is a systemic autoimmune disease affecting all organ systems. The aim was to present a cohort of lupus patients who in the course of the disease presented with Hashimoto's thyroiditis.

Methods A cohort of 10 patients, female, aged 21–42 years, suffering from SLE is presented. The patients were diagnosed with lupus and were either on treatment with hydroxychloroquine or with hydroxychloroquine and prednisone. Within this cohort a female patient aged 42 years had also antiphospholipid antibodies and had suffered a stroke at the age of 36.

Results Within this cohort 6 patients had positive both anti-thyroglobulin and thyroid peroxidase antibodies, 3 patients had positive only anti-thyroglobulin antibodies and 1 patient had positive only thyroid peroxidase antibodies. Within this group, 7 patients were euthyroid and were followed up, while 3 had hypothyroidism and were on treatment with thyroxine.

Conclusion In conclusion, SLE may be accompanied by Hashimoto's thyroiditis. In another cohort a two-fold increased risk of Hashimoto's thyroiditis was observed in lupus patients. The presence of anti-Sm antibodies was found to favor this association. In another cohort hypothyroidism, subclinical hypothyroidism and subclinical hyperthyroidism accompanied by the presence of thyroid autoantibodies was observed in a group of lupus patients. It appears that lupus patients may present with Hashimoto's thyroiditis with or without hypothyroidism and should be screened for this disorder during long-term observation.

PO.4.90 REMISSION AND CLINICAL PATTERNS OF SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) IN SOUTHERN PAKISTAN: A RETROSPECTIVE COHORT STUDY

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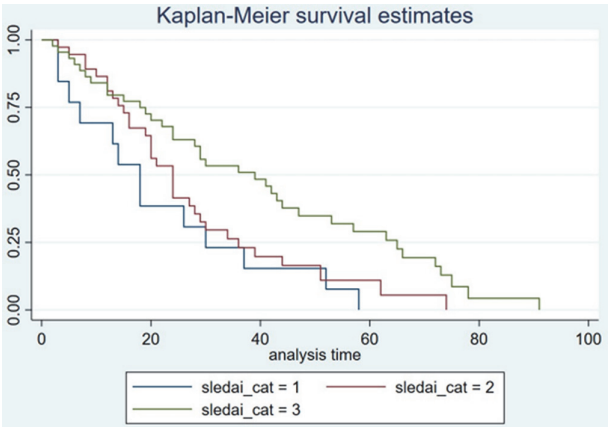
Background SLE is a difVcult to treat autoimmune disease due to clinical heterogeneity, unpredictability of disease iares and limited therapeutics. These challenges are worsened in a low-middle income country (LMIC) setting, yet clinical epidemiology from LMIC may have global beneVts.

Objectives To determine (i) the clinical pattern of SLE and (ii) the effect of SLE severity and treatment regimen on time to remission.

Methods A retrospective cohort study of 200 SLE patients' medical records (2014–20) from ImmunoCure clinic was conducted. Patients fulVlled ACR criteria 1997 for SLE classiVcation. SLEDAI-2K categories were used as outcome measure: mild (score <=6), moderate (7–10), severe (>10) to evaluate clinical pattern of SLE. Statistical analyses were performed using STATA v16.0. Kruskal-Wallis test was used for continuous measures, and Pearson's chi square test was used to compare categorical variables across SLEDAI severity. Remission status based on DORIS criteria and time to remission (>1year; n=94) was the secondary outcome.

Total doses of all drugs were calculated. Survival regression performed with Kaplan Meier curve.



Abstract PO.4.90 Figure 1

Results Most frequent antibodies are anti-dsDNA (63%), SSA (24%) and Ku (17.5%). Anti-cardiolipin (aCL) antibodies associate with severe SLE (OR = 3.6, P<0.01). Most common presentations were arthritis (85%), alopecia (53%), anemia (38%), rash (35%) and CNS disease (28%). Nephritis, CNS disease, cytopenias and oral ulcers are signiVcantly associated with severe SLE (P<0.01). ILD is in 10% of our cohort. Frequency of severe SLE was 47.5%, whereas mild disease was 16.5%. Mean duration of follow up was 41±19 months.

Every month of follow-up increased the odds of remission by 6% (P<0.05). Clinical remission on treatment (at Pred<=5mg) was successfully achieved in 62% patients. Complete remission (off all drugs & Pred) was achieved in 24 patients (14 in severe SLEDAI category) out of 200, with a mean post remission follow-up of 18 ± 15 months.. Hazard of time to remission is 61% (CI: 0.21–0.77, P=0.01) less in severe SLE as compared to mild SLE disease activity (Figure 1).

Conclusion Sustained remission is possible even in severe SLE in a LMIC setting if adequate immunosuppression is provided with persistent clinical follow-up.

PO.4.91 BANK1 AND IL-10+ B CELLS: BRINGING SOME LIGHT TO THE RELATIONSHIP BETWEEN MICROBIOTA COMPOSITION AND AUTOIMMUNITY DEVELOPMENT IN A MURINE MODEL OF LUPUS

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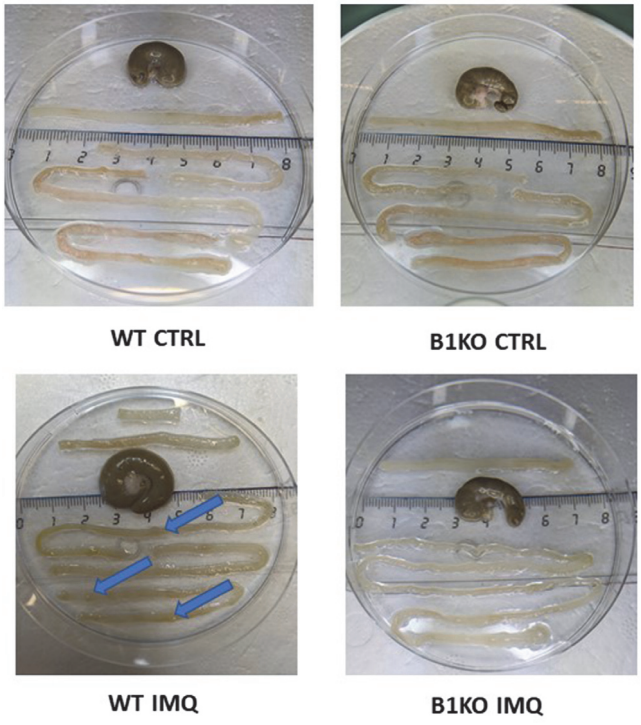
Background A new feature that seems to be decisive in autoimmune pathogenesis is the gut microbiota composition. However, its exact role remains to be determined. In systemic lupus erythematosus (SLE), an autoimmune disease characterized by persistent inflammation affecting multiple organs, the contribution of the gut microbiota is particularly elusive. The B cell scaffold with ankyrin repeats (Bank1) gene, which plays a role in TLR7 signalling, has been genetically associated with lupus in humans, and associated with a

reduction in disease severity in mice. In the recent years, IL-10 producing B cells (so-called regulatory B cells) are being characterised for their immunosuppressive role in autoimmune inflammation and possible relationship with microbiota composition.

Purpose We aimed to determine the influence of the gut microbiota composition, driven by Bank1, in IL-10+ B cell induction as well as the possible role of these cells in disease severity.

Methods We have characterised the manifestations of lupus in the gut associated lymphoid tissue using an imiquimod-induced murine model of lupus (TLR7-dependent). To analyse IL-10 producing B cells, immune cells isolated from Peyer's patches of C57Bl/6 WT mice and Bank1 -/- were stimulated with PMA/Ionomycin/Monensin and analysed by flow cytometry. To study microbiota influence in IL-10 production by B cells, we used single cage mice and littermates and sequenced the V4 region of 16S RNA gene.

Results and Conclusion The imiquimod-induced murine model of lupus leads to persistent gut inflammation with changes in microbiota composition in WT and Bank1 -/- mice. In these mice, the intestinal inflammation and local immune alterations resulted in increased gut permeability and caused intestinal blockade (image 1). This affectation is similar to that observed in human SLE patients. Mice deficient for Bank1 gene experienced a milder disease and exhibited a microbiota composition that was significantly different compared with their WT counterparts (principal components analysis). More specifically, the appearance of specific species belonging to the genus Porphyromonadaceae upon the induction of an inflammatory process only in Bank1 knock-out mice were related with reduced disease severity. To determine the contribution of gut microbiota to lupus inflammation, we induced the disease in littermate mice that inherited the Bank1 KO microbiota. We observed a normalized immune response that seems to be more alike to that observed in Bank1 KO mice grown separately from their WT counterparts. We then analysed the IL-10-producing cells in the gut and found that IL-10+ B cells readily increased in Peyer's patches upon lupus inflammation in Bank1 deficient mice, but not in WT mice. When lupus was induced in littermates, the levels of IL-10+ B cells were normalized across WT and Bank1 KO mice, suggesting a possible role of microbiota in regulatory B cell induction. Further studies are, however, needed to determine whether Bank1 is required for IL-10 producing B cell activation or if its contribution is mediated by changes in the gut microbiota composition.



Abstract PO.4.91 Figure 1 Representative intestinal tracts from WT and Bank1 KO (B1KO) mice at 20 weeks of age with (IMQ) and without (CTRL) lupus induction. It can be appreciated how the inflammation in WT mice after the treatment induces an increase in the caecal size and the Peyer's patches dimension (normally not visible without microscopic magnification, pointed with arrows in the WT IMQ image). In B1KO mice, however, the caecum remains unaltered and the inflammation at Peyer's patches levels is significantly lower than that achived by WT mice. Representation of three different experiments with 3–5 mice per group each experiment

PO.4.92 TAXONOMY, TREATMENT, TARGETS AND REMISSION IN SYSTEMIC LUPUS ERYTHEMATOSUS: THE 3TR-SLE STUDY PROTOCOL

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Purpose Taxonomy, Treatment, Targets and Remission (3TR) is a transdisciplinary consortium funded by the Innovative Medicine Initiative (IMI) and European Federation of Pharmaceutical Industries and Associations (EFPIA), aimed at performing a longitudinal multi-dimensional molecular analysis in patients with autoimmune, allergic, and inflammatory diseases. The main hypothesis of the 3TR project is that data obtained from multilevel omics analysis across seven different diseases will identify shared biological pathways that better predict response or non-response to therapies despite their differences in terms of clinical phenotypes and pathogenetic mechanisms. Systemic lupus erythematosus (SLE), rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease, asthma, and chronic obstructive pulmonary disease are the chronic disorders that will be investigated for shared biomolecular pathways.

Methods Centralised and standardised clinical data and sample collections will be a resource for studies and knowledge. Patients from multiple European centers are recruited for a longitudinal clinical follow-up and collections of blood, urine, stools, saliva, and relevant tissue samples at multiple timepoints. Among other analyses, we plan to perform transcriptome profiling in blood and tissues, including single-cell