

Overview of the participants categorized by age and disease

Men	All	NCD	Arthritis	No arthritis, no NCD
60-64	730	194	111	466
65-69	747	263	110	412
70-74	528	250	108	222
75-79	329	180	67	115
80-84	162	89	42	57
60-84	2496	976	438	1272
Women				
60-64	812	154	241	465
65-69	856	231	316	394
70-74	668	207	271	278
75-79	378	139	179	136
80-84	190	92	97	55
60-84	2904	823	1104	1328

NCD=non-communicable diseases

Reference values for the Timed Up and Go test in seconds

Age	All (n=5400)		NCD(n=1779)		Arthritis(n=1542)		No NCD, no arthritis (n=2600)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Men								
60	8.2	1.7	8.4	1.9	8.6	2.1	8.0	1.5
65	8.2	1.7	8.4	1.9	8.2	2.1	8.1	1.5
70	8.7	1.7	8.9	1.9	8.8	2.1	8.5	1.5
75	9.5	1.7	9.7	1.9	9.8	2.1	9.3	1.5
80	10.4	1.7	10.6	1.9	10.7	2.1	10.1	1.5
84	11.2	1.8	11.4	1.9	11.5	2.1	10.7	1.5
Women								
60	7.8	2.0	7.9	2.4	7.9	2.1	7.8	1.7
65	8.3	2.0	8.7	2.4	8.4	2.1	8.1	1.7
70	9.0	2.0	9.3	2.4	9.0	2.1	8.8	1.8
75	9.8	2.0	10.1	2.4	9.9	2.1	9.5	1.7
80	10.6	2.0	10.9	2.4	10.8	2.1	10.3	1.7
84	11.2	2.0	11.6	2.4	11.5	2.2	10.9	1.7

Values estimated from quantile regression, while mean (SD) was estimated from a linear regression model. In both regression settings age was included as a restricted cubic spline with 4 knots at default knot location (60, 66, 71, 80). Models were run separately for men and women.
SD = standard deviation, NCD = non-communicable diseases

catastrophizing, neuropathic-like pain, fatigue, sleep-disturbance, pain-distribution, or cognitive-impact. Presence/absence of pain persistence (pain present at baseline and year 1, n=1471) served as the primary pain outcome. In those reporting pain persistence, persistent pain severity (n=976) served as the secondary pain outcome. Residualised change scores were determined for persistent pain severity, and the four putative mediators of anxiety and depression (HADS anxiety and depression subscales), neuropathic-like pain (modified PainDetect Questionnaire), and Catastrophizing (Pain Catastrophizing Scale). A bias-corrected bootstrapping procedure with 5000 resamples estimated indirect effects of the Central Mechanisms trait on both pain outcomes through each of the putative mediators individually, and as a group. All analyses were conducted using mPlus version 8.2, and indirect effects are presented using standardised regression coefficients (β). **Results:** Increases from baseline to year-1 follow-up were observed for pain intensity, depression, pain catastrophizing and neuropathic-like symptoms (range of mean change=0.09 to 1.34, $p<0.1$), but not for anxiety (mean change=-0.37, $p<0.05$). Higher Central Mechanisms trait at baseline predicted persistent pain ($\beta=0.12$, 95%CI [0.10, 0.16], $p<0.001$), and worsening persistent pain severity at 1-year follow-up ($\beta=0.48$, 95%CI [0.42, 0.53], $p<0.001$). Significant ($p<0.001$) indirect effects of the Central Mechanisms trait on persistent pain severity were identified through Pain Catastrophizing ($\beta = 0.18$, 95%CI [0.11, 0.25]), anxiety ($\beta=-0.08$, 95%CI [-0.14, -0.02]); and Neuropathic-like pain ($\beta=0.18$, 95% CI [0.13, 0.24]), but not through depression. Significant indirect effects of the Central Mechanisms trait on pain persistence were not identified through any of the 4 putative mediators. Together, all four putative mediators had a significant indirect effect on the relationship between baseline Central Mechanisms trait and persistent pain severity ($\beta=0.22$, 95%CI [0.13, 0.30], $p<0.001$), but not on the relationship between baseline Central Mechanisms trait and pain persistence ($\beta=0.10$, 95%CI [-0.11, 0.12], $p=0.54$).

Conclusions: Worsening modifiable traits of anxiety, pain catastrophizing and neuropathic-like symptoms partially mediated prediction by the baseline Central Mechanisms trait of worsening knee pain severity at year-1 follow-up, but did not significantly affect whether knee pain would resolve or persist. Further research should investigate whether high Central Mechanisms trait can identify people for whom treatments that improve or prevent anxiety, pain catastrophizing or neuropathic-like symptoms might improve otherwise unfavourable knee pain progression.

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INTRA-ARTICULAR CORTICOSTEROIDS FOR KNEE AND HIP OA: A SYSTEMATIC REVIEW OF SERIOUS ADVERSE JOINT OUTCOMES

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Purpose: Intra-articular corticosteroids (IACS) are proven to provide short-term pain relief and functional improvement in individuals with knee and hip osteoarthritis (OA). However, the potential for serious adverse joint outcomes resulting from long-term use of IACS has been cause for concern. We conducted a systematic review of all RCT data comparing IACS to intra-articular saline to explore the incidence of accelerated OA progression, subchondral insufficiency fracture, complication of osteonecrosis, and/or rapid destruction of joint tissue among individuals with knee and hip OA.

Methods: Medline, PubMed Central, Google Scholar, and the Cochrane Databases were systematically searched from inception to November, 2019. Reference lists of relevant reviews were searched by hand, and we sought additional data within supplements of conference proceedings that had been published up until November, 2019. Randomized controlled trials comparing IACS to intra-articular saline in participants with knee or hip OA were included. Two reviewers screened potentially relevant references and independently assessed study quality using the Cochrane Risk of Bias tool. Relevant data were independently extracted from included studies by the same two reviewers. Outcomes of interest included incidence of any one of four target serious adverse joint outcomes: accelerated OA progression, subchondral insufficiency fracture, complication of osteonecrosis, and/or rapid destruction of joint tissue. Analysis of dichotomous outcomes using the Mantel-Haenszel method was planned *a priori*, and effect estimates were to be reported as risk ratios (RR) with 95% confidence intervals.

Results: Our search returned 516 potentially relevant references. Of these, 28 references were deemed to be potentially eligible for

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MEDIATORS OF THE PREDICTIVE RELATIONSHIP BETWEEN A SELF-REPORT MEASURE OF CENTRAL MECHANISMS AND FUTURE KNEE PAIN OUTCOMES IN THE KNEE PAIN IN THE COMMUNITY (KPIC) COHORT

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Purpose: Knee pain is a major source of disability in people over the age of 50 years, most commonly due to osteoarthritis. Central mechanisms can increase pain sensitivity, and be a barrier to response to peripherally targeted interventions, such as knee arthroplasty. Our previous research identified a self-report Central Mechanisms trait, which was associated with Quantitative Sensory Testing evidence of central sensitization. Baseline scores for the Central Mechanisms trait also predicted pain persistence at 1-year follow-up. We aimed to investigate which changes in modifiable self-report traits might mediate the predictive relationship between baseline scores for Central Mechanisms trait and future knee pain outcomes.

Methods: Data from participants consenting to the Knee Pain In the Community (KPIC) study who reported knee pain at baseline and responded at 1-year follow-up were analysed. A summary score for a latent 'central mechanisms' trait was derived from baseline scores for 8 items representing component traits of anxiety, depression,