Received: 8 January 2022 | Revised: 19 April 2022 | Accepted: 12 May 2022

DOI: 10.1002/smi.3165

RESEARCH ARTICLE

WILEY

Why is subjective stress severity a stronger predictor of health than stressor exposure? A preregistered two-study test of two hypotheses

Grant S. Shields[1] | Alyssa Fassett-Carman[2] | Zach J. Gray[1] | Joseph E. Gonzales[3] | Hannah R. Snyder[2] | George M. Slavich[4]

[1]Department of Psychological Science, University of Arkansas, Fayetteville, Arkansas, USA

[2]Department of Psychology and Neuroscience Program, Brandeis University, Waltham, Massachusetts, USA

[3]Department of Psychology and Center for Women and Work, University of Massachusetts Lowell, Lowell, Massachusetts, USA

[4]Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, California, USA

Correspondence
Grant S. Shields, Department of Psychological Science, University of Arkansas, Fayetteville, AR 72701, USA.
Email: gshields@uark.edu

Funding information
California Initiative to Advance Precision Medicine, Grant/Award Number: OPR21101; Office of the California Surgeon General and California Department of Health Care Services, Grant/Award Number: 21-10317; National Institutes of Health, Grant/Award Numbers: T32 MH019929, T32 GM084907

Abstract

Subjective stress severity appraisals have consistently emerged as better predictors of poor health than stressor exposure, but the reason for this is unclear. Subjective stress may better predict poor health for one of at least two reasons. First, because stressor exposure measures consider all stressors as equal, stress severity measures—which "weight" stressors by self-reported severity—might better predict poor health simply by not treating all stressors as being equally impactful. Second, subjective stress appraisals may index important individual differences in stress vulnerability. We tested these two possibilities in this preregistered, two-study manuscript. Across these two different studies, subjective stress severity was a better predictor of poor health than independently weighted stress severity or stressor exposure. These results demonstrate that, beyond weighting of stressful experiences, subjective stress severity indexes health-relevant individual differences. Moreover, the results suggest that subjective stress severity may be the preferred stress summary metric even when derived from imprecise stress assessment instruments.

KEYWORDS
assessment, health, measurement, mental health, physical health, stress, stress severity

1 | INTRODUCTION

Stress contributes to the onset or maintenance of numerous negative health outcomes, including depression, anxiety, autoimmune disorders, heart disease, and early mortality (Irwin, 2008; Miller et al., 2009; Shields & Slavich, 2017; Silverman & Sternberg, 2012; Slavich, 2015; Slavich & Irwin, 2014; Solomon et al., 2019). In this article, we define a *stressor* as an objectively measurable situation or set of circumstances that objectively threatens the wellbeing of an individual, and that requires that individual to expend resources in order to cope with or survive that situation or set of circumstances (Monroe & Slavich, 2016; Slavich, 2020; Slavich & Cole, 2013). Similarly, we define *stress* as a subjective experience of distress, fear, or negative affect that occurs as a result of a perceived threat to one's mental or physical wellbeing and is accompanied by a biological response that facilitates adaptive action (Juster et al., 2010; Kim & Diamond, 2002; McEwen & Wingfield, 2003; Shields et al., 2019, 2021). The subjective experience of stress can occur without stressor

exposure, and stressor exposure can occur without a subjective experience of stress. A wealth of research has investigated mechanisms through which stressor exposure and stress exert their effects (e.g., Silverman & Sternberg, 2012; Slavich & Cole, 2013; Slavich & Irwin, 2014), and this work has demonstrated that subjective stress severity (i.e., perceived stress during one or more stressors) is often a stronger predictor of negative health outcomes than measures of stressor exposure (i.e., the occurrence of one or more stressors) (e.g., Chen et al., 2009; Cole et al., 2011; Slavich & Cole, 2013). The reasons behind this effect, however, are unclear.

One possible reason for the relatively stronger effects of subjective stress severity as compared to stressor exposure is that not all stressors exert equal effects on health, and a simple sum of all stressor exposures, which does not weight stressors by any magnitude, might thus be a relatively less robust predictor of health outcomes than stressors "weighted" by their severities. Another possible explanation for this stronger association is that relatively greater subjective stress severity—given equivalent stressor exposure—is an indication of vulnerability to the negative effects of stress (e.g., greater stress reactivity, poorer stress recovery), whereas relatively lesser subjective stress severity may be an indication of resilience. To date, however, no study has examined whether either of these formulations accounts for the relatively greater association between subjective stress severity and negative health outcomes. We thus addressed this issue in the present preregistered two-study project.

Subjective (or, "perceived") stress severity has been examined in some detail given its role in the associations between stress and health (e.g., Epel et al., 2018; Fassett-Carman et al., 2020; Fassett-Carman et al., 2019; Lebois et al., 2016; Slavich & Shields, 2018). Severity and severity-like appraisals such as decreased desirability of events, increased perceived stress burden, and perceived severity of daily hassles have been associated with depression and anxiety symptoms (Cohen et al., 1983; Compas et al., 1987; Fassett-Carman et al., 2019; Rowlison & Felner, 1988). Furthermore, there is evidence that the perceived severity of recently experienced life stressors is transdiagnostically associated with internalising symptoms over and above exposure to stressors (Fassett-Carman et al., 2020).

Although controversy exists around the use of subjective (personal severity appraisals) versus independent (third person expert ratings) measures of stressor severity, stress appraisal research has played an important role in better understanding the relations between stress and mental health. Theories have described that although the occurrence of a stressor is an occasion-setter for the development of symptoms, an individual's appraisal of the stressor is what engenders symptoms (e.g., Abramson et al., 2002). Although these theories do not focus specifically on perceived stressor severity, they do describe stress-related cognitions that likely contribute to an individual's perception of stress severity, such as perceived control over the stressor, globality (vs. specificity) and stability of the cause of the stressor, and the stressor's relation to the self. Indeed, recent research demonstrates that the perceived controllability of recently experienced stressful life events correlates with their perceived severity (Fassett-Carman et al., 2019, 2020).

Stress appraisal theories further recognise that appraisals are shaped by both situational and individual factors (Fassett-Carman et al., 2019; Gaab et al., 2005; Lebois et al., 2016). Likewise, research has demonstrated that perceived stress severity is a function of both dispositional and personality-related factors (e.g., neuroticism) as well as situational factors (e.g., expectancy violations, threat) (Lebois et al., 2016; Monroe, 2008; Slavich & Shields, 2018). This makes the association of perceived stress severity with negative health outcomes over and above stressor exposure difficult to interpret: By these data alone, it is equally possible that this greater association could be driven by resilience/vulnerability to experienced stressors (i.e., because dispositional factors drive differences in ratings of perceived stress) or by experiences with stressors that are objectively more severe (i.e., because situational factors like threat drive differences in ratings of perceived stress), or both. Because it is possible to intervene on stress-related cognitions (Crum et al., 2017; Jamieson et al., 2012) and possibly thereby affect the link between stressor exposure and health (Banica et al., 2021, 2020; Shields et al., 2017b; Shields, Skwara, et al., 2020), there is a pressing need to understand the mechanisms underpinning the relatively stronger association of stress severity vs stressor exposure with negative health outcomes.

One potential method for adjudicating between the two explanations for the greater association between subjective stress severity (relative to stressor exposure) and negative health outcomes is to quantify an independent measure of stress severity by using sample-average ratings of severity for each experienced stressor. This would enable comparison between the predictive abilities of a stress severity measure based on how severe different stressors are typically experienced across the sample with the same subjective stress severity ratings made by each individual. If the primary driving forces behind the stronger negative health association with stress severity than with stressor exposure are individual differences conferring vulnerability or resilience to stress, then subjective stress severity should be a relatively stronger predictor than the sample-estimated stress severity metric. In contrast, if the driving force behind the stronger negative health association with stress severity than with stressor exposure is the fact that not all stressors are equally stressful or impactful and use of simple exposure variables (i.e., where each experienced stressor is weighted equally) misses this important variance, then sample-estimated stress severity measure may be as strong of a predictor of negative health outcomes as subjective stress severity. A sample-estimated stress severity measure could even be a stronger predictor of negative health outcomes in this case, since the subjective measure may contain nuisance variance from personality factors that affect stress appraisals but are unrelated to health (e.g., social desirability, Crutzen & Göritz, 2010; Slavich & Shields, 2018), or that have complicated relations with health (e.g., neuroticism, Slavich & Shields, 2018; Turiano et al., 2013; Weston & Jackson, 2015).

For stress assessment instruments that have high precision in definitions of stressors given to participants—such as the Stress and Adversity Inventory (STRAIN; Slavich & Shields, 2018), described in detail below—this sample-average approach will produce a more-or-

less participant-independent severity rating for a particular stressor. For stress assessment tools that are more general with regards to categorising stressors—such as stress "checklists" (e.g., the Adolescent Life Events Questionnaire [ALEQ]; Hankin & Abramson, 2002)—the above approach will contain both a relatively independent stress severity rating and a confound from the problem of *intracategory variability* (i.e., the issue that different people will take the same ambiguous event definition—for example, "sickness of a family member"—to mean different things, such as one person believing that a distant grandmother having the common cold fits the quoted example and another person believing that the question pertains to a parent having cancer) (Dohrenwend, 2006).

Given the information above, if subjective stress severity consistently emerged as a predictor as strong as, or stronger than, sample-estimated severity across multiple stress assessment tools—regardless of how precisely defined the stressors are—then this would help resolve why severity is often a stronger predictor of poor health than stressor exposure. In particular, this finding would suggest that the relatively stronger associations of negative health outcomes with stress severity than stressor exposure are because exposure indices fail to capture individual differences related to stress vulnerability or health, and not because exposure measures fail to capture variance associated with some stressors being more severe than others. In contrast, if sample-estimated stress severity consistently emerged as a stronger predictor of poor health than subjective stress severity, this finding would suggest that subjective stress severity scores have been more strongly associated with poor health than stressor exposure because subjective severity scores "weight" stressors, but also that they capture nuisance variance unrelated to health that sample-estimated or independent severity measures do not.

2 | PRESENT RESEARCH

In this preregistered project,[1] we examined the relative strength of associations between sample-estimated and individual subjective stress severity ratings with physical and mental health outcomes. In particular, in our first study, we examined the associations between an index of stress severity that is relatively independent of participants' personality characteristics and biases (sample-estimated stress severity) and individual subjective stress severity ratings with physical health outcomes using the STRAIN in a sample of adults. In our second study, we examined the associations of sample-estimated and subjective stress severity with mental health outcomes using the Adolescent Life Event Questionnaire – Revised (ALEQ-R) in a sample of emerging adults.

We focussed on our chosen mental and physical health outcomes because of their known links to stress and their links to potential shared mechanisms, such as inflammation (e.g., Furman et al., 2019; Shields & Slavich, 2017; Slavich & Irwin, 2014; Ye et al., 2021). Given the high co-occurrence of depression and anxiety, which are the mental health outcomes assessed in Study 2, we chose to use a latent variable model that parses depression and anxiety specific dimensions from their shared component, as this enabled us to examine transdiagnostic versus specific links with stress (Clark & Watson, 1991; Lin et al., 2014). To equate methodology as much as possible between studies, we analysed our health outcomes in Study 1 using a latent variable model as well. The pattern of results was similar when the observed variables were used as outcomes instead of latent variables (see Supporting Information S1).

The link between stress and health is likely to be mostly driven by the intersection of stressor exposure and subjective stress severity. We did not expect anything different. Instead, our goal in the present study was to examine what is unique or important about stress severity that has led to its relatively stronger association with health outcomes than stressor exposure in past studies.

We had two primary aims. First, to determine whether a "weighted" sample-estimated stress severity measure was a better predictor of poor health than stressor exposure. Drawing on work suggesting that some stressors are objectively more threatening or likely to cause upheaval than others (Brown & Harris, 1978), we hypothesised that the relatively stronger association between negative health outcomes and stress severity (vs. stressor exposure) previously observed would be partially driven by differences in the "objective" stressfulness of various stressors that are missed by unweighted exposure/count measures (which treat all stressors as equal). That is, the sample-estimated stress severity measure will more strongly predict negative health outcomes than stressor exposure measures in models with both predictors included. Second, we aimed to determine whether subjective stress severity differed from sample-estimated stress severity in predicting poor health. Drawing on research showing that stress appraisals are important for initiating biological stress responses (Slavich, 2020), we hypothesised that subjective stress severity scores would be more predictive of negative health outcomes than sample-estimated stress severity scores, though a relatively stronger association with either predictor would be informative for understanding why stress severity is more strongly associated with negative health outcomes than stressor exposure (e.g., due to accounting for the severity of the situation, or due to both accounting for the severity of the situation and individual differences in stress responsivity).

3 | STUDY 1

3.1 | Method

3.1.1 | Participants

This study is a secondary analysis of data collected from a prior study of stress and health (Slavich & Shields, 2018). Participants were 205 young, middle-aged, and older adults (mean age 37.82, *SD* = 11.72, range: 19–68 years-old; 46.8% men, 52.2% women, 1.0% other) with valid, useable data (i.e., who did not fail an attention check) who were recruited from the community to complete an "online study of stress

and health" that occurred from 23 March 2016, to 29 April 2016. Participants self-reported as 85.4% White, 5.9% Black or African American, 3.9% Asian, 2.4% Hispanic, 2.0% mixed/biracial, and 0.5% declined to answer.

3.1.2 | Procedure

Potential participants who saw an online advertisement were directed to the study website where they read an overview of the study that described the topics covered and expected time commitment (i.e., 45 min). The overview also noted that the survey would include several "attention checks" that they needed to pass for their responses to be valid (e.g., "If you are reading this question, please answer C"). Individuals who read these instructions and subsequently provided their electronic consent then began the study, which assessed their stress levels, personality and social desirability characteristics, demographic factors, and health status (see below). Participants completed all of the measures online, and data were retained for those who answered all of the questions without failing the attention checks (45.7% of respondents). All study procedures were approved by the institutional review board of the University of California, Los Angeles.

3.1.3 | Measures

Lifetime Stressor Exposure. Lifetime stressor exposure was assessed using the Stress and Adversity Inventory for Adults (Adult STRAIN; Slavich & Shields, 2018). The Adult STRAIN is an online, interview-based assessment tool that queries 55 stressors, including 29 chronic stressors (e.g., caregiving, social isolation) and 26 episodic negative life events (e.g., being fired, divorced). Stressor frequencies in these data are provided in the Supporting Information S1. Each stressor is given sufficient information to describe exactly what the stressor in question is (e.g., someone "close" to the participant is defined as someone they see nearly each day of the week), and some stressors entail additional follow-up questions to ascertain exactly what happened, as is typical in life stress interviews. For each endorsed stressor, users are asked a series of follow-up questions that ascertain the severity, frequency, timing, and duration of the stressor. For example, for each stressor experienced, participants are asked (retrospectively, thinking back to when it occurred), "At its worst, how stressful or threatening was this for you?" and they provide their responses using a scale ranging from 1 (*Not at all*) to 5 (*Extremely*). These follow-up questions allow for the production of sensitive summary scores, such as experienced (i.e., subjective) stress severity, which is created by summing a participant's severity rating for each experienced stressor. In addition to using subjective stress severity as a primary predictor of outcomes, we created a new summary score for this study: a measure of sample-estimated stress severity. Each stressor had its sample-estimated severity rating established by averaging the reported severity from all participants who had experienced that stressor. This average thus indicated how severe each stressor typically feels to participants who had experienced it in the sample. For each participant, both a sum total score and a mean score of the estimated severities were then calculated across the stressors they endorsed. The sum score provides the total sample-estimated severity, whereas the mean score was calculated to remove frequency from this score due to collinearity (see Study 1 Results).

Demographic and Potential Confounding Factors. Analyses with covariates included both demographic variables (self-reported age, sex, and race) and the following variables.

Socioeconomic Status. Participants reported their annual household income and personal highest educational achievement level, and answers to these questions were standardized and averaged to create an overall index of socioeconomic status.

Social Desirability. Participants' tendency to exhibit social desirability was assessed using the 17-item Social Desirability Scale (SDS-17; Stöber, 2001). Respondents indicate *True* (1) or *False* (0) for each item, and these responses are then summed to create an overall index of socially desirable responding. Internal consistency for the scale was excellent, $\alpha = 0.94$.

Negative Affect. Participants' levels of negative affect over the past week were assessed using the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). Participants were asked to report the extent to which they felt 10 negative and 10 positive emotions over the preceding week (20 items total). Responses to each item were provided on a 1 (*Very slightly or not at all*) to 5 (*Extremely*) scale, and responses to the 10 questions assessing negative affect were then averaged to create an overall index of negative affect, with higher scores indicating more negative affect. Internal consistency for the scale was excellent, $\alpha = 0.92$.

3.1.4 | Outcomes

Sleep Quality. Participants' sleep quality was assessed using the 10-item Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1989). The PSQI assesses both objective indices of sleep quality (e.g., how often participants wake up during the night) and subjective indices of sleep quality (e.g., how rested they typically feel after a night of sleep). Answers on the PSQI were scored using the standard scoring system and then summed to create a global PSQI score, with higher scores indicating worse sleep quality.

Mental and Physical Health Complaints. We used the Kessler-6 item psychological distress inventory (K-6; Kessler et al., 2002) and the Physical Health Questionnaire (PHQ; Schat et al., 2005) to assess mental health and physical health, respectively, over the preceding month. The K-6 is a 6-item scale that asks participants to self-report the frequency that they experienced various poor mental health symptoms (e.g., feeling hopeless, feeling worthless, feeling nervous or

fidgety) over the preceding month using a Likert scale ranging from 0 (*none of the time*) to 4 (*all of the time*). The K-6 shows good convergence with Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) based measures of mental health symptoms (Kessler et al., 2002), and the PHQ is a 14-item scale that shows good convergence with general health and divergence with work stress (Schat et al., 2005). For the PHQ, participants self-reported the frequency that they experienced various somatic symptoms (e.g., gastrointestinal sensitivity, sleep disturbances, headaches) over the past month using a Likert scale ranging from 1 (*not at all*) to 7 (*all of the time*). The K-6 and PHQ both demonstrated good internal consistency ($\alpha = 0.91$ and $\alpha = 0.84$, respectively), and their scores were summed to create indices of mental and physical health, respectively; higher scores indicated more complaints over the past month.

Doctor-Diagnosed General Health Problems. The presence of general health problems diagnosed by a medical doctor was assessed by asking participants whether a medical doctor had ever diagnosed them with any of the following conditions: anxiety, arthritis (not rheumatoid or psoriatic), asthma, cancer, chronic pain, coronary heart disease, depression, gastroesophageal reflex disease (GERD, or chronic heartburn), heart attack, high blood pressure, insomnia, kidney stone(s), migraines, overweight, post-traumatic stress disorder, stomach ulcer(s), and stroke. These conditions were selected due to their well-established links to stress (Shields & Slavich, 2017). Endorsed conditions were summed to create participants' general health problem scores, with higher scores indicating more health problems diagnosed by a medical doctor.

Doctor-Diagnosed Autoimmune Disorders. The presence of autoimmune disorders diagnosed by a medical doctor was assessed by asking participants whether a medical doctor had ever diagnosed them with any of the following conditions: Addison's disease, coeliac disease, dermatomyositis, Grave's disease, Hashimoto's thyroiditis, inflammatory bowel disease (i.e., Crohn's disease, ulcerative colitis), multiple sclerosis, myasthenia gravis, pernicious anaemia, psoriasis (or psoriatic arthritis), rheumatoid arthritis, Sjögren's syndrome, lupus (systemic lupus erythematosus), and other autoimmune disorder (specify). If "other autoimmune disorder" was endorsed, the specified response was manually examined by to determine if the condition was an autoimmune disorder. Endorsed conditions were summed to create a count of autoimmune disorders; higher scores indicate more autoimmune disorders diagnosed by a medical doctor.

3.1.5 | Data analysis

All analyses were conducted in **R**, version 4.0.2. Correlation and linear models were used to analyse continuous outcomes, and generalised linear models (GLMs) were used to analyse count outcomes (e.g., number of doctor-diagnosed general health problems). Structural equation modelling (SEM) was used to model a latent variable of poor health by extracting variance common to each health outcome. We conducted the following sets of analyses both with and without controlling for relevant covariates (i.e., age, sex, race, socioeconomic status, negative affect, social desirability) to determine if these associations were robust to covariate inclusion.

Our sample size of 205 provided power of 0.80 and higher for all correlations of $|r| > 0.137$. We also conducted a Monte Carlo simulation with 1000 iterations for a power analysis of a structural equation model using the paramtest and lavaan packages in **R**. This model consisted of one latent factor indicated by the five health outcomes included in this study, which we fixed to load on the respective factor at 0.35, and an observed variable predicting the latent variable with a standardized coefficient of 0.15. All loadings and observed variables were standardized. The model was based on a single group with 1000 replications of the simulated data. We achieved 0.86 power to detect the simulated loadings and coefficient with our sample size.

Data reduction. We used structural equation modelling to create a latent variable of poor health by extracting variance common to all the health-related outcomes for which we collected data in our prior study (i.e., poor sleep quality, mental and physical health complaints, doctor-diagnosed general health problems, and doctor-diagnosed autoimmune disorders). We expected these variables to be related at a latent level due to the influence of inflammatory activity on all of these variables (e.g., Slavich & Irwin, 2014). Fit statistics deemed to indicate acceptable fit for this sample size were comparative fit index (CFI) > 0.90, root mean square error of approximation (RMSEA) < 0.08, and standardized root mean square residual (SRMR) < 0.08 (Brown, 2006; Weston & Gore, 2006).

Analysis 1. We examined the associations of subjective stress severity and sample-estimated stress severity with each health outcome and the latent health factor, using correlations and GLMs.

Analysis 2. We determined the relative predictive strength of stressor exposure compared to subjective or sample-estimated stress severity. We fit two models: one with both stressor exposure and subjective stress severity as predictors of the poor health latent variable, and a second with both stressor exposure and sample-estimated stress severity as predictors of the poor health latent variable. We then tested whether the magnitudes of slopes of each stress severity measure were greater than the magnitude of the slope of stressor exposure predicting poor health using a test of difference for dependent regression slopes. This enabled us to determine if stress severity was in fact a better predictor of poor health than stressor exposure in our sample.

Analysis 3: We determined the relative predictive strength of subjective stress severity and sample-estimated stress severity (included in the same model) on the latent health variable. We then tested whether the magnitudes of the slope of subjective stress severity differed from the magnitude of the slope of sample-estimated stress severity predicting poor health using a test of difference for dependent regression slopes. This enabled us to determine if one measure of stress severity was a better predictor of poor health than the other.

4 | STUDY 1 RESULTS

Health latent variable. Due to less than acceptable model fit ($\chi^2(5) = 34.37$, $p < 0.001$, CFI = 0.911, RMSEA = 0.17, SRMR = 0.06), modification indices were examined, and covariances of PHQ with K-6, K-6 with autoimmune disorder diagnoses, and autoimmune disorder diagnoses with other stress-related disorder diagnoses were justifiable additions that improved model fit. With those covariances included, the model fit was acceptable: $\chi^2(2) = 4.22$, $p = 0.121$, CFI = 0.993, RMSEA = 0.07, SRMR = 0.02.

Analysis 1. We first conducted analyses that examined associations between our predictors and outcomes of interest. In these analyses, we found that when self-reported and sample-estimated stress severity each included frequency of exposure information (i.e., the "sum" scores in Table 1), the correlation between these scores was very high, $r = 0.97$, $p < 0.001$, as were the correlations between these variables and stressor exposure, $rs > 0.92$, $ps < 0.001$. Whether one should include these predictors in the same model, therefore, is debatable. On one hand, the substantial overlapping variance in these predictors may lead to suppression effects; on the other hand, however, associations between health outcomes and the unique variance in each of these predictors is precisely the variance that is needed to answer the question of why stress severity is a better predictor of health outcomes than stressor exposure—for reasons outlined above. Therefore, we present these results in two ways: first, with sum scores, and second, with mean scores, which remove frequency of exposure information from the severity variables. Correlations between each of the predictors and outcomes were as expected, except that sample-estimated stress severity mean scores were not significantly associated with the majority of the examined health outcomes (Table 1).

Controlling for relevant covariates (i.e., age, sex, race, socioeconomic status, negative affect, and social desirability) did not alter the associations between self-reported stress severity and health outcomes, $\beta s > 0.24$, risk ratios (RRs) > 1.012, $ps < 0.001$, or between sample-estimated stress severity and health outcomes, $\beta s > 0.23$, RRs > 1.013, $ps < 0.001$, which both remained significant predictors of poor health. In contrast, for mean scores (i.e., removing frequency of exposure), controlling for those same covariates, self-reported stress severity only significantly predicted recent physical health complaints, poor sleep quality, and doctor-diagnosed stress-related diseases, $\beta s > 0.13$, $RR = 1.54$, $ps < 0.046$, and sample-estimated stress severity only significantly predicted doctor-diagnosed stress-related diseases, $RR = 2.89$, $p = 0.006$. These analyses thus show that the sum sample-estimated stress severity score predicted health outcomes as expected, whereas the mean sample-estimated stress severity score

TABLE 1 Bivariate correlations for the primary variables of interest in study 1

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Stressor exposure (sum) *M* = 25.77; range = 1–83									
2. Self-reported stress severity (sum) *M* = 63.26; range = 0–167	0.93***								
3. Sample-estimated stress severity (sum) *M* = 63.42; range = 0–144.30	0.95***	0.97***							
4. Self-reported stress severity (mean) *M* = 3.78; range = 0–5	0.38***	0.55***	0.40***						
5. Sample-estimated stress severity (mean) *M* = 3.89; range = 0–4.32	0.22**	0.24***	0.25***	0.51***					
6. Poor sleep quality (PSQI-global) *M* = 13.88; range = 0–41	0.49***	0.56***	0.55***	0.27***	0.14*				
7. Recent physical health complaints (PHQ) *M* = 35.94; range = 13–73	0.47***	0.54***	0.50***	0.32***	0.07	0.73***			
8. Recent mental health complaints (K-6) *M* = 14.22; range = 6–30	0.32***	0.40***	0.37***	0.22**	0.02	0.44***	0.57***		
9. Stress-related doctor-diagnosed Illnesses *M* = 2.03; range = 0–11	0.51***	0.58***	0.56***	0.33***	0.15*	0.52***	0.46***	0.34***	
10. Doctor-diagnosed autoimmune disorders *M* = 0.17; range = 0–5	0.23**	0.25***	0.24***	0.14*	0.07	0.28***	0.17*	−0.05	0.29***

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

TABLE 2 Critical tests across models and studies

		Model 1			Model 2			Model 3		
Study	Outcome	Stressor exposure *β*	Subjective stress severity *β*	Test of coefficient difference *p* value	Stressor exposure *β*	Sample-estimated stress severity *β*	Test of coefficient difference *p* value	Subjective stress severity *β*	Sample-estimated stress severity *β*	Test of coefficient difference *p* value
Study 1	Poor health									
	Sum severity predictor	−0.323	**0.999**	<0.001	−0.301	**0.948**	<0.001	**0.920**	−0.229	0.001
	Mean severity predictor	**0.542**	0.162	<0.001	**0.597**	0.011	<0.001	**0.391**	−0.059	<0.001
Study 2	Depression-specific symptoms									
	Sum severity predictor	−0.215	**0.184**	0.010	−0.063	−0.002	0.624	**0.223**	−0.236	0.017
	Mean severity predictor	−0.081	**0.133**	0.025	−0.045	−0.019	0.769	0.104	−0.042	0.375
Study 2	Anxiety-specific symptoms									
	Sum severity predictor	−0.214	**0.428**	<0.001	−0.010	0.188	0.231	**0.444**	−0.217	<0.001
	Mean severity predictor	0.116	**0.171**	0.006	0.150	−0.084	0.118	**0.208**	−0.150	0.001
Study 2	Internalising symptoms									
	Sum severity predictor	**0.264**	0.046	0.023	**0.423**	−0.144	<0.001	**0.402**	−0.138	0.001
	Mean severity predictor	0.268	0.138	0.055	0.303	0.043	0.476	0.214	−0.055	0.103

Note: Because of the high collinearity between stressor exposure and stress severity sum scores, each model was run twice—once with stress severity as a sum score, and once with stress severity as a mean score. When the model coefficients significantly differed, the stronger predictor was bolded for emphasis. The critical comparisons between subjective stress severity and sample-estimated stress severity are in model three. As shown in model three, consistent across both studies, subjective stress severity was a stronger predictor of poor health outcomes than sample-estimated stress severity. These results therefore show that subjective stress severity contains important health-relevant variability that is not captured by "weighting" stressors to a greater extent than stressor exposure scores.

(i.e., how stressful the average stressor a person experienced was) was not associated with most of the expected outcomes.

Analysis 2. In the model with stressor exposure and subjective stress severity sum scores included as concurrent predictors of latent poor health, subjective stress severity sum scores ($\beta = 0.999$, $p < 0.001$) emerged as a stronger predictor of poor health than stressor exposure ($\beta = -0.323$, $p = 0.045$), $t(203) = 6.00$, $p < 0.001$ (see Table 2). Similarly, in the model with stressor exposure and sample-estimated stress severity sum scores included as concurrent predictors of poor health, sample-estimated stress severity sum scores ($\beta = 0.948$, $p < 0.001$) emerged as a stronger predictor of poor health than stressor exposure ($\beta = -0.301$, $p = 0.127$), $t(203) = 4.67$, $p < 0.001$. These analyses therefore show that stress severity (both subjective and sample-estimated) was a better predictor of poor health than stressor exposure in our sample.

When considering mean severity scores (i.e., removing frequency of exposure from severity scores) the results differed from the above. In particular, in the model with stressor exposure and mean subjective stress severity included as concurrent predictors of latent poor health, mean subjective stress severity ($\beta = 0.162$, $p = 0.018$) and stressor exposure ($\beta = 0.542$, $p < 0.001$) were both significant predictors of poor health, but stressor exposure was a significantly stronger predictor of poor health than mean subjective stress severity, $t(203) = 4.16$, $p < 0.001$. Moreover, in the model with stressor exposure and mean sample-estimated stress severity included as concurrent predictors of poor health, mean sample-estimated stress severity was not a significant predictor of poor health ($\beta = 0.011$, $p = 0.867$), whereas stressor exposure remained a significant predictor of poor health ($\beta = 0.597$, $p < 0.001$) and a stronger predictor of poor health than mean sample-estimated stress severity, $t(203) = 6.44$, $p < 0.001$. Controlling for covariates (i.e., age, sex, race, socioeconomic status, negative affect, and social desirability) did not alter the above results. These analyses therefore show that mean subjective stress severity explained variance in poor health besides that explained by stressor exposure, whereas mean sample-estimated stress severity did not.

Analysis 3. In a model comparing the relative predictive strength of subjective and sample-estimated stress severities, subjective stress severity sum scores emerged as a significant predictor of poor health ($\beta = 0.920$, $p < 0.001$), whereas sample-estimated stress severity sum scores did not ($\beta = -0.229$, $p = 0.369$); moreover, subjective stress severity was a significantly stronger predictor of poor health than sample-estimated stress severity, $t(203) = 3.27$, $p = 0.001$ (see Table 2). The same pattern of results emerged if mean severity scores (i.e., removing frequency of exposure from severity scores) were used instead. In particular, mean subjective stress severity emerged as a

significant predictor of poor health ($\beta = 0.391$, $p < 0.001$), whereas mean sample-estimated stress severity did not ($\beta = -0.059$, $p = 0.489$), and mean subjective stress severity was a significantly stronger predictor of poor health than mean sample-estimated stress severity, $t(203) = 4.65$, $p < 0.001$. Controlling for covariates did not alter any of the above results. These results are evidence that subjective stress severity (sum or mean) was a better predictor of poor health than sample-estimated stress severity in this sample.

5 | STUDY 1 DISCUSSION

Taken together, these results suggest that stressor exposure, subjective stress severity, and sample-estimated stress severity have unique relations to health. Stressor exposure and subjective stress severity each explained unique variance in poor health, even when frequency of exposure was removed from subjective stress severity. Sample-estimated stress severity, in contrast, was a better predictor of poor health than stressor exposure alone, but the sample-estimated severity of stressors experienced did not explain unique variance in health once frequency of exposure was removed (i.e., in the mean score analyses) or subjective stress severity was included within the model. Sample-estimated stress severity therefore is potentially useful as a predictor (i.e., being a better predictor than stressor exposure alone due to its accounting for stress severity, and it not being confounded with a participant's self-report biases and personality traits). However, its failure to explain variance in poor health both once frequency of exposure information was removed—or when accounting for variance explained by subjective stress severity—provides clues as to why stress severity scores explain more variance in health outcomes than stressor exposure scores. In particular, these findings suggest that subjective stress severity measures explain more variance in health for reasons *more than* accounting for the fact that in stressor exposure scores, all stressors are counted equally and some may be more impactful on average than others. Indeed, there seems to be something special about *subjective* stress severity that is not accounted for by what an independent sample would estimate a person's stress severity to be.

6 | STUDY 2

6.1 | Method

6.1.1 | Participants

Participants were recruited from treatment-seeking college students assessed at the Brandeis Counseling Center (BCC) between September 2017 and September 2019. Inclusion criteria were being in the emerging adult range (18–25 years) and screening into both the depression and anxiety questionnaires (see below). 476 participants met inclusion criteria. Of these participants, 16 did not complete the ALEQ, and four endorsed no stressors leaving them no opportunity to provide severity ratings so they were excluded from analysis. The total sample size was thus 456 ($M_{age} = 20.4$, 70.0% women, 29.8% men, 0.2% identified as other). Race and ethnicity information was not consented for research use, but the Brandeis University student body demographic composition is 51% non-Hispanic white, 25% East Asian, 8% Hispanic, 7% other Asian, 6% Black, 3% other ethnicity. Sample means for depression ($M = 24.56$, $SD = 6.02$) and anxiety ($M = 22.29$, $SD = 5.23$) were in the Moderate range based on the PROMIS questionnaire scoring guidelines. For depression and anxiety symptoms, 62.4% and 69.7% of the sample, respectively, scored in the Moderate to Severe range.

6.1.2 | Procedure

All participants registered for treatment at the BCC independently of this study. During the registration process, all students were asked to complete a series of questionnaires as part of standard procedures to inform their care, and could optionally consent for their responses to also be used for research. Students still received all the benefits of clinical care even if they did not wish to participate in research, and there were no downsides for not participating. Consent and questionnaires were administered online using Qualtrics.

6.1.3 | Measures

The Adolescent Life Event Questionnaire – Revised (ALEQ-R) Short Version. The ALEQ-R short version assesses 10 negative life events typically experienced by youth, occurring within the past 3 months. Example items include, "You had an argument with a close friend," and, "You did poorly on or failed a test or class project." For each item, participants rate how often the event occurred in the past 3 months on a Likert scale from 0 to 4 (0 = never; 4 = always), as well as perceived severity ("How stressful was it for you?") from 1 (*Not very stressful*) to 5 (*Very stressful*). Stressor frequencies are provided in the Supporting Information S1. We calculated four stress measures from these ratings. Stressor exposure scores for each participant were calculated by summing frequency ratings across items. Subjective stress severity scores were calculated in two ways: by summing ratings for each stressor endorsed by the participant, and by taking the mean of the severity ratings for each participant across the stressors that they endorsed. This dual approach provided a sum measure of cumulative perceived stress over the past 3 months and a mean score that ensured that the subjective severity ratings were statistically independent of stressor exposure. We also calculated a "sample-estimated stress severity" score, which used the sample-mean for each stressor on the ALEQ. To get this score, we first calculated mean severity rating for each ALEQ-R stressor across participants that endorsed that item. We then calculated a sample-estimated severity score for each participant by summing the sample means of the items that they endorsed.

Mental Health. These measures employed the DSM-5 Level 1 Cross Cutting Symptom Measure (CCM) (American Psychiatric Association, 2013), which is a self-report questionnaire designed to provide a comprehensive mental health assessment across multiple mental health domains. Each domain is represented by specific items within the CCM, and each domain has a pre-determined threshold for further inquiry. If a domain threshold is met, participants complete a Level 2 questionnaire for the domain to provide a more in-depth assessment.

Depression. To reach threshold for depression, a participant must respond "Mild (Several Days)" or greater to either of the two depression domain questions ("Little interest or pleasure in doing things?" or "Feeling down, depressed, or hopeless?"). The Level 2 depression questionnaire is composed of eight items that assess frequency symptoms (e.g. I felt worthless) within the past 7 days, rated on a 5-point Likert scale (1 = never; 5 = always).

Anxiety. To reach threshold for anxiety, a participant must respond "Mild (Several Days)" or greater to at least one of the three anxiety domain questions ("Feeling nervous, anxious, frightened, worried, or on edge?" or "Feeling panic or being frightened", or "Avoiding situations that make you anxious?"). The Level 2 anxiety questionnaire contains seven items that ask about frequency of anxiety symptoms (e.g. "I felt worried") experienced in the past 7 days, rated on a 5-point Likert scale (1 = never; 5 = always).

6.1.4 | Data analysis

Outliers +/− three standard deviations from the mean for each variable were removed from the dataset and marked as missing. One participant identified as "other" for gender, so they were marked as missing from the dichotomous gender variable.

Data reduction. As we have done previously with a subset of participants from this sample (Fassett-Carman et al., 2020), we used a bifactor model to parse depression and anxiety symptoms variance into a common internalising factor that represents variance shared across disorders as well as depression and anxiety specific factors which represent variance unique to each disorder. Specifically, this model is composed of a common internalising factor on which all indicators loaded (indicated by all depression and anxiety items), as well as a depression-specific factor (initially indicated by all depression items) and an anxiety-specific factor (initially indicated by all anxiety items). Because the common internalising factor captures the variance shared among all of the depression and anxiety items and the remaining covariance among depression items and among anxiety items is captured by the specific factors, the specific factors were specified as uncorrelated with the common latent factor and each other. Monte-Carlo simulations with 1000 iterations based on the loadings from the previous bifactor model built with a subset of this sample (Fassett-Carman et al., 2020), demonstrated that the sample size had adequate power (> 0.8) to detect standardized regression paths of $\beta = 0.2$ in the SEM for all analyses.

CFA and SEM were conducted using Mplus version 8.1 (Muthén & Muthén, 2017) using full information maximum likelihood estimation to handle missing data. Adequate fit for CFAs was RMSEA < 0.06, CFI > 0.95, TLI > 0.95, SRMR < 0.08 (Hu & Bentler, 1998).[2] If the fit did not meet criteria, we followed the modification indices until adequate fit (that is, residual correlations were added as needed, and any indicators that did not load significantly on a factor were eliminated.

Analysis 1. We first examined the associations of the stress measures with each mental health outcome at an observed variable level. These analyses served as a measure validity check to determine if the sample-estimated stress severity measure was associated with all of the same outcomes it would be expected to be based on associations with subjective stress severity.

Analysis 2. The bi-factor model from above was used to construct a SEM with stress variables as predictors, enabling us to test how stress severity measures were related to mental health outcomes. Age and gender were controlled for in all regressions. We fit two models: one with both stressor exposure and subjective stress severity as predictors of the common, depression-specific, and anxiety-specific latent factors, and a second with both stressor exposure and sample-estimated stress severity as predictors of those latent factors. We then tested whether the magnitudes of slopes of each stress severity measure were greater than the magnitude of the slope of stressor exposure predicting each of the common, depression-specific, and anxiety-specific latent factors using a test of difference for dependent regression slopes.

Analysis 3. Next, we determined the relative predictive strength of subjective stress severity and sample-estimated stress severity by using them as predictors of the latent variables described above. We then tested whether the magnitudes of the slope of subjective stress severity differed from the magnitude of the slope of sample-estimated stress severity predicting poor health using a test of difference for dependent regression slopes.

7 | STUDY 2 RESULTS

Internalising symptom latent factors. Three depression items ("I felt sad," "I felt depressed," and "I felt unhappy") had significant positive loadings only on the common internalising factor and were thus removed from the depression-specific factor. Two depression items ("I felt helpless" and "I felt hopeless") and two anxiety items ("I felt uneasy" and "I felt tense") with highly related content were allowed to covary as suggested by modification indices. The bifactor model had good to acceptable model fit after these modifications, $\chi^2(105) = 3222.67$, $p < 0.001$, CFI = 0.96, TLI = 0.94, RMSEA = 0.060, SRMR = 0.039, and was thus used for all SEM analyses.

Analysis 1. Depression and anxiety symptoms were positively correlated with each other ($r = 0.56$, $p < 0.001$) and with stress exposure and stress severity sum scores ($rs > 0.18$, $ps < 0.01$). As in Study 1, self-reported and sample-estimated stress severity each included frequency of exposure information (i.e., the "sum" scores in

Table 3), the correlation between these scores was very high, $r = 0.91$, $p < 0.001$, as were the correlations between these variables and stressor exposure, $rs > 0.84$, $ps < 0.001$. Therefore, as in Study 1, we have chosen to present these results in two ways: first, with sum scores, and second, with mean scores, which remove frequency of exposure information from the severity variables. As expected, both measures of perceived stress severity correlated significantly with depression ($rs > 0.20$, $ps < 0.001$) and anxiety ($rs > 0.22$, $ps < 0.001$) symptoms. Sample-estimated stress severity sum scores showed significant positive associations with depression symptoms ($r = 0.18$, $p < 0.001$) and anxiety symptoms ($r = 0.28$, $p < 0.001$), while sample-estimated stress severity mean scores did not correlate with depression symptoms ($p = 0.714$), and had a weak, negative correlation with anxiety symptoms ($r = -0.10$, $p = 0.039$).

Analysis 2. In separate models, each controlling for stressor exposure, subjective stress severity (model 1) and sample-estimated stress severity sum scores (model 2) were both significantly or marginally associated with the anxiety-specific factor (Model 1: $\beta = 0.428$, $p < 0.001$; Model 2: $\beta = 0.188$, $p = 0.070$), but not associated with the depression-specific (Model 1: $\beta = 0.184$, $p = 0.120$; Model 2: $\beta = -0.002$, $p = 0.989$) or common internalising (Model 1: $\beta = 0.046$, $p = 0.648$; Model 2: $\beta = -0.144$, $p = 0.133$) factors (see Table 2). Stressor exposure, in contrast, was associated with the common internalising factor (Model 1: $\beta = 0.264$, $p = 0.008$; Model 2: $\beta = 0.423$, $p < 0.001$), but unassociated or inversely associated with the depression-specific (Model 1: $\beta = -0.215$, $p = 0.072$; Model 2: $\beta = -0.063$, $p = 0.602$) and anxiety-specific (Model 1: $\beta = -0.214$, $p = 0.045$; Model 2: $\beta = -0.010$, $p = 0.924$) factors. The subjective stress severity sum score emerged as a stronger predictor of the anxiety-specific and depression-specific factors than stressor exposure, $ts(450) > 2.58$, $ps \leq 0.010$, but a weaker predictor of the common internalising factor, $t(450) = -2.28$ $p = 0.023$. Sample-estimated stress severity sum scores only differed from stressor exposure sum scores in that they were weaker predictors of the common internalising factor, $t(451) = -5.00$, $p < 0.001$. These results therefore indicate that in models with stressor exposure and stress severity without frequency of exposure removed from the severity variables, subjective and sample-estimated stress severity explained variance in anxiety-specific symptoms that was not explained by stressor exposure alone.

In models using mean severity scores (i.e., removing frequency of exposure from severity scores), and again controlling for stressor exposure, subjective stress severity (Model 1) mean scores were significantly associated with the depression-specific factor ($\beta = 0.133$, $p = 0.038$), the anxiety-specific factor ($\beta = 0.171$, $p = 0.002$), and the common internalising factor ($\beta = 0.138$, $p = 0.007$), whereas mean sample-estimated stress severity (Model 2) scores were unassociated with all latent factors ($ps > 0.126$). In both models, stressor exposure was significantly associated with the anxiety-specific factor (Model 1: $\beta = 0.116$, $p = 0.042$; Model 2: $\beta = 0.150$, $p = 0.009$), and the common internalising factor (Model 1: $\beta = 0.268$, $p < 0.001$; Model 2: $\beta = 0.303$, $p < 0.001$), but not the depression-specific factor (Model 1: $\beta = -0.081$, $p = 0.225$; Model 2: $\beta = -0.045$, $p = 0.509$). In addition, subjective stress severity mean scores were significantly stronger predictors of the depression-specific, $t(449) = 2.25$, $p = 0.025$, and anxiety-specific latent factors, $t(449) = 2.75$, $p = 0.006$, than stressor exposure, but not of the common internalising factor, $t(449) = -1.92$, $p = 0.055$. Sample-estimated stress severity mean scores did not differ from stressor exposure in associations with any of the latent variables, $ps > 0.122$. These analyses therefore indicate that a subjective stress severity mean score was a stronger predictor of depression-specific and anxiety-specific symptoms than stressor exposure, whereas a sample-estimated stress severity mean scores was not.

Analysis 3. When subjective stress severity and sample-estimated severity were tested in the same model (see Table 2), subjective stress severity was significantly associated with the common internalising ($\beta = 0.402$, $p = 0.001$) and anxiety-specific ($\beta = 0.444$, $p < 0.001$) factors, but not the depression-specific factor ($\beta = 0.223$, $p = 0.133$); sample-estimated stress severity sum scores were not associated with any latent factor ($\beta s = -0.237$ to -0.137, $ps > 0.087$). Subjective stress severity sum scores were more strongly associated with the depression-specific factor, $t(452) = 2.40$, $p = 0.017$, the anxiety-specific factor, $t(452) = 3.85$, $p < 0.001$, and the common internalising factor, $t(452) = 3.28$, $p = 0.001$, than sample-estimated stress severity sum scores.

Similarly, in the model considering mean severity scores (i.e., removing frequency of exposure from severity scores), mean subjective stress severity remained significantly associated with the

TABLE 3 Bivariate correlations for primary variables of interest in study 2

Variable	1.	2.	3.	4.	5.	6.
1. Level 2 depression						
2. Level 2 anxiety	0.56***					
3. Stressor exposure (sum)	0.26***	0.31***				
4. Self-reported stress severity (mean)	0.21***	0.23***	0.22***			
5. Sample-estimated stress severity (mean)	−0.02	−0.10*	−0.16***	0.21***		
6. Self-reported stress severity (sum)	0.26***	0.35***	0.86***	0.40***	−0.10*	
7. Sample-estimated stress severity (sum)	0.18***	0.28***	0.85***	0.04	−0.13***	0.91***

Note: Level 2 scores from the cross cutting symptom measure.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

anxiety-specific factor ($\beta = 0.208$, $p < 0.001$) and the common internalising factor ($\beta = 0.214$, $p < 0.001$), but not the depression-specific factor ($\beta = 0.104$, $p = 0.117$), whereas mean sample-estimated stress severity was negatively associated with the anxiety-specific factor ($\beta = -0.150$, $p = 0.006$) and was unassociated with the common internalising and depression-specific factors (βs = −0.056 to −0.043, ps > 0.296). Subjective stress severity mean scores were more strongly associated with the anxiety-specific factor, $t(444) = 3.27$, $p = 0.001$, than sample-estimated stress severity mean scores, whereas the two types of mean stress severity scores did not differ in their associations with the depression-specific factor, $t(444) = 0.89$, $p = 0.375$, or the common internalising factor $t(444) = 1.63$, $p = 0.103$. These results therefore show that subjective stress severity is a better predictor of common internalising and anxiety-specific symptoms than sample-estimated stress severity in this sample.

8 | STUDY 2 DISCUSSION

Taken together, the results of this second study had both similarities to and differences from Study 1. In particular, although stressor exposure, subjective stress severity, and sample-estimated stress severity were each related to one or more of the latent variables, stressor exposure was a stronger predictor of the common internalising factor than either of the stress severity scores, whereas subjective stress severity—but not sample-estimated stress severity—was a stronger predictor of depression-specific and anxiety-specific factors than stressor exposure. In a notable difference from Study 1, sample-estimated stress severity was a poor predictor of all of the mental health outcomes. One potential explanation for the lack of association between mental health outcomes and sample-estimated stress severity in Study two is the problem of intra-category variability seen in the study's stress measure: the ALEQ-R provides less precise definitions of stressors than the STRAIN used in Study 1. Alternatively, these differences in associations between health outcomes may be due to other differences between the two studies (e.g., different health outcomes, a clinical college student sample vs. a healthy adult sample). Despite these differences, a strikingly similar finding between Study 1 and Study 2 is that subjective stress severity was a better predictor of poor health than sample-estimated stress severity. Indeed, as in Study 1, the results of Study 2 suggest that there is something unique—and special—about *subjective* stress severity in predicting poor health that is not accounted for by the average perceived severity of the stressors to which an individual is exposed.

9 | GENERAL DISCUSSION

The present study tested two alternative theories regarding why perceived stress severity might be a stronger predictor of health outcomes than stressor exposure. First, some situations are objectively more likely to result in injury or death, and a stressor exposure variable (e.g., stressor count) would fail to "weight" an individual's stressor exposure by those likelihoods; in contrast, an individual's self-reported subjective stress severity would "weight" their experienced stressors by at least some factor, which could lead to a stronger association with health outcomes simply because not all stressors are treated equally. Alternatively, a stronger subjective perception of stress may exacerbate the biological and psychological responses to a stressor (and vice versa; LeMoult, 2020; Niedbala et al., 2018), leading to a stronger measured effect of stress. We tested these two hypotheses in two separate studies in the current article by comparing the associations of an independently weighted stressor exposure score with a subjective stress severity score with physical and mental health.

Although the results varied somewhat between studies, across both studies we found more support for the second hypothesis than the first: An individual's subjective stress severity was a better predictor of poor health outcomes than an individual's sample-estimated stress severity. This finding held both with and without controlling for important covariates, as well as when mean scores were used instead of sum scores. Moreover, in Study 2, subjective stress severity, but not sample-estimated stress severity, was a better predictor of poor health than stressor exposure; this was not the case in Study 1, however, which found that the sum score for sample-estimated stress severity was a significantly better predictor of poor health than stressor exposure. In short, despite some differences in findings between these studies, we showed for the first time that subjective stress severity contains important information for predicting stress-related health outcomes above and beyond the "objective" (i.e., average perceived) threat or severity of experienced stressors. Although prior research has examined the difference score between objectively and subjectively rated stress severity in relation to various health outcomes (e.g., Conway et al., 2016; Espejo et al., 2012), to our knowledge, the present study is the first to directly compare the predictive utility of objectively rated and subjectively rated stress severity scores against each other—and this comparison helps to determine why stress severity is a better predictor of health outcomes than stressor exposure. Our results suggest that subjective stress severity is a better predictor not just because it "weights" stressor exposure, but because it indexes important individual differences, presumably related to stress responsivity and vulnerability. Moreover, these results have important implications for dealing with the problem of intracategory variability, which we expand on below.

It should be noted, however, that the vast majority of variance explained in health outcomes was explained by the overlap between stressor exposure and stress severity. Nonetheless, this study helped to answer why stress severity is a slightly better predictor of health outcomes than stressor exposure.

The individual differences, either within an individual or within a situation, that lead an individual to appraise a stressor as more or less severe have been considered nuisance variables when considering relations among objective stressor exposure and subjective/perceived

stress (Shields & Slavich, 2017). For example, individual differences in the tendency to respond in socially desirable ways, pessimism, neuroticism, and agreeableness are each associated with individual differences in reports of perceived stress (Cazassa et al., 2020; Extremera et al., 2007; Slavich & Shields, 2018; Sturmbauer et al., 2019). Intuitively, these associations would seem to imply that perceived stress is a less "pure" measure of stress than stressor exposure. However, stress may also alter these traits in ways that subsequently increase perceived stress (Gonzalez-Liencres et al., 2016; Shields et al., 2016; Wu et al., 2020). Therefore, rather than these variables being "nuisance" variables that might detract from true associations between stress and some outcome, these variables may relate to stress perhaps because stress may alter these traits.

It is also possible that factors that are thought to be integral to links between stressor exposure and health, such as rumination, may contribute to the individual differences that lead to variation in stressor appraisal and subjective severity. For example, a tendency to ruminate on negative events is a stable individual difference (Marchetti et al., 2018; Yang et al., 2017) that has been linked to poor health outcomes following stress (Snyder et al., 2019; Snyder & Hankin, 2016). It may be a fruitful avenue for future research to examine whether factors that have previously been thought to explain the link between stressor exposure and poor health, such as rumination, predict variation in stress appraisals and subjective stress severity.

Our results have a number of important implications for stress assessment. For example, the gold-standard Life Events and Difficulties Schedule (Brown & Harris, 1978) uses objective raters to score the severity of stressors that participants have experienced. The use of these "objective" severity ratings has strong intuitive appeal, but our results suggest that these ratings may fail to capture important variance in the experiences of stress that confer poor health vulnerabilities—variance which is indexed by subjective stress severity reports. This is thus consistent with theories of stress and health that posit that subjective appraisals of stress are integral to initiating stress responses and thus producing detrimental consequences of stress (Abramson et al., 2002; Slavich, 2020, 2022; Slavich & Cole, 2013).

A second important implication these results have for stress assessment lies in the consistency between subjective stress severity results across studies. Notably, although the stress assessment measure used in Study 1 contained sufficient information to avoid the problem of intracategory variability, the stress assessment measure used in Study 2 did not. However, in both studies, the subjective stress severity measure emerged as a stronger predictor of poor health outcomes than stressor exposure. This consistent finding suggests that subjective stress severity may be relatively robust against the problem of intracategory variability. This interpretation makes intuitive sense; even though one person may understand "sickness" to mean a cold and another person may understand it to mean cancer, the subjective severity of a cold and cancer will presumably differ substantially, even though the exposure to "sickness" is the same given the lack of question specificity. Thus, our results suggest that subjective stressor exposure should be the outcome metric of choice when a stress assessment tool may suffer from the problem of intracategory variability.

Although we can only speculate about the potential reasons for the discrepant findings between our two studies, a few are important to consider. First, participants for Study 1 came from a nonclinical sample of healthy adults, whereas the participants for Study 2 came from a clinical sample of undergraduate students seeking treatment. Second, and relatedly, the outcome measure in Study 1 was a broad outcome measure of poor health that was indicated more by physical health (e.g., autoimmune disorder diagnoses) than mental health, whereas the outcome measures in Study 2 were exclusively mental health factors. Third, the stress assessment measure used in Study 1 (i.e., the STRAIN) suffers from less of a problem of intracategory variability (i.e., the same stressor description being interpreted in different ways by different people) than the stress assessment measure used in Study 2 (i.e., the ALEQ-R) due to the interview-based design of the measure used in Study 1 (Shields & Slavich, 2017). It is likely that each of these differences contributed to the discrepancy in findings between studies, but the discrepancy between Study 1 and Study 2 in how predictive sample-estimated severity scores were of outcomes while controlling for stressor exposure may be explained by differences between the stress assessment measures in sensitivity to the problem of intracategory variability—estimated stress severity is presumably less precise with less consistent understandings of stressors across participants.

Rather than a limitation, the discrepancy between results of our two studies further extends an important implication for stress assessment described above. In particular, the inconsistency in sample-estimated stress severity results across studies coupled with the consistency between the subjective stress severity results suggest that subjective stress severity may be relatively more robust to issues related to the problem of intracategory variability than sample-estimated stress severity. That is, even when a stress assessment measure contains insufficient information to avoid the problem of intracategory variability (as in Study 2), subjective stress severity still captures important predictive information, whereas sample-estimated stress severity does not. These results therefore suggest that when stress is assessed with an imprecise measure, subjective stress severity may be the most appropriate predictor variable that can be derived from that measure.

One potentially surprising result is that mean subjective severity scores, which remove frequency of exposure information from stress severity ratings, emerged as significant predictors of poor health in both studies, even though stressor exposure was included as a predictor in these models. This finding indicates that individual differences in the extent to which someone appraises *any* stressor as severe (or not), regardless of number of stressors experienced, is an important predictor of poor health outcomes (Fassett-Carman et al., 2019, 2020). However, this individual difference is a weaker predictor of poor health than simple exposure to more stressors. This finding is thus consistent with models of stress and health which argue that both stressor exposure and stress vulnerability factors

contribute to poor health outcomes (Deer et al., 2021; Harris, 2020; Helgeson & Zajdel, 2017; Shields et al., 2017a; Yang et al., 2020).

Our study has several strengths, including preregistered hypotheses, replication of primary results, and use of conceptually similar but concretely distinct measures, thus enhancing the generalisability of the results. However, several limitations should also be noted. First, the study is correlational, precluding any causal inference. It is certainly possible, for example, for health problems (e.g., depression) to make the experience of stress more severe. Although it is not possible to completely manipulate stress severity given exposure to a stressor, converting interventions that aim to decrease the perceived stressfulness of experienced stressors (Crum et al., 2017; Jamieson et al., 2012; Shields, Spahr, & Slavich, 2020) to longitudinal forms and assessing changes in health over time may help to further clarify the relations among stressor exposure, perceived stress severity, and health. Nonetheless, our results suggest that there is some variance in subjective stress severity linked to poor health outcomes that is not explained by independent ratings of severity for experienced stressors. Second, the sample-estimated severity ratings were not derived from trained experts in rating the severity of stressors, but by average severity ratings from all participants that had experienced each respective stressor. It is thus possible that findings with trained expert ratings of severity could diverge from what we observed. However, this divergence would lead to the question of why, since presumably severity ratings from people that had experienced a particular stressor would be more accurate in classifying the severity of a stressor than ratings from people that had not experienced that stressor—no matter their training. Third, as in every study of stress, self-report biases may have influenced the reporting of stress. Fourth, we did not assess medication usage in Study 1, and it is possible that some of the participants may have been taking medication for anxiety or depression, which could have altered associations that we observed. Fifth, we did not assess stress biomarkers, and it is possible that stressor exposure and stress severity may have evidenced different associations with physiological indicators of health related to stress, such as markers of allostatic load (e.g., Juster McEwen, & Wingfield, 2003), than the associations that we observed. Future work should thus attempt to determine how subjective appraisals might relate to physiological responses to stress (e.g., Ali et al., 2017; Niedbala et al., 2018; Skoluda et al., 2015). Sixth, our samples demographics did not permit assessment of the relevance of our results to health disparities. Seventh, we were unable to determine the temporal directionality of links between stress and health in this study. Our goal was to determine why subjective stress severity has been found to be a stronger predictor of stress-related outcomes than stressor exposure, and future work should examine the longitudinal links between stressor exposure and the development of health outcomes assessed in these studies (e.g., autoimmune disorder diagnoses) over time. Finally, although our samples were quite distinct, which serves to enhance the replicability of our findings, both of these samples were WEIRD (i.e., Western, Educated, Industrialised, Rich, and Democratic) relative to the majority of the world, and observed associations may differ in non-WEIRD samples (Henrich et al., 2010).

10 | CONCLUSION

In conclusion, across two studies, we found that subjective stress severity was a stronger predictor of poor health outcomes than either sample-estimated stress severity or stressor exposure. These findings suggest that the relatively stronger link between stress severity and poor health than stressor exposure (observed in prior research) were not due to a failure to "weight" stressors in frequency or exposure variables. Rather, there is likely something unique about an individual's perception of stress—more than the "objective" stressfulness of their experiences—that confers risk for poor health. In short, the findings illustrate that when attempting to understand links between stress and health, subjective appraisals of stressful experiences cannot be simply disregarded in favour of more "objective" measures. Moreover, the findings have important implications for stress assessment, as they suggest that subjective stress severity, not stressor exposure or estimated stress severity, are the most predictive summary metric derivable from stress measures, even when the assessment tool used to quantify stress suffers from the problem of intracategory variability.

ACKNOWLEDGEMENTS

George Slavich was supported by grant #OPR21101 from the California Initiative to Advance Precision Medicine and by contract #21-10317 from the Office of the California Surgeon General and California Department of Health Care Services, which supports the UCLA-UCSF ACEs Aware Family Resilience Network. Alyssa Fassett-Carman was supported by training grant T32 MH019929 from NIMH and T32 GM084907 from NIGMS. These organizations had no role in designing or planning this study; in collecting, analyzing, or interpreting the data; in writing the article; or in deciding to submit this article for publication.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data are available upon request.

ORCID

Grant S. Shields https://orcid.org/0000-0002-0827-4669
George M. Slavich https://orcid.org/0000-0001-5710-3818

ENDNOTES

[1] This project was preregistered after data collection but prior to hypothesis testing. The preregistration document can be found at https://osf.io/tmcj2/

[2] Although these fit statistic cutoffs differ from those used in Study 1, this is because the smaller sample size of Study 1 alter criteria for acceptable model fit (Brown, 2006; Weston & Gore, 2006).

REFERENCES

Abramson, L. Y., Alloy, L. B., Hankin, B. L., Haeffel, G. J., MacCoon, D. G., & Gibb, B. E. (2002). Cognitive vulnerability-stress models of

depression in a self-regulatory and psychobiological context. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (pp. 268–294). https://doi.org/10.1023/A

Ali, N., Nitschke, J. P., Cooperman, C., & Pruessner, J. C. (2017). Suppressing the endocrine and autonomic stress systems does not impact the emotional stress experience after psychosocial stress. *Psychoneuroendocrinology*, *78*, 125–130. https://doi.org/10.1016/j.psyneuen.2017.01.015

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5* (Vol. 5). American psychiatric association.

Banica, I., Sandre, A., Shields, G. S., Slavich, G. M., & Weinberg, A. (2020). The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. *International Journal of Psychophysiology*, *153*, 27–36. https://doi.org/10.1016/j.ijpsycho.2020.03.006

Banica, I., Sandre, A., Shields, G. S., Slavich, G. M., & Weinberg, A. (2021). Associations between lifetime stress exposure and the error-related negativity (ERN) differ based on stressor characteristics and exposure timing in young adults. *Cognitive, Affective & Behavioral Neuroscience*. https://doi.org/10.3758/s13415-021-00883-z

Brown, G. W., & Harris, T. (1978). Social origins of depression: A study of psychiatric disorder in women. In *Social origins of depression: A study of psychiatric disorder in women*. https://doi.org/10.4324/9780203714911

Brown, T. A. (2006). Confirmatory factor analysis for applied research. In *Methodology in social sciences* (2nd ed.). The Guilford Press. https://doi.org/10.1007/s11336-016-9548-3

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Cazassa, M. J., Oliveira, M., da, S., Spahr, C. M., Shields, G. S., & Slavich, G. M. (2020). The Stress and Adversity Inventory for Adults (Adult STRAIN) in Brazilian Portuguese: Initial validation and links with executive function, sleep, and mental and physical health. *Frontiers in Psychology*, *10*, 3083. https://doi.org/10.3389/fpsyg.2019.03083

Chen, E., Miller, G. E., Walker, H. A., Arevalo, J. M., Sung, C. Y., & Cole, S. W. (2009). Genome-wide transcriptional profiling linked to social class in asthma. *Thorax*, *64*, 38–43. https://doi.org/10.1136/thx.2007.095091

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*(3), 316–336. https://doi.org/10.1037/0021-843X.100.3.316

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385–396. https://doi.org/10.2307/2136404

Cole, S. W., Hawkley, L. C., Arevalo, J. M. G., & Cacioppo, J. T. (2011). Transcript origin analysis identifies antigen-presenting cells as primary targets of socially regulated gene expression in leukocytes. *Proceedings of the National Academy of Sciences of the United States of America*, *108*, 3080–3085. https://doi.org/10.1073/pnas.1014218108

Compas, B. E., Davis, G. E., Forsythe, C. J., & Wagner, B. M. (1987). Assessment of major and daily stressful events during adolescence: The Adolescent Perceived Events Scale. *Journal of Consulting and Clinical Psychology*, *55*, 534–541. https://doi.org/10.1037/0022-006X.55.4.534

Conway, C. C., Starr, L. R., Espejo, E. P., Brennan, P. A., & Hammen, C. (2016). Stress responsivity and the structure of common mental disorders: Transdiagnostic internalizing and externalizing dimensions are associated with contrasting stress appraisal biases. *Journal of Abnormal Psychology*, *125*(8), 1079–1089. https://doi.org/10.1037/abn0000163

Crum, A. J., Akinola, M., Martin, A., & Fath, S. (2017). The role of stress mindset in shaping cognitive, emotional, and physiological responses to challenging and threatening stress. *Anxiety, Stress & Coping*, *30*, 379–395. https://doi.org/10.1080/10615806.2016.1275585

Crutzen, R., & Göritz, A. S. (2010). Social desirability and self-reported health risk behaviors in web-based research: Three longitudinal studies. *BMC Public Health*, *10*, 720. https://doi.org/10.1186/1471-2458-10-720

Deer, L. B. K., Shields, G. S., Alen, N. V., & Hostinar, C. E. (2021). Curvilinear associations between family income in early childhood and the cortisol awakening response in adolescence. *Psychoneuroendocrinology*, *129*. https://doi.org/10.1016/j.psyneuen.2021.105237

Dohrenwend, B. P. (2006). Inventorying stressful life events as risk factors for psychopathology: Toward resolution of the problem of intracategory variability. *Psychological Bulletin*, *132*, 477–495. https://doi.org/10.1037/0033-2909.132.3.477

Epel, E. S., Crosswell, A. D., Mayer, S. E., Prather, A. A., Slavich, G. M., Puterman, E., & Mendes, W. B. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology*, *49*, 146–169. https://doi.org/10.1016/j.yfrne.2018.03.001

Espejo, E. P., Hammen, C., & Brennan, P. A. (2012). Elevated appraisals of the negative impact of naturally occurring life events: A risk factor for depressive and anxiety disorders. *Journal of Abnormal Child Psychology*, *40*(2), 303–315. https://doi.org/10.1007/s10802-011-9552-0

Extremera, N., Durán, A., & Rey, L. (2007). Perceived emotional intelligence and dispositional optimism-pessimism: Analyzing their role in predicting psychological adjustment among adolescents. *Personality and Individual Differences*, *42*(6), 1069–1079. https://doi.org/10.1016/j.paid.2006.09.014

Fassett-Carman, A. N., DiDomenico, G. E., von Steiger, J., & Snyder, H. R. (2020). Clarifying stress-internalizing associations: Stress frequency and appraisals of severity and controllability are differentially related to depression-specific, anxiety-specific, and transdiagnostic internalizing factors. *Journal of Affective Disorders*, *260*, 638–645. https://doi.org/10.1016/j.jad.2019.09.053

Fassett-Carman, A. N., Hankin, B. L., & Snyder, H. R. (2019). Appraisals of dependent stressor controllability and severity are associated with depression and anxiety symptoms in youth. *Anxiety, Stress & Coping*, *32*, 32–49. https://doi.org/10.1080/10615806.2018.1532504

Furman, D., Campisi, J., Verdin, E., Carrera-Bastos, P., Targ, S., Franceschi, C., Ferrucci, L., Gilroy, D. W., Fasano, A., Miller, G. W., Miller, A. H., Mantovani, A., Weyand, C. M., Barzilai, N., Goronzy, J. J., Rando, T. A., Effros, R. B., Lucia, A., Kleinstreuer, N., & Slavich, G. M. (2019). Chronic inflammation in the etiology of disease across the life span. *Nature Medicine*, *25*(12), 1822–1832. https://doi.org/10.1038/s41591-019-0675-0

Gaab, J., Rohleder, N., Nater, U. M., & Ehlert, U. (2005). Psychological determinants of the cortisol stress response: The role of anticipatory cognitive appraisal. *Psychoneuroendocrinology*, *30*, 599–610. https://doi.org/10.1016/j.psyneuen.2005.02.001

Gonzalez-Liencres, C., Breidenstein, A., Wolf, O. T., & Brüne, M. (2016). Sex-dependent effects of stress on brain correlates to empathy for pain. *International Journal of Psychophysiology*, *105*, 47–56. https://doi.org/10.1016/j.ijpsycho.2016.04.011

Hankin, B. L., & Abramson, L. Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Clinical Child and Adolescent Psychology*, *31*, 491–504. https://doi.org/10.1207/S15374424JCCP3104_8

Harris, B. N. (2020). Stress hypothesis overload: 131 hypotheses exploring the role of stress in tradeoffs, transitions, and health. *General and Comparative Endocrinology*, *288*, 113355. https://doi.org/10.1016/j.ygcen.2019.113355

Helgeson, V. S., & Zajdel, M. (2017). Adjusting to chronic health conditions. *Annual Review of Psychology*, *68*, 545–571. https://doi.org/10.1146/annurev-psych-010416-044014

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). Most people are not WEIRD. *Nature*, *466*, 29. https://doi.org/10.1017/S0140525X0999152X

Hu, L. T., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods*, *3*(4), 424–453. https://doi.org/10.1037/1082-989X.3.4.424

Irwin, M. R. (2008). Human psychoneuroimmunology: 20 Years of discovery. *Brain, Behavior, and Immunity*, *22*, 129–139. https://doi.org/10.1016/j.bbi.2007.07.013

Jamieson, J. P., Nock, M. K., & Mendes, W. B. (2012). Mind over matter: Reappraising arousal improves cardiovascular and cognitive responses to stress. *Journal of Experimental Psychology: General*, *141*(3), 417–422. https://doi.org/10.1037/a0025719

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*, 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002

Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S. L. T., Walters, E. E., & Zaslavsky, A. M. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine*, *32*, 959–976. https://doi.org/10.1017/S0033291702006074

Kim, J. J., & Diamond, D. M. (2002). The stressed hippocampus, synaptic plasticity and lost memories. *Nature Reviews Neuroscience*, *3*(6), 453–462. https://doi.org/10.1038/nrn849

Lebois, L. A. M., Hertzog, C., Slavich, G. M., Barrett, L. F., & Barsalou, L. W. (2016). Establishing the situated features associated with perceived stress. *Acta Psychologica*, *169*, 119–132. https://doi.org/10.1016/j.actpsy.2016.05.012

LeMoult, J. (2020). From stress to depression: Bringing together cognitive and biological science. *Current Directions in Psychological Science*, *29*(6), 592–598. https://doi.org/10.1177/0963721420964039

Lin, A., Yung, A. R., Wigman, J. T. W., Killackey, E., Baksheev, G., & Wardenaar, K. J. (2014). Validation of a short adaptation of the Mood and Anxiety Symptoms Questionnaire (MASQ) in adolescents and young adults. *Psychiatry Research*, *215*(3), 778–783. https://doi.org/10.1016/j.psychres.2013.12.018

Marchetti, I., Mor, N., Chiorri, C., & Koster, E. H. W. (2018). The brief state rumination inventory (BSRI): Validation and psychometric evaluation. *Cognitive Therapy and Research*, *42*(4), 447–460. https://doi.org/10.1007/s10608-018-9901-1

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, *43*(1), 2–15. https://doi.org/10.1016/S0018-506X(02)00024-7

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*, *60*, 501–524. https://doi.org/10.1146/annurev.psych.60.110707.163551

Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annual Review of Clinical Psychology*, *4*, 33–52. https://doi.org/10.1146/annurev.clinpsy.4.022007.141207

Monroe, S. M., & Slavich, G. M. (2016). Psychological stressors: Overview. In G. Fink (Ed.), *Stress: Concepts, cognition, emotion, and behavior* (pp. 109–115). Academic Press. https://doi.org/10.1016/B978-0-12-800951-2.00013-3

Muthén, B., & Muthén, L. (2017). Mplus. In *Handbook of item response theory* (pp. 507–518). Chapman and Hall/CRC.

Niedbala, E. M., Hohman, Z. P., Harris, B. N., & Abide, A. C. (2018). Taking one for the team: Physiological trajectories of painful intergroup retaliation. *Physiology & Behavior*, *194*, 277–284. https://doi.org/10.1016/j.physbeh.2018.06.011

Rowlison, R. T., & Felner, R. D. (1988). Major life events, hassles, and adaptation in adolescence: Confounding in the conceptualization and measurement of life stress and adjustment revisited. *Journal of Personality and Social Psychology*, *55*, 432–444. https://doi.org/10.1037/0022-3514.55.3.432

Schat, A. C. H., Kelloway, E. K., & Desmarais, S. (2005). The Physical Health Questionnaire (PHQ): Construct validation of a self-report scale of somatic symptoms. *Journal of Occupational Health Psychology*, *10*, 363–381. https://doi.org/10.1037/1076-8998.10.4.363

Shields, G. S., Dunn, T. M., Trainor, B. C., & Yonelinas, A. P. (2019). Determining the biological associates of acute cold pressor post-encoding stress effects on human memory: The role of salivary interleukin-1β. *Brain, Behavior, and Immunity*, *81*, 178–187. https://doi.org/10.1016/j.bbi.2019.06.011

Shields, G. S., Hostinar, C. E., Vilgis, V., Forbes, E. E., Hipwell, A. E., Keenan, K., & Guyer, A. E. (2021). Hypothalamic–pituitary–adrenal axis activity in childhood predicts emotional memory effects and related neural circuitry in adolescent girls. *Journal of Cognitive Neuroscience*, *33*(5), 872–886. https://doi.org/10.1162/jocn_a_01687

Shields, G. S., Moons, W. G., & Slavich, G. M. (2017a). Inflammation, self-regulation, and health: An immunologic model of self-regulatory failure. *Perspectives on Psychological Science*, *12*, 588–612. https://doi.org/10.1177/1745691616689091

Shields, G. S., Moons, W. G., & Slavich, G. M. (2017b). Better executive function under stress mitigates the effects of recent life stress exposure on health in young adults. *Stress: The International Journal on the Biology of Stress*, *20*, 75–85. https://doi.org/10.1080/10253890.2017.1286322

Shields, G. S., Skwara, A. C., King, B. G., Zanesco, A. P., Dhabhar, F. S., & Saron, C. D. (2020). Deconstructing the effects of concentration meditation practice on interference control: The roles of controlled attention and inflammatory activity. *Brain, Behavior, and Immunity*, *89*, 256–267. https://doi.org/10.1016/j.bbi.2020.06.034

Shields, G. S., & Slavich, G. M. (2017). Lifetime stress exposure and health: A review of contemporary assessment methods and biological mechanisms. *Social and Personality Psychology Compass*, *11*(8). https://doi.org/10.1111/spc3.12335

Shields, G. S., Spahr, C. M., & Slavich, G. M. (2020). Psychosocial interventions and immune system function: A systematic review and meta-analysis of randomized clinical trials. *JAMA Psychiatry*, *77*, 1031–1043. https://doi.org/10.1001/jamapsychiatry.2020.0431

Shields, G. S., Toussaint, L. L., & Slavich, G. M. (2016). Stress-related changes in personality: A longitudinal study of perceived stress and trait pessimism. *Journal of Research in Personality*, *64*, 61–68. https://doi.org/10.1016/j.jrp.2016.07.008

Silverman, M. N., & Sternberg, E. M. (2012). Glucocorticoid regulation of inflammation and its functional correlates: From HPA axis to glucocorticoid receptor dysfunction. *Annals of the New York Academy of Sciences*, *1261*, 55–63. https://doi.org/10.1111/j.1749-6632.2012.06633.x

Skoluda, N., Strahler, J., Schlotz, W., Niederberger, L., Marques, S., Fischer, S., Thoma, M. V., Spoerri, C., Ehlert, U., & Nater, U. M. (2015). Intra-individual psychological and physiological responses to acute laboratory stressors of different intensity. *Psychoneuroendocrinology*, *51*, 227–236. https://doi.org/10.1016/j.psyneuen.2014.10.002

Slavich, G. M. (2015). Understanding inflammation, its regulation, and relevance for health: A top scientific and public priority. *Brain, Behavior, and Immunity*, *45*, 13–14. https://doi.org/10.1016/j.bbi.2014.10.012

Slavich, G. M. (2020). Social safety theory: A biologically based evolutionary perspective on life stress, health, and behavior. *Annual Review of Clinical Psychology*, *16*, 265–295. https://doi.org/10.1146/annurev-clinpsy-032816-045159

Slavich, G. M. (2022). Social Safety Theory: Understanding social stress, disease risk, resilience, and behavior during the COVID-19

pandemic and beyond. *Current Opinion in Psychology*, *45*:101299. https://doi.org/10.1016/j.copsyc.2022.101299

Slavich, G. M., & Cole, S. W. (2013). The emerging field of human social genomics. *Clinical Psychological Science*, *1*, 331–348. https://doi.org/10.1177/2167702613478594

Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin*, *140*, 774–815. https://doi.org/10.1037/a0035302

Slavich, G. M., & Shields, G. S. (2018). Assessing lifetime stress exposure using the Stress and Adversity Inventory for Adults (Adult STRAIN): An overview and initial validation. *Psychosomatic Medicine*, *80*, 17–27. https://doi.org/10.1097/PSY.0000000000000534

Snyder, H. R., Friedman, N. P., & Hankin, B. L. (2019). Transdiagnostic mechanisms of psychopathology in youth: Executive functions, dependent stress, and rumination. *Cognitive Therapy and Research*, *43*(5), 834–851. https://doi.org/10.1007/s10608-019-10016-z

Snyder, H. R., & Hankin, B. L. (2016). Spiraling out of control: Stress generation and subsequent rumination mediate the link between poorer cognitive control and internalizing psychopathology. *Clinical Psychological Science*, *4*, 1047–1064. https://doi.org/10.1177/2167702616633157

Solomon, Z., Levin, Y., Crompton, L., & Ginzburg, K. (2019). Is acute stress reaction a risk factor for early mortality? *Health Psychology*, *38*, 606–612. https://doi.org/10.1037/hea0000744

Stöber, J. (2001). The Social Desirability Scale-17 (SDS-17): Convergent validity, discriminant validity, and relationship with age. *European Journal of Psychological Assessment*, *17*(3), 222–232. https://doi.org/10.1027//1015-5759.17.3.222

Sturmbauer, S. C., Shields, G. S., Hetzel, E.-L., Rohleder, N., & Slavich, G. M. (2019). The stress and adversity inventory for adults (adult STRAIN) in German: An overview and initial validation. *PLoS One*, *14*(5), e0216419. https://doi.org/10.1371/journal.pone.0216419

Turiano, N. A., Mroczek, D. K., Moynihan, J., & Chapman, B. P. (2013). Big 5 personality traits and interleukin-6: Evidence for "healthy Neuroticism" in a US population sample. *Brain, Behavior, and Immunity*, *28*, 83–89. https://doi.org/10.1016/j.bbi.2012.10.020

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–1070. https://doi.org/10.1037/0022-3514.54.6.1063

Weston, R., & Gore, P. A. (2006). A brief guide to structural equation modeling. *The Counseling Psychologist*, *34*(5), 719–751. https://doi.org/10.1177/0011000006286345

Weston, S. J., & Jackson, J. J. (2015). Identification of the healthy neurotic: Personality traits predict smoking after disease onset. *Journal of Research in Personality*, *54*, 61–69. https://doi.org/10.1016/j.jrp.2014.04.008

Wu, C. H., Wang, Y., Parker, S. K., & Griffin, M. A. (2020). Effects of chronic job insecurity on Big Five personality change. *Journal of Applied Psychology*, *105*(11), 1308–1326. https://doi.org/10.1037/apl0000488

Yang, Y., Cao, S., Shields, G. S., Teng, Z., & Liu, Y. (2017). The relationships between rumination and core executive functions: A meta-analysis. *Depression and Anxiety*, *34*, 37–50. https://doi.org/10.1002/da.22539

Yang, Y., Shields, G. S., Wu, Q., Liu, Y., Chen, H., & Guo, C. (2020). The association between obesity and lower working memory is mediated by inflammation: Findings from a nationally representative dataset of U.S. adults. *Brain, Behavior, and Immunity*, *84*, 173–179. https://doi.org/10.1016/j.bbi.2019.11.022

Ye, Z., Kappelmann, N., Moser, S., Davey Smith, G., Burgess, S., Jones, P. B., & Khandaker, G. M. (2021). Role of inflammation in depression and anxiety: Tests for disorder specificity, linearity and potential causality of association in the UK Biobank. *EClinicalMedicine*, *38*, 100992. https://doi.org/10.1016/j.eclinm.2021.100992

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Shields, G. S., Fassett-Carman, A., Gray, Z. J., Gonzales, J. E., Snyder, H. R., & Slavich, G. M. (2023). Why is subjective stress severity a stronger predictor of health than stressor exposure? A preregistered two-study test of two hypotheses. *Stress and Health*, *39*(1), 87–102. https://doi.org/10.1002/smi.3165