BJPsych | The British Journal of Psychiatry (2024)
224, 189–197. doi: 10.1192/bjp.2023.159

Original Article

Associations of an individual's need for cognition with structural brain damage and cognitive functioning/impairment: cross-sectional population-based study

Lotte S. Truin, Sebastian Köhler, Irene S. Heger, Martin P. J. van Boxtel, Miranda T. Schram, Walter H. Backes, Jacobus F. A. Jansen, Martien M. C. J. M. van Dongen, Nanne K. de Vries, Hein de Vries, Simone J. P. M. Eussen, Coen D. A. Stehouwer, Marjolein E. de Vugt and Kay Deckers

Background

High cognitive activity possibly reduces the risk of cognitive decline and dementia.

Aims

To investigate associations between an individual's need to engage in cognitively stimulating activities (need for cognition, NFC) and structural brain damage and cognitive functioning in the Dutch general population with and without existing cognitive impairment.

Method

Cross-sectional data were used from the population-based cohort of the Maastricht Study. NFC was measured using the Need For Cognition Scale. Cognitive functioning was tested in three domains: verbal memory, information processing speed, and executive functioning and attention. Values 1.5 s.d. below the mean were defined as cognitive impairment. Standardised volumes of white matter hyperintensities (WMH), cerebrospinal fluid (CSF) and presence of cerebral small vessel disease (CSVD) were derived from 3T magnetic resonance imaging. Multiple linear and binary logistic regression analyses were used adjusted for demographic, somatic and lifestyle factors.

Results

Participants ($n = 4209$; mean age 59.06 years, s.d. = 8.58; 50.1% women) with higher NFC scores had higher overall cognition scores ($B = 0.21$, 95% CI 0.17–0.26, $P < 0.001$) and lower odds for CSVD (OR = 0.74, 95% CI 0.60–0.91, $P = 0.005$) and cognitive impairment (OR = 0.60, 95% CI 0.48–0.76, $P < 0.001$) after adjustment for demographic, somatic and lifestyle factors. The association between NFC score and cognitive functioning was similar for individuals with and without prevalent cognitive impairment. We found no significant association between NFC and WMH or CSF volumes.

Conclusions

A high need to engage in cognitively stimulating activities is associated with better cognitive functioning and less presence of CSVD and cognitive impairment. This suggests that, in middle-aged individuals, motivation to engage in cognitively stimulating activities may be an opportunity to improve brain health.

Keywords

Dementias/neurodegenerative diseases; epidemiology; cognitive neuroscience; prevention; mild cognitive impairment.

Copyright and usage

© The Author(s), 2023. Published by Cambridge University Press on behalf of Royal College of Psychiatrists. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

Given the projected increase in the number of people with dementia worldwide and the absence of a curative treatment, dementia is considered one of the biggest public health concerns of the 21st century.[1] Observational research has shown that a substantial proportion of dementia cases are potentially attributable to modifiable risk factors, and the Lancet Commission on Dementia Prevention, Intervention and Care has emphasised that we should be ambitious about dementia risk reduction through lifestyle modifications.[1] This message is reinforced by several recent publications, such as World Health Organization (WHO) guidelines,[2] Alzheimer's Disease International's report[3] and the WHO position paper[4] on brain health.

Results from randomised controlled trials showed mixed findings regarding the influence of multi-domain lifestyle interventions on participants' cognitive functioning.[5] The FINGER trial suggested that an intensive 2-year multi-domain intervention consisting of diet improvement, exercise, cognitive training and cardiovascular risk monitoring could maintain or improve cognitive functioning in older persons at risk for dementia.[6] Some multi-domain intervention studies in different at-risk groups found similar results,[7–10] especially those that included cognitive training as part of the multi-domain intervention,[5] whereas others did not.[11] Two trials focused on incident dementia as an outcome, but individuals in the intervention group were as likely to develop dementia as those in the control group.[12,13] In other words, multi-domain interventions may have small beneficial effects on cognitive functioning in older persons, but it is still unclear whether such interventions can delay or prevent dementia. Thus, further insight into how environmental and lifestyle factors influence the onset and course of dementia is essential, for example for developing preventive interventions against dementia and its effective management.[2,14]

Engaging in cognitively stimulating activities is considered a promising modifiable protective factor for cognitive decline and dementia.[15,16] It is hypothesised that engaging in such activities contributes to the individual's 'cognitive reserve' by promoting synaptogenesis and strengthening of neural networks.[17] This concept aims to explain why some individuals tolerate brain pathology better than others and, consequently, whether the development of cognitive symptoms is delayed or ruled out.[18,19] Cognitively stimulating activities are activities in which information processing is a central component, such as listening to the radio, reading and playing strategic games like cards or puzzles.[20] An individual's need to engage in cognitively stimulating activities, which can be captured by the construct 'need for cognition' (NFC), includes

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

intrinsic motivation and enjoyment at being involved in cognitively stimulating activities. Also, persons with a higher NFC have the tendency to engage in and enjoy thinking, and make active efforts to structure relevant situations and increase understanding.[21] Importantly, NFC can thus be seen as a main driver for subsequent engagement in cognitively stimulating activities, which makes it an important primary intervention target.[21] Several studies have examined whether an individual's NFC is associated with cognitive functioning, but findings are mixed; however, most studies were relatively small (<350 participants) and were conducted in non-representative samples.[22–24]

As far as we know, there is no previous research on the association between NFC and structural brain damage. However, an association between engagement in cognitively stimulating activities and brain damage has been established.[25] For example, a systematic review by Anatürk et al[25] found that cognitive activity correlated with whole-brain assessments of white matter volume and number of lesions. Thus, it could be hypothesised that NFC is possibly associated with markers of brain damage and therefore might be a potential protective factor for overall brain health.

Therefore, the present study aimed to investigate the association between an individual's need to engage in cognitively stimulating activities and overall cognitive functioning, cognitive impairment and evidence of structural brain damage in the Dutch general population aged 40–75 years. In addition, we investigated the potential moderating effects of structural brain damage and cognitive impairment on the association between an individual's need to engage in cognitively stimulating activities and overall cognitive functioning to assess whether this association was different for people with and without existing brain damage or cognitive impairment.

Method

Study population and design

We used data from the Maastricht Study, an observational prospective population-based cohort study. The rationale and methodology have been described previously.[26] In brief, the study focuses on the aetiology, pathophysiology, complications and comorbidities of type 2 diabetes and is characterised by an extensive phenotyping approach. Eligible for participation were all individuals aged between 40 and 75 years who were living in the southern part of The Netherlands. Participants were recruited through mass media campaigns and from the municipal registries and the regional Diabetes Patient Registry via postal mailings. Recruitment was stratified according to known diabetes status, with an oversampling of individuals with type 2 diabetes, for reasons of efficiency. The present report includes cross-sectional data from 7689 participants who completed the baseline survey between November 2010 and December 2017. The examinations of each participant were performed within a time window of 3 months. Participants were included in the analyses if data on the Need for Cognition Scale, cognition, magnetic resonance imaging (MRI) outcomes, sociodemographic characteristics and lifestyle factors were available.

An individual's need to engage in cognitively stimulating activities

The 18-item Need for Cognition Scale is an instrument to detect an individual's 'tendency to engage in and enjoy thinking' and consists of various statements linked to the construct of NFC.[21] Items are evenly phrased positively and negatively, for example 'Thinking is not my idea of fun' (negative wording) and 'I would prefer complex to simple problems' (positive wording). Participants indicate their agreement with statements on a 9-point Likert scale, ranging from 'very strong disagreement' (score of −4) to 'very strong agreement' (score of +4); the total score range is −72 to 72). A high score on the Need for Cognition Scale implies that someone is highly motivated to engage in cognitively stimulating activities. In addition to looking at a continuous measurement of NFC (mean score on the Need for Cognition Scale; sum score divided by total number of completed items), participants were also classified into three groups based on tertiles (low, medium, high) of the mean Need for Cognition Scale score.

Cognitive functioning

Cognitive functioning was assessed by a 30-min neuropsychological test battery conducted by trained research assistants.[26] The individual neuropsychological tests scores were standardised and divided in three cognitive domains: memory, processing speed, and executive functioning and attention. For memory function, immediate and delayed recall on the Verbal Learning Test were used.[27] Information processing speed was based on the Stroop Color–Word Test Parts 1 and 2,[28] the Concept Shifting Test Parts A and B[29] and the Letter Digit Substitution Test.[30] Executive functioning and attention were assessed using the Concept Shifting Test Part C and Stroop Color–Word Test Part 3. Test scores were standardised into *z*-scores based on the means and standard deviations of individual variables across the study sample and then averaged in cognitive domain scores. The three domain scores were averaged to yield an overall cognition score. In addition, a score of ≤1.5 s.d. below the mean on any of the three cognitive domains based on normative data for age, gender and level of education was considered as indicative of cognitive impairment (yes/no).

Structural brain damage and cerebral small vessel disease

Structural brain damage was based on volumetric parameters measured using 3T MRI (MAGNETOM Prisma-fit Syngo MR D13D; Siemens Healthcare, Erlangen, Germany). The following MRI protocol was used: a three-dimensional T1-weighted sequence, a T2 fluid-attenuated inversion recovery (FLAIR) and a gradient recalled echo (GRE) pulse sequence with susceptibility-weighted imaging (SWI). The T1-weighted and T2 FLAIR images were used to assess participants' brain volume. The T1-weighted and T2 FLAIR images were also used to segment brain tissue into grey matter, white matter and cerebrospinal fluid volumes, and their sum indicates intracranial volume. The sum of grey matter and white matter volumes was used to calculate total brain volume. Volumetric measures of white matter hyperintensities (WMH; mL) and cerebrospinal fluid (CSF; mL) were used as a proxy of cerebrovascular damage and brain atrophy respectively. The combined cerebral small vessel disease (CSVD) score was defined as any of (a) WMH volume >3.0 mL, (b) presence of lacunar infarct or (c) presence of cerebral microbleeds, resulting in a binary score of CSVD present (yes/no).

Demographics and covariates

Information on age (years), gender (male/female), educational level (low/medium/high), smoking status (never/former/current) and history of cardiovascular disease (CVD; yes/no) was collected from study questionnaires. Participants were asked to bring their medication for review, from which the use of antihypertensive medication (yes/no), antidepressants (yes/no) and lipid-modifying medication (yes/no) was noted. Hypertension (yes/no) was based on average office blood pressure measurement using a blood pressure monitor (Omron 705IT, Japan; systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg) or current

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

antihypertensive medication use. The presence of a current minor or major depressive episode (yes/no) was assessed using the Mini-International Neuropsychiatric Interview (MINI).[31] Participants completed a Food Frequency Questionnaire, from which we obtained adherence to the Dutch Health Diet Index (DHDI; sum score), reflecting diet, and the Community Healthy Activities Model Program for Seniors questionnaire (CHAMPS; sum score), for assessment of physical activity.[32–35] The presence of type 2 diabetes (yes/no) was based on their glucose tolerance status measured using an oral glucose tolerance test (≥11.1 mmol/l), according to the WHO definition.[36] The total cholesterol to high density lipoprotein (HDL) cholesterol ratio was determined through laboratory assessment. Height and weight were measured, and body mass index (BMI) was calculated from these using the formula BMI = kg/m^2.

Statistical analysis

Differences in characteristics between the three NFC groups (tertiles) and between the final study sample and the excluded group were tested using one-way analysis of variance (ANOVA) for continuous variables and Pearson's χ^2-test for categorical variables. Multiple linear regression analyses were used to test the association between NFC (continuous score and tertiles) and (a) cognitive functioning, (b) WMH and (c) CSF. Binary logistic regression analyses were used to test the association between NFC and presence of (a) cognitive impairment and (b) CSVD. All analyses followed the same protocol of adding blocks of potential confounders. Model 1 was the crude model (adjusted for MRI lag-time, to correct for time between assessment and MRI, and intracranial volume for volumetric MRI markers, to correct for head size). In model 2, we adjusted for demographic factors (model 1 plus age, gender, education, type 2 diabetes (because of the oversampling of type 2 diabetes)). In model 3, we adjusted for somatic factors (model 2 plus BMI, hypertension, history of CVD, depression, antidepressants, cholesterol, lipid-modifying medication). In model 4, we adjusted for lifestyle factors (model 3 plus smoking status, physical activity, diet). Model 3 was considered the main model because missingness was relatively high for smoking status, physical activity and diet.

To examine whether there was an interaction effect between NFC and brain damage on cognitive functioning, interaction terms (NFC × WMH; NFC × CSF; NFC × CSVD) were included in the regression analyses. Similarly, to study whether there was an interaction effect between NFC and cognitive impairment on cognitive functioning and brain damage the interaction term 'NFC × cognitive impairment' was included in the regression analysis. Additional interaction terms were included in model 3 to investigate whether the associations between NFC and cognitive functioning/impairment and MRI markers were modified by age, gender and type 2 diabetes status. Associations are expressed by the unstandardised regression coefficients (*B*) and the 95% confidence intervals (CI) for linear regression and by odds ratios (OR) and 95% confidence intervals for logistic regression. The possibility of multicollinearity among NFC, age, gender and education was explored using the variance inflation factor (VIF). As WMH was skewed, a constant was added and the variable was log transformed. The possibility of a dose–response relationship or non-linear association was explored by replacing the continuous variable of NFC with a categorical (low/medium/high) variable of NFC and tested with a likelihood ratio test for a linear trend. A non-significant test suggests no deviation from a linear trend, which is consistent with a linear dose–response relation. In a sensitivity analysis, we restricted model 3 to participants with missing data on model 4 to explore potential selection bias in model 4. For similar reasons, we included participants with missing MRI data when testing the association between NFC and cognitive functioning/impairment. *P*-values <0.05 were considered statistically significant for two-sided tests. All statistical testing was performed with Stata Statistical Software, version 17.0 for Mac OS.

Standard protocol approvals, registrations, and patient consents

The Maastricht Study has been approved by the institutional medical ethical committee (NL31329.068.10) and the Ministry of Health, Welfare and Sports of The Netherlands (permit 131088-105234-PG) and conformed with the Declaration of Helsinki.[26] All participants gave written informed consent.

Results

General characteristics of the study population

Of the original 7689 participants, 3480 (45.3%) were excluded from this study, largely owing to missing data on NFC, cognitive functioning and MRI (Fig. 1). This resulted in a study population of 4209 participants (mean age 59.06 years, s.d. = 8.58; 50.1% female).

Compared with included participants for this study, excluded participants (n = 3480) had lower overall cognition scores (−0.1 *v.* 0.1; $F(1) = 172.2$, $P < 0.001$), lower education (40.7% *v.* 29.9%; $\chi^2(2) = 100.9$, $P < 0.001$) and a higher mean age (59.1 years *v.* 60.7 years; $F(1) = 70.1$, $P < 0.001$). Excluded participants more often had type 2 diabetes (18.9% *v.* 32.0%; $\chi^2(1) = 173.2$, $P < 0.001$), depression (3.0% *v.* 3.8%; $\chi^2(2) = 8.3$, $P = 0.016$), hypertension (49.2% *v.* 60.8%; $\chi^2(1) = 102.2$, $P < 0.001$) and CVD (12.0% *v.* 23.5%; $\chi^2(1) = 172.6$, $P < 0.001$) compared with the study sample. Furthermore, excluded participants more often used lipid-modifying medication (26.4% *v.* 40.0%; $\chi^2(1) = 160.2$, $P < 0.001$) and antidepressants (6.2% *v.* 8.4%; $\chi^2(1) = 13.2$, $P < 0.001$). Excluded participants had a higher BMI (26.5 kg/m^2 *v.* 27.6 kg/m^2; $F(1) = 117.1$, $P < 0.001$), were more often smokers (48.8% *v.* 50.0%; $\chi^2(2) = 51.0$, $P < 0.001$), were less physically active (13.5 h/week *v.* 14.1 h/week; $F(1) = 8.6$, $P = 0.003$) and less often adhered to a healthy diet (DHDI score 82.7 *v.* 84.5; $F(1) = 24.4$, $P < 0.001$) compared with the study sample.

Table 1 summarises the characteristics of the total study population and stratified by NFC tertiles (low, medium, high). Participants with a higher NFC score were more likely to be younger and male compared with participants with low or medium NFC scores. Participants with a low NFC score were more likely to have a lower level of education, have depression, use antidepressants, have a higher presence of CSVD and smaller CSF volume than participants with a medium or high NFC score. Participants with a low NFC score were more likely to have higher total hours of physical activity per week compared with participants with a higher NFC score. No crude differences between the NFC groups were observed with regard to type 2 diabetes status, BMI, hypertension, CVD history, smoking status and WMH volume.

NFC and overall cognition

After adjusting for demographic and somatic factors (model 3), a higher NFC score was associated with higher cognitive functioning (B = 0.213, 95% CI 0.169–0.258, $P < 0.001$; for full model metrics see the Supplementary material, available at https://dx.doi.org/10.1192/bjp.2023.159). After full adjustment (model 4), the associations between NFC score and cognitive functioning remained similar. Also, a higher NFC score was associated with higher levels in all three domains of cognitive functioning adjusted for demographic and somatic factors (model 3; memory: B = 0.187, 95% CI 0.116–0.257, $P < 0.001$; executive functioning and attention: B = 0.243,

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

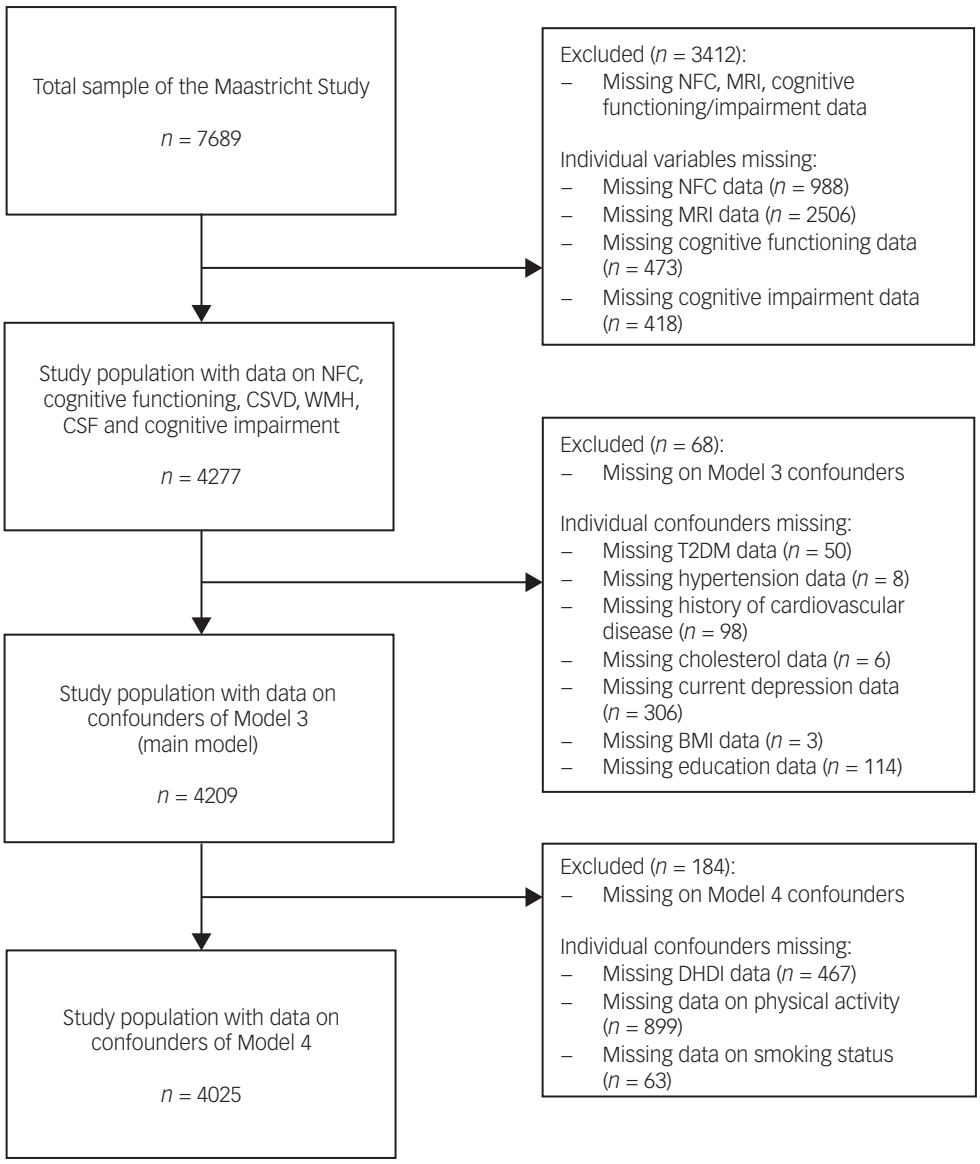


Fig. 1 Flowchart of participant selection.

NFC, need for cognition; MRI, magnetic resonance imaging; CSVD, cerebral small vessel disease; WMH, white matter hyperintensities; CSF, cerebrospinal fluid; T2DM, type 2 diabetes mellitus; BMI, body mass Index; DHDI, Dutch Healthy Diet Index.

95% CI 0.181–0.304, $P < 0.001$; processing speed: $B = 0.211$, 95% CI 0.156–0.266, $P < 0.001$; see Supplementary material for full model metrics). Again, associations remained similar when we additionally adjusted for lifestyle factors (model 4). Finally, cognitive functioning differed across NFC tertiles: participants with medium ($B = 0.073$, 95% CI −0.034 to 0.111, $P < 0.001$) and high NFC ($B = 0.169$, 95% CI −0.127 to 0.211; $P < 0.001$) showed better cognitive functioning compared with those with low NFC (model 3), suggesting a dose–response relationship (Fig. 2). This dose–response relationship was confirmed with a likelihood ratio (LR) test that showed a linear trend (LR $\chi^2(2) = -22.94$, $P = 1.000$).

A higher NFC score was associated with lower odds of cognitive impairment (OR = 0.602, 95% CI 0.479–0.757, $P < 0.001$) after adjusting for demographic and somatic factors (model 3). This association remained similar after full adjustment (model 4). The odds for cognitive impairment were lower for participants with a medium (OR = 0.846, 95% CI 0.696–1.029, $P = 0.094$) and high NFC score (OR = 0.707, 95% CI 0.572–0.873, $P = 0.001$) compared with participants with a low NFC score (model 3; see Supplementary material for full model metrics). The likelihood ratio test showed a linear trend (LR $\chi^2(2) = 2.98$, $P = 0.225$). No multicollinearity among NFC, age, gender and education was present (Supplementary Tables 13–15).

NFC and structural brain damage

After adjustment for demographic and somatic factors (model 3), a higher NFC score was associated with lower odds for CSVD (OR = 0.740, 95% CI 0.599–0.913, $P = 0.005$ (Table 2; see Supplementary material for full model metrics). After full adjustment (model 4), the association between NFC score and CSVD remained similar. No significant associations between NFC score and CSF or WMH volumes were observed (Table 2).

Participants with a high NFC score had lower odds for CSVD compared with participants with a low NFC score (model 3; OR = 0.820, 95% CI 0.675–0.996, $P = 0.046$). The association for the medium NFC tertile was directionally similar, but not significant (OR = 0.856, 95% CI 0.715–1.024, $P = 0.089$) in model 3. Again,

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

Table 1 Characteristics of the total study population and stratified by tertiles of need for cognition (NFC)[a]

	Total study population (*n* = 4209)	Low NFC (*n* = 1108)	Medium NFC (*n* = 1572)	High NFC (*n* = 1529)	*P*
Age, years: mean (s.d.)	59.1 (8.6)	59.8 (8.4)	59.1 (8.7)	58.6 (8.5)	0.002
Gender, female: *n* (%)	2109 (50.1)	711 (64.2)	802 (51.0)	596 (39.0)	<0.001
Educational level, *n* (%)					
Low	1260 (29.9)	555 (50.1)	505 (32.1)	200 (13.1)	<0.001
Middle	1210 (28.8)	326 (29.4)	492 (31.3)	392 (25.6)	
High	1739 (41.3)	227 (20.5)	575 (36.6)	937 (61.3)	
Type 2 diabetes, *n* (%)	796 (18.9)	222 (20.0)	308 (19.6)	266 (17.4)	0.159
BMI, mean (s.d.)	26.5 (4.2)	26.6 (4.4)	26.5 (4.1)	26.4 (4.0)	0.427
Hypertension, *n* (%)	2072 (49.2)	560 (50.5)	784 (49.9)	728 (47.6)	0.270
Total/HDL cholesterol, ratio (s.d.)	3.6 (1.2)	3.5 (1.2)	3.6 (1.2)	3.7 (1.2)	0.004
Use of lipid-modifying medication, *n* (%)	1113 (26.4)	323 (29.2)	433 (27.5)	357 (23.4)	0.002
Cardiovascular history, *n* (%)	506 (12.0)	143 (12.9)	185 (11.8)	178 (11.6)	0.570
Depression, *n* (%)	125 (3.0)	59 (5.3)	47 (3.0)	19 (1.2)	<0.001
Use of antidepressants, *n* (%)	261 (6.2)	110 (9.9)	89 (5.7)	62 (4.1)	<0.001
Smoking status, *n* (%)[b]					
Never	1677 (39.9)	451 (40.7)	595 (37.9)	631 (41.3)	0.248
Former	2054 (48.8)	528 (47.7)	789 (50.2)	737 (48.3)	
Current	476 (11.3)	129 (11.6)	188 (12.0)	159 (10.4)	
Total physical activities, h/week: mean (s.d.)[c]	14.1 (8.1)	14.4 (8.0)	14.2 (8.3)	13.8 (7.8)	0.040
DHDI score, mean (s.d.)[d]	84.5 (15.0)	84.1 (14.8)	84.5 (15.1)	84.8 (14.9)	0.434
Presence of CSVD, *n* (%)	1325 (31.5)	387 (34.9)	490 (31.2)	448 (29.3)	0.008
WMH volume, mL: mean (s.d.)	0.9 (2.8)	1.0 (2.8)	1.0 (3.2)	0.8 (2.4)	0.170
CSF volume, mL: mean (s.d.)	251.5 (47.0)	244.8 (46.2)	252.0 (46.4)	255.9 (47.7)	<0.001
Cognitive functioning score, mean (s.d.)	0.1 (0.6)	−0.1 (0.7)	0.1 (0.6)	0.2 (0.6)	<0.001
Executive functioning and attention score, mean (s.d.)	0.1 (0.8)	−0.1 (0.8)	0.1 (0.7)	0.3 (0.8)	<0.001
Memory score, mean (s.d.)	0.1 (0.9)	0.0 (0.9)	0.1 (0.9)	0.2 (0.9)	<0.001
Processing speed score, mean (s.d.)	0.1 (0.7)	−0.1 (0.8)	0.1 (0.7)	0.2 (0.7)	<0.001

BMI, body mass index; HDL, high-density lipoprotein; DHDI, Dutch Healthy Diet Index; CSVD, cerebral small vessel disease; WMH, white matter hyperintensities; CSF, cerebrospinal fluid.
a. Percentages do not total 100 owing to rounding.
b. Based on *n* = 4207 owing to missing values.
c. Based on *n* = 4208 owing to missing values.
d. Based on *n* = 4028 owing to missing values.

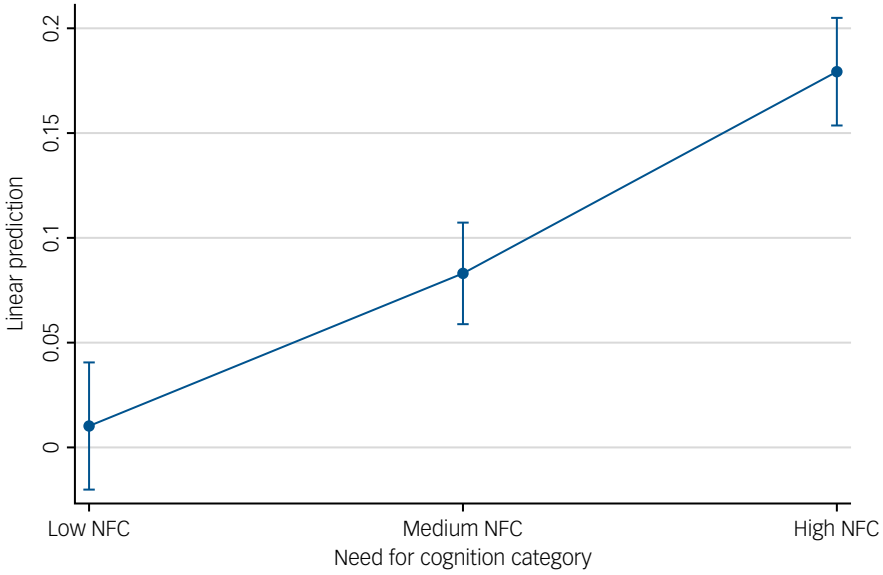


Fig. 2 Association between need for cognition (NFC) tertiles and overall cognitive functioning.

associations between NFC score tertiles and CSF or WMH volumes were not significant after adjusting for demographic and somatic factors (Table 3). No multicollinearity among NFC, age, gender and education was present (Supplementary Table 16).

Potential effect modifiers

Potential moderating effects between NFC and the three proxies of brain damage (CSVD, WMH, CSF) on cognitive functioning were tested, but no significant interaction was observed (CSVD: $B = -0.073$, 95% CI −0.160 to 0.015, $P = 0.104$; WMH: $B = -0.010$, 95% CI −0.081 to 0.064, $P = 0.794$; CSF: $B = 0.000$, 95% CI −0.001 to 0.001, $P = 0.615$). Using the tertiles of NFC score, we found an interaction effect on cognitive functioning between NFC score and CSVD but not between NFC score and WMH or CSF (CSVD × medium NFC: $B = -0.081$ (95% CI −0.161 to −0.001, $P = 0.048$; CSVD × high NFC: $B = -0.098$, 95% CI −0.179 to 0.016, $P = 0.019$), suggesting that higher NFC was less strongly

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

Table 2 Association between need for cognition score (continuous) and cerebral small vessel disease, white matter hyperintensities volume and cerebrospinal fluid volume

	Cerebral small vessel disease			White matter hyperintensities			Cerebrospinal fluid		
	OR	95% CI	*P*	*B*	95% CI	*P*	*B*	95% CI	*P*
Model 1	0.656	0.546–0.787	<0.001	−0.100	−0.145 to −0.055	<0.001	−3.217	−6.253 to −0.180	0.038
Model 2	0.736	0.597–0.908	0.004	−0.036	−0.080 to 0.008	0.110	0.063	−2.626 to 2.752	0.963
Model 3[a]	0.740	0.599–0.913	0.005	−0.030	−0.074 to 0.014	0.185	0.216	−2.479 to 2.910	0.875
Model 4	0.725	0.584–0.900	0.004	−0.026	−0.072 to 0.020	0.263	0.462	−2.290 to 3.214	0.742

OR, odds ratio; *B*, unstandardised regression coefficient; model 1: Adjusted for magnetic resonance imaging (MRI) lag time and intracranial volume for volumetric MRI markers; model 2: model 1 plus adjustment for age, gender, education, type 2 diabetes; model 3: model 2 plus adjustment for body mass index, hypertension, history of cardiovascular disease, depression, antidepressants, cholesterol, and lipid-modifying medication; model 4: model 3 plus adjustment for smoking status, physical activity, Dutch Health Diet Index sum score.
a. Main model.

Table 3 Association between need for cognition (NFC) score (tertiles) and cerebral small vessel disease, white matter hyperintensities volume and cerebrospinal fluid volume in model 3 (main model)[a]

	Cerebral small vessel disease			White matter hyperintensities			Cerebrospinal fluid		
	OR	95% CI	*P*	*B*	95% CI	*P*	*B*	95% CI	*P*
Low NFC		Reference			Reference			Reference	
Medium NFC	0.856	0.715–1.024	0.089	−0.000	−0.039 to 0.038	0.989	0.310	−2.027 to 2.649	0.795
High NFC	0.820	0.675–0.996	0.046	−0.018	−0.059 to 0.023	0.392	−0.437	−2.954 to 2.080	0.734

OR, odds ratio; *B*, unstandardised regression coefficient.
a. Adjusted for magnetic resonance imaging (MRI) lag time and intracranial volume for volumetric MRI markers, age, gender, education, type 2 diabetes, body mass index, hypertension, history of cardiovascular disease, depression, antidepressants, cholesterol and lipid-modifying medication.

associated with cognition in those with CSVD. We observed no interaction effects on cognitive functioning between NFC score and cognitive impairment ($B = 0.037$, 95% CI −0.044 to 0.117, $P = 0.372$) or CSVD (OR = 1.002, 95% CI 0.312–3.948, $P = 0.872$). These results remained similar when using NFC tertiles. Lastly, interactions were tested between NFC score (both continuous measure and tertiles) and age, gender and type 2 diabetes, and between WMH, CSF and CSVD and age, gender and type 2 diabetes, but no significant interactions were observed.

Sensitivity analysis

When participants with missing data on lifestyle factors (model 4 covariates smoking status, physical activity and diet; $n = 184$) were excluded from the regression analyses in model 3, associations remained unchanged. When including participants with missing MRI data ($n = 2506$), associations between NFC and cognitive functioning/impairment remained similar.

Discussion

Main findings

This cross-sectional population-based study investigated the associations between an individual's NFC score, cognition and structural brain damage in mid-life. Individuals with a higher NFC score, representing higher motivation for and pleasure in engaging in cognitively stimulating activities, showed better cognitive functioning and had lower odds for CSVD and cognitive impairment. No associations were observed between NFC score and WMH or CSF volumes. No potential effect modifiers were identified, other than that the association between the tertiles of NFC score and cognitive functioning differed for persons without CSVD compared with persons with CSVD. Finally, associations did not differ between those with and without prevalent cognitive impairment.

Interpretation of our findings and comparison with the literature

Our results are supported by previous research. A prospective study by Baer et al[23] suggested that NFC was associated with change in cognitive functioning in recent retirees ($n = 333$): participants with a higher NFC showed greater improvement in cognitive status 2 years after retirement than participants with a lower NFC. Likewise, Maldonato et al[22] found that NFC and neurocognitive ability were strongly correlated in 1174 healthy elderly participants in the USA. In contrast, a study by Gärtner et al did not find evidence for an association between NFC and executive functions in young adults.[24] A possible explanation could be that the sample was relatively small ($n = 189$), participants were on average relatively young (mean age 23.8 years) and had relatively low variation in their cognitive abilities and likely educational level as participants were recruited on a university campus. Furthermore, our findings suggested a dose–response relationship between NFC and cognitive functioning: the higher someone's NFC, the better their cognitive functioning.

NFC was significantly associated with the presence of CSVD, but there were no associations between NFC and WMH or CSF volumes, the latter a measure of overall brain atrophy. The association between NFC score and CSVD was independent of demographic, somatic and lifestyle factors, and did not differ by type 2 diabetes status, gender or cognitive impairment. Although a link between engagement in cognitively stimulating activities and brain damage has been described in the literature, to the best of our knowledge, there is no previous research on the association between NFC score and brain damage. For example, a systematic review from Anatürk et al[25] showed that high socio-intellectual activities were associated with larger global white matter volume and fewer white matter lesions, although effect sizes were small. Also, higher engagement in cognitively stimulating activities has been associated with larger global grey matter volume, which in turn is associated with better cognitive functioning.[25,37] Although previous literature did not specifically focus on the association between NFC and brain damage, results of this study build on existing evidence that higher engagement in cognitively stimulating activities is associated with less presence of brain damage markers.

The association between NFC score and cognitive functioning were similar for men and women, people with and without type 2 diabetes, and for individuals with and without prevalent cognitive impairment. The last is of particular importance and suggests that individuals could benefit from increasing NFC regardless of their

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

cognitive status and that efforts at enlarging NFC might benefit persons with and without prevalent cognitive impairment. Next, there were no interactive effects between NFC score and WMH or CSF volumes on cognitive functioning, which suggests that the association between NFC score and cognitive functioning also did not depend on inter-individual difference in volumetric measures of structural brain integrity, including subtle vascular damage or atrophy. An interaction was observed between NFC tertiles and CSVD on cognitive functioning: NFC might be more beneficial for cognitive functioning in persons without CSVD compared with persons with CSVD. However, this effect was not found in our main analysis, where we used NFC score as a continuous measure, and it requires replication.

Clinical implications

The results of our study add to the growing body of literature suggesting that a cognitively active lifestyle is protective against cognitive decline and dementia.[16,38–43] NFC in both adolescents and adults (mid-life and later in life), assessed using the Need for Cognition Scale, has been shown to be indicative of the individual's engagement in cognitively stimulating activities.[21,44,45] Yet, it is also important to distinguish an individual's NFC and their actual engagement in these activities. Current preventive strategies against cognitive decline and dementia mostly aim at promoting engagement in cognitively stimulating activities, but this might be less effective if NFC is low. Increasing intrinsic motivation is a crucial first step towards behavioural change and therefore strengthening one's NFC should be a primary target for improving or maintaining cognitive functioning and brain health. Mental health clinicians and policymakers should put more emphasis on the potential of NFC and could capitalise on this by creating awareness of this topic as a first step. In line with behaviour change models,[46] people with high NFC might already be in the next stage of behavioural change (more intention/motivation to change their health behaviour) compared with people with low NFC. A first step for clinicians is to screen for individuals with high NFC in order to engage them in cognitively stimulating activities. Next, individuals with low NFC need to be motivated by clinicians to prevent them remaining stuck in the early stages of behavioural change. This can be done by person-centred counselling approaches such as motivational interviewing.[47]

Strengths and limitations

Strengths of this study include the population-based design, the large sample with a wide age range (40–75 years) and availability of data on NFC, multiple domains of cognitive functioning and state-of-the-art MRI images. Further, many covariates were assessed, which allowed us to adjust for major confounders, including other dementia risk and protective factors. Associations between NFC and CSVD, CSF and WMH and between NFC and cognitive functioning/impairment remained unchanged in model 4; furthermore, when rerunning model 3 by excluding participants with missing data on model 4 covariates, the associations in model 3 remained similar. When including participants with missing MRI data, associations between NFC and cognitive functioning/impairment remained similar. This suggested that not including participants with missing data on model 4 covariates or MRI data in the main analysis (model 3) did not introduce selection bias.

This study also had several limitations. First, self-selection might have led to a study population that is healthier and more health-conscious than the general population, which is a common problem in observational cohort studies. Second, excluding participants with missing data on NFC, cognitive functioning and MRI and the possible confounders included in model 3 may have introduced selection bias. Compared with our study sample, the excluded group was indeed older, had lower overall cognition scores, lower levels of education and seemed to be burdened with more comorbidities, such as depression, hypertension and CVD. Together, these forms of selection bias most likely led to an underestimation of the associations explored in this study and lower generalisability. Third, despite log transformation of WMH volume, non-normal distribution and heteroskedasticity of the variable was still present, which might have made study outcomes involving WMH less precise. This might lead to an underestimation of the associations explored in this study. Last, since analyses were cross-sectional, temporal relationships between variables in our models cannot be established, and the observational study design cannot rule out residual confounding. Hence, no statements on cause and effect can be made. Further research is needed to better inform about possible causality, specifically large-scale longitudinal studies and cohorts that are followed over the lifespan.

Lotte S. Truin, MSc, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Sebastian Köhler**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Irene S. Heger**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Martin P. J. van Boxtel**, MD, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Miranda T. Schram**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; School for Cardiovascular Diseases (CARIM), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; Department of Internal Medicine, Maastricht University Medical Center, Maastricht, The Netherlands; and Maastricht Heart and Vascular Center, Maastricht University Medical Center, Maastricht, The Netherlands; **Walter H. Backes**, PhD, MSc, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; School for Cardiovascular Diseases (CARIM), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Radiology & Nuclear Medicine, Maastricht University Medical Center, Maastricht, The Netherlands; **Jacobus F. A. Jansen**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Martien M. C. J. M. van Dongen**, PhD, Department of Epidemiology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Nanne K. de Vries**, PhD, Department of Health Promotion, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Hein de Vries**, PhD, Department of Health Promotion, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Simone J. P. M. Eussen**, PhD, School for Cardiovascular Diseases (CARIM), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; Department of Epidemiology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Care and Public Health Research Institute (CAPHRI), Maastricht University, Maastricht, The Netherlands; **Coen D. A. Stehouwer**, MD, PhD, School for Cardiovascular Diseases (CARIM), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Marjolein E. de Vugt**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; **Kay Deckers**, PhD, School for Mental Health & Neuroscience (MHeNs), Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands; and Department of Psychiatry and Neuropsychology, Faculty of Health, Medicine and Life Sciences, Maastricht University, Maastricht, The Netherlands

Correspondence: Kay Deckers. Email: kay.deckers@maastrichtuniversity.nl

First received 7 Jul 2023, final revision 20 Oct 2023, accepted 6 Nov 2023

Supplementary material

Supplementary material is available online at https://doi.org/10.1192/bjp.2023.159.

Data availability

The data used in this study are unsuitable for public deposition owing to ethical restrictions and regulation of the privacy of participants' data. Data from the Maastricht Study are available to

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

any interested researcher who meets the criteria for access to confidential data. Data requests may be submitted to the Maastricht Study Management Team.

Acknowledgements

We thank all participants in the Maastricht Study for their willingness to participate in the study, as well as all the study's funders.

Author contributions

L.S.T.: analyses and interpretation of the data, drafting the manuscript. S.K.: study concept and design, analyses and interpretation of the data, study supervision, critical revision of the manuscript for intellectual content. I.S.H., M.P.J.v.B., M.T.S., W.H.B, J.F.A.J., M.M.C.J.M.v.D., N.K.d.V., H.d.V., S.J.P.M.E., C.D.A.S. and M.E.d.V: critical revision of the manuscript for intellectual content. K.D.: study concept and design, analyses and interpretation of the data, study supervision, critical revision of the manuscript for intellectual content.

Funding

This study was supported by the European Regional Development Fund via OP-Zuid, the Province of Limburg, the Dutch Ministry of Economic Affairs (grant 31O.041), Stichting De Weijerhorst (Maastricht, The Netherlands), the Pearl String Initiative Diabetes (Amsterdam, The Netherlands), CARIM, School for Cardiovascular Diseases (Maastricht, The Netherlands), School CAPHRI, Care and Public Health Research Institute (Maastricht, The Netherlands), NUTRIM, School of Nutrition and Translational Research in Metabolism (Maastricht, The Netherlands), Stichting Annadal (Maastricht, The Netherlands), Health Foundation Limburg (Maastricht, The Netherlands) and by unrestricted grants from Janssen-Cilag B.V. (Tilburg, The Netherlands), Novo Nordisk Farma B.V. (Alphen aan den Rijn, The Netherlands) and Sanofi-Aventis Netherlands B.V. (Gouda, The Netherlands). This publication is also part of the ABOARD project. ABOARD is a public-private partnership that receives funding from ZonMw (#73305095007) and Health~Holland, Top Sector Life Sciences & Health (PPS allowance; #LSHM20106). More than 30 partners contribute in kind and/or in cash. ABOARD also receives funding from Gieskes-Strijbisfonds and Edwin Bouw Fonds.

Declaration of interest

None.

References

1 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**: 413–46.

2 World Health Organization. *Risk Reduction of Cognitive Decline and Dementia: WHO Guidelines*. WHO, 2019.

3 Alzheimer's Diseases International. *Risk Factors and Risk Reduction*. Alzheimer's Diseases International, 2020 (https://www.alzint.org/about/risk-factors-risk-reduction/).

4 World Health Organization. *Optimizing Brain Health across the Life Course: WHO Position Paper*. WHO, 2022.

5 Hafdi M, Hoevenaar-Blom MP, Richard E. Multi-domain interventions for the prevention of dementia and cognitive decline. *Cochrane Database Syst Rev* 2021; **11**(11): CD013572.

6 Ngandu T, Lehtisalo J, Solomon A, Levalahti E, Ahtiluoto S, Antikainen R, et al. A 2 year multidomain intervention of diet, exercise, cognitive training, and vascular risk monitoring versus control to prevent cognitive decline in at-risk elderly people (FINGER): a randomised controlled trial. *Lancet* 2015; **385**: 2255–63.

7 Komulainen P, Kivipelto M, Lakka TA, Savonen K, Hassinen M, Kiviniemi V, et al. Exercise, fitness and cognition – a randomised controlled trial in older individuals: the DR's EXTRA study. *Eur Geriatr Med* 2010; **1**: 266–72.

8 Lee KS, Lee Y, Back JH, Son SJ, Choi SH, Chung YK, et al. Effects of a multidomain lifestyle modification on cognitive function in older adults: an eighteen-month community-based cluster randomized controlled trial. *Psychother Psychosom* 2014; **83**: 270–8.

9 Li M, Liu L, Song S, Shi A, Ma Y, Zhang S, et al. Effect of long-term lifestyle intervention on mild cognitive impairment in hypertensive occupational population in China. *Medicine (Baltimore)* 2018; **97**(34): e11975.

10 Chen LK, Hwang AC, Lee WJ, Peng LN, Lin MH, Neil DL, et al. Efficacy of multidomain interventions to improve physical frailty, depression and cognition: data from cluster-randomized controlled trials. *J Cachexia Sarcopenia Muscle* 2020; **11**: 650–62.

11 Andrieu S, Guyonnet S, Coley N, Cantet C, Bonnefoy M, Bordes S, et al. Effect of long-term omega 3 polyunsaturated fatty acid supplementation with or without multidomain intervention on cognitive function in elderly adults with memory complaints (MAPT): a randomised, placebo-controlled trial. *Lancet Neurol* 2017; **16**: 377–89.

12 Moll van Charante EP, Richard E, Eurelings LS, van Dalen JW, Ligthart SA, van Bussel EF, et al. Effectiveness of a 6-year multidomain vascular care intervention to prevent dementia (preDIVA): a cluster-randomised controlled trial. *Lancet* 2016; **388**: 797–805.

13 Espeland MA, Luchsinger JA, Baker LD, Neiberg R, Kahn SE, Arnold SE, et al. Effect of a long-term intensive lifestyle intervention on prevalence of cognitive impairment. *Neurology* 2017; **88**: 2026–35.

14 Sheardova K, Vyhnalek M, Nedelska Z, Laczo J, Andel R, Marciniak R, et al. Czech Brain Aging Study (CBAS): prospective multicentre cohort study on risk and protective factors for dementia in the Czech republic. *BMJ Open* 2019; **9** (12): e030379.

15 Deckers K, van Boxtel MP, Schiepers OJ, de Vugt M, Munoz Sanchez JL, Anstey KJ, et al. Target risk factors for dementia prevention: a systematic review and Delphi consensus study on the evidence from observational studies. *Int J Geriatr Psychiatry* 2015; **30**: 234–46.

16 Duffner LA, Deckers K, Cadar D, Steptoe A, de Vugt M, Köhler S. The role of cognitive and social leisure activities in dementia risk: assessing longitudinal associations of modifiable and on-modifiable risk factors. *Epidemiol Psychiatr Sci* 2022; **31**: e5.

17 Reuter-Lorenz PA, Park DC. How does it STAC up? Revisiting the scaffolding theory of aging and cognition. *Neuropsychol Rev* 2014; **24**: 355–70.

18 Stern Y, Arenaza-Urquijo EM, Bartrés-Faz D, Belleville S, Cantilon M, Chetelat G, et al. Whitepaper: defining and investigating cognitive reserve, brain reserve, and brain maintenance. *Alzheimers Dement* 2020; **16**: 1305–11.

19 DeJong NR, Jansen JFA, van Boxtel MPJ, Schram MT, Stehouwer CDA, Dagnelie PC, et al. Cognitive resilience depends on white matter connectivity: the Maastricht Study. *Alzheimers Dement* 2023; **19**: 1164–74.

20 Stern C, Munn Z. Cognitive leisure activities and their role in preventing dementia: a systematic review. *Int J Evid Based Healthc* 2010; **8**: 2–17.

21 Cacioppo J, Petty R. The need for cognition. *J Pers Soc Psychol* 1982; **42**: 116–31.

22 Maldonato NM, Sperandeo R, Costa V, Cioffi V, Cozzolino P, De Santo RM, et al. Does brain sweat pay off?: the association between the need for cognition and cognitive function among the American elderly. *J Psychol Psychother* 2017; **7** (5): 1000326.

23 Baer LH, Tabri N, Blair M, Bye D, Li KZ, Pushkar D. Longitudinal associations of need for cognition, cognitive activity, and depressive symptomatology with cognitive function in recent retirees. *J Gerontol B Psychol Sci Soc Sci* 2013; **68**: 655–64.

24 Gärtner A, Grass J, Wolff M, Goschke T, Strobel A, Strobel A. No relation of need for cognition to basic executive functions. *J Pers* 2021; **89**: 1113–25.

25 Anatürk M, Demnitz N, Ebmeier KP, Sexton CE. A systematic review and meta-analysis of structural magnetic resonance imaging studies investigating cognitive and social activity levels in older adults. *Neurosci Biobehav Rev* 2018; **93**: 71–84.

26 Schram MT, Sep SJ, van der Kallen CJ, Dagnelie PC, Koster A, Schaper N, et al. The Maastricht Study: an extensive phenotyping study on determinants of type 2 diabetes, its complications and its comorbidities. *Eur J Epidemiol* 2014; **29**: 439–51.

27 van der Elst WIM, van Boxtel MPJ, van Breukelen GJP, Jolles J. Rey's Verbal Learning Test: normative data for 1855 healthy participants aged 24–81 years and the influence of age, sex, education, and mode of presentation. *J Int Neuropsychol Soc* 2005; **11**: 290–302.

28 van der Elst W, van Boxtel MPJ, van Breukelen GJP, Jolles J. The Stroop Color-Word Test: influence of age, sex, and education; and normative data for a large sample across the adult age range. *Assessment* 2006; **13**: 62–79.

29 van der Elst W, van Boxtel MP, van Breukelen GJ, Jolles J. The Concept Shifting Test: adult normative data. *Psychol Assess* 2006; **18**: 424–32.

30 van der Elst W, van Boxtel MP, van Breukelen GJ, Jolles J. The Letter Digit Substitution Test: normative data for 1,858 healthy participants aged 24–81 from the Maastricht Aging Study (MAAS): influence of age, education, and sex. *J Clin Exp Neuropsychol* 2006; **28**: 998–1009.

31 Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry* 1998; **59**(suppl 20): 22–33.

32 Looman M, Feskens EJ, de Rijk M, Meijboom S, Biesbroek S, Temme EH, et al. Development and evaluation of the Dutch Healthy Diet Index 2015. *Public Health Nutr* 2017; **20**: 2289–99.

33 Stewart AL, Mills KM, King AC, Haskell WL, Gillis D, Ritter PL. CHAMPS physical activity questionnaire for older adults: outcomes for interventions. *Med Sci Sports Exerc* 2001; **33**: 1126–41.

34 Resnicow K, McCarty F, Blissett D, Wang T, Heitzler C, Lee RE. Validity of a modified CHAMPS physical activity questionnaire among African-Americans. *Med Sci Sports Exerc* 2003; **35**: 1537–45.

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press

35 van Dongen MC, Wijckmans-Duysens NEG, den Biggelaar LJ, Ocké MC, Meijboom S, Brants HA, et al. The Maastricht FFQ: development and validation of a comprehensive food frequency questionnaire for the Maastricht Study. *Nutrition* 2019; **62**: 39–46.

36 Wprld Health Organization, International Diabetes Foundation. *Definition and Diagnosis of Diabetes mellitus and Intermediate Hyperglycaemia: Report of a WHO/IDF Consultation*. WHO, 2006.

37 Bartres-Faz D, Sole-Padulles C, Junque C, Rami L, Bosch B, Bargallo N, et al. Interactions of cognitive reserve with regional brain anatomy and brain function during a working memory task in healthy elders. *Biol Psychol* 2009; **80**: 256–9.

38 Parrott MD, Carmichael PH, Laurin D, Greenwood CE, Anderson ND, Ferland G, et al. The association between dietary pattern adherence, cognitive stimulating lifestyle, and cognitive function among older adults from the Quebec Longitudinal Study on Nutrition and Successful Aging. *J Gerontol B Psychol Sci Soc Sci* 2021; **76**: 444–50.

39 Almeida-Meza P, Steptoe A, Cadar D. Is engagement in intellectual and social leisure activities protective against dementia risk? Evidence from the English Longitudinal Study of Ageing. *J Alzheimers Dis* 2021; **80**: 555–65.

40 Fratiglioni L, Paillard-Borg S, Winblad B. An active and socially integrated lifestyle in late life might protect against dementia. *Lancet Neurol* 2004; **3**: 343–53.

41 Fritsch T, Smyth KA, Debanne SM, Petot GJ, Friedland RP. Participation in novelty-seeking leisure activities and Alzheimer's disease. *J Geriatr Psychiatry Neurol* 2005; **18**: 134–41.

42 Scarmeas N, Levy G, Tang MX, Manly J, Stern Y. Influence of leisure activity on the incidence of Alzheimer's disease. *Neurology* 2001; **57**: 2236–42.

43 Wang HX, Karp A, Winblad B, Fratiglioni L. Late-life engagement in social and leisure activities is associated with a decreased risk of dementia: a longitudinal study from the Kungsholmen Project. *Am J Epidemiol* 2002; **155**: 1081–7.

44 Strobel A, Luong C, Fleischhauer M. Predicting everyday life behavior by direct and indirect measures of need for cognition. *J Individ Dif* 2018; **39**: 107–14.

45 Cacioppo JT, Petty RE. Dispositional differences in cognitive motivation: the life and times of individuals varying in need for cognition. *Psychol Bull* 1996; **119**: 197–253.

46 Prochaska JO, Velicer WF. The transtheoretical model of health behavior change. *Am J Health Promot* 1997; **12**: 38–48.

47 Hettema J, Steele J, Miller WR. Motivational interviewing. *Ann Rev Clin Psychol* 2005; **1**: 91–111.

EXTRA CONTENT ONLINE OPEN ACCESS CC BY

https://doi.org/10.1192/bjp.2023.159 Published online by Cambridge University Press