) belongs to the group of Spondyloarthritis (SpA) typically associated with inflammatory bowel disease (IBD). SpA are divided into axial (ax) or peripheral disease. Ax-SpA are classified as