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# Social Health and Change in Cognitive Capability among Older Adults: Findings from Four European Longitudinal Studies

Jane Maddock[a] Federico Gallo[b, c] Frank J. Wolters[d, e] Jean Stafford[a] Anna Marseglia[f] Serhiy Dekhtyar[b] Marta Lenart-Bugla[g] Eline Verspoor[h] Marieke Perry[h] Suraj Samtani[i] Myrra Vernooij-Dassen[j] Karin Wolf-Ostermann[k, l] Rene Melis[h] Henry Brodaty[i] Mohammad Arfan Ikram[d] Anna-Karin Welmer[b, m, n] Daniel Davis[a] George B. Ploubidis[o] Marcus Richards[a] Praveetha Patalay[a, o] on behalf of the SHARED Consortium

[a]MRC Unit for Lifelong Health and Ageing at UCL, Faculty of Population Health, University College London, London, UK; [b]Aging Research Center, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, and Stockholm University, Stockholm, Sweden; [c]Centre for Cognition and Decision Making, Institute for Cognitive Neuroscience, National Research University Higher School of Economics, Moscow, Russian Federation; [d]Department of Epidemiology, Erasmus MC Rotterdam, Rotterdam, The Netherlands; [e]Department of Radiology and Nuclear Medicine, Erasmus MC Rotterdam, Rotterdam, The Netherlands; [f]Division of Clinical Geriatrics, Center for Alzheimer Research, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Stockholm, Sweden; [g]Department of Psychiatry, Wroclaw Medical University, Wroclaw, Poland; [h]Department of Geriatric Medicine, Radboud Institute for Health Sciences, Radboud University Medical Center, Nijmegen, The Netherlands; [i]Centre for Healthy Brain Ageing (CHeBA), Discipline of Psychiatry and Mental Health, School of Clinical Medicine, University of New South Wales Sydney, Sydney, NSW, Australia; [j]Scientific Center for Quality of Healthcare (IQ Healthcare), Radboud University Medical Center, Nijmegen, The Netherlands; [k]Institute for Public Health and Nursing Research, University of Bremen, Bremen, Germany; [l]Leibniz ScienceCampus Digital Public Health, Bremen, Germany; [m]Division of Physiotherapy, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Stockholm, Sweden; [n]Women's Health and Allied Health Professionals Theme, Medical Unit Medical Psychology, Karolinska University Hospital, Stockholm, Sweden; [o]Centre for Longitudinal Studies, UCL Institute of Education, University College London, London, UK



**Keywords**
Social support · Social network · Cognition · Longitudinal study · Cohort

**Abstract**

***Introduction:*** In this study, we examine whether social health markers measured at baseline are associated with differences in cognitive capability and the rate of cognitive decline over an 11-to-18-year period among older adults and compare results across studies. ***Methods:*** We applied an integrated data analysis approach to 16,858 participants (mean age 65 years; 56% female) from the National Survey for Health and Development (NSHD), the English Longitudinal Study of Aging (ELSA), the Swedish National Study on Aging and Care in Kungsholmen (SNAC-K), and the Rotterdam Study. We used multilevel models to examine social health in relation to cognitive capability and the rate of

karger@karger.com
www.karger.com/ger



Correspondence to:
Jane Maddock, jane.maddock@ucl.ac.uk

cognitive decline. ***Results:*** Pooled estimates show distinct relationships between markers of social health and cognitive domains, e.g., a large network size (≥6 people vs. none) was associated with higher executive function (0.17 standard deviation [SD] [95% CI: 0.00, 0.34], $I^2$ = 27%) but not with memory (0.08 SD [95% CI: −0.02, 0.18], $I^2$ = 19%). We also observed pooled associations between being married or cohabiting, having a large network size, and participating in social activities with slower decline in cognitive capability. However, estimates were close to zero, e.g., 0.01 SD/year (95% CI: 0.01, 0.02) $I^2$ = 19% for marital status and executive function. There were clear study-specific differences: results for average processing speed were the most homogenous, and results for average memory were the most heterogeneous. ***Conclusion:*** Overall, markers of good social health have a positive association with cognitive capability. However, we found differential associations between specific markers of social health and cognitive domains and differences between studies. These findings highlight the importance of examining between-study differences and considering the context specificity of findings in developing and deploying interventions.


## Introduction

Cognitive capability, the capacity to undertake the mental tasks of daily living, is an important aspect of healthy ageing [1–3]. Numerous factors across the life course can affect cognitive capability and decline [4–7]. Most research has focused on biological and medical correlates [8]. Understanding if and how aspects of an individual's social environment and well-being can affect cognitive capability may provide cost-effective complementary targets to support interventions to delay cognitive decline.

Our paper draws upon recent conceptual advancements to better understand the role of social environment and well-being in the context of dementia research [9]. Vernooij-Dassen et al. [9] use the term social health to denote the capacity to engage in meaningful social interactions, maintain supportive and fulfilling social relationships, and experience a sense of belonging and connectedness to one's community or social group. This evokes a relational concept at the individual and social-environmental level. The inclusion of the term health in this context emphasises that social well-being and the ability to adapt to challenges is a crucial component of overall health, alongside physical and mental health, in the context of dementia research.

Empirical research may encounter challenges when attempting to operationalise certain aspects of social health, like the ability to fulfil one's potential [9]. However, some aspects of the immediate social environment have been operationalised for empirical examination. Social network models have previously identified structural and functional domains of the immediate social environment [10, 11]. As Vernooij-Dassen et al. [9] outline, structure refers to the social ties between individuals within networks (e.g., marital status, social network size, and frequency of social contact), while function refers to the actual exchanges between network members (e.g., social engagement and social support). Previous research has used this classification of structural and functional domains when examining aspects of social networks with health outcomes [12–15].

There is empirical evidence that markers of social health contribute to cognitive capability, the rate of cognitive decline and risk of dementia [15–20]. A systematic review highlighting the complexities of associations between social health and cognition [18] reported that social activity, larger social networks, and greater social support were associated with higher global cognitive capability, with mixed findings for domain-specific cognitive abilities and less evidence for associations with cognitive decline. Further systematic reviews and meta-analyses focusing on cognitive decline found that poor social health (as measured by, small social network size, low social engagement, and loneliness) was associated with faster rates of decline, but there was heterogeneity between the studies and evidence for publication bias [15, 20].

A recent study using data from Sweden has shown that a composite measure of social health can offer valuable insights into the relationship between overall social health and cognitive capability [21]. However, associations between different markers of social health may differ across cognitive domains depending on the pathway evoked and the cognitive demand involved. Researchers have theorised that markers of social health can influence cognition through a combination of physiological, psychological, or behavioural pathways. For example, participating in social activities involves cognitive and mental stimulation [22]. Positive aspects of social support may act as a buffer against stress, which itself can impair cognition [23, 24]. Further, health-related behaviours can be influenced by the normative behaviours of a social environment, a phenomenon known as the social control theory [24].

For several reasons, it is difficult to ascertain the exact nature of the association between social health and cognitive capability. Firstly, the term social health refers

to a multitude of markers that have been variously defined, operationalised, assessed, and analysed. Secondly, cognitive capability has often been examined as a composite measure in relation to markers of social health, and differential effects between specific cognitive domains (e.g., memory, processing speed) observed in some previous studies are worth exploring [18, 25]. Thirdly, results of earlier meta-analyses may have overlooked important between-study differences by combining results from different types of studies that measured and analysed data in different ways.

The current study sought to overcome some of these challenges. We used a clear framework to operationalise markers of social health and examined both specific cognitive domains (memory, executive function, and processing speed) and global or composite cognitive function where possible. Importantly, we applied integrative data analyses across four large European longitudinal studies. Integrated data analysis involves coordination of measurement and analytical protocols between independent studies which maximises comparability between results, while accounting for study-specific strengths and design features [26]. The studies and countries chosen are part of the SHARED consortium (https://www.shared-dementia.eu/), sampled from the general population, containing the necessary exposure and outcome measures and multiple time points required to examine decline, and were homogeneous enough to pool findings and analyse replication. This paper further builds on a recent study that examined a similar question in different studies involving fewer time points and greater heterogeneity of populations sampled from the COSMIC consortium [27]. Our overall aim was to examine whether social health markers measured at baseline are associated with differences in cognitive capability and the rate of cognitive decline over an 11- to-18-year period among older adults and compare results across studies.

## Materials and Methods

*Participants*

Participants came from four European population-based longitudinal studies located in the UK, Sweden, and the Netherlands: the National Survey of Health and Development (NSHD) [28] and the English Longitudinal Study of Ageing (ELSA) [29] in the UK, the Swedish National Study on Aging and Care-Kungsholmen (SNAC-K) [30] in Sweden, and the Rotterdam Study [31] in the Netherlands (online suppl. Fig. 1 for details; for all online suppl. material, see https://doi.org/10.1159/000531969). Briefly, NSHD is a British birth cohort where participants of the same age have been followed up 24 times since birth in 1946. Cognitive capability was measured in 1999, 2006–2010, and 2014–2015 when participants were aged 53, 60–64, and 68–69 years. ELSA is a study of adults aged 50 years and over living in private households in England. The original sample was contacted in 2002–2003 and participants are followed up every 2 years. Cognitive capability was measured in waves 1–9. SNAC-K is a study of people aged 60 years and older living at home or in nursing homes in Kungsholmen (central Stockholm). Baseline assessment took place between 2001 and 2004. Those aged between 60 and 72 years were followed every 6 years, and those aged 78 years and older every 3 years, up until a maximum follow-up period of 12 years. The Rotterdam Study is a study of adults aged 45 years or older that began in 1990–1993. Participants are followed up every 4 years. For each of these studies, our analytical sample consisted of participants with information on at least one social health variable at baseline and cognitive capability from at least two time points in one domain. Participants with dementia at baseline and those with missing covariate data (<9%) were excluded (see online suppl. File 1). Table 1 outlines the analytical sample size for each study.

*Measures*

We harmonised all measures across studies as much as possible. We provide full details of the harmonisation process and the original questions in online supplementary File 1. We briefly describe variables used in analyses in the following sections. Note that some studies did not include every outcome and exposure of interest (Table 1).

*Cognitive Capabilities*

We included three tests of cognitive capability that were similarly measured across studies. Memory was assessed in all cohorts using an immediate word list recall. In ELSA, SNAC-K, and the Rotterdam Study, executive function was assessed using a semantic verbal fluency test. Processing speed was assessed in NSHD and ELSA using a letter cancellation task, a digit cancellation task in SNAC-K and letter-digit substitute task in the Rotterdam Study. We standardised each test across time points and within studies on a common standard deviation (SD)-based scale with mean of 0 and SD of 1. This enhances comparability of estimates between studies while allowing examination of changes over time within studies. Total follow-up time in each study ranged between 11 and 18 years (online suppl. File 1 provides detailed follow-up time for each cognitive outcome within each study). Study-specific global or composite cognitive scores were constructed for sensitivity analyses (full details in online suppl. File 1).

*Social Health: Structural and Functional Markers*

We focus on the social environment aspects of social health. As outlined in the introduction, we distinguish between structural and functional domains. Structural social health markers at baseline included: marital or cohabitation status (married or cohabiting vs. unmarried and alone); social network size (1–2 people, 3–6 people, or ≥6 people vs. none); contact frequency (more than once a year, about once to twice a month, weekly or more than twice a month, at least 2 to 3 times per week vs. never or almost never). Functional social relationship variables included: participation in social activities (moderate or high vs. low); perceived received positive support (coded as a standardised score and categorised as −1 SD to 0 SD, 0 to 1 SD and >1 SD vs. <−1 SD); and perceived received negative support (coded as a standardised score and categorised as −1 SD to 0 SD, 0 to 1 SD and >1 SD vs. <−1 SD). We categorised the

**Table 1.** Descriptive statistics across studies

| | NSHD | ELSA | SNAC-K | Rotterdam study |
|---|---|---|---|---|
| *N* | 2,109 | 8,460 | 1,892 | 4,397 |
| Total follow-up time, years | 16 | 18 | 12 | 11 |
| Age at baseline, years (SD) | 53.4 (0.17) | 64.3 (9.8) | 71.4 (9.6) | 72.2 (7.3) |
| *Demographics* | | | | |
| Female, % (*n*) | 53 (1,116) | 55 (4,625) | 62 (1,182) | 56 (2,466) |
| Occupational social class, % (*n*) | | | | |
| Manual | 33 (699) | 44 (3,732) | 15 (279) | 27 (1,193) |
| Non-manual | 67 (1,410) | 56 (4,728) | 85 (1,613) | 73 (3,204) |
| Education, % (*n*) | | | | |
| Lower | 39 (820) | 47 (4,018) | 12 (222) | 11 (484) |
| Secondary | 50 (1,053) | 28 (2,390) | 49 (928) | 43 (1,909) |
| Higher | 11 (236) | 24 (2,052) | 39 (742) | 46 (2,004) |
| IADL, % (*n*) | | | | |
| None | N/A | 93 (7,841) | 91 (1,722) | 55 (2,436) |
| At least one | | 7 (619) | 9 (170) | 45 (1,961) |
| Vascular-related health conditions, % (*n*) | | | | |
| None | 96 (2,029) | 87 (7,334) | 74 (1,397) | 67 (2,954) |
| At least one | 4 (80) | 13 (1,126) | 26 (495) | 33 (1,443) |
| *Structural social health variables* | | | | |
| Marital/cohabitation status, % (*n*) | | | | |
| Unmarried and alone | 9 (189) | 24 (2,011) | 48 (907) | 31 (1,386) |
| Married or cohabiting | 91 (1,920) | 76 (6,449) | 52 (983) | 68 (3,010) |
| Missing | 0 | 0 | 0.001(2) | 0.02 (1) |
| Network size, % (*n*) | | | | |
| None | 4 (78) | 2 (145) | 1 (21) | N/A |
| 1–2 people | 13 (269) | 12 (975) | 14 (273) | |
| 3–6 | 30 (626) | 39 (3,330) | 42 (799) | |
| ≥6 | 54 (1,136) | 40 (3,443) | 37 (690) | |
| Missing | 0 | 7 (567) | 6 (109) | |
| Contact frequency, % (*n*) | | | | |
| Never or almost never | 0.9 (18) | 1 (49) | 0.6 (11) | N/A |
| More than once a year | 8 (161) | 15 (1,271) | 10 (181) | |
| About once to twice a month | 12 (261) | 55 (4,618) | 45 (853) | |
| Weekly or more than twice a month | 48 (1,013) | 20 (1,722) | 36 (682) | |
| At least two to three times per week | 29 (620) | 4 (311) | 5 (97) | |
| Missing | 2 (36) | 6 (489) | 4 (68) | |
| *Functional social health variables* | | | | |
| Participation in social activities, % (*n*) | | | | |
| Low (≤1 activity) | N/A | 21 (1,751) | 3 (56) | N/A |
| Moderate (2–3 activities) | | 33.3 (2,816) | 9 (160) | |
| High (≥4 or more activities) | | 46 (3,893) | 82 (1,557) | |
| Missing | | 0 | 6 (119) | |
| Positive support* | | | | |
| Range | 0–9 | 0–9 | 0–10 | 0–10 |
| Median (IQR range) | 6.0 (5.0–8.0) | 7.0 (5.8–8.0) | 10 (9–10) | 10 (9–10) |
| Missing, % (*n*) | 3 (70) | 6 (516) | 4 (91) | 0.1 (5) |
| Negative support* | | | | |
| Range | 0–9 | 0–9 | N/A | N/A |
| Median (IQR range) | 8.0 (6.0–8.0) | 7.3 (6.3–8.0) | | |
| Missing, % (*n*) | 3 (70) | 6 (479) | | |

**Table 1** (continued)

| | NSHD | ELSA | SNAC-K | Rotterdam study |
|---|---|---|---|---|
| *Cognitive capability* | | | | |
| Memory* | | | | |
| Range | 0–15 | 0–10 | 0–16 | 0–15 |
| At baseline, mean (SD) | 6.0 (2.0) | 5.6 (1.7) | 7.3 (2.3) | 6.8 (2.0) |
| **Rate of decline, β (95% CI) | −0.025 (−0.028, −0.022) | −0.03 (−0.04, 0.03) | −0.049 (−0.054, −0.045) | −0.007 (−0.011, −0.003) |
| Executive function* | | | | |
| Range | N/A | 0–50 | 2–50 | 2–47 |
| At baseline, mean (SD) | | 19.9 (6.2) | 22.0 (6.3) | 20.6 (5.3) |
| **Rate of decline, β (95% CI) | | −0.01 (−0.02, −0.01) | −0.046 (−0.050, −0.042) | −0.021 (−0.025, −0.017) |
| Processing speed* | | | | |
| Range | 0–780 | 0–780 | 2–34 | 1–55 |
| At baseline, mean (SD) | 283.9 (73.9) | 309.7 (94.3) | 18.0 (4.1) | 26.8 (7.0) |
| **Rate of decline, β (95% CI) | −0.020 (−0.023, −0.017) | −0.03 (−0.04, −0.03) | −0.045 (−0.049, −0.041) | −0.056 (−0.059, −0.053) |

Full details on how each variable was coded are in the online supplementary File 1. IADL: Instrumental Activities of Daily Living. *Raw scores presented for descriptive purposes; standardised scores used in analyses. For negative support, higher scores indicate less feelings of negative support. **Based on standardised outcomes from mixed-effects multilevel models, e.g., −0.03 indicates a 0.03 SD lower cognitive score per year.

standardised scores for support since positive support in the Rotterdam Study and SNAC-K had a skewed distribution, i.e., most participants reported very high levels of support (Table 1).

*Confounders*

We defined baseline confounders a priori (based on prior literature and knowledge): sex (female vs. male); occupational-based social class (non-manual vs. manual occupation); education (secondary or higher vs. lower); impairment in instrumental activities of daily living (at least one vs. none); and vascular-related health conditions excluding hypertension, e.g., diabetes, stroke, heart disease. Mental health may be considered a mediator or confounder; therefore, we have not included it in our main models. However, we adjusted for standardised mental health scores in sensitivity analyses. For details of mental health measurement in each study, see online supplementary File 1.

*Analyses*

We applied integrated data analysis in this paper [26]. Following harmonisation of all variables after agreement between authors (online supplementary File 1), JM wrote an exemplar script using Stata 17 and an analyst within each country (J.M., F.G., and F.J.W.) adapted and ran the script for their study. Within each study, change in each cognitive domain and individual social health marker was modelled using linear mixed-effects models with random intercepts, slopes, and time centred on baseline date and coded as years. This method accounts for within-person correlation between repeated cognitive scores over time. We used two main approaches when applying these linear mixed-effects models. Firstly, we included the social health marker and time in the model and interpreted the coefficient for social health as the association with levels of cognitive capability on average over time, hereinafter referred to as average cognitive capability. Secondly, we included an interaction term between the social health marker and time and interpreted the coefficient as association with rate of cognitive decline.

We applied three levels of adjustment to the specified linear mixed-effects models. After estimating associations adjusted for sex and age at baseline, we adjusted first for socio-demographic information (social class and education) and second for health-related information at baseline (instrumental activities of daily and vascular-related health conditions). We decided to use three levels of adjustment as the identity of key confounding variables remains uncertain. We also wanted to explicitly show the effect of health-related information above socio-demographic information on the observed associations. It is plausible that associations between social health and cognitive capability might vary by sex [32, 33]; therefore, each model was repeated stratifying by sex.

Within-study analyses were conducted using Stata. Overall and stratified results from each study were sent to J.M. who pooled the estimates using random effects meta-analysis with restricted maximum likelihood (*meta* command in Stata 17). Sex-differences were tested using the subgroup option in the *meta* command in Stata 17. We report heterogeneity using the $I^2$ statistic, where 0% indicates estimates were similar across studies and values closer to 100% represent greater heterogeneity. Figures 1 and 3 were created using R.

Sensitivity Analyses

In sensitivity analyses, we examined if associations were robust to additional adjustment for depressive symptoms. We also repeated analyses with a global or composite measure of cognitive function.

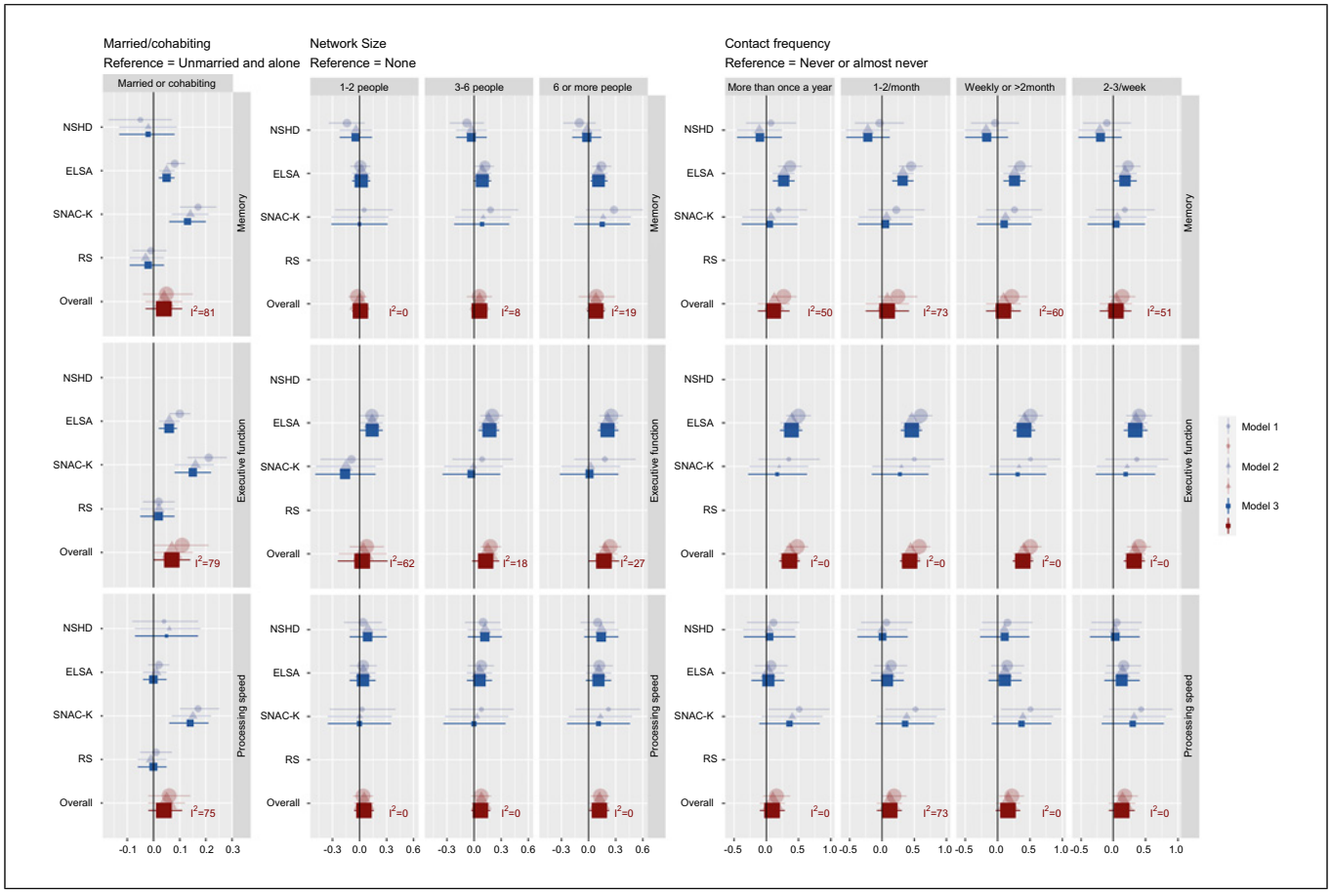



**Fig. 1.** Associations between baseline structural social relationship variables and standardised cognitive capability on average over time. Size of the estimate markers represents study weight. See online supplementary File 2 for this information in table format. Model 1: sex, age at baseline. Model 2: sex, age at baseline, social class, education. Model 3: sex, age at baseline, social class, education, Instrumental Activities of Daily Living, and vascular-related health conditions.

## Results

Our analyses included 16,858 participants (mean age 65.3 years; 56% female) with follow-up times ranging from 11 to 18 years across four studies (Table 1). Most participants came from a non-manual social class, particularly in SNAC-K (85%). The proportion of married or cohabiting participants was somewhat higher in the two UK studies (NSHD and ELSA) than in SNAC-K and the Rotterdam Study, which included on average older participants. Most people had extensive social networks (>37% had more than six people in their network) and reported having high positive support (median ranged from 6 to 10 across studies where scales ranged from 0–9 to 0–10). A large proportion of SNAC-K participants (82%) had high levels of social participation. Markers of social health were weakly correlated within studies ($p < 0.03$, online suppl. File 2). Cognitive domains were moderately correlated within studies ($r > 0.3$) except for memory and processing speed in NSHD, ELSA, and SNAC-K ($r < 0.3$, online suppl. File 2). We observed decline in all cognitive domains across all studies ranging from −0.001 to −0.06 SD per year (Table 1).

Results for the linear mixed-effects models are discussed in the following sections. We organise results by cognitive domain. We report results within each subsection for structural social health markers and then functional social health markers. We focus on the fully adjusted models as we did not observe large differences in results between different adjustment levels (online suppl. File 2). We did not observe any statistically significant sex-differences, however when stratifying by sex, estimates tended to be stronger for married males than for married females (all sex-stratified results are found in online suppl. File 2).

## Memory

*Structural Social Health Markers*

Being married or cohabiting was associated with 0.05 (95% CI: 0.02, 0.08) and 0.13 (95% CI: 0.06, 0.2) SD higher average memory scores in ELSA and SNAC-K, respectively. As seen in Figure 1 and online supplementary file 2, estimates were opposite in sign and confidence intervals crossed the null in NSHD and the Rotterdam Study, and a relationship was not observed in the pooled results (0.04 [95% CI: −0.03, 0.11]). This heterogeneity was reflected in the high $I^2$ statistic (81%). We observed a similar degree of heterogeneity ($I^2$ = 76%) when examining how being married or cohabiting associated with the rate of memory decline. As shown in Figure 2 and online supplementary file 2, clear associations were observed in ELSA and SNAC-K where being married or cohabiting at baseline was associated with a slower rate of memory decline, although the effect size was small (e.g., 0.02 SD/year 95% CI: 0.01, 0.02 in ELSA).

There were no clear associations between having a large network size and average memory scores in NSHD or SNAC-K (Fig. 1 and online suppl. file 2). In ELSA, only a very large network size (≥6 people compared with none) was associated with 0.11 (95% CI: 0.02, 0.21) SD higher memory score. The Rotterdam Study did not have information about network size. Overall, results across studies were less heterogeneous than for marital or cohabitation status ($I^2$ = ≤19%) with effect sizes being similar in ELSA and SNAC-K and opposite in sign in NSHD (Fig. 1 and online suppl. file 2). Results were also relatively homogeneous across studies when examining the rate of decline in memory ($I^2$ = ≤0.09%). Pooled results from NSHD, ELSA, and SNAC-K suggest that having a larger network size was associated with a slower rate of decline in memory (e.g., 0.02 SD/year [95% CI: 0.01, 0.03] for 3–6 people compared with none).

Overall pooled estimates suggest no association between frequent social contact and average memory, however, results were heterogenous ($I^2$ = ≥50%). In ELSA, having frequent social contact was associated with higher memory scores (Fig. 1; online suppl. file 2). Estimates in SNAC-K were smaller and confidence intervals crossed the null and were opposite in sign in NSHD. The Rotterdam Study did not have information about social contact frequency. Results from analyses examining decline in memory were less heterogenous ($I^2$ = ≤0.08%, Fig. 2; online suppl. file 2). Overall, there was no evidence that frequency of social contact was associated with the rate of memory decline.

*Functional Social Health Markers*

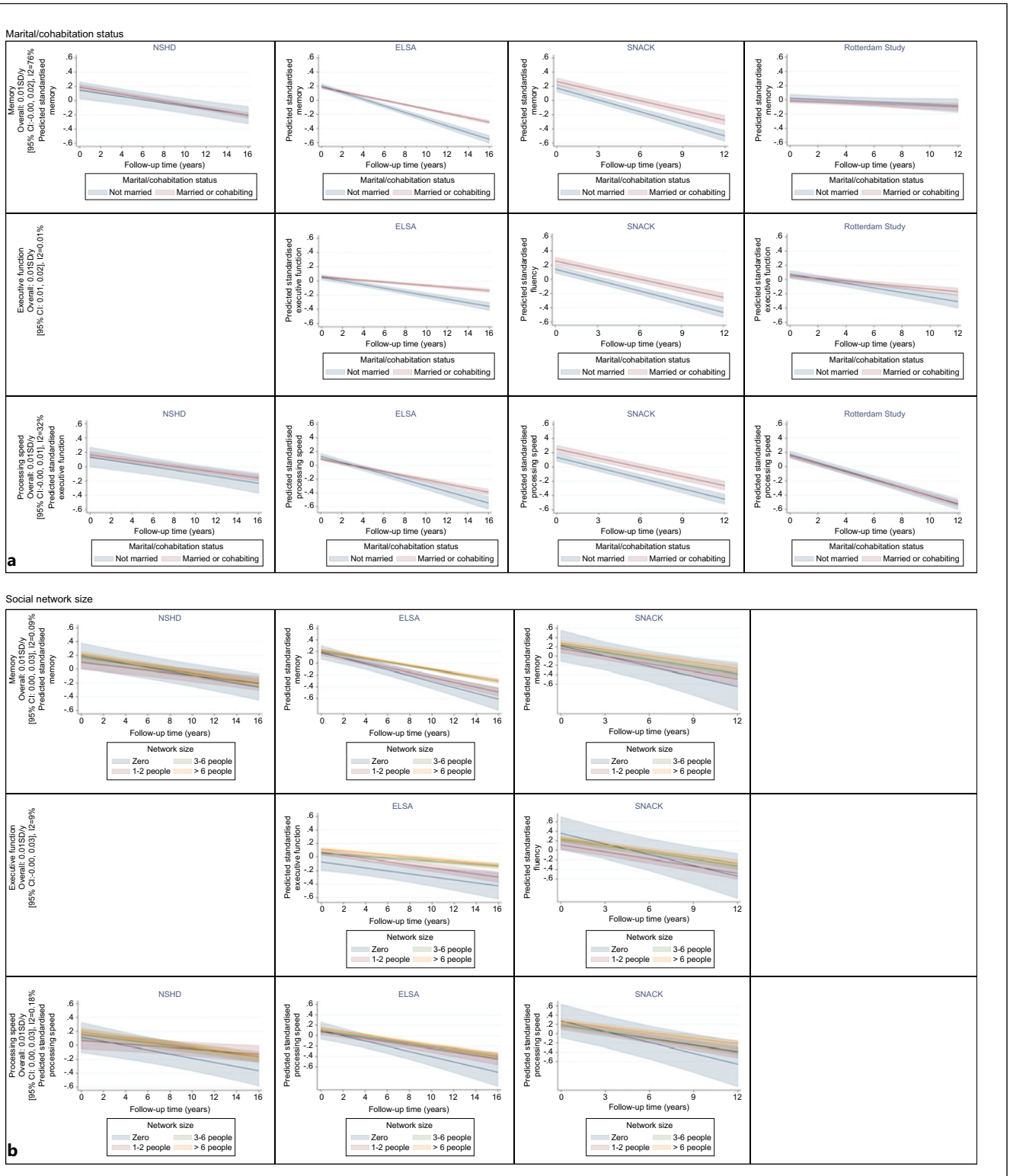
Among the two studies with relevant information (ELSA and SNAC-K), participation in social activities was associated with higher average memory scores (Fig. 3 and online suppl. file 2). Effect sizes were similar across the two studies ($I^2$ = ≤8%), with pooled estimates indicating that participating in ≥4 social activities was associated with 0.32 (95% CI: 0.28, 0.35) SD higher average memory score compared with not participating in any social activities. Effect sizes were also similar ($I^2$ = ≤0.09%) across the studies when examining rate of memory decline (Fig. 4 and online suppl. file 2). Pooled estimates suggest a slower rate of decline in memory for those participating in ≥4 social activities compared with none (0.01 SD/year 95% CI: 0.01, 0.02, $I^2$ = 0%).

Pooled estimates in Figure 3 and online supplementary file 2, show that compared with participants who reported having low levels of positive social support (i.e., <−1 SD) those in the middle of the distribution (between −1 SD to 0 SD and 0 SD to 1 SD) had higher average memory. Estimates were relatively consistent across all studies ($I^2$ =≤ 0.02%). However, results were more heterogeneous ($I^2$ = 89%) when comparing individuals reporting high levels of positive support (>1 SD vs. <−1 SD). Participants from the Rotterdam Study did not contribute to this due to the distribution of positive social support in the sample. Unexpectedly, estimates were smaller compared with people in the middle of the distribution for ELSA and SNAC-K. There was no strong evidence for an association between positive social support and rate of memory decline across studies ($I^2$ =< 0.02%, Fig. 4; online suppl. File 2).

Only the UK studies (ELSA and NSHD) had information about perceived negative aspects of social support. Overall pooled results show that those in the middle of the distribution (i.e., −1 SD to 0 SD and 0 SD to 1 SD) had 0.12 (95% CI: 0.08, 0.16) and 0.16 (0.11, 0.20) SD higher average memory scores compared with participants reporting high negative social support (i.e., <−1 SD) (Fig. 3 and online suppl. file 2). Estimates attenuated to 0.05 (95% CI: 0.00, 0.10) for those in reporting low levels of negative social support (>1 SD vs. < −1 SD). Estimates were similar between studies ($I^2$ < 11%). There was no clear evidence for an association between negative social support and rate of memory decline across studies (Fig. 4; online suppl. file 2).

## Executive Function

NSHD did not have information about executive function over the follow-up period.

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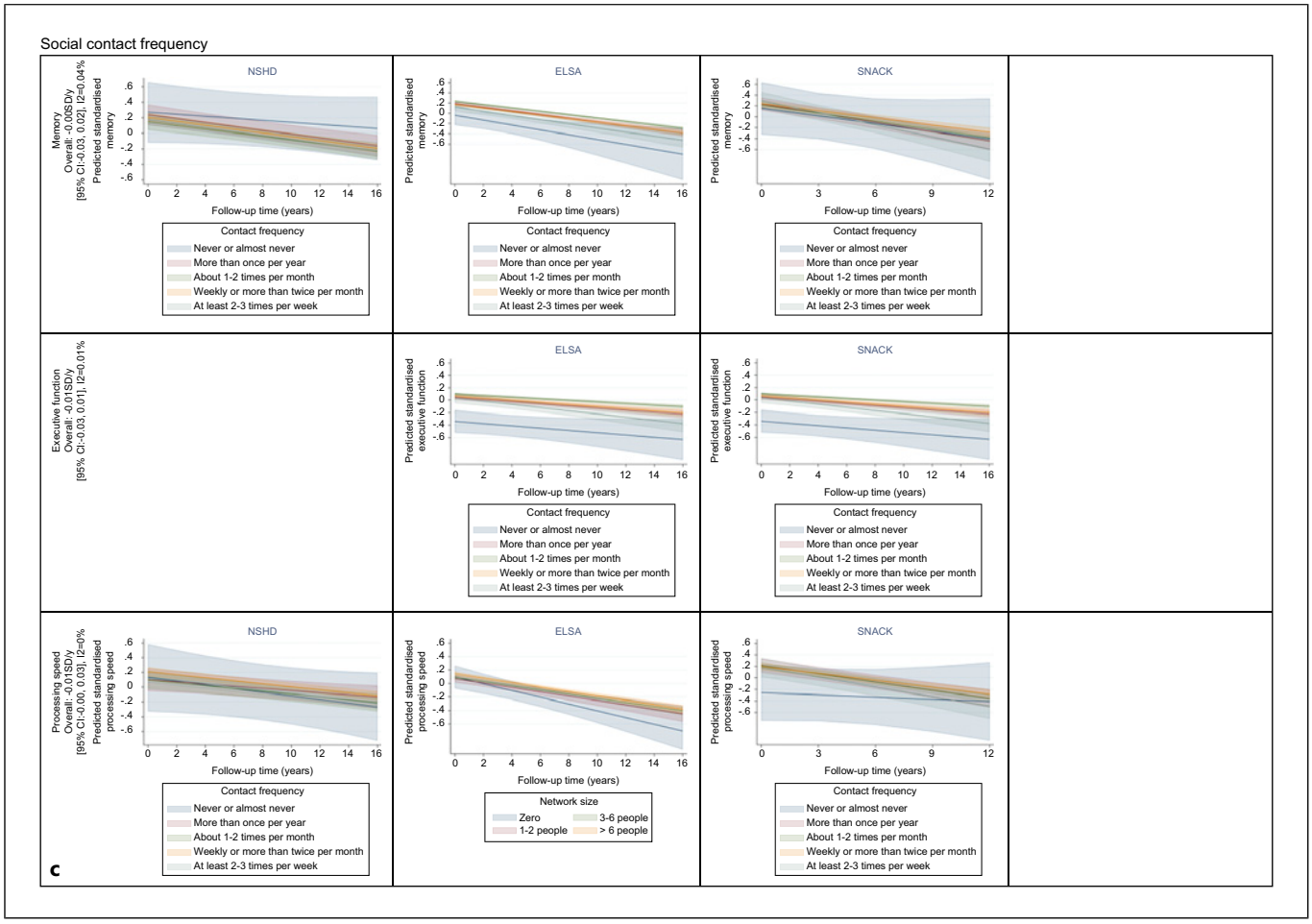



**Fig. 2.** Association between (**a**) marital and cohabitation status (**b**) social network size (**c**) social contact frequency and decline in cognitive function in each study. Estimates are adjusted for sex, age at baseline, social class, education, Instrumental Activities of Daily Living, vascular-related health conditions. Overall result reflects the meta-analysed value for decline. Where there is more than one exposure category, the overall value reflects the result from the final category.

*Structural Social Health Markers*

Being married or cohabiting was associated with higher average executive function scores in ELSA and SNAC-K, with the largest estimate found in SNAC-K (0.15 SD [95% CI: 0.08, 0.22]) as seen in Figure 1 and online supplementary file 2. The smallest estimate was found in the Rotterdam Study where the confidence interval crossed the null (0.02 [95% CI: −0.05, 0.08]). The difference in effect sizes was reflected in the high $I^2$ statistic (79%). Being married or cohabiting was also associated with slower rate of decline in executive function (0.01 SD/year [95% CI: 0.01, 0.02]) with similar results observed across all studies ($I^2$ = 0.01%) as shown in Figure 2 and online supplementary File 2.

Only ELSA and SNAC-K had information on network size and executive function. A larger network size was associated with higher average executive function in ELSA, but estimates were in the opposite direction and crossed the null for SNAC-K ($I^2 \geq 18\%$, Fig. 1 and online suppl. File 2). There was little evidence for an association between network size and the rate of decline in executive function, with results being slightly more homogenous ($I^2 = \leq 20.9\%$) than for average executive function (Fig. 2; online suppl. File 2).

Frequent social contact was associated with higher average executive function (Fig. 1; online suppl. File 2). Only ELSA and SNAC-K contributed to these analyses and findings were similar across the studies ($I^2$ = 0%), although estimates were larger and more precise for ELSA. The largest pooled estimate was observed for participants who had social contact once to twice a month compared with never or almost never (0.43

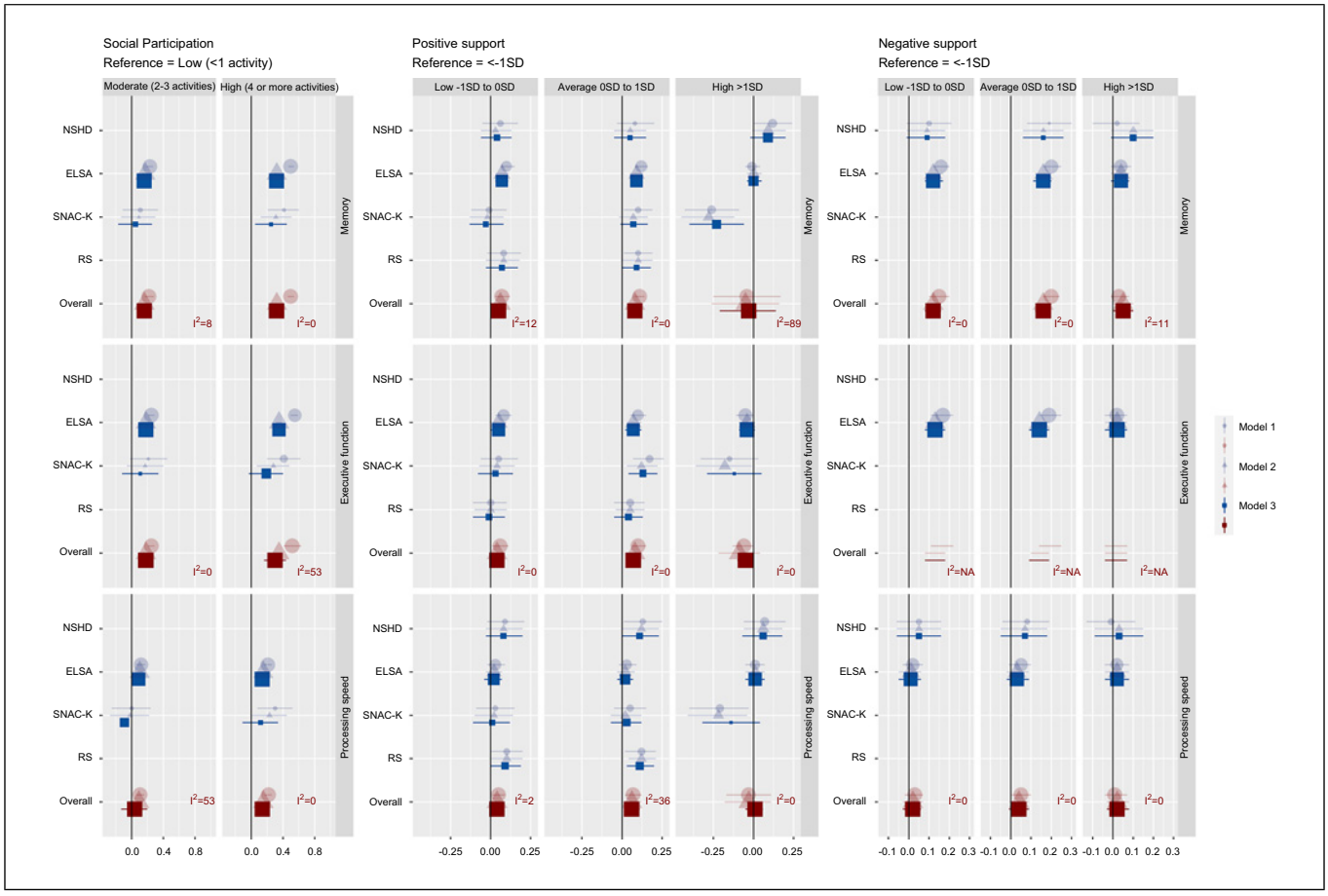



**Fig. 3.** Associations between baseline functional social relationship variables and standardised cognitive capability on average over time. Size of the estimate markers represents study weight. See online supplementary File 2 for this information in table format. Model 1: sex, age at baseline. Model 2: sex, age at baseline, social class, education. Model 3: sex, age at baseline, social class, education, Instrumental Activities of Daily Living, and vascular-related health conditions.

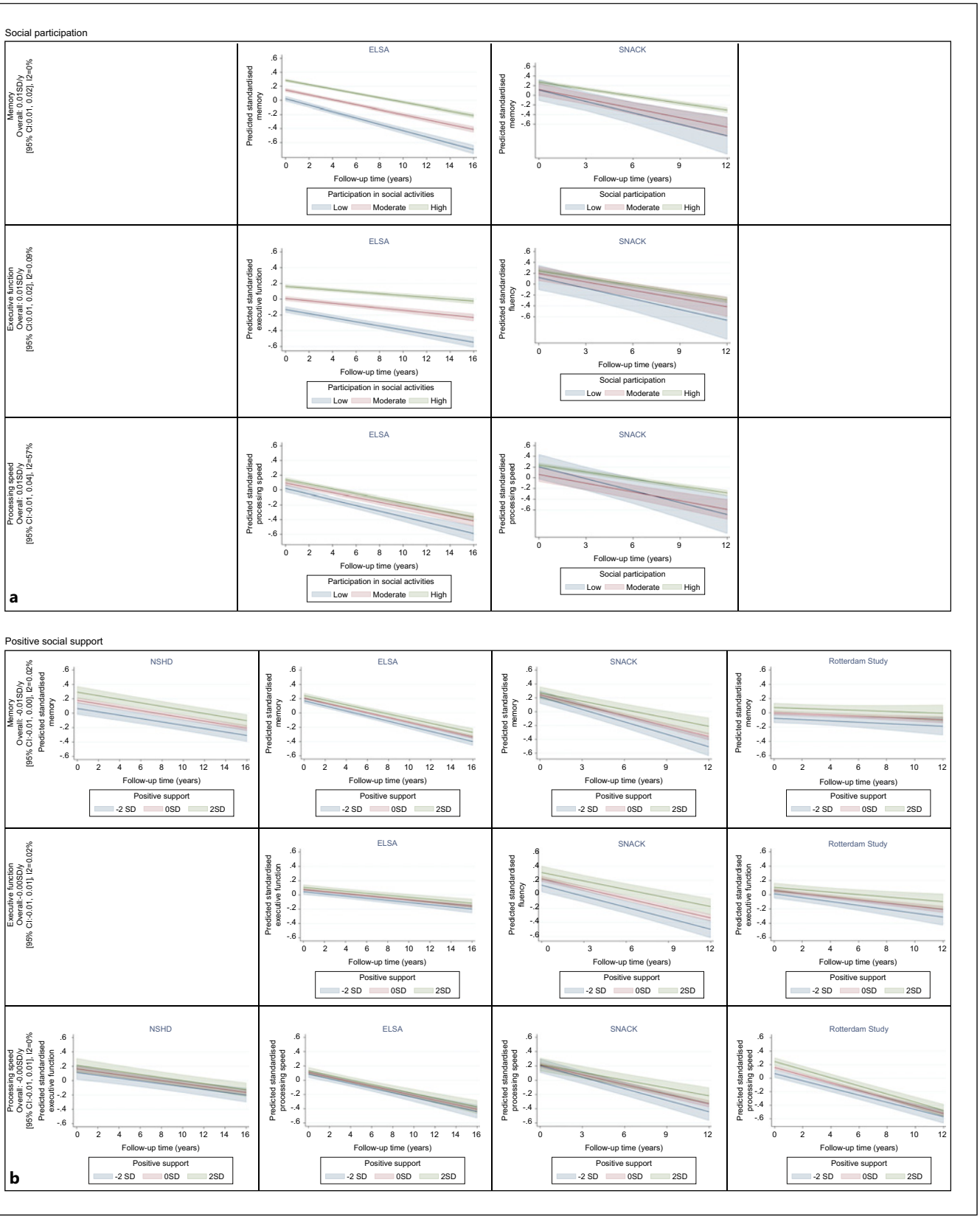
[95% CI: 0.28, 0.59]). We did not observe associations between frequency of social contact and the rate of decline in executive function ($I^2$ = ≤0.06%).

*Functional Social Health Markers*

Pooled results showed participating in social activities was associated with higher average executive function (e.g., 0.30 [95% CI: 0.16, 0.44] for ≥4 activities vs. none, Fig. 3, online suppl. File 2). ELSA and SNAC-K contributed to these analyses and results were homogeneous ($I^2$ = 0%), however, estimates from ELSA tended to be larger and more precise. More social participation at baseline slowed the rate of decline in executive function (e.g., 0.01 [95% CI: 0.01, 0.02], $I^2$ = 0.09%) (Fig. 4 and online suppl. File 2).

Pooled results in Figure 3 showed that compared with those reporting low levels of perceived positive social support, those in the middle of the distribution (i.e., −1 SD to 0 SD and 0 SD to 1 SD) had higher average executive function (0.04 [95% CI: 0.00, 0.08] and 0.07 [95% CI: 0.04, 0.11], $I^2$ < 0.01%). The association was no longer observed when comparing those in the highest end of the distribution (e.g., >1 SD) with those in the lowest (<−1 SD): −0.05 (95% CI: −0.1, 0.00, $I^2$ = 0.01%). Similarly, those in the middle of the distribution at baseline had a slower decline in executive function, which was not observed among those at the top of the distribution (Fig. 4; online suppl. File 2).

ELSA was the only study with information on perceived negative social support and executive function. Results from this study showed that those in the middle of the distribution had higher average executive function which attenuated for those in the top end of the distribution (Fig. 3). While there was no evidence for perceived negative social support at baseline




*(Figure continued on next page.)*

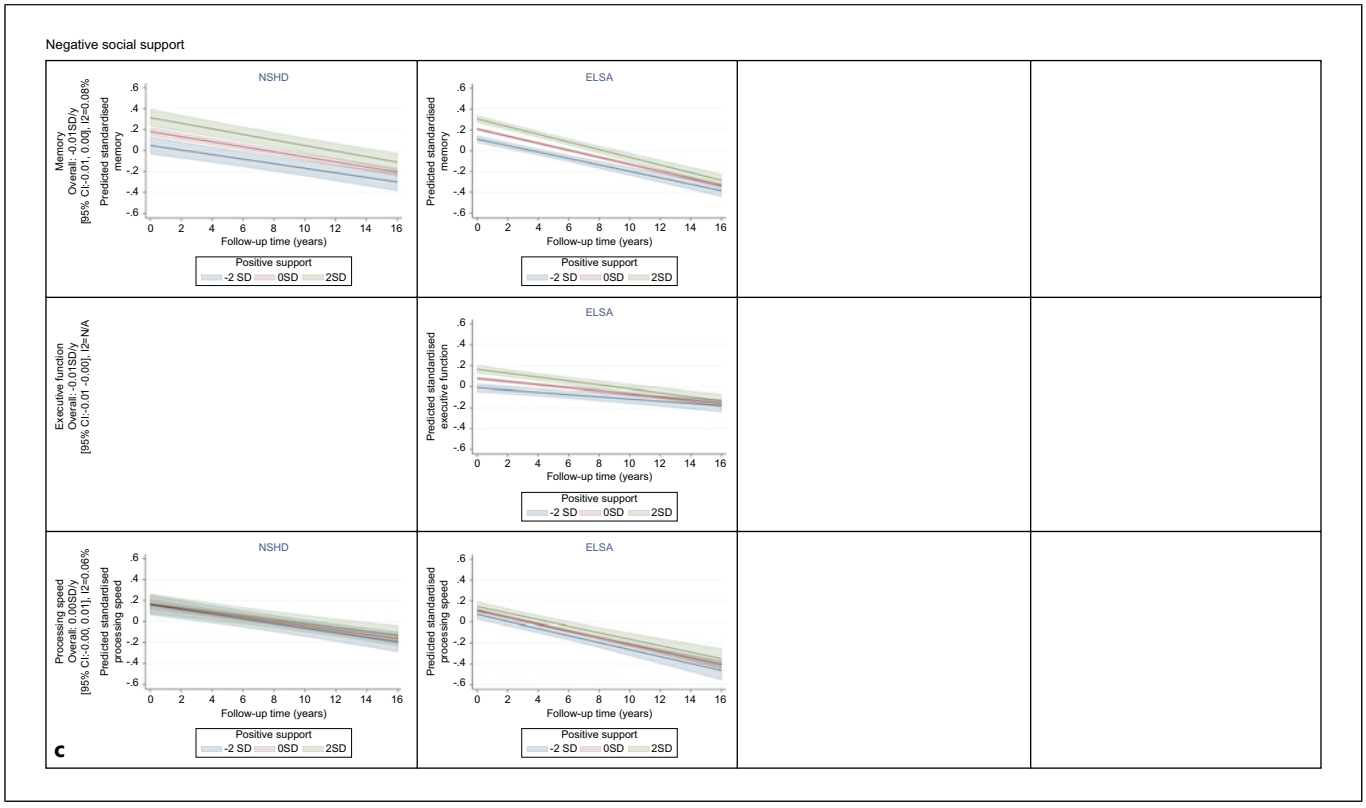



**Fig. 4.** Association between (**a**) social participation (**b**) positive social support (**c**) negative social support and decline in cognitive function in each study. Estimates are adjusted for sex, age at baseline, social class, education, Instrumental Activities of Daily Living, vascular-related health conditions. Overall result reflects the meta-analysed value for decline. Where there is more than one exposure category, the overall value reflects the result from the final category.

and the rate of decline in executive function, those in the top end of the distribution tended to have a faster decline (−0.01 SD/year 95% CI: −0.01, −0.00, Fig. 4; online suppl. File 2).

## Processing Speed

*Structural Social Health Markers*

Being married or cohabiting was associated with higher average processing speed scores in SNAC-K only (0.14 SD [95% CI: 0.06, 0.21], Fig. 1). Other estimates crossed the null and overall, results were heterogenous ($I^2 = 75\%$). Results were also slightly heterogeneous for the rate of decline ($I^2 = 32\%$), with ELSA showing the clearest associations between being married or cohabiting at baseline and 0.01 SD/year (95% CI: 0.00, 0.02) slower rate of decline in processing speed with small estimates from other studies (Fig. 2; online suppl. File 2).

There was some evidence for pooled associations between having large network size (i.e., ≥6 people) and average processing speed (Fig. 1, $I^2 \leq 0.01\%$). Confidence intervals from individual studies examining network size and decline in processing speed cross the cross the null, however, pooled analyses found having a larger network size was associated with slower rate of decline in processing speed (Fig. 2, e.g., 0.01 SD/year [95% CI: 0.00, 0.03] for ≥6 people vs. none, $I^2 = 0.18\%$)

There was no evidence for associations between frequency of social contact and average processing speed scores among included studies (Fig. 1, $I^2 = 0\%$). Similarly, there was no evidence for frequency of social contact and rate of decline in processing speed (Fig. 2, $I^2 \leq 0.02\%$).

*Functional Social Health Markers*

Only ELSA and SNAC-K contributed to analyses examining social participation and processing speed. In ELSA, high social participation (≥4 activities) was associated with 0.14 SD (95% CI: 0.03, 0.19) higher average processing speed scores. Estimates were

smaller and confidence intervals crossed the null for SNAC-K (Fig. 3; online suppl. File 2). We did not observe that social participation at baseline was associated with the rate of decline of processing speed (Fig. 4; online suppl. File 2); however, this was heterogenous for those participating in ≥4 activities with SNAC-K showing a 0.03 SD/year (95% CI: 0.00, 0.06) slower rate of decline that was not observed in ELSA.

While confidence intervals for estimates in all studies, expect for the Rotterdam Study, crossed the null, pooled results indicated that those in the middle of the distribution for perceived positive social support had higher average processing speed (Fig. 3; online suppl. File 2, $I^2$ < 36%). Like memory and executive function, this attenuated for those at the highest end of the distribution. There was no clear evidence for associations between perceived social support and rate of decline in processing speed, however, for those in the middle of the distribution, results were heterogenous ($I^2$ ≥ 59%, Fig. 3l; online suppl. File 2).

We did not observe evidence for associations between perceived negative social support and average processing speed, which only included the two UK studies (Fig. 3; online suppl. File 2). Those in the middle of the distribution tended to have slower decline in processing speed, but this was not observed for those in the highest (Fig. 4; online suppl. File 2).

### Sensitivity Analyses

Overall, additional adjustment for depressive symptoms slightly attenuated estimates but did not change general interpretation (online suppl. File 2). Estimates for the global or composite measures of cognitive capability were of a similar magnitude as the domain-specific estimates and showed similar heterogeneity between studies (online suppl. File 2). Being married or cohabiting was associated with 0.07 SD (95% CI: 0.01, 0.15, $I^2$ = 81%) higher average composite or global score and slower rate of decline (0.01 SD/year [95% CI: 0.00, 0.02], $I^2$ = 84%) decline per year, with the average association being stronger for males (0.15 SD [95% CI: 0.06, 0.25]) than for females (0.03 SD [95% CI: −0.05, 0.11]; $p$ value for subgroup difference = 0.05). Having a larger network size (≥6 people vs. none) was associated with a 0.15 SD (95% CI: 0.05, 0.24, $I^2$ = 0%) higher average composite or global score and a slower rate of decline (0.02 SD/year [95% CI: 0.01, 0.03], $I^2$ = 0.02%). We observed no associations between frequency of contact and the composite or global score of cognitive capability. More frequent participation in social activities was associated with 0.37 SD (0.32, 0.41, $I^2$ = 0.01%) higher average composite score and a slower rate of decline (0.02 SD/year [95% CI: 0.01, 0.02], $I^2$ = 0.09%). Compared with participants who reported having low levels of positive social support or high levels of negative support, those in the middle of the distribution (between −1 SD and 1 SD) had a higher average composite or global score (e.g., 0.09 [95% CI: 0.05, 1.12, $I^2$ = 0.2%] for positive support and 0.14 [95% CI: 0.09, 0.18, $I^2$ = 0.01%] for negative support), but we did not observe associations with high positive support and low negative support, nor with rate of decline for either of the support measures.

## Discussion

We applied an integrated analysis approach to data from participants aged 50 years and older from four longitudinal European studies. Overall, we found that markers of good social health were associated with higher average cognitive capability and slower rate of cognitive decline. However, despite harmonisation of measures and application of the same analytic protocol, associations varied across studies, exposure type, and cognitive domains.

When focusing on pooled estimates from our study, findings support conclusions from previous research suggesting distinct relationships between markers of social health and cognitive domains [18, 27]. Findings for structural markers of social health (marital or cohabitation status, social network size, contact frequency) were mixed — all were associated with executive function, and network size was also associated with processing speed. We did not observe associations between structural markers of social health and memory. We did find associations between functional aspects of social health (social participation, perceived received positive and negative social support) across all cognitive domains (except for negative social support and processing speed). This result may reflect distinct mechanisms underlying the associations between social health and cognitive capability.

Active engagement and quality of interpersonal relationships are key components in our definition of functional aspects of social health. While engagement may be beneficial for cognitive capability through cognitive stimulation [34], positive interpersonal relationships may act as a buffer against stressful life events [23, 24]. This implies that functional aspects of social health

may affect different aspects of cognitive capability through several pathways. Our research also suggests that social health may have distinct effects on specific cognitive domains. We found that the functional aspects of social health have a universal effect across all cognitive domains, while the structural aspects of social health may not be as relevant to aspects of memory.

One specific result requires further discussion. For both positive and negative aspects of perceived received social support, we observed non-linear relationships. Compared to those in the lower end of the distribution (<−1 SD), those in the middle scored higher on cognitive tests, but this effect attenuated for the higher part of the distribution (>1 SD). There are several potential explanations for this. There may be a threshold for the beneficial effects of social support on cognitive capability. Alternatively, this could reflect reverse causality; participants with lower cognitive capability at baseline may already receive higher social support at baseline.

We also observed pooled associations between marital or cohabitation status, network size, and social participation with the rate of decline in cognitive capability; however, estimates were close to zero. It is possible that, although the follow-up time in our study ranged from 11 to 18 years, it may take longer for many participants, especially those who were younger at baseline, to experience the effect of social health on cognitive decline. Alternatively, specific social health markers may convey a protective effect on the baseline level of cognitive capability, namely by affecting its peak levels during the life course, but less on the rate of its decline. US researchers observed a similar association between education and initial levels of global cognition but not with the rate of cognitive change among older participants [35].

The goal of pooling estimates from different studies is to provide a unified and precise estimate to support clear conclusions [36]. There are several reasons why estimates may not be the same between studies; variables may be measured differently, statistical analyses might be different, characteristics of study populations may be different, and there might be a true difference in the effect of interest between studies. A random effects meta-analysis aims to incorporate the underlying between-study variation [36]. With the coordinated approach taken, we minimised heterogeneity due to measurement and analytical approaches and focused our interpretation on heterogeneity between studies as well as the pooled estimate. NSHD is a birth cohort, and, therefore, is an age homogeneous sample (all 53 years at baseline). A noteworthy observation from our analyses is the absence of associations between social health and cognitive capability in this cohort. This may be attributed to the fact that participants were slightly younger at baseline compared to those in other studies. Overall, results for average processing speed were the most homogenous and results for average memory were the most heterogenous. We found heterogenous estimates across studies for marital or cohabitation status and all cognitive domains. Being married or cohabiting was associated with higher average cognitive capability in ELSA and SNAC-K. This was not observed in NSHD or the Rotterdam Study. Conversely, we observed relatively consistent associations between positive social support and average cognitive capability. Similarly, we observed associations participating in social activities and higher average cognitive capability across all included studies, although this may be because NSHD and the Rotterdam Study did not contribute data to these analyses. Results for social health and decline in cognitive capability were relatively homogeneous across studies, except for marital and cohabitation status.

A major strength of our study was the inclusion of four European studies, resulting in a large sample size and the ability to examine replication of findings across datasets. We applied a single framework and coordinated analyses to multiple datasets, selected outcomes that were similarly measured across studies, and harmonised variables as far as possible. This approach has the advantage of reducing conceptual and analytical heterogeneity. Associations that are consistently observed across all studies support the existence of a true effect (or lack thereof). This approach also highlights the importance of not relying on single datasets. Where findings diverge between studies, further exploration of study-specific or context-specific consideration is needed, and caution is required when interpreting the overall pooled effect. However, there are limitations to our study. Assuming that attrition predominantly affected people with poor social health and poor cognition, this could have led to an underestimation of the relationships of interest. Despite our efforts to harmonise measures, some of the heterogeneity in findings between studies observed could be due to differences in the wording of social health questions and the distribution of social relationship variables. Importantly, while we examine social health markers at baseline and cognitive capability over a long follow-up, a bidirectional association between social health and cognitive capability cannot be ruled out. Although impairments in social functioning have been described as part of the dementia prodrome [37], few previous studies have directly examined this. Several studies have made inferences about

reverse causality based on the length of follow-up, with mixed findings. For example, in their paper examining social contact and dementia risk, Sommerlad et al. [38] argue that the 28-year follow-up in the Whitehall II study is unlikely to be explained by reverse causality. Conversely, in the Betula study, a Swedish population-based cohort, the protective effect of social relationships on dementia risk was no longer observed after excluding those with a survival time of less than 3 years. This suggests that the association may reflect reverse causality [39]. While further studies examining the direction of the relationship between social health and cognitive capability are needed, it is also likely that the way in which social health and cognitive capability are related will depend on the dementia disease phase.

Inconsistencies in previous research exploring how various facets of social health relate to cognitive ability may be due to differences in methodology or analytical techniques. While our study has progressed the subject by reducing this methodological noise as much as possible through the integrated data analysis approach, we focused primarily on two domains of social health due to data constraints. We undertook these analyses through the lens of the social health framework. However, further work exploring different aspects such as reciprocity and autonomy are required to fully understand the role of social health in cognitive capability.

In conclusion, we provide evidence that markers of good social health have a positive association with cognitive capability in later life. However, we found differential associations between specific markers of social health and cognitive domains, and not all findings replicated consistently across datasets, highlighting the importance of examining between-study differences and considering context specificity of findings in developing and deploying any interventions. Social contact is recognised as a key component in dementia prevention, intervention, and care [40]. Understanding the mechanisms through which markers of social health can affect cognitive capability is a key next step to identify effective policy-level interventions that target social health. As such, our findings may guide future studies to determine if promoting social health at old age may delay cognitive decline.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## Statement of Ethics

Ethical approval for the MRC National Survey of Health and Development study was obtained from Research Ethics Committees. Written, informed consent was obtained from the study member for each component of data collection. ELSA has received ethical approval from a Research Ethics Committee. SNAC-K was approved by the Ethical Review Board in Stockholm and written informed consent was obtained from participants or their next of kin. The Rotterdam Study has been approved by an Ethics Committee. The Rotterdam Study has been entered into the Netherlands National Trial Register (NTR; www.trialregister.nl) and into the WHO International Clinical Trials Registry Platform (ICTRP; www.who.int/ictrp/network/primary/en/) under shared catalogue number NTR6831. Written informed consent was obtained from all participants.

## Conflict of Interest Statement

The authors report no conflicts of interest.


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## Author Contributions

J.M., P.P., A.M., S.D., and A.-K.W. conceptualised the study. J.M., F.G., F.J.W., A.M., S.D., and A.-K.W. were involved in data curation. J.M., F.G., and F.J.W. conducted formal analyses. K.W.O., R.M., H.B., M.A.I., A.-K.W., and D.D. were involved in funding acquisition. J.M. and P.P. designed the methodology for this study. K.W.-O. and MAI were involved in SHARED project administration. P.P. supervised this study. J.M. was responsible for visualisation. JM wrote original draft. J.S., M.L.B., S.V., M.P., G.B.P., M.R., and all other authors provided critical review and editing of the original manuscript.

## Data Availability Statement

NSHD data used in this study are available to bona fide researchers upon request to the NSHD Data Sharing Committee via a standard application procedure. Further details can be found at http://www.nshd.mrc.ac.uk/data doi: 10.5522/NSHD/Q101, 10.5522/NSHD/Q102 and doi: 10.5522/NSHD/Q103. ELSA data used in this study are available to download through the UK data service. doi.org/10.5255/UKDA-SN-5050-16. SNAC-K data used in this study are available to researchers upon approval by the SNAC-K data management and maintenance committee. Applications for accessing these data can be submitted to Maria Wahlberg (Maria.Wahlberg@ki.se) at the Aging Research Center, Karolinska Institutet, Stockholm, Sweden. Requests for access to the Rotterdam Study data reported in this paper can be addressed to the data management team of the Rotterdam Study (secretariat.epi@erasmusmc.nl).

## References

1 Batty GD, Deary IJ, Zaninotto P. Association of cognitive function with cause-specific mortality in middle and older age: follow-up of participants in the English Longitudinal Study of Ageing. Am J Epidemiol. 2016;183(3):183–90.
2 Davis D, Cooper R, Terrera GM, Hardy R, Richards M, Kuh D. Verbal memory and search speed in early midlife are associated with mortality over 25 years' follow-up, independently of health status and early life factors: a British birth cohort study. Int J Epidemiol. 2016;45(4):1216–25.
3 Richards M, Deary IJ. A life course approach to cognitive capability. In: Kuh D, Cooper R, Hardy R, et al editors. A life course approach to healthy ageing. Oxford; 2014. p. 32–46.
4 Seifert I, Wiegelmann H, Lenart-Bugla M, Łuc M, Pawłowski M, Rouwette E, et al. Mapping the complexity of dementia: factors influencing cognitive function at the onset of dementia. BMC Geriatr. 2022;22(1):507.
5 Plassman BL, Williams JW, Burke JR, Holsinger T, Benjamin S. Systematic review: factors associated with risk for and possible prevention of cognitive decline in later life. Ann Intern Med. 2010;153(3):182–93.
6 Ritchie SJ, Tucker-Drob EM, Cox SR, Corley J, Dykiert D, Redmond P, et al. Predictors of ageing-related decline across multiple cognitive functions. Intelligence. 2016;59:115–26.
7 Salthouse TA. Correlates of cognitive change. J Exp Psychol Gen. 2014;143(3):1026–48.
8 Lenart-Bugla M, Łuc M, Pawłowski M, Szcześniak D, Seifert I, Wiegelmann H, et al. What do we know about social and non-social factors influencing the pathway from cognitive health to dementia? A systematic review of reviews. Brain Sci. 2022;12(9):1214.
9 Vernooij-Dassen M, Verspoor E, Samtani S, Sachdev PS, Ikram MA, Vernooij MW, et al. Recognition of social health: a conceptual framework in the context of dementia research. Front Psychiatry. 2022;13:1052009. Epub ahead of print 2022.
10 Berkman LF, Glass T, Brissette I, Seeman TE. From social integration to health: durkheim in the new millennium. Soc Sci Med. 2000;51(6):843–57.
11 Huber M, André Knottnerus J, Green L, van der Horst H, JadadAR, Kromhout D, et al. How should we define health? BMJ. 2011;343:1–3.
12 Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. PLoS Med. 2010;7:e1000316. Epub ahead of print 2010.
13 Kawachi I, Berkman LF. Social ties and mental health. J Urban Health. 2001;78(3):458–67.
14 Amieva H, Stoykova R, Matharan F, Helmer C, Antonucci TC, Dartigues JF. What aspects of social network are protective for dementia? Not the quantity but the quality of social interactions is protective up to 15 years later. Psychosom Med. 2010;72(9):905–11.
15 Kuiper JS, Zuidersma M, Zuidema SU, Burgerhof JG, Stolk RP, Oude Voshaar RC, et al. Social relationships and cognitive decline: a systematic review and meta-analysis of longitudinal cohort studies. Int J Epidemiol. 2016;45(4):1169–206.
16 Bielak AAM. How can we not 'lose it' if we still don't understand how to 'use it'? unanswered questions about the influence of activity participation on cognitive performance in older age: a mini-review. Gerontology. 2010;56(5):507–19.
17 Brown CL, Gibbons LE, Kennison RF, Robitaille A, Lindwall M, Mitchell MB, et al. Social activity and cognitive functioning over time: a coordinated analysis of four longitudinal studies. J Aging Res. 2012;2012:287438. Epub ahead of print 2012.
18 Kelly ME, Duff H, Kelly S, McHugh Power JE, Brennan S, Lawlor BA, et al. The impact ofsocial activities, social networks, social support and social relationships on the cognitive functioning of healthy older adults: a systematic review. Syst Rev. 2017;6(1):259. Epub ahead of print 2017.
19 Kuiper JS, Zuidersma M, Oude Voshaar RC, Zuidema SU, van den Heuvel ER, Stolk RP, et al. Social relationships and risk of dementia: a systematic review and meta-analysis of longitudinal cohort studies. Ageing Res Rev. 2015;22:39–57.
20 Piolatto M, Bianchi F, Rota M, Marengoni A, Akbaritabar A, Squazzoni F. The effect of social relationships on cognitive decline in older adults: an updated systematic review and meta-analysis of longitudinal cohort studies. BMC Public Health. 2022;22(1):278.

21 Marseglia A, Kalpouzos G, Laukka EJ, Maddock J, Patalay P, Wang HX, et al. Social health and cognitive change in old age: role of brain reserve. Ann Neurol. 2023;93(4):844–55.
22 Stern Y. Cognitive reserve in ageing and Alzheimer's disease. Lancet Neurol. 2012;11:1006–12.
23 Cohen S, Wills TA. Stress, social support, and the buffering hypothesis. Psychol Bull. 1985;98(2):310–57.
24 Cohen S. Social relationships and health. Am Psychol. 2004;59(8):676–84.
25 Gow AJ, Corley J, Starr JM, Deary IJ. Which social network or support factors are associated with cognitive abilities in old age? Gerontology. 2013;59(5):454–63.
26 Hofer SM, Piccinin AM. Integrative data analysis through coordination of measurement and analysis protocol across independent longitudinal studies. Psychol Methods. 2009;14(2):150–64.
27 Samtani S, Mahalingam G, Lam BCP, Lipnicki DM, Lima-Costa MF, Blay SL, et al. Associations between social connections and cognition: a global collaborative individual participant data meta-analysis. Lancet Healthy Longev. 2022;3(11):e740–53.
28 Wadsworth M, Kuh D, Richards M, Hardy R. Cohort profile: the 1946 national birth cohort (MRC National Survey of Health and Development). Int J Epidemiol. 2006;35(1):49–54.
29 Steptoe A, Breeze E, Banks J, Nazroo J. Cohort profile: the English longitudinal study of ageing. Int J Epidemiol. 2013;42(6):1640–8.
30 Lagergren M, Fratiglioni L, Hallberg IR, Berglund J, Elmståhl S, Hagberg B, et al. A longitudinal study integrating population, care and social services data. The Swedish National study on Aging and Care (SNAC). Aging Clin Exp Res. 2004;16(2):158–68.
31 Ikram MA, Brusselle G, Ghanbari M, Goedegebure A, Ikram MK, Kavousi M, et al. Objectives, design and main findings until 2020 from the Rotterdam Study. Eur J Epidemiol. 2020;35(5):483–517.
32 Lee H, Ang S. Productive activities and risk of cognitive impairment and depression: does the association vary by gender? Sociol Perspect. 2020;63(4):608–29.
33 Pillemer S, Ayers E, Holtzer R. Gender-stratified analyses reveal longitudinal associations between social support and cognitive decline in older men. Aging Ment Health. 2019;23(10):1326–32.
34 Hultsch DF, Hertzog C, Small BJ, Dixon RA. Use it or lose it: engaged lifestyle as a buffer of cognitive decline in aging? Psychol Aging. 1999;14(2):245–63.
35 Wilson RS, Yu L, Lamar M, Schneider JA, Boyle PA, Bennett DA. Education and cognitive reserve in old age. Neurology. 2019;92(10):e1041–50.
36 Sutton AJ, Higgins JPT. Recent developments in meta-analysis. Stat Med. 2008;27(5):625–50.
37 Ismail Z, Smith EE, Geda Y, Sultzer D, Brodaty H, Smith G, et al. Neuropsychiatric symptoms as early manifestations of emergent dementia: provisional diagnostic criteria for mild behavioral impairment. Alzheimers Dement. 2016;12(2):195–202.
38 Sommerlad A, Sabia S, Singh-Manoux A, Lewis G, Livingston G. Association of social contact with dementia and cognition: 28-year follow-up of the Whitehall II cohort study. PLoS Med. 2019;16(8):e1002862.
39 Sörman DE, Rönnlund M, Sundström A, Adolfsson R, Nilsson LG. Social relationships and risk of dementia: a population-based study. Int Psychogeriatr. 2015;27(8):1391–9.
40 Livingston G, Huntley J, Sommerlad A, Ames D, Ballard C, Banerjee S, et al. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. Lancet. 2020;396(10248):413–46.