Background The circulating free DNA (cfDNA) originating mostly from the abnormal cell apoptosis, necrosis or netosis contains sequences of microorganisms encountered previously by the patients. Therefore, it may be a source of information about past infections and may become a tool to evaluate human microbiome in relation to specific diseases. The aim of the study was to identify bacterial sequences in cfDNA of patients with different types of glomerulopathies.

Methods Blood samples from 9 patients with lupus nephritis (LN), 5 with IgA nephropathy, 4 with membranous nephropathy and 3 healthy controls were collected once. cfDNA was isolated (QI Amp, Qiagen) and quantified (Thermo Fisher Scientific, Waltham). Sequencing libraries were constructed, and quality checked (KAPA-Roche, Basel). Samples were sequenced on NextSeq 550 (Illumina, San Diego), before a multi-step bioanalysis.

Results Bacterial sequences represented 0.031% of the cfDNA. The most frequent bacterial genera in the cfDNA of patients with glomerulopathies included: Escherichia, Streptococcus, Klebsiella, Brevundimonas and Moraxella and they varied between the studied patient groups. The cluster of four LN and one MN patients had distinctive bacterial cfDNA pattern which was observed on the species, family, order, class and phylum level.

Conclusions Bacterial sequences in cfDNA of patients with lupus nephritis differ from patients with IgA nephropathy and membranous nephropathy. Validation in a larger patient population is warranted.
External validity is tested by replication in independent cohorts from the Danish Blood Donor Study and Copenhagen Biobank.

Conclusion Clinical tools based on genetics predictive of SARDs are in wanting but have generally been judged of little to no useful information. In this study, we will provide a validated predictive model of SARD based on multiple genes and interaction with non-genetic factors.

Acknowledgement Supported by the Danish Rheumatism Association

**P91 THE DEVELOPMENT AND VALIDATION OF A POLYGENIC RISK SCORE FOR MYOCARDIAL INFARCTION IN SLE**

Sarah Reid, Johanna K Sandling, Andrei Alexsson, Pascal Pucholt, Christopher Spjøtvoll, Karoline Lerang, Andreas Jønsen, Iva Gunnarsson, Ann-Christine Syvänen, Anne Trolleborg, Anne Voss, Anders A Bengtsson, Øyvind Molberg, Søren Jacobsen, Elisabet Svenungsson, Lars Rönnblom, Dag Leonard, Uppsala University, Uppsala; Linköping University, Linköping, Sweden; University of Oslo, Oslo, Norway; Lund University, Lund; Karolinska Institutet, Solna, Sweden; Aarhus University, Aarhus; Odense University Hospital, Odense; Copenhagen University Hospital, Copenhagen, Denmark

10.1136/lupus-2020-eurolupus.135

Background Patients with SLE have increased morbidity and mortality due to cardiovascular disease. Here, we construct and validate a polygenic risk score (PRS) for myocardial infarction (MI) in SLE.

Methods Patients with SLE (European decent, ≥4 ACR-criteria) were genotyped using a 200K Immunochip SNP array (discovery cohort, Sweden, n=776) and custom MassARRAY assays (replication cohort, Norway/Denmark, n=890). In the discovery cohort, 57 SNPs with previously established association with SLE development (p<5.0×10^-8) were investigated for associations with MI using a cox regression model. Significant SNPs were included in a PRS, weighted by their ORs for MI development. The PRS was subsequently validated in the replication cohort.

Abstract P90 Figure 1

External validity is tested by replication in independent cohorts from the Danish Blood Donor Study and Copenhagen Biobank.

Conclusion Clinical tools based on genetics predictive of SARDs are in wanting but have generally been judged of little to no useful information. In this study, we will provide a validated predictive model of SARD based on multiple genes and interaction with non-genetic factors.

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