analysis revealed a positive correlation between ADA levels and biochemical remission [AUC (95% CI) 0.67 (0.58–0.75), p = 0.0004]. An optimum ADA level of >8.9 µg/ml was identified for predicting biochemical remission (81.2% sens, 50.0% spec, positive LR 1.6). ADA levels but not ATA independently predicted biochemical remission in a multivariate logistic regression model.

**Conclusions:** Higher ADA levels were independently associated with biochemical remission; levels of >8.9 µg/ml, higher than previously suggested, might be an appropriate target in the maintenance treatment of CD.

## DOP Session 9: Advances in IBD Pathophysiology

### DOP073

### Results of the sixth ECCO Scientific Workshop: The pathogenesis of inflammatory extraintestinal manifestations of inflammatory bowel disease: implications for research, diagnosis, and therapy

C.R.H. Hedin[1]*, S.R. Vavricka[2], A.J. Stagg[3], A. Schoepfer[4], T. Raine[5], L. Puig[6], U. Pleyer[7], A. Navarini[8], A.E. van der Meulen - de Jong[9], J. Maul[10], K.H. Katsanos[11], A. Kagramanova[12], T. Greuter[13,14], Y. Gonzalez Lama[15], F.A. van Gaalen[16], P. Ellul[17], J. Burisch[18], D. Bettenworth[19], M. Becker[20], G. Bamias[21], F. Rieder[22]

*[1]Karolinska University Hospital, Gastroenterology Unit, Patient Area Gastroenterology, Dermatovenereology and Rheumatology, Stockholm, Sweden, [2]Centre for Gastroenterology and Hepatology, Zurich, Switzerland, [3]Barts and The London, Queen Mary's School of Medicine and Dentistry, Centre for Immunobiology, London, UK, [4]CHUV - Centre Hospitalier Universitaire Vaudois, Department of Gastroenterology, Lausanne, Switzerland, [5]University of Cambridge, Department of Medicine, Cambridge, UK, [6]Universitat Autònoma de Barcelona, Department of Dermatology, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain, [7]Charite - University Hospital Berlin, University Eye Clinic, Uveitis Center, Berlin, Germany, [8]University Hospital Zurich, Department of Dermatology, Zurich, Switzerland, [9]Leiden University Medical Center (LUMC), Department of Gastroenterology and Hepatology, Leiden, The Netherlands, [10]Gastroenterologie am Bayerischen Platz, Berlin, Germany, [11]University and Medical School of Ioannina, Department of Internal Medicine I, Division of Gastroenterology and Hepatology, Ioannina, Greece, [12]The Loginov Moscow Clinical Scientific Centre, IBD Department, Moscow, Russian Federation, [13]Mayo Clinic, Gastroenterology Research Unit, Rochester, MN, USA, [14]University of Zurich, Department of Gastroenterology and Hepatology, USZ, Zurich, Switzerland, [15]Hospital Universitario Puerta De Hierro, Intestinal Inflammatory Disease Unit / IBD Unit, Gastroenterology and Hepatology Service / Gastroenterology and Hepatology Department, Madrid, Spain, [16]Leiden University Medical Center (LUMC), Department of Rheumatology, Leiden, The Netherlands, [17]Mater Dei Hospital, Department of Medicine, Division of Gastroenterology, Triq Dun Karm, L-Imsida, Malta, [18]North Zealand University Hospital, Department of Gastroenterology and Hepatology, Frederikssund, Denmark, [19]University Hospital Münster, Department of Medicine B, Gastroenterology and Hepatology, Münster, Germany, [20]Triemli Hospital, Department of Ophthalmology, Zurich, Switzerland, [21]Sotiria Hospital, National and Kapodistrian University of Athens, GI Unit, 3rd Academic Dpt. Of Internal Medicine, Athens, Greece, [22]Cleveland Clinic Foundation, Department of Gastroenterology and Hepatology, Cleveland, USA*

**Background:** Approximately 50% of inflammatory bowel disease (IBD) patients experience at least one extraintestinal manifestation (EIM) during their disease course. The pathogenic mechanisms that cause EIM are incompletely understood, however unravelling these pathways has the potential to enhance our understanding of the pathogenesis of IBD overall and improve patient care.

**Methods:** The workshop comprised gastroenterologists of ECCO as well as experts from rheumatology, ophthalmology and dermatology. One group critically appraised the scientific evidence supporting a range of proposed pathogenic mechanisms of EIM, whilst the second group defined strategies and clinical tools that could be employed in future research in EIM. Unanswered questions in the field of EIM were identified.

**Results:** In order to facilitate systematic inclusion of patients in scientific and clinical studies and to align outcome measures across studies, research evaluating EIM needs to be underpinned by widely agreed definitions of the pathology being studied. However, such clear criteria are often lacking in EIM. We considered the evidence supporting the role of the gut microbiota and genotype in EIM pathogenesis as well as the differential therapeutic efficacy of drugs in IBD and EIMs, which may reveal pathogenic mechanisms. We defined two broad groups of speculative immune pathways: 1. Extension of immune responses from the intestine and 2. EIM as independent, but associated inflammatory events. The adequacy of the currently available animal models for the investigation of pathogenic mechanisms in EIM was appraised. The biomarkers and clinical and patient-reported outcome measures currently available to assess inflammation in different somatic systems were compared and suggestions made for future clinical and research use.

**Conclusions:** There is an urgent need to increase understanding of pathogenesis of EIM both to identify new molecular treatment targets, as well as to enhance the strategic application of the currently available drugs according to the character and somatic distribution of inflammation in individual patients. New knowledge challenges traditional organ-based paradigms and we predict that consideration of the total inflammatory burden will drive treatment decisions and define clinical and research outcomes in the future. Many currently available clinical and patient-reported outcome measures are organ-based; therefore novel tools for diagnosis and monitoring of inflammation are needed.

### DOP074

### IL-33 promotes gut mucosal wound healing by inducing miRNA-320 to stimulate epithelial restitution and repair

L.R. Lopetuso[1,2]*, C. De Salvo[2], L. Di Martino[2], N. Rana[2], W. Goodman[2], F. Scaldaferri[1], A. Armuzzi[1], A. Gasbarrini[1], T.T. Pizarro[2]

*[1]Catholic University of Sacred Heart, Internal Medicine Department, Gastroenterology Division, Rome, Italy, [2]Case Western Reserve University, Pathology, Cleveland, USA*

**Background:** IL-33 and ST2 are crucial factors in IBD.However, animal studies to uncover their mechanistic function(s) have yielded ambiguous results. The aim was to characterise the precise role of the IL-33/ST2 axis following acute epithelial injury and mucosal repair in DSS-induced colitic mice.