

Cognition and arthritis in adults over 50: Evidence from CHARLS, HRS, and ELSA

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ABSTRACT

Introduction: Arthritis and cognitive decline are significant health challenges in ageing populations, yet their association across diverse socioeconomic and cultural contexts remains insufficiently understood. This study examines the relationship between cognitive function and arthritis prevalence among adults aged 50 years and older, utilising data from 3 longitudinal ageing cohorts: the China Health and Retirement Longitudinal Study (CHARLS), the Health and Retirement Study (HRS), and the English Longitudinal Study of Ageing (ELSA).

Methods: The analysis included 18,562 participants from CHARLS, 23,238 from HRS, and 9848 from ELSA, all aged ≥ 50 years with physician-diagnosed arthritis and cognitive assessments. Cognitive function was evaluated across memory, orientation, and executive function domains, standardised as z-scores within each cohort. Generalised Estimating Equations (GEE) and Generalised Linear Mixed Models assessed the longitudinal association between cognition and arthritis, adjusting for socio-demographic factors, lifestyle variables, and comorbidities. Sensitivity analyses validated the findings' robustness.

Results: In fully adjusted GEE models, executive function was significantly associated with arthritis. Each 1-standard deviation (SD) lower executive score was associated with higher arthritis odds: CHARLS odds ratio (OR) 1.000 (95% confidence interval [CI] 1.000–1.001, $P=0.016$), HRS OR 1.13 (95% CI 1.01–1.28, $P=0.037$), and ELSA OR 1.07 (95% CI 1.02–1.12, $P=0.009$). Neither episodic memory nor orientation showed significant associations (all $P>0.10$), and total cognition in HRS was significant. Mixed-model estimates were concordant, and alternative correlation structures yielded consistent executive–arthritis links.

Conclusion: Across 3 major ageing cohorts, impaired executive function is independently associated with higher odds of arthritis in adults aged ≥ 50 years. Targeting executive deficits may enhance early identification and integrated management of arthritis and cognitive health in ageing populations.

Keywords: ageing, arthritis, cognitive function, cross-national analysis, epidemiology, geriatric medicine, longitudinal cohorts, statistics

CLINICAL IMPACT

What is New

- This study demonstrates a consistent association between impaired executive function and higher odds of arthritis across 3 large ageing cohorts from China, US, and the UK.
- Among cognitive domains, executive function—but not memory or orientation—was independently linked to arthritis risk in adults aged ≥ 50 years.

Clinical Implications

- Executive function assessment may serve as a practical tool for early identification of individuals at increased risk of arthritis in ageing populations.
- Integrating cognitive evaluation into routine clinical care could support more comprehensive management strategies addressing both cognitive and musculoskeletal health.

INTRODUCTION

The world is transitioning towards older age groups, with the share of people aged 60 years and above expected to increase 2-fold by 2050.¹ This demographic transition poses significant public health challenges, notably the rising incidence of cognitive impairment and dementia, which severely impact the well-being of older adults and place considerable strain on healthcare systems.² Identifying modifiable risk factors associated with cognitive decline is critical for devising strategies to mitigate its onset and progression. Among these factors, arthritis—a chronic condition affecting approximately 23% of adults in the US, with prevalence escalating with age—has garnered increasing attention.³ Beyond its well-documented effects on physical mobility and quality of life, arthritis may also influence

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cognitive function through multiple pathways, warranting further investigation.

Arthritis, encompassing conditions such as osteoarthritis (OA) and rheumatoid arthritis (RA), is characterised by chronic pain and inflammation, both of which have been implicated in cognitive decline. Research indicates that persistent pain can impair cognitive performance by disrupting sleep, elevating depressive symptoms, and necessitating medications with cognitive side effects.⁴ Systemic inflammation, a common feature of arthritis, is also recognised as a contributor to neurodegenerative processes, including those underlying dementia.⁵ Moreover, arthritis often restricts physical activity and social participation—lifestyle factors known to bolster cognitive resilience.⁶ Preliminary studies have reported associations between arthritis and increased risks of cognitive impairment, yet the evidence remains fragmented, often limited to specific populations or lacking longitudinal depth.^{7,8}

Despite these insights, significant gaps persist in understanding the relationship between arthritis and cognitive function, particularly across diverse socioeconomic and cultural contexts. Much of the existing research originates from high-income countries, leaving the association underexplored in low- and middle-income settings where healthcare access and lifestyle factors may differ markedly. Cross-national investigations are thus essential to determine whether this relationship holds universally or varies due to regional influences. To address these deficiencies, this study harnesses data from 3 prominent longitudinal ageing cohorts: the China Health and Retirement Longitudinal Study (CHARLS), the Health and Retirement Study (HRS) in the US, and the English Longitudinal Study of Ageing (ELSA) in the UK. These datasets offer detailed, representative information on health, socioeconomic status, and cognitive outcomes among middle-aged and older adults, facilitating robust, comparative analyses.

This study primarily aims to examine the overall association between arthritis and cognitive function in adults aged 50 years and older, utilising data from CHARLS, HRS, and ELSA. Secondary objectives include exploring potential mediators, such as pain, inflammation, and physical inactivity, and assessing whether the association differs across countries or demographic subgroups. By integrating these rich, multinational datasets, this research seeks to provide a comprehensive understanding of how arthritis relates to cognitive health, offering insights that could guide targeted interventions to preserve cognitive function in ageing populations globally.

METHODS

Study population and data sources

A longitudinal cohort analysis was conducted using harmonised data from 3 nationally representative ageing surveys: CHARLS, HRS, and ELSA. CHARLS is a biennial survey of Chinese adults aged ≥ 45 years and their spouses, with baseline data collected in 2011–2012 using multistage probability sampling and face-to-face interviews.⁹ Initiated in 1992, HRS is a biennial panel survey of US adults born before 1947 and their spouses, aimed at investigating health, economic, and family transitions associated with retirement.¹⁰ ELSA commenced in 2002 with follow-up waves every 2 years, enrolling English residents aged ≥ 50 years in a multidisciplinary protocol aligned with HRS.¹¹

For each cohort, the baseline is defined as the first wave in which both arthritis status and cognitive measures were concurrently available (CHARLS wave 1 [2011], HRS wave 9 [2008], and ELSA wave 5 [2010]). Participants were eligible if they were aged ≥ 50 years, had physician-diagnosed arthritis information, completed at least 2 waves of cognitive assessments, and had non-missing data on key sociodemographic covariates (gender, education, marital status). After sequential exclusions for age < 50 years, missing arthritis or cognitive measures, missing covariates, and fewer than 2 waves of follow-up, the analytic samples comprised 18,562 (CHARLS), 23,238 (HRS), and 9848 (ELSA) participants (Fig. 1).

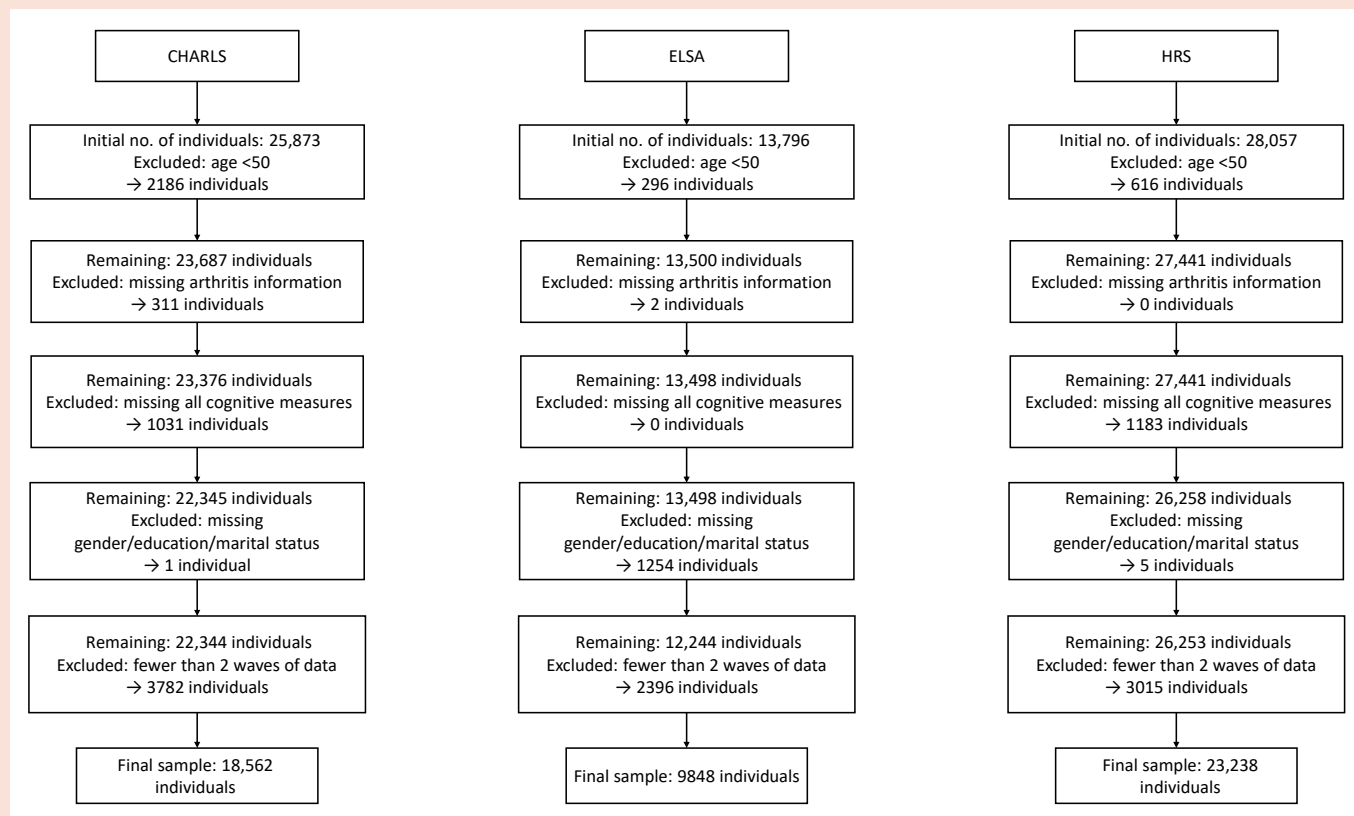
Assessment of arthritis

Arthritis status was ascertained via self-report of a physician diagnosis in all 3 cohorts, a method validated against medical records with high specificity for OA and RA in older adults.¹² In CHARLS and HRS, participants answered “Has a doctor ever told you that you have arthritis?”; ELSA used a parallel question in its health interview module. Affirmative responses were treated as prevalent arthritis at each wave. To account for incident cases, arthritis status was updated at each follow-up and included as a time-varying exposure.

Cognitive function measures

Cognitive performance was evaluated across 3 domains—episodic memory, orientation, and executive function—as well as a composite total score. Episodic memory comprised immediate and delayed recall of a 10-word list, scored 0–10 in CHARLS (mean of the 2 trials) and 0–20 in HRS and ELSA (sum).¹³ Orientation was assessed via questions on date, day of week, and

Fig. 1. Participants selection flowchart.



season (scored 0–5) in CHARLS and HRS; ELSA did not include an orientation subscale, so orientation data were from CHARLS and HRS only. Executive function was measured by verbal fluency (animals named in 1 minute; truncated at 45 seconds in HRS and ELSA) and a pentagon-drawing test (0–1) in CHARLS.¹³ Total cognition scores were created by summing available domain scores within each cohort. To facilitate cross-cohort comparison, all domain and total scores were standardised (z-scores) within each dataset, with higher values indicating better cognition. The substantial heterogeneity in cognitive measurement across the cohorts is acknowledged, such as the absence of an orientation subscale in ELSA and variations in specific executive function items. Analytically, standardising domain scores mitigates some of these variabilities, though conceptual differences in the underlying constructs remain.

Covariates

The authors adjusted for baseline sociodemographic factors, lifestyle behaviours, and comorbidities known to confound the arthritis–cognition relationship. Covariates included age (continuous), gender (male/female), residence (urban/rural in CHARLS; region in HRS/ERCLA; England only in ELSA), marital status (married versus [vs] other), and education (primary,

secondary, tertiary).^{9,11} Lifestyle factors comprised self-reported smoking (yes/no) and drinking (yes/no) status, and body mass index (BMI, kg/m²) derived from objectively measured height and weight. Chronic health conditions—hypertension, diabetes, heart disease, stroke, cancer, and psychiatric disorders—were self-reported physician diagnoses. Depressive symptoms were assessed using the 10-item Center for Epidemiologic Studies-Depression (CES-D) in CHARLS (cut-off ≥10) and HRS/ELSA (8-item CES-D cut-off ≥3; 12-item EURO-D cut-off ≥4). All covariates were measured at baseline.

Statistical analysis

The primary analysis employed Generalised Estimating Equations (GEE) to examine the longitudinal association between cognitive function and arthritis, accounting for repeated measures within individuals.⁴ An exchangeable correlation structure was specified, and 5 models were fitted per cohort: one for each cognitive domain (memory, orientation, executive function, total cognition) and one for the multi-domain model. The GEE model was defined as:

$$\text{logit}(P(\text{arthritis}_{it})) = \beta_0 + \beta_1 \text{cognition}_{it} + \beta_2 \text{covariates}_{it}$$

Results are expressed as odds ratios (ORs) accompanied by their 95% confidence intervals (CIs).

To assess robustness, Generalised Linear Mixed Models (GLMM) with random intercepts for participant ID were conducted as a secondary analysis⁵:

$$\text{logit}(P(\text{arthritis}_{it})) = \beta_0 + \beta_1 \text{cognition}_{it} + \beta_2 \text{covariates}_{it} + u_i$$

Sensitivity analyses tested the stability of findings. (1) Cognition as tertiles: cognitive scores were categorised into tertiles (low/mid/high) and analysed using multinomial GEE to evaluate dose–response relationships; (2) alternate correlation structures: GEE models were refitted using independence and first-order autoregressive (AR-1) working correlation matrices to verify that findings were not contingent on the exchangeable assumption.¹⁴ All sensitivity models included the same covariate set as the main analysis. Results across CHARLS, HRS, and ELSA were synthesised descriptively, rather than pooled, due to differences in covariate distributions and measurement protocols.

Analyses were conducted in R version 4.5.0 (R Foundation for Statistical Computing, Vienna, Austria), utilising geepack for GEE and lme4 (R Foundation for Statistical Computing, Vienna, Austria) for GLMM. Statistical significance was set at a two-tailed $P < 0.05$.

RESULTS

This study included 51,648 participants aged 50 years and older from 3 longitudinal cohorts: 18,562 from CHARLS (China), 23,238 from HRS (US), and 9848 from ELSA (UK). The median baseline age was 58 years (interquartile range [IQR] 52–64) in CHARLS, 60 years (IQR 54–71) in HRS, and 63 years (IQR 57–72) in ELSA. Females comprised 48.8% of CHARLS, 42.1% of HRS, and 45.8% of ELSA participants. Across all cohorts, participants with arthritis were significantly older, more likely to be female, and had lower education levels compared to those without arthritis (all $P < 0.001$). Cognitive function scores were consistently lower in the arthritis groups, with total cognition z-scores of 0.02 (IQR -0.74–0.64) vs 0.27 (IQR -0.49–0.88) in CHARLS, 0.07 (IQR -0.63–0.54) vs 0.19 (IQR -0.41–0.66) in HRS, and 0.90 (IQR 0.44–1.25) vs 1.02 (IQR 0.67–1.37) in ELSA (all $P < 0.001$). Participants with arthritis also exhibited higher BMI, greater prevalence of hypertension, diabetes, heart disease, stroke, cancer, psychiatric disorders, and depressive symptoms, alongside lower rates of drinking and smoking (all $P < 0.05$). These findings underscore significant demographic, cognitive, and health-related differences associated with arthritis in older adults across the cohorts (Fig. 1) (Supplementary Tables S3–S5).

The overall association between domain-specific cognitive performance and odds of physician-diagnosed arthritis was first examined using GEE with an exchangeable correlation structure. In the fully

adjusted GEE models, impaired executive function emerged as the cognitive domain significantly associated with elevated arthritis risk across all 3 cohorts. Specifically, each 1-standard deviation (SD) decrement in executive score corresponded to a 1.000-fold increase in odds of arthritis in CHARLS (OR 1.000, 95% CI 1.000–1.001, $P = 0.016$), a 1.13-fold increase in HRS (OR 1.13, 95% CI 1.01–1.28, $P = 0.037$), and a 1.07-fold increase in ELSA (OR 1.07, 95% CI 1.02–1.12, $P = 0.009$). In contrast, neither memory nor orientation scores demonstrated significant associations with arthritis (all $P > 0.10$), and the total cognition score models lacked consistent significant associations across cohorts (Supplementary Table S1, Fig. 2).

To assess the robustness of these findings when accounting for intra-individual variability, the authors fitted GLMM with random intercepts for participant ID. The GLMM results paralleled those of the GEE analyses, further underscoring the specific role of executive impairment in arthritis risk. In HRS, a 1-SD lower executive score was associated with a 1.13-fold higher odds of arthritis (OR 1.13, 95% CI 1.02–1.26, $P = 0.021$), whereas CHARLS and ELSA yielded ORs of 1.17 (95% CI 1.05–1.32, $P = 0.007$) and 1.07 (95% CI 1.03–1.11, $P = 0.0003$), respectively. Neither memory nor orientation domains reached statistical significance under the GLMM framework (all $P > 0.05$), and the total cognition GLMM estimates remained consistent in HRS but imprecise in CHARLS and ELSA ($P > 0.05$). These parallel analytic approaches confirm that the observed executive–arthritis association is not an artifact of model selection or unmeasured individual-level heterogeneity (Supplementary Table S2, Fig. 3).

In sensitivity analyses, the authors first varied the GEE working correlation structure to independence and AR-1 specifications. Across CHARLS, HRS, and ELSA, the inverse relationship between executive function and arthritis remained robust: each 1-SD decrement in executive score continued to predict significantly higher odds of arthritis under both independence and AR-1 structures (all $P < 0.01$). By contrast, when overall cognition was recoded into tertiles (low, middle, high), no significant associations emerged in any cohort (all $P > 0.10$), confirming that the tertile categorisation attenuated the effect. These findings underscore that the executive-domain result is not an artifact of the chosen correlation matrix and that dichotomising or trichotomising global cognition obscures the specific executive–arthritis linkage observed in the main analyses (Supplementary Table S6).

DISCUSSION

In this large, multinational cohort analysis of 51,648 adults aged ≥ 50 years from CHARLS, HRS, and ELSA,

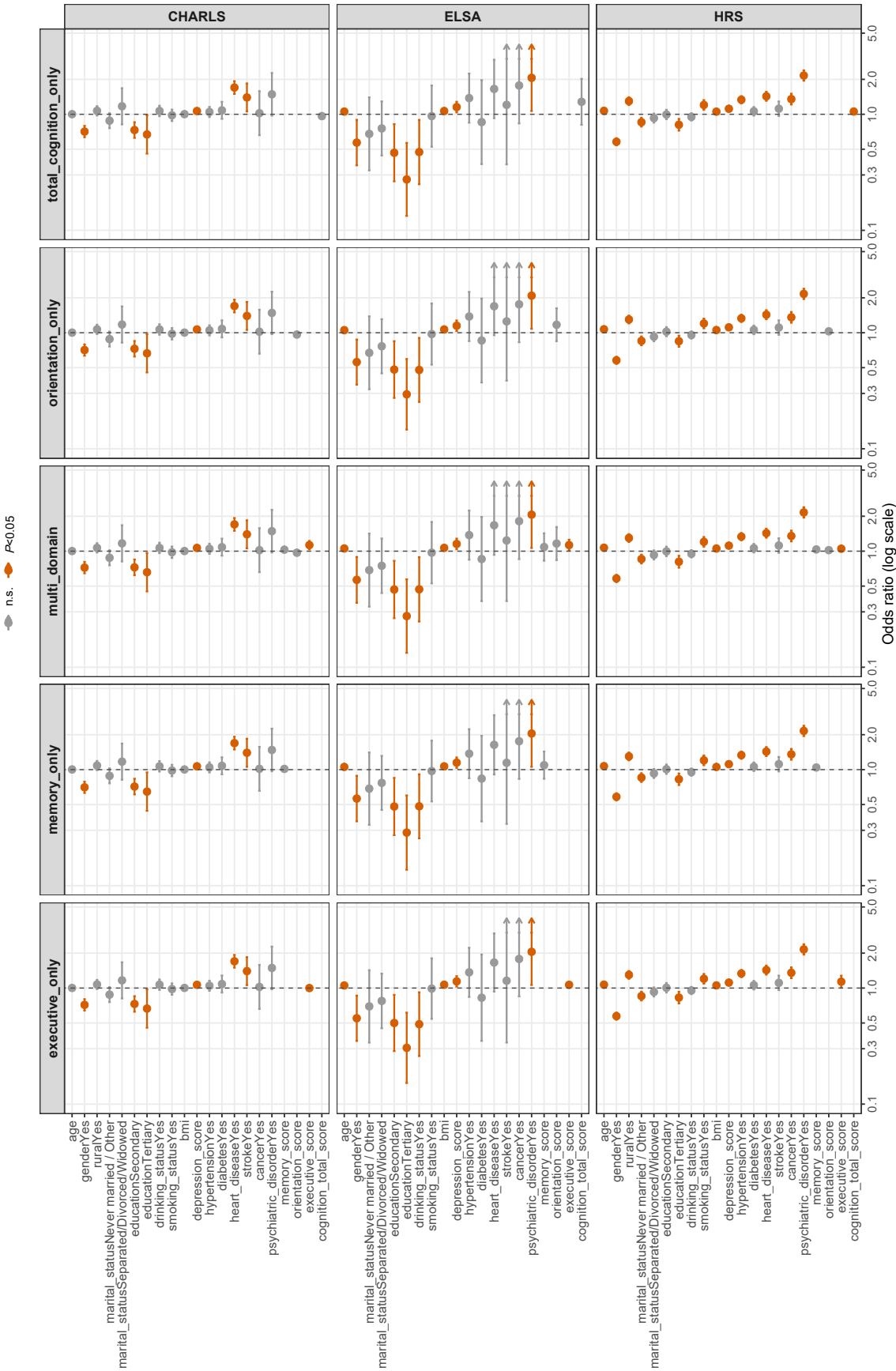


Fig. 2. Associations between cognitive function and arthritis in CHARLS, HRS, and ELSA using GEE.

BMI: body mass index; CHARLS: China Health and Retirement Longitudinal Study; ELSA: English Longitudinal Study of Ageing; GEE: Generalised Estimating Equations; HRS: Health and Retirement Study; OR: odds ratio

Results are derived from 5 GEE models per dataset (15 models total), adjusted for covariates including age, gender, rural residence, marital status, education, drinking status, smoking status, BMI, depression score, hypertension, diabetes, heart disease, stroke, cancer, and psychiatric disorders, with an exchangeable correlation structure. The x-axis displays ORs on a logarithmic scale, while the y-axis lists cognitive domains and datasets. Significant correlations ($P < 0.05$) are marked with orange dots and non-significant correlations are marked with grey dots.

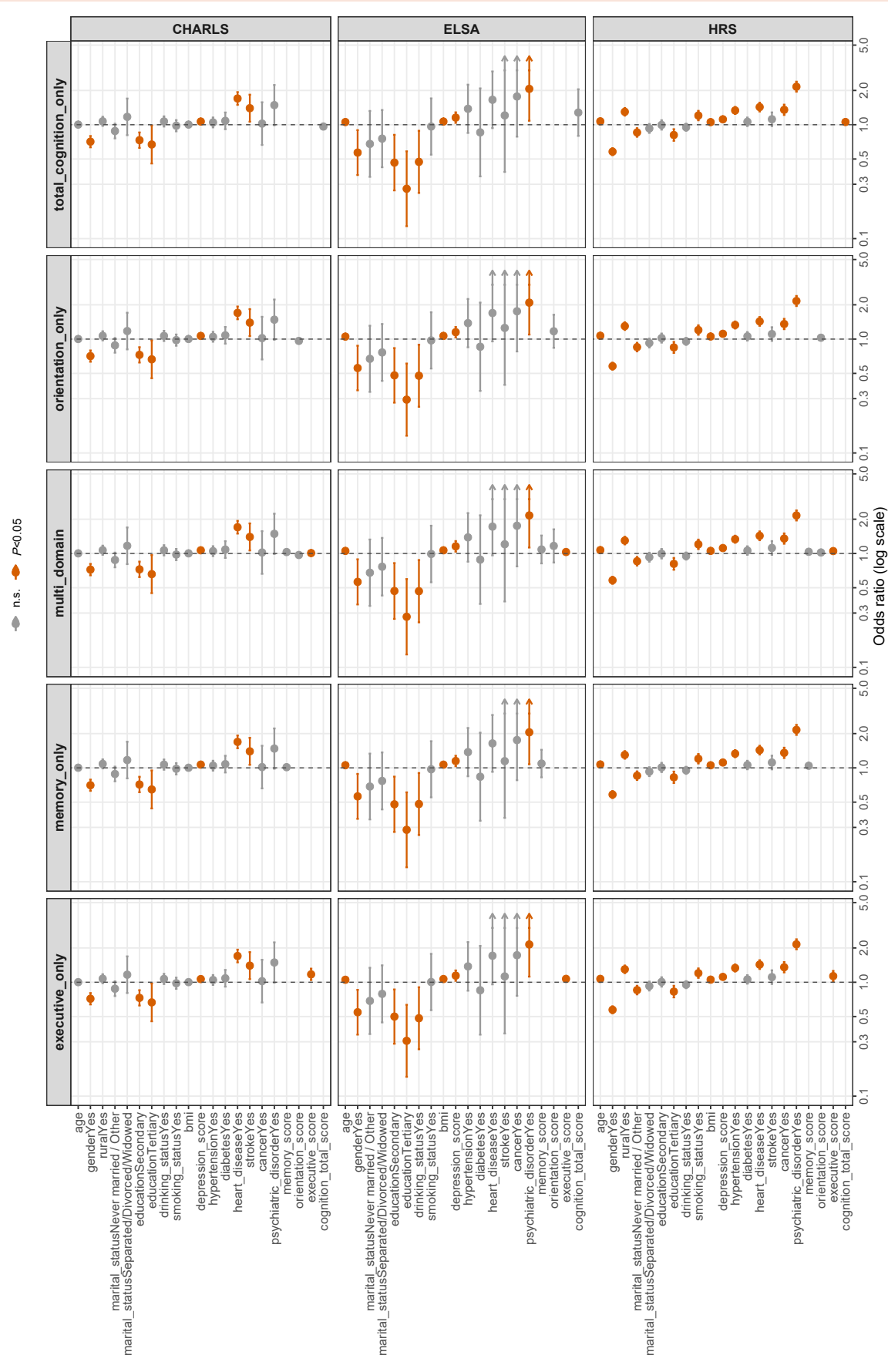


Fig. 3. Associations between cognitive function and arthritis in CHARLS, HRS, and ELSA using GLMM.

BMI: body mass index; CHARLS: China Health and Retirement Longitudinal Study; ELSA: English Longitudinal Study of Ageing; GLMM: Generalised Linear Mixed Models; HRS: Health and Retirement Study; OR: odds ratio

Models are adjusted for the same covariates as in the GEE analysis: age, gender, rural residence, marital status, education, drinking status, smoking status, BMI, depression score, hypertension, diabetes, heart disease, stroke, cancer, and psychiatric disorders. The x-axis shows ORs on a logarithmic scale, and the y-axis categorises results by cognitive domain and dataset. Significant correlations ($P<0.05$) are marked with orange dots and non-significant correlations are marked with grey dots.

a consistent, independent association between lower executive function and higher odds of physician-diagnosed arthritis was observed across 3 culturally distinct populations. Each 1-SD decrement in executive function predicted a 7–17% increase in arthritis risk, findings that were robust across GEE and GLMM frameworks and persisted under alternative correlation structures. Notably, neither episodic memory nor orientation exhibited significant associations after full adjustment, and global cognition showed only a significant positive trend in HRS. These results highlight the specificity of executive dysfunction as a correlate of arthritis, suggesting that processes subserved by prefrontal networks may be particularly vulnerable to the systemic and behavioral sequelae of joint disease.

Comparison with previous studies

The results align with emerging evidence that systemic joint disease may herald or exacerbate cognitive decline. A recent Korean longitudinal study found that individuals with cognitive impairment had a 47.6% incidence of arthritis over 12 years vs 30.1% for those with normal cognition, underscoring bidirectional risk.¹⁵ A recent meta-analysis demonstrated that OA confers a 1.25- to 1.80-fold elevated risk of dementia, alongside neuroimaging evidence of focal cerebral atrophy.^{16,17} In RA, patients exhibited higher rates of cognitive impairment—up to 71% in some cohorts—linked to chronic inflammation and white matter changes.^{18,19} This study complements these findings by focusing on arthritis as a predictor rather than cognitive decline, and by controlling for a comprehensive set of confounders, including depressive symptoms and cardiovascular comorbidities.

Potential mechanisms

Multiple pathways may underpin the cognitive–arthritis nexus. First, chronic inflammation is a shared hallmark; elevated C-reactive protein (CRP) and interleukin-6 in RA patients have been associated with poorer executive and memory performance.²⁰ Low-grade systemic inflammation was also linked to long-term cognitive decline in a community-based cohort of 2000 adults over 18 years.^{21–23} Second, persistent pain inherent to OA and RA can disrupt sleep and increase depressive symptoms, both detrimental to cognition; rodent models of OA demonstrate measurable deficits in object recognition tasks following joint pain induction.²⁴ Third, arthritis often curtails physical activity and social engagement—behaviours shown to bolster cognitive reserve—suggesting that reduced stimulation may accelerate cognitive ageing.²⁵ Finally, arthritis treatments may modulate neuroinflammation; disease-modifying antirheumatic drugs and tumor necrosis factor (TNF) inhibitors have shown promise in reducing neuroinflammatory markers and improving cognitive metrics in pilot studies.^{20,26}

Strengths and limitations

The key strengths of this study include the use of 3 nationally representative cohorts with harmonised cognitive and arthritis measures, long follow-up, and rigorous sensitivity analyses confirming the stability of the findings. By employing both GEE and GLMM, the authors accounted for within-person correlation and unobserved heterogeneity. However, certain limitations merit consideration. The observational design precludes causal inference, and residual confounding—particularly by unmeasured factors such as pain severity, specific medication use (e.g. analgesics), and objective physical activity levels—cannot be excluded. Dementia incidence was not uniformly defined across cohorts, potentially introducing misclassification, although the primary focus on executive–arthritis associations reduces dependence on dementia ascertainment. Reverse causality is another concern. Early executive declines might impair arthritis self-management, reducing treatment adherence and leading to worse joint outcomes. Nonetheless, sensitivity analyses excluding early arthritis cases produced similar estimates, mitigating this possibility. Last, cognitive domain measures varied slightly by cohort (e.g. absence of orientation subscale in ELSA), although consistent executive associations across datasets suggest robustness. Furthermore, the reliance on self-reported physician diagnosis of arthritis may introduce non-differential misclassification. However, such bias would likely push the observed associations toward the null, suggesting that the true relationship between executive function and arthritis might be even stronger than reported here.

Clinical and public health implications

The findings have several practical implications. First, clinicians managing older adults with arthritis should be alert to potential concomitant cognitive deficits and consider routine cognitive screening, as early identification may facilitate interventions to preserve function. While the individual-level effect sizes observed in this study are modest (e.g. ORs ranging from 1.07 to 1.17), the high prevalence of both arthritis and cognitive decline in ageing populations means that this association translates into a substantial clinical and public health burden. Second, integrated strategies targeting inflammation and pain management—such as optimised use of disease-modifying anti-rheumatic drugs or non-steroidal anti-inflammatory drugs (NSAID)—could yield cognitive benefits, as suggested by a 12% reduction in dementia risk among long-term NSAID users in a Rotterdam study.²⁷ Third, interventions promoting physical activity and social participation in arthritis populations may confer dual benefits for joint health and cognitive

resilience. Finally, public health efforts should emphasise arthritis prevention and early treatment as part of broader dementia risk-reduction frameworks.

Future directions

Prospective trials are needed to test whether arthritis therapies can slow cognitive decline. Mechanistic studies should elucidate the relative contributions of systemic vs neuroinflammation, perhaps leveraging imaging biomarkers and cerebrospinal fluid analyses. Additionally, research exploring genetic predispositions linking arthritis and Alzheimer's-type pathology—such as the role of human leukocyte antigen DR (HLA-DR) alleles—could reveal novel therapeutic targets. Finally, expanding cross-cultural studies to low- and middle-income settings will clarify the generalisability of the cognitive–arthritis link and inform resource-appropriate interventions.

CONCLUSION

Across 3 major ageing cohorts, impaired executive function emerged as a specific and consistent correlate of arthritis in middle-aged and older adults. This executive–arthritis link likely reflects intertwined pathways of pain, systemic inflammation, and physical inactivity, with significant implications for identifying at-risk individuals and designing targeted interventions. Addressing executive deficits in arthritis care may offer a valuable strategy to preserve both joint and cognitive health in ageing populations.

Supplementary materials

Table S1. Summary of GEE model results for the association between cognitive function and arthritis in CHARLS, HRS, and ELSA.

Table S2. Summary of GLMM model results for the association between cognitive function and arthritis in CHARLS, HRS, and ELSA.

Table S3. Baseline characteristics of the study population in CHARLS (n=18,562).

Table S4. Baseline characteristics of the study population in HRS (n=23,238).

Table S5. Baseline characteristics of the study population in ELSA (n=9848).

Table S6. Sensitivity analysis of the association between cognitive function and arthritis using GEE models with different specifications in CHARLS, HRS, and ELSA.

Ethics statement

CHARLS, HRS, and ELSA each obtained approval from their institutional review boards, and informed consent was secured from all participants at the time of data collection. This analysis employed anonymised, publicly

accessible datasets, obviating the requirement for further ethical clearance.

Declaration

The authors have no affiliations with or involvement in any organisation or entity with any financial interest in the subject matter or materials discussed in this manuscript. There is no conflict of interest and funding to declare. Generative artificial intelligence (AI) tools were used in the preparation of this manuscript for tasks such as improving grammar, refining language, and suggesting phrasing. The authors retain full responsibility for the scientific analysis, interpretation of results, and conclusions. No AI was involved in data analysis or the generation of scientific content.

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