Original Article

AMERICAN SOCIOLOGICAL ASSOCIATION

Journal of Health and Social Behavior
1–18


DOI: 10.1177/00221465211055993
jhsb.sagepub.com
SAGE

Stressful Life Events, Differential Vulnerability, and Depressive Symptoms: Critique and New Evidence

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Abstract

Depressive symptoms are disproportionately high among women and less educated individuals. One mechanism proposed to explain this is the differential vulnerability hypothesis—that these groups experience particularly strong increases in symptoms in response to stressful life events. We identify limitations to prior work and present evidence from a new approach to life stress research using the UK Household Longitudinal Study. Preliminarily, we replicate prior findings of differential vulnerability in between-individual models. Harnessing repeated measures, however, we show that apparent findings of differential vulnerability by both sex and education are artifacts of confounding. Men and women experience similar average increases in depressive symptoms after stressful life events. One exception is tentative evidence for a stronger association among women for events occurring to others in the household. We term this the "female vulnerability to network events" hypothesis and discuss with reference to Kessler and McLeod's related "cost of caring" hypothesis.

Keywords

depression, gender, life events, mental health, stress

Stressful life events (SLEs) are central to the etiology of mental health problems such as depression, which impose a large burden of disability on populations worldwide (World Health Organization 2017). Early case-control studies routinely found that around 80% of cases of depression were preceded by an SLE (Mazure 1998), and more sophisticated recent designs present strong evidence that SLEs exert a causal effect (Hammen 2005; Kendler and Gardner 2010; Kendler, Karkowski, and Prescott 1999).

Early research on stress and depression further highlighted that most people who experience such an event do not become depressed, leading to a subsequent focus on effect heterogeneity or "differential vulnerability." Psychiatric epidemiologists made progress by focusing on high-risk subpopulations—particularly working-class mothers—and identifying "vulnerability factors" whose presence correlated with a larger impact of SLEs on depressive symptoms (Bifulco et al. 1998; Brown and Harris 1978)—for instance low mastery, lack of social support, and a genetic diathesis (Kessler 1997; Thoits 2010).

Symptoms of depression and anxiety exhibit a socially stratified distribution. A substantial sex difference and educational gradient are robust and valid findings (Fryers, Melzer, and Jenkins 2003; Hyde, Mezulis, and Abramson 2008; Lorant et al. 2003; Mirowsky and Ross 1995). Sociologists

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have studied whether differential vulnerability to SLEs might underlie inequalities in mental health by sex, socioeconomic position, race, and other characteristics. Extending the reasoning of the psychiatric epidemiology literature, resources and characteristics identified as influencing vulnerability within high-risk groups may also be unevenly distributed between groups (Thoits 1995). More generally, certain groups may lack other broad categories of resources that allow individuals to mitigate the effects of SLEs (Link and Phelan 1995). Integrative models of depression have emphasized affective, biological, and particularly cognitive sources of greater vulnerability to SLEs among women (Hankin and Abramson 2001; Hyde et al. 2008). In particular, it is often claimed that women employ less effective coping strategies, being more likely to ruminate and less likely to either accept or attempt to control a given problem (Rosenfield and Mouzon 2013). Many early studies found evidence for differential vulnerability to SLEs by sex and socioeconomic position (Aneshensel 1992; Kessler 1979; McLeod and Kessler 1990).

The current study revisits the question of whether differential vulnerability to SLEs may be a mechanism generating mental health inequalities. We apply data and methods new to the topic to produce substantially stronger evidence than is currently available. In short, whereas prior research has used checklist measures of stressors that in many cases lack an objective standard of measurement, we harness the specific questions available in UK Household Longitudinal Study (UKHLS) data to construct more objective measures; while prior research has attempted to account for the confounding influence of chronic (ongoing) stressors by measuring them with items that are explicitly subjective as well as endogenous to psychological distress, we account for them by using a first-differences panel model on the assumption that such chronic stressors tend to exhibit stability over the course of 1 year; and while prior research has assumed all stressors equally severe, we harness over 150,000 observations of over 35,000 individuals to model and take into account variation in severity between different SLEs. Our approach also has substantial advantages with respect to the measurement of SLEs that occur to individuals close to the focal respondent.

The next section discusses prior evidence and sets out our research questions. We then present a more detailed critique of this prior evidence as motivation for our own empirical analysis.

BACKGROUND

In a landmark set of articles, Turner and colleagues raised the concern that early findings of differential vulnerability may be artifactual (Avison, Ali, and Walters 2007; Turner 2003; Turner and Avison 2003; Turner, Wheaton, and Lloyd 1995). Unmeasured group differences in the level of exposure to stressors, arising in part from a focus on SLEs and a lack of attention to chronic stressors in prior research, may "masquerade" as findings of differential vulnerability (Turner and Avison 2003:499).[1] That is, a larger association between SLEs and depression among women, for instance, may arise because women experiencing SLEs typically endure a disproportionately high level of chronic stressors—ongoing stressful situations such as an abusive relationship or a family member with an addiction.

These authors accordingly attempted to measure stressors comprehensively before testing the relative contribution of differential vulnerability and differential exposure to group differences in mental health. Although they found that "vulnerability differences account for over 50 percent of the total gap [in depressive symptoms between men and women]" (Turner et al. 1995:116), their overall conclusion—that "differences in exposure to stress alone account for between 23 and 50 percent of observed differences in mental health by sex, marital status, and occupation…[in] contrast with the prevailing view [emphasizing the role of] differences in vulnerability" (Turner et al. 1995:104)—appears to have been functionally canonized by Thoits's (2010) review, now cited over 1,800 times.[2]

Interest in differential vulnerability remains high in the literature on physical health inequalities, where there have been supportive findings, including reports of stronger associations between health behaviors and outcomes among the less educated (Christensen et al. 2017; Diderichsen, Hallqvist, and Whitehead 2019; Hoven and Siegrist 2013; Katikireddi et al. 2017; Veronesi et al. 2017). Since Thoits's (2010) review, however, sociological work on stress and depression has largely focused on specific stressors or, when considering stress holistically, on the theorization and exploration of "anticipatory stressors" (Grace 2020; Wheaton et al. 2013).

In sum, the work by Turner and colleagues has come to be viewed as the last word on social stratification in stress and depression. We agree with their methodological criticism of prior work. However, more recently collected data and a different approach allow us to more rigorously test the

implications of that criticism as far as it concerns the hypothesis that women and individuals with relatively low education suffer more depressive symptoms in response to SLEs.

Our first research question, then, is:

Research Question 1: Do men and women and individuals with differing levels of education vary in their vulnerability to SLEs?

A limited version of the differential vulnerability hypothesis holds that rather than being "pervasively more vulnerable," women show more depressive symptoms than men only in response to network stressors—that is, events that occur to close others (Kessler and McLeod 1984:620). With the aim of bringing theoretical clarity, we term this the "female vulnerability to network events" (FVNE) hypothesis. Gender role and identity theories provide a theoretical rationale, emphasizing that women are disproportionately socialized to privilege others over the self and expected to provide empathy and support in times of trouble (Rosenfield, Lennon, and White 2005; Taylor 2015).

Having first identified this pattern, Kessler and McLeod (1984:620) described further suggestive findings consistent with a particular mechanism for FVNE: "the greater emotional investment of women in the lives of those around them." This has come to be known as the "cost of caring" hypothesis. Although it is difficult to identify a precise statement of this thesis, it is clear that it posits certain specific mechanisms for FVNE and rejects others, specifically women's higher likelihood of being sought out for help: "Men do provide a substantial amount of support to people in crisis. Yet men are somehow able to avoid the personal distress felt by women" (Kessler, McLeod, and Wethington 1985:497). The limited subsequent research has often conflated the FVNE and cost of caring hypotheses and has yielded mixed results (Aneshensel, Rutter, and Lachenbruch 1991; Taylor 2015; Turner and Avison 1989). The most recent work—albeit importantly drawing attention to the role of gender orientation in the differential vulnerability debate—used cross-sectional data collected by Turner and colleagues from 1990 to 1991 (Taylor 2015).

Household panel data present an opportunity to test the FVNE hypothesis in a manner that improves on prior work, in which individuals are typically asked about SLEs that occurred to "anyone close to you." The definition of "close" is subjective and may be influenced by the effect an event has on the respondent's well-being. With household data, although we miss some SLEs occurring to indisputably close network members, a clear, objective constraint is applied to the definition of network membership, allowing a test of the FVNE hypothesis uncontaminated by any differential propensity to define others as close. Moreover, unlike in prior research, these network members are themselves interviewed so that measures of SLEs are constructed from their own responses and do not depend on the focal respondent's idiosyncrasies.

Our second research question, then, is:

Research Question 2: Do men and women vary in their vulnerability to SLEs occurring to others within their household?

CRITIQUE

Background: The Conceptualization and Measurement of Stress and Stressors

The rich development of the study of stress in humans has been lucidly reviewed elsewhere (Cohen, Murphy, and Prather 2019; Hammen 2016; Monroe 2008; Wheaton et al. 2013). There remain multiple perspectives on the conceptualization of stress, with differing emphases, but researchers now widely endorse a stress process of the following description. Environmental stimuli perceived as threatening, challenging, or harmful (stressors) trigger psychological and physiological responses adapted for enabling a reaction that avoids harm and maintains core regulatory processes (the stress response). This allostatic response is costly to the body and, if sustained continually, increases risk of subsequent health problems. Psychological distress is a "manifest maladaptive response pattern in the presence of stress, such as anxiety, depression, anger, fear, or aggression" (Wheaton et al. 2013:300) and occurs in the context of insufficient coping resources. To a degree, such responses may in fact be adaptive (Nesse 2019), but they are nevertheless usually undesirable affective states and bring significant impairment if sustained.

A central problem in the stress literature is the measurement of stressors. Research can only inform policy and practice if it produces evidence on the associations between objective circumstances and health outcomes. These associations exhibit a high degree of heterogeneity—people respond differently when faced with the same stressor. But this heterogeneity of response cannot be understood without reliable measurement of the stimulus.

The link from potential stressor to stress is conditional on an individual's appraisal of that stressor. As usually conducted, however, the *measurement* of stressors is also subject to the idiosyncratic appraisals of respondents. The most common approach has been checklists of SLEs (and later, other sorts of stressor; Dohrenwend 2006; Monroe 2008). Respondents indicate which of a set of SLEs has occurred in their life in the recent past (usually a year). Often, stressors are only vaguely defined. This creates a problem of intracategory variability—the set of recorded instances of a given SLE may vary substantially in terms of their objective severity. For instance, "minor illnesses are included along with major events such as heart attacks in response to an item like *serious physical illness or injury*" (Dohrenwend 2006:480). Other respondents tend in the other direction, failing to indicate SLEs that objectively did occur. Monroe (2008:42) gives the example of a woman whose husband suffered a heart attack but who did not tick "serious illness in close family member" because to her, "the event wasn't stressful. As a result of his heart attack, her husband had… become more patient, and they were getting along much better than previously."

In small-scale psychiatric epidemiology studies, a credible solution emerged in the form of intensive structured interviewing with independent postinterview transcript coding (Dohrenwend 2006). The most widely used such instrument is the Life Events and Difficulties Schedule (LEDS; Brown and Harris 1978). This painstaking approach has proven fruitful in many respects. Its focus on homogeneous, high-risk populations is suitable for the important project of building and testing models of the etiology of depression (Kendler and Gardner 2016). However, it offers little insight into *between*-group differences in mental health problems—that is, health inequality.

The sociological literature on stress and mental health has sought to describe and explain these between-group differences, requiring larger samples and making administration of the LEDS or similar instruments prohibitively expensive. The work of Turner and colleagues (Avison et al. 2007; Turner 2003; Turner and Avison 2003; Turner et al. 1995) marked an advance on prior research and remains the most recent treatment of inequalities in stress and depression by sex, socioeconomic position, marital status, and race. Due in large part to the cost and time demands of comprehensively measuring stressors, this research was on a relatively small scale and limited to single cities in the United States and Canada, using analytic samples of 1,393 adult residents of metropolitan Toronto (Turner et al. 1995), 899 non-Hispanic white and African American 18- to 22-year-olds originally sampled from the Miami-Dade public school system (Turner and Avison 2003), and 817 mothers living in London, Ontario (Avison et al. 2007). These represent valuable original data but nonetheless limit generalizability on a number of dimensions. It is also not clear that these samples yield sufficient statistical power to adequately test for interactions. We focus on three further important limitations.

Subjective Measurement of Stressors to Self and to Others

Returning to the issue of appraisal, a first major limitation of these studies is the use of survey items that in many cases lack an objective standard of measurement. For example, among the SLE measures are "a serious accident or injury," "a serious illness," "trouble with the law," "a major financial crisis," and "a change of job for a worse one." No definition is given of each stressor. Rather, the interviewer reads a list to the respondent, and they indicate whether each one occurred. Interpretation of the meaning of items is in the hands of the respondent, inviting misunderstanding and idiosyncrasy. Monroe (2008:40) writes of such checklist measures that "people quite often interpret the life event descriptors in highly personal and idiosyncratic ways."

In addition to random error, this space for subjective interpretation yields important risks of endogeneity. These arise, first, from the influence of the dependent variable—a common concern is that distressed individuals may seek to locate an explanation or justification of their distress. A second potential source is any systematic difference in interpretation between the groups being compared.

Of particular importance, third, is endogeneity arising from the magnitude of the effect a given event has on an individual's well-being. For example, it is highly plausible that people who were distressed by a financial crisis of a given objective level of intensity are more likely to report that item as having occurred than people who experienced the same objective circumstance but were not distressed by it. This threatens our ability to estimate associations between objective circumstances and experiences of distress and thus also to test whether these vary by sociodemographic characteristics. If there *is* systematic differential vulnerability, we are unlikely to be able to detect it if we do not capture

the disproportionate number of occasions when (as a hypothetical example) men experience (an objectively defined level of) financial problems but are not distressed by it.

Furthermore, respondents' awareness that they are taking part in a survey focused on stress can lead to false positives. Validation studies report that "[participants] felt their task was to provide information, and consequently they often 'stretched' their experiences to fit the categories of the checklist" (McQuaid et al. 1992:53). The same "commonly" happens due simply to a wish to seem interesting: "people who also had very few if any events, but upon questioning rather sheepishly and spontaneously indicated that they did not want us to think their lives were boring" (Monroe 2008:42). Interview (rather than self-completion) administration of checklists may exacerbate these social desirability biases.

Checklists used as part of self-report questionnaires have been extensively criticized for these and many other reasons (Dohrenwend 2006; Monroe 2008). There has been less attention to the validity of stress checklists administered as part of an interview, but it is clear that many of the same issues apply. Although according to Turner et al. (1995:107) "each reported event required an additional series of probing questions," these appear to be limited to asking the respondent to indicate the month in which each event began and ended, aided by the use of a calendar and by encouragement to place significant dates on it to aid recall.

The problems described so far are magnified for items where respondents are asked "about some things that happened to you or anyone close to you (that is your spouse/partner, children, relatives or close friends)" (Turner et al. 1995:120). Definition of "close friends" is highly subjective and likely to be affected by the impact a given event had on the respondent: If an acquaintance experienced an accident *and the respondent was stressed by this*, it is more likely to be mentioned than if it had no effect (i.e., the accident is more likely to meet the respondent's understanding of "serious," *and* the acquaintance is more likely to meet the respondent's understanding of "close").

Such network events can be reliably measured only by giving a strict, objective definition of network membership or by prespecifying "close" network members in a prospective design. McQuaid and colleagues (1992:53) found that "subjects would often lower the 'threshold' for events such as serious illnesses or deaths associated with close relatives or friends; upon interview, these turned out to be events of distant relatives or casual friends (or friends of relatives or friends of friends)." Cohen et al. (2019:589) note that "women are more likely to also report exposure to stressful life events that occur to close others." For the reasons given, it is impossible to know whether this truly reflects a pattern of differential exposure, differences in reporting due to differing levels of distress or vulnerability, or even some other mechanism.

Endogenous Measures of Chronic Stressors

A second, related, major limitation of this set of studies is the endogeneity of measures of chronic stressors arising from their substantial conceptual overlap with the outcome. A distinctive feature of the work of Turner and colleagues (1995:121) has been its explicit embrace of the use of subjective measures of chronic stressors—items such as "You find it is too difficult to find someone compatible with you," "You are alone too much," "Your partner doesn't show enough affection," and "Your job often leaves you feeling both mentally and physically tired." Without presenting the results in a table or figure, Wheaton (1994:97–98) gives a very brief report of some analyses using a sample of 530 Canadians which support "the measurement integrity of assuming [distress and chronic stress are] separate concepts", but also imply substantial overlap between the two: "In no case did the factor loading of a chronic stress item on a distress factor reach as high as 0.30"; "reciprocal causation [between chronic stress and distress] held for most types of chronic stress." The typical cognitive hallmarks of depression would imply substantial overlap, especially for items that may be influenced by negative self-perception and pessimism about the future (Wisco 2009).

The advantage of these measures is a more comprehensive sampling of the different sources of stress, which plainly extend well beyond discrete events such as deaths, job losses, and divorces, and reflect the myriad consequences of systems of social organization for the difficulties people face in their day-to-day lives (Pearlin 1989; Wheaton 1994). This strategy is appropriate for accurately describing the social distribution of stress (Turner and Avison 2003). On the other hand, the inclusion of these items is less clearly appropriate for estimating causal associations between stressors and mental health outcomes.

Equal Weighting of Stressors

A third major limitation is the operationalization of stressors in the models of Turner and colleagues (Avison et al. 2007; Turner 2003; Turner and Avison 2003; Turner et al. 1995). Each indicated stressor is counted as of equal weight within its broad category (SLEs, chronic stressors, childhood traumas, and lifetime traumas).[3] That is, the life event index is constructed as a "simple count," equally weighting items from the death of a child to a child moving back into the house (Turner et al. 1995:120). To be clear, moving out of your city, your partner "experienc[ing] a change of job for a worse one," and a relative having a serious illness are together measured as 3 times as stressful as the death of a child.

Furthermore, this assumption of interchangeability among stressors may introduce error into comparisons of groups' exposure and vulnerability if the distribution of particular stressors varies across the groups being analyzed. In other words, some groups may be disproportionately exposed to the more objectively severe stressors on the list independent of differences in the number of stressors reported. Turner and colleagues (Avison et al. 2007; Turner and Avison 2003; Turner et al. 1995) do not provide descriptive statistics on each stressor item, which leaves in doubt the validity of their description of differential exposure to stressors by gender, race, and socioeconomic position.

To a certain extent, these limitations were unavoidable or represented considered trade-offs. We conclude, however, that these features of the work of Turner and colleagues call into question the validity of their tests of the differential vulnerability hypothesis. On this specific issue, we are able to present new evidence of greater validity. We now turn to the presentation of this evidence.

METHODS

Data

The nationally representative UK Household Longitudinal Study (UKHLS, also known as Understanding Society) comprises nine annual waves of data collection, from 2009–11 to 2017–19. Each adult member of approximately 40,000 households (at Wave 1) is interviewed annually on a wide range of topics.

Although we included information from all interviewed household members ages 16 and above in constructing the SLE measures (e.g., household member had a miscarriage or termination), our analytic sample was restricted to those ages 25 to 64 so that education was a stable characteristic in almost all cases.

Stressful Life Events

From Wave 2 of the survey onward, binary measures of the occurrence over the past year of the following SLEs were constructed from the UKHLS data: job loss (due to being fired, made redundant, or a temporary job ending), becoming unemployed, union dissolution (of a marriage or cohabiting union lasting at least 3 months), eviction or other forced move, new child, low birthweight or premature baby, miscarriage or termination, stillbirth or infant death, onset of a serious health condition, hospital stay lasting more than 2 days, onset or deterioration of a disability, becoming a full-time carer, and household financial problems (the household has been behind with rent, council tax, or other bills in the past 12 months). We also constructed indicators for each of these events happening to anyone else in the household (where applicable), plus measures of whether a household member died and whether a household member went to prison. This final item was available only from Wave 6 onward.

Details of the construction of these measures are given in the Appendix in the online version of the article. It should be noted that these items were not presented to the respondent in the form reported in the previous paragraph, but constructed post hoc from a series of answers to more specific questions through which respondents are routed as applicable. Moreover, items applying to other household members were coded from those other individuals' own responses, unlike in checklist approaches to SLEs.

Education

We used a sex- and 5-year birth-cohort-relativized measure of education as a broad indicator of socioeconomic position. Access to different educational credentials, as well as their social meaning and import, has changed drastically across the birth cohorts included in our sample (1946–1993). To achieve similarity in the size of educational groups across cohorts given the distributions of actual qualifications (see Appendix Figures S1 and S2 in the online version of the article), we created a high education group that corresponds to approximately the most educated 33%, a low education group corresponding approximately to the least educated 20%, and a medium education group, the remainder (see Appendix Figures S3 and S4 in the online version of the article). Unlike the available absolute measures of education, this relativized measure was negligibly correlated with age ($\rho = -.01$).

Outcome: General Health Questionnaire

The General Health Questionnaire 12-item version (GHQ-12) is a validated and widely used survey instrument designed to screen for nonspecific psychiatric morbidity and measure mental health in general (Goldberg et al. 1997; McCabe et al. 1996; Pevalin 2000). It is a "valid and reliable measure of general psychological distress, depressive symptoms, and anxiety" (Elovainio et al. 2020:1). Respondents answered each item on a 4-point scale (for positively phrased items such as "Over the past few weeks, have you been able to concentrate on whatever you're doing?": "better than usual," "same as usual," "less than usual," "much less than usual"). We followed the 0–0–1–1 scoring method and analyzed GHQ score as a continuous variable ranging from 0 to 12, with a higher score indicating more depressive symptoms.

Analytical Approach

The first step of the analysis was to combine the individual SLEs into an overall measure of SLE exposure. We approached this problem by constructing a data-driven SLE index. Using the eight available waves (2–9) of SLE and GHQ data, we applied a change score model (Allison 1990) to estimate the expected within-individual change in GHQ for each SLE, independent of all other SLEs, across the entire sample:

$$GHQ_{it} - GHQ_{i,t-1} = \boldsymbol{\beta}_k \boldsymbol{SLE}_{kit} + \left(\varepsilon_{it} - \varepsilon_{i,t-1}\right). \quad (1)$$

GHQ_{it} is the GHQ score of individual i at wave t, and $\boldsymbol{SLE}_{kit}$ is a vector of 24 dummy variables for the SLEs we measure, with k indexing the particular SLE. $\boldsymbol{\beta}_k$ is a vector of coefficients representing, for each of the 24 events, the effect of that event's occurrence in the interval from $t-1$ to t on change in GHQ score in the same interval. ε is an idiosyncratic error term. Note that individual-level unmeasured characteristics υ_i that are constant across (at least) adjacent waves would appear in the equation for GHQ_{it} but are absent from Equation 1 by construction: $\upsilon_i - \upsilon_i = 0$.[4]

With the coefficients from this model (shown in Figure 1), we constructed an SLE index (SLEI) that was adjusted for varying mean severity across different types of events, for time-invariant unobserved confounding, and for possible confounding among different events. Coefficients for each event experienced by individual i in wave t were summed:

$$SLEI_{it} = \beta_1 SLE_{1it} + \beta_2 SLE_{2it} + \ldots + \beta_K SLE_{Kit}. \quad (2)$$

By construction, then, a one-unit change in SLEI is the amount of SLE exposure that correlates with a one-unit within-individual change in GHQ score, on average, across our whole sample.

Recall Turner and colleagues' (1995:106) concern that unmeasured chronic stressors that are unevenly distributed across groups "parade" in research as findings in support of differential vulnerability to SLEs. This concern is raised in the context of prior cross-sectional (and thus necessarily between-individual) analyses, and Turner et al.'s (1995) proposed remedy—more comprehensive measurement of stressors—is also applied to cross-sectional data. We argue that for the purposes of testing the differential vulnerability hypothesis, a preferable approach to the problem is a within-individual analysis that excises stable differences in unmeasured stressor exposure and thus does not depend on the quixotic task of measuring chronic stressors.

As an intermediate step linking our results to those of prior work, we explored what we would find if we were similarly restricted to a between-individual analysis. In these "between-effects" models, we ignored within-variation and used each individual's mean value of each variable as if we had a large cross-sectional sample:

$$\overline{GHQ}_i = \alpha + \beta \overline{SLEI}_i + \gamma \bar{G}_i + \delta \overline{SLEI}_i \bar{G}_i + \upsilon_i + \bar{\varepsilon}_i \,. \quad (3)$$

Here, α is an intercept, G_i represents one or more indicator variables for group membership (e.g., *female*, with reference group *male; high education* and *mid education*, with reference group *low education*) with coefficient(s) γ. Note that G_i is time-invariant in the vast majority of cases. A small number of individuals do gain further education, however, and thus change education category. The coefficient(s) δ gives the interaction effect between SLEI and group membership, constituting a test of the differential vulnerability hypothesis.

The final step in our analysis assessed whether the within-individual change in GHQ associated with a given burden of SLE exposure differed across groups. This was our preferred test of the differential vulnerability hypothesis.

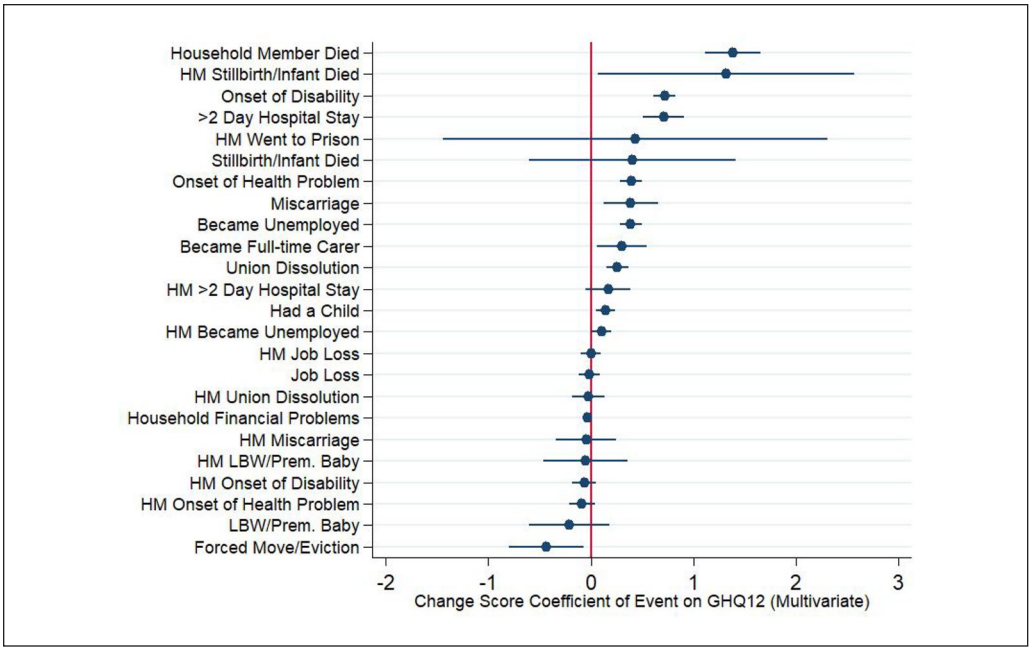

Figure 1. Coefficients Used to Construct SLE Index, from Change Score Regression of GHQ on SLEs, With Data from the UK Household Longitudinal Study.
Note: N (observations) = 159,055; *N* (individuals) = 36,341; SLE = stressful life event; GHQ = General Health Questionnaire 12-item version; HM = household member; LBW = low birthweight. For the coefficients from separate, bivariate models regressing GHQ change score on each SLE, see Figure S5 in the online version of the article.

$$GHQ_{it} - GHQ_{i,t-1} = \beta(SLEI_{it} - SLEI_{i,t-1}) + \gamma(G_{it} - G_{i,t-1}) + \delta(SLEI_{it}G_{it} - SLEI_{i,t-1}G_{i,t-1}) + (\varepsilon_{it} - \varepsilon_{i,t-1}). \quad (4)$$

In this case, we applied a first-differences model rather than a change score model. Whereas each individual SLE rarely recurs across consecutive waves, SLEI amalgamates all measured SLEs, making it far more likely to be nonzero across consecutive waves. It may then be important to adjust for the influence of the previous wave's SLEI on the baseline GHQ, $GHQ_{i,t-1}$. This is achieved by including *change in SLEI* rather than the absolute SLEI as in a change score model.[5]

RESULTS

We first comment briefly on Figure 1, which shows results from the model described in Equation 1. These coefficients are used to construct the SLE index as described in Equation 2. Although many events are clearly associated with an increase in depressive symptoms, many others show a small or null association. However, these associations are averaged across the whole sample. As discussed previously, modest average effects in the general population alongside strong other evidence indicating the importance of SLEs for mental health originally spurred the focus on effect heterogeneity, which is to say differential vulnerability. It should also be noted that null average effects are concentrated among SLEs occurring to others in the household rather than to the self.

Descriptives for the observations from Wave 2 are shown in Table 1. Approximately one-third of the sample experience an SLE in this first wave of SLE measurement. The SLEs we measure occur to men and women in similar numbers in most cases, but a clear educational gradient is evident, particularly for economic and health-related events such as becoming unemployed or the onset of a serious medical condition. For both men and women and across education levels, the percentage of individuals with an SLE index close to zero is 80 or above.

Table 2 shows the results of our main analyses. With Model 1, the first column, we confirm that

Table 1. Descriptive Statistics: Stressful Life Events and SLE Index by Sex and Education ($N = 29,066$), with Data from the UK Household Longitudinal Study Wave 2.

	Overall		Sex		Education		
			Male	Female	High	Mid	Low
	%	Frequency	%	%	%	%	%
			43.4	56.6	31.2	45.7	23.1
0 Stressful life events	67.3	19,567	67.3	67.3	74.0	68.3	56.4
1 Stressful life event	23.9	6,944	23.9	23.9	20.3	23.2	30.1
≥2 Stressful life events	8.8	2,555	8.8	8.8	5.7	8.5	13.5
HH financial problems	14.7	4,261	14.2	15.0	8.9	14.5	22.6
Had a child	4.1	1,195	4.3	4.0	4.5	3.5	4.8
Job loss	3.1	892	4.2	2.2	3.0	3.0	3.1
Onset of serious health condition	3.0	882	2.8	3.2	2.2	3.1	4.1
HH member: job loss	2.9	848	2.5	3.2	2.7	3.0	3.1
Became unemployed	2.6	745	2.7	2.5	1.5	2.1	4.9
HH member became unemployed	2.4	710	2.4	2.4	1.7	2.3	3.8
HH member: onset of serious health condition	2.3	667	2.5	2.1	1.7	2.3	3.0
Onset/deterioration of disability	1.7	507	1.6	1.9	1.1	1.6	2.8
Union dissolution	1.6	464	1.4	1.8	1.2	1.6	2.0
HH member: onset/deterioration of disability	1.2	361	1.4	1.1	.9	1.2	1.8
Hospital stay (>2 days)	1.0	295	1.1	1.0	.6	1.2	1.2
HH member: hospital stay (>2 days)	.8	243	.8	.8	.7	.8	1.3
HH member: union dissolution	.6	177	.6	.6	.5	.6	.8
Miscarriage or termination	.4	129	.0	.8	.6	.4	.4
HH member: miscarriage or termination	.4	109	.8	.1	.4	.4	.4
Onset of full-time caring responsibilities	.3	101	.2	.4	.2	.3	.6
HH member died	.2	68	.2	.3	.2	.3	.2
Had a premature or low birthweight child	.2	57	.0	.3	.2	.1	.3
HH member had a premature or low birthweight child	.2	57	.4	.1	.2	.2	.2
Eviction or other forced move	.1	31	.1	.1	.1	.1	.1
Stillbirth or infant death	.0	13	.0	.0	.1	.0	.0
HH member: stillbirth or infant death	.0	9	.0	.0	.0	.0	.0
HH member went to prison	.0	0	.0	.0	.0	.0	.0
SLE index <.10	85.5	24,864	86.4	84.9	87.9	86.7	80.0
SLE index 0.10–0.50	11.2	3,249	10.6	11.6	9.9	10.1	15.1
SLE index 0.50–1.00	2.0	590	1.7	2.3	1.5	1.9	3.2
SLE index 1.00–1.50	1.1	320	1.1	1.1	.7	1.2	1.5
SLE index >1.50	.2	43	.2	.1	.1	.2	.1

Note: In Waves 2 through 5, *household member went to prison* is not measured. Frequency of this SLE in Waves 6 through 9 is 8, 1, 2, and 7, respectively. SLE = stressful life event; HH = household.

the SLE index behaves similarly in a first-differences model as in the change score approach under which its coefficient is 1 by construction. Here, the coefficient is 1.10, indicating that a unit change represents approximately the amount of life stress associated on average with a one-unit increase in GHQ score in a first-differences model as well as in a change score model.

Table 2. Testing the Differential Vulnerability Hypothesis: Depressive Symptoms (GHQ Score) Regressed on Stressful Life Event Index, Group Indicators, and Their Interactions, with Data from the UK Household Longitudinal Study.

Model	(1) FD	(2) BE	(3) BE	(4) FD	(5) BE	(6) BE	(7) FD
SLE index	1.10***		5.95***	1.04***		6.72***	.93***
	(.05)		(.17)	(.07)		(.20)	(.09)
Female		.53***	.41***				
		(.03)	(.03)				
Female × SLE index			1.04***	.10			
			(.22)	(.10)			
High education					–.63***	–.30***	–.27
					(.04)	(.04)	(.22)
Mid education					–.41***	–.26***	–.18
					(.03)	(.04)	(.16)
High education × SLE index						–1.77***	.36**
						(.31)	(.13)
Mid education × SLE index						.22	.19
						(.26)	(.11)
Constant		1.59***	1.29***		2.27***	1.75***	
		(.02)	(.02)		(.03)	(.03)	
N (observations)	140,487	182,605	182,605	140,487	182,266	182,266	140,226
N (individuals)	33,202	33,202	33,202	33,202	33,139	33,139	33,139

Note: Standard errors in parentheses. Reference categories are *male*, *low education*, and *low education* × *SLE index*. GHQ score ranges from 0 to 12; a higher score indicates more symptoms. SLE = stressful life event; FD = first-differences (see Equation 4); BE = between-effects (see Equation 3); GHQ = General Health Questionnaire 12-item version.
**$p < .01$, ***$p < .001$.

Differential Vulnerability by Sex

Model 2 reproduces the standard finding of sex differences in depression. The constant shows that men on average report a GHQ score of 1.59. Women report an additional .53; that is, 33% higher. Model 3 adds the SLE index and its interaction with sex in a between-individual analysis. At 5.95, the coefficient for the SLE index is far larger than in the within-individual model. There may be several reasons for this discrepancy, among them unmeasured chronic stressors that correlate with or result from SLEs. Notably, we reproduce the between-individual finding of higher vulnerability to SLEs among women reported by early work (e.g. Kessler 1979) and by Turner et al. (1995).

Model 4 shows the first-differences specification of the test of differential vulnerability by sex. Here, there is only extremely weak evidence for greater vulnerability to SLEs among women ($\beta = .10$, SE = .10). This suggests that unmeasured confounding generated the apparent differential vulnerability in Model 3.

Differential Vulnerability by Education

Model 5 evidences lower levels of depressive symptoms among those with high and medium levels of education relative to their sex and birth cohort. Model 6, like Model 3, presents a between-individual test of differential vulnerability and reproduces earlier findings that those of lower socioeconomic position are more impacted by SLEs (e.g. McLeod and Kessler 1990).

In the case of education, the differential vulnerability evident in the between-effects model not only disappears but changes sign in the first-differences model (Model 7). The increase in depressive symptoms over the course of a year associated with a given burden of SLE exposure is estimated at 39% greater for the high educated relative to the low educated. Again, it appears that unmeasured confounding generated the differential vulnerability apparent in the previous model. In Model 7, we also see imprecisely estimated decreases in symptoms associated with gaining education and moving up in the relative distribution.

Table 3. Testing the Female Vulnerability to Network Events Hypothesis: Depressive Symptoms (GHQ Score) Regressed on Indices of Stressful Life Events Occurring to Self and Stressful Life Events Occurring to Others and Their Interaction with Sex, with Data from the UK Household Longitudinal Study.

Model	(8) FD	(9) BE	(10) FD
SLE index (events to self)	1.11***	6.69***	1.09***
	(.05)	(.18)	(.08)
SLE index (events to others)	1.08***	1.96***	.84***
	(.11)	(.44)	(.15)
Female		.42***	
		(.03)	
Female × SLE index (self)		1.04***	.03
		(.24)	(.11)
Female × SLE index (others)		.43	.39
		(.60)	(.21)
Constant		1.28***	
		(.02)	
N (observations)	140,487	182,605	140,487
N (individuals)	33,202	33,202	33,202

Note: Standard errors in parentheses. GHQ score ranges from 0 to 12; a higher score indicates more symptoms. SLE = stressful life event; FD = first-differences (see Equation 4); BE = between-effects (see Equation 3); GHQ = General Health Questionnaire 12-item version.
***$p < .001$.

The Female Vulnerability to Network Events Hypothesis: Disaggregating Events Occurring to the Self and to Others

The FVNE hypothesis is a limited version of the hypothesis of differential vulnerability by sex, according to which women suffer more distress on average in response to SLEs occurring to close friends and family. We test this by creating separate versions of the SLE index based on events occurring to the self and events occurring to household members. The coefficients used in this construction are still those shown in Figure 1, but whereas the main SLEI sums the expected change in GHQ from all events, the two new indices each sum the expected change from the relevant events only.

Table 3 shows the results. We present a series of models analogous to Models 1, 3, and 4 in Table 2. The main coefficient of interest is the interaction in Model 10 between sex and events to others. There is some indication of the positive interaction predicted by the hypothesis ($\beta = .39$), but the p value is .059. This tentative evidence contrasts with the clear zero interaction between events to self and sex, suggesting that if there is any differential vulnerability, it is limited to the experience of SLEs occurring to others.

Robustness Checks

We have chosen to use change score and first-differences models in our main within-individual approach. A key reason we preferred these to fixed effects (FE) models is that the aim is to adjust for unobserved ongoing sources of stress (in contrast to more objectively measurable *events*), and although these are characterized in the literature as often chronic situations that persist for many years (Hammen 2016), it is more conservative to assume that they may change over the course of up to 8 years but are less likely to change over the course of 1 year. Nonetheless, there may be good reasons for preferring an FE specification. For estimation of the SLEI, FE may better capture the impact of an event in the presence of anticipation effects, such as may precede a union dissolution or some economic and health problems. By estimating deviations from individual means, FE may also evaluate the impact of life events with respect to a more reliably measured "baseline" GHQ than does FD, which takes previous year's GHQ as the baseline.

Appendix Figure S6 in the online version of the article shows the coefficients from a multivariate FE model estimating the impact of each SLE, which are generally slightly larger than those shown in Figure 1. These are used to construct an FE-based version of the SLE index. This is then used in FE

models presented in Appendix Tables S1 and S2 in the online version of the article, replicating our main analyses. Results are very similar, with two minor divergences of note. First, Model 7 from Appendix Table S1 shows that under FE, the reversal of differential vulnerability by education is less pronounced. The high education interaction with SLEI is positive but smaller (β = .13 vs. .36 in Table 2) and no longer significant (p = .100), although the interaction with the medium-education term is positive and marginally significant under this specification (p = .048). Second, Model 10 in Appendix Table S2 in the online version of the article finds a coefficient of similar size for the test of the FVNE hypothesis (β = .37 vs. .39 in Table 3), but under FE, this is statistically significant (p = .017).

Although the first-differences approach adjusts for the potentially confounding influence of circumstances that do not vary over the course of 1 year, this cannot fully account for the role of chronic stressors because these too exhibit some degree of temporal variation through onsets, endings, and changes of intensity. In this regard, it should be noted that the onset of chronic stressors is often an indirect pathway by which SLEs exert their effects. For example, union dissolution may precipitate a move to a more stressful neighborhood, the loss of friends, and single-parent status (Leopold 2018; McDermott, Fowler, and Christakis 2013). As Wheaton and colleagues (2013:311) put it, chronic stress (inter alia) "nicely describes the stressful situations that proceed from the sudden onset of major life events that are not resolved." Chronic stressors may play a mediating as well as a confounding role.

Importantly, this implies that measuring and adjusting for chronic stressor onsets to some extent means conditioning on posttreatment variables and thereby obscuring the effects of SLEs. Nevertheless, in Appendix Tables S3 and S4 in the online version of the article, we describe and present models in which we include a set of seven chronic stressor measures as covariates. Among these are subjective financial situation and job satisfaction. The results are highly similar except that the estimate of interest for the FVNE hypothesis is moderately attenuated (β = .27 vs. .39 in Table 3). However, this may be because of increases in chronic stressors caused by others in the household suffering SLEs. Specifically, for women, male partners' SLEs such as job loss and health problems may disproportionately affect household income and thus financial strain because men are more likely to be the sole or majority wage earner in the contemporary UK. Appendix Table S5 in the online version of the article reports regressions of our chronic stressor measures on SLE indices for events to self and events to others interacted with sex. These results confirm that for women in particular, SLEs to others are associated with deteriorations in subjective financial situation. This, then, is one mechanism underlying our FVNE finding. More generally, it suggests that controlling away these chronic stressor pathways is not desirable or at least depends on an assumption that chronic stressors play a more important confounding than mediating role.

The chronic stressor measures we use in this robustness check are described in the Appendix in the online version of the article. They are far from comprehensive and therefore leave open the possibility of confounding by unmeasured time-varying factors. However, such factors would need to exhibit a very particular constellation of sex-specific and educationally stratified associations to explain away our findings.

DISCUSSION

Differential vulnerability refers to the idea that a given stressor may have a greater psychological impact on individuals with certain demographic characteristics. This has been proposed as a mechanism behind inequality in mental health outcomes between men and women and across levels of education.

We identify three major problems with existing evidence testing for differential vulnerability by sex and education: first, the use of problematic checklist instruments that in many cases do not give objective criteria for the measurement of stressors. These leave interpretation of the definition of events such as "serious illness" up to respondents. This introduces error and is prone to confounding by the effect a given event has on the respondent, among other issues. Second, in attempting comprehensive measurement of all types of stressors, prior research has explicitly embraced subjective measures that overlap theoretically and empirically with the dependent variable of psychological distress. This represents a trade-off of specificity for sensitivity in measuring stressors that is appropriate for some purposes. For testing the differential vulnerability hypothesis, however, it is undesirable. The third issue is that prior work has used the simple sum of reported stressors, giving equal weight to types of events that differ wildly in their severity.

We contribute by testing the differential vulnerability hypothesis in a manner that overcomes these limitations using a multitopic annual

longitudinal household survey. Measures of objectively defined life events are constructed from highly specific questions. Each household member age 16 or over is interviewed separately, decoupling the measurement of events happening to others from focal respondents' perceptions and giving a strict and consistent definition of the network within which we consider events happening to others. We construct an index of stressful life events in which events are weighted by the expected within-individual change in mental health associated with them, estimated from a multivariate model adjusted for unobserved time-invariant confounders and any confounding among events. Finally, we test for differential vulnerability to the effects of stressful life events in a within-individual design by applying a first-differences model on the assumption that potential confounders, such as chronic stressors (and their effects on mental health), are generally stable over the course of a year.

Our results indicate no overall greater effect of SLEs on women compared with men. Preliminary models using only between-individual variation did indicate a substantial interaction between life event stressors and sex, vindicating the concern raised by Turner and colleagues (Turner and Avison 2003; Turner et al. 1995) that unmeasured group differences might lead to false-positive findings of differential vulnerability. When drawing only on within-individual variation, however, this interaction becomes indistinguishable from zero. As noted earlier, Turner et al. (1995) in fact reported a finding of differential vulnerability by sex; our finding suggests that theirs may be an artifact of the very sort they identify.

We observe some support, albeit sensitive to model specification, for higher vulnerability among women to events occurring to others, in line with what we term the "female vulnerability to network events" hypothesis. The point estimates from our two different specifications each suggest that a given level of exposure to events occurring to other household members leads to an increase in depressive symptoms that is close to 50% greater for women than men. Because our design drastically reduces the possibility of endogenous influences determining the set of measured events to others, we believe this is a substantially stronger test than has previously been possible of the idea that women exhibit greater vulnerability to events occurring to close others.

A theoretical contribution is that we distinguish the FVNE hypothesis from Kessler and McLeod's (1984:620) narrower "cost of caring" hypothesis, which similarly posits a greater vulnerability among women to events to others but further holds that this is due to "greater emotional involvement of women in the lives of those around them." We were not able to test this specific mechanism, but our robustness checks did raise a noteworthy point: The mechanisms behind FVNE may also include more mundane considerations, such as that because men tend to be the primary wage earner in contemporary UK households (and many other contexts), events that lead to loss of income for men may manifest in the results as a disproportionately large impact of events to others for women, with their effects mediated through loss of household income and potentially an increase in relationship conflict. Further research should seek to replicate this finding, explore the mechanisms involved, and clarify the implications for intervention.

Finally, we find no support for greater vulnerability to SLEs among those less educated than their contemporaries. In fact, we find some—again somewhat sensitive—evidence for a stronger impact on the higher educated. A possible explanation is a quasi-ceiling effect whereby lower-status individuals experience higher levels of stressors and distress overall so that the marginal impact of a further event is relatively low.

We note some limitations to our study. First, we assume that changes in depressive symptoms associated with events mostly reflect a causal impact of events, but some portion of this is likely due to reverse causality whereby mental health problems precipitate certain events, such as job losses or union dissolutions. We believe that this is plausible only for a small subset of the SLEs we measure.

Second, it should be noted that we succeed in testing differential vulnerability to stressful life events only. Further research using high-quality measurement should explore differential vulnerability to chronic stressors and early life traumas.

A third potential limitation is that the available measures miss certain important types of SLEs, such as criminal victimization or a major breach of trust by one's partner. In considering this issue, it must be borne in mind that our focus is on testing the differential vulnerability hypothesis. To invalidate our results, it would need to be the case that the sorts of SLEs we do not measure exhibit a clearly contrary pattern to those we do measure. We see no strong reason to expect this because the latter cover a broad range of life domains. Note also that unmeasured events that intuitively loom larger in the lives of women—for instance, sexual assault and harassment—may do so mainly because of differential exposure rather than vulnerability.

Fourth, we are not able to measure SLEs that occur to close others outside the household. Respondents' understandings of "closeness" may pose an endogeneity problem, as discussed. Even so, it is noteworthy that in the limited research literature on FVNE, we do find the result—in a survey of 720 individuals from the late 1960s—that this differential vulnerability was limited to network events occurring to others *outside* the household (Kessler and McLeod 1984). It is therefore possible that our estimate of greater vulnerability among women to events to others represents a lower bound. Further research might explore this question with a design in which respondents prespecify others outside their household whom they care about or consider close, with follow-up measures of SLEs and mental health outcomes.

Fifth, our data would ideally include diagnostic instruments to assess whether individuals meet clinical thresholds for disorder. Because the GHQ is an effective screening instrument for a wide range of clinical diagnoses (Goldberg et al. 1997) and prior research has shown a high correlation between distress and disorder (Payton 2009), we believe our results are at least somewhat informative about clinically relevant mental illness as well as psychological distress more generally.

The main implication of our findings is that differential vulnerability to SLEs is unlikely to play a major role in the generation of mental health inequality by education, but vulnerability to SLEs occurring to friends and family may be implicated in sex differences in mental health outcomes. Testing whether differential exposure and vulnerability to stressors explain between-group inequality will require measurement of as wide as possible a range of stressors and must be sensitive to the issues raised in the critique presented here.

We have argued that measures of chronic stressors that are endogenous to psychological distress may be suitable for the description of inequalities but that they are not a valid basis for testing causal mechanisms that may generate these inequalities. Yet the inclusion of these chronic stressor measures has been central to two of Turner and colleagues' main *causal* claims about stress, mental health, and inequality, summarized as the first two headline findings in Thoits's (2010:S42–S43) review: "Finding 1: With more comprehensive stress measurement, the impacts of stressors on health are substantial" and "Finding 2: Exposure to stress is unequally distributed in the general population and fosters inequalities in physical and psychological well-being."

Finding 2 is based on Turner and colleagues' (1995:106) argument that if chronic stressors are more prevalent among disadvantaged social groups (as they find) but are left unmeasured, the resulting unmeasured differences may "parade within research findings as vulnerability differences" when in fact, inequalities of *exposure* are the key mechanism. Against this, however, results utilizing measures of stressors that are endogenous to distress are biased in favor of indicating that differential exposure to stressors generates inequalities in distress: Groups with higher levels of distress will, by construction of the measures, report higher exposure to chronic stressors. Future research must, so far as is possible, evaluate the extent to which these headline findings derive from confounded measurement.

Elements of our critique apply beyond the particular articles on which we have focused. One way is that the same measurement instruments have been employed in other surveys, producing questionable results. McDonough and Walters (2001: 553) report that "[s]ocial life stress emerging from the pressures and expectations of others was overwhelmingly the most important predictor" of distress in their sample. "Social life stress" is constructed as the sum of five chronic stressor items, including "You are trying to take on too many things at once" and "Too much is expected of you by others." In addition, more recently created measurement instruments face similar issues. For instance, the Trier Inventory for Chronic Stress includes items likely to measure distress, such as "Sometimes I am consumed by my worries," alongside others more oriented toward stressors, such as "I have no opportunity to discuss things with others" (Petrowski et al. 2018:3).

Our findings highlight that apparent differential vulnerability when adjusting on observables may in fact reflect unmeasured differences in exposures between groups, as argued by Turner and colleagues (Turner and Avison 2003; Turner et al. 1995). This may then also raise concerns for the physical health literature reporting differential vulnerability cited earlier in the article (e.g., Katikireddi et al. 2017; Veronesi et al. 2017). These studies typically involve between-individual designs, measuring a risk factor once at baseline and then linking records to administrative health data to observe hospital admissions and mortality. Outcomes such as cirrhosis, stroke, and mortality are clearly difficult to incorporate into a within-individual design, but research might consider repeated measures of both exposures (e.g., alcohol

intake, smoking) and biomarkers that are both upstream of disease states and reversible.

This study represents a proof of concept for using UKHLS and potentially other household surveys in life stress studies. Almost all other empirical studies of human life stress come from far smaller, mostly cross-sectional surveys using checklists or an intensive interview similar to the LEDS. Further use of alternative data sources may provide more insights into the stratification of stress and well-being.

Finally, our main finding of no overall differential vulnerability by sex concerns men's mental health as well as women's. In spite of prior findings and theoretical reasons to expect greater vulnerability among women (Rosenfield and Mouzon 2013), we find that SLEs are associated with men's depressive symptoms just as much as women's. Practitioners and policymakers concerned with mental health may find this a striking framing, especially in a context of rising male suicide rates in the United Kingdom and United States (Hedegaard, Curtin, and Warner 2020; Office for National Statistics 2020) and increasing public concern around male norms of stoicism and silence.

ACKNOWLEDGEMENTS

The authors would like to thank members of the Oxford Sociology Monday Meeting Group and three anonymous reviewers for helpful comments.

FUNDING

The author disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was conducted as part of the CRITEVENTS project (Critical Life Events and the Dynamics of Inequality: Risk, Vulnerability, and Cumulative Disadvantage). CRITEVENTS is part of the New Opportunities for Research Funding Agency Cooperation in Europe (NORFACE) Joint Research Programme on the Dynamics of Inequality Across the Life-Course (DIAL), which is co-funded by the European Commission through Horizon 2020 under grant agreement 724363. Christiaan Monden also gratefully acknowledges support from the Leverhulme Trust.

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NOTES

1. Unstated is the further assumption that unmeasured chronic stressors correlate with stressful life events at the individual level.
2. According to Google Scholar. Also, this review does not mention Grzywacz and colleagues' (2004:11) study of daily stressors, or "hassles," which found that "better-educated individuals' physical and mental health were influenced less by daily stressors (i.e., they were less vulnerable)."
3. Lifetime traumas are (extreme) events, such as experiencing or witnessing violence or sexual abuse, that have ever occurred to the respondent during adulthood, whereas SLEs are usually conceptualized as recent events.
4. We do not apply a fully first-differenced model here (i.e., including instead of $\boldsymbol{\beta}_k \boldsymbol{SLE}_{kit}$ a term for *change* in whether each SLE occurs: $\boldsymbol{\beta}_k[\boldsymbol{SLE}_{kit} - \boldsymbol{SLE}_{ki,t-1}]$) because this would involve an unnecessary assumption that GHQ returns fully to normal in the following year, when $SLE_{it} - SLE_{i,t-1} = -1$ for that particular event. (Each SLE rarely recurs in consecutive waves.)
5. In the following, we also discuss and present results from a fixed effects specification.

SUPPLEMENTAL MATERIAL

The Appendix, Figures S1 through S6, Tables S1 through S5, and a reproduction package (materials to reproduce our results) are available in the online version of the article.

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