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Cognitively Stimulating Environments and Cognitive Reserve: The Case of Personal Social Networks

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Abstract

Cognitively stimulating environments are thought to be protective of cognitive decline and onset of Alzheimer's disease and related dementias (ADRD) through the development of cognitive reserve (CR). CR refers to cognitive adaptability that buffers the impact of brain pathology on cognitive function. Despite the critical need to identify cognitively stimulating environments to build CR, there is no consensus regarding which environmental determinants are most effective. Rather, most studies use education as proxies for CR and little is known about the association between older adults' personal social networks and CR. Using neuroimaging data from 135 older adults participating in the Social Networks in Alzheimer Disease (SNAD) study, this article adopted a residual method for measuring CR and found that large network size, high network diversity, and loosely connected networks were positively associated with greater CR. These results suggest that expansive social networks in later life may constitute cognitively stimulating environments which can be leveraged to build CR and reduce the burden of ADRD.

Keywords

Cognitive reserve; cognitively stimulating environments; social networks; Alzheimer's disease

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1. Introduction

The cognitive reserve hypothesis has been a prominent theory explaining heterogeneity in the relationship between brain structure and cognitive function. It suggests that individual differences in cognitively stimulating environments can lead to different levels of cognitive reserve (CR) that enable some people to withstand considerable brain atrophy without exhibiting declines in cognition or displaying symptoms (Stern, 2002; Stern et al., 2020). This study focuses on social networks as a cognitively stimulating environment. The networks perspective is distinctive in that it embeds individuals in the larger social context of relationships, group membership, and community, and this context affects their outcomes and life chances, including cognitive function (Perry et al., 2018). In line with this, evidence suggests that more social engagement (Krueger et al., 2009; Sauter et al., 2019), larger network size (Barnes et al., 2004; Kelly et al., 2017; Perry et al., 2021), and higher network diversity [i.e., count of types of social relationships in a network] (Ali et al., 2018; Ellwardt et al., 2015) are associated with better cognitive function. Researchers often explain this association by arguing that diverse social networks can expose people to cognitive challenges (e.g., novel information and perspectives) which in turn build up people's CR (Shankar et al., 2013). However, this link between social networks and CR is rarely tested empirically.

Despite the critical need to identify cognitively stimulating environments that build CR, there is no consensus regarding which environmental determinants are most promising, due in large part to limitations in the measurement of CR (Chapko et al., 2018; Reed et al., 2011). In many studies, indicators of cognitively stimulating environments (e.g., education) are conceptualized as proxies of CR and used as predictors of cognitive health or dementia (Nilsson and Lövdén, 2018; Opdebeeck et al., 2016; Schwartz et al., 2016). If a significant association between cognitively stimulating environments and cognitive health is observed, the result is interpreted as evidence for CR. In these studies, there is no direct measure of underlying CR itself, and the association between cognitively stimulating environments and CR is inferred. Thus, the reliance on proxy measures of CR prevents direct observation of the link between CR and cognitively stimulating environments because the two concepts are assumed to be synonymous.

To directly test the link between CR and cognitively stimulating environments, this study adopts a residual method to measure CR (Habeck et al., 2017; Reed et al., 2010). In theory, CR assesses to what extent cognitive function is better or worse than what would be expected given someone's pathology burden. Using this definition, CR is quantified as residuals in cognitive function that remain after accounting for structural brain health and any neuropathologic changes. In other words, individuals who perform better than predicted have high CR, and individuals who perform worse than predicted have low CR (as shown in Figure 1). The current study aims to estimate the association between social networks and CR using this residual method.

2. Methods

2.1 Participants

Data were drawn from the Social Networks in Alzheimer Disease (SNAD) Project, which recruited participants from the Indiana Alzheimer's Disease Research Center (IADRC). The IADRC cohort consists of approximately 450 participants in three main groups: roughly half are cognitively normal (CN), about one quarter have mild cognitive impairment (MCI), and about one quarter have Alzheimer's disease and related dementias (ADRD). Beginning in March 2015, all eligible IADRC participants were approached to voluntarily complete the SNAD protocol (92% response rate). Exclusion criteria for SNAD included: (1) IADRC participants with a Montreal Cognitive Assessment (MoCA) score <10; (2) participants with known family history of dominantly inherited dementia genes; (3) people with age<45. Data for SNAD were collected face-to-face using computer-assisted personal interviewing by trained professional interviewers.

This analysis used data from 135 participants who completed MRI scans and the first wave of SNAD interviews. Of these participants, 67% were cognitively normal, 24% were diagnosed with mild cognitive impairment, and 9% were diagnosed with ADRD. The healthier sample relative to the IADRC cohort as a whole reflects exclusion of participants with severe dementia who may have been unable to accurately report on their social networks.

2.2 Social Network and Cognitive Assessments

The SNAD project collected data on egocentric social networks by interviewing participants using an expanded PhenX Social Network Battery (SNB) tailored to the case of dementia ("PhenX Toolkit," 1991). Interviewers elicited names of network members with whom participants interacted with in the past six months for discussions about important matters and health matters (Perry et al., 2018). After network members' names were provided, interviewers asked follow-up questions about each person in the network. Four measures of network characteristics were examined in this study. *Network size* was measured by the number of network members in participants' network. *Network density* was measured by the mean of the closeness of tie between each network member in participants' network, which includes 0 = do not know, 1 = not very close, 2 = sort of close, or 3 = very close. Higher scores signified higher closeness of ties between network members. *Proportion of kin* was measured by calculating the proportion of friends in participants' network. *Network diversity* was measured using a modified version of the Social Network Index (Cohen et al., 1997), which assessed participation in 12 types of social relationships (ranged from 0–12). These included relationships with a spouse, parents, parents-in-law, children, other close family members, close neighbors, friends, workmates, schoolmates, members of groups without religious affiliations, members of religious groups, and professionals. All 4 measures were Z-score standardized.

Global cognitive function was measured by the *Montreal Cognitive Assessment (MoCA)* (Nasreddine et al., 2005), a widely used brief cognitive screening tool. The MoCA included tests of memory, visuospatial ability, executive functions, attention, language,

and orientation to time and place. Higher scores indicated better cognitive function. *The Craft Story 21* is a paragraph story recall test that assesses episodic memory (Craft et al., 1996), which was treated as a secondary cognitive outcome in the sensitivity analyses. The examiner read a story aloud and then asked the participant to repeat the details of the story in the same words read by the examiner or in his/her own words. Points for verbatim (exact content words) and paraphrase recall (similar contextual story units) were summed separately and yielded substantively identical results. After approximately 20 minutes, the participant was asked to recall the story again. For this study, we used the delayed paraphrase scores for their proven validity in measuring global cognition and memory (Dodge et al., 2020). Higher scores indicated better episodic memory.

2.3 Structural Neuroimaging

All scans were performed on a research dedicated Siemens Prisma 3T scanner using the ADNI protocol (http://adni.loni.usc.edu) by the Indiana ADRC Neuroimaging Core. Structural scan data analyzed here included T1-weighted MPRAGE scans that were postprocessed using FreeSurfer, version 6.0, to extract intracranial volume, gray matter volume, and cortical thickness by regions of interests (ROIs), and white matter hyperintensities (WMH) volume. ROIs included: hippocampal volume, amygdala volume, frontal, parietal, temporal, and occipital mean cortical thickness. WMH volumes were log transformed to normalize their distribution.

2.4 Data Analysis

2.4.1 Computation of CR—We adopted a linear-regression based residual approach to measure CR (Habeck et al., 2017; Reed et al., 2010). CR was computed as the residual (ε), when regressing cognitive function on brain structural measures, including intracranial volume, gray matter volume, cortical thickness, and WMH. In other words, CR is measured as the residual of cognitive performance unexplained by brain structure. The mathematical expression is detailed below:

$$\text{Cognitive function} = \beta_{\text{Brain}} * \text{Brain} + \varepsilon$$

Cognitive function is the cognitive measure of interest (i.e., MoCA score), Brain is a 1 × 8 matrix of MRI data with an associated 8 × 1 vector of regression coefficients β_{Brain}, and ε is residuals representing CR.

2.4.2 Prediction of CR—Once CR was calculated, 4 measures of social networks (network size, network density, network diversity, and proportion of kin) were used to predict CR in separate models while controlling for age, gender, and race. We chose to control for demographics in prediction models rather than the computation model of CR. This allows us to observe the relationship of the derived CR measures to these demographics.

Missing data were handled by the full information maximum likelihood estimation method using structural equation models (Enders and Bandalos, 2001). All analyses were done in Stata 16.0.

3. Results

As shown in Table 1, 35% of our participants were men, 76% were White, mean age was 71 years old, and mean education was 16 years. On average, participants had about 5 members in their networks, a diversity score of 4 (i.e., 4 different types of relationships across their networks), 65% of kin in the network, and mean density of 1.76. The average MoCA score was about 24 out of 30.

As shown in Table 2, education was weakly correlated with network measures ($r <= 0.2$). Therefore, using education alone as a proxy of CR would not account for other important sources of CR (e.g., social networks) and therefore could be biased.

3.1 Cognitive Reserve

We calculated CR measures using the residual method. In the computation model, intracranial volume, ROIs' gray matter volume and thickness, and WMH explained 40.8% variance in cognitive function, which is consistent with other studies using the residual method (Boyle et al., 2013; Nilsson and Lövdén, 2018).

3.2 Social Networks as Predictors of CR

Table 3 showed that larger network size ($b = 0.15$, $p < 0.05$), lower network density ($b = -0.22$, $p < 0.05$), and higher network diversity ($b = 0.26$, $p < 0.01$) were associated with higher CR, whereas proportion of kin was not associated with CR controlling for age, gender, and race.

As shown in Figure 2, older adults with CR higher than the mean had larger networks, more diversity and were less densely connected than those below the mean.

3.3 Sensitivity Analyses

Although the two-step model (i.e., first estimating residuals and then using them as a dependent variable in a separate regression model) for estimating CR is easy to implement, it is subject to measurement error in the step computing CR. A simultaneous estimation approach is less likely to be biased (Bollen, 1989). To examine the robustness of our findings to measurement error, we simultaneously regressed observed cognitive outcomes on brain and social network variables (Table S1 in Supplementals). The results were very similar to the two-step method and suggested minimal bias from measurement error.

An alternative way to test whether social networks are associated with cognitive reserve is through an analysis of interactions between social network and brain variables. We conducted interaction analyses between network diversity and two saliant brain variables (hippocampal volume and amygdala volume). Hippocampal volume and amygdala volume were chosen because they are connected to memory, social behavior, and onset of ADRD (Apostolova et al., 2006; Porcelli et al., 2019). Both interactions were statistically significant (beta=−0.15** for hippocampal volume interaction and beta=−0.14* for amygdala volume interaction). Results showed a diminished association between brain structural measures and cognitive function among those with high diversity, suggesting network diversity may confer cognitive reserve against neurodegeneration (Figure 3). In other words, these results depict

the interaction of network diversity with brain volumes, and show that brain effects are substantially reduced in those with higher network diversity.

In addition, MoCA is often skewed to the left and tends to have ceiling effects (Figure S1 in Supplementals). This may interfere with estimating effects across the full spectrum of cognitive function. To check the robustness of our results, we conducted sensitivity analyses using a different cognitive outcome: Craft Story 21 delayed recall (paraphrase). As shown in the Figure S1, Craft Story delayed recall was less subject to ceiling or floor effects. The results using Craft Story recall (Table S2) were similar to that of MoCA (beta=0.21** vs, 0.15* for network size, beta=−0.18 vs. −0.22* for density, beta=0.25** vs. 0.26** for diversity, and beta=−0.14 vs. −0.03 for prop. kin). In general, both results suggested that social network measures were related to CR.

Given gender differences in social engagement and Alzheimer's disease (Nebel et al., 2018; Valenzuela et al., 2013), it is possible that the associations between social networks and CR vary by gender. We examined the interaction between gender and social networks, but we found no statistical significance. Similarly, we explored whether this relationship is the same depending on disease severity. We examined the interaction between cognitive status (normal vs. impaired) and social networks, but we found no statistical significance (Table S3 in Supplementals).

4. Discussion

This is the first study to examine the link between social networks and CR using a residual measure of CR rather than a proxy measure, such as educational attainment. We found that people with larger networks, lower network density, and higher network diversity had higher CR. These results support the idea that large and diverse social networks are cognitively stimulating environments which build CR. Our findings provide evidence that CR is likely to be an important pathway underlying the association between cognitive function and network size and diversity documented in the prior research (Ali et al., 2018; Barnes et al., 2004; Ellwardt et al., 2015; Kelly et al., 2017). According to theories of social networks, sharing ties to loosely connected and diverse groups of others can expose individuals to novel information and life experiences. Further, because diverse groups of others usually hold different social norms and information, maintaining ties to them requires an individual to switch between different cognitive frameworks associated with distinct social groups (Mische and White, 1998). Therefore, there are theoretical grounds to argue that large, loosely connected, and diverse network can bolster cognitive reserve by promoting access to cognitively stimulating social environments (Ali et al., 2018; Burt, 2000; Pan and Chee, 2020).

The issue of how to measure CR is a largely neglected topic. Research aimed at studying CR requires the inclusion of three components: the status of the brain (reflecting brain health and the extent or severity of any neuropathological condition), clinical or cognitive outcomes, and a measure of reserve. A simple correlation of cognitive function with a sociobehavioral proxy of CR is not sufficient to establish evidence of CR because it provides no insight into how CR influences the relationship between the brain and cognitive

function. In response to the call for better operationalization of CR (Stern et al., 2020), we implemented a residual measure of CR as the excess or shortfall of cognitive performance over its brain-based prediction (Habeck et al., 2017; Reed et al., 2010) and demonstrated its use in studying the link between cognitively stimulating environments and CR.

There is a critical need to investigate modifiable determinants of CR beyond education and occupation (Chapko et al., 2018; Nilsson and Lövdén, 2018; Opdebeeck et al., 2016; Schwartz et al., 2016). Education, the most commonly used proxy measure of CR (Opdebeeck et al., 2016), is static and does not change over time, which prevents researchers from studying changes in CR and how to improve CR beyond the early life course. Therefore, although education plays a key role in CR research, more empirical studies are needed to study modifiable cognitively stimulating environments (e.g., social engagement, network size, and network diversity) in later life that are potential targets for intervention. This study showed that social network size, diversity, and density were linked to CR, which suggests opportunities in personal social networks for possible intervention early in AD trajectories.

This finding is especially important for older adults. With children leaving home, retirement, and other life changes, this population has fewer opportunities for exposure to cognitively stimulating social environments (e.g., work related stimulations) (Elder, 1985; Moen et al., 2000). Significant life course transitions may lead social networks to play a more critical role in exposing older adults to stimulating environments (Roth, 2020). However, as people age, they tend to limit their social worlds to a small group of close friends and family who are most important to them (Carstensen, 1993; Lang and Carstensen, 2002). Older adults' preference for strong ties over weak ties can reduce their opportunities for interacting with diverse groups of others at a point when such interactions are paramount for maintaining brain health. Therefore, it is critical to understand the complex relationships between aging, social networks, and CR to promote social policies and programs that keep older adults cognitively stimulated in maximally effective ways.

Caveats include that all the findings are correlational, and that causal inference should be made with caution. Reverse causation (i.e., that people with more CR have larger, less dense, and more diverse networks) cannot be ruled out by the cross-sectional design of this study. However, this is not a pathway that is predicted by the theory of CR. Future research using longitudinal designs is needed to study how changes in social networks are related to changes in CR over time.

In this study, we did not evaluate the association between perceived social support and CR. Given the reported association between perceived social support and cognitive decline in prior research (Boss et al., 2015; Kelly et al., 2017), it is possible that perceived social support can also promote cognitive reserve. One potential mechanism could be that social support fosters a sense of belonging and promotes coping, reducing the harmful effects of stress on cognitive function (Cohen et al., 1997; Thoits, 2011).

A reporting bias, such that people with impaired cognitive function underreport their social networks, is a concern. That said, a comparison of network corroboration between

participants' reports and study informants' reports found that cognitively impaired older adults showed no difference in their ability to corroborate accounts of their social networks with their study informants, compared to cognitively normal older adults (Roth et al., 2021). This suggests that self-reported network data offer a reasonable account of the core network members in one's life, even in the early stages of dementia. Nevertheless, we attempted to mitigate this concern by dropping participants with MoCA score < 10.

In this study, we only measured CR using MoCA and Craft Story 21. In theory, people can have different levels of CR in different dimensions of cognition. Moreover, different stimulating environments may be more or less effective in building CR depending on the dimension of CR being measured. Therefore, future studies should examine CR across a wide range of cognitive outcomes.

In addition, our data are not representative. Rather, they are based on a convenience sample of participants in an NIH-funded ADRC cohort. As such, participants are more likely to be white, live in a metropolitan area, and have higher socioeconomic status relative to the general population. It is possible that these characteristics of our sample yielded an underestimate of the association between social network characteristics and CR given the relatively lower risk for dementia in this subpopulation.

In sum, using a residual method to measure CR, this study found that large, loosely connected, and diverse social networks were related to higher CR. While other analytic approaches may yield similar results, having a direct, quantitative measure of CR facilitates hypothesis testing regarding which cognitively stimulating environment is effective in building CR. This work contributes to the CR literature by providing support to the proposition that social networks in later life can expose people to cognitive challenges (e.g., novel information and life experiences) which in turn build up CR and lead to delayed onset of cognitive decline associated with underlying ADRD neuropathology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Social networks are positively associated with cognitive reserve (CR).
- Larger network size, higher diversity, and lower density predict higher CR.
- The residual measurement of CR is useful in testing CR building environments.

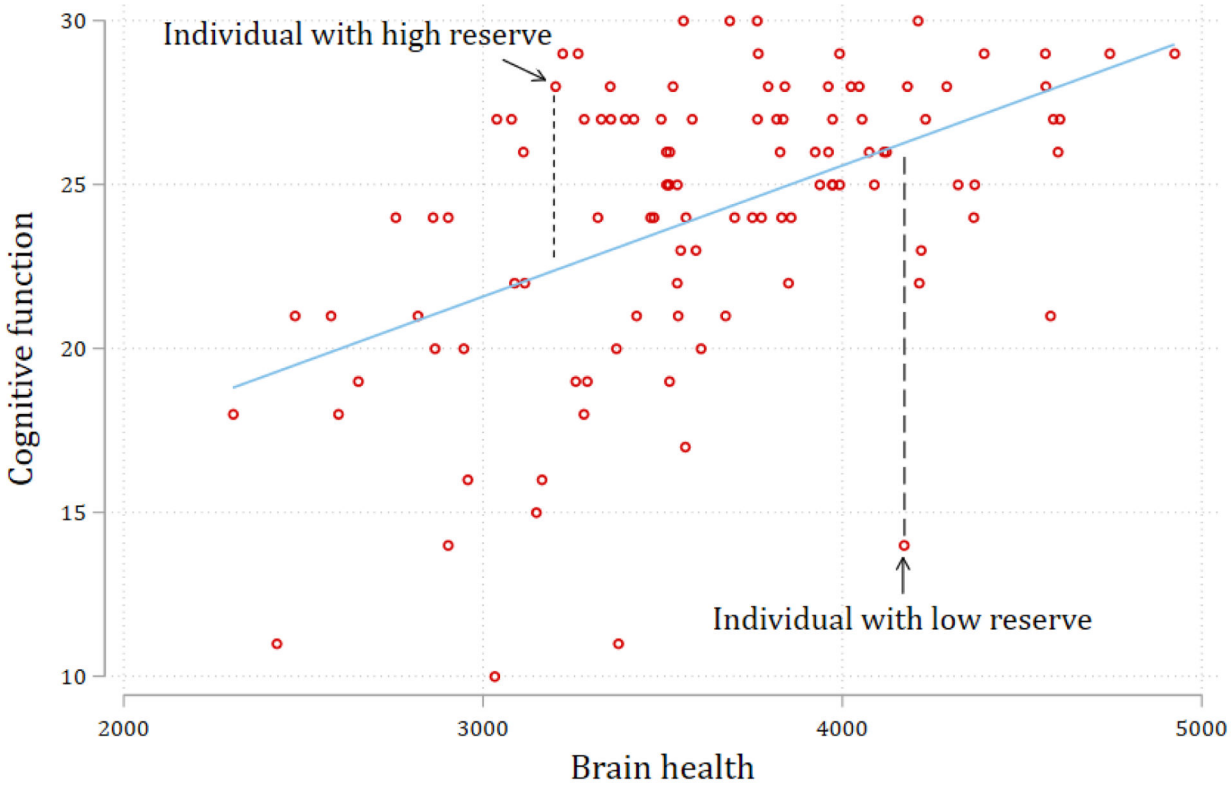

Figure 1.
A residual method for measuring cognitive reserve (CR)

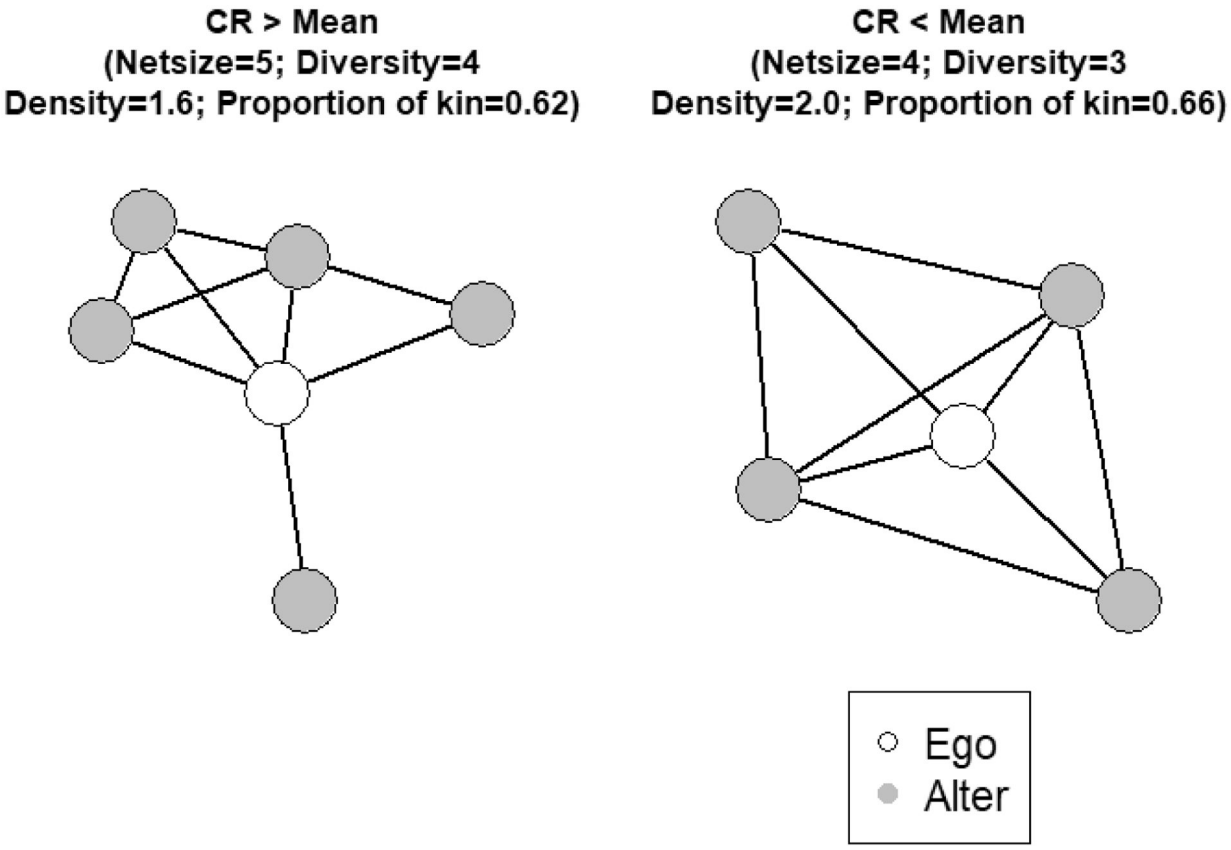

Figure 2.

Visualization of network characteristics of respondents with CR > mean vs. CR < mean.

Note: CR = cognitive reserve; Ego = a focal respondent; Alter = a focal respondent's network members; Line = presence of a tie between people.

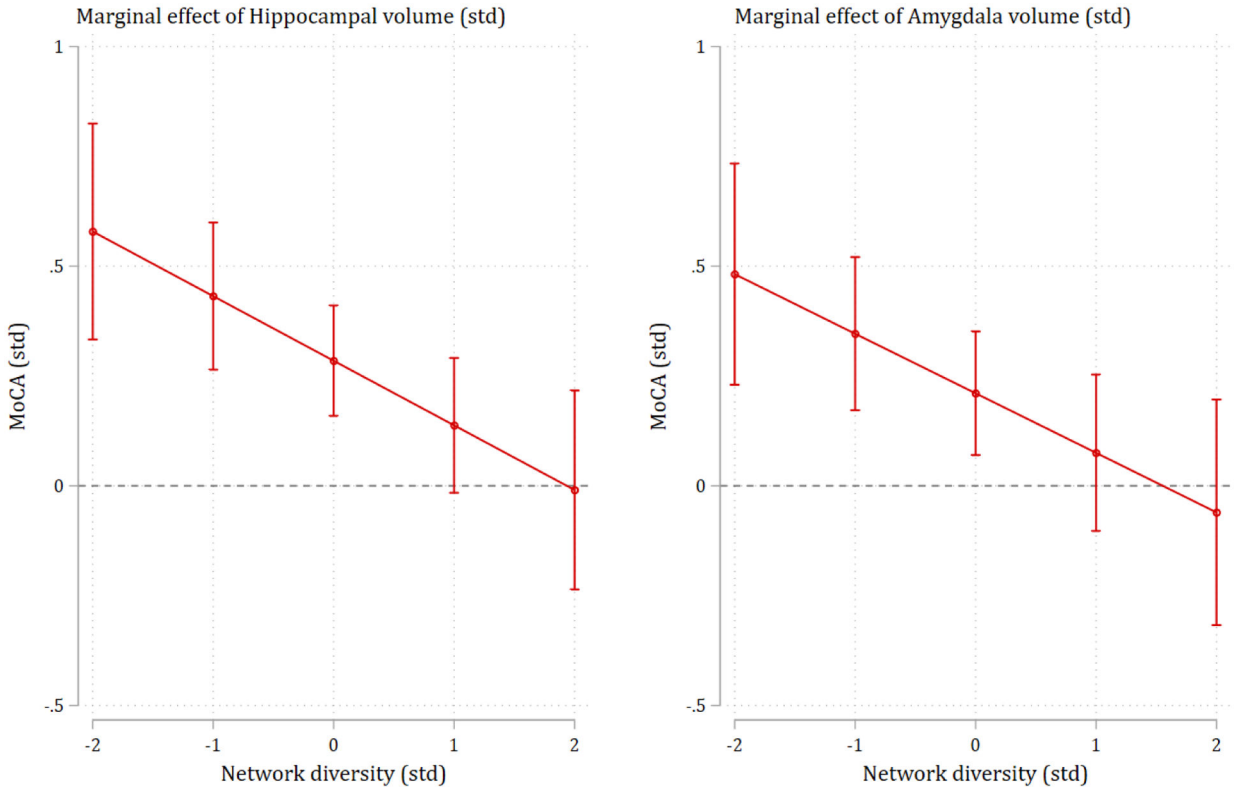

Figure 3:

Marginal effect of brain structure on cognitive function by network diversity.

Note: The plotted values are the beta coefficients for the brain variables at different values of network diversity. Effects above zero indicate higher cognitive function for people with higher brain volume.

Table 1.

Descriptive statistics (N = 135)

	Mean/Proportion	Standard Deviation
Network size	5.05	2.72
Network density	1.76	.77
Network diversity	3.62	1.54
Proportion of kin	.65	.28
Men	.35	
White	.76	
Age	70.64	8.37
Education	16.33	2.75
MoCA total raw score	24.08	4.31

Table 2.

Pairwise correlations

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(1) Network size	1.00							
(2) Network density	−0.37***	1.00						
(3) Network diversity	0.68***	−0.41***	1.00					
(4) Proportion of kin	−0.31***	0.61***	−0.33***	1.00				
(5) Years of education	0.12	−0.10	0.20**	−0.19**	1.00			
(6) White	0.00	0.19**	0.06	−0.05	0.20**	1.00		
(7) Men	−0.14*	0.20**	−0.15*	−0.01	0.16*	0.16*	1.00	
(8) Age	−0.15*	0.11	−0.23***	0.03	−0.20**	0.08	0.12	1.00

*** $p<0.01$

** $p<0.05$

* $p<0.1$

Table 3.

Prediction of CR using social networks ***

	Coef. (SE)	Coef. (SE)	Coef. (SE)	Coef. (SE)
Network size (std)	0.15*			
	(0.07)			
Network density (std)		−0.22*		
		(0.09)		
Network diversity (std)			0.26**	
			(0.09)	
Proportion of kin (std)				−0.03
				(0.09)
Men	−0.31	−0.28	−0.27	−0.36
	(0.19)	(0.18)	(0.19)	(0.19)
White	0.56**	0.64**	0.50*	0.56**
	(0.21)	(0.21)	(0.20)	(0.21)
Age	0.01	0.01	0.01	0.01
	(0.01)	(0.01)	(0.01)	(0.01)
R-squared	0.099	0.122	0.142	0.079
AIC	2036.07	2006.48	2043.23	2064.97
BIC	2053.50	2058.77	2095.52	2117.26

Note: std refers to standardization

* $p<0.05$

** $p<0.01$

*** $p<0.001$.