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Research paper

Understanding the relationship between post-stroke cognitive impairments and depression: The role of loneliness

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ABSTRACT

Background: Post-stroke cognitive impairments have been shown to increase the risk of depression, however the mechanisms underpinning this association are not well understood. The theory of social isolation in chronic illness proposes that the relationship between symptom severity in chronic conditions and mood disorders is mediated by social isolation. This study therefore aimed to explore the impact of social isolation on depression in stroke survivors with and without cognitive impairments.

Methods: Stroke survivors were recruited ≥6 months post-stroke from the Oxford Screening Programme and completed assessments of cognitive function, social isolation, and depression. Measures of social isolation evaluated both subjective feelings of loneliness and objective social disconnectedness. Multiple linear regressions examined associations of cognition and social isolation with depression symptoms, and serial mediation analyses assessed potential mediating effects of loneliness and social disconnectedness.

Results: Eighty-five participants completed the study measures. Chronic cognitive impairments predicted depressive symptoms in stroke survivors ($\beta = 1.11$, $p < 0.001$). This relationship was mediated by feelings of loneliness, which were associated with higher depression scores in participants with more severe cognitive impairments (indirect effect [IE] = 0.294, $p < 0.05$). Whilst significant direct effects were observed between all variables in the mediation analyses (all $p < 0.05$), there was no evidence for indirect effects of social disconnectedness.

Conclusions: Subjective experiences of loneliness, but not objective social disconnectedness, may increase the risk of depression in stroke survivors with cognitive deficits. These findings suggest that feelings of loneliness may be a suitable target for intervention in post-stroke depression.

1. Introduction

Stroke is one of the leading medical causes of disability worldwide, influencing physical, cognitive, and communicative functioning (Feigin et al., 2025; Goljar et al., 2010). Mental health is strongly affected in both acute and long-term recovery, with estimates suggesting 29% of stroke survivors present with depression and rates remaining stable up to 10 years after the event (Ayerbe et al., 2013). Post-stroke depressive symptoms negatively impact on survival, functional recovery, and quality of life (Dafer et al., 2008; Gbiri et al., 2010). In order to develop preventative strategies and effective interventions, it is crucial that factors contributing to post-stroke depression are identified. Importantly, post-stroke cognitive impairments have been shown to predict poorer mental health outcomes (Nys et al., 2006; Turunen et al., 2018; Williams and Demeyere, 2021). Chronic cognitive impairments affect 20%to 85% of stroke survivors (Demeyere et al., 2016; Sexton et al., 2019) and encompass a wide range of domains, including executive functioning, memory, language, and visuospatial abilities (Jokinen et al., 2015). Most stroke survivors present with multi-domain deficits (Milosevich et al., 2024) which have additive effects on the risk of depression (Williams and Demeyere, 2021). However, the mechanisms by which cognitive impairments may lead to depression are not well understood.

1.1. Social isolation in chronic illness

The middle-range theory of social isolation in chronic illness (Iovino et al., 2023) posits that social isolation is a driving factor in the

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development of depression in persistent physical health conditions. It suggests that precipitating and predisposing factors, such as the frequency and severity of symptoms, increase the risk of social isolation which in turn affects health outcomes including depression. Social isolation is a complex construct, which consists of two interacting elements: social disconnectedness and loneliness. Social disconnectedness is defined as an *objective* measure of social isolation, which reflects a lack of contact with other individuals (Cornwell and Waite, 2009a). In contrast, loneliness refers to the *subjective* experience of social isolation, resulting from a perception of a discrepancy between the relationships one expects and the actual relationships one has (Peplau and Perlman, 1982). Whilst they can co-occur, it has been demonstrated that social disconnectedness and loneliness are distinct concepts which are independently associated with different health outcomes (Cornwell and Waite, 2009b). The physical presence of others is not in itself sufficient to prevent feelings of loneliness (Cacioppo et al., 2015), and conversely it is possible to be socially disconnected without feeling lonely (Hawkley and Cacioppo, 2010). Congruently, the correlation between loneliness and disconnectedness has been found to be weak to moderate (Cornwell and Waite, 2009b; Golden et al., 2009). It has therefore been proposed that there are four categories of social isolation: neither disconnected nor lonely, disconnected but not lonely, lonely but not disconnected, and both disconnected and lonely (Andersson, 1986; Iovino et al., 2023; see Fig. 1A). Evidence for the existence of these subgroups has been provided by Smith and Victor (2019) using latent class analysis. Both social disconnectedness and loneliness are associated with depression in healthy adults, although the underlying mechanisms may differ (Cornwell and Waite, 2009b) and loneliness typically has a stronger effect (Ge et al., 2017). Research on social isolation should therefore assess both social disconnectedness and loneliness to ascertain whether they exert independent and/or additive effects on mental health (Taylor et al., 2018).

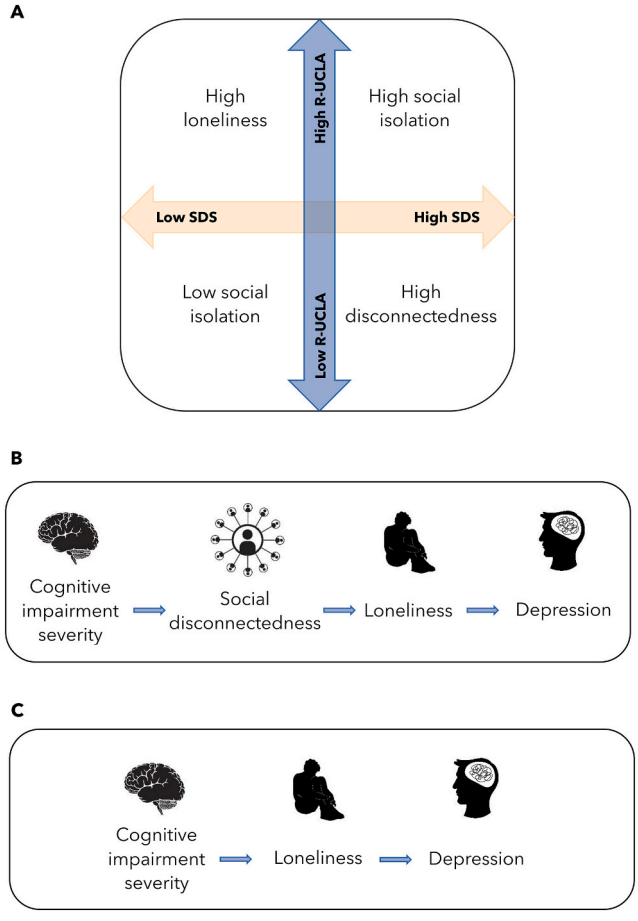

Fig. 1. **(A)** Patterns of social isolation based on social disconnectedness (Social Disconnectedness Scale; SDS) and loneliness (Revised UCLA Loneliness Scale; R-UCLA) scores. **(B)** Serial mediation with cognitive impairment severity causing reduced social connectedness, which results in feelings of loneliness that lead to depression. **(C)** Simple mediation with cognitive impairments causing loneliness (independent of objective social connectedness) which increases the risk of depression.

1.2. The impact of social isolation on post-stroke depression

Iovino et al. (2023) emphasise that their theory remains to be tested empirically for specific illnesses. Applying their hypotheses to chronic symptoms of stroke, we predict that greater severity of cognitive impairments may increase social isolation, which consequently heightens the risk of depressive symptoms. There is emerging evidence that social isolation presents a risk factor for depression in stroke survivors (Goh et al., 2019; Petitte et al., 2015). However, the contribution of cognitive impairments to this relationship remains to be explored. Qualitative studies suggest that there are different pathways by which cognitive impairments may lead to social isolation. First, cognitive impairments may produce practical barriers to social participation. For example, stroke survivors with cognitive impairments indicated that dependence on others restricts their engagement in social and leisure activities (Norlander et al., 2022). Moreover, difficulties with attention and memory interfered with stroke survivors' ability to participate meaningfully in social interactions, leading to a reduced number of social relationships (Bennett et al., 2024). Accordingly, Iovino et al. (2023) suggest a serial mediation process by which this social disconnectedness causes feelings of loneliness, which then result in depressive symptoms (see Fig. 1B). A second pathway involves a direct effect of loneliness through changes in stroke survivors' perceptions of themselves and the quality of their relationships. Individuals with acquired brain injuries who have cognitive impairments report feeling psychologically distant from and misunderstood by others, and alone in their experience of brain injury (Dunne et al., 2023; Salter et al., 2008; Yang et al., 2022). In addition, they describe attempting or feeling pressure to conceal perceived deficits (Crowe et al., 2016; Lowe et al., 2021) which further reduces the quality of social relationships. Thus, there may be a simple mediation process in which cognitive impairments directly heighten experiences of loneliness and thereby depressive symptoms (Iovino et al., 2023; see Fig. 1C) even when the individual objectively has a large social network or participates in social activities.

1.3. Study aims and hypotheses

From a clinical perspective, a better understanding of the effects of social isolation on depression in stroke survivors with chronic cognitive impairments could help identify new and more timely targets and strategies for interventions. This study therefore seeks to clarify the relationship between chronic post-stroke cognitive impairments, social isolation, and depression. We hypothesise that (1) results will replicate and extend previous findings that the severity of cognitive impairments predicts symptoms of depression (Williams and Demeyere, 2021) in the chronic phase post-stroke, (2) social isolation will be associated with higher depression scores, with a stronger effect of loneliness compared with social disconnectedness as previously suggested (Ge et al., 2017), and (3) the relationship between cognitive impairments and depression will be mediated by social isolation.

2. Methods

2.1. Patient and public involvement

Three stroke survivors were consulted regarding the main research question, participant information sheets, and measures of social

isolation to ensure project relevance and accessibility. These experts by experience indicated that the main research questions were relevant for this population based on their personal experiences and contact with other stroke survivors. They emphasised the importance of clarifying the inclusion criteria, for example that individuals did not have to feel depressed or socially isolated to be eligible. In addition, they suggested providing different options for completing questionnaires on sensitive topics where possible (e.g. reading out the questions, filling out questionnaires independently, or asking a relative/carer to assist). Procedures were adjusted to incorporate the recommendations.

2.2. Recruitment and participants

Eighty-five participants were recruited from the Oxford Screening Programme (Milosevich et al., 2024). Stroke survivors were included if they were (1) 18 years or older, (2) admitted with a suspected or confirmed stroke ≥6 months previously, (3) able and willing to provide informed consent, (4) able to sufficiently understand English to complete the study questionnaires, and (5) able to concentrate for approximately 30 min consecutively.

2.3. Study procedure

Ethical approval for the study was obtained from the Central University Research Ethics Committee (CUREC; Ethics Ref: 94589/RE002). Stroke survivors were contacted by phone or email, provided with information sheets, and given the opportunity to ask questions before deciding whether to take part. All participants provided informed consent in accordance with the 1964 Helsinki declaration and its later amendments. In-person sessions were arranged in which participants completed a cognitive screening tool along with questionnaires assessing social isolation and depression. Participants were offered regular breaks throughout the session to minimise effects of fatigue. At the end of the session participants were offered signposting information to mental health support services.

2.4. Demographics and stroke characteristics

Demographic details and stroke characteristics were obtained from participants' records in the Oxford Screening Programme. Information collected included participant age, sex, education (years), type of stroke, lesion hemisphere, time since stroke, acute stroke severity as measured with the National Institutes of Health Stroke Scale (NIHSS), and acute level of functional impairment as indicated by the Barthel Index (BI; Mahoney and Barthel, 1965).

2.5. Cognitive assessment

The Oxford Cognitive Screen (OCS; Demeyere et al., 2015) is a screening tool which was designed to evaluate common cognitive impairments associated with stroke. The OCS assesses six cognitive domains: visuospatial attention, executive function, language, memory, number processing, and praxis. It takes approximately 15–20 min to administer and has good content validity, specificity, and sensitivity for impairments across cognitive domains in stroke survivors (Demeyere et al., 2015). Subtest outcomes were binarized as impaired or unimpaired according to cut-off scores based on normative data. Domains were considered impaired if there was at least one subtest score outside the cut-off range.

2.6. Social isolation measures

Social disconnectedness was measured with the 8-item Social Disconnectedness Scale (SDS; Cornwell and Waite, 2009a) which evaluates social network size and range, frequency of interaction with members of the personal network, household size, number of friends, frequency of socialising with friends, family, and groups, and volunteering activities. The SDS has satisfactory internal consistency (Cronbach's $\alpha = 0.73$) and construct validity (Cornwell and Waite, 2009a). SDS outcomes were converted into z-scores across the sample, with higher scores representing greater social disconnectedness. Loneliness was assessed with the 11-item Revised University of California, Los Angeles Loneliness Scale (R-UCLA; Lee and Cagle, 2017). The R-UCLA uses a three-point Likert scale, with higher scores suggesting greater levels of subjective loneliness. The 11-item R-UCLA has good internal consistency (Cronbach's $\alpha = 0.87$) and content validity (Lee and Cagle, 2017).

2.7. Mental health outcomes

The depression and anxiety subscales from the Hospital Anxiety and Depression Scale (HADS-D and HADS-A, respectively; Zigmond and Snaith, 1983) were used to assess mental health outcomes. The HADS is a 14-item questionnaire with scores ranging from 0 to 21 for each subscale. Higher scores represent greater symptomatology, with scores ≥8 indicating symptoms in the clinical range. This measure has good psychometric properties with sensitivity and specificity at the level of 0.80 (Bjelland et al., 2002) and is suitable for assessing symptoms of mood disorders specifically in stroke survivors (Aben et al., 2002; Ayis et al., 2018).

2.8. Sample size

A power analysis was carried out using the 'mc_power_med' package in R software (version 4.1.2). The required sample size was calculated for a mediation model with two serial mediators using standardised coefficients derived from the existing literature (Cacioppo et al., 2006; Franco-O'Byrne et al., 2023; Glymour et al., 2008; Taylor et al., 2018; Williams and Demeyere, 2021) with 10,000 replications and 20,000 Monte Carlo draws per replication. Results indicated that a minimum sample of 78 participants was needed to achieve 80% power to detect the main effects of interest, namely simple and serial mediation for the relationship between cognitive impairment severity, social isolation, and depression.

2.9. Statistical analyses

All statistical analyses were completed in R (version 4.1.2). Descriptive statistics were used to summarise sample demographics and clinical characteristics. Pairwise Pearson correlations were performed between HADS scores and age, education (years), time since stroke (years), and NIHSS scores, with a two-tailed independent *t*-test run to determine potential sex differences in depression scores. Imputation of missing NIHSS data was carried out using predictive mean matching (PMM) procedures with the 'mice' package. A data-driven approach was used to select covariates for analyses in order to increase predictive accuracy whilst minimising the risk of overfitting analysis models. Specifically, variables which were significantly (alpha < 0.05) correlated with the main outcome of interest (HADS depression scores) were included as covariates in all analyses.

Multiple linear regressions were used to assess whether previous findings of a significant relationship between cognitive impairments and depression at 6 months post-stroke (Williams and Demeyere, 2021) were replicated in this sample of chronic stroke survivors. To account for non-normal distributions of residuals, robust multivariate regression models were created using the '*lmrob*' function from the 'robustbase' package. Number of impaired cognitive domains was specified as the predictor and HADS-D score as the outcome variable. Secondary analyses explored the relationship between impairments on individual cognitive domains with depression scores. False discovery rate (FDR) corrections were applied to these analyses using the Benjamini-Hochberg procedure to reduce Type-I error inflation associated with performing multiple tests

(Glickman et al., 2014).

Next, the association between social isolation and depression was examined. As discussed above, social isolation and loneliness are hypothesised to be related but distinct constructs. Exploratory analyses therefore evaluated the impact of specific patterns of social isolation on post-stroke depression (see Fig. 1A). Participants were categorised into four groups based on median splits of SDS and R-UCLA data to represent the subgroups proposed by Andersson (1986) and enhance clinical interpretability: (1) high social isolation (high SDS and R-UCLA scores), (2) high loneliness (high R-UCLA and low SDS scores), (3) high disconnectedness (high SDS and low R-UCLA scores), and low social isolation (low SDS and R-UCLA scores). The relationship between social isolation patterns and depression scores was examined with multiple linear regression analyses. Sensitivity analyses using continuous SDS and R-UCLA outcomes are reported in the Supplementary Materials (see Appendix A). The main analyses investigated potential mediation effects of social disconnectedness and loneliness in the relationship between post-stroke cognitive impairments and depression. Serial mediation models were generated using the 'lavaan' package, with number of impaired cognitive domains as the predictor, SDS and R-UCLA scores as causally ordered mediators, and HADS-D score as the outcome variable. All mediation models were fitted using non-parametric bootstrap resampling with 10,000 iterations. Finally, exploratory multiple regression and mediation models were run to assess the impact of cognitive impairment severity and social isolation on anxiety symptoms as measured with the HADS-A. As depression was the main outcome of interest and depression and anxiety scores were highly correlated, results for HADS-A scores are reported in the Supplementary Materials (see Appendix B).

3. Results

Eighty-five participants completed the cognitive assessment and questionnaires. Descriptive statistics for demographic variables, clinical characteristics, and outcome measures are provided in Table 1. All participants were assessed in the chronic stroke phase, with assessments completed an average of 4.5 ($SD \pm 2.8$) years post-stroke. Forty-eight (56.4%) participants presented with one or more impaired cognitive domains upon assessment. Cognitive impairments assessed using the OCS were prevalent across domains except for executive dysfunction, which was observed in only four (4.8%) participants. A total of 27 (31.8%) participants scored in the clinical range on the HADS-D and 23 (27.1%) participants had scores in the clinical range on the HADS-A. Comorbidity of depression and anxiety was high, with 15 (17.6%) participants presenting with clinical symptoms on both scales ($r(83) = 0.54$, $p < 0.001$). Significant correlations were observed for time since stroke ($r(83) = -0.14$, $p < 0.05$) and years of education ($r(83) = -0.227$, $p < 0.05$) with depression (see Appendix C), which were therefore included as covariates in the analyses. Adding stroke severity as measured by the NIHSS as a covariate did not alter the results (see Appendices C-F). HADS-D scores did not differ between male and female participants ($t(83) = 1.26$, $p = 0.210$).

3.1. Cognitive impairment and post-stroke depression

A multiple linear regression adjusted for covariates was conducted to determine effects of cognitive impairment severity on depression scores. A greater number of impaired cognitive domains predicted higher depression scores in chronic stroke ($\beta = 1.11$, $t = 3.63$, $p < 0.001$; see Fig. 2A). Additional analyses by domain demonstrated that specific impairments in visuospatial attention ($\beta = 2.58$, $t = 3.81$, FDR-corrected $p < 0.05$) and language ($\beta = 4.83$, $t = 4.57$, FDR-corrected $p < 0.001$) were associated with greater depressive symptomatology (see Figs. 2B-2G). No significant associations with depression were observed for any of the remaining cognitive domains following adjustment for covariates (all FDR-corrected $p > 0.05$, see Appendix D).

Table 1
Participant characteristics and outcome scores ($N = 85$).

		Median (IQR)	Range
Age – mean (*SD*)	72.8 (12.6)	75.6 (17.0)	32.6–98.0
Sex – *n* (%)			
Male	54 (63.5%)		
Female	31 (36.5%)		
Years of education – mean (*SD*)	13.6 (3.8)	12.0 (5.0)	9–23
Ethnicity – *n* (%)			
White British	80 (94.1%)		
White Irish	1 (1.2%)		
White other	2 (2.4%)		
Black other	1 (1.2%)		
Indian	1 (1.2%)		
Stroke hemisphere – *n* (%)			
Left	38 (44.7%)		
Right	32 (37.7%)		
Bilateral	8 (9.4%)		
Unknown	7 (8.2%)		
Stroke subtype – *n* (%)			
Ischaemic	64 (75.3%)		
Haemorrhagic	18 (21.2%)		
Unknown	3 (3.5%)		
First or recurrent stroke – *n* (%)			
First	59 (69.4%)		
Recurrent	23 (27.1%)		
Unknown	3 (3.5%)		
Years post-stroke – mean (*SD*)	4.5 (2.8)	3.6 (3.8)	0.8–13.3
Acute NIHSS score[a] – mean (*SD*)	7.4 (5.2)	6.0 (8.0)	0–21
Acute BI score[b] – mean (*SD*)	10.63 (5.65)	9.00 (8.0)	0.0–20.0
OCS total – *n* (%)			
0 domains impaired	37 (43.5%)		
1 domain impaired	20 (23.5%)		
≥2 domains impaired	28 (32.9%)		
OCS subdomain impaired – *n* (%)			
Visuospatial attention	26 (30.1%)		
Executive functioning	4 (4.8%)		
Language	19 (22.4%)		
Memory	19 (22.4%)		
Number processing	14 (16.5%)		
Praxis	21 (24.7%)		
Social isolation scores – mean (*SD*)			
SDS	0.0 (0.4)	0.0 (0.6)	−1.1–1.4
R-UCLA	17.4 (5.3)	16.0 (6.0)	11.0–22.0
HADS total scores – mean (*SD*)			
Depression subscale	5.7 (4.2)	5.0 (6.0)	0.0–17.0
Anxiety subscale	5.5 (3.5)	5.0 (5.0)	0.0–15.0
HADS-D range – *n* (%)			
Normal	58 (68.2%)		
Clinical	27 (31.8%)		
HADS-A range – *n* (%)			
Normal	62 (72.9%)		
Clinical	23 (27.1%)		

IQR = Interquartile range; *SD* = standard deviation; NIHSS = National Institutes of Health Stroke Scale; BI = Barthel Index; OCS = Oxford Cognitive Screen, SDS = Social Disconnectedness Scale; R-UCLA = Revised University of California, Los Angeles Loneliness Scale; HADS-D = HADS depression subscale; HADS-A = HADS anxiety subscale.
[a] *n* assessed = 74; [b] *n* assessed = 49.

3.2. Impact of social isolation on post-stroke depression

The impact of social isolation on post-stroke depression scores was assessed across social isolation categories (see Fig. 3 and Appendix E). Multiple linear regressions adjusted for covariates showed that participants with high overall social isolation ($\beta = 5.17$, $t = 5.68$, $p < 0.001$) or loneliness ($\beta = 3.02$, $t = 5.68$, $p < 0.01$) scores were more depressed than participants with low social isolation scores. In contrast, participants who were more socially disconnected (but had low loneliness scores) did not differ significantly from individuals who had low overall social isolation scores ($\beta = 1.35$, $t = 1.35$, $p = 0.18$). This suggests that the presence of subjective feelings of loneliness is critical in the relationship between social isolation and post-stroke depression.

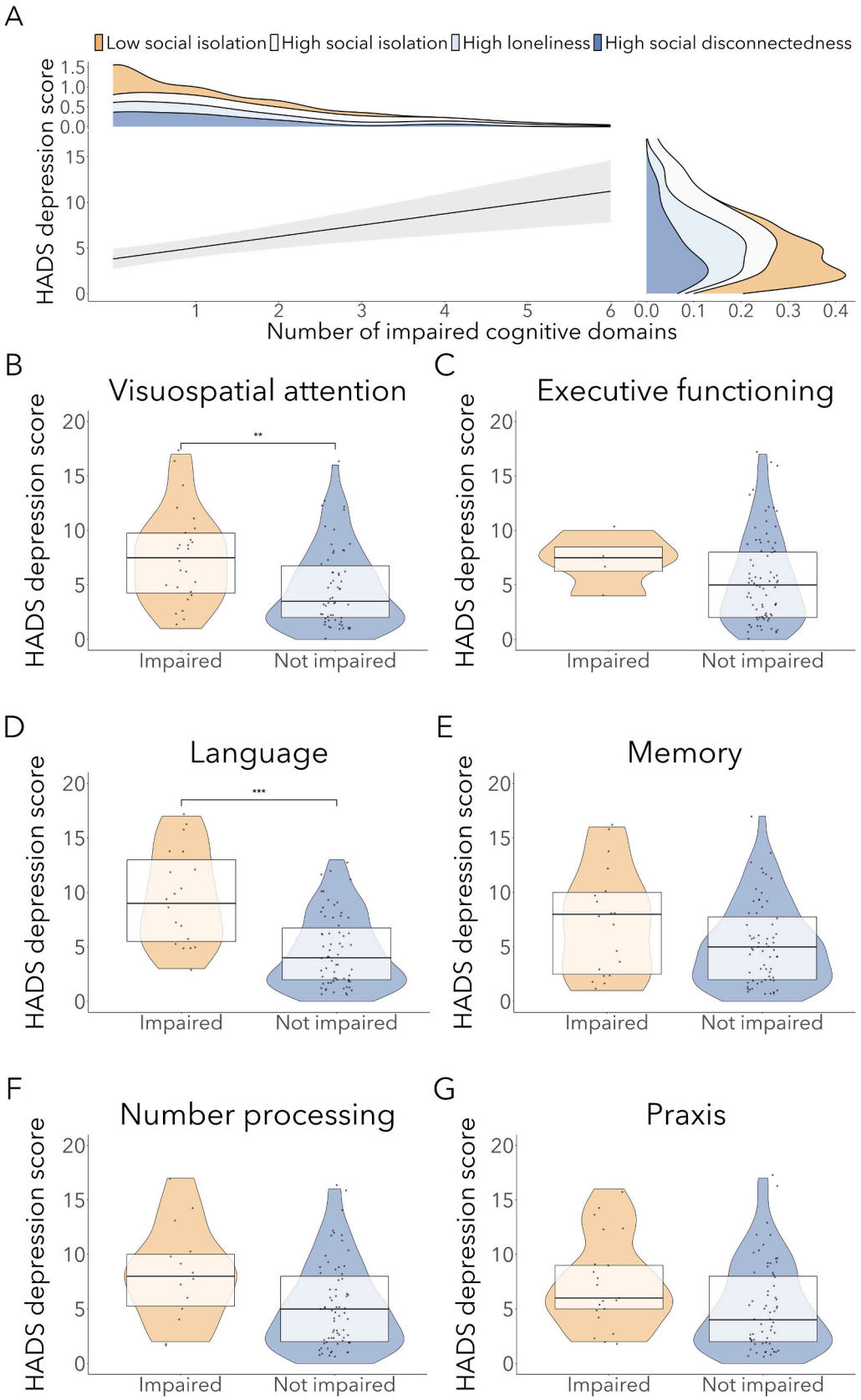

Fig. 2. **(A)** A greater number of impaired cognitive domains on the OCS predicts higher depression scores. The solid line represents the multiple linear regression formula, with the shaded area indicating standard errors. Density plots indicate the distribution of HADS-D scores and number of cognitively impaired domains across social isolation categories. **(B-G)** Violin plots demonstrating the distributions of depression scores by impairment across cognitive domains. Impairments in the visuospatial attention and language domains predicted higher depression scores (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ for FDR-corrected p-values).

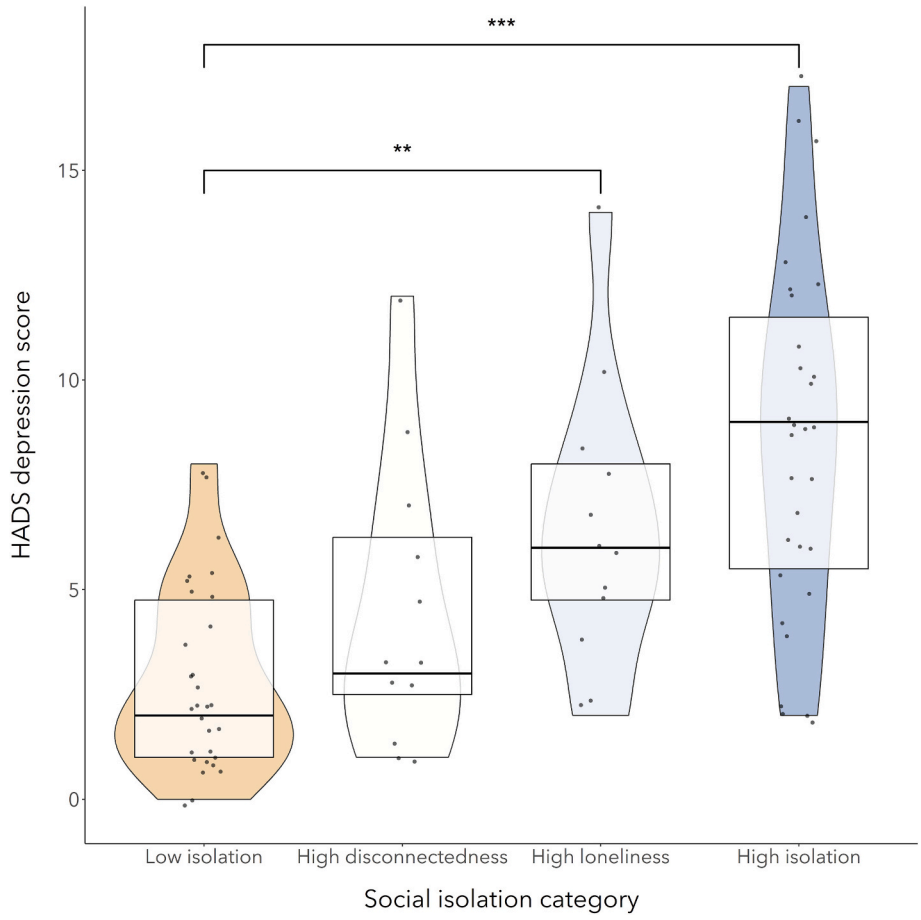

Fig. 3. Violin plots showing the distributions of depression scores by social isolation category (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$). Small dots represent raw data points, with box plots indicating median scores alongside the first and third quartiles of HADS-D scores.

3.3. *Loneliness mediates effects of cognitive impairment on depression*

The relationship between cognitive impairments, social isolation, and depression was evaluated with serial mediation models. All pathways showed significant direct effects (see Fig. 4), such that greater cognitive impairment severity predicted higher social disconnectedness, loneliness, and depression scores. In addition, social disconnectedness and loneliness had significant direct effects on depression outcomes.

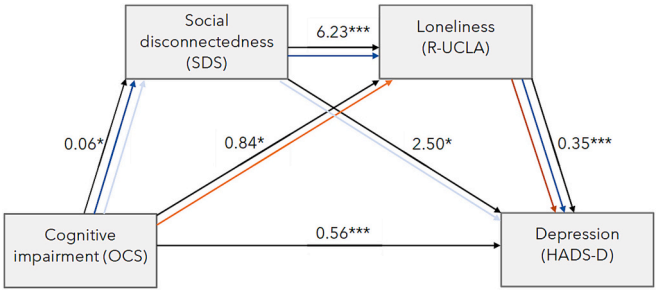

Fig. 4. Graphical representation of the serial mediation analysis adjusted for covariates, with coefficients for direct effects (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$) indicated by black arrows. Loneliness was a significant mediator in the relationship between cognitive impairment and depression ($p < 0.05$; orange arrows). A serial mediation pathway via social disconnectedness and loneliness was not significant following adjustment for covariates ($p = .0.08$; dark purple arrows). Finally, there was no evidence that social disconnectedness mediated associations between cognitive impairment and depression ($p = 0.125$; light purple arrows).

Participants with a greater number of impaired cognitive domains reported stronger feelings of loneliness, which predicted higher depression scores (see Table 2). This indirect effect was maintained following adjustment for significant covariates. In non-adjusted models a serial mediation effect was observed, with the effects of cognitive impairment on depression being sequentially mediated by social disconnectedness and loneliness. However, this effect was not significant after adjusting for years of education and time since stroke. Consistent with the analyses of social isolation categories reported above, there was no evidence that social disconnectedness independently mediated the relationship between cognitive impairment severity and depression.

4. Discussion

This study explored the role of chronic cognitive impairments and social isolation in post-stroke depression. More severe cognitive deficits predicted higher depression scores in stroke survivors. Consistent with the theory of social isolation in chronic illness (Iovino et al., 2023), this relationship was mediated by feelings of loneliness. However, there was no evidence that social disconnectedness significantly altered the association between cognitive impairments and depression after stroke. Taken together, these findings suggest that loneliness presents a possible mechanism by which cognitive impairments increase the risk of post-stroke depression.

4.1. *Cognitive impairment severity predicts depression in chronic stroke*

Stroke survivors with more severe, multi-domain cognitive deficits

Table 2
Indirect effects of the serial mediation model of the relationship between cognitive impairment, social isolation, and depression after stroke.

	Model 1[a]				Model 2[b]			
	Estimate (*SE*)	z-score	*p*-value	*CI*	Estimate (*SE*)	z-score	*p*-value	*CI*
Indirect effect 1: Cog → Disc → Dep	0.182 (0.109)	1.665	0.096	0.040–0.481	0.155 (0.101)	1.535	0.125	0.022–0.427
Indirect effect 2: Cog → Lon → Dep	0.361 (0.152)	2.374	<0.05	0.123–0.743	0.294 (0.144)	2.044	<0.05	0.071–0.656
Indirect effect 3: Cog → Disc → Lon → Dep	0.197 (0.090)	2.197	<0.05	0.076–0.452	0.135 (0.077)	1.757	0.079	0.030–0.362
Total effect	1.331 (0.294)	4.533	<0.001	0.811–1.960	1.140 (0.292)	3.909	<0.001	0.625–1.762

Abbreviations: Cog = number of impaired cognitive domains on the OCS; Disc = social disconnectedness (SDS) score; Dep = depression (HADS-D) score; Lon = loneliness (R-UCLA) score; *SE* = standard error; *CI* = confidence interval
[a] Unadjusted model; [b] Adjusted for education and time since stroke.

have been reported to be at higher risk of depression at 6 months post-stroke (Williams and Demeyere, 2021). The present study, in which assessments were completed an average of 4.5 years post-stroke, shows that this relationship is maintained throughout the chronic stroke phase. Cognitive impairment severity is therefore a consistent predictor of mental health after stroke.

Analyses by cognitive domain suggested that language and visuospatial attention deficits most strongly influenced post-stroke depression. This corresponds with a longitudinal study by Nys et al. (2006), which showed that acute visuospatial and language deficits increased risk of depression at 6 months post-stroke. In contrast, previous studies using the OCS found that language, memory, number processing, visuospatial attention, and praxis impairments predicted depression symptoms (Kelleher et al., 2025; Williams and Demeyere, 2021). These divergent results may be due to the larger sample sizes and consequently greater power of previous studies to detect smaller effects. It should be noted that sample size for the present study was calculated based on overall cognitive impairment severity, and analyses for individual domains were therefore exploratory by nature. It is possible that a larger-scale study would have revealed effects of other cognitive domains on depression in addition to language and visuospatial attention deficits.

Evidence for the impact of executive dysfunction as measured by the OCS has been mixed. Whilst significant effects on depression were reported by Williams and Demeyere (2021), this finding was not replicated by Kelleher et al. (2025). The low number of participants presenting with executive dysfunction in the current study limited our ability to test associations between this domain and depressive symptoms. Additional research is needed to clarify potential associations between executive function and depression after stroke.

4.2. Differential effects of social disconnectedness and loneliness

As well as cognitive impairment severity, social isolation was a significant predictor of higher depression scores after stroke. When investigating specific patterns of social isolation, participants with stronger feelings of loneliness presented with more depressive symptoms. Importantly, whilst concurrent social disconnectedness numerically increased predicted depression scores, this additive effect was not significant. Moreover, stroke survivors who were objectively socially disconnected but had low subjective loneliness scores did not differ in mood ratings from participants who were not socially isolated. Consistent with research in healthy populations (Lee et al., 2021), this suggests that the experience of loneliness is an important predictor of post-stroke depression independent of individuals' social network and/or extent of social participation.

Crucially, mediation analyses showed that loneliness mediates the previously observed relationship between cognitive impairment severity and depression. A higher number of impaired cognitive domains was associated with stronger feelings of loneliness, which in turn predicted increased depressive symptoms. In contrast, serial mediation effects in which social disconnectedness preceded loneliness were not significant after adjusting for demographic and clinical covariates. Altogether, these findings suggest that the impact of cognitive impairments on stroke survivors' perception of and/or satisfaction with their relationships, rather than on their social connectedness, contributes to poorer mental health outcomes. There was thus evidence for the simple, but not the serial mediation pathway proposed by Iovino et al.'s (2023) theory of social isolation in chronic illness in this population.

4.3. Clinical implications

These findings have clear implications for the treatment of post-stroke depression. First, they demonstrate that effects of cognitive impairments on depression do not dissipate over time. It is therefore crucial that effective strategies are developed to support the mental health of stroke survivors with cognitive deficits. Whilst cognitive rehabilitation may improve neuropsychological functioning to some extent (Gillespie et al., 2015), pervasive cognitive impairments are common even in stroke survivors who have made a successful clinical recovery (Jokinen et al., 2015; Planton et al., 2012). For individuals with chronic cognitive impairments, social isolation may present a modifiable factor which could improve mental health outcomes. When designing suitable interventions for this population, it is important to consider that loneliness but not disconnectedness significantly mediated the relationship between cognition and depression. This suggests that strategies aiming to increase stroke survivors' social networks and participation may not be sufficient to ameliorate depressive symptoms, and interventions may be more effective when targeting feelings of loneliness and the cognitions associated with these.

In clinical practice, this could be implemented by using measures to assess for the presence of feelings of loneliness and testing effects of targeted interventions for loneliness. A meta-analysis of loneliness interventions indicated that approaches based on cognitive behavioural therapy (CBT) models are most effective in reducing the experience of loneliness (Masi et al., 2011). According to the social cognition model of loneliness, subjective perceptions of being socially isolated lead to negative cognitive biases towards social threats as well as behaviours which confirm maladaptive thoughts and beliefs (Cacioppo et al., 2015). Qualitative research suggests that, in adults with cognitive impairments following an acquired brain injury, common cognitions and behaviours may include a reluctance to share distressing thoughts and feelings with others (Crowe et al., 2016; Yang et al., 2022), a perceived lack of understanding of their experience by other people (Lowe et al., 2021; Salter et al., 2008), a heightened awareness of differences between oneself and other people (Yang et al., 2022), and attempts to conceal cognitive deficits (Lowe et al., 2021). CBT approaches aim to address maladaptive cognitions and behaviours which inadvertently maintain feelings of loneliness. To date, the application of CBT for loneliness has predominantly been assessed in cognitively unimpaired older adults (Cohen-Mansfield and Parpura-Gill, 2007; Dworschak et al., 2024; Theeke et al., 2016) and young people (Käll et al., 2020). Further research is needed to determine the effectiveness of and potential

adaptations (e.g. Ponsford et al., 2020) to this intervention for loneliness and associated symptoms of depression in stroke survivors with cognitive impairments.

4.4. Strengths and limitations

The participant sample recruited in this study presented with similar rates of depression and cognitive impairments reported in previous large-scale studies (Ayerbe et al., 2013; Demeyere et al., 2016; Sexton et al., 2019), indicating that clinical characteristics were representative of this population. However, there was a lack of ethnic diversity and acute stroke severity was predominantly in the minor to moderate range. Additional work is needed to explore the generalisability of our findings across demographic characteristics and individuals with more severe stroke.

Whilst grounded in a clear theoretical framework, it was not possible to determine causality of the associations observed in the present study. Previously, it has been shown that cognition, loneliness, and depression have reciprocal influences (Cacioppo et al., 2006; Liang et al., 2023; Sbarra et al., 2023), complicating investigations of the relationship between these three constructs. However, longitudinal studies have consistently demonstrated that baseline cognitive impairments can precede both depression (Nys et al., 2006) and social isolation (Duan et al., 2023), as well as loneliness scores predicting the onset of depression (Kraav et al., 2021; Mann et al., 2022; Martín-María et al., 2021; Van Winkel et al., 2017). Moreover, analyses using cross-lagged analytical models indicated that loneliness predicted changes in depressive symptoms, whereas the reverse relationship was not significant (Cacioppo et al., 2010). The current evidence base therefore suggests that loneliness is a causal factor in the development of depression. In the present study, a stroke-specific screening tool was used to assess common cognitive sequelae associated with stroke, such as hemispatial neglect, apraxia, and aphasia, in addition to more general impairments in memory and executive functioning. Whilst depression has been linked with changes in a range of domains, including memory, attention, and executive functioning (Rock et al., 2014), it is unlikely that low mood caused more stroke-specific impairments as measured with the OCS. Going forward, longitudinal studies and clinical trials will be instrumental for establishing the directional effects of domain-general cognitive impairments and loneliness on post-stroke depressive symptoms.

Finally, all analyses were adjusted for relevant clinical and demographic characteristics. Although beyond the scope of the present study, more in-depth explorations would be valuable for understanding the impact of specific variables. First, whilst the data-driven approach to covariate selection did not include sex as a potential confounding variable, there is emerging evidence that relationships between cognition, social isolation, and depression may differ between men and women (Chi et al., 2024; Wan et al., 2025). It would therefore be worth investigating whether the pathways outlined here are similar or vary by sex or gender. In addition, due to a high proportion of missing data on the Barthel Index it was not possible to impute missing values for this measure and determine the potential impact of functional abilities on social isolation or depression. However, as Barthel Index scores are strongly associated with NIHSS scores (Kwakkel et al., 2010), it was attempted to account for possible confounding effects of functional impairments by including stroke severity as a covariate. The addition of NIHSS scores did not alter the main results, suggesting that the effects of cognitive impairments and loneliness on depression were independent of stroke severity. This is consistent with our previous finding that the inclusion of NIHSS and Barthel Index scores did not affect the observed relationship between post-stroke cognition and depression (Williams and Demeyere, 2021). Given the high prevalence of physical disability after stroke, however, it would be of interest to explore potentially independent contributions of physical dysfunction to social isolation and depression in future studies.

5. Conclusions

This study showed that the extent of stroke survivors' cognitive impairments consistently predicts symptoms of depression. Feelings of loneliness mediated this relationship and may therefore present a modifiable target for interventions aiming to improve depression in this population. Priorities for further research include establishing causality of these relationships and examining the potential for interventions targeting loneliness in depressed stroke survivors with cognitive impairments.

CRediT authorship contribution statement

Margot Juliëtte Overman: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Reena Vohora:** Writing – review & editing, Supervision. **Nele Demeyere:** Writing – review & editing, Supervision.

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Declaration of competing interest

ND is a developer of the Oxford Cognitive Screen.

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Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2026.121305.

Data availability

Raw data and analysis scripts can be accessed on https://osf.io/gu4f5.

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