Post-Stroke Fatigue and Its Psychological Correlates: Anxiety Components and Quality of Life

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Thesis Portfolio Abstract

Aims: This portfolio explores the aetiology of Post-Stroke Fatigue (PSF) by exploring the predictive strength of two risk factors: anxiety and quality of life (QOL). Anxiety is categorised into three domains: cognitive, physical and behavioural, allowing for a detailed examination of its predictive strength.

Structure: This portfolio includes a systematic review of studies examining the relationship between PSF and QOL among stroke survivors. An empirical paper follows, which examined how three components of anxiety (behavioural, physical and cognitive) accounted for individual differences in PSF. Hierarchical regression models were used to determine whether behavioural, cognitive, and physical anxiety symptoms accounted for additional variance in PSF once demographic, clinical, and emotional factors were consideredThe portfolio finishes by reflecting on this research.

Results: The systematic review found a significant relationship between QOL and PSF levels, consistent across all studies. It also found that demographic factors, such as female sex and younger age, were related to higher PSF and lower QOL. The empirical paper found that depressive symptoms were the strongest emotional predictor of PSF, with behavioural, cognitive, and physical anxiety symptoms showing meaningful associations at the unadjusted level but not contributing independently once depression was included. The paper also found that stroke type (haemorrhagic) significantly improved the model's prediction of PSF.

Conclusions: We found evidence that depressive symptoms were the strongest emotional predictor of PSF, with behavioural, cognitive, and physical symptoms of anxiety only showing meaningful associations before controlling for depression. This may indicate that the relationship between anxiety and PSF is mediated by co-occurring depressive

symptoms. Overall, this study's findings support the need for routine screening of non-motor symptoms and for ensuring that emotional distress is considered within PSF formulation.

The potentially elevated risk observed in specific demographic groups offers a further area for future research. Future work could examine whether demographic characteristics help identify which stroke survivors may be more likely to develop PSF. This would allow for closer monitoring of individuals at higher risk, ensuring that their fatigue is recognised early and not overlooked within rehabilitation pathways.

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Declaration

Material submitted by the portfolio author for the Thesis Proposal as part of the Doctorate in Clinical Psychology at the University of East Anglia has been updated, adapted and used in the completion of the overall thesis portfolio.

Chapter 1. Introduction to the Thesis Portfolio

This thesis portfolio presents research that explores the condition of Post-Stroke Fatigue (PSF) and its predictors. The systematic review appraised studies examining the link between PSF and Quality of life (QOL). Regression analysis was applied in the empirical paper to provide insight into how different components of anxiety contribute differently to understanding the aetiology of PSF.

Background and rationale

Over the past three decades, interest in PSF has grown considerably within the stroke literature. Early stroke rehabilitation research focused primarily on motor impairment, mortality, and secondary prevention, leaving non-motor sequela, such as PSF, under-examined (Lerdal & Gay, 2013). As qualitative and clinical reports accumulated, clinicians began to recognise PSF as a distinct and debilitating symptom rather than a secondary consequence of neurological injury (Staub & Bogousslavsky, 2001). Stroke survivors consistently described PSF as qualitatively different from ordinary tiredness, naming it as persistent and unresponsive to rest, prompting researchers to reconsider its clinical significance (Ingles & Phillips, 1999; Lynch et al., 2007).

Epidemiological evidence further accelerated interest in PSF. Large cohort studies demonstrated that PSF affects a substantial proportion of stroke survivors, with prevalence estimates commonly exceeding 50% (Cumming et al., 2016). Importantly, PSF was shown to persist long after the acute phase of recovery and to exert a profound influence on return to work, cognitive functioning, and overall QOL (Van der Werf et al., 2001). Reflecting its substantial long-term impact, the James Lind Alliance (2016) Stroke Priority Setting Partnership identified PSF as one of the top unanswered research priorities for improving life after stroke, emphasising the need for better understanding and management of this

debilitating symptom. The combination of high prevalence and substantial functional impact positioned PSF as a major unmet need within stroke rehabilitation, causing a need for more targeted research.

Methodological challenges also played a central role in shaping this research target. Early studies used inconsistent definitions and measurement tools, making it difficult to compare findings or synthesise evidence (Chen et al., 2023). This issue led to the development of more standardised approaches, including validated fatigue scales and systematic reviews (Lerdal et al., 2009).

Fatigue is a common and debilitating symptom across many long-term conditions, including multiple sclerosis, chronic fatigue syndrome (CF) and myalgic encephalomyelitis (ME/CFS) (Chaudhuri & Behan, 2004; Kuppuswamy, 2024), Thyroid diseases (Lindstedt et al., 2001) and diabetes (Fritschi & Quinn, 2010). Evidence suggests that PSF may have differences in etiology and phenomenology to fatigue caused by other clinical conditions (Nacul et al., 2011). Phenotyping work indicates that PSF is highly multidimensional, with subtypes that appear to cluster according to type of stroke and functional changes, emotional influences, and altered sensory attenuation, rather than the immune–autonomic and post-exertional profile characteristic of ME/CFS (Kuppuswamy, 2024; Kuppuswamy et al., 2024). In contrast to ME/CFS, where fatigue is typically persistent, unified, and closely tied to post-exertional symptom exacerbation (Nacul et al., 2011), PSF often presents as separable exertional and chronic components with partly distinct predictors (De Doncker et al., 2018; Kuppuswamy et al., 2024). These differences support the conceptualisation of PSF as a stroke-specific fatigue syndrome that warrants dedicated investigation rather than being assumed equivalent to other fatigue conditions.

Despite substantial growth in research on PSF, a significant gap remains in understanding its underlying aetiology, which continues to hinder the development of effective treatments (Hinkle et al., 2019). Although numerous mechanisms have been proposed, including neuroinflammation, altered sensory processing, dysregulated effort perception, depression, and sleep disturbance, none fully explain the occurrence of PSF (De Doncker et al., 2018; Lerdal et al., 2009). One emerging but underexplored factor is anxiety. Several studies have identified anxiety as a significant predictor of PSF, yet the specific pathways linking anxiety to PSF have not been examined in detail (Glader et al.,2002; Chen et al., 2023; Snaphaan et al., 2011). As a result, intervention research has struggled to progress beyond exploratory or small-scale trials, and no treatment has achieved consensus support (Harrison et al., 2021). Without clarity on what drives PSF, it remains challenging to design targeted interventions, evaluate treatment targets, or develop personalised rehabilitation strategies, leaving PSF a highly prevalent yet therapeutically neglected condition.

PSF has also been reported as having a significant impact on QOL. Survivors frequently describe PSF as one of the most disruptive long-term consequences of stroke, often more limiting than motor or cognitive impairments (Thomas et al., 2019). However, despite this consistent patient-reported burden, a systematic review of the relationship between PSF and QOL has not yet been conducted. This lack of synthesis represents a critical gap in the literature. Without a clear understanding of how PSF affects QOL, clinicians cannot determine the true impact of PSF or identify which patients are most vulnerable to poor outcomes. This limits their ability to prioritise screening, allocate resources to areas of greatest need, and design interventions that target the aspects of life most affected by PSF. This prevents clinicians from delivering care that meaningfully improves the QOL of stroke survivors. A systematic review is therefore essential to consolidate existing findings, clarify

the magnitude of the association, and identify methodological limitations that future research must address.

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Chapter 2. The Impact of Post-stroke Fatigue on Quality of Life in Stroke Survivors: a Systematic Review and Meta-Analysis

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Keywords: Post-stroke fatigue, Stroke, Quality of life.

Abstract

Post-Stroke Fatigue (PSF) is a common and burdensome sequela of stroke, yet its impact on Quality of Life (QOL) has not been systematically synthesised. This systematic review examined the association between PSF and QOL in adult stroke survivors. A comprehensive search of four databases (Medline Ultimate, APA PsycINFO, CINAHL Ultimate, and EBSCO EJournals) identified 20 eligible studies. Fifteen studies contributed data to the meta-analysis, and five were examined narratively. Methodological quality was assessed using the Mixed Methods Appraisal Tool (MMAT). Effect sizes were pooled using a random effects model. Results demonstrated a moderate, statistically significant negative association between PSF and QOL ($r = –0.36$, 95% CI [–0.46, –0.25], $p < .001$), indicating that higher fatigue is consistently linked to poorer QOL and shares approximately 13% of its variance. The five remaining papers supported these findings and highlighted potential moderators, including age and sex. Overall, PSF appears to be a substantial and under-recognised determinant of QOL after stroke. These findings underscore the need for routine fatigue assessment, greater integration of PSF into stroke care pathways, and further longitudinal research to clarify causal mechanisms and inform intervention development.

Introduction

Post-Stroke Fatigue (PSF) is defined within the literature as a multidimensional, subjective experience of physical, cognitive, and emotional exhaustion that is disproportionate to effort, not alleviated by rest, and cannot be fully explained by neurological impairment (Lerdal et al., 2011; Staub & Bogousslavsky, 2001).

PSF is a highly prevalent condition among stroke survivors. Although there are conflicting reports of the exact rates, a recent longitudinal observational cohort study reported prevalence rates of 39.11% (Huang et al., 2025), whilst other studies report higher prevalence rates of 48% (Alghamdi et al., 2021). Whilst there is some variation in reported prevalence rates, it is clear that PSF affects many stroke survivors.

Evidence suggests that several demographic factors influence PSF, although findings are not fully consistent across studies. Higher levels of PSF have been reported more frequently among women than men, a pattern observed across multiple observational studies and systematic reviews (Cumming et al., 2016; Wu et al., 2015). Associations between age and PSF are mixed, with some studies identifying greater fatigue in older stroke survivors (commonly defined as those aged 65 and above; Feigin et al., 2014), while others report no significant relationship or elevated fatigue in younger individuals (under 65 years old, Feigin et al.,2014), possibly reflecting differing social and occupational demands (Lerdal et al., 2009). Socioeconomic factors, including lower educational attainment and reduced social support, have also been associated with increased PSF severity, suggesting that demographic and social context may interact with biological and psychological contributors to PSF (Mead et al., 2011).

Despite the high prevalence and clinical significance of PSF, there is currently no universally agreed or recommended measurement tool for its assessment, which presents substantial barriers to research and clinical practice. Multiple self-report instruments are used across studies, including the Fatigue Severity Scale and the Multidimensional Fatigue Inventory, but these tools vary in conceptual focus, psychometric properties, and validation in stroke populations, limiting comparability across studies (Lerdal et al., 2009;

Wu et al., 2015). The absence of a standardised measure hinders routine clinical screening, reduces consistency in outcome reporting, and may drive inconsistencies between studies exploring prevalence, mechanisms, and treatment effectiveness (Cumming et al., 2016). Consequently, PSF remains under-recognised in clinical services and is insufficiently integrated into stroke audit programmes and outcome frameworks. For instance, the Sentinel Stroke National Audit Programme (SSNAP), which provides information on the quality and timeliness of stroke care across England, Wales, and Northern Ireland, does not specifically comment on PSF (SSNAP, 2025).

Whilst there are historic challenges in the measurement of PSF, the significant impact of PSF on stroke survivors is clear. Among stroke survivors, PSF is reported as one of the most bothersome symptoms (Sawant, 2023). Unlike fatigue in the general population, PSF is characterised by extreme exhaustion, which is typically not alleviated by rest (De Groot et al., 2003). PSF has a significant functional impact on stroke survivors. Usman et al (2024) showed that individuals with higher levels of PSF demonstrated poorer mobility, reduced walking capacity, and lower engagement in physical activity, all of which hinder functional recovery and limit participation in meaningful roles. This impact on physical abilities is further supported by Larsson et al. (2025), who showed that PSF can perpetuate a cycle of deconditioning and functional limitation over time. Clinical guidelines also emphasise that PSF affects cognitive functioning, emotional wellbeing, and the capacity to manage everyday tasks (National Clinical Guideline for Stroke, 2023).

The broad impact of PSF is further explored within a biopsychosocial framework, which offers a multidimensional lens for understanding PSF, integrating neurobiological mechanisms, psychological distress, and social environmental factors (Delbridge et al., 2024). This model emphasises that PSF is not a singular entity but reflects a complex interaction between physical deconditioning, mood disturbance, cognitive demands, and reduced social participation (Aarnes et al., 2020; Kuppuswamy, 2020). Empirical evidence supports this perspective; for example, van Ginkel et al. (2015) found that PSF was strongly associated with depressive symptoms and lower levels of physical activity, as well as reduced participation in everyday activities. These findings highlight the interconnected physical and psychosocial nature of fatigue.

PSF is not only relevant on objective functional aspects of stroke survivors lives such as those already discussed, but also because of its influence on more subjective outcomes such as quality of life (QoL). Beyond these observable impairments, PSF also shapes how individuals perceive their own health and wellbeing (Choi-kwon et al., 2005). QoL provides an important subjective indicator of recovery that offers insight into the personal impact of symptoms that may not be visible in clinical assessments. Lower QoL among stroke survivors is itself linked to poorer long-term outcomes, including increased mortality, disability, and reduced social participation (Haley et al., 2011), highlighting the importance of understanding how PSF shapes both functional and subjective aspects of recovery. Despite this, many QoL measures do not explicitly assess PSF, meaning the unique burden of PSF may be under-represented in standard evaluations. Emerging evidence shows that PSF is associated with lower QoL scores independent of stroke severity or disability (Naess et al., 2012; Matérne et al., 2025), suggesting that PSF contributes to diminished wellbeing in ways that extend beyond functional limitations alone. This under-recognised influence is further

compounded by the chronic and fluctuating nature of PSF, which can reduce motivation, restrict participation, and undermine engagement with rehabilitation services (Delbridge et al., 2024; Fan et al., 2025).

Despite consistent evidence linking PSF with poorer outcomes, the relationship between PSF and QOL has not been systematically reviewed. Existing reviews have examined PSF prevalence, correlates, and mechanisms, but none have synthesised the extent to which PSF specifically contributes to QoL outcomes or quantified its independent impact. This represents a critical gap, as PSF is not routinely assessed within QoL evaluations in stroke services, despite growing evidence that it may be a key determinant of survivors' subjective wellbeing. A systematic synthesis is therefore needed to clarify the magnitude and consistency of the PSF and QoL association and to determine how much variance in QoL can be explained by PSF. Understanding this relationship would allow clinicians and researchers to understand how much of QOL can be accounted for by fatigue, and therefore contextualise its impact and relevance for health outcomes. This review therefore aims to examine whether PSF negatively impacts QoL across studies.

Methodology

The protocol for this review was registered with the International Prospective Register of Systematic Reviews (PROSPERO) on the 20th October 2025 (Registration Number: CRD42024530658). Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines (Page et al., 2021) were followed for this review.

Search strategy

An electronic literature search was completed across four databases (Medline Ultimate, APA PsychINFO, CINAHL Ultimate, and EBSCO E-Journals) to retrieve studies published between inception and the 20th of October 2025, when the search was completed. The search terms were generated through consultation with a specialist librarian. The search terms, which included a mixture of keywords and MESH terms, were as follows

(Stroke OR "cerebrovascular accident" OR cva OR post-stroke OR thrombosis) AND (“Post-stroke fatigue”* OR “PSF” OR “Fatigue”) AND (adult OR aged* OR elderly*) AND (“quality of life” OR “QOL”)

A manual search of reference lists of included articles was completed. All articles were extracted to Rayyan (Ouzzani et al., 2016) for screening. Duplicated articles were removed manually using Rayyan’s duplicate identifier function.

Eligibility Criteria

Population

Inclusion Criteria.

Articles examining adult participants (≥18 years) with a current diagnosis or documented history of stroke, including both ischemic and haemorrhagic subtypes, at any stage post-stroke.

Outcomes

Inclusion Criteria.

Articles that employed validated quantitative measures of PSF and QOL. Decisions regarding measure validity were based on validation studies and clear evidence of applicability to stroke or acquired brain injury populations. To ensure conceptual

and psychometric appropriateness, only measures that had undergone validation within stroke cohorts were considered eligible for inclusion.

Study Design and Analysis

Inclusion Criteria.

Quantitative studies (cross-sectional, cohort, case-control, randomised control trials) that report baseline data on both PSF and QOL. Studies were required to include statistical analyses capable of informing the relationship between PSF and QOL.

Exclusion Criteria.

Qualitative studies, narrative reviews, protocols, editorials, and conference abstracts lacking full-text data. Studies with unextractable, incomplete data, or no relational data on PSF or QOL. Studies not published in English were excluded due to lack of translation resources.

Screening and Selection

Citation searching was conducted on included studies, and no additional eligible records were identified. A Kappa Agreement Procedure (McHugh., 2012) was applied to ensure screening reliability. All titles and abstracts were screened against predefined eligibility criteria, with 25% independently reviewed by a second reviewer.

Quality assessment

Quality assessment was conducted to evaluate the methodological rigor and potential risk of bias across included studies. The Mixed Methods Appraisal Tool (MMAT; Hong et al., 2018), version 2018, was selected for the present review because of its capacity to appraise varying study designs within a unified framework, its emphasis on core methodological criteria, and its structured approach to assessing validity, relevance, and

coherence. The MMAT includes a set of screening questions followed by five methodological domains, each containing five criteria tailored to specific study designs (qualitative, quantitative non-randomised, quantitative randomised, quantitative descriptive, and mixed methods, full screening questions can be found in (Appendix B) . These criteria are designed to determine whether key aspects of study design, conduct, and reporting have been implemented in a way that minimises the risk of bias. A percentage score was calculated to indicate the proportion of criteria met within the relevant domain(s). Higher percentages reflect a lower risk of bias. Risk of bias was assessed for all studies by the primary reviewer (AM), with the secondary reviewer (EB) reviewing 25%; disagreements were resolved between the two reviewers without the need of a third reviewer.

Data extraction

The following data was extracted from the included studies by the primary researcher: author, year, country, study design, sample size, population, setting, age, sex, outcome measure type. Fifteen of the included studies had the required data for meta-analyses; outcome measure means scores (SD), effect size type, effect size, confidence intervals and standard error. The named author contact of the remaining five were contacted requesting further data with a two-week time limit to return this for inclusion in meta-analysis. No authors responded with required data. These five papers are therefore commented on qualitatively in relation to any additional contribution they make to the current evidence base.

Data synthesis

The included studies reported associations using a variety of effect size metrics (such as correlations, regression coefficients and odds ratios). Effects were converted into a common metric to allow quantitative synthesis. Fisher's converted effect size was chosen because it provides a variance-stabilized transformation of correlation coefficients and allows effects from diverse statistical outputs to be expressed on a comparable scale.

When studies reported Pearson's correlation coefficients (r), these were transformed to Fisher's z using the standard Fisher transformation (Borenstein et al., 2009). When only standardized regression coefficients (β) were available, these were converted to approximate correlations using established formulas (Lüdecke, 2019; Borenstein et al., 2009) before applying the Fisher's z transformation. For studies reporting odds ratios or risk ratios, effect sizes were converted to their corresponding correlation estimates using recommended methods (Borenstein et al., 2009; Cleophas
& Zwinderman., 2017)) and subsequently transformed to Fisher's z.

For studies reporting multiple effect sizes from the same sample, effect sizes were combined into a single composite correlation to avoid unit-of-analysis errors (Borenstein et al., 2009), using the mean of correlations.

Standard errors for each Fisher's z value were calculated using its sampling variance, and 95% confidence intervals were computed accordingly. Because Fisher's z is normally distributed, all meta-analytic models were estimated using the transformed values. A copy of the data conversion table can be found in Appendix C.

Meta-analysis

Effect sizes were synthesized using a random-effects model, reflecting anticipated heterogeneity across studies in terms of populations, measurement methods, and study designs. Analyses were conducted using R (R Core Team., 2024)

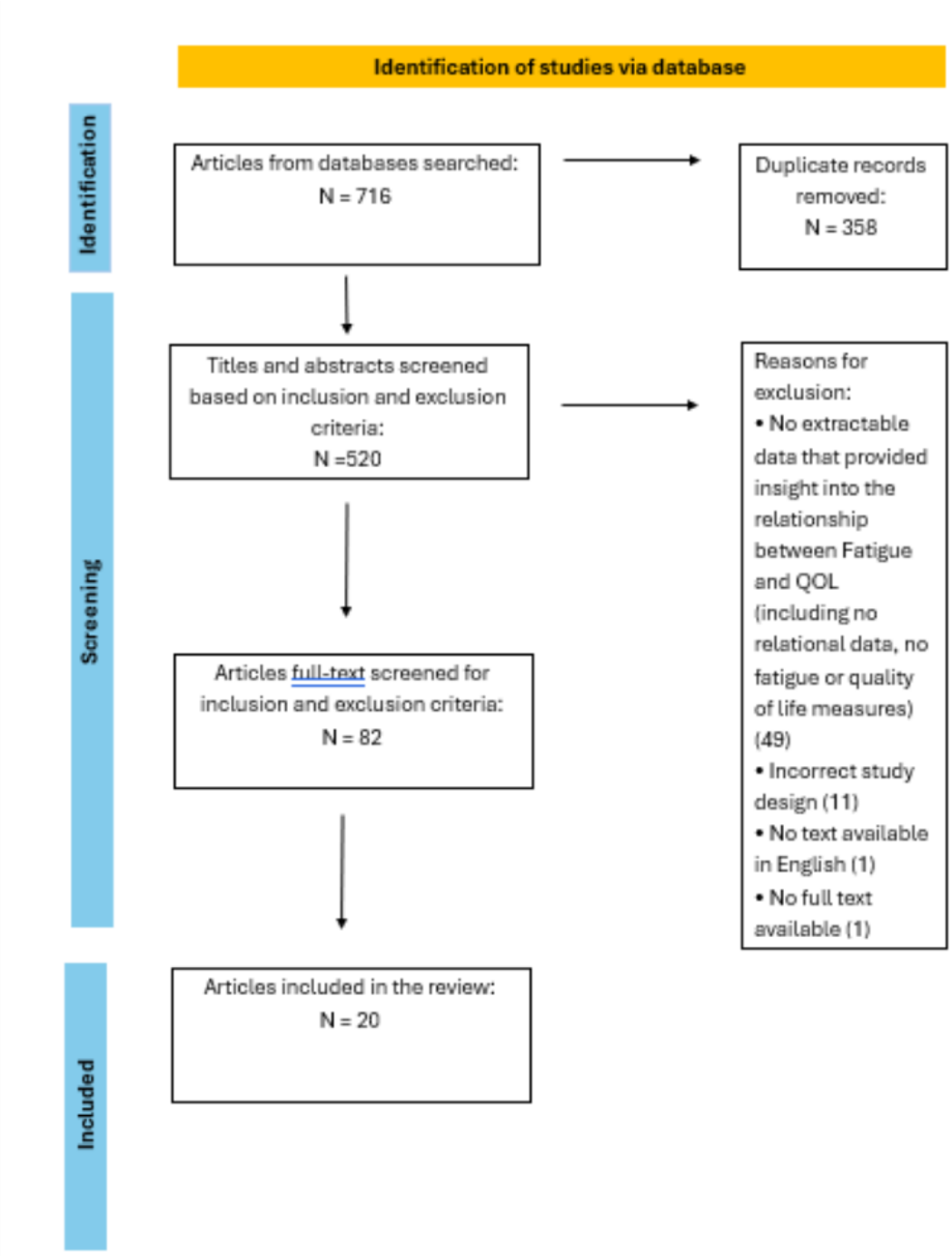
Results

Study inclusion

At title and abstract screening agreement for inclusion was 78.33% (102/130) comprising 96 exclude–exclude matches and 6 include–include matches. All discrepancies were resolved through discussion without escalation to a third reviewer. From 716 original articles identified, 82 articles were selected for full-text screening. During full text screening, 25% of papers were again double screened, achieving 100% agreement. In total, 20 studies were included after screening. Common reasons for ineligibility included the non-reporting of relational statistics between PSF and QOL. Twenty eligible studies were included in this review, 15 were included in meta-analysis, and five were commented on qualitatively. A flowchart of the screening and selection process can be seen in figure 1.

Figure 1

Flowchart of the article identification, screening and selection process, adapted from guidelines and templates published by Page et al. (2021)



Quality Assessment and Risk of Bias

Table 1 shows the paper's outcomes for each MMAT question, with an overall quality percentage score.

Table 1

Quality assessment results using the MMAT

Study	MMAT design classification	Criterion 1	Criterion 2	Criterion 3	Criterion 4	Criterion 5
Abdulla et al., 2019	Quantitative descriptive	Y	N	Y	Y	Y
Aked et al., 2025	Quantitative non-randomised	Y	Y	Y	Y	Y
Bruijn et al., 2015	Quantitative descriptive	Y	N	Y	N	Y
Chen et al., 2014	Quantitative descriptive	Y	N	Y	N	Y

Dong et al., 2025	Quantitative non-randomised	Y	Y	Y	Y	Y
Duncan et al., 2015	Quantitative non-randomised	Y	Y	Y	Y	Y
Ghosh et al., 2024	Quantitative descriptive	Y	N	Y	Y	Y
Kang et al., 2025	Quantitative non-randomised	Y	Y	Y	Y	Y
Kim et al., 2020	Quantitative non-randomised	Y	Y	Y	Y	Y
Kusec et al., 2023	Quantitative descriptive	Y	N	Y	N	Y
Moreno et al., 2018	Quantitative non-randomised	N	Y	Y	Y	Y
Naes et al., 2012	Quantitative descriptive	Y	N	Y	N	Y
Odetunde et al., 2025	Quantitative descriptive	Y	N	Y	Y	Y

Ogwumike et al., 2022	Quantitative descriptive	N	N	Y	Y	N
Pan et al., 2023	Quantitative non-randomised	Y	Y	Y	Y	Y
Port et al.,2007	Quantitative Non-Randomised	Y	Y	Y	Y	Y
Pederson et al., 2022	Quantitative non-randomised	Y	N	Y	N	Y
Sibbritt et al., 2022	Quantitative non-randomised	N	Y	Y	Y	Y
Tang et al., 2010	Quantitative non-randomised	Y	Y	Y	Y	Y
Visser-Meily et al., 2008	Quantitative non-randomised	N	Y	Y	Y	Y

Note.

Quantitative descriptive criteria:(1) Is the sampling strategy relevant to address the quantitative research questions? (2) Is the sample representative of the population under study?(3) Are measurements appropriate?(4) Is there an acceptable

response rate?Mixed-methods criteria: (1) Is the mixed- methods research design relevant to address the qualitative and quantitative research questions, or the qualitative and quantitative aspects of the mixed-methods question? (2) Is the integration of qualitative and quantitative data relevant to address the research questions? (3) Is appropriate consideration given to the limitations associated with this integration, in a triangulation design?

Characteristics of each of the final studies are provided in Table 2. One study did not report demographic characteristics (Sibritt et al., 2022)

Table 2

Study Characteristics

Study ID (Meta)	Authors	Country	Study Design	Sample Size (N)	Population/Condition	Setting	Outcome measures	Participant Characteristics	Notes/finding
1	Abdulla et al., 2019	Saudi Arabia	Cross-sectional	147 stroke, 70 controls	Subacute/chronic stroke	Hospital	FSS SSQOL-A-E	Mean age 59.63 (10.97) 53.1% female	FSS correlated with QOL (broader finding for the study was that the FSS showed good psychometric properties)

2	Pedersen et al., 2024	Norway	Prospective cohort (1y & 4y)	144	Predominantly mild/moderate strokes	Used 4-year-old data from stroke survivors who were in medical files from hospital stay	FSS SSQOL	Mean age 67.3 (10.9) 36% Female	Fatigue is higher in worse QOL groups
3	Sibbritt et al., 2022	Australia	Cross-sectional	576	Stroke survivors	Data was obtained from a sub-study of the 45 and Up Study, including 576 Australian adults who had been diagnosed with a stroke	MFIS-5 EQ-5D	Not provided	Higher fatigue scores correlated with lower quality of life

4	Dong et al., 2025	China	Cross-sectional	271	Stroke survivors	Hospital	FSS SS-QOL	Age 18-39:13.7% 40-59: 86.3% 47.2% Female	FSS associated with QOL
5	Kusec et al., 2023	UK	Longitudinal	105	Long-term stroke survivors (>2y)	Community.	FSS EQ-5D-5L	Mean age 72.9 59% male	Fatigue prevalence 51.4%; QOL associations
6	Odetunde et al., 2025	Nigeria	Cross-sectional	102	Stroke survivors	Community	FSS SS-QOL	Mean age 59.98 (11.59) 52.9% Female	FSS correlated with QOL

7	Kim et al., 2020	South Korea	Cross-sectional	112	Mild/moderate stroke in rehab	Rehabilitation unit	FSS WHO-QOL	Without Insomnia Mean age 66.11(12.8) 48.6% female With Insomnia: Mean age 66.37(15.7) 40% female	Higher fatigue – poorer QOL.

									Fatigue group Mean age 57 (51,62) 59.9% Male	
8	Kang et al., 2025	China	Cross-sectional	300	Stroke patients	Hospital	FSS SS-QOL	Non-Fatigue group	Mean age 56 (47.5,61) 62.8% Male	Higher fatigue correlated with lower quality of life.

9	Chen et al., 2014	Taiwan	Cross-sectional	218	Stroke survivors	Neurology Department – Hospital	FSS SS-QOL	Mean age 61·2(11·4) 73.48%	Fatigue correlated with QOL
10	Pan et al.,2023	China	Cross-sectional	176	Stroke survivors	Hospital	SRF-S HRQOL	Mean age 65.44 (12.43) 36.36% female	Fatigue correlated with QOL.
11	Moreno et al.,2018	Spain	Case–control	181 (92 stroke, 89 control)	First-ever TIA/minor stroke	Stroke unit - hospital	FAS EQ-5D-DL	(Stroke cohort) Mean age 59.5 (8.2) 72.5% male	Fatigue correlated with quality of life

12	Aked et al.,2025	Sweden	Quantitative non-randomised	167	First-ever stroke	Clinic/home	FAS HRQOL	Median age 72 40% female	Higher fatigue correlated with worse QOL
13	Ogwumike et al., 2022	Nigeria	Cross-sectional	110	Stroke survivors ≤1y	Physiotherapy clinics	FSS SS-QOL	Mean age 60.9 58.2% male	FSS and QOL correlated
14	Naes et al., 2012	Norway	Cross-sectional	328	Stroke survivors	Hospital/community	FSS HRQOL	Mean age 67.7 37% female	A significant association between worse HRQOL scores and an increasing number of fatigue symptoms
15	Visser-Meily et al., 2008	Netherlands	Cross sectional	141	Stroke patients (aneurysmal subarachnoid haemorrha	Community	SSQOL and FSS	Mean age 51.4 (12.3) 66.7% female	Depression, anxiety and fatigue are strongly related to decreased SSQOL.

					ge), previously inpatients, now living in the community			66.7% Female	
16	Duncan et al., 2015	UK	Prospective cohort	136	Acute stroke	Clinical research facility/home	FAS EUROQOL	Mean age 72 65% men	QOL predicted fatigue
17	Tang et al., 2010	Hong Kong	Cross-sectional	458	Acute ischemic stroke	Acute stroke unit	FSS HRQOL	Mean age 66.2 62% male	Fatigue and quality of life correlated
18	Port et al.,2007	Netehrlands	Prospective cohort	223	Stroke patients	Rehabilitation centre	FSS HRQOL	Mean age 57.3 (11.1) 40% male	fatigue correlated with QOL

19	Bruijn et al., 2015	Netherlands	Cross-sectional	171 stroke patients, 61 controls	Young ischemic stroke patients	Hospital	FAS WHOQOL-BREF 26)	Mean age 46.3 (7.1) 44.1% male	Higher fatigue is associated with lower QOL
20	Ghosh et al., 2024	India	Cross-sectional	100	Stroke patients	neurology outpatient department - community	FSS SSQOL	Mean age 45.8 (8.54) 82% male	Higher fatigue is associated with worse QOL

Design and Methodology

Fifteen studies were cross-sectional, and five used other designs: Duncan et al. (2015) and Pederson et al. (2024) used a prospective study design. Port et al. (2007) used a prospective cohort design, Aked et al. (2025) used a population-based cohort design, and Kusec et al (2023) used a longitudinal design.

Participants

The included studies featured 3,922 stroke survivors. Across studies, samples consisted of adult stroke survivors spanning a broad age range, with most studies reporting mean ages between the mid-50s and late-60s, although several focused specifically on younger or working-age adults (de Bruijn et al., 2015; Kang et al., 2025; Pedersen et al., 2024). Sex distribution was generally mixed, with most samples including a slightly higher proportion of men.

Participants were from 12 countries. Ethnicity was inconsistently reported and, therefore, could not be analysed. The time elapsed since the stroke event varied considerably between studies, from within the last year to four years post-stroke.

Stroke participants were sampled from a range of settings, including hospital inpatient settings and community outpatient clinics. Stroke sample sizes ranged from 92 (Moreno et al.,2018) to 576 (Sibritt et al., 2022).

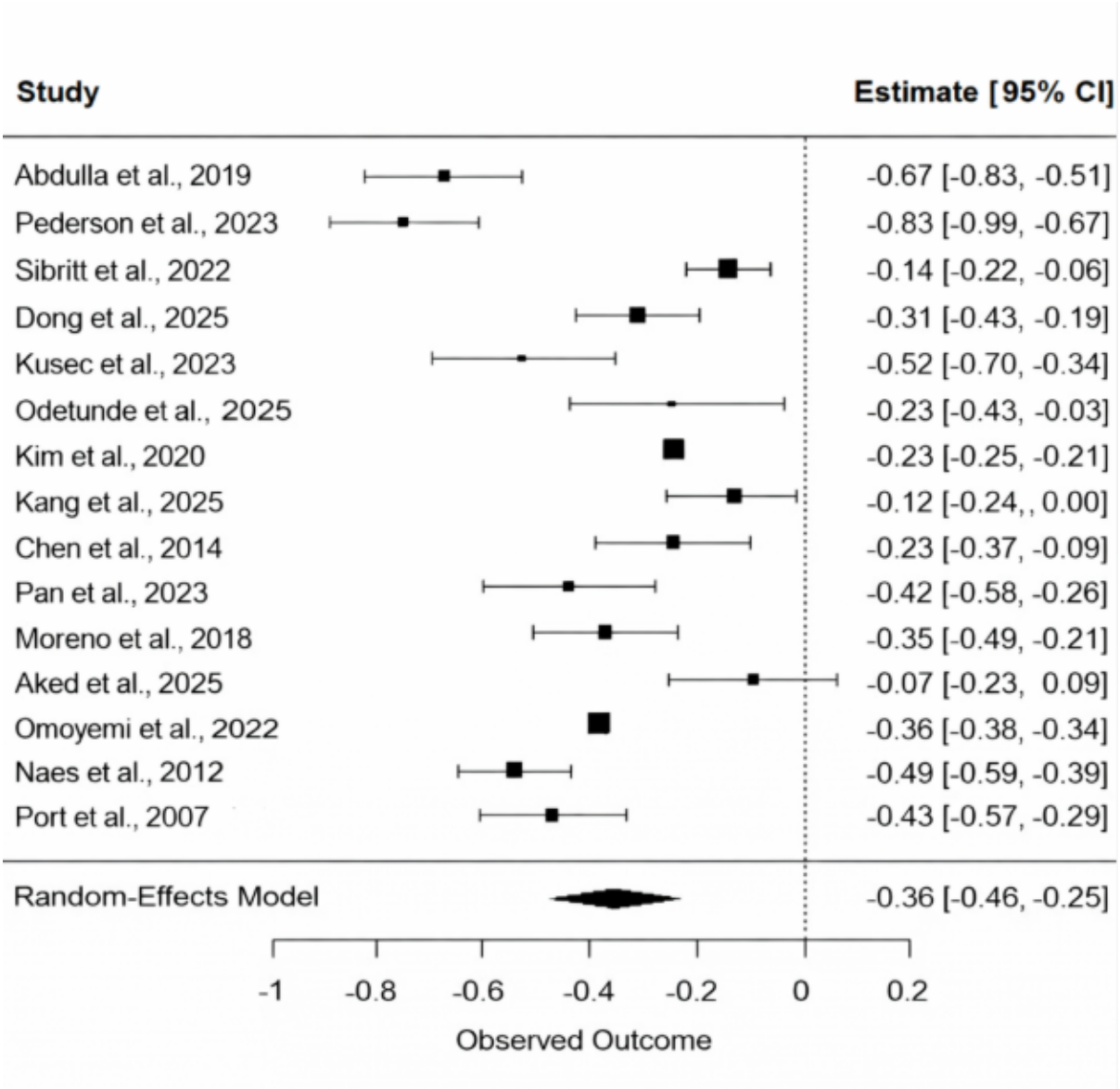
Main Findings

A random-effects meta-analysis was conducted to synthesise the association between PSF and QOL across the available studies. Figure 2 presents the forest plot summarising effect sizes across all included studies. The pooled effect indicated a moderate, statistically significant negative correlation between PSF and QOL ($r = –0.36$, 95% CI [–0.46, –0.25], $p < .001$), demonstrating that higher levels of PSF were reliably linked to poorer QOL outcomes. Despite the consistency in effect direction, there was moderate heterogeneity among studies ($I^2 = 62\%$), suggesting meaningful variability in sample characteristics, assessment methods, and study contexts.

Although the magnitude of effects varied, the majority of confidence intervals did not cross zero, reinforcing the robustness of the observed association. This pattern supports PSF as a meaningful and consistent determinant of reduced QOL after stroke, even across heterogeneous study designs and measurement tools.

Figure 2

Forest plot of effect sizes across included studies



*Note***.** All effects were coded so that higher PSF corresponds to higher QOL. When necessary (opposite scale direction or reversed predictor/outcome), effect sizes were reversed to ensure a consistent direction of effects for comparison.

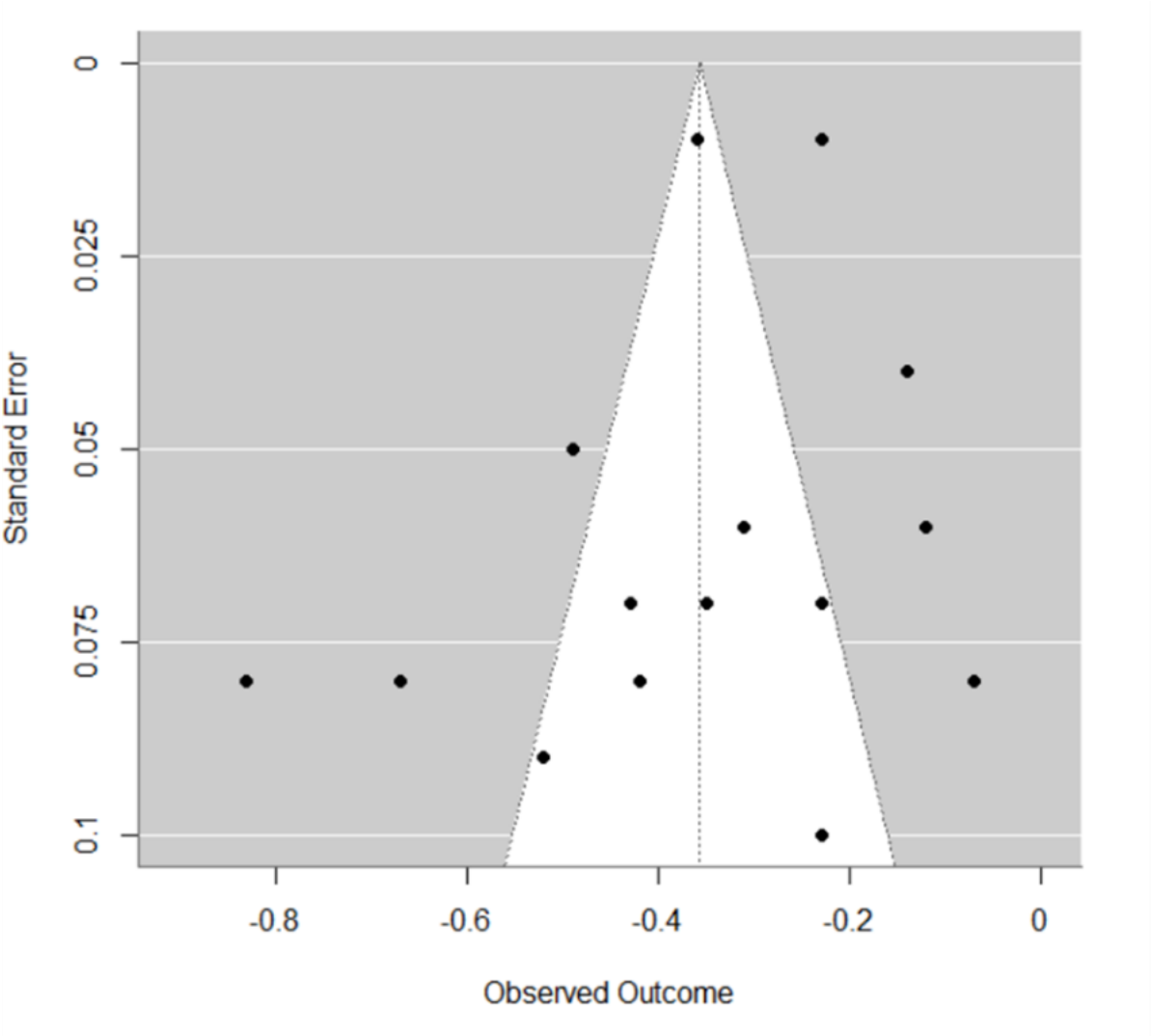
The funnel plot shown in Figure 3 displays a slight degree of asymmetry, suggesting the potential presence of publication bias. Specifically, several smaller studies appeared to report stronger negative correlations than larger ones, which may indicate preferential

publication of studies finding more substantial effects. However, the asymmetry was mild, meaning that while bias cannot be ruled out, it is unlikely to fully account for the observed effect. The plot nonetheless highlights the need for larger, methodologically rigorous studies to improve precision and to reduce the influence of publication bias on future meta-analytic estimates.

Because studies one and two presented as strong outliers, potential factors contributing to this variation (setting, time since stroke, stroke type, measurement tools, country, and site characteristics) were examined, but none appeared to account for the unusually strong effect sizes observed in these studies.

Figure 3

Funnel plot of included studies



Note. Figure three shows a funnel plot.

Because of the strong effect sizes observed in Studies 1 and 2, a sensitivity analysis was conducted to assess their influence on the overall meta-analytic estimate. Excluding these two studies yielded a reduced pooled effect size ($r = –0.30$, 95% CI –0.38 to –0.22, $p < .0001$), compared to the full sample ($r = –0.36$). This suggests that the excluded studies

contributed to the strength of the original association. However, heterogeneity increased substantially ($I^2 = 94.73\%$), indicating greater variability among the remaining studies.

Narrative synthesis

The five studies that could not be included in the meta-analysis all reported findings consistent with the overall direction of the pooled estimate, showing that higher PSF was associated with poorer QOL. Beyond this general pattern, their results offered additional insight into how this relationship varies across recovery stages, demographic groups, and co-occurring symptoms.

Several studies highlighted the importance of time since stroke. Visser-Meily et al. (2008) found that lower PSF was consistently linked with better long-term QOL following subarachnoid haemorrhage, while Duncan et al. (2015) similarly reported that individuals with higher PSF experienced significantly poorer QOL over time. Tang et al. (2010) further demonstrated that PSF was negatively correlated with QOL in the early post-stroke period, suggesting that PSF affects well-being across both the acute and chronic phases of recovery.

Other studies emphasised demographic influences. De Bruijn et al. (2015) showed that the association between PSF and QOL was particularly pronounced in younger stroke survivors (aged 18–49), indicating a potentially greater psychosocial impact in this age group. Ghosh et al. (2024) reported that the relationship was strongest among female stroke survivors, pointing to possible sex-based differences in vulnerability.

Across all five studies, co-occurring psychological symptoms, particularly anxiety and depression, were consistently identified as additional factors that further reduced QOL.

Discussion

The meta-analysis demonstrated a moderate and statistically significant negative association between PSF and QOL, indicating that higher PSF severity is consistently linked to poorer QOL amongst stroke survivors. The size of the pooled effect ($r = –0.36$, 95% CI [–0.46, –0.25], $p < .001$) suggests that PSF is not merely a secondary or transient symptom but a substantial determinant of QOL, explaining approximately 13% of its variance. This proportion is clinically meaningful. Most widely used QOL measures do not directly assess fatigue, meaning that PSF is influencing domains that are not explicitly assessed, such as mobility, emotional well-being, social participation, and perceived health status. It also highlights that fatigue is significant to individuals' subjective perception of their health. Clinically, this underscores the importance of routinely assessing PSF, rather than assuming it will be captured indirectly through general QOL scores.

In relation to the further nuanced findings from the narrative synthesis, there may be various explanations for these. For instance, de Bruijn et al., (2015) findings of a pronounced association within younger stroke survivors, a possible explanation may be that adults under 50 are more likely to be employed, raising children, or engaged in education at the time of stroke, meaning that even mild fatigue can interfere substantially with work participation, family responsibilities, and social functioning (Naess et al., 2006; Putaala et al., 2009). Younger survivors also report a stronger expectation of full recovery and a greater sense of identity disruption when invisible symptoms such as PSF persist (Stone et al., 2019). Some research suggests that younger adults experience more psychosocial distress and role limitation after stroke than older adults, despite having milder physical impairments (Marini et al., 1999; Varona et al., 2004). Overall, the evidence suggests that PSF may have a

disproportionately disruptive impact on younger adults, strengthening the case for considering demographic context when interpreting the relationship between PSF and QOL.

In relation to Ghosh et al. (2014), who found that the PSF and QOL relationship was strongest among female stroke survivors, some cohort studies have shown that women experience more persistent fatigue than men after stroke and are more likely to develop post-stroke depression and anxiety (Hinkle et al., 2017; Schepers et al., 2006). Morris et al. (2013) reported that women also face greater caregiving and domestic role expectations, which can intensify the functional impact of fatigue and limit opportunities for rest and recovery. Additionally, women often report poorer long-term QOL and greater psychosocial burden after stroke, even when physical impairment is similar to that of men (Gall et al., 2012; Bushnell et al., 2014). These factors align with findings such as those from Ghosh et al. (2024), where the PSF and QOL relationship was strongest among female participants.

Taken together, the meta-analysis and narrative synthesis suggest PSF is a substantial and clinically meaningful determinant of QOL across stroke survivors. While the pooled effect demonstrates a consistent moderate association between higher PSF and poorer QOL, the narrative findings suggest that this relationship may be amplified in certain groups, such as younger adults and females. Understanding these broader and subgroup-specific patterns is essential for informing future research aimed at reducing the QOL burden associated with PSF.

Methodological Limitations

The moderate heterogeneity suggests that differences in study design may influence the observed effect, limiting the certainty and precision of the findings. For example,

QOL was measured using diverse tools (for example, SF36, EQ5D, SSQOL, WHOQOLBREF), which differ in domain structure, sensitivity to psychosocial versus physical functioning, and measurement of, and response to, stroke-specific deficits. These conceptual differences limit the comparability of findings and may partly explain between-study variability. Slight funnel-plot asymmetry also raises the possibility of publication bias, with smaller studies showing stronger associations potentially overrepresented.

Second, many included studies used cross-sectional designs and single-time-point self-report measures. Such designs limit causal inference and are vulnerable to mood effects, recall bias, and the influence of co-occurring symptoms. Given that anxiety and depression are strongly associated with both PSF and QOL, the absence of consistent adjustment for these confounders may inflate observed associations.

There was also considerable inconsistency in how PSF was conceptualised and measured. Several studies treated PSF as a unitary construct without distinguishing it from overlapping emotional or cognitive symptoms. Only a minority statistically adjusted for anxiety or depression, and few attempted to separate PSF from these domains at the measurement level. This raises the possibility that some of the variance attributed to PSF may reflect unmeasured emotional distress, limiting confidence in the association's specificity.

A further limitation concerns the inconsistent reporting and adjustment for stroke severity across the included studies. More severe strokes are associated with greater disability and lower QoL (Haley et al., 2011; Naess et al., 2012), and higher stroke severity has in some studdies been linked to increased PSF levels (Lerdal & Gay, 2013; Cumming et al., 2016). However, many studies in this review did not control for severity. As a result, part of the observed association between PSF and QoL may reflect underlying differences in stroke severity rather than the independent contribution of fatigue. Although some individual studies

have demonstrated that PSF predicts QoL even after adjusting for severity (Naess et al., 2012; Matérne et al., 2025), the lack of consistent control across the evidence base limits the strength of inferences that can be drawn.Finally, the absence of standardised cut-offs or scoring approaches further complicates synthesis. Studies variously used continuous scores, dichotomised thresholds (such as FSS > 4; FAS > 22), or regression-based classifications.

Clinical Implications

The findings reinforce that PSF is central to people's subjective ratings of their health and quality of life, even though fatigue is not explicitly captured in many QOL instruments. This association between PSF and poorer QOL supports the need for routine screening for PSF across all stages of stroke recovery, including long-term follow-up. PSF is often underrecognized and undertreated in clinical practice. However, given its substantial impact on QOL, PSF should be routinely monitored, with thought into possible treatable factors, and considered a core rehabilitation target, in line with The National Institute for Health and Care Excellence guidance (NICE, 2023) rather than an inevitable consequence of stroke.

Moreover, the results suggest that fatigue-targeted interventions may represent a potential pathway to improving QOL outcomes. Interventions addressing behavioural, psychological, and physical contributors to PSF, such as cognitive-behavioural strategies, and graded physical activity, may produce benefits that extend beyond PSF reduction alone. However, the current evidence base for PSF interventions remains limited and mixed. For example, trials such as the POSTIF study (Mead et al., 2022) did not demonstrate a significant treatment effect on fatigue outcomes, highlighting the challenges of effectively modifying PSF in clinical practice. Many existing studies are small, heterogeneous, and vary

in intervention content and outcome measurement, limiting firm conclusions. Therefore, while integrating PSF management into multidisciplinary stroke rehabilitation programmes appears conceptually justified, particularly given the substantial association between PSF and QOL observed in this meta-analysis, stronger, methodologically rigorous trials are needed before definitive clinical recommendations can be made.

Future Research

Future research should prioritise longitudinal study designs with repeated assessments of PSF and QOL to clarify temporal relationships and potential causal pathways. Such designs would allow researchers to examine how PSF trajectories evolve and how changes in PSF relate to subsequent changes in QOL.

The limitations noted within the measures highlight an area of difficulty in assessing the impact of stroke in particular clinical domains and for research purposes. The challenge posed by studies that differ in their conceptualisation of PSF and QOL poses a significant barrier to synthesising and understanding the data. Whilst the included measures pose these difficulties, there is currently no agreed-upon gold standard measurement tool for PSF. This results in researchers having to make individual judgments about the best fits for their research, without actual evidence to back them. This poses significant barriers to needed research into stroke, and PSF is noticeably affected. Creating a widely accessible, accurate, and dimensional PSF measure should be a focus of future research to ensure our understanding of PSF continues to grow reliably.

Conclusion

This systematic review adds to the current evidence base, suggesting that PSF is a prevalent, persistent, and under-addressed determinant of poor QOL among stroke survivors. The moderate negative association observed across diverse studies highlights the broad and multidimensional impact of PSF on QOL. These findings underscore the importance of recognising PSF as a central component of post-stroke care. Integrating systematic PSF assessment that is accessible to all stroke survivors regardless of abilities, utilising flexible means of assessment and adaptations, and further research into management strategies in stroke rehabilitation care has the potential to improve QOL and overall recovery outcomes for this growing population.

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Chapter 3: Bridging Chapter

The systematic review examined the relationship between PSF and QOL, identifying a consistent pattern in which greater PSF severity was associated with poorer QOL outcomes. However, variability emerged in the extent to which studies accounted for psychological comorbidities, such as anxiety, which are known to influence both fatigue perception. Anxiety symptoms were inconsistently measured or controlled for, despite theoretical and empirical evidence suggesting that anxiety may amplify or maintain PSF. This raised the possibility that the observed relationship between PSF and QOL may be partially confounded by unmeasured anxiety related mechanisms.

If anxiety contributes uniquely to the experience of PSF, then studies that treat PSF as a unitary construct may obscure important underlying heterogeneity. For example, cognitive aspects of anxiety (such as, interpretations of bodily sensation, intense worrying and rumination) may heighten perceived fatigue severity, while behavioural components (such as, avoidance or activity restriction) may reduce physical conditioning and thereby exacerbate PSF over time. Similarly, physiological arousal may be misattributed to fatigue, inflating symptom reports. Without distinguishing these pathways, the true nature of the PSF and QOL relationship remains uncertain, and interventions may fail to target the mechanisms most responsible for maintaining fatigue.

This conceptual ambiguity highlighted a methodological gap, to the best of our knowledge, no research has examined the differential contribution of specific anxiety components to PSF. As a result, it is unclear whether anxiety causes a general influence on PSF or whether symptom clusters (cognitive, behavioural, or physical) play a more prominent role. Addressing this gap is needed to better understand PSF and for informing more person-centred interventions.

The empirical paper that follows therefore aimed to investigate the unique contributions of the three core components of anxiety to PSF, and whether these associations persist when controlling for other psychological conditions, such as depression. This approach directly addresses the concerns raised in the systematic review by clarifying the extent to which anxiety related mechanisms may underpin or exacerbate PSF, offering a more nuanced understanding of the pathways linking PSF to reduced QOL in stroke survivors.

Chapter 4. Exploring the link between components of anxiety and Post-Stroke Fatigue

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Keywords; Post-stroke fatigue, Stroke, Anxiety.

Abstract

Post-stroke Fatigue (PSF) is a prevalent and debilitating yet poorly understood condition that significantly impairs quality of life (QOL). There remains no evidence-based treatment. Emerging evidence suggests anxiety may contribute to PSF, but the aetiology of this relationship has not been studied. This study aimed to examine the predictive strength of different components of anxiety on PSF severity in stroke survivors. Seventy-five participants completed a cross-sectional online survey, using validated measures of fatigue, anxiety, and depression. . Hierarchical multiple linear regression was used to examine the contribution of behavioural, cognitive, and physical anxiety symptoms to PSF after accounting for demographic and clinical covariates. Across three preliminary models, behavioural, physical, and cognitive anxiety symptoms each showed meaningful associations with PSF severity; however, none remained independently predictive once depressive symptoms were included, with depression emerging as the strongest emotional predictor. Stroke type (haemorrhagic/ischaemic) significantly improved prediction of PSF. Findings highlight the importance of routine psychological screening and suggest that both depression and anxiety warrant consideration in PSF formulation. The interaction observed between anxiety and depression in fatigue levels also offers new insight as to why current fatigue treatments focusing solely on depression may be showing no effect. Future research should include randomised control trials, which could test the causal mechanisms involved in this relationship. This would offer more definitive conclusions than can be drawn from observational designs and would directly inform the development of targeted rehabilitation pathways.

Introduction

Worldwide stroke's account for approximately 5.5 million deaths annually (Mukherjee & Patil, 2011) and one hundred thousand people have a stroke every year in the United Kingdom (Stroke Association, 2013). In addition to the substantial impacts on individuals and their families, there can be significant health and social care service implications. The current UK annual societal cost of stroke is £25.6 billion (King et al., 2020). The two most common strokes are ischaemic and haemorrhagic, with around 87% of strokes being ischaemic (Johns Hopkins Medicine., 2022). The effects of strokes differ between individuals, primarily based on the location and extent of damage to the brain. However, strokes commonly result in physical, behavioural, cognitive, and psychological difficulties.

Estimates show that one in four people will experience an anxiety disorder in their lifetime. Following a stroke, the chances of experiencing anxiety rise to one in three people (Raften et al., 2018). The cognitions in post-stroke anxiety often involve worrying about recurrence of stroke, having a fall/injury, returning to work, and one's self-image or identity (Raften., et al 2018). Physically, individuals can experience a range of symptoms that may include but are not limited to palpitations, dizziness, or trembling (Campbell et al., 2013). Behaviourally it has been noted that some people may avoid activities because of worry about what may happen during them and adopting safety behaviours that can lead to long-term maintenance of the anxiety (Ahrens et al., 2022). Collectively these components of anxiety negatively impact on the quality of life in stroke survivors (Tang et al., 2013).

Post stroke anxiety has been reported as highest in the first two weeks following stroke but often extends for up to and surpassing five years (Tang et al., 2013). Difficulty often lies in the support that individuals receive for anxiety post-stroke.

Post-stroke anxiety receives significantly less attention compared to other psychological problems (Campbell et al., 2013). This is reiterated in reports from the Stroke Association (2021) who found that only two in ten stroke survivors reported being given any information or support in coping with anxiety. This impacts stroke survivors' recovery in relation to emotional wellbeing, but also in relation to other post-stroke complications, to which anxiety is increasingly being shown within the literature to contribute (Wright et al., 2017). Further to this, research has begun looking at links between stroke type and risk of developing anxiety, Ojabemi et al., (2020) study found haemorrhagic stroke (or a subtype of it) is associated with a higher risk of post-stroke anxiety. It is important that we develop our understanding of post-stroke anxiety and its contributions to other difficult symptoms after stroke, so that appropriate interventions can be developed and delivered.

Post-Stroke Fatigue (PSF) is another common stroke sequela. PSF is characterised by extreme exhaustion or tiredness, which can be cognitive (defined as a decline in task performance that requires sustained mental activity) or physical (the body's sensation of extreme, persistent tiredness) and is not typically alleviated by rest (De Groot et al., 2003). PSF is defined in many ways throughout the current literature, which creates different estimates of how many people are affected by it. A recent study estimated on average 48% of individuals will experience PSF and found that up to 40% of individuals report it as one of their worst symptoms (Cumming et al., 2016). PSF has been shown to be a barrier to people's everyday life, impacting negatively on social aspects, career aspects, sleep, and increasing risk of suicidal ideation and attempts (Zhang et al., 2020). Acciarresi et al. (2014) found that patients who felt they had not made a full recovery following stroke, were significantly more likely to experience PSF than those who made a full recovery. Furthermore, for patients who showed excellent neurological and physiological recovery, PSF was often one of the only

persistent effects of stroke, and severely impacted these individuals return to pre-stroke functioning. This suggests that PSF is ubiquitous and persistent across all levels of recovery.

Studies into the aetiology of PSF have considered neurological deficits, medical comorbidities, demographic factors, and other lifestyle considerations (Hinkle et al., 2017). Research on neurological factors appear consistent in findings that severity of stroke as well as infarct volume do not appear to be associated with PSF (Amelilie et al., 2015). However, there is some research that suggests leucoaraiosis is predictive of developing fatigue (Acciarresi et al., 2014, Scopelliti et al., 2023, Naess et al., 2012). Beyond neurological mechanisms, several studies highlight the role of psychological factors, particularly post-stroke depression , in the development and maintenance of PSF. Depression is one of the most consistently reported correlates of PSF after stroke, with evidence showing that depressive symptoms predict both the presence and severity of PSF independent of neurological impairment (Choi-Kwon et al., 2005; Snaphaan et al., 2011; Lerdal et al., 2011). Emotional distress and reduced motivation have also been identified as contributing factors, suggesting that PSF may arise partly from cognitive emotional processes rather than solely from structural brain changes (Schepers et al., 2006; van der Werf et al., 2003).

Recently, research has focused on the contribution of anxiety to PSF. Mutai et al. (2017) collected data from 101 stroke patients to investigate aspects of PSF and the clinical factors contributing to its development. Their study showed that anxiety was independently associated with multidimensional aspects of PSF, more specifically they found anxiety to be strongest in predicting general fatigue and physical fatigue among stroke survivors, suggesting there are detailed links between anxiety and PSF, as opposed to a general link that remains the same across different subtypes. However, this offered no further details about the nature of this relationship or causality. Galligan et al. (2016) investigated the

link between depression, pain, anxiety, and PSF to understand this relationship further. Their results found that these variables accounted for 32% of the variance in fatigue scores. More specifically, their results support that the relationship between anxiety and PSF stayed significant when controlling for the influence of other study variables (depression and pain). Whilst Galligan et al. (2016) and other PSF studies have shown the importance of anxiety related factors, the contribution of the different components of anxiety, or how these may differ in their contributions to individuals' PSF levels, remains unknown.

A deeper understanding of the aetiology of PSF, including anxiety's role, is needed because of current difficulties in treatment. Despite PSF creating difficulties for stroke survivors, there is no clear agreed upon treatment protocol, and the current treatment methods are showing low success in the reduction of symptoms. Treatment of PSF has been identified as one of the top ten research priorities in stroke research (Pollock et al., 2012). This research priority is reiterated in a recent meta-analysis looking at the management strategies for PSF, which concluded that studies were urgently needed to understand the aetiology of PSF to provide better-targeted treatments (Chen et al., 2023). Wu et al. (2015) conducted a meta-analysis of 12 registered control trials for PSF treatment's, these included pharmacological interventions, psychological interventions, and physical training (Wu et al., 2015). Their findings showed there was no sufficient evidence for the use of any intervention to treat PSF. Based on this, they recommended further research into treatment effectiveness be conducted. However, other research has suggested psychological treatment, such as cognitive behavioural therapy (CBT) has been shown to be more effective than pharmacological interventions (Su et al., 2020).

A recent study evaluating the effectiveness of cognitive behavioural therapy (CBT) for post-stroke anxiety and depression also reported a significant reduction in fatigue, despite

fatigue not being a direct target of the intervention (Sharma & Varshney, 2022). This incidental improvement suggests that anxiety symptoms may play a contributory or maintaining role in PSF. Notably, CBT primarily addresses cognitive, behavioural, and physiological components of anxiety, implying that improvements in these domains may indirectly alleviate PSF. In contrast, The Nottingham Post-Stroke Fatigue Intervention (POST-IF) used a brief, CBT based manualised programme to support people experiencing post-stroke fatigue. It combined psychoeducation with behavioural and cognitive self-management strategies, including activity monitoring, graded activity scheduling, pacing and energy-conservation techniques, problem-solving, sleep-hygiene advice, and cognitive reframing of unhelpful beliefs about fatigue. In contrast to Sharma & Varshney's (2022) CBT style intervention that also addressed anxiety, the POSTIF trial focused predominantly on depression-based intervention mechanisms such as behavioural activation and long-term condition self-management models, aiming to stabilise routines and increase engagement in meaningful activity. The POSTIF trial in contrast did not find any significant reduction in PSF, suggesting a more complex underlying emotional mechanism may exist for the development of PSF. Together, these findings underscore the importance of examining which specific dimensions of anxiety are most strongly associated with PSF. Understanding these mechanistic pathways could help refine therapeutic approaches and improve targeted interventions for individuals experiencing PSF after stroke.

Although anxiety is frequently identified as a correlate of PSF, little is known about whether specific components of anxiety are differentially associated with PSF after stroke. Evidence from general and clinical populations suggests that anxiety is not a unitary construct, and that subdomains characterised by physical or somatic tension may have unique relationships with PSF. For example, a longitudinal population-based study found that an "anxious tension" symptom category, reflecting somatic tension, was among the strongest

predictors of burnout symptoms, including exhaustion and fatigue (Hagström et al., 2025). Similarly, in occupational samples, somatic arousal associated with chronic burnout has been linked to persistent fatigue, sleep disturbance, and elevated cortisol, pointing to physiological hyperarousal as a contributor to long-term physical exhaustion (Melamed et al., 1999). Furthermore, a recent study among athletes demonstrated that somatic anxiety (physical symptoms rather than worry) was significantly associated with burnout, supporting the idea that physiological anxiety components may drive fatigue and exhaustion even in non-clinical populations (Yang et al.,2024). These mechanisms may be especially relevant after stroke, a context in which worry about recurrence and long-term uncertainty is highly prevalent (Townend et al., 2006; Chun et al., 2021), potentially heightening the impact of somatic anxiety on the experience of fatigue.

Given that PSF remains one of the most common and disabling consequences of stroke and considering that previous research has typically treated anxiety as an influencing variable of fatigue, testing whether specific anxiety components, cognitive, physical and behavioural, differentially relate to PSF represents a clear and important gap. If certain anxiety subcomponents are especially predictive of PSF after stroke, this could have direct clinical relevance. For example, guiding the development or tailoring of psychological interventions to reduce PSF and improve recovery outcomes.

The aim of this study is to understand the relationships between anxiety and PSF in adults in more detail, looking at each component of anxieties contribution to PSF levels. We propose that understanding detailed predictors could help us understand which components of anxiety to focus on in relation to treatment of PSF, for instance if there was higher predictive

strength of cognitive symptoms of anxiety, this could indicate cognitive symptoms should be a treatment target to reduce PSF levels.

Research questions

Are there differences in the predictive strength of the different components of anxiety (cognitive, physical and behavioural) on PSF levels in stroke survivors? How are different components of anxiety related to PSF?

Materials and Method

Design

The study used a cross-sectional correlational design. Participants were recruited and took part via the completion of an online survey, using convenience sampling.

Participants

Seventy-five participants were recruited. Based on previous research and recommendations (Towfighi et al., 2023), the following participant characteristics were recorded; age, ethnicity (based on the 18 categories used by the Office of National Statistics), and gender. Also collected were participants stroke characteristics, including time since last stroke, type of stroke (ischaemic or haemorrhagic) and location of stroke (left cerebral hemisphere, right hemisphere, cerebellum, corpus callosum and brainstem).

Inclusion criteria

We included adult stroke survivors who had been diagnosed with stroke by a medical professional, were eighteen years of age or older, were able to access and complete online activities, and were able to respond to questions independently**.**

Exclusion criteria

We excluded individuals who had another neurological condition (other than stroke) that could affect fatigue levels.

Procedure

The study was advertised through social media, such as posts on Facebook and X stroke support pages, with a short description of the study, a study poster and the link to complete the questionnaire. The study was advertised twice on each support page, a month apart. The study was also advertised through a local brain injury (including strokes) rehabilitation charity, posters of the study with a QR code to take part were placed in the rehabilitation centre waiting rooms. This was achieved through contacting the senior psychologist from this centre and requesting this assistance with recruitment.

Further advertisement was achieved through two celebrities, who had experienced a stroke and shared this publicly, sharing the study poster and a link to complete the study on their X social media page.

Measures

Fatigue Severity Scale (FSS)

The FSS (Appendix B) is a unidimensional self-report questionnaire designed to assess the impact of fatigue on daily functioning (Krupp et al., 1989). It consists of nine items rated on a 7-point Likert scale, with higher scores indicating greater fatigue severity. The measure has demonstrated strong internal consistency (Cronbach's $\alpha = .89$) and good construct validity across clinical populations, including individuals with stroke (Lerdal et al., 2005). Following feedback from patient and public involvement (PPI), a brief explanation clarifying that the measure assesses the severity and functional impact of fatigue, rather than simple tiredness was added.

Behavioural Outcomes of Anxiety (BOA)

The Behavioural Outcomes of Anxiety scale, developed by Kneebone et al. (2012) (Appendix C), has 10 questions, which are rated on a scale of often (3) – never (0), with higher scores indicating higher anxiety. The measure includes the required questions related to behavioural aspects of anxiety, which are absent from other measures. The measure offers high internal consistency $\alpha = .81$ (Linley-Adams et al., 2014). The BOA was used as an indicator of behavioural manifestations of anxiety, such as the degree of behavioural avoidance if situations, restlessness, and being easily startled. However, it does contain other non-behavioural items.

Beck Anxiety Inventory (BAI)

The Beck Anxiety Inventory (BAI; Osman et al., 1997) (Appendix D) contains 21 questions, rated on a scale of not at all (0) – severely (3), with higher scores indicating higher anxiety. The measure offers good coverage of the symptoms of anxiety that we aim to

measure. The measure offers high internal consistency α= .92 (Muntingh et al., 2011). Factor analytic investigations of the BAI consistently demonstrate its multidimensional structure. In the original manual, Beck et al.,(1988) described it as capturing somatic, affective, and cognitive symptoms. Subsequent exploratory and confirmatory studies have identified between two symptoms of anxiety, physiological and cognitive (Osman et al., 1997), supporting the splitting of symptoms into somatic and cognitive domains. Therefore, this measure will be used as a measure of both physiological and cognitive anxiety.

Patient Health Questionnaire-8 (PHQ-8)

A measure of depression was also operationalised into the study to identify its influence on fatigue and anxiety, and to enable us to factor out this as a confounding variable, as evidence suggests depression also contributes to fatigue (Gilligan et al., 2016). The Patient Health Questionnaire-8 (PHQ-8; Kroenke & Spitzer, 2002) (Appendix E) was used. The PHQ-8 consists of 8 questions based on the DSM-IV criteria for major depression. Participants rate their responses to questions on a scale of 0 (not at all) to 3 (nearly every day). The PHQ-8 has high internal consistency α=.84-.88 (Tore et al., 2023)

Ethics

Ethical and information governance processes have been followed in line with the British Psychological Society (BPS) codes of conduct (The British Psychological Society. 2021, Health & Care Professions Council., 2016). The project received ethical approval through the University of Easy Anglia Faculty of Medicine and Health Sciences Research Ethics subcommittee (ETH2425-1844). A copy of this can be found in appendix F.

Patient and Public Involvement

Patient and public involvement (PPI) from one stroke survivor helped shape the project to be accessible to the target population. Through feedback given on possible measure we had considered using, our chosen measures were then chosen based on those identified by this

individual as the most accessible regarding cognitive load. We included a short explanation, as to why our fatigue measure asked questions that are not purely about being physically tired, as this was identified as something that could be confusing if you were not aware fatigue extended to other domains.

Data Analysis

A prior power analysis was not conducted because the study analysis plan involved hierarchical multiple regression with several predictors, and there was insufficient prior evidence to specify reliable expected effect sizes for each predictor. Instead, Green's (1991) rule of thumb was used, as it provides a widely accepted and design-appropriate minimum sample size criterion for multiple regression when effect sizes are uncertain. Green's guidelines (50 + 8m for testing the overall model) is specifically intended for exploratory regression models with multiple correlated predictors, such as those used in this study. Using this approach allowed us to determine whether the available sample was adequate for evaluating overall model effects while acknowledging that the study would be underpowered to detect very small unique contributions of individual predictors.

Collected data were inspected and prepared for regression and mediation analysis. Variables were cleaned to meet assumptions for multivariate analysis. The process included missing data handling, variable coding, composite scale computation, linearity checks and confounder variable checks. Missing data were inspected for each scale. Checks for missing data followed the recommendations by Tabachnick and Fidell (2019) for maintaining validity while minimizing case loss. No missing data were identified; therefore, all cases were retained

for analysis and no listwise or casewise deletion procedures were necessary.All rows from the original dataset were retained, as no participants exceeded the missing-item thresholds established for the FSS, BAI, PHQ-8, or BOA measures.

Hierarchical multiple linear regression was selected as the primary analysis to examine the extent to which different components of anxiety explained variance in PSF after accounting for demographic and clinical factors. In line with Green's (1991) recommendations for regression sample adequacy, the available sample size was sufficient for detecting small–medium effects at the model level. Anxiety components were operationalised using the subscale structures. Scores from the Behavioural Outcomes of Anxiety (BOA) measure were used to index behavioural anxiety symptoms. To run planned analysis, total scores on the behavioural outcomes of anxiety measure were categorised as behavioural symptoms of anxiety. In line with literature supporting this categorisation (Linley-Adams et al., 2014) the BAI was split into cognitive and physical symptoms of anxiety in accordance with literature supporting this splitting (Osman et al., 1997). Variables were entered in hierarchical blocks so that demographic, clinical, depressive, and anxiety variables were added sequentially. This structure allowed examination of the incremental predictive value of the anxiety components over and above demographic, stroke-related, and depressive factors known to be associated with PSF

Confounders were explored by identifying relationships between various factors and the anxiety outcomes. Any significant confounders were added to the regression models as covariates.

Additionally, separating the models aligned with the planned sample size recommendations. According to Green (1991), a minimum of $50 + 8\ m$ participants is

recommended for testing overall model fit, where *m* is the number of predictors. With 75 participants and no more than three predictors per model, all models met this criterion.

This modelling strategy also allowed for clearer interpretation of each anxiety dimension's unique contribution to the outcome variables (anxiety and fatigue scores), without the confounding influence of overlapping variance.

This analysis plan was checked through discussion with course staff with expertise in regression analysis, an independent university statistician who supported the planned analysis, as well as the universities associate Professor in Medical Statistics (MK).

Results

Table 1 Demographic overview

		N	%	M	SD
Sex					
	Male	19	25.3%		
	Female	56	74.7%		
Age				49.47	(11.39)
Ethnicity	White	68	90.67%		
	Asian	2	2.67%		
	Black	2	2.67%		
	Mixed	2	2.67%		
	Other	1	1.33%		
Time since stroke	0-6 Months	17	22.7%		
	7-12 Months	13	17.3%		
	>12 Months	45	60%		
Type of stroke	Ischaemic	53	70.7%		
	Haemorrhagic	15	20%		
	Don’t know	7	9.3%		
Location of Stroke					

Left side	25	33%
Right side	30	40%
Cerebellum	3	4%
Corpus Callosum	1	1%
Brainstem	7	9%
Don’t Know	9	12%

All data met required linearity checks. Type of stroke and the PHQ-8 were correlated significantly with PSF scores and were therefore included as confounders within the regression models. Correlation checks also showed high correlations ($r > .68$) between anxiety measures, suggesting multicollinearity. Observations of multicollinearity were considered, given the conceptual similarity of the anxiety subscales. Where multicollinearity was detected, variance inflation factor (VIF) values were examined, with initial models exceeding the commonly accepted threshold ($VIF > 5$), indicating potential instability in coefficient estimates (Field, 2018). Further inspection revealed that the cognitive symptoms subscale contributed most strongly to multicollinearity (Condition Index = 44.165, Variance Proportion = .99). To address this, the analysis was restructured into three separate regression models, which reduced VIF values to below 2, indicating acceptable multicollinearity and improved model stability. As all models used the same standardized beta estimates, results were comparable across models. Table 2 shows raw correlations.

Table 2

Raw Correlations

Variable	Cognitive total score	Physical total score	Behavioural total score	Stroke type	Fatigue total score	PHQ score
Cognitive total score	1	r=.75**	r=.74**	r=.09	r=.36**	r=.60**
Physical total score	r=.75**	1	r=.68**	r=.12	r=.43**	r=.61**
Behavioural total score	r=.74**	r=.68**	1	r=.08	r=.43**	r=.48**
Stroke type	r=.09	r=.12	r=.08	1	r=.35**	r=.25*
Fatigue total	r=.36**	r=.43**	r=.43**	r=.35**	1	r=.48**
PHQ total	r=.60**	r=.61**	r=.48**	r=.25*	r=.48**	1

Note: *p < .05, **p < .01

Primary analysis

A series of hierarchical regressions were conducted to examine whether a particular anxiety subtype best predicted fatigue severity after controlling for stroke type and PHQ-8 scores.

A hierarchical multiple regression was conducted to examine whether behavioural symptoms of anxiety predicted fatigue severity. Model 1 showed that behavioural symptoms significantly predicted fatigue, $F(1, 73) = 16.69$, $p < .001$, accounting for 18.6% of the variance. Adding stroke type in Model 2 significantly improved the model, $F(2, 72) = 14.47$, $p < .001$, increasing the explained variance to 28.7%. In Model 3, the addition of depressive symptoms resulted in a significant overall model, $F(3, 71) = 11.24$, $p < .001$, explaining 32.2% of the variance. However, in the final model, behavioural symptoms were not a significant predictor of fatigue ($\beta = .245$, $p = .062$). Stroke type remained a significant predictor ($\beta = .267$, $p = .011$), while depressive symptoms were not significant at the $p < .05$ level ($\beta = .255$, $p = .059$).

Table 3

Hierarchical Multiple Regression Predicting Fatigue Severity from Stroke Type and Behavioural Anxiety symptoms

Predictor	B	SE B	β	t	p
Model 1					
(Constant)	29.90	5.22		5.73	< .001
Behaviour total score	0.99	0.24	.431	4.09	< .001
Model 2					
(Constant)	22.71	5.41		4.20	< .001
Behaviour total score	0.93	0.23	.406	4.06	< .001
Stroke type	6.07	1.91	.318	3.19	.002
Model 3					
(Constant)	25.32	5.48		4.62	< .001
Behaviour total score	0.56	0.30	.245	1.90	.062
Stroke type	5.09	1.94	.267	2.62	.011
PHQ score	0.52	0.27	.255	1.92	.059

Note. N = 75. Model 1 predictors: Behaviour total. Model 1: $R^2 = .186$, Adjusted $R^2 = .175$, $F(1, 73) = 16.69$, $p < .001$. Model 2 predictors: Behaviour total, Stroke type. Model 2: $R^2 = .287$, Adjusted $R^2 = .267$, $F(2, 72) = 14.47$, $p < .001$. Model 3 predictors: Behaviour total, Stroke type, PHQ score. Model 3: $R^2 = .322$, Adjusted $R^2 = .293$, $F(3, 71) = 11.24$, $p < .001$.

A hierarchical multiple regression was conducted to examine whether physical anxiety symptoms predicted fatigue severity. Model 1 showed that physical anxiety significantly predicted fatigue, $F(1, 73) = 16.26$, $p < .001$, accounting for 18.2% of the variance. Adding stroke type in Model 2 significantly improved the model, $F(2, 72) = 13.59$, $p < .001$, increasing the explained variance to 27.4%. In Model 3, the addition of depressive

symptoms resulted in a significant overall model, $F(3, 71) = 11.11$, $p < .001$, explaining 32.0% of the variance. However, in the final model, physical anxiety symptoms were not a significant predictor of fatigue ($\beta = .227$, $p = .072$), whereas stroke type ($\beta = .254$, $p = .014$) and depressive symptoms ($\beta = .278$, $p = .033$) remained significant.

Table 4

Hierarchical Multiple Regression Predicting Fatigue Severity from Stroke Type and Physical Anxiety Symptoms

Predictor	B	SE B	β	t	p	95% CI
Model 1						
(Constant)	40.44	2.82		14.32	< .001	34.82, 46.07
Physical total score	0.66	0.16	.427	4.03	< .001	0.33, 0.98
Model 2						
(Constant)	33.22	3.59		9.25	< .001	26.06, 40.38
Physical total score	0.60	0.16	.391	3.87	< .001	0.29, 0.91
Stroke type	5.82	1.93	.305	3.02	.004	2.04, 9.60
Model 3						
(Constant)	31.44	3.60		8.74	< .001	24.29, 38.58
Physical total score	0.35	0.19	.227	1.83	.072	-0.03, 0.72
Stroke type	4.85	1.93	.254	2.51	.014	1.01, 8.69
PHQ score	0.57	0.26	.278	2.18	.033	0.05, 1.08

*Note***.** N = 75. Model 1 predictors: Physical total score. Model 1: $R^2 = .182$, Adjusted $R^2 = .171$, $F(1, 73) = 16.26$, $p < .001$. Model 2 predictors: Physical total score, Stroke type. Model 2: $R^2 = .274$, Adjusted $R^2 = .254$, $F(2, 72) = 13.59$, $p < .001$. Model 3 predictors: Physical total score, Stroke type, PHQ score. Model 3: $R^2 = .320$, Adjusted $R^2 = .291$, $F(3, 71) = 11.12$, $p < .001$.

A hierarchical multiple regression was conducted to examine whether cognitive anxiety symptoms predicted fatigue severity. Model 1 showed that cognitive anxiety significantly predicted fatigue, $F(1, 73) = 11.02$, $p = .001$, accounting for 13.1% of the variance. Adding stroke type in Model 2 significantly improved the model, $F(2, 72) = 10.99$, $p < .001$, increasing the explained variance to 23.4%. In Model 3, the addition of depressive symptoms resulted in a significant overall model, $F(3, 71) = 10.13$, $p < .001$, explaining 30.0% of the variance. However, in the final model, cognitive anxiety symptoms were not a significant predictor of fatigue ($\beta = .139$, $p = .268$), whereas stroke type ($\beta = .255$, $p = .016$) and depressive symptoms ($\beta = .332$, $p = .012$) remained significant.

Table 5

Hierarchical Multiple Regression Predicting Fatigue Severit from Stroke Type and Cognitive Anxiety Symptoms

Predictor	B	SE B	β	t	p
Model 1					
(Constant)	8.14	12.84		.634	.528
Cognitive total score	1.02	.306	.362	3.32	.001
Model 2					
(Constant)	2.96	12.25		.241	.810
Cognitive total score	.938	.291	.334	3.22	.002
Stroke type	6.14	1.96	.322	3.11	.003
Model 3					
(Constant)	19.1	13.3		1.43	.157
Cognitive total score	.392	.351	.139	1.12	.268
Stroke type	4.86	1.97	.255	2.47	.016
PHQ score	.677	.262	.332	2.59	.012

Note. N = 75. Model 1 predictors:Cognitive total score. $R^2 = .131$, Adjusted $R^2 = .119$, $F(1, 73) = 11.020$, $p = .001$. Model 2 predictors: Cognitive total score, Stroke type. $R^2 = .234$, Adjusted $R^2 = .213$, $F(2, 72) = 10.989$, $p < .001$. Model 3 predictors: Cognitive total score, Stroke type, PHQ score. $R^2 = .300$, Adjusted $R^2 = .270$, $F(3, 71) = 10.134$, $p < .001$.

A one-way ANOVA was conducted to examine whether PSF severity differed between participants with ischaemic and haemorrhagic stroke. The analysis showed a significant effect of stroke type on PSF, $F(1, 66) = 6.12$, $p = .016$. Stroke type accounted for approximately 8.5% of the variance in PSF scores ($\eta^2 = .085$). Participants with haemorrhagic stroke reported higher PSF severity than those with ischaemic stroke.

Discussion

The aim of this study was to examine whether different components of anxiety (cognitive, physical, and behavioural) independently predicted PSF and to examine how different components of anxiety are related to PSF. The anxiety components did not independently predict PSF once depressive symptoms were included in the models. Although each anxiety symptom was correlated with PSF, their effects were no longer significant after adjusting for PHQ-8 scores. This suggests that the relationship between anxiety and PSF may be largely explained by shared variance with depressive symptoms rather than unique contributions of anxiety itself. This pattern aligns with theoretical models proposing substantial overlap between anxiety and depression in stroke populations, particularly in relation to somatic and cognitive symptoms (Ayerbe et al., 2013; Barker-Collo, 2007).

Although the separate components of anxiety did not remain, significant independent predictors of PSF once depressive symptoms were included in the final regression models, this does not imply that anxiety is irrelevant to PSF. In all three models, anxiety significantly predicted PSF when entered alone, indicating that higher anxiety is associated with greater

PSF. The loss of significance after adjusting for depression likely reflects the substantial overlap between anxiety and depressive symptoms, consistent with the tripartite model, which proposes that both constructs share a core dimension of negative affect (Clark & Watson, 1991). This shared variance also means that anxiety may contribute to PSF indirectly, for example, by increasing rumination, worry, and cognitive load, which are known to worsen depressive symptoms (Nolen-Hoeksema, 2000), and depression in turn is strongly associated with PSF (Ponchel et al, 2015). Anxiety may also influence PSF through behavioural pathways, such as avoidance and reduced activity, which can reinforce low mood and energy depletion, or through physiological mechanisms such as heightened arousal and HPA-axis dysregulation, which have been implicated in other fatigue studies (Chaudhuri & Behan, 2004). Thus, while the individual components of anxiety did not explain unique variance beyond depression in the multivariable models, the present findings suggest that anxiety may still play an important role within broader emotional and behavioural processes that contribute to PSF. Our study, however, does not have the capacity to determine the directionality of this interaction; that is, we cannot say whether it is that anxiety symptoms may worsen depression, which may worsen fatigue, or that it is more that depression influences anxiety levels and therefore fatigue.

Our findings differ to some of the current literature. For instance, Mutai et al (2017), that found anxieties effects on PSF remained significant when accounting for depression. A possible explanation for why the present findings differ from this study relates to methodological and sample differences that may influence the extent to which anxiety uniquely predicts PSF. Mutai et al (2017) used the HADS-Anxiety scale, which focuses intentionally on psychological symptoms whilst minimises somatic content (Zigmond & Snaith, 1983), whereas the current study employed the BAI subscales that included physical

and cognitive symptoms. Given that physical symptoms of anxiety overlap more strongly with both depressive symptoms and physical consequences of stroke (Kohlmann et al, 2016), this may reduce its unique predictive value once depression is controlled (Clark & Watson, 1991). Differences in sample characteristics may also contribute: Mutai et al (2017) participants were recruited from inpatient rehabilitation settings and were earlier in their recovery, whereas the present sample was mainly recruited from a community setting, and PSF mechanisms may shift across the recovery trajectory (Chaudhuri & Behan, 2004). Additionally, depressive symptoms were higher in the current sample in comparison, which may have increased the proportion of fatigue variance attributable to depression, leaving less unique variance for anxiety. Mutai et al (2017) also had a substantially larger sample, providing greater power to detect small independent effects. Taken together, these factors may explain why anxiety remained a significant predictor in Mutai et al (2017) models but not in the present study.

The finding that none of the individual anxiety components predicted PSF independently of depressive symptoms offers clinically meaningful insight. Rather than supporting explanations that PSF may be caused by unmanaged worry or poor coping skills (Wu et al., 2014), the results point toward a more nuanced emotional profile in which depression emerges as a key correlate of PSF. At the same time, the significant associations observed between anxiety and PSF indicate that anxiety may still play an important role, albeit possibly indirectly through its influence on mood, behavioural avoidance, and somatic symptoms (Jacobson et al., 2014). This interpretation aligns with broader evidence that anxiety and depression share substantial variance and often interact to shape post-stroke adjustment (Mitchel et al., 2017). Clinically, this perspective may help counter stigmatising narratives that minimise the legitimacy of fatigue (Eilertsen et al, 2013), while still

underscoring the importance of assessing and supporting individuals who experience high levels of anxiety after stroke. Rather than dismissing anxiety as irrelevant, these findings suggest that addressing emotional distress in an integrated way, rather than focusing on anxiety in isolation, may be most beneficial for people living with PSF.

Consistent with previous research, stroke type also significantly predicted PSF severity, with individuals who experienced haemorrhagic stroke reporting higher fatigue than those with ischaemic stroke (English et al., 2024; Lerdal et al., 2011). This reinforces evidence that biological and neurological factors associated with stroke subtype contribute meaningfully to PSF outcomes (Staub & Bogousslavsky, 2001). Whilst the current sample size limits the ability to draw definitive clinical recommendations, these findings highlight stroke type as a potentially important factor in PSF and suggest that future research should further investigate the mechanisms and pathways linking haemorrhagic stroke to PSF.

Study limitations

A limitation of the study is that the sample size (N = 75) restricts the ability to draw firm conclusions about the comparative predictive strength of the emotional variables. Although the regression model identified depression as the only independent predictor of PSF, the study was not sufficiently powered to disentangle the shared variance between depression and the anxiety subtypes or to determine the extent to which each construct uniquely contributes to PSF. Larger samples are required to permit more sophisticated approaches, such as relative importance analysis, that can clarify whether depression is genuinely the strongest predictor, or whether some anxiety components exert smaller but meaningful independent effects. Replication with larger cohorts would therefore strengthen confidence in the pattern observed here and allow a more precise evaluation of the weighted contribution of each emotional factor to PSF.

The sample was predominantly White (90.67%), which limits the generalizability of the findings to more ethnically diverse populations. Cultural differences in the experience and reporting of fatigue and anxiety may influence outcomes, and future studies should aim to recruit more representative samples.

All measures were self-reported, which introduces the possibility of response bias. Participants may have under or overestimated their symptoms because of recall limitations or misunderstanding of questionnaire items. Incorporating clinician rated assessments or objective physiological measures could strengthen future research. Further to this study specific limitation, a wider limitation of current anxiety and PSF measures is considered. Whilst anxiety and fatigue measures are widely validated in general populations, there is still a limited set of measures validated within stroke populations. This was a key point of reflection from the initial stages of this study, with the stroke survivor involved in informing the design of the study, noting the language and structure of self-report questionnaires felt inaccessible despite proposed measures being validated in stroke populations. Further to this, the included measures do not perfectly capture the domains they are intended to represent. For example, the Behavioural Outcomes of Anxiety subscale includes items that extend beyond pure behavioural symptoms.

A statistical limitation of the current study is noted as running three separate regressions instead of a single unified model, because of the presence of high multicollinearity. Multicollinearity undermines the reliability of coefficient estimates, inflating standard errors and making it difficult to determine the true effect of each predictor. By splitting the analysis into three regressions, the model avoided multicollinearity but sacrificed the ability to assess joint effects and interactions between all anxiety variables.

Clinical implications

The study findings emphasise the importance of recognising emotional factors within post-stroke care, particularly given that depression and anxiety remain under-assessed despite their clear influence on recovery and quality of life (Ozkan et al., 2024; de Man-van Ginkel et al., 2015). Although anxiety symptoms were associated with PSF at the unadjusted level, they did not independently predict PSF once depressive symptoms were included, highlighting depression as an emotional correlate of PSF. This pattern is consistent with broader evidence showing depression to be one of the most significant predictors of PSF after stroke (Ayerbe et al., 2013; Ponchel et al., 2015). However, the presence of significant associations suggests that both depression and anxiety remain clinically relevant when developing an individualised formulation of a patient's PSF, as emotional distress may shape how PSF is experienced, interpreted, and managed even when not uniquely predictive. Clinicians should therefore ensure that both constructs are routinely screened and considered within holistic assessments, rather than focusing solely on physical or neurological contributors.

Although the current study is not powered to make treatment recommendations, the findings support the value of psychologically informed care. Stroke survivors frequently report that fatigue is misunderstood or dismissed (Stroke Association, 2017; Townend et al., 2007), and clear psychoeducation may help patients differentiate between emotional distress, bodily sensations, and genuine fatigue. This may reduce unnecessary worry, support more confident engagement in rehabilitation, and help clinicians frame PSF as a legitimate and multifactorial consequence of stroke rather than a sign of poor motivation. Overall, the findings highlight the need for comprehensive emotional assessment and sensitive interpretation of PSF within routine stroke care.

Future research

Several areas for future research present from the current study. Although anxiety subtypes were associated with fatigue at the unadjusted level, none remained significant once depressive symptoms were included, suggesting that depression may be the more central emotional correlate. Future research should therefore prioritise clarifying the mechanisms through which emotional distress contributes to PSF, with particular attention to shared variance between anxiety and depression. Before further attempts are made to develop targeted PSF interventions, greater theoretical clarity is needed regarding the pathways linking emotional symptoms and PSF. Recent feasibility work, such as the POSTIF trial, which primarily targeted behavioural and cognitive mechanisms associated with depression and inactivity(Mead et al., 2022), highlights both the promise and the challenges of delivering fatigue interventions after stroke, underscoring the need for a stronger mechanistic foundation. Given that the present findings indicate that anxiety symptoms were significantly associated with fatigue severity until depressive symptoms were included in the model, suggests that the two constructs share substantial overlapping variance in relation to PSF. Rather than operating as isolated predictors, anxiety and depression appear to form an interconnected emotional profile that is relevant to the experience of PSF after stroke. This has implications for existing interventions. For instance, the Nottingham POST-IF trial, did not improve fatigue outcomes. The intertwined nature of anxiety and depression, as shown in our study, may offer one possible explanation for this lack of efficacy, as interventions focused predominantly on depression-related mechanisms may fail to address the broader emotional processes that contribute to PSF. These findings therefore support the need for research trials that consider the combined and interacting roles of anxiety and depression in PSF, rather than treating them as independent or competing predictors.

Future research should move beyond observational designs and begin to test causal mechanisms directly. Interventional studies could help to determine whether targeting anxiety symptoms produces measurable improvements in PSF, and whether fatigue-focused interventions yield secondary benefits for anxiety. Randomised controlled trials would provide stronger evidence of causality and help identify which psychological processes represent viable treatment targets. This would offer more definitive conclusions than can be drawn from observational designs and would directly inform the development of targeted rehabilitation pathways.

Future research would benefit from the development or refinement of PSF and anxiety measures that more accurately and comprehensively assess behavioural, cognitive, and physiological domains within stroke populations. Ensuring that measures are both clinically feasible and inclusive is essential for accurately capturing the lived experiences of all stroke survivors.

The finding that haemorrhagic stroke was associated with higher fatigue severity than ischaemic stroke warrants further investigation in larger, adequately powered samples. Replication of this pattern would strengthen the case for exploring subtype-specific mechanisms and assessment needs, particularly given that stroke type is rarely considered in fatigue research despite emerging evidence of different cognitive, emotional, and recovery profiles across subtypes (Girgenti et al.,2024). Larger studies could also clarify whether stroke related biological factors interact with emotional distress to shape fatigue trajectories.

Efforts were made during recruitment to include participants from ethnically minoritised backgrounds, in recognition of the well documented underrepresentation of these groups in stroke research (Emett et al.,2025; Nanavati et al., 2024; Vindrola-Padros et al.,

2025). Recruitment was primarily achieved through stroke specific support groups, a method consistent with the approach used for other subgroups such as young stroke survivors. However, despite extensive searching, no support groups specifically serving ethnically minoritised stroke survivors were identified. This raises important questions about where and how individuals from these communities are accessing post-stroke support. While it cannot be assumed that such individuals are not receiving support, the absence of identifiable, targeted support networks suggests a potential gap in service provision. Future research aiming to recruit more diverse samples may need to explore alternative recruitment strategies and community-based outreach methods, such as visits to faith centres, or community hubs, to effectively engage underrepresented populations.

Conclusion

The study provides new insight into the emotional correlates of PSF, showing that although the three components of anxiety were associated with fatigue at the unadjusted level, none remained significant once depressive symptoms were included. These findings suggest that depression may be the more central emotional contributor to PSF, while anxiety may influence PSF indirectly or through shared variance. Both constructs therefore remain important when developing a clinical formulation of an individual's PSF experience. Larger, studies are needed to replicate these findings, clarify the relative contribution of different emotional factors, and strengthen the evidence base for how psychological symptoms should be incorporated into assessment and treatment for PSF.

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Chapter 5: Discussion and Critical Analysis

This thesis portfolio aimed to critically examine the concept of PSF and explore the contributions of anxiety to its occurrence, as well as examining its impact on QOL in stroke survivors. Two approaches were used to address this aim: (1) a systematic review and synthesis of studies that provided relational data on QOL and PSF and (2) an examination of how each separate component of anxiety contributed to PSF levels.

This chapter summarises the paper's findings, followed by a broader discussion of the significance of the portfolio in relation to relevant areas. A critical analysis follows, and the chapter is concluded with areas for future research.

Research Summary

Systematic Review

PSF impacting QOL had been commonly noted in studies (Omuwa et al., 2025, Naess et al., 2012, Visser-Meily et al.,2009), however no attempt had yet been made to synthesise these results. The present review demonstrated that research in this area is dominated by cross-sectional correlational studies, with very limited use of longitudinal methodologies capable of clarifying directionality or causal pathways. Despite this narrow methodological base, the meta-analysis revealed a consistent and moderately strong negative association between PSF and QOL, indicating that higher PSF is reliably linked to poorer QOL across studies. However, substantial heterogeneity complicated interpretation, and sensitivity analyses showed that two outlier studies inflated the pooled effect.

Qualitative synthesis of studies excluded from the meta-analysis supported the overall direction of findings and highlighted additional nuances. De Bruijn et al., (2015) reported

that the PSF and QOL association was particularly pronounced in younger stroke survivors, suggesting that PSF may have a disproportionate psychosocial impact in groups facing greater disruption to employment, family roles, and identity (Putaala., 2020). Ghosh et al., (2024) study indicated stronger associations among women, aligning with broader evidence of sex based differences in poststroke psychological outcomes (Gall et al., 2026).

Methodological inconsistencies across studies were noted as a limitation of this review. PSF was operationalised using a range of outcome measures, raising questions about the conceptual equivalence of these measures. QOL measures also varied widely, with some emphasising physical functioning and others capturing broader psychosocial domains. These inconsistencies, combined with incomplete demographic reporting, made it difficult to determine whether studies assessed comparable constructs or whether observed differences reflected measurement issues.

Despite these limitations, the review provides clear evidence that PSF is a significant correlate of reduced QOL following stroke, accounting for approximately 13% of its variance. The findings highlight the need for more rigorous longitudinal research to clarify causal pathways, identify modifiable contributors, and inform targeted interventions aimed at improving long term wellbeing for stroke survivors.

Although anxiety has frequently been cited as a correlate of PSF (Schrage et al., 2025; Galligan et al., 2015), no study had previously examined whether specific components of anxiety (cognitive, physical, and behavioural) all contribute to, or differentially predict, PSF in stroke survivors. The present study attempted to address this gap by operationalising anxiety into these three domains and assessing their associations with PSF. With no current effective treatment, understanding PSF aetiology remains crucial for moving towards more effective care.

Initial attempts to model all anxiety components simultaneously were hindered by substantial multicollinearity. This meant that the three anxiety subscales could not be entered into the same model without compromising coefficient stability, and therefore three separate hierarchical regression models were run, each controlling for stroke type and depression, which had been identified as confounders.

Across all models, anxiety symptoms significantly predicted fatigue severity at the unadjusted level. However, once depressive symptoms were included in the models, none of the anxiety components remained significant predictors. This indicates that depression is the more central emotional correlate of PSF in this sample, and that the apparent effects of anxiety may be attributable to shared variance with depressive symptoms.

A key limitation relates to the substantial multicollinearity between the anxiety and depression variables. Because the anxiety subscales were highly correlated with each other and with depressive symptoms, it was not possible to determine whether any anxiety component exerted a unique effect on PSF once shared variance was accounted for. This means that although each anxiety subtype showed a significant association with fatigue when

entered alone, the study cannot establish whether anxiety contributes independently to PSF beyond depression.

Despite these methodological constraints, the study provides evidence that anxiety across cognitive, physical, and behavioural domains is meaningfully associated with PSF, however it does not independently predict fatigue when depressive symptoms are accounted for. The findings support the view that PSF is not solely a neurological or physiological phenomenon but is shaped by broader emotional processes that may influence how PSF is experienced and managed. This underscores the importance of comprehensive psychological assessment in stroke services and highlights the need to consider both anxiety and depression when formulating an individual's fatigue profile.

Further research using larger samples and longitudinal or structural modelling approaches is needed to clarify causal pathways, disentangle shared emotional variance, and determine whether changes in specific emotional symptoms lead to measurable improvements in PSF.

Synthesis of findings

Combined, the findings of the two papers presented in this portfolio suggest that PSF has a significant impact on the well-being and QOL of stroke survivors and highlights that emotional factors, particularly depression, but also anxiety, are relevant to its aetiology. The findings together build upon the current understanding of PSF and offer possible insight into the complex biopsychosocial nature of its occurrence.

The systematic review findings offer evidence of PSF and QOL relationship being strongest among young female stroke survivors. Taken alone, this would appear to be a

possible risk factor based purely on gender and age. However, when considered alongside the empirical paper findings, where depressive symptoms emerged as the primary emotional correlate of PSF, with anxiety associated only at the unadjusted level, there is a possibility that these findings are more complex and highlight a wider biopsychosocial effect. Epidemiological research consistently shows that women are around twice as likely as men to experience both anxiety and depression across adolescence and adulthood (Kuehner, 2017; Piccinelli & Wilkinson, 2000). These sex differences are most pronounced in younger and mid-adult age ranges, where women report higher rates of internalising symptoms, including worry, low mood, and fatigue. Biological factors (such as HPA axis reactivity and hormonal fluctuations), psychological processes (rumination), and social factors (such as role strain, caregiving expectations, and exposure to interpersonal stressors) all contribute to heightened vulnerability (Kudielka & Kirschbaum, 2005; Pinquart & Sörensen, 2006). Together, these factors mean that younger female stroke survivors may enter recovery with a higher baseline vulnerability to depression and anxiety, which can interact with PSF and amplify its impact on perceived QOL.

The findings on the associations between PSF, anxiety, and QoL may also be better understood when situated within this broader, condition-specific literature. Phenotyping and mechanistic reviews suggest that PSF arises from an interplay of early inflammatory processes, dopaminergic and sensorimotor network disruption, and impaired sensory attenuation, which together increase perceived effort and vulnerability to fatigue during everyday activities (De Doncker et al., 2018; Kuppuswamy et al., 2024). By contrast, in ME or CFS and other chronic fatigue conditions, anxiety and low mood are typically conceptualised as secondary consequences of prolonged disability and uncertainty rather than primary drivers of fatigue, and QoL impairment is often profound across physical, social, and

role domains (Nacul et al., 2011). The more heterogeneous, subtype-dependent QoL impact observed in PSF therefore aligns with the view that PSF is mechanistically and phenomenologically distinct from ME/CFS and other fatigue syndromes, reinforcing the importance of stroke-specific assessment and intervention targets when addressing anxiety and QoL in this population (Chaudhuri & Behan, 2004; Kuppuswamy, 2024).

Critical Evaluation

In this section, specific strengths and weaknesses of this portfolio will be briefly considered.

Strengths

A key strength of this research lies in its ability to use both the findings from the empirical project and the systematic review together. By combining findings, we move beyond isolated observations and instead offer a cohesive account of how psychological, behavioural, and emotional processes shape PSF and QOL after stroke. This methodological breadth enhances the robustness of the conclusions and strengthens confidence in the central claim that PSF is a modifiable, clinically meaningful symptom rather than an inevitable consequence of stroke.

Another strength, more specifically, is that this portfolio creates insight for future research and thoughtfulness in completing assessments post-stroke. Highlighting the need for routine fatigue screening and psychologically informed interpretation of cognitive and functional assessments, recognising that emotional distress, particularly depression, but also anxiety, can shape how PSF is experienced and expressed. The systematic review further

reinforces the global consistency of the PSF and QOL relationship, underscoring the importance of embedding fatigue management within standard rehabilitation pathways.

Weaknesses

A limitation identified in the empirical study was the substantial overlap between the anxiety subscales, reflected in high multicollinearity. This indicated that the measures were not functioning as fully independent constructs but instead shared considerable variance. This lack of discriminant validity meant it was not possible to examine the relative predictive strength of the anxiety components within a single model, and the separate regressions used may still reflect shared underlying variance rather than unique effects. This limitation highlights the challenge of disentangling closely related psychological constructs in poststroke populations, where symptoms often co-occur. This issue was not unique to the empirical study. A limitation of the systematic review was also that many of the included studies relied on PSF and QOL measures that demonstrated conceptual overlap, inconsistent operationalisation, and variable psychometric quality. PSF was assessed using a range of tools, some unidimensional, others multidimensional, and each capturing slightly different aspects of the construct. Similarly, QOL measures varied widely in scope, with some emphasising physical functioning and others capturing broader psychosocial domains. Few studies reported conducting checks for discriminant validity or assessing whether their chosen measures adequately separated fatigue from related constructs such as depression, anxiety, or type of stroke. As a result, the associations reported in the literature may be influenced by extraneous measurement bias. The moderate heterogeneity observed in the meta-analysis may partly reflect these unexamined psychometric inconsistencies, as well as differences in measurement tools, sample characteristics, and timing poststroke.

The empirical paper also has significant limitation in relation to the ethnic representation of participants. Despite efforts during recruitment to reach underrepresented ethnic groups, most participants were White English, Welsh, Scottish, Northern Irish or British. Therefore, findings of this thesis may not be generalisable to populations outside of these ethnicities.

Implications for Research and Clinical Practice

Research

Together, these issues highlight a broader challenge within the PSF literature: the field lacks standardised, psychometrically robust measures that cleanly differentiate PSF from related psychological constructs. Recent reviews have shown that many fatigue instruments used in stroke research lack evidence of structural validity and discriminant validity, making it difficult to determine whether they measure PSF itself or overlap with emotional symptoms such as depression and anxiety (Wu & Jin, 2024). This complicates attempts to synthesise findings across studies and may obscure the true nature of the relationships between anxiety, PSF, and QOL. The empirical study's experience with multicollinearity underscores the importance of future research adopting more rigorous measurement practices, including the use of validated subscales, confirmatory factor analysis, and checks for discriminant validity, as well as further work into the development of more representative and accessible outcome measures for stroke populations. Without such methodological improvements, the risk remains that observed associations reflect measurement differences rather than genuine underlying mechanisms.

The findings combined offer potential future research focuses into specific demographic groups. For instance, further research is warranted to examine the possible link between gender, age and PSF, examining whether this finding stays consistent if low levels of depression and anxiety are observed.

A key finding of note was the difficulty reaching ethnic minority groups who remain underrepresented in stroke research (Emett et al.,2025). This was not a key focus of this project, so resources could not be given to understanding this further. To ensure future research can include these groups, research is warranted to examine if these stroke survivors are not receiving support through the areas that other non-minority groups are, or if they are receiving support and where from?

Clinical Practice

The combined findings from the empirical study and the systematic review underscore the central role of psychological factors in shaping PSF and, consequently, QOL. The empirical study demonstrates that depressive symptoms are correlates of PSF, with anxiety across behavioural, cognitive, and physical symptoms showing associations at the unadjusted level. This pattern reinforces the need for systematic screening of emotional distress throughout the stroke pathway, ensuring that both depression and anxiety are considered when assessing and formulating PSF.

Conclusion

The thesis portfolio aimed to understand the relationship between anxiety and PSF as well as PSF's influence on QOL. Evidence was found that PSF has significant implications for QOL, and that emotional factors, particularly depression, but also anxiety, are meaningfully associated with PSF. The empirical findings therefore support the need for

consistent psychological assessment and tailored rehabilitation planning that considers the broader emotional context in which PSF occurs. Future research should improve the available psychometric options, explore potential collective biopsychosocial influences, and evaluate interventions targeting the emotional mechanisms that may interact with or exacerbate PSF.

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Appendix Systematic Review

Appendix A

Journal submission guidance

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Appendix B: MMAT screening questions

1 = Is the qualitative approach appropriate to answer the research question?; 2 = Are the qualitative data collection methods adequate to address the research question?; 3 = Are the findings adequately derived from the data?; 4 = Is the interpretation of results sufficiently substantiated by the data?; 5 = Is there coherence between qualitative data sources, collection, analysis, and interpretation?; 6 = Is randomization in randomized controlled trials appropriately performed?; 7 = Are RCT groups comparable at baseline?; 8 = Are there complete outcome data in RCTs?; 9 = Are outcome assessors blinded in RCTs?; 10 = Did participants adhere to assigned interventions in RCTs?; 11 = Are participants in non-randomized studies representative of the target population?; 12 = Are measurements in non-randomized studies appropriate for outcome and intervention/exposure?; 13 = Are outcome data complete in non-randomized studies?; 14 = Have confounders been accounted for in non-randomized studies?; 15 = Is the intervention/exposure administered as intended in non-randomized studies?; 16 = Is the sampling strategy in quantitative descriptive studies relevant?; 17 = Is the sample in descriptive studies representative of the target population?; 18 = Are measurements in descriptive studies appropriate?; 19 = Is the risk of nonresponse bias low in descriptive studies?; 20 = Is the statistical analysis appropriate in descriptive studies?; 21 = Is there an adequate rationale for using a mixed-methods design?; 22 = Are the components of mixed-methods studies effectively integrated?; 23 = Are the outputs of the integration adequately interpreted?; 24 = Are divergences/inconsistencies between quantitative and qualitative results adequately addressed?; 25 = Do the components adhere to quality criteria of each method involved?

Appendix C

Effect sizes

Study	Study ID forn Meta		(Original Effect)	(SE)	Approx Partial r (converted r) or r as reported	Fisher's z (SD)
Abdulla et al., 2019	1	147	(-0.59) P (rho)	(0.07)	-0.59	-0.67 (0.08)
Pederson et al., 2023	2	144	(-0.69) P (rho)	(0.06)	-0.69	-0.83 (0.08)
Sibritt et al., 2022	3	566	(0.07) β	(0.01)	0.14	0.14 (0.04)
Dong et al., 2025	4	271	(0.26) β	(0.05)	0.30	0.31 (0.06)
Kusec et al., 2023	5	105	(-0.61) β	(0.11)	-0.48	-0.52 (0.09)
Odetunde et al., 2025	6	102	(-0.21) β	(0.09)	-0.23	-0.23 (0.10)

Kim et al., 2020	7	112	(-0.31) β	(0.12)	-0.23	-0.23 (0.01)
Kang et al., 2025	8	300	(-0.01) OR	(0.01)	-0.12	-0.12 (0.06)
Chen et al., 2014	9	218	(0.39) β	(0.11)	0.23	0.23 (0.07)
Pan et al., 2023	10	176	(0.41) β	(0.07)	-0.40	-0.42 (0.08)
Moreno et al., 2018	11	181	(0.01) β	(0.06)	−0.3317	−0.35 (0.07)
Aked et al., 2025	12	167	(-0.65) β	(0.20)	0.07	0.07 (0.08)
Omoyemi et al., 2022	13	110	(0.26) OR	(0.38)	-0.35	-0.36 (0.01)
Naes et al., 2012	14	328	(0.16)	(0.17)	-0.45	0.49 (0.05)

			HR			
Port et al., 2007	15	223	(2.77) β	(0.42)	0.41	0.43 (0.07)

Appendix Empirical Project

Appendix A

Journal Submission Guidance

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Appendix B

Fatigue Severity Scale (FSS)

FATIGUE SEVERITY SCALE

During the past week, I have found that:	Strongly Disagree			Neither Agree Nor Disagree			Strongly Agree
1. My motivation is lower when I am fatigued.	1	2	3	4	5	6	7
2. Exercise brings on my fatigue.	1	2	3	4	5	6	7
3. I am easily fatigued.	1	2	3	4	5	6	7
4. Fatigue interferes with my physical functioning.	1	2	3	4	5	6	7
5. Fatigue causes frequent problems for me.	1	2	3	4	5	6	7
6. My fatigue prevents sustained physical functioning.	1	2	3	4	5	6	7
7. Fatigue interferes with carrying out certain duties and responsibilities.	1	2	3	4	5	6	7
8. Fatigue is among my three most disabling symptoms.	1	2	3	4	5	6	7
8. Fatigue interferes with my work, family, or social life.	1	2	3	4	5	6	7

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Appendix C

Behavioural outcomes of anxiety (BOA)

	Often 3 points	Sometimes 2 points	Rarely 1 point	Never 0 points	Score (points)
I feel tense or on edge	☐	☐	☐	☐	☐
I have a strained face	☐	☐	☐	☐	☐
I have had trouble falling asleep	☐	☐	☐	☐	☐
I have been getting tired easily	☐	☐	☐	☐	☐
I feel restless as if I have to be constantly on the move (e.g., pacing)	☐	☐	☐	☐	☐
Worrying thoughts go through my mind	☐	☐	☐	☐	☐
I get sudden feelings of panic	☐	☐	☐	☐	☐
I'm scared of falling over	☐	☐	☐	☐	☐
I tend to avoid activities or social engagements	☐	☐	☐	☐	☐
I feel jumpy or easily startled	☐	☐	☐	☐	☐

Note. Adapted from the carer version in "Screening for depression and anxiety after stroke: Developing protocols for use in the community" by I. I. Kneebone, L. Neffgen, and S. Pettyfer, 2012, *Disability and Rehabilitation, 34*, p. 117. Copyright 2012 by Informa UK, Ltd. Items were rendered to the first person and instructions to consider behaviour during the past week were added. All items were rephrased into the past tense and the items on sleep, social activities and panic were simplified.

Appendix D

Beck Anxiety Inventory

Beck Anxiety Inventory (BAI)

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past month, including today, by circling the number in the corresponding space in the column next to each symptom.

	Not at all	Mildly, but it didn't bother me much	Moderately – it wasn't pleasant at times	Severely – it bothered me a lot
Numbness or tingling	0	1	2	3
Feeling hot	0	1	2	3
Wobbliness in legs	0	1	2	3
Unable to relax	0	1	2	3
Fear of worst happening	0	1	2	3
Dizzy or lightheaded	0	1	2	3
Heart pounding / racing	0	1	2	3
Unsteady	0	1	2	3
Terrified or afraid	0	1	2	3
Nervous	0	1	2	3
Feeling of choking	0	1	2	3
Hands trembling	0	1	2	3
Shaky / unsteady	0	1	2	3
Fear of losing control	0	1	2	3
Difficulty in breathing	0	1	2	3
Fear of dying	0	1	2	3
Scared	0	1	2	3
Indigestion	0	1	2	3
Faint / lightheaded	0	1	2	3
Face flushed	0	1	2	3
Hot / cold sweats	0	1	2	3

Appendix E

Patient Health Questionnaire – 8 (PHQ-8)

Patient Health Questionnaire –8 (PHQ-8)

Name:_____

Date of Birth: _____ Today's Date:_____

Over the last 2 weeks, how often have you been bothered by any of the following problems?	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	☐	☐	☐	☐
2. Feeling down, depressed, irritable or hopeless	☐	☐	☐	☐
3. Trouble falling or staying asleep, or sleeping too much	☐	☐	☐	☐
4. Feeling tired or having little energy	☐	☐	☐	☐
5. Poor appetite or overeating	☐	☐	☐	☐
6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down	☐	☐	☐	☐
7. Trouble concentrating on things, such as school work, reading or watching television	☐	☐	☐	☐
8. Moving or speaking so slowly that other people could have noticed? Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual	☐	☐	☐	☐

Appendix F

Ethics approval

University of East Anglia
Norwich Research Park
Norwich. NR4 7TJ

Email: ethicsmonitor@uea.ac.uk
Web: www.uea.ac.uk

Study title: Exploring the link between components of anxiety and Post-Stroke fatigue

Application ID: ETH2324-3002

Dear Anna,

Your application was considered on 14th February 2025 by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee).

The decision is: **approved**.

You are therefore able to start your project subject to any other necessary approvals being given.

If your study involves NHS staff and facilities, you will require Health Research Authority (HRA) governance approval before you can start this project (even though you did not require NHS-REC ethics approval). Please consult the HRA webpage about the application required, which is submitted through the IRAS system.

This approval will expire on **3rd March 2026**.

Please note that your project is granted ethics approval only for the length of time identified above. Any extension to a project must obtain ethics approval by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) before continuing.

It is a requirement of this ethics approval that you should report any adverse events which occur during your project to the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) as soon as possible. An adverse event is one which was not anticipated in the research design, and which could potentially cause risk or harm to the participants or the researcher, or which reveals potential risks in the treatment under evaluation. For research involving animals, it may be the unintended death of an animal after trapping or carrying out a procedure.

Any amendments to your submitted project in terms of design, sample, data collection, focus etc. should be notified to the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) in advance to ensure ethical compliance. If the amendments are substantial a new application may be required.

Approval by the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee) should not be taken as evidence that your study is compliant with the UK General Data Protection Regulation (UK GDPR) and the Data Protection Act 2018. If you need guidance on how to make your study UK GDPR compliant, please contact the UEA Data Protection Officer (dataprotection@uea.ac.uk).

Please can you send your report once your project is completed to the FMH S-REC (fmh.ethics@uea.ac.uk).

I would like to wish you every success with your project.

On behalf of the FMH S-REC (Faculty of Medicine and Health Sciences Research Ethics Subcommittee)

Yours sincerely,

Dr Paul Linsley