Results: The study included 4886 SLE patients and 24430 age- and sex-matched controls. The SLE group had a significantly higher proportion of FMF patients compared to non-SLE controls (0.68% and 0.21% respectively; $p < 0.001$).

Table 1. All study population

Table 1. SLE patients and matched controls basic characteristics

	No SLE <i>N</i> =24430	SLE <i>N</i> =4886	<i>p</i> .overall
Age	51.2±16.5	51.2±16.5	1.000
Gender: Female	20100 (82.3%)	4020 (82.3%)	1.000
FMF	52 (0.21%)	33 (0.68%)	<0.001

Table 2. Stratification

Table 2. comparison of FMF patients with and without SLE

	FMF without SLE <i>N</i> =52	FMF with SLE <i>N</i> =33	<i>p</i> .overall
Age	44.6±13.7	50.5±17.7	0.106
Gender: Female	45 (86.5%)	26 (78.8%)	0.523

Conclusion: FMF was found to be more common amongst SLE patients compared to matched controls. The current study results suggest that the occurrence of SLE turn patients with an appropriate genetic and environmental setting to develop also FMF. This cross-sectional study sheds light on the coexistence of these two diseases, autoimmune and autoinflammatory.

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AB0420 RISK OF DEVELOPING TYPE 2 DIABETES MELLITUS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Patients with systemic lupus erythematosus (SLE) have higher than in general population prevalence of diabetes mellitus (DM). Hyperinsulinemia is a predictor of developing type 2 DM, however routine measurement of insulin levels for DM risk assessment is uncomfortable in daily clinical practice. International Diabetes Federation recommends the use of patient questionnaires to quickly identify people who may be at a higher risk of DM development.

Objectives: To determine the 10-years risk of developing type 2 DM in SLE patients using dedicated questionnaire - Finnish Type 2 Diabetes Risk Assessment Form (FINDRISK) data.

Methods: The study included 92 SLE patients without DM (83 women, 9 men, 39 [34; 47] years old). The median disease duration was 6 [2,14] years, SLE-DAI-2K was 4[2;8]. SLE pts were treated with glucocorticoids (GC) (89%) and hydroxychloroquine (78%), immunosuppressive drugs (28%) and biological agents (10%). The control group consisted of 88 subjects without systemic rheumatic diseases, inflammatory arthritis or DM, matched by age and sex with SLE patients. Eight items of FINDRISK questionnaire (age, overweight, abdominal obesity, family history of diabetes, physical inactivity, eating habits, history of antihypertensive drugs treatment, history of hyperglycemia) were taken into account to calculate the total risk score (TS). The risk of developing DM within following 10 years is regarded as low (1%) or slightly elevated (4%) with TS ≤11 points, as moderate (17%), high (33%) or very high (50%) with TS ≥12 points.

Results: The risk of developing DM was low or slightly elevated in 65 (71%) SLE pts and moderate, high or very high in 27 (29%) pts. The difference was significant compared with the control group, in which 76 (86%) subjects had a

low or slightly elevated risk and 12 (14%) had a moderate, high or very high risk ($p=0,01$). The number of risk factors (4[2;5]) and the median TS of SLE pts (9[5;12] points) were higher than values in control subjects (3[2,4] factors and 6[3;9] points, respectively) ($p<0,01$ for both). DM risk factors profiles were similar in two groups, except for higher prevalence of abdominal obesity (66% vs 41%, $p<0,01$) and history of antihypertensive drugs treatment (57% vs 17%, $p<0,01$) in SLE. There were positive correlations between TS and CRP levels ($r=0,25$, $p=0,02$), SLICC ($r=0,36$, $p<0,01$), HAQ ($r=0,29$, $p<0,01$), and negative correlations between TS and SLEDAI-2K ($r=-0,32$, $p<0,01$), glomerular filtration rate by CKD-EPI ($r=-0,23$, $p=0,03$). Current GC use had no influence on TS values in SLE.

Conclusion: Patients with SLE were more likely than individuals without systemic rheumatic diseases to have a moderate, high and very high risk of developing DM, and therefore, required interventions to prevent the metabolic disease. Increased risk of developing DM was associated with most common traditional factors, especially by abdominal obesity and regular use of antihypertensive drugs that can be considered a kind of equivalent to the presence of hypertension. Current contribution of inflammation, lupus activity and irreversible damage index can't be ignored. Clarification of SLE-specific phenomena in DM pathogenesis requires further research.

Disclosure of Interests: : None declared

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AB0421 EFFECT OF BODY WEIGHT ON COMPLEMENT LEVELS IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: The complement system is a recognized biomarker for diagnosis or monitoring of disease activity in systemic lupus erythematosus (SLE) patients (pts). But on the other hand, it has been linked to insulin resistance and obesity in general population.

Objectives: To find out whether overweight/obesity can modify C3 or C4 levels in SLE pts.

Methods: A total of 92 SLE pts (83 women, 9 men, 39 [34;47] years old) were enrolled in the study. Median disease duration was 6[2;14] years, and SLE activity using SLEDAI-2K was 4[2;8]. SLE pts were treated with glucocorticoids (89%), hydroxychloroquine (78%), immunosuppressants (28%), biologics (10%). The overweight/obesity status was determined by World Health Organization criteria in patients with body mass index (BMI) ≥25kg/m².

Results: Overweight/obesity were established in 46% SLE pts. Overweight/obese SLE pts were older than pts with normal BMI (40[36;48] vs 37[31;44] years, $p=0,02$), and had lower SLEDAI-2K (3[2;6] vs 6[4;8], $p<0,01$). Lower C3 concentrations were found in 36% overweight/obese pts vs 68% pts with normal weight ($p<0,01$), decreased C4 levels - in 19% vs 30% pts ($p=0,33$), median C3 concentrations were 0,98[0,81;1,14] g/l vs 0,84[0,69;0,96] g/l ($p<0,01$), and C4 levels were 0,15[0,10;0,19] g/l vs 0,12[0,09;0,16] g/l, respectively ($p=0,03$). C3 and C4 levels negatively correlated with SLEDAI-2K ($r=-0,5$, $p<0,01$ for both), the effect was more strongly pronounced in patients with BMI≥25kg/m² ($r=-0,6$, $p<0,01$ for both) than in those with normal weight ($r=-0,2$, $p=0,09$ for C3, $r=-0,3$, $p=0,04$ for C4).

Conclusion: Overweight/obesity status in SLE pts was associated with increased levels of complement proteins, therefore decreased C3 or C4 levels in patients with BMI≥25kg/m² are more likely related to disease activity and, can potentially induce SLE flares.

Disclosure of Interests: : None declared

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AB0422 LEFT VENTRICULAR ABNORMALITIES IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS FOLLOWED BY SEQUENTIAL ECHOCARDIOGRAPHY

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Background: Cardiovascular disease (CVD) is detected in up to 50% of systemic lupus erythematosus (SLE) patients¹ and major cause of death². Even clinically silent SLE patients can develop left ventricular (LV) diastolic dysfunction³. Proper echocardiographic follow up of SLE patients is required.

Objectives: To clarify how the prevalence of LV abnormalities changes over follow-up period and identify the associated clinical factors, useful in suspecting LV abnormalities.

Methods: 29 SLE patients (24 females and 5 men, mean age 52.8±16.3 years, mean disease duration 17.6±14.5 years) were enrolled. All of them underwent echocardiography as the baseline examination and reexamined over more than a year of follow-up period (mean 1075±480 days) from Jan

2014 to Sep 2019. Patients complicated with pulmonary artery hypertension, deep venous thrombosis or pulmonary embolism and underwent cardiac surgery during the follow-up period were excluded. Left ventricular(LV) systolic dysfunction was defined as ejection fraction (EF) < 50%. LV diastolic dysfunction was defined according to ASE/EACVI guideline⁴. LV dysfunction (LVD) includes one or both of LV systolic dysfunction and LV diastolic function. Monocyte to HDL ratio (MHR) was calculated by dividing monocyte count with HDL-C level.

Prevalence of left ventricular abnormalities was analysed at baseline and follow-up examination. Clinical characteristics and laboratory data were compared among patient groups as follows; patients with LV dysfunction (Group A) and without LV dysfunction (Group B) at the follow-up echocardiography, patients with LV asynergy at any point of examination (Group C) and patients free of LV abnormalities during the follow-up period (Group D).

Results: At the baseline examination, LV dysfunction (5/29 cases, 13.8%), LV asynergy (6/29 cases, 21.7%) were detected. Pericarditis was detected in 7 patients (24.1%), LVD in 3 patients, LV asynergy in 2 patients) and 2 of them with subacute onset had progressive LV dysfunction, while 5 patients were normal in echocardiography after remission induction therapy for SLE. At the follow-up examination, LV dysfunction (9/29 cases, 31.0%), 5 new-onset and 1 improved case), LV asynergy (6/29 cases, 21.7%, 2 new-onset and 2 improved cases) were detected. Though any significant differences were observed between Group A and Group B at the baseline, platelet count (156.0 vs 207.0, $p=0.049$) were significantly lower in LV dysfunction group (Group A) at the follow-up examination. Group C patients had significantly higher uric acid ($p=0.004$), monocyte count ($p=0.009$), and MHR ($p=0.003$) than Group D(results in table).

Conclusion: LV dysfunction is progressive in most of patients and requires regular follow-up once they developed. Uric acid, monocyte count and MHR are elevated in SLE patients with LV asynergy. Since MHR elevation was reported as useful marker of endothelial dysfunction⁵, our future goal is to analyse involvement of monocyte activation and endothelial dysfunction in LV asynergy of SLE patients.

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	Group C(LV asynergy +, n=8)	Group D(no abnormality, n=10)	P
Age, (y)	63.0(51.3-71.0)	49.0(45.8-61.8)	ns
SLE duration, (y)	22.9(13.4-33.9)	7.0(4.9-25.6)	ns
SLEDAI	1.0(0.0-2.0)	2.0(0.0-6.3)	ns
White blood cell count, ($\times 10^9/L$)	7.0(5.4-11.2)	6.1(4.7-7.9)	ns
Lymphocyte count, ($\times 10^9/L$)	1.2(0.88-2.1)	0.89(0.67-1.2)	ns
Monocyte count, ($\times 10^9/L$)	0.48(0.29-0.57)	0.24(0.17-0.29)	0.009
Hb, (g/dL)	11.7(10.5-14.2)	13.1(12.3-14.2)	ns
Platelet, ($\times 10^9/L$)	195.0(157.3-270.8)	225.0(183.3-284.8)	ns
Uric acid, (mg/dL)	7.2(5.8-8.1)	4.5(3.8-6.0)	0.004
IgG, (mg/dL)	1201(807.0-1347)	1260(1034-1786)	ns
Anti-DNA antibodies, (mg/dL)	0.0(0.0-7.3)	3.0(0.0-26.0)	ns
CH ₂ O, (mg/dL)	42.4(38.6-44.4)	39.7(28.9-46.9)	ns
Triglyceride, (mg/dL)	127.0(80.0-156.0)	109.0(75.5-155.0)	ns
HDL-Cholesterol, (mg/dL)	53.0(5.1-25-62.0)	54.5(42.8-82.7)	ns
LDL-Cholesterol, (mg/dL)	102.0(89.0-103.0)	118.5(82.8-134.3)	ns
MHR	0.0087(0.0063-0.012)	0.0039(0.0026-0.0051)	0.003

Numbers are median (interquartile range), Mann-Whitney u test were performed, p value less than 0.05 was considered statistically significant.

Disclosure of Interests: : None declared

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AB0423

NEUROPSYCHIATRIC OUTCOME OF CHILDREN BORN TO WOMEN WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) WOMEN AND EXPOSED IN UTERO TO AZATHIOPRINE: A CASE-CONTROL STUDY.

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Background: A possible increase in neurodevelopmental (ND) and learning disorders (LD) in the offspring of mothers affected by SLE have been suggested in some studies, along with the identification of different possible risk factors. Azathioprine (AZA) is commonly used during pregnancy, based on its non-teratogenicity and extended experience in women with different diseases. However, a few small studies suggested an association between in utero exposure to AZA and possible increased frequency of ND/LD in children, indirectly derived from increased request of supportive educational services.

Objectives: To evaluate the medium-long term outcome in terms of ND/LD in children of school age (≥ 6 years) born to SLE women treated with AZA during pregnancy, as compared to that of children born to SLE mothers not treated with AZA during pregnancy.

Methods: Data from our Pregnancy Clinic registry were collected for prospectively followed pregnancies of SLE women treated with AZA (cases) and compared to pregnancies of SLE women not treated with AZA (controls), that were matched for age at pregnancy, presence of renal involvement and aPL positivity. SLE patients (cases and controls) were interviewed by phone to collect data about their children, focusing on the presence of ND/LD certified by Neuropsychiatrists.

Results: Data were collected for 14 SLE mothers in the AZA group and 31 in the control group, with similar age at pregnancy (30.3 $\pm$ 5.21 vs 31.4 $\pm$ 4.70 years, $p=0.45$) and frequency of renal involvement (50.0% vs 44.1%, $p=0.77$), aPL positivity (33.3% vs 29.4%, $p=0.76$) and anti-Ro/SSA positivity (27.8% vs. 26.5%, $p=0.55$). A SLE flare during pregnancy was more frequently recorded in the AZA group (27.8% vs. 2.94%, $p=0.02$). Other medications included HCQ (55.6% vs. 70.6%, $p=0.36$) and corticosteroids (100% vs 79.4%, $p=0.08$).

We collected data for 18 children in the AZA group and 34 children in the control group, that had a similar mean age at the time of the interview (12.7 $\pm$ 4.80 vs. 12.9 $\pm$ 5.61 years, $p=0.91$). The two groups had also similar gestational age (37.4 $\pm$ 2.20 weeks vs. 38.0 $\pm$ 1.29 weeks, $p=0.23$), birth weight (3003 $\pm$ 433 g vs 3011 $\pm$ 453 g, $p=0.95$) and rate of male sex (61.1% vs 44.1%, $p=0.38$).

We recorded similar frequency of ND/LD in the two groups. In particular, a ND was present in 2/18 (11.1%) of children exposed to AZA vs. 2/34 (5.88%) in the control group ($p=0.60$). A LD was present in 1/18 cases (5.56%) and 6/34 controls (17.6%) ($p=0.40$).

Conclusion: The medium-long term outcome of children born to SLE mothers in the whole cohort was characterized by the presence of ND in 4/54 (7.69%) and LD in 7/52 (13.5%). ND/LD do not seem to be related to in utero exposure to AZA.

Disclosure of Interests: None declared

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AB0424

PREDICTORS OF SLE FLARE-UP AND PREMATURE DELIVERY IN PREGNANCY

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Background: Systemic lupus erythematosus (SLE) is a life-threatening autoimmune disease that affects many women of child-bearing age, with potentially severe consequences on pregnancy outcome. SLE flare-ups may occur during pregnancy and the post-partum period. Previous studies documenting the incidence and risk factors of flare-up of SLE during pregnancy and puerperium had partially discordant results.

Objectives: We aimed to delineate the pregnancy complications of women with SLE, as well as neonatal outcomes of their offspring, and hoped to clarify the incidence and risk factors of SLE flare-ups during pregnancy and puerperium.

Methods: We retrospectively reviewed the medical records of SLE patients with previous records of pregnancies in our institution. Flare events during pregnancy and puerperium were documented. The pregnancy outcomes recorded include live births, intra-uterine fetal death (IUFD), premature delivery (< 36 weeks of gestational age), NICU admission, and small for gestational age (SGA, <10th percentile). Univariate logistic regression was performed to determine the factors associated with disease relapse and pregnancy outcomes.

Results: From January, 2000 to December, 2019, a total of 94 SLE patients with 139 pregnancies were identified. The overall live birth rate was 92.4% (134/145). Forty-six (34.3%) of the neonates were delivered prematurely. Forty-six (34.3%) of them were SGA. The admission rate to the neonatal intensive care unit was 25% (30/120). Nine (6.4%) were diagnosed to have SLE during pregnancy. The flare rate during pregnancy was 20% while post-partum 9.4%. The majority of the relapses during pregnancy occurred in the second trimester (46.2%), followed by the first trimester (30.8%), and the third trimester (23.1%). Low complement C3 (C3 < 80mg/dl), thrombocytopenia (PLT < 100 $\times 10^3/uL$) at conception, and low serum albumin level at the first trimester were associated with antepartum flare. Presence of disease flare and pre-eclampsia in pregnancy, and low serum albumin level at conception were significantly associated with premature delivery.

Conclusion: Low complement C3 and thrombocytopenia at conception, and low serum albumin level at the first trimester were associated with disease flare-up during pregnancy. Patients with relative low serum albumin level at conception, or presence of eclampsia or disease flare-up during pregnancy had a higher risk of premature delivery.

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