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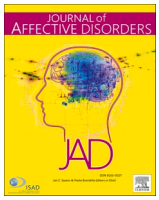

Review article

Meta-analytic review of longitudinal depression severity in stroke survivors

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ABSTRACT

Background: Depression is a prevalent and debilitating condition among stroke survivors, yet it is potentially treatable and preventable. Previous meta-analyses have primarily focused on prevalence rates, and thus lacked the nuanced insights needed to optimize timing for interventions and prevention strategies. In this meta-analysis, we examined the progression of depression severity from the acute and sub-acute stages up to 36 months post-stroke.

Methods: We included only studies involving stroke survivors not undergoing treatment interventions. A multilevel meta-analysis was conducted across 41 eligible studies to assess natural changes in depression severity over time using standardized mean change. We analysed data across all timepoints and conducted sensitivity analyses on potential sources of bias, including publication bias, methodological quality, and low-powered timepoints.

Results: In the full model including all eligible studies, depression severity did not significantly change over time, and time since stroke was not a significant moderator. However, sensitivity analyses excluding studies identified as outliers revealed a small but significant overall reduction in depression severity (*95 % CI* [−0.123, –0.030]; $p = 0.002$), and a small increase in depression severity at 12-months post-stroke (*95 % CI* [0.006, 0.374]; $p = 0.043$).

Conclusions: Although the overall model did not show significant longitudinal change, sensitivity analyses revealed a potential pattern of overall improvement in depression, with a small resurgence at 12-months post-stroke. The findings underscore the importance of evaluating depression in stroke survivors on a continuous scale, which demonstrates a pattern of post-stroke depression not previously observed using prevalence rates. Future studies should more closely examine the influence of continuous longitudinal post-stroke depression severity on stroke recovery.

1. Introduction

Depressive symptoms following stroke affect approximately one-third of stroke survivors and are highly debilitating (Medeiros et al., 2020). Post-stroke depression (PSD) not only heightens the risk of disabilities and mortality but also reduces motivation to engage in rehabilitation, which is essential for resuming independent living and returning to work (Robinson and Jorge, 2016). It is well established within the depression literature that early prevention and intervention, when depressive symptoms are relatively mild, lead to better long-term outcomes and are often more effective and cost-efficient relative to treating florid depression (Ormel et al., 2020; McLaughlin, 2012). Studies in stroke survivors indicate that early screening and timely intervention before depression becomes clinically severe can enhance participation in therapy (Devereux and Berns, 2023). This approach mitigates the adverse effects of PSD on physical and cognitive rehabilitation, ultimately leading to better functional outcomes and reduced mortality risk.

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In the past, PSD has primarily been studied through prevalence rates. In a meta-analysis by Liu et al. (2023), patients were classified as either "depressed" or "not depressed" based on cut-off scores from self-report measures. The study reported that those classified as clinically depressed within the first three months post-stroke were substantially more likely to experience clinical depression within one year of stroke. This method provides useful information about the prevalence of depression over time, but cannot quantify the influence of depression severity on patient outcome. For example, if individuals with severe depression at three months remained depressed at one year, while those with moderate depression recovered, this difference would not be detected by prevalence rates alone. Developing effective early interventions and prevention strategies for PSD requires a more nuanced understanding of depression etiology. Identifying when patients first exhibit signs of depression would enable timely interventions to halt the progression to severe, florid depression and mitigate secondary consequences. Furthermore, continuous measures of PSD offer pragmatic utility in a clinical setting since they can easily be acquired in time-sensitive care, such as in the emergency room or with follow-up care, whereas binary classifications based on a formal diagnosis can often require time-consuming clinical interviews. Binary classification also limits the ability to detect fluctuations toward remission, relapse, or chronicity. Continuous measures preserve variability and allow for more nuanced analyses of within-person change, offering better predictive utility for long-term functional and psychological outcomes. By binning patients into "depressed" or "not depressed", more comprehensive patterns of PSD etiology are lost in analysis. A meta-analytic approach focused on continuous symptom severity allows for generalization of trajectory trends across studies, laying the groundwork for identifying high-risk periods and subgroups in future longitudinal and person-centered research. This necessitates a shift from studying prevalence to focusing on PSD severity over time.

Previous studies have reported that PSD is most severe between three to six months following stroke (Dong et al., 2021; Alajbegovic et al., 2014; Whyte and Mulsant, 2002), suggesting that the acute phase may be optimal for early interventions and prevention, but the reliability of this timing is not well-established. Assessments of depression are often conducted three to six months post-stroke for practical reasons – this period usually follows hospital and rehabilitation discharge, preceding a return to work. This makes it a convenient time for research involvement rather than a clinically confirmed peak for PSD (Winstein et al., 2016). Measuring depression in the sub-acute phase with cross-sectional study designs prevents full longitudinal characterization of depressive symptom severity and does not identify when PSD symptoms are mild enough for optimal intervention.

No meta-analysis to date has examined continuous PSD severity over time at a generalizable level, from the acute and sub-acute phases to long-term progression. This approach allows identification of fluctuations in symptom severity that binary classifications cannot capture, thereby offering new insights into timing and trajectory of depressive symptoms following stroke. Here, we examined changes in depression severity over time along continuous self-report measures relative to baseline depression measured within the acute and sub-acute phases, i.e. within the first 10-weeks of stroke. Based on previous studies, which have reported that PSD peaks between three to six months post stroke (Dong et al., 2021; Alajbegovic et al., 2014; Whyte and Mulsant, 2002), and that clinical levels of PSD at three months are likely to persist at clinical levels one year later (Liu et al., 2023), we hypothesised that PSD severity in stroke survivors would increase significantly from baseline to three and six months post-stroke, and remain severe at 12 months post-stroke.

2. Methods

2.1. Search strategy and selection criteria

Our search included studies available on PubMed, PsychInfo, and Web of Science from database inception to the 15th of March 2025. All databases were searched with the following search terms: (depress*) AND (stroke) AND ((trajectory*) OR (longitudinal)). The search was conducted by two researchers independently (MDT and DW). Any disagreement between the two researchers was arbitrated by a third reviewer (LO). Inclusion criteria required that studies involved a sample of stroke survivors aged 18 years or older, with depression severity measured using a self-report scale at two or more time points. Other inclusion criteria involved baseline depression recorded within 10 weeks of stroke (acute/subacute stage) to compare future timepoints with acute/subacute stages of stroke, and medical documentation confirming stroke diagnosis. Exclusion criteria were depression recorded as a binary variable only (depressed/not depressed), samples with aphasia or other communication impairments that may erroneously influence self-reported depression scores, depression assessed by a third person (e.g. nurse, partner, etc.), and duplicate study samples. We further excluded randomized controlled trials with placebo versus treatment effects (e.g. antidepressants, psychotherapy, etc.), because we were interested in the natural progression of depression severity in the absence of targeted interventions. After removing studies that did not meet inclusion criteria, further studies were removed due to insufficient reporting of depression severity. In case of insufficient data reporting (missing means or standard deviations), missing data were requested from study authors.

2.2. Statistical analysis

To map the progression of PSD severity, a multi-level meta-analysis was conducted comparing baseline measures (depression severity measured within 10 weeks of stroke) with all timepoints. Timepoints were compared using standardized mean change (SMC) to allow comparisons of multiple continuous measures of depression, despite differences in scoring ranges. The SMC was calculated by subtracting the basline PSD score from the post-acute PSD score and dividing the result by the standard deviation of the baseline PSD score within every study (Becker, 1988). Hedge's g was used as the effect size measure to account for small sample sizes (Marfo and Okyere, 2019). Given that within-subjects measures can be influenced by auto-correlation, correlation coefficients were included in standard error corrections. This correction requires the pre-post correlation coefficient of individual samples in the meta-analysis. As this value was not reported in the majority of included manuscripts, meta-analyses were repeated multiple times across five correlation coefficients, 0 (uncorrelated), 0.25, 0.50, 0.75, and 0.90 (Becker, 1988).

We used a random-effects model to test pooled estimates across studies. Chi-squared and I^2 heterogeneity tests were performed for each meta-analysis. A funnel plot and Egger's test of asymmetry were conducted to determine potential publication bias. The Risk of Bias in Non-Randomized Studies of Exposures (ROBINS-E; Higgins et al., 2024) was performed on all included studies. We further conducted three sensitivity analyses of our multi-level meta-analysis: one in which all high risk studies from the ROBINS-E were removed, one in which all timepoints with a sample size of less than three were removed, and a third in which all studies with publication bias in our funnel plot were removed. Follow-up time moderator analysis was conducted on all models. All analyses were conducted in R 4.4.0 (R Core Team, 2021).

3. Results

Of 2,244 studies, 41 studies including 6,979 stroke patients met inclusion criteria (see Fig. 1 and Table 1). Studies reported depression

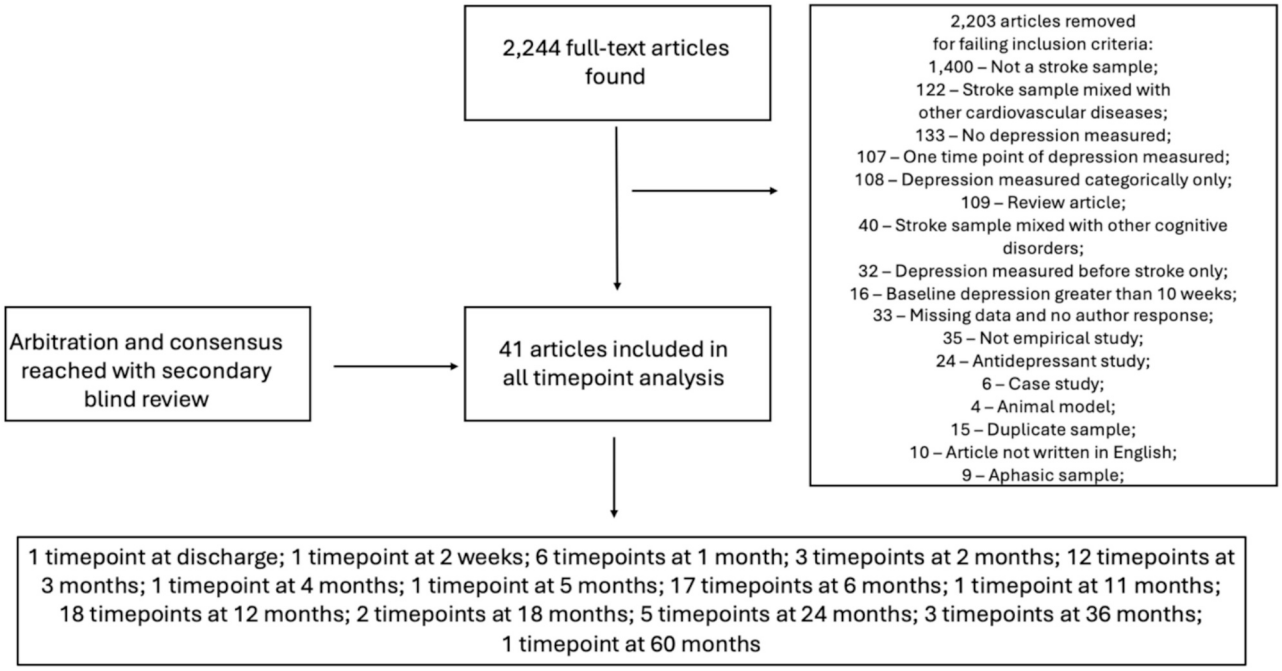

Fig. 1. Flow diagram of literature review.

severity from a multitude of depression self-report measures (see Table 1) across 14 different times post-stroke onset, ranging from 2 weeks to 60 months post-stroke. Across studies, post-stroke timepoints reported by three or more studies were: 3-months ($n = 12$), 6-months ($n = 17$), 12-months ($n = 18$), 24-months ($n = 5$) and 36-months ($n = 3$). A multi-level meta-analysis was therefore conducted between baseline (acute/subacute) and across all timepoints ($n = 72$). Our sensitivity analysis of high risk of bias removed 5 timepoints ($n = 67$), our sensitivity analysis of low power removed 8 timepoints ($n = 64$), and our sensitivity analysis of publication bias removed 18 timepoints ($n = 54$).

3.1. Meta-analysis

Differences in effects between correlation coefficients were observed only in our low power sensitivity analysis. We therefore report all effects at a correlation coefficient of 0. Details of all tested models are reported in Supplemental Table 2. The meta-analysis of longitudinal depression severity in stroke survivors across all timepoints showed no overall significant improvement in depression severity relative to baseline ($p = 0.075$, SMC = −0.078, *95 % CI* [−0.165, 0.008]; see Supplemental Fig. 1), however a within-study effect was found ($p = 0.026$, SMC = −0.074, *95 % CI* [−0.138, –0.009]), and no between-study effect ($p = 0.075$, SMC = −0.078, *95 % CI* [−0.165, 0.008]). No moderation of time was observed ($p > 0.05$). Significant heterogeneity was observed among the 72 assessments ($\chi^2(71) = 24{,}799.9$, $p < 0.001$, $I^2 = 99.7$ %). Sensitivity analysis of publication bias showed a significant improvement in depression severity relative to baseline ($p = 0.002$, SMC = −0.076, *95 % CI* [−0.123, –0.030]; see Fig. 2). A within-study effect, ($p < 0.001$, SMC = −0.073, *95 % CI* [−0.111, –0.034]) and a between-study effect were found, ($p = 0.002$, SMC = −0.076, *95 % CI* [−0.123, –0.030]). While a time moderator analysis revealed no overall effect ($p = 0.22$), an increase in depression severity at 12-months post-stroke was indicated ($p = 0.043$, SMC = 0.190, *95 % CI* [0.0064, 0.374]). However, significant heterogeneity was still observed among the 54 assessments ($\chi^2(53) = 3159.3$, $p < 0.001$, $I^2 = 98.3$ %). No further variations in significance were identified in our sensitivity analyses of low power, nor of high risk of bias.

3.2. Publication bias

The funnel plot for the overall meta-analysis used to identify potential studies of publication bias can be seen in Supplemental Fig. 2. The few outliers may indicate heterogeneity within smaller sample studies. Egger's test of asymmetry further indicated reasonable symmetry across our included studies, $t(70) = 0.14$, $p = 0.89$. The risk of bias assessment showed minimal risk of bias across all 41 included studies. In total, 22 studies demonstrated low risk of bias, 14 studies demonstrated some risk of bias, 5 studies demonstrated high risk of bias, and no studies demonstrated very high risk of bias. Details of these results can be found in Supplemental Table 1.

4. Discussion

This meta-analysis examined the longitudinal course of PSD across multiple timepoints. In the full model that included all eligible studies, depression severity did not significantly differ over time, and time since stroke was not a significant moderator. However, sensitivity analyses revealed a small but statistically significant overall reduction in depression overtime with a brief increase in depression severity at 12-months post-stroke when outliers were excluded, suggesting a possible deterioration effect that may be obscured in the presence of extreme values. Together, these findings suggest that there is an increased risk for the development of severe depression at 12-months after stroke that clinical care may need to account for. This pattern of PSD progression contradicts the findings of previous studies, which reported that depression severity typically peaks at three to six months post-stroke (Dong et al., 2021; Alajbegovic et al., 2014; Whyte and Mulsant, 2002). Although the overall improvement across timepoints remained significant, the pattern at 12-months, may reflect a meaningful shift in mood deterioration during this period. While this pattern should be interpreted as a preliminary general trend rather than definitive evidence of a novel clinical trajectory, it could represent a transitional phase in emotional adjustment that needs to be accounted for in preventative clinical care.

When controlling for outliers, there was an overall improvement in depression severity from the acute and subacute phases of stroke over time. This finding is consistent with the general understanding of Major

Table 1
Included studies.

Authors	Baseline Timepoint	Baseline M (SD), n	6-month timepoint M (SD), n	Depression Measure
Liang et al. (2023)	2–7 days post-stroke	8.15(6.77), $n = 610$	NA	HAMD
Wang et al. (2022)	Within 30 days of admission	35.31 (7.52), n = 101	32.73(8.06), $n = 85$	SDS
Sagen-Vik et al. (2022)	Admission	3.60(2.60), $n = 150$	NA	HADS
Fong et al. (2022)	Admission	7.25(6.59), $n = 188$	NA	HADS
Liu et al. (2022)	72 h	9.43(4.02), $n = 122$	8.13(4.16), $n = 111$	HADS
Volz et al. (2023)	6 weeks	3.68(3.54), $n = 176$	4.15(4.01), $n = 118$	GDS
Orman et al. (2022)	Admission	3.90(3.26), $n = 280$	NA	HADS
Liu et al. (2021a)	Admission	6.20(3.00), $n = 250$	NA	HADS
Kang (2021)	Admission	1.98(2.01), $n = 766$	NA	CESD
Rutkowski et al. (2021)	Admission	11.35 (8.60), n = 105	10.94(9.54), $n = 105$	BDI - II
Hu et al. (2022)	1 Month	3.25(3.11), $n = 65$	NA	PHQ-9
Stolwyk et al. (2022)	Admission	5.56(4.89), $n = 87$	NA	HADS
Liu et al. (2021b)	Admission	4.76(3.80), $n = 217$	6.03(5.93), $n = 209$	HADS
Jones et al. (2020)	Admission	6.40(4.20), $n = 56$	NA	HADS
Lee et al. (2019)	2 Weeks	6.50(6.20), $n = 396$	NA	HADS
Limampai et al. (2017)	Discharge	6.86(3.73), $n = 200$	NA	HADS
Malhotra et al. (2016)	0–10 Weeks	21.30 (10.00), $n = 191$	NA	CES-D
Lewin-Richter et al. (2015)	Admission	4.33(3.61), $n = 96$	4.63(4.27), $n = 71$	GDS-15
Kruithof et al. (2015)	Admission	13.70 (9.30), $n = 206$	NA	CES-D
Eriksen et al. (2016)	Admission	9.60(7.50), $n = 94$	9.70(7.40), n = 94	BDI-II
Brodaty et al. (2013)	Admission	2.90(2.32), $n = 135$	NA	GDS-15
De Ryck et al. (2014)	Admission	4.96(4.38), $n = 125$	NA	CSD
Kim et al. (2013)	2 Weeks	4.70(4.30), $n = 423$	NA	HADS
Yiu et al. (2012)	1 Month	15.29 (9.11), $n = 98$	12.77(9.96), $n = 82$	CES-D
Sibon et al. (2012)	Admission	7.05(4.64), $n = 43$	NA	HAMD
Haley et al. (2011)	Admission	0.95(1.82), $n = 136$	NA	CES-D
Hadidi et al. (2011)	Admission	6.60(4.40), $n = 23$	NA	CES-D
Perrin et al. (2009)	1 Month	9.04(6.63), n = 135	8.88(6.55), $n = 124$	GDS
Secrest and Zeller (2007)	1 Month	19.73 (11.43), $n = 51$	9.08(9.40), $n = 33$	CES-D
Ski and O'Connell (2007)	3 Weeks	44.30 (7.30), n = 13	NA	SDS
Townend et al. (2007)	2–5 Days	2.10(3.91), n = 125	NA	HADS
Pickard et al. (2005)	Admission	18.00 (9.00), n = 124	13.00(10.00), n = 98	CES-D
King et al. (2002)	Admission	12.90 (9.60), $n = 53$	NA	CES-D
Robinson-Smith et al. (2000)	1 Month	13.40 (9.00), $n = 63$	8.60(8.00), n = 63	CES-D
Clark and Smith (1999)	Admission	49.70 (12.40), $n = 60$	48.10(7.50), n = 60	ZDS
Ladwig et al. (2022)	Admission	5.60(4.60), $n = 226$	5.60(4.80), $n = 183$	PHQ-9
Bakken et al. (2012)	Admission	9.70(7.80), $n = 119$	9.90(7.50), $n = 90$	BDI-II
Marsh et al. (2022)	1 Month	4.50(5.40), $n = 80$	3.70(4.30), $n = 48$	PHQ-9
Kang et al. (2013)	2 Weeks	4.60(4.20), n = 423	NA	HADS
Morrison et al. (2000)	Admission	7.80(4.20), $n = 101$	6.40(4.10), n = 71	HADS
Campbell et al. (2023)	7 Days	4.10(4.10), $n = 67$	NA	DASS

Depressive Disorder, which often demonstrates a natural tendency for symptoms to improve over time if left untreated (Kessler et al., 2003). It is important to note that this improvement typically involves prolonged periods of significant suffering for patients, as untreated depression can lead to a chronic course that exacerbates distress and impairs functioning (Warden et al., 2009). While some individuals may experience spontaneous recovery, many endure substantial emotional pain during this period, underscoring the need for timely and effective interventions.

Contrary to previous literature, which indicates that PSD symptoms peak at three to six months post-stroke (Dong et al., 2021; Alajbegovic et al., 2014; Whyte and Mulsant, 2002), our meta-analyses revealed a different pattern. After removing outliers from our analyses, we found that depression severity slowly reduced overtime, with a small increase in depression observed at 12-months, suggesting a non-linear trajectory with potential fluctuations. This discrepancy with prior studies, which implied an increase in PSD severity at three to six months, may be due to the fact that many studies begin measuring PSD at this time point, potentially missing the early stages of PSD progression. Without a baseline reference from the subacute stage, previous studies might not have fully captured the trajectory of depression severity from stroke onset. Our findings suggest that depression severity remains relatively stable during the first three-six months, possibly reflecting the initial period of adjustment to the consequences of the stroke. Notably, the six to 12-month mark often coincides with the completion of rehabilitation for many patients (Langhorne et al., 2011). Providing additional support from six to 12-months could be crucial in preventing a relapse into depression as patients transition out of structured care. These results should not be interpreted as prescriptive for clinical practice but rather as preliminary signals to inform future trial design. Future studies might consider measuring the effect of various clinical intervention strategies on the actualization and sustained reduction of PSD at the six to 12-month interval. Influences of pathological cardiovascular and cognitive comorbidities are also worthy of future consideration.

Commonly comorbid with PSD are anhedonia and fatigue, creating an additional barrier to effective treatment for both patient and clinician (Ning et al., 2024; Sato and Hyakuta, 2023; Segura et al., 2024). A recent study by Ashaie et al. (2023) found that individuals with severe anhedonia or fatigue at three months post-stroke had an increased risk of continued severe anhedonia and fatigue by six to 12-months post-stroke. Cardiovascular comorbidities have also been shown to be predictive of PSD severity (Ma et al., 2024). These include measures of health behavior (diet, physical activity, nicotine consumption, and sleep

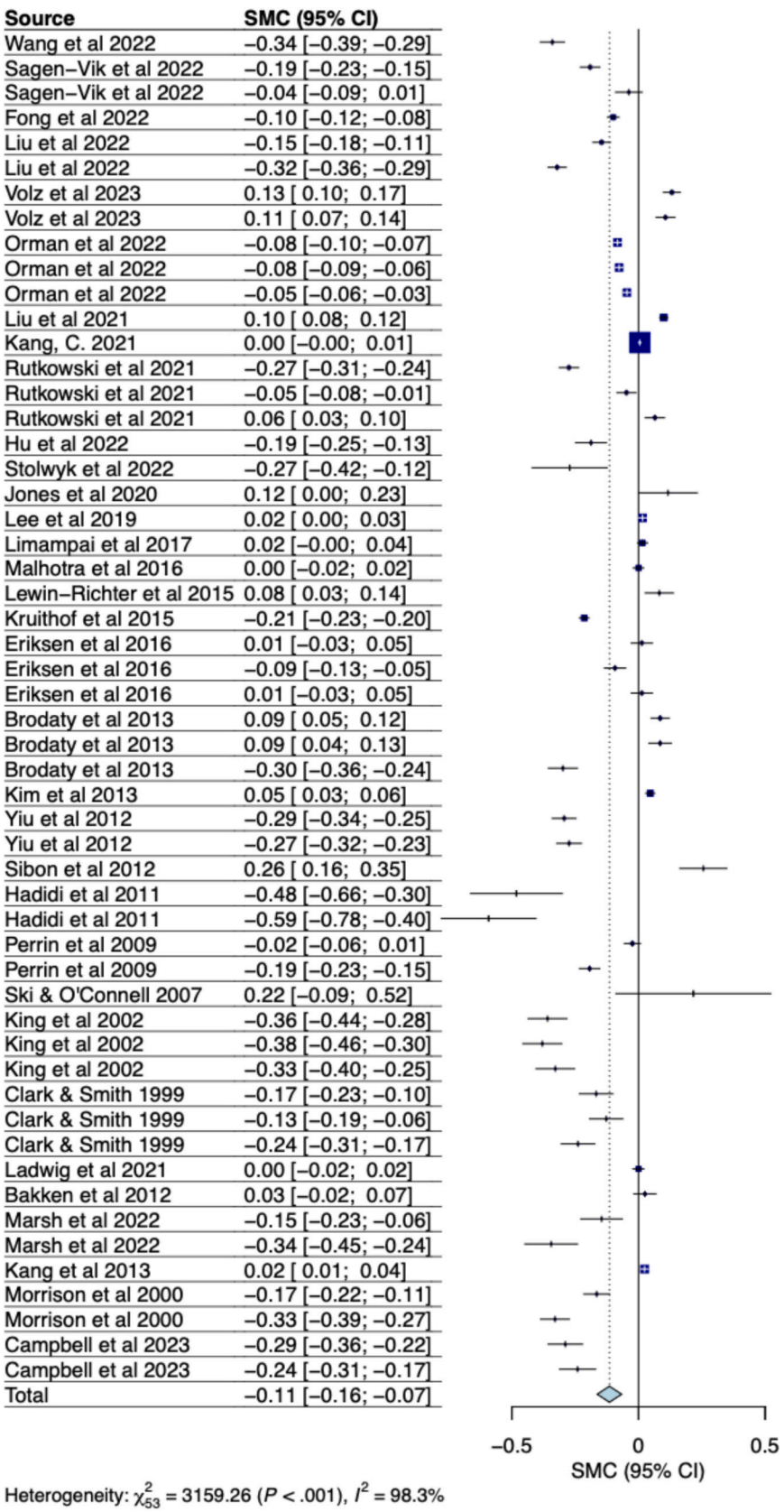

Fig. 2. Severity of depression across all timepoints after heterogeneity sensitivity analysis, relative to baseline; SMC = Standard Mean Change.

habits) and physiology (body mass index, cholesterol, blood glucose, and blood pressure) linked to cardiovascular well-being. While our study has demonstrated clear patterns of PSD on the generalizable scale, there is enough evidence to recommend that future studies of PSD explore detailed affective symptoms, as well as somatic and cardiovascular comorbities that can be linked to patients who suffer from more long-term severe PSD versus patients who recover from PSD. This further underscores the benefit of measuring PSD on a continuous scale rather than a binary one, as comorbities linked to minor changes in longitudinal PSD severity can be clearly identified. Importantly, our analysis is limited to participants who were functionally capable of completing self-report measures, and future research should explore how factors such as stroke severity and functional recovery interact with PSD symptom progression. Moreover, future work should incorporate stroke severity, cognitive functioning, and social support to better understand how these domains moderate or mediate PSD recovery.

By measuring PSD across continuous scales rather than in a binarized format, our findings reveal a gradual improvement in PSD severity over time, which has not previously been reported on a global population level in the PSD literature. Our findings might relate to those of Dong et al. (2021), who reported that individuals without severe PSD symptoms tend to recover at around six months post-stroke, while those with severe PSD symptoms in the first six months post-stroke tend to still have those symptoms at 12-months. Yet other studies suggest there may be several sub-classes of depression trajectory over time (Ayis et al., 2016; Liu et al., 2023). While being able to map the nuanced fluctuations of depression severity globally is still of considerable value, our meta-analyses cannot identify different sub-classes of patients with varying depression trajectories. Future studies should apply person-centered approaches or latent class modeling to examine whether certain subgroups follow distinct symptom trajectories, such as recovery, relapse, or chronic depression. While this meta-analysis does not stratify participants by intervention windows or test intervention effects directly, it reveals longitudinal shifts in depression severity that can inform hypotheses about optimal timing. Future randomized controlled trials should account for our findings when assessing windows of clinical opportunity to test.

A potential limitation of the current meta-analysis is that the within-subjects design might have increased model error due to autocorrelation between pre-and post-measures. However, we took appropriate precautions by testing our models with multiple correlation coefficient corrections for our SMC calculation (see Supplemental Table 2). Notably, accounting for these corrections revealed no difference in the results, thereby enhancing our confidence in the robustness of the effects observed. Our tests also indicated substantial heterogeneity. This is likely due to the natural autocorrelation present in longitudinal data, which can disrupt the normality of variance distributions when comparing pre- and post-scores. As a result, we expected our test to exhibit high degrees of heterogeneity because we used a random effect estimator, which accounts for the relationships between correlated data points. Although study-level attrition was not directly examined, our multi-level model accounts for between-study variance, and the large overall sample helps mitigate concerns of attrition bias. Nonetheless, differential dropout rates remain a potential limitation of the current work. Finally, exclusion of patients suffering from aphasia-related symptoms means that the observed pattern of PSD may not generalize to this important subgroup. Future studies should explore how continuous PSD symptoms manifest in stroke survivors with aphasia, and develop methods to enable their inclusion in longitudinal research.

In conclusion, our study highlights a new pattern in the natural progression of PSD severity over time, with a small decline in depression severity across all timepoints and a small increase at 12-months not previously observed in the literature. Although the magnitude of change was small, continuous PSD severity measures may serve as a scalable clinical tool for monitoring at-risk patients, especially around the 12-month mark where a transient increase in symptoms may signal vulnerability to relapse. A gradual decline in PSD severity over time might be linked to specific predictive factors that can be identified in future studies and harnessed by clinicians, offering hope that recovery is attainable for individuals suffering from severe PSD.

Credit authorship contribution statement

Matthew D. Thurston: Writing – original draft, Formal analysis, Data curation. **Jason B. Mattingley:** Writing – review & editing, Supervision. **Dihua Wu:** Data curation. **Margaret J. Moore:** Writing – review & editing, Supervision. **Lena K.L. Oestreich:** Writing – review & editing, Supervision, Conceptualization.

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Declaration of competing interest

The authors have no conflicts of interest to declare.

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None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2025.120479.

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