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Relationship of subjective and objective cognition with post-stroke mood differs between early and long-term stroke

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ABSTRACT

Objective: Depression and anxiety affects approximately 1 in 3 stroke survivors. Performance on standardized objective cognitive tests and self-reported subjective cognitive symptoms are associated with concurrent depression and anxiety, but longitudinal data on whether and how objective and subjective cognition relate to emotional outcomes are lacking. **Method:** $N=99$ stroke survivors (M age = 68.9, $SD=13.1$; Median NIH Stroke Severity = 5) completed measures of depression and anxiety (Hospital Anxiety and Depression Scale; HADS), objective cognition (Oxford Cognitive Screen) and subjective cognitive symptoms (Cognitive Failures Questionnaire) at 6-months, 4.5, and 5.5 years post-stroke. The contribution of objective and subjective cognition to depression and anxiety was determined *via* mixed-effects models. **Results:** We found no evidence that age, stroke severity, years of education, and participant sex related to changes in HADS-Depression or HADS-Anxiety scores (Marginal R^2=0.03 and 0.05, respectively). Objective cognitive impairments at 6-months and increases in subjective cognitive symptoms at 5.5 years significantly related to increased HADS-Depression scores (Marginal R^2=0.22). Only increases in subjective cognitive symptoms at 5.5 years significantly related to increased HADS-Anxiety scores (Marginal R^2=0.20). When conducting models in reverse, HADS-Depression and HADS-Anxiety scores did not reciprocally explain changes in subjective cognitive symptoms. **Conclusions:** Objective cognitive abilities are more strongly associated with depression at 6-months post-stroke, while subjective cognitive symptoms are more relevant to both long-term post-stroke depression and anxiety. There may be a unique unidirectional influence of subjective cognitive symptoms to post-stroke depression and anxiety.

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Introduction

Globally, stroke is the leading cause of chronic disability in physical, psychological, and functional domains (Feigin et al., 2014). The impact of stroke often causes emotional distress, with post-stroke depression and anxiety prevalence rates nearly double the general population (Schöttke & Giabbiconi 2015; Cumming et al., 2016). Elevated depression and anxiety can persist for at least up to 10 years post-stroke (Ayerbe et al., 2014). Given this, investigating causes of post-stroke depression and anxiety has been ranked as the number one long-term research priority by stroke survivors, carers, and clinicians (Hill et al., 2022).

In addition to mood disorders, stroke commonly causes cognitive impairment, such as attention, memory, language, and executive function difficulties. Stroke survivors often experience multi-domain cognitive impairments (Demeyere et al., 2016, 2019; Leśniak et al., 2008) that can negatively affect everyday activities and participation (Bickerton et al., 2015; Demeyere et al., 2019; Mole & Demeyere, 2020; Nys et al., 2005). Performance on objective, neuropsychological cognitive tasks more strongly correlates with post-stroke depression over traditional predictors such as age, sex years of education and physical independence (Barker-Collo, 2007; Hackett & Anderson, 2005) and can more than double depression risk at 6 months post-stroke (Williams & Demeyere, 2021). Though post-stroke anxiety is less frequently researched, some studies report associations to objective cognitive impairment (Fure et al., 2006; Shimoda & Robinson, 1998; Tang et al., 2021).

Post-stroke depression and anxiety are not only related to objective cognitive performance on standardized tests. Subjective cognitive symptoms, defined as whether individuals self-report cognitive difficulties, are thought to be in part related to emotional concern about cognitive impairment, and have been shown to relate to everyday functional outcomes. In systematic reviews, subjective cognitive symptoms have weak associations with objective cognitive performance (van Rijsbergen et al., 2014), but have a stronger correlation with post-stroke depression (Duits et al., 2008; Nijsse et al., 2017), work and leisure activities (McKevitt et al., 2011), self-efficacy (Aben et al., 2011) and fatigue (Maaijwee et al., 2014; van Rijsbergen et al., 2019). Subjective cognitive symptoms are important because they tend to increase over time post-stroke (van Rijsbergen et al., 2014) and can be a precursor to cognitive decline and dementia risk (Mitchell, 2008; Juncos-Rabadán et al. 2012).

The relationship between emotional distress and both objective and subjective cognition post-stroke has several potential pathways. Given stroke causes objective cognitive impairment, at least some unidirectionality can be assumed in that greater severity of objective cognitive impairment causes emotional distress. For example, difficulties recalling positive events that sustain a good mood (Murphy et al., 2019). Alternatively, sufficiently severe objective cognitive impairments could cause functional impairments in daily activities that result in lowered mood (Mole & Demeyere, 2020), or potentially increase worry about future cognitive status and its implications on daily life (Salter et al., 2008). Outside of stroke, severe levels of depression can also cause "pseudodementia" (Brodaty & Connors, 2020) leading to impaired performance on neuropsychological tests; it possible this pattern emerges post-stroke as well. Similarly, generalized anxiety has been shown to relate to reduced episodic memory and executive function abilities (Beaudreau & O'Hara, 2008); further meta-analyses have demonstrated that high levels of anxiety can elevate risk for future mild cognitive impairment and dementia (Desai et al., 2021).

The relationship between subjective cognition to emotional distress could also be bidirectional. Possibly, the onset of post-stroke depression and anxiety increases cognitive resources toward rumination, worry, and metacognitive beliefs that lead to cognitive lapses during functional tasks (e.g., Szyszkowska & Bala, 2023). Outside of stroke, theoretical models of anxiety (Koerner & Dugas, 2006) and depression (Beck, 1974) state that negative subjective evaluations and interpretations of abilities, including overly negative perceptions of cognitive abilities such as forgetfulness and inattention, increase risk of emotional distress, and over time, worsen depression and anxiety symptoms. Collectively, both objective and subjective cognitive symptoms may be a risk factor for post-stroke depression and anxiety

Though objective cognitive performance relates to depression cross-sectionally within early stroke (Maaijwee et al., 2014; Williams & Demeyere, 2021; Hackett & Anderson, 2005), subjective cognitive symptoms may be a stronger correlate (van Rijsbergen et al., 2014). Comparatively, the relationship between objective and subjective cognition and anxiety has been less frequently researched. In a systematic review, Menlove et al. (2015) reported that objective cognitive impairment is associated with concurrent anxiety post-stroke. However, to our knowledge, a potential link between subjective cognition and post-stroke anxiety has not yet been examined. Importantly, objective and subjective cognition have not been considered together as predictive factors for post-stroke mood. Examining these variables together in understanding post-stroke mood outcomes is important given the common risk of negative subjective evaluations to depression and anxiety severity outside of stroke (Cisler & Koster, 2010; Kircanski et al., 2012), and overlap between depression and anxiety symptoms post-stroke (Barker-Collo, 2007; Wright et al., 2017).

Finally, links between objective and subjective cognition to post-stroke mood have predominantly been examined cross-sectionally. Existing longitudinal research has focused only on objective cognitive impairments (Downhill & Robinson, 1994; Nys et al., 2006) and within the first-year post-stroke (Lo et al., 2022). Longitudinal data allows for modelling within-individual change over time, rather than comparing potentially very different stroke cohorts cross-sectionally at varying time points, and can provide stronger evidence for potential causality. Importantly, the documented improvements in objective cognitive abilities, and changes in subjective cognitive symptoms over time post-stroke (Hochstenbach et al., 2003; van Rijsbergen et al., 2014) may impact depression and anxiety outcomes.

Study aim

The study aim was to determine whether changes in subjective cognitive symptoms and objective cognitive impairment from 6 months to long-term (>2 years post-stroke) differentially predict changes in depression and anxiety severity post-stroke. We made no hypotheses regarding how changes in objective and subjective cognition over time relate to post-stroke depression and anxiety severity, given the lack of longitudinal data examining this relationship.

Methods

Data collection at 6 months (Oxford Cognitive Screening Programme; Reference 14/LO/0648 & 18/SC/0550) and in long-term stroke (OX-CHRONIC Study; Reference 19/SC/0520) was approved by National Research Ethics Committees. All participants provided informed consent to take part.

Participants and data structure

This study is a secondary observational analysis of the OX-CHRONIC study, a longitudinal stroke cohort of post-stroke psychological outcomes (Demeyere et al., 2021; Kusec et al., 2023). Participants were initially recruited from the John Radcliffe Hospital acute stroke ward in Oxford, UK as part of the Oxford Cognitive Screening programme (2012 – 2020). Inclusion criteria were (1) diagnosis of stroke (first ever or recurrent); (2) age 18 years or older; (3) ability to remain alert for 20 min and (4) ability to provide informed consent. Participants consenting to future research opportunities at a 6-month follow-up assessment (Time 1) and who were ≥2 years post-stroke ($N = 208$) were approached for participation in OX-CHRONIC. Participants consenting to OX-CHRONIC completed a battery of self-report and neuropsychological measures remotely across two time points, one year apart (Time 2 [*M* years since stroke = 4.56] and Time 3 [*M* years since stroke = 5.56 years). OX-CHRONC data were combined with 6-month assessment data to form the current dataset. To account for any language impairments affecting instruction comprehension, 6-month assessments were conducted with a researcher present so as to provide any additional support necessary. Similarly, in OX-CHRONIC, cognitive assessments were conducted live with a researcher. Given the study sample had low prevalence of semantic impairments (6-months 3.03%; 4.5 years 0.00%, 5.5. years 1.12%; see Supplementary Materials) it is unlikely that difficulties following instructions affected completion of study measures.

Participant demographic information and stroke characteristics were obtained from hospital records with participant consent.

Study measures

Participants completed the below measures at each time point.

Depression and anxiety were assessed using the *Hospital Anxiety and Depression Scale* (HADS; Zigmond & Snaith, 1983), a 14-item measure (7 items HADS-Depression; 7 items HADS-Anxiety). Each item is rated on 0—3 Likert scale, with scale anchors varying by question (e.g., “not at all” to “most of the time” for item 1; “hardly at all” to “definitely as much” for item 2). Higher scores indicate greater symptom severity. Cronbach’s alpha for the HADS-Depression scale was 0.80, and 0.79 for HADS-Anxiety.

Subjective cognitive symptoms were assessed using the *Cognitive Failures Questionnaire* (CFQ; Broadbent et al., 1982), a 25-item measure of day-to-day cognitive difficulties (e.g. in memory, attention, perception). Items are answered on a 0 ("Never") to 4 ("Very often") Likert scale. Previous studies have indicated that the CFQ is best represented as a global measure of subjective cognitive difficulties (Goodman et al., 2022); here we use CFQ total scores in analyses. Higher scores indicate greater subjective cognitive symptoms. Cronbach's alpha in current sample was 0.91.

Objective cognitive impairment was assessed using the *Oxford Cognitive Screen* version A (OCS; Demeyere et al., 2015), a validated stroke-specific measure of cognition in domains of memory, spatial attention, praxis, numeracy, language, and executive function. Due to the COVID-19 pandemic, long-term follow up at Time 2 and 3 used the Tele-OCS version A (Webb et al., 2023), which contains 10 OCS subtests (picture naming, semantics, sentence reading, orientation, verbal memory, episodic memory, number writing, calculations, hearts cancellation, and trail making). To ensure similar testing conditions between the in-person OCS and Tele-OCS, at the start of OX-CHRONIC assessments participants were instructed to sit at a table in a quiet setting without interference (e.g. from television). OCS stimuli were posted in a sealed envelope prior to the assessments. Participants were instructed to keep these materials sealed until directed to open them during the assessment. Participants were instructed when to turn each page in the booklet to see the stimuli pertaining to each OCS subtask. Participants scoring <5th centile relative to normative performance by healthy adults were considered impaired in that subtest. Total proportion of subtests impaired was used as a measure of cognitive impairment severity (e.g. Milosevich et al., 2024).

Data management and sample power

For any self-report questionnaire with <25% missing item-level data, totals were estimated using mean imputation based on responses from non-missing items. Any participant with ≥25% of HADS or CFQ items missing was treated as a missing observation for that questionnaire. Proportion of OCS subtests impaired was used in analyses (versus number of subtests impaired) to accommodate for any missingness. CFQ scores and OCS proportion of subtests impaired were standardized using z-scores (*Mean* = 0, standard deviation = 1) to allow for direct comparisons between independent predictors in analyses.

In a post-hoc power analysis, the sample was adequately powered to detect a moderate effect size of at least 0.31 with 80% power and an alpha of 0.05 in a within-subjects repeated measures ANOVA.

Statistical analyses

Analyses were performed using R version 4.2.1 (R Core Team, 2022). Analysis code can be found at https://osf.io/6hdcs/. OX-CHRONIC study data has been deposited in full to Dementia Platforms UK where it can be accessed: https://portal.dementiasplatform.uk/.

Descriptive statistics for demographic variables were calculated. Attrition analyses, including whether participants consenting to OX-CHRONIC differed from those who did not consent, were conducted. Depression and anxiety prevalence rates from

6-months to long-term stroke are reported. To characterize depression and anxiety trajectories from 6 months to long-term stroke, and the degree to which they were predicted by post-stroke demographic variables, "baseline" mixed-effects models were conducted using the *nlme* R package (Pinheiro & Bates, 2022). We then conducted the final longitudinal mixed-effects models to determine whether changes in objective and subjective cognition over time predicted depression and anxiety severity above the baseline model.

Because depression and anxiety severity, as well as subjective cognitive symptoms, are assessed by self-report, it is possible that these measures may be (at least in part) reflecting the same underlying construct. To determine the direction of influence between these measures, exploratory longitudinal mixed-effects models were conducted in reverse (depression and anxiety predicting subjective cognitive symptoms) and examined relative to models conducted above.

For all mixed-effects models, a random intercepts, random slopes structure (with participant ID grouped by time point) was used. Using the *performance* R package (Lüdecke et al., 2021), adjusted intraclass correlation coefficients (ICCs) were estimated to determine random effects variance (Nakagawa et al., 2017), and marginal R^2 for fixed effects variance. Interaction terms in the models (i.e. between Timepoint and objective cognition) were evaluated both in terms of statistical significance to the model ($p < 0.05$) and whether inclusion of the interaction term improved model fit to the study data. Model fit statistics including the Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC), the Root Mean Squared Error (RMSE) and the Sigma (Σ) are reported, with lower values in each statistic indicating better fit. Mixed-effects models are robust to missing datapoints and provide unbiased model parameter estimates with all available information using maximum likelihood estimation. Therefore, all individuals who contributed data were included in analyses, except those lost to attrition due to death as this was not considered to meet the Missing at Random assumption of mixed-effects models.

In addition to mixed effects models, cross-sectional correlations between objective and subjective cognition to depression and anxiety severity, age, time post-stroke, stroke severity, years of education, and participant sex were conducted to aid interpretation and for full transparency of the data.

Results

$N = 105$ stroke participants were recruited to OX-CHRONIC (Kusec et al., 2023, for a detailed description), with 6 lost to attrition due to death[1] (present study $N = 99$; Table 1). Participants consenting to OX-CHRONIC did not differ from those not consenting in terms of sex, acute NIHSS scores, type of stroke, lesion hemisphere, emotional distress, or subjective cognition at 6-months post-stroke. OX-CHRONIC participants were younger, had lower levels of objective cognitive impairment, and a greater number of years of education (see Supplementary Materials). Across all assessment points, 81.8% of participants had complete data points ($N = 243$ of 297 data points). Those with incomplete

Table 1. Participant characteristics. Scores higher than 7 on the HADS-D and HADS-A were considered clinically elevated (i.e., warranting clinical attention), as per published cutoffs.

Participants ($N = 99$)		Min–Max	% Clinically elevated
Sex – *n* (%)			
Male	56 (56.6%)		
Female	43 (43.4%)		
Age at stroke – Mean (*SD*)	68.9 (13.1)	18–92	
Years of education – Mean (*SD*)	13.9 (3.7)	9–23	
Stroke type – *n* (%)			
Ischaemic	82 (82.8%)		
Haemorrhagic	17 (17.2%)		
Lesion hemisphere – *n* (%)			
Left	40 (40.4%)		
Right	39 (39.4%)		
Bilateral	7 (7.1%)		
Undetermined from scan	13 (13.1%)		
Vasculature – *n (%)*			
Middle cerebral artery	47 (47.5%)		
Multiple	9 (9.1%)		
Posterior cerebral artery	6 (6.1%)		
Superior cerebellar artery	6 (6.1%)		
Posterior inferior cerebellar artery	4 (4.0%)		
Basilar artery	3 (3.0%)		
Anterior cerebral artery	2 (2.0%)		
Undetermined/not reported	22 (22.2%)		
First or recurrent stroke – *n* (%)			
First	67 (67.7%)		
Recurrent	32 (32.3%)		
Acute NIHSS score – Mean (*SD*)	7.49 (6.35)	0–27	
HADS-depression – Mean (*SD*)			
Time 1 (6 months post-stroke)	4.71 (3.54)	0–16	18.2
Time 2 (~4.5 years post-stroke)	4.70 (3.59)	0–17	21.5
Time 3 (~5.5 years post-stroke)	4.73 (3.60)	0–18	17.8
HADS-anxiety – Mean (*SD*)			
Time 1 (6 months post-stroke)	5.32 (3.55)	0–16	25.0
Time 2 (~4.5 years post-stroke)	5.06 (3.62)	0–15	21.5
Time 3 (~5.5 years post-stroke)	5.21 (3.93)	0–20	25.6

HADS: Hospital Anxiety and Depression Scale; NIHSS: National Institute of Health Stroke Severity.

data did not differ in terms of demographic variables or study measures (see Supplementary Materials). We considered the data to be missing at random.

A total of 73 participants (73.7%) completed OX-CHRONIC assessments (Time 2 and Time 3) via telephone (26.3% completed via videoconferencing). Through testing, 3 participants were noted to have a carer or family member present to support assessments. These 3 participants were retained in analyses for study power and sample representativeness. Participants predominantly had ischaemic strokes (82.8%) and moderate stroke severity (median NIHSS = 5, Interquartile Range = 6.75).

Cross-sectional correlations between HADS subscales to OCS and CFQ scores are in Figure 1. At all time points, objective cognition and subjective cognitive symptoms did not correlate with each other, nor with age, time-post stroke or years of education ($ps = 0.13 – 0.94$) nor were there any observed sex differences (see Supplementary Materials for details).

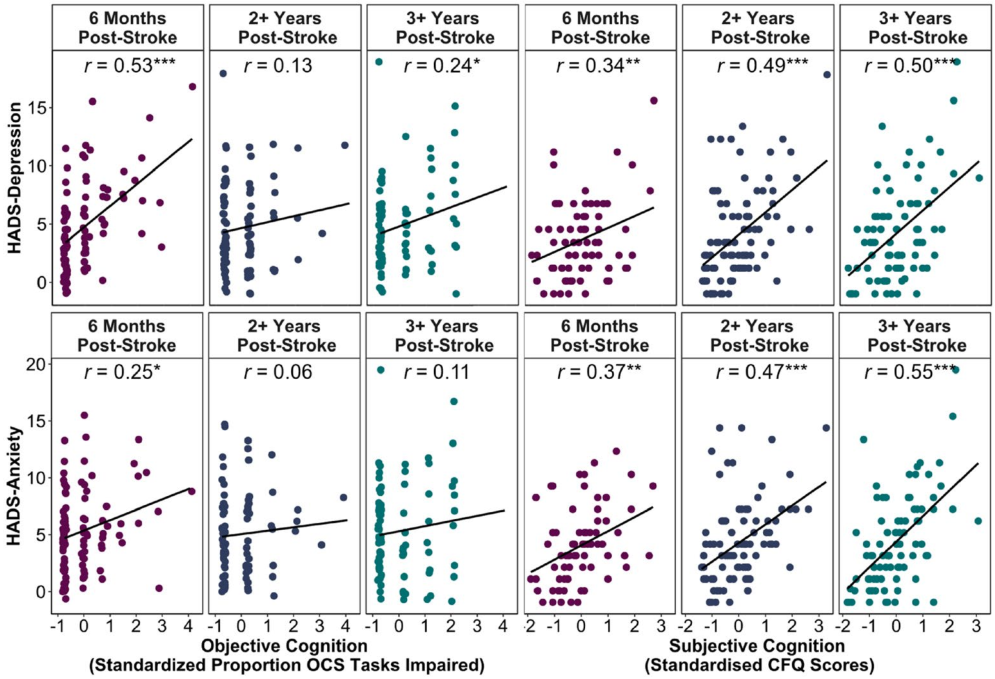

Figure 1. Cross-sectional pairwise complete Pearson correlations between objective and subjective cognition to depression severity (top row) and to anxiety severity (bottom row) across all study time points (6-months $N = 96$, 2+ Years $N = 93$, 3+ Years $N = 85$). OCS: Oxford Cognitive Screen; CFQ: Cognitive Failures Questionnaire; HADS: Hospital Anxiety and Depression Scale. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Baseline model – depression and anxiety trajectories

There was no mean change in post-stroke depression from Time 1 to Time 2 ($b = -0.04$, SE = 0.33, p=.90; VC = 7.91, $SD = 2.81$) nor to Time 3 ($b = 0.24$, SE = 0.36, p=.51; VC = 9.34, $SD = 3.06$; ICC_{adj}=0.89 [AIC = 1396.93, BIC = 1433.17, Marginal $R^2 = 0.001$, RMSE = 0.59, $\Sigma = 1.21$]). Similarly, there was no change in anxiety scores to Time 2 ($b = -0.35$, SE = 0.38, p=.36; VC = 9.89, $SD = 3.15$) or Time 3 ($b = -0.02$, SE = 0.39, p=.0.96; VC = 10.39, $SD = 3.22$; ICC_{adj}=0.87 [AIC = 1442.54, BIC = 1478.78, marginal $R^2 = 0.002$, RMSE = 0.64, $\Sigma = 1.34$]). Depression and anxiety were thus stable on average from 6 months to chronic stroke. However, there appeared to be large variation in both improvement and worsening subgroups between timepoints (Figure 2).

When adding demographic variables to baseline models, HADS-Depression and HADS-Anxiety scores were not predicted by acute NIHSS scores (HADS-D $b = 0.06$, SE = 0.06, p=.32; HADS-A $b = -0.06$, SE = 0.05, p = .29), participant concurrent age (HADS-D $b = 0.01$, SE = 0.03, $p = 0.63$; HADS-A $b = -0.03$, SE = 0.03, p=.31), years of education (HADS-D $b = -0.10$, SE = 0.09, p=.34; HADS-A $b = -0.18$, SE = 0.10, p=.07), participant sex (HADS-D $b = -0.02$, SE = 0.76, p=.98; HADS-A $b = -0.14$, SE = 0.75, p=.85), stroke type (HADS-D $b = -0.31$, SE = 0.94, p=.75; HADS-A $b = 0.24$, SE = 0.94, p=.79) or lesion hemisphere (HADS-D $bs = -0.19 - 0.92$, SEs = $0.82 - 2.32$, $ps = .26 - .94$; HADS-A $bs = -0.15$—–0.75, SEs = $0.81 - 2.31$, ps=.56 – .95). Inclusion of demographic variables

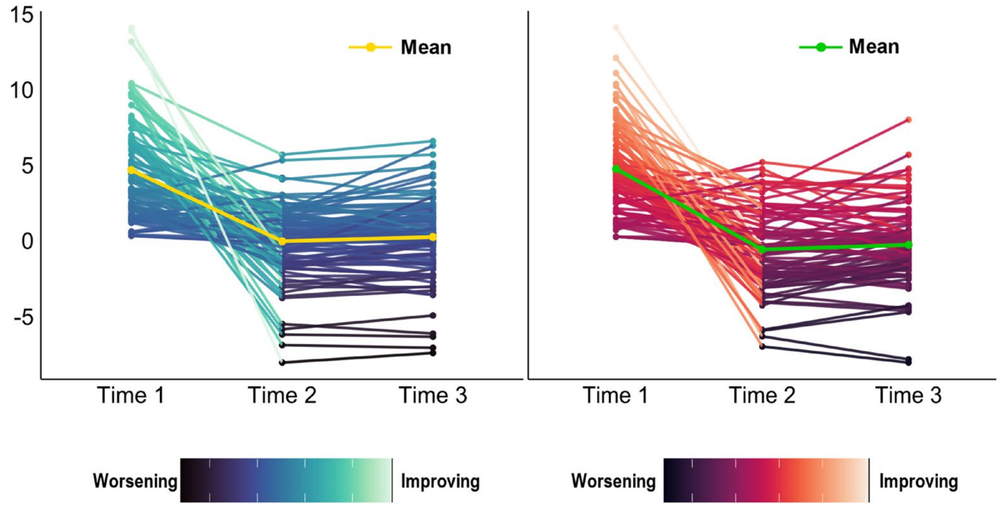

Figure 2. Participant-level trajectories of depression (left hand panel) and anxiety severity (right hand panel) from 6-months (Time 1) to approximately 4.5 (Time 2) and 5.5 years (Time 3) post-stroke ($N = 98$). The y-axis shows individual-level change in HADS-D and HADS-A after accounting for expected change over time and between-participant variability (random slopes). Darker colours represent worse depression or anxiety and lighter colours represent better scores. Mean trajectory values are shown in yellow for depression severity, and in green for anxiety severity.

explained little variance and did not improve fit for HADS-Depression (marginal R^2=0.04; AIC = 1163.71, BIC = 1225.20; RMSE = 0.62, $\Sigma = 1.26$; ICC_{adj}=0.88) nor HADS-Anxiety (marginal R^2=0.05; AIC = 1204.78, BIC = 1266.27; RMSE = 0.66, $\Sigma = 1.40$; ICC_{adj}=0.87).

Longitudinal association of objective and subjective cognition to depression

When including objective and subjective cognition to the baseline depression model, both objective ($B = 1.01$, SE = 0.29, $p < .001$) and subjective cognition ($B = 0.83$, SE = 0.27, $p < .01$) had main effects on depression severity, indicating a stable relationship between these variables regardless of timepoint.

In terms of interaction effects, objective cognitive impairments significantly related to HADS-Depression scores at 6-months but this weakened at 4.5 years ($B = -0.79$, SE = 0.37, $p < .05$). This indicates that improvements in objective cognition from 6-months to long-term stroke had a lesser effect on depression compared to objective cognitive ability at 6-months post-stroke. The impact of subjective cognition to HADS-Depression did not change between Time 1 to Time 2, though a trend was noticed ($B = 0.60$, SE = 0.34, $p = .08$). Subjective cognitive symptoms at Time 3 appeared to have a stronger effect on HADS-D scores ($B = 0.77$, SE = 0.35, $p < .05$). Inclusion of the interaction term between subjective cognitive symptoms over time provided the best fit to the study data (see Figure 2; AIC = 1160.15, BIC = 1215.90, RMSE = 0.43, $\Sigma = 0.99$, marginal R^2=0.22) compared to modelling only an interaction between objective cognition over time (AIC = 1334.99, BIC = 1381.73, RMSE = 0.52, $\Sigma = 1.12$, marginal R^2=0.08). Variance due to random slopes was moderate at Time 2 (VC = 5.04, *SD* = 2.25) and Time 3 (VC = 6.29, *SD* = 2.51), with an ICC_{adj} of 0.89.

Exploratory analyses were conducted investigating whether changes in any specific cognitive domains predicted depression (see Supplementary Materials for full results). Only attention abilities, as measured via the OCS Broken Hearts cancellation subtask, related to depression outcomes at Time 1 relative to Time 2 ($b = -1.81$, SE = 0.81, $p < .05$) and Time 3 ($b = -1.90$, SE = 0.80, $p < .05$).

Longitudinal association of objective and subjective cognition to anxiety

Only subjective cognition had a main effect on HADS-Anxiety scores ($B = 1.14$, SE = 0.34, $p < .01$) whereas objective cognition did not ($B = 0.32$, SE = 0.37, $p = .40$).

This lack of relationship between objective cognition and HADS-Anxiety remained constant from Time 1 to Time 2 ($B = -0.49$, SE = 0.48, $p = .30$) and from Time 2 to Time 3 ($B = -0.15$, SE = 0.45, $p = .73$). Though the relationship between subjective cognition and HADS-Anxiety from Time 1 to Time 2 was stable ($B = 0.17$, SE = 0.44, $p = .69$), this relationship strengthened at Time 3 ($B = 0.96$, SE = 0.44, $p < .05$). Variance due to random slopes was moderate at Time 2 (VC = 10.62, *SD* = 3.26) and Time 3 (VC = 9.76, *SD* = 3.12), with an ICC_{adj} of 0.86. As in the depression model, inclusion of the interaction term between subjective cognitive symptoms over time provided the best fit to the data (AIC = 1226.20, BIC = 1281.96, RMSE = 0.52, $\Sigma = 1.19$, marginal $R^2 = 0.20$; see Figure 2) compared to modelling only an interaction between objective cognition over time (AIC = 1394.48, BIC = 1441.21, RMSE = 0.59, $\Sigma = 1.23$, marginal $R^2 = 0.02$).

Exploratory domain-specific cognitive impairment models were not conducted due to objective cognitive impairment not demonstrating a main effect in anxiety severity.

Exploring direction of influence between mood and subjective cognition

It is possible that a relationship between subjective cognition to depression and anxiety exists because self-report measures capture similar response tendencies. Cognitive theories of depression and anxiety (Beck, 1974, Koerner & Dugas, 2006) state that overly negative evaluations of abilities increases negative affect, instead of the reciprocal. When conducting models in reverse, there was no interaction between changes in HADS-Depression from Time 1 to Time 2 ($B = 0.05$, SE = 0.12, $p = .70$) or at Time 3 ($B = -0.01$, SE = 0.12, $p = .94$) to changes in subjective cognition. Similarly, there were no interaction effects in changes in HADS-Anxiety over time from Time 1 to Time 2 ($B = -0.03$, SE = 0.03, $p = .33$) nor at Time 3 ($B = -0.05$, SE = 0.03, $p = .13$). See the Supplementary Materials for a full account.

Summary of results

Taken together, objective cognitive impairments, in particular attention impairments, at 6 months post-stroke were related to depressive symptoms only. However, this relationship weakened in long-term stroke (~4.5 years). Subjective cognitive symptoms had a stronger relationship to both depression and anxiety in long-term (≥2 years) stroke compared to 6-months post-stroke (Table 2). When exploring potential direction of influence between subjective cognitive symptoms to mood outcomes, changes in depression and anxiety severity did not have a reciprocal effect on subjective cognition (Figure 3).

Table 2. Mixed-effects model results across $N = 99$ participants

	HADS-depression		HADS-anxiety	
Variable	*B* (SE)	*t* (*p*-Value)	*B* (SE)	t (*p*-Value)
Intercept	**4.26 (0.30)**	**14.14 (<.001)**	**4.99 (0.35)**	**14.15 (<0.001)**
Time 2	0.34 (0.31)	1.11 (0.27)	−0.03 (0.42)	−0.07 (0.94)
Time 3	0.57 (0.34)	1.69 (0.09)	0.32 (0.42)	0.76 (0.45)
Proportion OCS tasks impaired	**1.01 (0.29)**	**3.48 (<0.001)**	0.32 (0.37)	0.85 (0.40)
CFQ	**0.83 (0.27)**	**3.08 (<0.01)**	**1.14 (0.34)**	**3.34 (<0.01)**
Time 2 * proportion OCS tasks impaired	**−0.79 (0.37)**	**−2.13 (<0.05)**	−0.49 (0.48)	−1.03 (0.30)
Time 3 * proportion OCS tasks impaired	−0.43 (0.36)	−1.19 (0.24)	−0.16 (0.45)	−0.35 (0.73)
Time 2 * CFQ	0.60 (0.34)	1.79 (0.08)	0.17 (0.44)	0.40 (0.69)
Time 3 * CFQ	**0.77 (0.35)**	**2.19 (<0.05)**	**0.96 (0.43)**	**2.17 (<0.05)**

Bolded text indicates statistically significant terms.
OCS: Oxford Cognitive Screen; CFQ: Cognitive Failures Questionnaire.

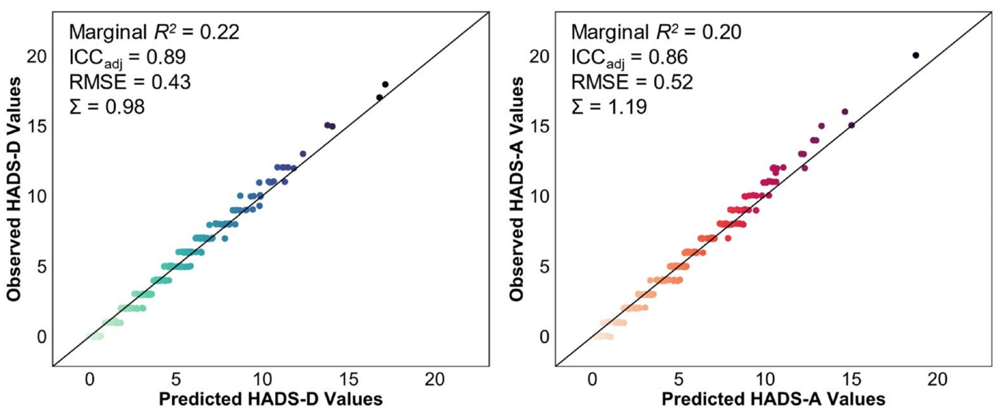

Figure 3. Predicted versus observed values of HADS-D scores (left) and HADS-A scores (right). Predicted values are based on model fit indices ($N = 96$). HADS-D: Hospital Anxiety and Depression Scale – Depression; HADS-A: Hospital Anxiety and Depression Scale – Anxiety; ICC: intraclass correlation coefficient; RMSE: root mean square error.

Discussion

This study examined whether the relationship between objective cognitive impairment and subjective cognitive symptoms to post-stroke depression and anxiety differed from 6 months to long-term stroke (average ~5.5 years, range 2—10 years). We found that typical predictors of post-stroke function such as age, years of education, sex, and stroke severity did not relate to post-stroke depression and anxiety (collective marginal R^2=0.03 – 0.05). Instead, depression and anxiety severity was better explained by objective and subjective cognition (marginal R^2=0.20 – 0.22). While both objective and subjective cognition related to post-stroke depression, only subjective cognition related to post-stroke anxiety. Specifically, objective cognitive impairments at 6 months post-stroke related to depression, but this effect weakened in long-term stroke. Subjective cognitive symptoms mostly strongly influenced post-stroke depression and anxiety in long-term stroke.

Relationship between objective and subjective cognition over time

Though both objective cognitive impairment and subjective cognitive symptoms related to depression and anxiety severity, they did not relate to each other at any time point. Many studies report no correlation between objective cognitive impairments and subjective cognitive symptoms both in acute stroke (Duits et al., 2008; Kliem et al., 2022) and onwards post-stroke (Lamb et al., 2013). This may be explained by the content of subjective cognition self-report measures. In systematic reviews, the highest correlation between subjective cognitive symptoms and objective performance is on more salient cognitive impairments such as memory (Davis et al., 1995; Duits et al., 2008), whereas executive function difficulties for example may be more difficult to self-report. Alternatively, anosognosia or "impaired self-awareness" may also weaken a correlation between objective and subjective cognition. Stroke survivors with difficulties monitoring cognitive abilities may self-report good cognitive functioning, despite objectively impaired performance (Nurmi & Jehkonen, 2014). We note, however, whilst anosognosia is observed in early stroke, there is little evidence to suggest this persists in long-term stroke (Jehkonen et al., 2006) aside from specific case reports (e.g., Venneri & Shanks, 2004). However, outside of stroke, meta-analytic evidence suggests only a modest correlation between self-reported abilities and objective performance (r = 0.29, Zell & Krizan, 2014), indicating that discrepancies may be due to pre-stroke difficulties in self-assessing abilities.

The weak relationship between objective and subjective cognition may indicate that each has distinct influences on post-stroke depression and anxiety. Our findings on objective cognitive impairment align with previous research showing that improvements in cognitive ability enhances function and thus mood (Mole & Demeyere, 2020; Wheeler et al., 2023). It is less clear how subjective cognition changes affect mood outcomes. Lowered access to psychological care in the long-term post-stroke (Lin et al., 2021) may result in increased distress over unaddressed subjective cognitive symptoms. Alternatively, self-reports of cognitive difficulties may represent worry about possible cognitive decline post-stroke (Tang et al., 2019). Qualitative literature has highlighted that uncertainty about future cognitive status can increase post-stroke anxiety (Salter et al., 2008). These increases in uncertainty in cognitive abilities may explain the stronger relationship between subjective cognitive symptoms and mood outcomes in longer-term stroke.

Objective cognitive performance and post-stroke depression and anxiety

We found that the relationship between objective cognitive impairment to post-stroke depression severity is strongest at 6 months post-stroke and lessens into the long term. The relationship between objective cognitive impairments and post-stroke depression is well-documented in systematic reviews (Ayerbe et al., 2013; Chun et al., 2022; Hackett & Anderson, 2005). Objective cognitive impairments are more prevalent at 6 months compared to long-term stroke (Kusec et al., 2023), therefore recovery made may have a more noticeable influence and therefore improve depression, relative to potentially minor shifts in objective cognitive ability in long-term stroke. However, there are alternative explanations. First, although cognitive impairment is still common in long-term stroke (~45% prevalence; Kusec et al., 2023), it may no longer be severe enough to substantially impact self-rated depression. Second, individuals may become more adept at managing the impact of cognitive impairment on daily life (Ch'Ng et al., 2008), weakening its link to depression. Finally, improvements in specific cognitive impairments may better explain improvements in mood status than overall severity of cognitive impairment, as evidenced in cross-sectional research (Williams & Demeyere, 2021). In our exploratory analyses, we found that only attention impairments relate to depression outcomes from 6 months to long-term stroke. Though replication is warranted, this result aligns with research outside of stroke, where concentration difficulties and increased distractibility are core features of depression (Keller et al., 2019).

We found no role of objective cognitive impairment to post-stroke anxiety. This is in line with some previous research demonstrating no unique correlation between these variables (Scopelliti et al., 2022; Williams & Demeyere, 2021), though other studies report strong predictive effects of objective cognitive performance to post-stroke anxiety in both early (Barker-Collo, 2007) and long-term stroke (Lee et al., 2019). Additionally, we found that demographic factors do not reliably predict post-stroke anxiety, contrary to previous research demonstrating younger age as a risk factor (Menlove et al., 2015). Possibly, demographic factors contribute to only a small amount of variance in anxiety (as found here) that are only statistically detectable in meta-analyses with far larger samples (e.g., Chun et al., 2022).

Subjective cognitive symptoms and post-stroke depression and anxiety

Subjective cognitive symptoms have long been associated with post-stroke depressive symptoms (van Rijsbergen et al., 2014). Possibly, in the very long-term post-stroke, individuals become more adept at *recognizing* cognitive changes, and begin ruminating on the implications of this on everyday life. Alternatively, long-term stroke may result in greater social isolation due to decreases in occupational and leisure activities (Mole & Demeyere, 2020; Stolwyk et al., 2021). Greater social isolation can result in more time ruminating on stroke-related changes, including increased focus on cognitive changes (Turner et al., 2019; White et al., 2008).

We found that *only* changes in subjective cognitive symptoms related to post-stroke anxiety, particularly in long-term stroke. This aligns with previous work demonstrating a link between subjective cognition and anxiety (van Rijsbergen et al., 2019; Maaijwee et al., 2014; Nijsse et al., 2017). Similar to the depression models, changes in anxiety post-stroke did not reciprocally relate to changes in subjective cognitive symptoms. In qualitative research, stroke survivors report difficulties accepting not being able to predict if and how their cognitive abilities may change in the future, leading to worry

(Carlsson et al., 2009; Crowe et al., 2016). Further quantitative research has shown that difficulties coping with uncertainty are uniquely associated with post-stroke anxiety (Kusec et al., 2024) as has been shown outside of stroke (Koerner & Dugas, 2006).

Elevated reports of subjective cognitive symptoms could also represent an "over-awareness" of post-stroke abilities—that is, endorsing a relatively mild cognitive change as distressing despite objective performance corresponding to expected, or pre-stroke, performance (e.g., Smeets et al., 2017; Wheeler et al., 2023). Another explanation is that *pre-stroke* negative evaluation tendencies extend to post-stroke abilities more generally (Beck, 1974; Koerner & Dugas, 2006). Whether negative evaluations are unique to cognitive difficulties post-stroke, compared to physical abilities for example, would help determine whether a more general versus cognition-specific negative evaluation tendency is important to depression and anxiety outcomes post-stroke.

Subjective cognitive symptoms are thought in part to be manifestations of depressed mood (Nijsse et al., 2017) rather than independent from depression. Notably, we found that whilst depression and anxiety severity are correlated with subjective cognition over time, changes in depression and anxiety severity did not reciprocally relate to changes in subjective cognition. These findings are in line with cognitive models of depression in non-stroke populations, in which negative evaluations of abilities worsen depression outcomes (Beck, 1974) rather than the reverse. It is also possible that the unidirectional effect observed here is specific to the measure used. The CFQ assesses frequency of cognitive lapses in everyday life rather than concern over these lapses. Other measures of subjective cognition (e.g., Checklist for Cognitive and Emotional Symptoms, van Heugten et al., 2007) assess the impact of such lapses. Therefore, it may be more precise to state that we find evidence that frequency of cognitive symptoms is unidirectionally associated with emotional distress. Whether concern over cognitive symptoms has differential relationships with post-stroke depression and anxiety should be examined in future research. Additionally, subjective cognitive symptoms could have a more context-dependent or dynamic relationship to depression and anxiety, while the effect of depression/anxiety on subjective cognition is comparably constant over time.

Clinical implications

A top unmet need of long-term stroke survivors is mental health support (Hill et al., 2022; Lin et al., 2021). Using longitudinal analyses, we have demonstrated addressing subjective or objective cognition in mood interventions may depend on time-point post-stroke. At 6 months post-stroke, including cognitive rehabilitation strategies within mood interventions, specifically attentional management strategies, may be beneficial for depression. However, in long-term (>2 years) stroke, managing subjective cognitive symptoms may be more valuable to incorporate into both depression and anxiety interventions. Our results hold particular promise for developing anxiety interventions, as subjective cognitive symptoms are potentially modifiable, unlike other reported risk factors such as age and sex (Chun et al., 2022).

In developing mood interventions for long-term stroke, subjective cognitive symptoms likely relate to depression and anxiety severity differently. Depression is characterized by ruminative tendencies over past events, while anxiety is characterized by pathological worry about events that may occur in the future. Individuals with post-stroke depression may ruminate over the loss of cognitive function compared to pre-stroke abilities, while those with post-stroke anxiety worry about whether cognitive abilities will change or deteriorate in the future. These distinctions may result in similar scores on subjective cognitive symptoms measures, but could have different treatment implications (i.e., in targeting the content of concern driving such reporting).

Future directions

Given that previous reviews have demonstrated that more salient cognitive impairments have greater concordance with subjective cognition (van Rijsbergen et al., 2014), future research could explore whether different within-domain objective cognitive abilities relate differentially to global subjective cognition (as in the CFQ) in early versus long-term stroke. Further, we find that attentional impairments uniquely relate to post-stroke depressive symptoms. Future research could explore whether attentional impairments aggravate depression-related concentration and distractibility symptoms and worsen depression. With regards to anxiety, though we find no evidence of a relationship between objective cognition and anxiety here, given inconsistent correlations in existing research larger scale research is needed prior to accepting or rejecting this relationship. However, as subjective cognitive symptoms did correlate with anxiety over time here, identifying mediators of the relationship can be a fruitful avenue for future research.

As we observed large variation when visualizing depression and anxiety trajectories post-stroke, future research could examine whether potential subgroups exist. It is also possible that co-occurring diagnoses post-stroke affect emotional distress levels. For example, diagnoses that affect the ability to identify and consider the impact of cognitive impairment to everyday life (e.g., anosognosia, Wernicke's aphasia), or have independent impact on levels of emotional distress (e.g., paresis, pain); these should be explored in future studies. Additionally, potential causal mechanisms of the differential temporal relationship between objective and subjective cognition should be explored. Though there is evidence in the non-stroke population that negative interpretations of abilities worsen depression and anxiety (Beck, 1974, Koerner & Dugas, 2006), it is not yet clear from the present data whether the relationship between high levels of subjective cognition and mood are driven by such overly negative interpretations, or whether they represent concern over future cognitive abilities, or have better ecological validity in capturing post-stroke cognitive impairment. These potential mediators of the relationship between subjective cognition to mood should be explored. It is also possible that there are interactions between recovery of objective cognition and increases in subjective cognition over time; given that attentional abilities seem to confer a unique risk to depression here, it is possible that improvements in this domain relate to not just changes in depression, but also in subjective cognition over time. Finally, both objective and subjective cognition have reported impacts on post-stroke function; the degree to which both impact functional impairment should be investigated as a moderator of emotional distress in future research.

Study generalizability and limitations

Our study comprises a sample of moderate severity stroke survivors with a large proportion of left-hemisphere stroke, enhancing generalizability. Using a stroke-specific, aphasia-friendly screen to assess cognition minimized confounds associated with language. Encouragingly, demographic variables, levels of emotional distress and subjective cognition did not differ from participants not consenting to OX-CHRONIC, suggesting good generalizability in interpreting these variables. OX-CHRONIC participants were however on average younger, better educated, and had lower levels of objective cognitive impairment compared to those not consenting. It is possible that differing relationships between objective cognition and emotional distress would be observed in a more cognitively impaired sample.

Though time post-stroke did not correlate with changes in depression and anxiety, data time points 2 and 3 comprised a wide range of participants from 2 to 9 years post-stroke. We additionally had no data on sample ethnicity or on socioeconomic status, which potentially limits interpretation and generalizability of the findings. Although pre-stroke emotional status is known to relate to post-stroke mood outcomes, such data was not available here. The OCS and the HADS are screening tools rather than diagnostic instruments, thus stroke survivors with more subtle cognitive impairments for example may have been missed in the present study assessments. Longitudinal results should be replicated with repeated assessments of neuropsychological batteries and interview-based mood disorder diagnoses. Further, the unidirectional influence of subjective cognition to emotional distress found here should be replicated in a separate cohort.

Conclusions

Objective cognitive impairment and subjective cognitive symptoms better predict changes in depression symptoms from 6 months to the long-term post-stroke than demographic variables. Subjective cognitive symptoms relate uniquely to post-stroke anxiety. This research may have clinical implications regarding (i) the inclusion of cognitive strategies for depression interventions at 6 months post-stroke and (ii) addressing subjective cognitive symptoms in both depression and anxiety interventions in long-term stroke.

Note

1. There were no differences in demographics and study measures between those lost to attrition due to death by Time 3 ($N = 6$) and those retained ($N = 99$) (see *Supplementary Table 1*).

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CRediT authorship statement

A. Kusec: Conceptualization, Formal analysis, Methodology, Visualization, Data Curation, Project administration, Writing—original draft, writing—review & editing; **N. Demeyere:** Conceptualization, Writing—review & Editing, Supervision, Project administration, Funding acquisition

Disclosure statement

No potential conflict of interest was reported by the author(s). ND is a developer of the Oxford Cognitive Screen but does not receive any remuneration from its use.

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