Title

Lifetime and Acute Stress Predict Functional Outcomes Following Stroke: Findings From the Longitudinal STRONG Study.

Journal

Stroke, 54(11)

Authors

Holman, Ellen

Cramer, Steven

Publication Date

2023-11-01

DOI

10.1161/STROKEAHA.123.043356

Peer reviewed

HHS Public Access

Author manuscript

Stroke. Author manuscript; available in PMC 2024 November 01.

Published in final edited form as:

Stroke. 2023 November ; 54(11): 2794–2803. doi:10.1161/STROKEAHA.123.043356.

Lifetime and acute stress predict functional outcomes following stroke: Findings from the longitudinal STRONG Study

E. Alison Holman, PhD, FNP[1,2], **Steven C. Cramer, MD**[3] **The STRONG Study Investigators**

[1]Sue & Bill Gross School of Nursing, Room 4517, Nursing & Health Sciences Hall, University of California, Irvine, CA 92697

[2]Department of Psychological Science, 4201 Social & Behavioral Sciences Gateway, University of California, Irvine, CA 92697

[3]Department of Neurology, University of California, Los Angeles; and California Rehabilitation Institute

Abstract

Background: Stroke is a sudden-onset, uncontrollable event; stroke-related stress may impede rehabilitation and recovery. Lifetime stress may sensitize patients to experiencing greater stroke-related stress and indirectly affect outcomes. We examine lifetime stress as predictor of post-stroke acute stress and examine lifetime and acute stress as predictors of 3- and 12-month functional status, and we compare acute stress and baseline National Institute of Health Stroke Scale (NIHSS) as predictors of post-stroke functional status.

Methods: Between 2016–2020 the STRONG Study enrolled adults with new radiologically-confirmed stroke 2–10 days post-stroke onset at 28 acute care U.S. hospitals. Participants were interviewed three times: acute admission (acute stress; Acute Stress Disorder Interview), 3-months (Fugl-Meyer Upper Extremity motor impairment [FM;N=431], modified Rankin Scale [mRS;3-mo N=542], Stroke Impact Scale-Activities of Daily Living [SIS-ADL;3-mo N=511], Lifetime Stress Exposure Inventory) and 12-months (mRS,N=533; SIS-ADL,N=485; Telephone-Montreal Cognitive Assessment,N=484) post-stroke. Structural equation models examined whether acute stress predicted 3- and 12-month functional outcomes, and/or mediated an association between lifetime stress and outcomes controlling for demographics and initial NIHSS. Standardized betas are reported.

Results: Sample (N=763) was 19–95 years old (M=63;SD=14.9); 448(58.7%) male. Acute stress scores ranged from 0–14 (M=3.52,95%CI=3.31,3.73). Controlling for age, gender, baseline NIHSS, and race/ethnicity, higher lifetime stress predicted higher acute stress (β=.18,p<.001), which predicted lower 3-mo FM scores (β=−.19,p<.001), lower SIS-ADL scores at 3-mo (β=−.21,p<.001) and 12-mo (β=−.21,p<.001), higher mRS scores at 3-mo (β=.23,p<.001) and 12-mo (β=.22,p<.001), and lower 12-mo tMOCA scores (β=−.20,p<.001). Acute stress predicted 12-mo tMoCA (χ^2 (1)=5.29,p=.022) more strongly, 3-mo and 12-mo mRS and SIS scores as

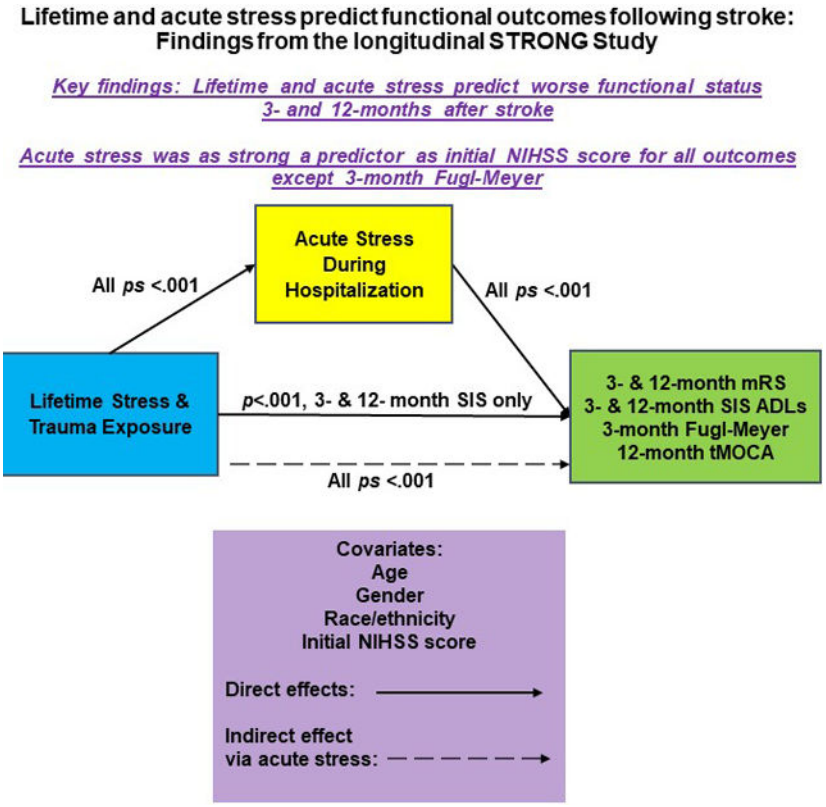
Corresponding author: E. Alison Holman, Sue & Bill Gross School of Nursing, Room 4517, Nursing & Health Sciences Hall, University of California, Irvine, CA 92697, aholman@uci.edu, aholman@hs.uci.edu, Phone: 949-370-1980.

Disclosures: Dr. Cramer serves as a consultant for Abbvie, Constant Therapeutics, BrainQ, Myomo, MicroTransponder, Neurolutions, Panaxium, NeuExcell, Elevian, Helius, Omniscient, Brainsgate, Nervgen, Battelle, and TRCare.

strongly (all *p*s>.18), but Fugl-Meyer scores (χ^2 (1)=7.01,*p*=.008) less strongly than baseline NIHSS.

Conclusions: Lifetime stress/trauma is associated with more post-stroke acute stress, which is associated with greater motor and cognitive impairment and disability 3- and 12-months post-stroke. Post-stroke interventions for acute stress may help mitigate stroke-related disability.

Graphical Abstract



Keywords

acute stress; functional impairment; stroke rehabilitation; lifetime stress

Introduction

Stroke is a leading cause of human disability and the leading neurological cause of lost disability-adjusted life years.[1] It is also a sudden-onset, unexpected, uncontrollable, highly disruptive life event. These features make having a stroke extremely stressful and potentiate its debilitating effects.[2–4] Indeed, stroke-related stress is likely to place stroke survivors at great risk for functional impairments, independent of the stroke-related damage to the brain.[5,6] Voluminous evidence links stressful life events (SLEs) with physical ailments, including stroke, highlighting the importance of studying how SLEs may affect psychosocial and functional outcomes over time.[7–9] A small, growing body of research has examined stroke-related post-traumatic stress symptoms (PTSS) or disorder (PTSD), documenting

wide variability (6%-~40%) in PTSS/PTSD among stroke patients.[10–13] Yet few have studied how lifetime exposure to stressful or traumatic events is associated with functional sequelae of stroke,[14] and we found no studies juxtaposing early post-stroke psychological sequelae with more traditional early assessments (e.g., NIHSS scores) as predictors of longer-term functional outcomes in stroke survivors.[15,16]

Rehabilitation therapy following stroke emphasizes early, preventative interventions. As such, optimal post-stroke rehabilitation may require identifying highly-stressed individuals so that appropriate care can begin immediately after the stroke. However, most studies addressing stress-related symptoms in patients who had a stroke are not methodologically rigorous as they often used small samples,[3,17–19] with either cross-sectional or retrospective designs.[4,10,18–20] or had limited short-term follow-up assessments.[21,22] Prospective, longitudinal studies are needed to identify early acute-phase predictors of chronic impairment and disability. Toward this end, this paper reports findings from a study designed to examine the unique, independent roles of lifetime stress/trauma and acute stress as early predictors of long-term functional sequelae following a stroke and to calibrate early psychological response known to complicate post-stroke recovery[10] with the NIHSS score. Calibrating early psychological responses with the NIHSS as predictors of functional outcomes could inform development of more comprehensive early predictive tools for guiding post-stroke interventions.

Methods

Recruitment

The multi-center longitudinal STRONG Study recruited new stroke patients at 28 hospitals across the U.S. (see Supplementary Materials) to examine genetic markers, lifetime stress/trauma, and acute stress experiences that may affect stroke recovery. Genetic analyses will be reported elsewhere. Entry criteria included age ≥18 years, ischemic stroke or intracerebral hemorrhage confirmed radiologically, onset 2–10 days before initial interview, able to understand/respond in English, and able to respond to study assessments. Excluded patients had expected survival <1 year, experienced a stroke <90 days before index stroke, moderate-severe disability *before* index stroke (pre-stroke modified Rankin Scale [mRS] score>2), and significant psychiatric/neurological diagnosis before index stroke. To ensure consistency across sites, interviewers at each site underwent standardized training before enrolling/interviewing patients. The standardized protocol did not monitor who conducted which interviews over time, so stressful life events (SLE), acute stress, and outcome assessments may have been assessed by the same person over time. However, acute stress—a baseline assessment—was not consistently assessed by the same person who conducted 3-mo and 12-mo interviews. Patient attrition was due to mortality, inability to return to enrollment hospital, and COVID-19 restrictions (see Supplemental Figure 1). This study was approved by the local IRB at each participating institution. All participants provided informed consent with no surrogate consent permitted. The data that support the findings of this study are available from the corresponding author upon reasonable request.

Measures

Visit 1 (acute) was a live exam 2–10 days after stroke onset that included the well-validated and reliable 14-item Acute Stress Disorder Interview (ASDI),[23] a dichotomous assessment of the presence/absence of early stroke-related post-traumatic stress symptoms (e.g., intrusions, hypervigilance, avoidance). This interview also included demographics, medical history, and extraction of initial NIH Stroke Scale[24,25] (NIHSS) score from the medical record. Positive ASDI symptoms were summed to create a total score ranging from 0 to 14, with higher scores indicating greater acute stress.

Visit 2 (3-mo) was a live exam approximately 3 months post-stroke. Measures included Fugl-Meyer Upper Extremity (FM) assessment[26–28], a 66-point scale where higher scores indicate less upper extremity motor impairment; the mRS[29], a 7-level scale where higher scores indicate greater global disability, the Stroke Impact Scale 3.0 Activities of Daily Living subsection (SIS-ADL)[30], a 40-point scale where higher scores indicate less functional disability; and a modified version of the Lifetime Stress Exposure Inventory[31] of respondents' exposure to 31 major stressful/traumatic life events (heretofore SLEs; e.g., domestic violence, bereavement, childhood maltreatment, discrimination, living in dangerous neighborhood, sexual assault, disasters) in childhood, and in adulthood before ***or*** after the index stroke with higher scores indicating greater SLE exposure. The SLEs measure was developed by a member of the team in a study of 1456 chronically ill primary care patients,[32] and has reliably produced comparable rates of population-level SLEs as standardized measures.[33] SLEs occurring during childhood and adulthood before the index stroke were summed to create an index of *pre-stroke* lifetime SLE exposure. Visit 3 data were collected 6-months post-stroke but are not relevant to the current paper.

Visit 4 (12-mo) occurred via telephone, approximately 12 months post-stroke, and included three measures of mental and physical functioning: the mRS,[29] the SIS-ADL,[30] and the Telephone Montreal Cognitive Assessment (tMOCA).[34,35]

Data Analysis

Analyses were conducted using the Stata version 17[36] structural equation modeling program. Mediation path models were constructed predicting each outcome: mRS scores (3-mo and 12-mo), SIS-ADL scores (3-mo and 12-mo), FM scores (3-mo) and tMOCA scores (12-mo). Lifetime SLE was the main predictor with total acute stress score (ASDI) tested as an explanatory mediator linking lifetime SLEs with post-stroke functional outcomes. Each model included age, gender, race-ethnicity, and initial NIHSS total score as covariates. All models were estimated using Full Information Maximum Likelihood (FIML) to retain participants by using all available data to estimate a likelihood function for each participant. This approach analyzes the full sample and minimizes bias on parameter estimates due to missing data.[37] Indirect effects of lifetime SLEs (Lifetime Stress Exposure Inventory score) on each outcome through acute stress (ASDI score) were then tested. For each model, a Wald χ^2 test compared the strengths of acute stress versus baseline NIHSS scores as predictors of each outcome. Continuous variables were standardized before analyses to provide standardized betas as effect sizes. Analyses were repeated on cases with complete data and the findings were essentially identical across all outcomes. FIML

data are presented. Analyses were rerun controlling for hypertension and diabetes, and again to examine the independent associations of childhood, adulthood, and post-stroke SLE. In all analyses findings and effect sizes for lifetime SLE, acute stress, and NIHSS were essentially identical to those presented herein, but model fit worsened, so analyses are presented with pre-stroke lifetime SLE and acute stress as key predictors of 3- and 12-month outcomes. This manuscript follows the STROBE[38] reporting guidelines.

Results

Sample demographics

We enrolled 763 patients between 10/2016 and 2/2020. Complete data were available in 510 patients at 3-mo; and 482 at 12-mo. Table 1 displays sample demographics. Participants were enrolled a median of 4 [IQR: 3–6] days post-stroke. This was the first stroke in 80.9%; 32.4% received acute reperfusion therapy (IV tPA or neurointerventional procedure or both). Hypertension was present in 71.6%; diabetes mellitus, in 30.0%.

Lifetime and acute stress

SLEs ranged from 0–31 events (M=7.25, SD=6.10; 95% CI=6.75,7.76; Md=6). Acute stress symptoms ranged from 0–14, where higher scores indicate higher acute stress (M=3.52, SD=2.94; 95% CI[3.31,3.73]; Md=3), with 6.61% (N=50) reporting nine or more symptoms consistent with DSM-V acute stress disorder diagnosis. In all models reported, lifetime SLEs directly predicted patient reports of stroke-related acute stress (ASDI score) 2–10 days post-stroke: As SLEs increased, post-stroke acute stress symptoms also increased significantly (β=.19, 95% CI[.11,.26], p<.001).

Visit 2 outcomes

Modified Rankin Scores.—Acute stress predicted 3-mo functional outcomes; ASDI scores were associated with poorer 3-mo mRS scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. The mediation path model predicting 3-mo mRS scores from lifetime SLE and acute stress had good model fit: χ^2(4, N=763)=5.23, p=.264, with acute stress R^2=.07, mRS score R^2=.15, and model R^2=.18.

As acute stress increased, mRS scores also increased (β=.23,95% CI[.15,.31],p<.001). There was a small but significant positive indirect effect of lifetime SLEs on 3-mo mRS scores through acute stress--more lifetime SLEs were associated with poorer functioning (β=.04,95% CI[.02,.07],p<.001; Figure 1a). Finally higher baseline NIHSS scores directly (β=.20,95% CI[.12,.28],p<.001) and indirectly (β=.03,95% CI[.01,.05],p=.006) predicted higher 3-mo mRS scores. When compared to the baseline NIHSS scores, acute stress was as strong a predictor of 3-mo mRS scores after controlling for SLEs and covariates, Wald χ^2 test χ^2(1, N=763)=0.25, p=.615.

Stroke Impact Scale-ADL.—Acute stress predicted 3-mo disability outcomes, as higher acute ASDI scores were associated with poorer 3-mo SIS-ADL scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline

NIHSS scores. The mediation path model predicting 3-mo SIS-ADL scores from lifetime SLEs and acute stress had good model fit: $\chi^2(4, N=763)=5.28$, $p=.26$, with acute stress $R^2=.07$,SIS-ADL scores $R^2=.22$, and model $R^2=.23$. Both higher acute stress (β=−.21,95% CI[−.29,−.14],$p<.001$) and lifetime SLEs (β=−.15,95% CI[−.23,−.07],$p<.001$) directly predicted lower 3-mo SIS-ADL scores. Lifetime SLEs also indirectly predicted lower SIS-ADL scores through acute stress (β=−.04,95% CI[−.06,−.02],$p<.001$;Figure 1b). Finally, higher baseline NIHSS scores directly (β=−.22, 95% CI[−.30,−.14], $p<.001$) and indirectly (β=−.03,95% CI[−.04,−.01],$p=.006$) predicted lower 3-mo SIS-ADL scores. When compared to the baseline NIHSS scores, acute stress was as strong a predictor of 3-mo disability after controlling for SLEs and covariates, Wald χ^2 test $\chi^2(1, N=763)=0.02$, $p=.88$.

Fugl-Meyer Scores.—Acute stress predicted 3-mo motor impairment outcomes, as higher acute ASDI scores were associated with poorer 3-mo FM scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. The mediation path model predicting 3-mo FM total scores from lifetime SLE and acute stress scores had good model fit: $\chi^2(4, N=763)=5.24$, $p=.26$, with acute stress $R^2=.07$, FM scores $R^2=.21$, and model $R^2=.23$. Acute stress directly predicted 3-mo FM scores—as acute stress increased, FM scores decreased (β=−.19, 95% CI[−.28,−.10], $p<.001$). There was a small but significant indirect effect of lifetime SLEs on 3-mo FM scores through acute stress (β=−.04, 95% CI[−.06,−.01], $p=.001$; Figure 1c). Higher baseline NIHSS scores also directly (β=−.37, 95% CI[−.46,−.28], $p<.001$) and indirectly (β=−.02, 95% CI[−.04,−.01], $p=.010$) predicted lower 3-mo FM scores. When compared to baseline NIHSS scores, acute stress was a weaker a predictor of 3-mo impairment after controlling for SLEs and covariates, Wald χ^2 test χ^2 (1, N=763)=6.61, $p=.01$.

Visit 4 outcomes

Acute stress also predicted 12-mo functional and cognitive impairment outcomes, as acute ASDI scores were associated with 12-mo mRS, SIS-ADL, and tMOCA scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. When compared to baseline NIHSS scores, acute stress was as strong a predictor of 3-mo mRS and SIS-ADL scores, but a stronger predictor of the 12-mo tMOCA scores.

Modified Rankin Scores.—Higher ASDI scores were associated with poorer 12-mo mRS scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. The mediation path model predicting 12-mo mRS scores from lifetime SLEs and acute stress had good model fit: $\chi^2(10, N=763)=13.43$, $p=.201$, with acute stress $R^2=.07$, mRS score $R^2=.13$, and model $R^2=.16$. As acute stress increased, mRS scores also increased (β=.22,95% CI[.14,.31],$p<.001$). Lifetime SLEs did not directly predict 12-mo mRS scores (β=−.04,95% CI[−.13,.04],$p = .329$), however, a small but significant indirect effect emerged linking lifetime SLEs with 12-mo mRS scores through acute stress; more lifetime SLEs were associated with poorer functioning (β=.04,95% CI[.02,.06],$p<.001$; Figure 2a). Finally, higher baseline NIHSS scores directly (β=.18,95% CI[.10,.26],$p<.001$) and indirectly (β=.03, 95% CI[.01,.05], $p=.007$) predicted higher 12-mo mRS scores. When compared to baseline NIHSS scores, acute stress was as strong a predictor of 12-mo mRS scores after controlling for SLEs and covariates, Wald χ^2 (1,N=763)=0.60, $p=.439$.

Stroke Impact Scale-ADL.—Higher ASDI scores predicted poorer 12-mo SIS-ADL scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. The mediation path model predicting 12-mo SIS-ADL from lifetime SLEs and acute stress had good model fit: $\chi^2(10, N=763)=13.43$, $p=.20$, with acute stress $R^2=.07$, SIS-ADL scores $R^2=.15$, and model $R^2=.16$. Higher acute stress (β=−.21, 95% CI[−.29,−.13], $p<.001$) and lifetime SLEs (β=−.19, 95% CI[−.28,−.10], $p<.001$) directly predicted lower 12-mo SIS-ADL scores. Lifetime SLEs also indirectly predicted lower SIS-ADL scores through acute stress (β=−.04, 95% CI[−.06,−.02], $p=.001$; Figure 2b). Finally, higher baseline NIHSS scores directly (β=−.13, 95% CI[−.21,−.04], $p=.003$) and indirectly (β=−.02, 95% CI[−.04,−.01], $p=.008$) predicted lower 12-mo SIS-ADL scores. When compared to baseline NIHSS scores, acute stress was as strong a predictor of 12-mo disability after controlling for lifetime trauma and covariates, Wald χ^2 (1, $N=763$)=1.78, $p=.182$.

tMOCA scores.—ASDI scores predicted poorer 12-mo tMOCA scores after controlling for age, gender, self-reported race/ethnicity, lifetime SLEs, and baseline NIHSS scores. The mediation path model predicting 12-mo tMoCA scores from lifetime SLEs and acute stress scores had good model fit: χ^2 (10, $N=763$)=13.67, $p=.189$, with acute stress $R^2=.07$, tMoCA scores $R^2=.13$, and model $R^2=.18$. Higher acute stress directly predicted lower 12-mo tMOCA scores (β=−.20, 95% CI[−.28,−.11], $p<.001$). Lifetime SLEs were not directly associated with 12-mo tMOCA (β=.06, 95% CI[−.03,.15], $p=.215$), but indirectly predicted 12-mo tMOCA scores through their association with acute stress (β=−.04, 95% CI[−.06,−.01], $p=.001$; Figure 2c). Similarly, baseline NIHSS scores were not directly associated (β=−.05, 95% CI[−.14,.04], $p=.263$) but were indirectly associated with lower 12-mo tMOCA scores through acute stress (β=−.02, 95% CI[−.04,.01], $p=.010$). When compared to baseline NIHSS scores, acute stress was a stronger predictor of 3-mo impairment after controlling for lifetime trauma and covariates, Wald $\chi^2(1, N=763)=5.29$, $p=.022$.

Discussion

Identifying early predictors of long-term disability following stroke is essential for developing early interventions that enable a personalized approach to optimizing long-term outcomes. The NIHSS score is commonly used as the best deficit rating scale of long-term outcomes amongst data available during the acute stroke admission.[15,16] In this prospective longitudinal study of 763 patients with stroke, greater lifetime exposure to stress/trauma was associated with higher acute psychological stress symptoms reported 2–10 days after stroke onset, which in turn were a strong predictor of 3-month disability and motor impairment as well as 12-month disability and cognitive impairment. Effect sizes show acute stress symptoms are as strong a predictor of mRS and SIS-ADL scores at 3- and 12-months, and a stronger predictor of cognitive impairment at 12-months post-stroke, when compared to initial NIHSS scores. The NIHSS was the strongest predictor of 3-month motor impairment, as expected given that the NIHSS is strongly driven by motor deficits. These novel findings suggest that above and beyond current rehabilitation to address early physical impairments (e.g., NIHSS score), early psychological sequelae of stroke may serve as an important target

for *early* intervention to mitigate the negative downstream disability often seen in stroke survivors. Given the link between lifetime SLEs and acute stress, early interventions that connect stroke survivors with a support system to bolster coping with both the psychological sequelae of the stroke and any lifetime/chronic stress affecting patients' rehabilitation would be best.

Acute stress is independently associated with short- and long-term disability

An early psychological sequela of stroke was consistently one of the two strongest predictors of 3-month outcomes alongside NIHSS score and age. At 12-months post-stroke, the same pattern emerged: acute stress was either the strongest predictor (tMoCA) or was one of two equally-strong top predictors of post-stroke disability alongside baseline NIHSS (for mRS and SIS-ADL). Substantial evidence suggests that post-stroke rehabilitation therapy treatment gains are: (a) highly variable in stroke survivors,[39] and (b) often smaller than the effects of key covariates (i.e., age, severity) included in the model.[40] This suggests that some important predictors have yet to be included in these predictive models. To address this problem, several prediction tools have been developed in recent years to improve clinicians' ability to accurately identify treatment outcomes.[41–44] Findings reported here suggest that incorporating assessments of acute stress in these tools may further strengthen their prognostic utility for predicting functional and perhaps motor outcomes. New strategies are needed to optimize prescription of rehabilitation therapy and better predict the wide variability in response to post-stroke rehabilitation therapy; our findings suggest these strategies need to address the acute stress experienced by stroke patients as a possible mechanism through which lifetime SLE exposure may exacerbate long-term disability. Doing so may improve patients' post-stroke long-term functioning.

These findings extend decades of research documenting stress-related mental and physical health outcomes from basic[7] and epidemiological research[9] to shed light on psychological processes that may impact stroke recovery. While post-stroke depression is an extensively-studied known contributor to poor recovery outcomes among stroke survivors[45], the role of PTSD–a common comorbid disorder–has heretofore received far less attention among stroke survivors.[10] This study draws from the rich prior literature on stress-related disorders to identify early PTSD symptoms as a risk factor that can be measured during initial hospitalization, opening the possibility of using an early intervention to reduce post-stroke disability. As is the case with disaster response, receiving early psychological first aid–perhaps implemented by a trained nurse–may prove beneficial for acute stroke patients.[46] Encouraging use of Chronic Disease Self-Management Programs[47–50] may also benefit patients by improving their quality of life and self-efficacy.[51] Future research is needed to identify the most appropriate early interventions to mitigate stress-related symptoms experienced acutely during hospitalization and examine their potential impact on long term disability among stroke survivors.

Prior lifetime SLE as a predictor of stroke outcomes

A growing body of research identifies lifetime SLEs as a risk factor for subsequent stroke,[12,52–54] but comprehensive assessments of lifetime SLE exposure is rare. We document that patient reports of pre-stroke lifetime SLE directly predict stroke-related acute

stress as measured within days of stroke onset, with the latter being a strong predictor of wide-ranging 3-mo and 12-mo functional outcomes. Indirect effects identified through mediation path modeling further show that acute stress is one of the mechanisms linking lifetime SLE with these outcomes. These findings support calls for screening patients about their lifetime and ongoing SLE exposure[53,55] so that appropriate follow-up care can be provided to offset any negative impact of these exposures.

Importantly, lifetime stress and trauma exposure is a well-documented social determinant of health. Assessing and addressing the structural racism,[56] other forms of discrimination,[57] experiences of financial strain, food/housing insecurity, neighborhood safety – all components of our SLE assessment – should be implemented in research and clinical settings, with attention paid to informing policymakers about the significant cardiovascular benefits of tackling these issues. Moreover, greater understanding of the mechanisms by which social determinants adversely affect the health of people from marginalized communities could also inform more effective stroke prevention efforts. Recent multi-disciplinary teams have started to identify specific biological processes associated with discrimination that may contribute to health disparities.[58] These findings also highlight the critical issue of providing person-centered care for each patient.[59] While recognizing each patient as a unique individual with unique life experiences may improve post-stroke care and outcomes, few patients are screened for lifetime stress/trauma exposure.[60] Implementing such screening would provide evidence for the value of consistent, in-depth support from a social worker and/or psychologist and in so doing, could support providers' care planning by documenting the need for more comprehensive, multi-disciplinary team approaches to planning patient care.

Finally, prior research documents biological pathways that may underlie these findings. Psychological stress is known to precipitate immune responses that promote inflammatory processes (e.g., atherosclerosis),[61–63] and increase risk for both stroke and post-stroke depression, dementia, or other neuropsychiatric disease.[64–66] Animal studies further document that the link between stress exposure and stroke-related outcomes may be modulated by expression of genes related to growth and transcription factors involved in brain plasticity.[14] Future research is needed to identify genetic and other stress-related physiologic pathways that may be targeted for early intervention to prevent poor stroke-related outcomes. One physiologic system for which there is a solid body of evidence linking stress with cerebrovascular outcomes is the renin-angiotensin-aldosterone system, making it a plausible target for such research.[66]

Limitations

This large, nationwide, prospective longitudinal study of stroke survivors provides a robust examination and comparison of acute stress with NIHSS score as key predictors of long-term functional outcomes following stroke. The sample was diverse with racial/ethnic breakdown similar to the U.S population. Nonetheless, a sizable minority (~1/3) of patients enrolled as the COVID-19 pandemic began, forcing us to conduct 3-mo assessments by phone, foregoing the 3-mo Fugl-Meyer assessment in some patients. Baseline FM assessments, a potentially valuable covariate, were not included in study

protocol. The sample excluded (a) non-English-speaking patients, many of whom may be recent immigrants who experienced the stress of immigration; (b) patients unable to provide consent, some of whom may have had aphasia; and (c) the most severe stroke patients, who would not have been able to complete study protocols. Hence, selection bias restricts generalization of our findings, and probably underestimates the amount of stress reported, which could limit the range of our lifetime and acute stress assessments and the strength of our findings. Retrospective recall of SLE may also be biased by current life circumstances, which could have affected our findings.[67] Participants' pre-stroke cognitive status and living in an area with regional deprivation were not assessed; both may help explain the findings reported. Approximately one-third of the sample was lost to follow-up due to mortality, inability to return to enrollment hospital, and COVID-related factors, however analyses used FIML to retain the full analytic sample and prevent bias from missing data.[37]

Conclusion

Lifetime SLE exposure and stroke-related acute stress symptoms were associated with greater disability and impairment at 3- and 12-months post-stroke. When compared to the baseline NIHSS score, acute stress was a weaker predictor of 3-month motor impairment, but as strong a predictor of 3- and 12-month mRS and SIS-ADL, and a stronger predictor of 12-month cognitive impairment. Assessments of lifetime stress and early post-stroke acute stress symptoms could help identify patients at risk for subsequent disability and be an appropriate target for early intervention to mitigate this risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments:

We would like to thank Ms. Breann McGee and Ms. Ashley Jubb for their many contributions to the successful conduct of the STRONG Study, and Dr. Dan Relihan for his assistance with statistical analyses.

Source of Funding:

NIH R01 NR015591

APPENDIX

Appendix:

List of the STRONG Study Investigators, in alphabetical order by study site

Author Name	Study Site
Shreyansh Shah, MD	Duke University
Christoph J. Griessenauer, MD	Geisinger Health; Geisinger Commonwealth School of Medicine
Nirav Patel, MD	Los Alamitos Medical Center
David J. Lin, MD	Massachusetts General Hospital
Joey Gee, DO	Providence Mission Hospital Mission Viejo

Author Name	Study Site
Johnson Moon, MD	Providence St. Jude Medical Center
Julie Schwertfeger, PT, PhD, DPT, MBA	Rosalind Franklin University of Medicine and Science
Arun Jayaraman, PT, PhD	Shirley Ryan Ability Lab
Robert Lee, MD	St. David's Medical Center
Maarten Lansberg, MD, PhD	Stanford University
Jeremy Payne, MD, PhD	University of Arizona
Carolynn Patten, PhD, PT	University of California, Davis
Steven C. Cramer, MD, MMSc; E. Alison Holman, PhD, FNP	University of California, Irvine
Kunal Agrawal, MD	University of California, San Diego
Stacey DeJong, PT, PhD	University of Iowa
John Cole, MD, MS	University of Maryland
Brian Silver, MD	University of Massachusetts Chan
Brett Cucchiara, MD	University of Pennsylvania
Ania Busza, MD, PhD	University of Rochester
Sook-Lei Liew, PhD, OTR/L	University of Southern California
Susan Alderman, PhD, RN	University of Texas, Houston
Heather Hayes, PT, PhD, DPT; Jennifer J. Majersik, MD, MS	University of Utah
Brad Worrall, MD	University of Virginia
David Tirschwell, MD, MSc	University of Washington
Cheryl Bushnell, MD, MHS; Nada El Husseini, MD, MHS	Wake Forest School of Medicine
Jin-Moo Lee, MD, PhD	Washington University in St. Louis
Guido J. Falcone, MD, ScD, MPH	Yale University

Non-standard Abbreviations and Acronyms

ASDI	Acute Stress Disorder Interview
FM	Fugl-Meyer Upper Arm Assessment
NIHSS	National Institutes of Health Stroke Scale
PTSD	Post-traumatic stress disorder
SIS-ADL	Stroke Impact Scale 3.0 Activities of Daily Living
SLE	Stressful Life Events
STRONG	Stroke, Stress, Rehabilitation, and Genetics
tMOCA	Telephone Montreal Cognitive Assessment

References

1. Tsao CW, Aday AW, Almarzooq ZI, Alonso A, Beaton AZ, Bittencourt MS, Boehme AK, Buxton AE, Carson AP, Commodore-Mensah Y, et al. Heart disease and stroke statistics—2022 update: a report from the American Heart Association. Circulation. 2022;145(8):e153–639. [PubMed: 35078371]
2. Ben Assayag E, Tene O, Korczyn AD, Solomon Z, Bornstein NM, Shenhar-Tsarfaty S, Seyman E, Niry D, Molad J, Hallevi H. Posttraumatic stress symptoms after stroke: the effects of anatomy and coping style. Stroke. 2022;53(6):1924–1933. [PubMed: 35264011]
3. Jiang C. Posttraumatic stress disorder after a first-ever intracerebral hemorrhage in the Chinese population: A pilot study. Appl Neuropsychol Adult. 2020; 27:1–8. DOI: 10.1080/23279095.2018.1451334. [PubMed: 29617165]
4. Kronish IM, Edmondson D, Goldfinger JZ, Fei K,& Horowitz CR. Posttraumatic stress disorder and adherence to medications in survivors of strokes and transient ischemic attacks. Stroke. 2012;43(8):2192–2197. [PubMed: 22618380]
5. Karam EG, Friedman MJ, Hill ED, Kessler RC, McLaughlin KA, Petukhova M, Sampson L, Shahly V, Angermeyer MC, Bromet EJ, et al. Cumulative traumas and risk thresholds: 12-month PTSD in the World Mental Health (WMH) Surveys. Depression and Anxiety. 2014;31:130–142. [PubMed: 23983056]
6. Rodriguez P, Holowka DW and Marx BP. Assessment of posttraumatic stress disorder-related functional impairment: a review. J Rehabil Res Dev. 2012;49:649–665. DOI: 10.1682/jrrd.2011.09.0162. [PubMed: 23015577]
7. Lupien SJ, McEwen BS, Gunnar MR, Heim, C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci. 2009;10:434–445. [PubMed: 19401723]
8. McEwen BS. Protective and damaging effects of stress mediators. N Engl J Med. 1998;338:171–179. [PubMed: 9428819]
9. Suglia SF, Koenen KC, Boynton-Jarrett R, Chan PS, Clark CJ, Danese A, Faith MS, Goldstein BI, Hayman LL, Isasi CR, et al. Childhood and adolescent adversity and cardiometabolic outcomes: a scientific statement from the American Heart Association. Circulation. 2018;137(5):e15–28. [PubMed: 29254928]
10. Edmondson D, Richardson S, Fausett JK, Falzon L, Howard VJ, Kronish IM. Prevalence of PTSD in survivors of stroke and transient ischemic attack: a meta-analytic review. PloS One. 2013;8(6):e66435.
11. Garton AL, Gupta VP, Pucci JU, Couch CK, Connolly ES Jr. Incidence and predictors of post-traumatic stress symptoms in a cohort of patients with intracerebral hemorrhage. Clinical Neurol Neurosurg. 2020;190:105657.
12. Edmondson D, von Känel R. Post-traumatic stress disorder and cardiovascular disease. Lancet Psychiatry. 2017;4:320–329. DOI: 10.1016/S2215-0366(16)30377-7 [PubMed: 28109646]
13. Zhang S, Xu M, Liu ZJ, Feng J, Ma Y. Neuropsychiatric issues after stroke: Clinical significance and therapeutic implications. World J Psychiatry. 2020;10(6):125–138. [PubMed: 32742946]
14. Zucchi FC, Yao Y, Ilnytskyy Y, Robbins JC, Soltanpour N, Kovalchuk I, Kovalchuk O, Metz GA. Lifetime stress cumulatively programs brain transcriptome and impedes stroke recovery: benefit of sensory stimulation. PloS One. 2014;9(3):e102489.
15. Mistry EA, Yeatts S, de Havenon A, Mehta T, Arora N, De Los Rios La Rosa F, Starosciak AK, Siegler JE III, Mistry AM, Yaghi, et al. Predicting 90-day outcome after thrombectomy: baseline-adjusted 24-hour NIHSS is more powerful than NIHSS score change. Stroke. 2021;52(8):2547–2553. [PubMed: 34000830]
16. Kurmann CC, Beyeler M, Grunder L, Lang MF, Piechowiak EI, Meinel TR, Jung S, Hoffmann A, Seiffge DJ, Heldner MR, et al. Association of the 24-hour National Institutes of Health Stroke Scale after mechanical thrombectomy with early and long-term survival. Stroke: Vascular and Interventional Neurology. 2022;2(4):1–10.
17. Stein LA, Goldmann E, Zamzam A, Luciano JM, Messé SR, Cucchiara BL, Kasner SE, Mullen MT. Association between anxiety, depression, and post-traumatic stress disorder and outcomes after ischemic stroke. Frontiers in Neurology. 2018;890:1–9.

18. Bruggimann L, Annoni JM, Staub F, Von Steinbüchel N, Van der Linden M, Bogousslavsky J. Chronic posttraumatic stress symptoms after nonsevere stroke. Neurology. 2006;66(4):513–516. [PubMed: 16505303]
19. Speck V, Noble A, Kollmar R, Schenk T. Diagnosis of spontaneous cervical artery dissection may be associated with increased prevalence of posttraumatic stress disorder. J Stroke Cerebrovasc Dis. 2014;23:335–342. DOI:10.1016/j.jstrokecerebrovasdis.2013.03.033. [PubMed: 23849487]
20. Kiphuth IC, Utz KS, Noble AJ, Köhrmann M, Schenk T. Increased prevalence of posttraumatic stress disorder in patients after transient ischemic attack. Stroke. 2014;45:3360–3366. DOI: 10.1161/STROKEAHA.113.004459. [PubMed: 25278556]
21. Letamendia C, LeBlanc NJ, Pariente J, Simon NM, Thomas CL, Chabrol H, Chollet F, Raposo N, Schmitt L, Birmes P, et al. Peritraumatic distress predicts acute posttraumatic stress disorder symptoms after a first stroke. General Hospital Psychiatry. 2012;5(34):e11–3.
22. Wang X, Chung MC, Hyland ME, Bahkeit M. Posttraumatic stress disorder and psychiatric co-morbidity following stroke: the role of alexithymia. Psychiatry Res. 2011;188:51–57. [PubMed: 21036403]
23. Bryant RA. (2016). Acute stress disorder: What it is and how to treat it. Guilford Publications.
24. Kwah LK, Diong J. National Institutes of Health Stroke Scale (NIHSS). J Physiother. 2014;60:61. [PubMed: 24856948]
25. Lyden P, Raman R, Liu L, Emr M, Warren M, Marler J. National Institutes of Health Stroke Scale certification is reliable across multiple venues. Stroke.2009;40:2507–2511. [PubMed: 19520998]
26. Duncan PW, Propst M, Nelson SG. Reliability of the Fugl-Meyer assessment of sensorimotor recovery following cerebrovascular accident. Phys Ther. 1983;63:1606–1610. [PubMed: 6622535]
27. Sullivan KJ, Tilson JK, Cen SY, Rose DK, Hershberg J, Correa A, Gallichio J, McLeod M, Moore C, Wu SS, et al. Fugl-Meyer assessment of sensorimotor function after stroke: standardized training procedure for clinical practice and clinical trials. Stroke. 2011;42(2):427–432. [PubMed: 21164120]
28. See J, Dodakian L, Chou C, Chan V, McKenzie A, Reinkensmeyer DJ, Cramer SC. A standardized approach to the Fugl-Meyer assessment and its implications for clinical trials. Neurorehabil Neural Repair. 2013;27:732–741. [PubMed: 23774125]
29. Banks JL, Marotta CA. Outcomes validity and reliability of the Modified Rankin Scale: Implications for stroke clinical trials: A literature review and synthesis. Stroke. 2007;38:1091–1096. [PubMed: 17272767]
30. Richardson M, Campbell N, Allen L, Meyer M, Teasell R. The Stroke Impact Scale: Performance as a quality of life measure in a community-based stroke rehabilitation setting. Disabil Rehabil. 2016;38:1425–1430. [PubMed: 26517368]
31. Seery MD, Holman EA, Silver RC. Whatever does not kill us: cumulative lifetime adversity, vulnerability, and resilience. J Pers and Soc Psychol. 2010;99(6):1025–1041. [PubMed: 20939649]
32. Holman EA, Silver RC, & Waitzkin H. Traumatic life events in primary care patients: A study in an ethnically-diverse sample. Arch Fam Med. 2000; 9:802–810. [PubMed: 11031385]
33. Breslau N. The epidemiology of trauma, PTSD, and other posttrauma disorders. Trauma, Viol, & Abuse 2009;10(3):198–210.
34. Pendlebury ST, Welch SJ, Cuthbertson FC, Mariz J, Mehta Z, Rothwell PM. Telephone assessment of cognition after transient ischemic attack and stroke: modified telephone interview of cognitive status and telephone Montreal Cognitive Assessment versus face-to-face Montreal Cognitive Assessment and neuropsychological battery. Stroke. 2013;44(1):227–229. [PubMed: 23138443]
35. Wong A, Nyenhuis D, Black SE, Law LS, Lo ES, Kwan PW, Au L, Chan AY, Wong LK, Nasreddine Z, et al. Montreal Cognitive Assessment 5-minute protocol is a brief, valid, reliable, and feasible cognitive screen for telephone administration. Stroke. 2015;46(4):1059–1064. [PubMed: 25700290]
36. StataCorp. 2021. Stata Statistical Software: Release 17. College Station, TX: StataCorp LLC.
37. Enders CK, & Bandalos DL The relative performance of full information maximum likelihood estimation for missing data in structural equation models. Structural Equation Modeling, 2001;8(3):430–457.

38. STROBE Statement. Strengthening the Reporting of Observational Studies in Epidemiology. https://www.strobe-statement.org/. Accessed March 10, 2023.
39. Winstein CJ, Stein J, Arena R, Bates B, Cherney LR, Cramer SC, Deruyter F, Eng JJ, Fisher B, Harvey RL, et al. Guidelines for adult stroke rehabilitation and recovery: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2016;47(6):e98–169. [PubMed: 27145936]
40. Bath PM, Lees KR, Schellinger PD, Altman H, Bland M, Hogg C, Howard G, Saver JL. Statistical analysis of the primary outcome in acute stroke trials. Stroke. 2012;43(4):1171–1178. [PubMed: 22426314]
41. Stinear CM, Smith MC, Byblow WD. Prediction tools for stroke rehabilitation. Stroke. 2019;50(11):3314–3322. [PubMed: 31610763]
42. de Ridder IR, Dijkland SA, Scheele M, den Hertog HM, Dirks M, Westendorp WF, Nederkoorn PJ, van de Beek D, Ribbers GM, Steyerberg EW, et al. Development and validation of the Dutch Stroke Score for predicting disability and functional outcome after ischemic stroke: a tool to support efficient discharge planning. European Stroke Journal. 2018;3(2):165–173. [PubMed: 29900414]
43. Vagal A, Sucharewv H, Lindsell C, Kleindorfer D, Alwell K, Moomaw CJ, Woo D, Flaherty M, Khatri P, Adeoye O, et al. Predicting Mortality and Functional Outcomes after Ischemic Stroke: External Validation of a Prognostic Model. J Behav Brain Sci. 2018;8(10):587–597.
44. Cooray C, Mazya M, Bottai M, Dorado L, Skoda O, Toni D, Ford GA, Wahlgren N, Ahmed N. External validation of the ASTRAL and DRAGON scores for prediction of functional outcome in stroke. Stroke. 2016;47(6):1493–1499. [PubMed: 27174528]
45. Medeiros GC, Roy D, Kontos N, Beach SR. Post-stroke depression: a 2020 updated review. Gen Hosp Psychiatry. 2020;66:70–80. [PubMed: 32717644]
46. Ruzek JI, Brymer MJ, Jacobs AK, Layne CM, Vernberg EM, Watson PJ. Psychological first aid. J Ment Health Couns. 2007;29(1):17–49.
47. Brady TJ, Murphy L, O'Colmain BJ, Beauchesne D, Daniels B, Greenberg M, House M, Chervin D. A meta-analysis of health status, health behaviors, and health care utilization outcomes of the chronic disease self-management program. Preventing Chronic Disease. 2013;10:1–14. DOI: 10.5888/pcd10.120112.
48. Jones F, Riazi A. Self-efficacy and self-management after stroke: A systematic review, Disabil Rehab. 2011;33(10):797–810. DOI:10.3109/09638288.2010.511415.
49. Marks R, Allegrante JP, Lorig K. A review and synthesis of research evidence for self-efficacy-enhancing interventions for reducing chronic disability: implications for health education practice (part II). Health Promotion Practice. 2005;6(2):148–156. [PubMed: 15855284]
50. Warner G, Packer T, Villeneuve M, Audulv A, Versnel J. A systematic review of the effectiveness of stroke self-management programs for improving function and participation outcomes: self-management programs for stroke survivors, Disabil Rehab. 2015;37(23):2141–2163, DOI: 10.3109/09638288.2014.996674.
51. Fryer CE, Luker JA, McDonnell MN, Hillier SL. Self-management programs for quality of life in people with stroke. Stroke. 2016;47:e266–e267. [PubMed: 27895302]
52. Reddin C, Murphy R, Hankey GJ, Judge C, Xavier D, Rosengren A, Ferguson J, Alvarez-Iglesias A, Oveisgharan S, Iversen HK, et al. Association of Psychosocial Stress With Risk of Acute Stroke. JAMA Network Open. 2022;5(12):e2244836.
53. Perkins JD, Wilkins SS, Kamran S, Shuaib A. Post-traumatic stress disorder and its association with stroke and stroke risk factors: A literature review. Neurobio Stress. 2021;14:100332.
54. Krantz DS, Shank LM, Goodie JL. Post-traumatic stress disorder (PTSD) as a systemic disorder: Pathways to cardiovascular disease. Health Psychology. 2022;41(10):651–662. [PubMed: 34807673]
55. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;140(11):e596–646. [PubMed: 30879355]

56. Churchwell K, Elkind MS, Benjamin RM, Carson AP, Chang EK, Lawrence W, Mills A, Odom TM, Rodriguez CJ, Rodriguez F, et al. Call to action: structural racism as a fundamental driver of health disparities: a presidential advisory from the American Heart Association. Circulation. 2020;142(24):e454–468. [PubMed: 33170755]
57. Caceres BA, Streed CG Jr, Corliss HL, Lloyd-Jones DM, Matthews PA, Mukherjee M, Poteat T, Rosendale N, Ross LM, American Heart Association Council on Cardiovascular and Stroke Nursing; Council on Hypertension; Council on Lifestyle and Cardiometabolic Health; Council on Peripheral Vascular Disease; and Stroke Council. Assessing and addressing cardiovascular health in LGBTQ adults: a scientific statement from the American Heart Association. Circulation. 2020;10;142(19):e321–332. [PubMed: 33028085]
58. Dong TS, Gee GC, Beltran-Sanchez H, Wang M, Osadchiy V, Kilpatrick LA, Chen Z, Subramanyam V, Zhang Y, Guo Y, et al. How Discrimination Gets Under the Skin: Biological Determinants of Discrimination Associated With Dysregulation of the Brain-Gut Microbiome System and Psychological Symptoms. Biological Psychiatry. 2023;94(3):203–214. [PubMed: 36754687]
59. Yun D, Choi J. Person-centered rehabilitation care and outcomes: a systematic literature review. Int J Nurs Studies. 2019;93:74–83.
60. Magruder KM, McLaughlin KA, Elmore Borbon DL. Trauma is a public health issue. Eur J Psychotraumatology. 2015;8(1):1375338 DOI:10.1080/20008198.2017.1375338.
61. Marsland AL, Walsh C, Lockwood K, John-Henderson NA. The effects of acute psychological stress on circulating and stimulated inflammatory markers: a systematic review and meta-analysis. Brain Behav and Immun. 2017;64:208–219.
62. Rohleder N. Stimulation of systemic low-grade inflammation by psychosocial stress. Psychosom Med. 2014;76(3):181–189. [PubMed: 24608036]
63. Steptoe A, Hamer M, Chida Y. The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis. Brain Behav Immun. 2007;21(7):901–912. [PubMed: 17475444]
64. Endres M, Moro MA, Nolte CH, Dames C, Buckwalter MS, Meisel A. Immune pathways in etiology, acute phase, and chronic sequelae of ischemic stroke. Circulation Research. 2022;130(8):1167–1186. [PubMed: 35420915]
65. Iadecola C, Buckwalter MS, Anrather J. Immune responses to stroke: mechanisms, modulation, and therapeutic potential. J Clin Invest. 2020;130(6):2777–2788. [PubMed: 32391806]
66. Saavedra JM, Sánchez-Lemus E, Benicky J. Blockade of brain angiotensin II AT1 receptors ameliorates stress, anxiety, brain inflammation and ischemia: therapeutic implications. Psychoneuroendocrinology. 2011;36(1):1–18. [PubMed: 21035950]
67. Danese A, Widom CS. Associations Between Objective and Subjective Experiences of Childhood Maltreatment and the Course of Emotional Disorders in Adulthood. JAMA Psychiatry. 2023: Epub ahead of print: E1-E8. doi:10.1001/jamapsychiatry.2023.2140

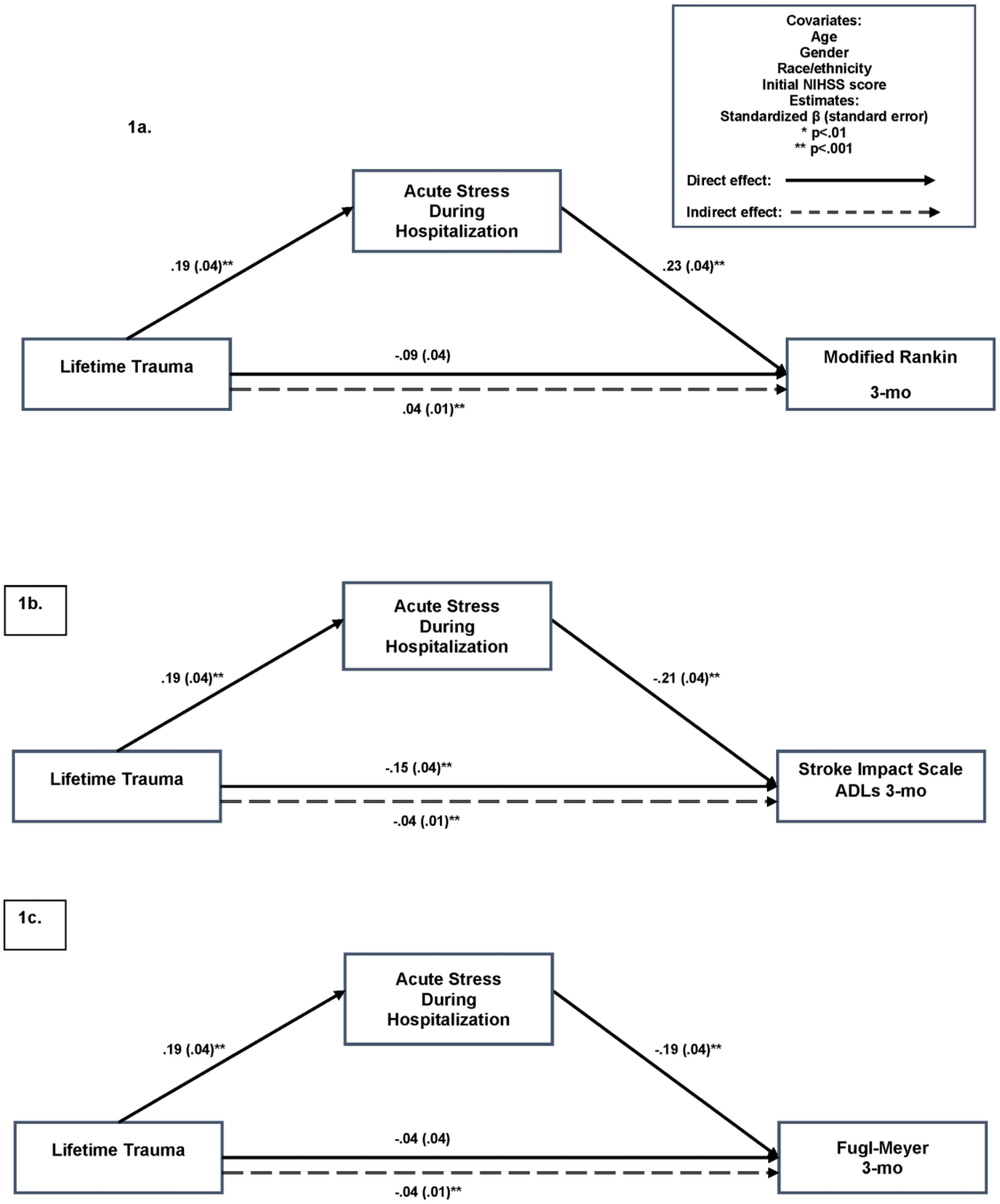


Figure 1.

Prospective standardized associations among lifetime SLEs, acute stress, and 3-month (a) mRS, (b) SIS-ADL, and (c) FM scores. Solid lines = direct effects, dashed line = indirect effect. Continuous variables were standardized prior to analysis; model was estimated using a maximum likelihood approach.

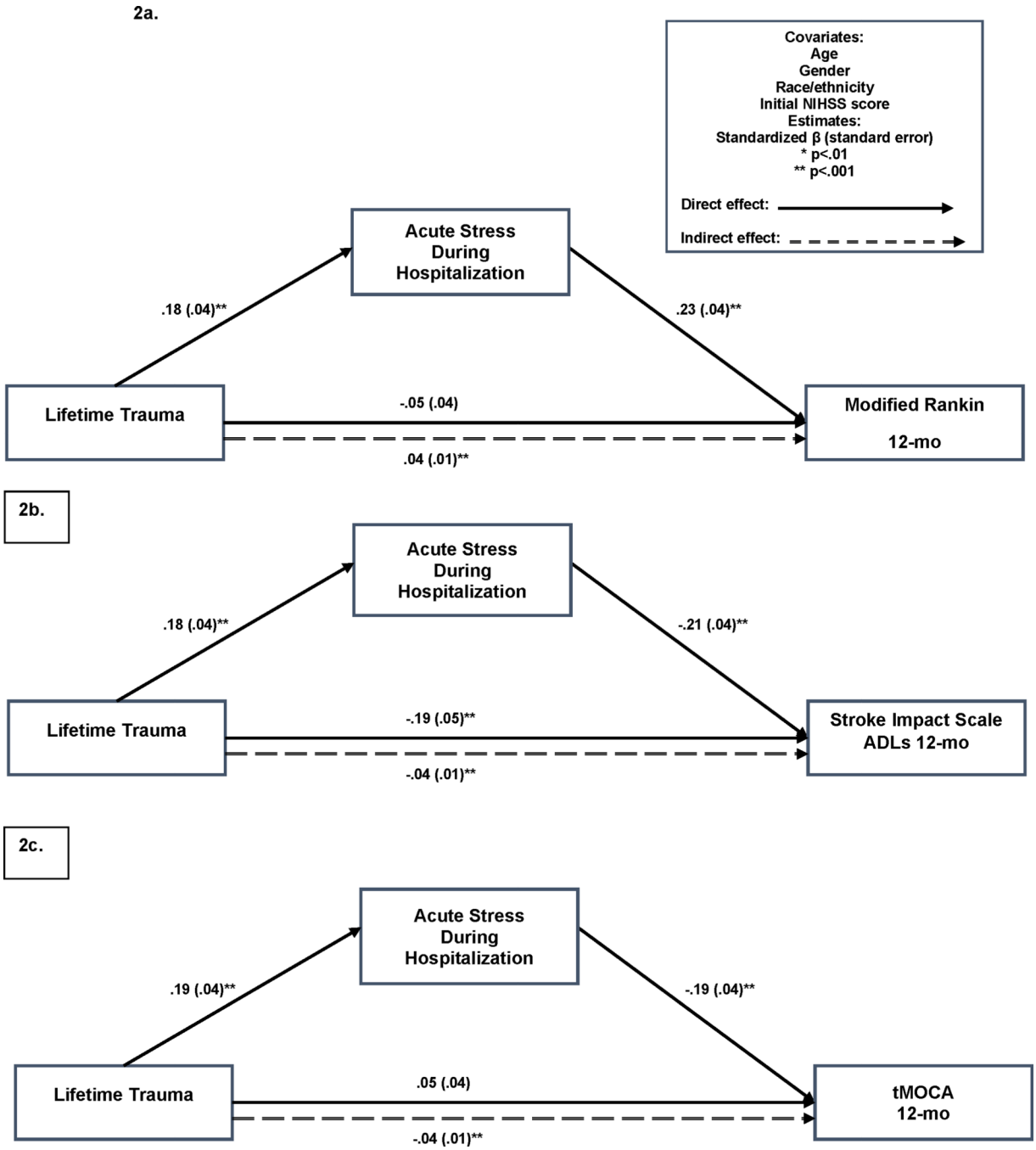


Figure 2.

Prospective standardized associations among lifetime SLEs, acute stress, and 12-month (a) mRS scores, (b) SIS-ADL scores, and (c) tMOCA scores. Solid lines = direct effects, dashed line = indirect effect. Continuous variables were standardized prior to analysis; model was estimated using a maximum likelihood approach.

Table 1.

Demographics of the sample (N=763)

Age (years)		Mean 63 (SD14.9), Range 19–95
Gender		
	Male	448 (58.7%)
	Female	314 (41.2%)
	Other	1 (0.01%)
Race		
	White	529 (69.4%)
	Black	127 (16.7%)
	Asian	41 (5.4%)
	Mixed race	37 (4.9%)
	American	12 (1.6%)
	Indian/Hawaiian or Pacific Islander	
	Other	17 (2.1%)
Ethnicity		
	Non-Hispanic	656 (86%)
	Hispanic	99 (13%)
	Unknown	8 (1%)
Marital status		
	Married	388 (50.8%)
	Divorced/widowed	185 (24.3%)
	Single	168 (22.0%)
	Other	22 (2.9%)