of BMD at the left and right hip, and at the lumbar spine, as well as average percent fat and other risk factors for osteoporosis, including the FRAX risk factors. We used only the measures collected at baseline (time of first scan). We modelled the T scores of the BMD measurements using a linear regression model including percentage fat and BMI as explanatory variables, and adjusting for gender, age at scan, and other known risk factors for osteoporosis, including the FRAX risk factors. BMI and average percent fat were standardised.

Results: The number of patients included was 33037, (82% female). Results of both regression models are shown in table 1 below. We show the standardised effect size estimates for average percent fat and BMI.

Anatomical location	Effect size estimate for average percent fat (95% confidence intervals)	P value	Effect size estimate for BMI (95% confidence intervals)	P value
Left neck	-0.156 (-0.171, -0.141)	<0.001	-0.0255 (-0.0441, -0.00701)	0.00692
Left total	-0.225 (-0.241, -0.208)	<0.001	-0.0680 (-0.0882, -0.0477)	<0.001
Left Ward's	-0.181 (-0.196, -0.166)	<0.001	-0.0268 (-0.0456, -0.00813)	0.00493
Left trochanter	-0.263 (-0.281, -0.246)	<0.001	-0.0667 (-0.0882, -0.0451)	<0.001
Right neck	-0.139 (-0.154, -0.124)	<0.001	-0.0131 (-0.0317, 0.00549)	0.167
Right total	-0.221 (-0.237, -0.204)	<0.001	-0.0611 (-0.0811, -0.0411)	<0.001
Right Ward's	-0.180 (-0.196, -0.165)	<0.001	-0.0193 (-0.0381, -0.000586)	0.0433
Right trochanter	-0.261 (-0.278, -0.243)	<0.001	-0.0598 (-0.0810, -0.0386)	<0.001
Spine (averaged L1-L4)	0.219 (0.195, 0.242)	<0.001	-0.00846 (-0.0379, 0.0206)	0.563

Conclusion: The analysis shows that average percent fat is a statistically significant predictor for BMD at different anatomical locations, and a larger predictor in comparison to BMI when evaluated in the same model. In the right hip neck and the spine, BMI was not predictive of changes in BMD. Higher average percent fat increases the BMD in the spine, compared to a decline at the hip. Further research is needed to characterise the relationship more precisely and identify whether there is a causal link.

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SAT0486

BONE MINERAL DENSITY IN PATIENT WITH SHRUNKEN PORE SYNDROME IS SIGNIFICANTLY LOWER THAN THAT IN PATIENT WITHOUT, HOWEVER SERUM PARATHYROID HORMONE DOES NOT CORRELATED MUCH WITH IT

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Background: Shrunken pore syndrome (SPS), defined by cystatin C (CysC) based estimated glomerular filtration rate ($eGFR_{CysC}$) < 60% of creatinine (Cr) based $eGFR$ ($eGFR_{Cr}$) in the absence of extrarenal influences on the plasma levels of CysC or Cr, is associated with a higher increase in mortality. SPS often causes reduced bone mineral density (BMD).

Objectives: In this study, relationship between BMD and SPS was investigated.

Methods: Patient with rheumatic diseases who were measured BMD with dual-energy X-ray absorptiometry and at the same time, CysC and Cr were also measured, were picked up. $eGFR_{CysC}$ and $eGFR_{Cr}$ were calculated, and a patient group with SPS were recruited. Relationship between serum PTH and CysC, or Cr was evaluated with univariate linear regression analysis. Between the SPS groups and the other patient group, statistical difference was evaluated regarding sex, age, Cr, CysC, serum Cr/CysC ratio (Cr/CysC), serum calcium corrected with albumin (Ca), creatinine phosphokinase (CPK), parathyroid hormone (PTH), $eGFR_{CysC}$, $eGFR_{Cr}$, BMD in the lumbar spine (BMD_LS) and femoral neck (BMD_FN) were evaluated with Mann-Whitney U-test. Relationship between BMD for each bone and sex, age, CPK, PTH, Cr/CysC, $eGFR_{CysC}$ and being SPS was statistically evaluated with multivariate linear regression analysis. Further, sensitivity and specificity regarding being SPS for T-score < -2.5, that is defined as diagnosis criteria of osteoporosis calculated from BMD, was evaluated with chi square test.

Results: A total of 819 participants with 75 males and 744 females joined. Patient with SPS counted 31 and without SPS counted 782. Underlying diseases are shown in Table 1. Average age, CysC, Cr, PTH, $eGFR_{Cr}$, and $eGFR_{CysC}$ were 76.5, 1.18, 0.76, 42.1, 66.2 and 59.0, respectively. PTH significantly correlated with CyC ($p=0.015$), but not correlated with Cr ($p=0.079$). SPS demonstrated significantly higher ratio for being male ($P<1.0\times10^{-10}$), higher age ($p=1.07\times10^{-3}$), higher titer of CysC ($p=5.5\times10^{-10}$), lower titer of CPK ($p=1.5\times10^{-4}$), lower Cr/CysC ($P<1.0\times10^{-10}$), lower $eGFR_{CysC}$ ($p=2.8\times10^{-7}$), BMD_LS ($P<1.0\times10^{-10}$) and BMD_FN ($P<1.0\times10^{-10}$), however no significant difference demonstrated for Cr (2.4×10^{-1}), PTH ($p=1.7\times10^{-1}$) and Ca ($p=6.3\times10^{-1}$). BMD_LS demonstrated significant positive correlation with CPK ($p=2.6\times10^{-4}$), and negative correlation with being female ($p=4.9\times10^{-7}$), age ($p=2.1\times10^{-3}$), PTH ($p=2.2\times10^{-2}$), $eGFR_{CysC}$ ($p=2.5\times10^{-9}$) and being SPS ($p=4.9\times10^{-3}$), while BMD-FN demonstrated significant positive correlation with Cr/CysC ($p=7.3\times10^{-4}$), and negative correlation with being female ($p=1.5\times10^{-6}$), age ($p=4.9\times10^{-8}$) and being SPS (7.3×10^{-3}). Sensitivity and specificity of T-score < -2.5 in the LS regarding SPS was 50.0% and 74.0% ($p=6.9\times10^{-3}$), while in the FN 67.9% and 61.7% ($p=1.7\times10^{-3}$), respectively.

Table 1. Number of patients who were picked up in the study
Abbreviations: RA, rheumatoid arthritis; PsA, psoriatic arthritis; AJA, Sjoren Syndrome; SLE, systemic lupus erythematosus; PPP, pustulosis palmaris et plantaris; AS, ankylosing spondylitis; SSc, systemic sclerosis; UA, ulcerative colitis; PM/DM, polymyositis/dermatomyositis; MCTD, mixed connective tissue disease; FMF, Familial Medditeranean fever; PAN, polyarteritis nodosa.

Diseases	Number of patient
RA	512
PsA	110
SJS	67
SLE	66
PPP	17
AS	16
SSc	13
UA	11
Behcet	8
PM/DM	3
MCTD	2
FMF	2
PAN	1
total	828

Conclusion: These results suggested that SPS has serious potential risk of osteoporosis. BMD_LS loss may correlate elevation of PTH due to filtration disorder, however BMD_FN loss is not affected. Split of Cr and CysC is more important.

Disclosure of Interests: None declared

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Basic and translational science in paediatric rheumatology

SAT0487

PREDICTIVE BIOMARKERS OF IGA VASCULITIS WITH NEPHRITIS BY METABOLOMIC ANALYSIS

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Background: IgA vasculitis/ Henoch Schönlein Purpura (IgAV/HSP) is the most common vasculitis of childhood and renal involvement is the most serious long-term complication. A better understanding of the pathophysiology of the progression to kidney disease is required for better treatment to be achieved and current biomarkers of Ig A vasculitis with nephritis (IgAVN) lack the predictive value.

Objectives: In this study, an untargeted metabolomics approach was performed to reveal the underlying molecular mechanism of disease pathogenesis and to find potential biomarkers of plasma samples from patients with IgAV and IgAVN.

Methods: IgAV/HSP was diagnosed according to the Ankara criteria in 2008 (1). Forty-five patients, including 39 active IgAV patients (H), 6 IgAVN (N), and 6 age- and

gender-matched healthy controls (C), were enrolled in the study. Plasma samples from subjects were collected on the same day of IgAV(HSP) diagnosis and before steroid or other immunosuppressive treatment initiated. This study has utilized liquid chromatography-mass spectrometry (LC-MS/ Q-TOF) to investigate the alterations in plasma metabolomic profiles. Three separate pools, health controls, active IgAV, and IgAVN were created. Peak picking, grouping, and comparison parts were performed (metabolite profiling) via XCMS (<https://xcmsonline.scripps.edu/>) software.

Results: Totally 2618 peaks were detected for group H, N and C. Among them 355 peaks were found to be statistically significant and reliable ($p<0.05$) and 155 of these peaks were found to be changed (fold change >1.5) between the groups C and H. On the other hand, 66 peaks were found to be changed (fold change >1.5) between the groups H and N. The number of the peaks on the intersection of the peaks found to be changed between the groups (C and H) and (H and N) was 39. This situation was illustrated in Figure 1. Based on putative identification results, 15 peaks were matched with 24 metabolites. The list of these metabolites is given in Table 1.

Table 1. Putative identification of 15 peaks found to be statistically different and having fold changes.

Peak	Putative Identification	KEGG Codes	rt (min)	N/H
1	5-Aminopentanamide	C00990	2,10	0,36
1	5-Aminopentanoic acid	C00431	2,10	0,36
1	2-Keto-6-aminocaproate	C03239	2,10	0,36
1	(S)-5-Amino-3-oxohexanoate	C03656	2,10	0,36
1	D-1-Piperidine-2-carboxylic acid	C04092	2,10	0,36
2	Oxalurate	C00802	15,52	0,50
3	Porphobilinogen	C00931	14,78	9,62
4	(+)-cis-3,4-Dihydrophenanthrene-3,4-diol	C04468	15,52	0,02
5	(-)-trans-Carveol	C00964	12,69	1,82
6	DHAP(18:0)	C03805	12,70	1,92
6	N-Acetyl-b-glucosaminylamine	C01239	12,70	1,92
7	5-Methyltetrahydrofolic acid	C00440	14,88	2,41
8	N2-Succinyl-L-ornithine	C03415	15,52	0,05
8	N6-Acetyl-L-2,6-diaminoheptanedioate	C04390	15,52	0,05
9	Estrone	C00468	12,71	1,90
10	N-Acetyl-4-O-acetylneuraminic acid	C04015	14,89	2,41
10	N-Acetyl-7-O-acetylneuraminic acid	C04016	14,89	2,41
11	Oleoyl-CoA	C00510	15,52	0,26
12	Saccharopine	C00449	15,52	0,26
13	Prostaglandin D2	C00696	17,81	2,05
13	Prostaglandin I2	C01312	17,81	2,05
14	Glycocholic acid	C01921	13,78	0,61
15	Galactosylsphingosine	C01747	24,41	0,45
15	Glucosylsphingosine	C03108	24,41	0,45

Conclusion: Certain differences in metabolites were identified between controls and IgAV patients and between those with and without kidney involvement.

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Paediatric rheumatology

SAT0488

ETANERCEPT CONCENTRATION IN PATIENTS WITH JUVENILE IDIOPATHIC ARTHRITIS

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Background: Etanercept (ETN) is the most used TNF blocker in children with JIA. There is still limited real-life data of etanercept concentrations in children, especially in association with dosing.

Objectives: The aim of the study was to investigate association between ETN dosing and serum trough concentration in children with non-systemic JIA.

Methods: We conducted a multicenter retrospective study of 180 Finnish JIA patients (Table 1) receiving ETN either as monotherapy or in combination with one or more DMARDs (Table 2). Prior biologicals were used by 17 % of the patients. Patients were divided into two groups, ETN started before or after one year of diagnosis (Figure 1). ETN concentration samples (collected 2014-2017) were analyzed using validated enzyme-linked immunosorbent assay (ELISA) in Sanquin Diagnostics, Amsterdam, the Netherlands.

Results: Demographics at etanercept start and diagnoses are shown in Table 1. Duration of the treatment with ETN, ETN doses, concentrations and concomitant medications at the time of concentration measurement are shown in Table 2.

Association between ETN dose and concentration is shown in Figure 1.

Those who started medication early (< 1 year from diagnosis) were younger than those who started later (Table 1) and association between ETN dose and concentration was more obvious (Figure 1).

Table 1	N=68, Disease duration <1 year	N=112, Disease duration ≥ 1 year	P-value
Girls, n (%)	39 (57)	70 (62)	0.49
Age, mean (SD), years	6.6 (3.8)	8.9 (3.5)	<0.001
BSA, mean (SD)	0.86 (0.32)	1.03 (0.30)	<0.001
Disease duration, mean (SD), months	0.7 (0.2)	3.9 (2.7)	..
Dg n (%)			0.078
Oligoarthritis, persistent	12 (18)	23 (21)	
Oligoarthritis, extended	7 (10)	22 (20)	
Polyarthritis, RF-neg	42 (62)	55 (49)	
Polyarthritis, RF-pos	3 (4)	0 (0)	
Enthesitis-related arthritis	3 (4)	8 (7)	
Psoriatic arthritis	0 (0)	3 (3)	
Undifferentiated arthritis	1 (1)	1 (1)	
HLA-B27 pos, n (%)	19 (29)	33 (30)	0.86
ANA pos, n (%)	21 (31)	33 (29)	0.84

Table 2	Disease duration at ETN start <1 year	Disease duration at ETN start ≥ 1 year	P-value
BSA, mean (SD)	0.98 (0.33)	1.18 (0.31)	<0.001
ETN duration, mean (IQR), months	11.7 (4.3-29.2)	10.9 (4.7-32.9)	0.68
ETN dose (mg/BSA/week), mean (SD)	23.7 (6.4)	23.9 (5.0)	0.90
ETN concentration (ug/ml), mean (SD)	1.91 (1.00)	1.74 (0.85)	0.24
Concomitant treatments, n (%)			
MTX	43 (63)	54 (48)	0.050
LEF	10 (15)	14 (13)	0.67
HCQ	5 (7)	3 (3)	0.16
SSZ	4 (6)	6 (5)	0.88
AZA	0 (0)	1 (1)	0.99
Glucocorticoids	9 (13)	2 (2)	0.003