Conclusion: We demonstrated that the prevalence of CVD is approximately 1.5 times higher in patients with rheumatic diseases compared to healthy controls (11% vs. 7%, respectively). This corresponds with previous research, although the reported prevalence of CVD in PsA and AS patients is even higher compared to prior studies. This suggests that the CVD risk of patients with rheumatic diseases is still elevated in 2020 compared to the general population, despite the improved management of rheumatic disease activity. Therefore, adequate and timely treatment of CV risk factors remains relevant, not only in patients with RA, but in patients other rheumatic diseases as well.

REFERENCES:

- [1] Hooijberg F et al. (2020) Patients with rheumatic diseases adhere to COVID-19 isolation measures more strictly than the general population. *The Lancet rheumatology* 2(10), 583-585.

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POS0525

ARTHRITIC PAIN AS A SURROGATE MARKER FOR ASYMPTOMATIC CARDIOVASCULAR RISK FACTORS: OFFERING PRACTITIONERS A 'TEACHABLE MOMENT'

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Background: Cardiovascular diseases (CVD) are the number one cause of death worldwide. CVDs are linked to well established risk factors: obesity, hypertension (HTN), dyslipidaemia (DL) and diabetes mellitus (DM)¹. While targeting risk factors reduces the burden of CVD, this is often challenging because they are largely asymptomatic and patients are therefore unlikely to seek medical attention. Arthritis, in contrast, causes pain and functional impairment prompting presentation to a healthcare practitioner. Patients with arthritis of varying aetiologies (such as osteoarthritis², gout³, rheumatoid arthritis⁴) have been shown to have an increased risk of CVD.

Objectives: To examine the relationship between arthritis and DM, HTN and DL in adults of all age groups. A secondary objective was to examine whether this relationship existed independent of obesity.

Methods: Data from the 2017-18 Australian Bureau of Statistics National Health Survey included 13,776 participants, categorised into young (18-39 years), middle aged (40-64 years) and older (≥65 years) adults. Blood pressure, height and weight were measured. BMI was calculated and participants classified as obese (≥30kg/m²) or non-obese. HTN was defined as > 140/90mmHg. Participants were asked if they had arthritis of any form, DL or DM diagnosed by a doctor. Logistic regression models estimated odds ratios with 95% CI for prevalence of arthritis associated with CVD risk factors.

Results: Arthritis was reported by 3.9% of young adults, 28.8% of middle-aged adults, and 54.5% of older adults. In all three age groups, arthritis was associated with significantly increased odds of obesity, HTN, DL and DM. For example, in middle-aged adults, having arthritis was associated with increased odds of obesity (1.75, 95% CI 1.54-2.01), HTN (1.78, 1.60-2.04), DL (2.14, 1.84-2.49) and DM (1.64, 1.33-2.03). These associations remained statistically significant after adjustment for obesity.

Conclusion: Compared to those without arthritis, adults with arthritis were at increased risk of obesity, HTN, DM and DL. The increased risk of HTN, DM and DL was independent of obesity and tended to be higher in younger adults. These data suggest that a patient's presentation with symptomatic arthritis of any aetiology and at any age, may be used opportunistically as a "teachable moment" for screening for asymptomatic CVD risk factors in higher-risk individuals. This provides practitioners an opportunity to manage both arthritis and CVD risk in parallel, rather than in silos.

REFERENCES:

- [1] World Health Organisation. (2017). "Cardiovascular Diseases." from [https://www.who.int/en/news-room/fact-sheets/detail/cardiovascular-diseases\(cvds\)](https://www.who.int/en/news-room/fact-sheets/detail/cardiovascular-diseases(cvds)).
- [2] Wang, H., et al. (2016). "Osteoarthritis and the risk of cardiovascular disease: a meta-analysis of observational studies." *Scientific reports* 6: 39672.
- [3] Singh, J. (2015). "When gout goes to the heart: does gout equal a cardiovascular disease risk factor?" *Annals of the Rheumatic Diseases* 74: 631-634.
- [4] England, B. R., et al. (2018). "Increased cardiovascular risk in rheumatoid arthritis: mechanisms and implications." *British Medical Journal* 361: k1036.

Table 1. Prevalence of CVD risk factors in adults with and without arthritis.

	Young 18-39 yo			Middle Aged 40-64 yo			Older ≥65 yo		
	No arthritis n = 3773	Arthritis n = 157	OR (95% CI) *adjusted for obesity	No arthritis n = 4055	Arthritis n = 1638	OR (95% CI) *adjusted for obesity	No arthritis n = 1891	Arthritis n = 2262	OR (95% CI) *adjusted for obesity
Obesity	473	32	2.07 (1.36-3.16)	868	527	1.75 (1.54-2.01)	326	610	1.89 (1.62-2.21)
HTN	131	14	2.72 (1.53-4.84)	745	496	1.78 (1.60-2.04)	730	1091	1.48 (1.31-1.68)
			2.35 (1.17-4.70)*			1.59 (1.37-1.84)			1.35 (1.18-1.55)*
DM	19	4	5.7 (1.74-15.37)	236	151	1.64 (1.33-2.03)	256	399	1.37 (1.15-1.62)
			4.87 (1.34-17.69)*			1.37 (1.08-1.73)*			1.15 (0.95-1.39)*

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POS0526

SEXUAL FUNCTION IN MALE AND FEMALE PATIENTS WITH RHEUMATOID ARTHRITIS: A POST-HOC ANALYSIS OF THE FINCH STUDIES

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Background: While sexual function is impaired in a high proportion of patients with rheumatoid arthritis (RA), it is often neglected in patient care.¹ FINCH 1 (NCT02889796), FINCH 2 (NCT02873936) and FINCH 3 (NCT02886728) were Phase 3 studies to assess the safety and efficacy of filgotinib (FIL) for moderate-to-severe RA; patient-reported sexual function was also evaluated.

Objectives: To analyse disease characteristics associated with sexual function and explore the effect of FIL and adalimumab (ADA) on sexual function in males and females in the FINCH studies.

Methods: Post-hoc analyses included data from patients who were randomised and received ≥1 dose of study drug in the FINCH studies. Male and female subgroup analyses were performed to describe the correlation between baseline disease characteristics and baseline visual analogue scale (VAS) sexual function score (using Pearson correlation coefficient) and to assess the treatment effect on the change from baseline in VAS sexual function (mm) up to Week 52 (FINCH 1 and 3) or Week 24 (FINCH 2). Patients indicated how RA affected their ability to have sex during the last week using an exploratory 0–100 VAS (0: no effect; 100: complete inhibition). Changes from baseline were analysed with a mixed-effects model for repeated measures. All P values are nominal for exploratory purposes.

Results: Baseline characteristics are shown in the Table 1. Univariate analyses revealed significant positive correlations (P<0.05) between disease duration and baseline VAS sexual function score in male and female subgroups in FINCH 1; no significant correlations were seen in male and female subgroups of FINCH 2 and 3. In all studies, significant correlations (P<0.05) were observed between baseline VAS sexual function score and baseline disease characteristics (swollen/tender joint count 28, Disease Activity Score-28, Health Assessment Questionnaire Disability Index, 36-Item Short Form Survey, patient global VAS, pain VAS or fatigue) in males or females. In all studies, analysis of least-squares mean changes from baseline in VAS sexual function revealed improvements in both males and females on FIL as early as Week 2, until Week 52 (Week 24 in FINCH 2). Figure 1 shows data for FINCH 1.

Conclusion: Sexual function should be considered as an important patient outcome in RA treatment. At baseline in the FINCH studies, disease activity

negatively impacted sexual function in both male and female patients. Active treatment with FIL or ADA resulted in early and sustained improvements from baseline in sexual function.

REFERENCES:

- [1] Tristano AG. *World J Orthop* 2014;5:107–11

Table 1. Mean (standard deviation) baseline characteristics

	FINCH 1		FINCH 2		FINCH 3	
	Male n=258	Female n=990	Male n=79	Female n=270	Male n=233	Female n=691
Duration of RA, yr	6.8 (7.38)	8.0 (7.63)	11.3 (8.53)	12.7 (9.35)	2.1 (5.35)	2.2 (4.85)
SJC28	11 (5.0)	11 (5.1)	13 (6.2)	12 (6.1)	11 (5.4)	11 (5.7)
TJC28	14 (6.5)	15 (6.4)	15 (7.5)	16 (7.0)	14 (6.6)	15 (6.7)
HAQ-DI	1.35 (0.614)	1.64 (0.601)	1.41 (0.689)	1.73 (0.634)	1.37 (0.651)	1.62 (0.617)
DAS28 (CRP)	5.6 (0.95)	5.8 (0.90)	5.8 (1.08)	5.9 (0.92)	5.7 (1.00)	5.7 (0.99)
SF-36 PCS	34.3 (7.72)	33.0 (7.34)	31.7 (8.48)	30.9 (7.75)	34.4 (7.72)	33.4 (7.47)
SF-36 MCS	45.9 (10.15)	43.6 (10.65)	43.7 (11.20)	44.5 (11.71)	46.2 (11.75)	43.0 (10.89)
FACIT-fatigue	30.0 (10.00)	26.8 (10.49)	26.3 (11.15)	24.0 (11.64)	30.7 (10.93)	26.6 (10.89)
Patient global VAS (mm)	64 (19.8)	67 (19.0)	66 (20.0)	70 (19.5)	65 (22.4)	66 (20.3)
Pain VAS (mm)	61 (20.6)	66 (19.5)	62 (22.3)	68 (20.6)	64 (22.3)	66 (20.9)
VAS sexual function score	44 (30.2)	49 (32.3)	48 (34.6)	49 (36.8)	42 (34.4)	48 (35.1)

Variables in bold significantly correlated with VAS sexual function score ($P < 0.05$) DAS28 (CRP), Disease Activity Score-28 using C-reactive protein; FACIT, Functional Assessment of Chronic Illness Therapy; HAQ-DI, Health Assessment Questionnaire Disability Index; MCS, mental component summary; PCS, physical component summary; RA, rheumatoid arthritis; SF-36, 36-Item Short Form Survey; S/TJC28, swollen/tender joint count based on 28 joints; VAS, visual analogue scale

Figure. Change from baseline in VAS sexual function over time for female and male subgroups in FINCH 1

Patients receiving placebo at Week 24 were re-randomised 1:1 to receive filgotinib 200 mg or 100 mg (data not shown)
* $P \leq 0.05$, ** $P \leq 0.01$ vs placebo
ADA, adalimumab; CI, confidence interval; FIL, filgotinib; LS, least-squares; MTX, methotrexate; PBO, placebo; VAS, visual analogue scale

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POS0527

ACUTE KIDNEY INJURY (AKI) IN PATIENTS WITH RHEUMATOID ARTHRITIS (RA)

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Background: Methotrexate has been an anchor drug for patients with rheumatoid arthritis (RA). However, it is strictly prohibited to prescribe MTX to patients with severely decreased renal function because it can induce a fatal adverse event such as pancytopenia in these patients. On the other hand, since the average age of RA patients is gradually increasing, and many of them already have mildly to moderately impaired renal function, their renal function can easily decrease to below the critical level of the estimated glomerular filtration rate. Therefore, new development of acute kidney injury (AKI) during MTX administration might induce a fatal adverse event, making the identification of patients susceptible to AKI very important.

Objectives: To clarify the frequency of AKI and the factors involved in it in RA patients.

Methods: Two hundred and fifty-two RA patients (211 females, 41 males, mean age 62.3 ± 12.5 years, disease duration 11.0 ± 9.5 years) diagnosed more than 3 years earlier and followed for more than 5 years, and also, others diagnosed ≥ 3 years earlier but followed for ≤ 5 years were enrolled. We measured BUN, Cr, RF and aCCP in patient serum, urinary proteins, urinary blood, and urinary casts and evaluated CDAI, SDAI, disease activity score (DAS) 28-CRP and DAS28-ESR. Steinbrocker functional classification and radiological grading were evaluated. History of diabetes mellitus, hypertension and hyperlipidemia was determined from the medical records. Medications for RA, including non-steroid anti-inflammatory drugs (NSAIDs), prednisolone, csDMARD (MTX, Tacrolimus, etc.), bDMARDs and tsDMARDs were evaluated. Estimated glomerular filtration rate (eGFR) was calculated by the new Japanese coefficient-modified Modification of Diet in Renal disease (MDRD) study equation. The criteria of AKI were that serum Cr increased by 0.3 mg/dl or increased by 1.5-fold between consecutive visits according to the KIDIGO criteria¹⁾ and the report of Leither et al²⁾.

Results: Twenty (7.9%) patients developed AKI, 22 times. The causes of AKI were 10 infections, 6 dehydrations, 2 enteritis, 1 urticaria, 2 hypercalcemia due to VitD administration, and 1 ureteral stone. We divided our patients into group A (with AKI) and group B (without AKI). Group A was older (69.9 ± 10.1 vs 61.7 ± 12.6 years), had greater physician VAS (29.5 ± 27.7 vs 15.7 ± 18.3 mm), higher serum creatinine (0.79 ± 0.19 vs 0.60 ± 0.16 mg/dl), higher BUN (18.4 ± 5.7 vs 15.1 ± 4.4 mg/dl), lower eGFR (65.5 ± 23.3 vs 86.4 ± 22.4 ml/min), more frequent prednisolone administration (75.0% vs 41.9%), more frequent hyperlipidemia (50.0% vs 19.2%) and more frequent hypertension (60.0% vs 30.6%) than Group B by univariate analysis significantly ($p < 0.01$). We then performed multifactorial analysis using logistic regression analysis. Greater physician VAS (OR 1.02, 1.00-1.04), lower eGFR (OR 1.04, 1.01-1.08) and prednisolone administration (OR 3.29, 1.02-10.63) were found as independent relevant factors for group A.

Conclusion: Our study indicated that AKI developed in RA patients and suggested that renal function decline and prednisolone administration may be implicated. RA patients with impaired renal function and prednisolone administration need to be treated with special attention to the onset of AKI.

REFERENCES:

- [1] Kidney Disease: Improving Global Outcomes (KDIGO) Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl* 2: 1-138, 2012
[2] Leither MD, Murphy DP, Bicknese L et al. The impact of outpatient acute kidney injury on mortality and chronic kidney disease: a retrospective cohort study. *Nephrol Dial Transplant*. 34:493-501, 2019

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