also increased from 28.7 to 38.2 per 100 000 adults ($p<0.001$). The mean duration of hospitalisation was 12.5 ± 2.7 days (13 days; 2–26 days) and increased from 11.6 to 12.7 ($p=0.03$).

Abstract FRI0253 – Table 1

	2011	2012	2013	2014	2015
Population size in Minsk, adults	1537880	1555210	1568320	1585480	1598120
Incidence per 100 000	25.0	26.2	35.3	38.0	35.5
Prevalence per 100 000	179	228	248	255	290
Hospitalisation rate per 100 000	28.7	36.8	33.5	33.9	38.2
Hospitalisation duration, days	11.6	12.5	13.1	12.7	12.7

Conclusions: We revealed significant increase in incidence, prevalence as well as hospitalisation rates for gout in adults in Minsk (the Republic of Belarus) for the 5 year period (2011–2015) in 1.4, 1.6 and 1.3 times, correspondingly.

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FRI0254 CLINICAL FEATURES AND RISK FACTORS FOR DEVELOPING GOUT ATTACKS DURING ANTI-TUBERCULOSIS MEDICATION: A CASE-CONTROL STUDY IN SOUTH KOREA

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Background: Gout is a form of inflammatory arthritis that develops in some people with hyperuricemia. Several drugs may induce hyperuricemia and gout attack. Some anti-tuberculosis (TB) medications are also known to increase uric acid level and trigger acute gout attack. However, there have been no well-described studies for gout during anti-TB therapy.

Objectives: To investigate the clinical features and risk factors of gout attacks following anti-TB treatment in South Korea.

Methods: We conducted the case-control study including 49 patients who developed gout attack following anti-TB medications and 147 age- and sex- matched TB controls without gout attack. Demographic, clinical features and laboratory findings of patients with gout attacks following anti-TB medication were analysed and compared to those of controls in order to establish risk factors associated with gout attack during anti-TB treatment.

Results: The mean age was 67.7 ± 13.2 years and 39 patients (79.6%) were male. Twenty-four patients (49.0%) had a previous history of gout. The mean time interval to develop gout attack from the initiation of anti-TB therapy was 4.13 ± 4.49 months. The attacks tended to involve lower extremity joints (43/49, 87.8%), especially the ankle and the first metatarsophalangeal joint. Factors associated with experiencing gout attack during anti-TB medication were higher body mass index (BMI, 23.7 ± 3.6 vs 21.8 ± 3.6 , $p=0.003$), higher pre-treatment uric acid level (7.2 ± 2.5 vs 6.2 ± 1.7 mg/dL, $p<0.001$), previous history of gout (49.0% vs 1.4%, $p<0.001$), absence of dyslipidemia (4.1% vs 16.3%, $p=0.008$), and decreased renal function (eGFR 63.6 ± 27.5 vs 82.6 ± 29.9 , $p=0.003$). In multivariate model, higher BMI, chronic kidney disease (CKD, eGFR <60 ml/min/1.73m²), previous history of gout, and pre-treatment hyperuricemia (uric acid ≥ 6.8 mg/dL) were

found to be independent risk factors for gout attack following anti-TB medication (table 1).

Abstract FRI0254 – Table 1. Multivariate analysis of risk factors for post-TB medication gout

	Odds ratio	95% CI	p-value
Body mass index (BMI)	1.202	1.029–1.403	0.020
Previous history of gout	71.39	12.790–398.467	<0.001
Chronic kidney disease (eGFR<60 ml/min/1.73m ²)	2.794	1.012–7.719	0.047
Pre-treatment hyperuricemia (Serum uric acid ≥ 6.8 mg/dL)	4.866	1.770–13.378	0.002

Conclusions: TB patients with obesity, history of gout, pre-treatment hyperuricemia, and CKD have higher risk for having gout attack following anti-TB medication. When starting anti-TB therapy in TB patients with these risk factors, physicians should pay attention to the development of gout attack and educate the patients.

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SLE, Sjögren's and APS – etiology.3, pathogenesis and animal models

FRI0255 INJECTION OF CD40 DNA VACCINE AMELIORATES THE AUTOIMMUNE PATHOLOGY OF NON-OBESE DIABETIC MICE WITH SJOGREN'S SYNDROME

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Background: In the patients with primary Sjögren's syndrome (pSS), overexpression of CD40 was reported. The increased CD40 expression contributes to enhance CD40-CD40L interaction and promotes the inflammatory response in various ways.

Objectives: To investigate whether CD40 DNA vaccine could inhibit the immune response and slow the disease progression of SS in non-obese diabetic (NOD) mice.

Methods: Female 8-week-old NOD mice were randomly divided into 3 groups. CD40 DNA vaccine group received pTaget CD40 DNA vaccine at a weekly dose of 50 ug for 4 weeks. Vector and NS group were administered an equivalent amount of empty vector or NS. Serum anti-CD40 antibody was measured by ELISA. Lymphocytes infiltration in the salivary glands was monitored by focus score (FS) calculation. Salivary CD40 was stained by immunohistochemistry. Splenic lymphocyte phenotypes were analysed by flow cytometry. CD40, TNF- α and IL-6 levels in the salivary glands were detected by PCR. Serum ANA was monitored by immunofluorescence.

Results: 1) CD40 DNA vaccination induced anti-CD40 antibody response. We found the positive rate on anti-CD40 antibody in vaccine group was 80% at week 6% and 100% at week 10, which was significantly increased compared with that in nonvaccinated ones ($p<0.05$ and $p<0.01$, respectively)

2) Expression of CD40 decreased in salivary glands in vaccine group