bladder, 2,590 for lung, 4,488 for bowel, 6,474 for prostate and 10,274 for breast cancer). The definitions for cancer endpoints were matched for ICD9 and 10 codes between MDC/MPP and UK-Biobank cohorts. For both MR settings, a set of 26 urate-associated single nucleotide variants was selected to build-up the SU instrument (*SU-instr*) to test for a causal effect of SU on cancer outcomes. Statistical analysis, adjusted for age and sex, was done using multiple conventional MR methodologies and MR package in R (v4.0.2). A $p < 0.05$ was designated as statistically significant.

Results: We found no causal effect of our *SU-instr* on neither all-cause nor site-specific cancer across all MR analyses (all $p > 0.05$). In MDC/MPP, *SU-instr* did not show a causal effect on the risk of all-cause [OR = 1.06, $p = 0.32$], bladder [OR = 0.96, $p = 0.84$], lung [OR = 1.26, $p = 0.17$], bowel [OR = 0.96, $p = 0.81$], prostate [OR = 1.05, $p = 0.62$], and breast [OR = 0.99, $p = 0.98$] cancer. Similar findings were made in the two-sample settings. Detailed results are provided in Table 1.

Conclusion: Our MR study, using a series of causal inference approaches, does not support a causal effect of genetically determined SU for major cancer outcomes. There is no evidence to support changing SU levels by lifestyle or pharmacological intervention to attenuate the risk of major cancer types.

Table 1. Results from a range of MR analyses for causal effect of SU on cancer risk in MDC/MPP (one-sample MR) and GUGC and UK-Biobank (two-sample MR) cohorts

One-sample MR							
Cancer type	IVW			2SLS		GRS	
	OR (95% CI)	p -causal	p -Het	OR (95% CI)	p -causal	OR (95% CI)	p -causal
Bladder	0.96 (0.68; 1.36)	0.84	0.55	0.99 (0.70; 1.42)	0.99	0.94 (0.67; 1.34)	0.76
Lung	1.26 (0.90; 1.77)	0.17	0.89	1.29 (0.91; 1.82)	0.14	1.26 (0.90; 1.77)	0.17
Bowel	0.96 (0.72; 1.28)	0.81	0.44	0.95 (0.72; 1.27)	0.77	0.97 (0.73; 1.28)	0.83
Prostate	1.05 (0.84; 1.31)	0.62	0.84	1.04 (0.83; 1.30)	0.72	1.04 (0.83; 1.30)	0.69
Breast	0.99 (0.74; 1.33)	0.98	0.11	0.98 (0.73; 1.32)	0.92	0.98 (0.73; 1.32)	0.94
All-cause	1.06 (0.93; 1.21)	0.32	0.6	1.07 (0.94; 1.21)	0.28	1.07 (0.94; 1.21)	0.29
Two-sample MR							
Cancer type	IVW			Weighted median		MR-Egger	
	OR (95% CI)	p -causal	p -Het	OR (95% CI)	p -causal	OR (95% CI)	p -causal
Bladder	1.02 (0.88; 2.16)	0.77	0.36	1.03 (0.86; 2.10)	0.7	1.03 (0.83; 2.13)	0.75
Lung	0.86 (0.73; 1.08)	0.07	0.04	0.86 (0.73; 1.09)	0.09	0.84 (0.66; 1.16)	0.15
Bowel	0.82 (0.69; 1.02)	0.03	< 0.0001	0.89 (0.78; 1.06)	0.06	0.92 (0.72; 1.67)	0.52
Prostate	1.00 (0.91; 2.64)	0.97	0.72	0.98 (0.88; 2.17)	0.77	0.94 (0.83; 1.45)	0.37
Breast	0.97 (0.88; 1.95)	0.67	0.0002	1.00 (0.91; 2.70)	0.99	0.95 (0.81; 1.59)	0.46
All-cause	0.96 (0.90; 1.20)	0.18	< 0.0001	0.97 (0.92; 1.23)	0.21	0.93 (0.86; 1.12)	0.11

OR; odds ratio, 95% CI; 95% confidence interval, IVW; Inverse variance weighted method, 2SLS; two-stage least square, GRS; genetic risk score, p -Het; p -value for heterogeneity.

Disclosure of Interests: None declared

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Public Health, Health Services and Health Economics

POS0296 DOSING OF BDMARDS IN AXSPA AND PSA IN A REAL WORLD SETTING

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Background: The treatment of patients with axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA) has been revolutionised by the introduction of biologic DMARDs targeting TNF, IL17, and IL23 inhibitors (i). In Germany, about 30-50% of axSpA and PsA patients receive treatment with bDMARDs. Although many patients benefit from these drugs, in some patients effectiveness of the standard dose may be insufficient and higher doses are used.

Objectives: To describe dosing of TNFi and non-TNFi bDMARDs over a 2 year period in a real world cohort of patients with axSpA and PsA managed by rheumatologists.

Methods: RABBIT-SpA is a prospective longitudinal cohort study including axSpA and PsA patients enrolled at the start of a new conventional treatment (including NSAID) or b/tsDMARD treatment. Description of dosing of TNFi (adalimumab bio-original (bo), adalimumab bio-similar (bs), etanercept bo, etanercept bs, golimumab, certolizumab) in comparison to nonTNFi-bDMARDs (secukinumab, ustekinumab, ixekizumab, guselkumab) in axSpA and PsA. Standard dosing was defined according to the current labels for axSpA and PsA.

Results: 1628 patients (axSpA: n=903, PsA: n=725) were included in this analysis. At inclusion mean age was 44 years in axSpA and 51 years in PsA. 44% of patients with axSpA and 58% of those with PsA were female. The mean disease duration of axSpA was 7.6 years, of PsA 6.4 years.

Standard doses of TNFi were used during a 2 year period in > 90% of patients with axSpA and PsA (Figure 1). In contrast, standard doses of non-TNFi-bDMARDs were only used in 70-80% of patients. The percentage of documented higher doses in patients with axSpA ranged from 20-30% at different time points. In PsA, this percentage increased from 27% at baseline to 44% at 2 years. On the other hand, TNFi were used in lower doses than the label in up to 9% and 7% of patients with axSpA and PsA, respectively, after 2 years.

Figure 1. Percentages of patients with axSpA or PsA who received less than, equal to, or more than the approved doses of bDMARDs at baseline and at 5 follow-up visits.

Conclusion: While TNFi are used in licensed doses in most patients, non-TNFi-bDMARDs were often used in higher doses, which corresponds to higher doses approved in other indications like psoriasis. The effectiveness of this treatment strategy in axSpA and PsA needs to be analysed further.

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POS0297 HOSPITAL ADMISSIONS FOR PATIENTS WITH RHEUMATOID ARTHRITIS IN WESTERN AUSTRALIA HOSPITALS HAVE DECLINED OVER TIME

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Background: Rheumatoid arthritis (RA) represents a substantial burden on patients and society in terms of morbidity, enduring disability, and medical expenses (1). RA prevalence is poorly described in Australia, and linked health datasets can provide a more meaningful picture for RA epidemiology in the Australian population.

Objectives: To describe the period prevalence rate of RA patients per 1000 hospital separations coded as RA primary or secondary diagnosis in Western Australia (WA) hospitals between 1995 and 2014.