Table 2. Descriptive data of MNA and NRS-2002 in rheumatic diseases.

	NUTRITIONAL SCREENING TOOLS				
	MNA	NRS-2002	NRS-2002*	Rho	p
All Rheumatic Diseases					
Total Score, n(SD)	24.31 (3.48)			.23	.004
Malnutrition, n(%)	4 (2.6)	5 (3.3)	5 (3.3)		
Risk, n(%)	55 (36.2)	147 (96.7)	41 (27)		
Normal, n(%)	93 (61.2)		106 (69.7)		
Rheumatoid Arthritis					
Total Score, n(SD)	24.43 (3.23)			.43	.05
Malnutrition, n(%)	1 (1.4)	2 (2.9)	2 (2.9)		
Risk, n(%)	22 (31.4)	68 (97.1)	15 (21.4)		
Normal, n(%)	47 (67.1)		53 (75.7)		

Conclusion: MNA was a more sensible tool for detecting risk of malnutrition when compared to NRS-2002. Screening tools play an important role at nutritional evaluation and should be complemented with objective methods such as BIA.¹ Rheumatologists must be aware that nutritional disorders affect the state of rheumatic diseases; and selecting an appropriate tool to detect malnutrition is of vital importance.

References:

- [1] Elkan, A. C., Engvall, I. L., Tengstrand, B., Cederholm, T., & Hafström, I. (2008). Malnutrition in women with rheumatoid arthritis is not revealed by clinical anthropometrical measurements or nutritional evaluation tools. *European journal of clinical nutrition*, 62(10), 1239-1247.

Figure 1. Graphic 1. Nutritional screening tools in rheumatic disorders. Total N= 152 patients. MNA= Mini Nutritional Assessment NRS-2002= Nutritional Risk Screening Tool-2002. NRS-2002*= Nutritional Risk Screening Modified with 3 parameters.

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AB1301-HPR COMORBIDITIES AND FACTORS INFLUENCING RECURRENT GOUT ATTACK IN PATIENTS WITH GOUT: A CROSS-SECTIONAL STUDY

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Background: Gout attack is characterized by painful arthritis, loss of function and reduced quality of life. Frequent gout attacks can exert negative an influence on gout management [1].

Objectives: The objective was to identify the comorbidities of gout, to compare gender difference and to identify independent factors of multiple gout attacks.

Methods: A cross-sectional study was performed to collect demographic, clinical variables, self-reported comorbidities, and relevant testing. Group comparison and correlation of serum uric acid (sUA) levels with other variables was performed. Univariate and multivariate logistic regression was used to detect independent risk factors of sUA.

Results: 653 gout patients were enrolled, including 553 (84.7%) males. The mean age was 48.3±15.8 years old, with a disease duration of 8.0±6.4 years. 170 (26.0%) patients had hypertension, and 57 (8.7%) had hyperlipidemia. Elevated total cholesterol (TC) was observed in 173 (26.5%) cases. 42 (37.1%) cases presented with increased triglycerides (TG) and 270 (41.3%) had increased low-density lipoprotein (LDL-C). Abnormalities including nephrolithiasis (29.4%), hydronephrosis (3.2%), and gallstones (11.9%) were detected in the patients who underwent ultrasound examination. Although female patients had a longer disease duration, they had lower levels of sUA, creatine and C-reactive protein (CRP). A positive correlation with sUA was found in TG and CRP ($P<0.05$) in

female patients, which was not observed in males. Only gout duration (OR=1.406, $P<0.001$), sUA (OR=1.006, $P<0.001$) and LDL-C (OR=0.530, $P=0.006$) were independent factors of gout attack (>20 times).

Conclusion: Comorbidity screening involving dyslipidemia is often neglected in gout patients. Gout duration and sUA level are risk factors of multiple gout attacks.

References:

- [1] Wang Y, Yan S, Li C et al. Risk Factors for Gout Developed From Hyperuricemia in China: A Five-Year Prospective Cohort Study. *Rheumatol Int*. 2013; 3: 705-10.

Table 1 Comparisons of clinical and laboratory variables between male and female patients with gout

Variable	Male (n=553)	Female (n=100)	p
Age (years)	47.7±15.9	51.3±14.5	0.040*
BMI (kg/ m2)	24.5 (22.0-27.0)	23.3 (20.3-25.3)	0.001*
Gout duration (years)	6.0 (3.0-11.0)	9.5 (4.0-15.0)	0.001*
Gout attack times			
•≤5	209 (37.5)	33 (33.0)	
•6-10	97 (17.5)	22 (22.0)	
•11-20	70 (12.7)	22 (23.0)	
•>20	177 (32.0)	23 (23.0)	
Comorbidities			
Hypertension, n (%)	138 (25.0)	32 (32.0)	0.140
Coronary heart disease, n (%)	57 (10.3)	10 (10.0)	0.926
Fatty liver disease	53 (9.6)	4 (4.0)	0.069
Laboratory testing			
•ALT (U/L)	25.0 (17.0-40.0)	19.2 (14.6-29.0)	0.003*
•ALB (g/L)	43.5±6.3	42.4±3.9	0.025*
•TB (μmol/L)	12.7 (9.0-17.3)	14.5 (12.1-17.4)	0.002*
•BUN (mmol/L)	4.85 (3.98-6.27)	4.85 (3.91-4.82)	0.588
•Creatine (mmol/L)	94.0 (81.4-108.1)	73.8 (67.4-87.2)	<0.001*
•UA (μmol/L)	528.4±141.1	363.8±122.9	<0.001*
•Glucose (mmol/L)	5.62±1.67	5.43±1.17	0.317
•TC (mmol/L)	5.05±1.15	5.09±1.10	0.726
•TG (mmol/L)	2.11±1.66	1.94±1.86	0.335
•HDL-C (mmol/L)	1.14±0.30	1.52±0.41	<0.001*
•LDL-C (mmol/L)	3.25±0.97	3.39±0.92	0.182
•ESR (mm/h)	40.1±32.4	37.4±20.8	0.402
•CRP (mg/L)	28.0±40.3	10.5±18.6	<0.001*

* $p < 0.05$; BMI: body mass index; sUA: serum uric acid; ALT: alanine aminotransferase; ALB: albumin; TB: Total bilirubin; BUN: blood urea nitrogen; UA: uric acid; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein; LDL-C: Low-density lipoprotein; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein

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AB1302-HPR ASSOCIATIONS OF SLEEP ON GOUT PREVALENCE AND SERUM URIC ACID CONCENTRATIONS

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Background: Sleep deprivation and sleep inconsistency have been associated with modulation of the immune and inflammatory response systems with effects that include higher risk for psychosis, cardiovascular disease, and increased susceptibility to infection. One hypothesized mechanism to explain these findings is an association with pro-inflammatory states.

Objectives: In a first-of-its kind study, we examined associations between sleep quantity and gout using the largest public US dataset available; the National Health and Nutrition Examination Survey (NHANES).

Methods: NHANES data from 2009 to 2014 was selected based on questionnaire responses and laboratory studies. The resulting data set was analyzed for serum uric acid (SUA) levels (cut-off >6.0 mg/dL in females and >7.0 mg/dL in males), and gout risk factors. Analysis of variance, means, proportions, as well as univariate and multiple logistic regression models were used to evaluate the associations between sleep quantity, SUA and gout.

Results: 10,410 survey participants from 2003 to 2014 were included in the study. A total of 280 (2.7%) had gout. Gout prevalence ranged between 4.0% and 4.7% between high, normal, and low sleep groups. Multivariate analyses showed a significant association between extremely low sleep levels (OR = 0.53 and CI = 0.34 to 0.83), and gout, which was also seen with SUA level association (OR = 0.41 and CI = 0.32 to 0.52). However, in the broader sense, lower than normal (<7 hours) and higher than normal (>9 hours) levels of sleep had no significant associations with either gout prevalence or SUA, likely due to a non-linear relationship of SUA to sleep levels.

Conclusion: Despite numerous adverse effects found with low sleep levels, our study does not show a strong association between sleep quantity and gout prevalence or SUA levels.

References: