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Cumulative stress: A general "s" factor in the structure of stress

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ABSTRACT

Objective: The present study tested a hierarchical model of cumulative stress in a large probability sample of adults from the United States.
Methods: Exploratory factor analysis (EFA) and confirmatory factor analysis (CFA) models were used to develop and test a hierarchical model of cumulative stress. Structural equation models were used to estimate concurrent associations with demographic factors, polygenic risk scores, and physical health outcomes, as well as prospective associations with physical health outcomes.
Results: A hierarchical model of cumulative stress was the best-fitting model, with a general "s-factor" capturing the tendency for subordinate dimensions of stress to correlate. Associations with demographic factors and polygenic risk scores for physical and psychological phenotypes provide evidence for the convergent validity of a general s-factor of cumulative stress. The general s-factor and subordinate factors of cumulative stress were also associated with physical health outcomes, concurrently and prospectively, including number of chronic conditions, body mass index, and difficulty with activities of daily living.
Conclusions: Like other human individual differences, the co-occurrence of social stressors can be understood using a hierarchical model.

1. Introduction

1.1. The structure of cumulative stress

Psychosocial stressors—internal or external stressful events that cause physiological or psychological response and trigger a disruption in homeostasis—can have a major influence on physical health (Schneiderman et al., 2005). The failure to cope with acute stressors (e.g., natural disasters or social stressors like the death of a loved one) can have prolonged and impairing consequences to immune responses, which can lead to poor health, including cardiovascular disease (Dimsdale, 2008; Hokimoto, 2018) and post-traumatic stress disorder (Bountress et al., 2020; Raker et al., 2019). In addition, stressors that induce repeated or continuous activation of stress responses are thought to be the most potent as they can lead to permanent physiological and psychological changes that give rise to tissue damage and disease (Schneiderman et al., 2005; Cohen et al., 2007). Researchers have long sought to identify the key psychosocial stressors that pose threat to public health and contribute to health disparities (Dimsdale, 2008; Cohen et al., 2007; Slavich, 2016). Traditionally, research has focused on single stressors in isolation, but this is likely to underestimate and mischaracterize the impact of stress on physical health because a single stressor is rarely experienced in isolation of other stressors. Therefore, research has begun to shift focus to multiple or repeated stressors (i.e., cumulative stress; Sternthal et al., 2011).

1.2. The measurement and structure of cumulative stress

Given that many psychosocial stressors often coexist in individuals' lives, researchers are beginning to capture the cumulative impact of multiple stressors on physical health (Burroughs Peña et al., 2019; Slavich, 2016; Slopen et al., 2018). Growing research has shown that exposure to multiple stressors, or repeated exposure to the same stressor, exceeds the detrimental health consequences of a single exposure (Evans and Kim, 2010; Evans et al., 2013; Slopen et al., 2018; Turner and Lloyd, 1995; Turner et al., 1995). Drawing from life course epidemiology principles and models (Slopen et al., 2018), a common strategy to evaluate the impact of cumulative stress on health is to create a

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composite index by summing indicators for multiple stressors, or recurrence of the same stressor, into a summary score. Using this strategy, studies show that the accumulation of multiple stressors is associated with sleep problems (Slopen and Williams, 2014) and chronic health conditions (Albert et al., 2017; Burroughs Peña et al., 2019; Slopen et al., 2018).

Typically, composite scores weigh all environmental risk factors the same and therefore eschew assumptions about the relative strength of different risk factors. Further details on the calculation of unit-weighted composite scores to operationalize cumulative stress are described elsewhere (Slopen et al., 2018; Sternthal et al., 2011). Some researchers suggest that documenting the quantity of exposure, in addition to the type of exposure, may have greater utility in identifying associations with physical health (Shields and Slavich, 2017). The assumptions among researchers are that using composite scores provides (a) robust prediction of a wide array of health outcomes, (b) parsimonious modeling with greater statistical power, given the composite score is one independent predictor rather than multiple collinear variables, and (c) ease of interpretation for laypersons and policymakers (Evans et al., 2013; Slopen et al., 2018).

Despite these advantages, composite scoring also has its disadvantages. For one, interpreting findings with the goal of developing or improving prevention and intervention programs can become perplexing and difficult—researchers may not be able to address all environmental stressors in a single program. Programs may be more efficacious if a subdimension of stress or specific "target" is identified, particularly in a manner that is generalizable across developmental stages or health outcomes. Composite scores also lack face validity because unit-weighted summary scores assume that the variable being measured is unidimensional. However, acknowledging the multidimensional nature of stress has become common in stress research (Evans et al., 2013; Slavich, 2016; Slopen et al., 2018). In addition, by creating a composite score, unsystematic measurement error is compounded, potentially decreasing predictive accuracy and, in turn, failing to identify those who are at risk of experiencing deleterious physical health outcomes. Finally, by creating a composite score, the correlated structure of the experience of multiple stressors is lost and every stressor is treated as an equally sound indicator of cumulative stress.

The first step to overcoming these limitations is to adopt a factor analytic approach, which partitions the covariances among many individual measures into a smaller number of dimensions that capture variance that is common to subsets of related measures. When these empirically derived dimensions of covariation are themselves correlated, then the relations among the individual measures might be effectively organized and understood within a hierarchical taxonomy. A hierarchical taxonomy provides an organizational structure, whereby more specific or subordinate dimensions or taxa are subsumed by or related to one or more superordinate dimension or taxon that is more broad or general. Although more analytically complex than calculating a unit-weighted composite score, such an approach has been fruitful in delineating the phenotypic structure, developmental course, and correlates of many human individual differences, including cognition (Carroll, 2003), personality (Chang et al., 2012), and psychopathology (Kotov et al., 2017). Crucially, a factor analytic approach can help address limitations to existing approaches to measure cumulative stress.

Compared to calculating a unit-weighted summary score (mean or sum), a factor analytic approach neither weighs all stressors equally nor does it ignore the pattern of correlations among different stressors. A factor analytic approach also does not assume that cumulative stress is unidimensional. Instead, patterns of correlations among subsets of stressors are used to empirically extract one or more dimension of cumulative stress. A factor analytic approach can also begin to untangle common from residual variance among individual measures of stress, which has at least two advantages. Researchers can begin to distinguish the tendency to experience multiple dimensions of stress (i.e., cumulative stress) from subordinate dimensions of stress and individual stressors, which may exhibit varied associations with physical and mental health. Further, the compounding of unsystematic measurement error is avoided by distinguishing common from residual variance in individual measures of stress. Still, it remains an open question whether patterns of correlations among different stressors are adequately captured by a hierarchical model.

1.3. Gene-environment correlation

Although social, relational, and economic stress are associated with physical health, individuals do not play a passive role in the experience of stress. Instead, individuals take an active role is shaping and responding to their social and ecological environments, a process sometimes called "niche-picking" in personality psychology (Roberts and Nickel, 2017) or an "extended phenotype" in evolutionary biology (Dawkins, 1982). To the extent that such a process is undergirded, at least partly, by genetic factors, this results in gene-environment correlations as individual genotypes become correlated with environmental exposures through active and evocative transactions between individuals and their environments (Plomin et al., 1977; Scarr and McCartney, 1983).

The possibility that the experience of cumulative stress may not be purely environmental has been underexamined. Although polygenic risk scores do not fully capture the underlying processes that contribute to gene-environment correlations, nor do they capture the effects of indels, rare variants, epistasis, or moderating environments, polygenic risk scores can be used to provide a statistical adjustment for genetic confounds that has the potential to clarify whether associations between cumulative stress and physical health are influenced by underlying genetic factors. Significant associations between polygenic risk scores and cumulative stress would indicate that individual differences in cumulative stress are not purely environmental. Instead, individual differences in cumulative stress would be partially explained by polygenic liability for physical and psychological phenotypes. Associations that remain between cumulative stress and physical health after controlling for the effects of polygenic scores may help provide insight into the magnitude of links between cumulative stress and physical health by adjusting estimated associations for potential genetic confounds.

1.4. Goals of the present study

The present study tests a hierarchical model of cumulative stress using a large probability sample of aging adults. Different hierarchical models are compared to a model with factors scores that are a linear transformation of unit-weighted sum scores (Fig. 1;(McNeish and Wolf, 2020). We assess whether cumulative stress is unidimensional and evaluate whether a hierarchical model of cumulative stress replicates across different cohorts. We also test the convergent and predictive validity of a hierarchical model by estimating cohort differences and associations with demographic factors, polygenic risk scores, and physical health outcomes. Finally, by implementing statistical controls for polygenic liabilities for a wide breadth of psychological and physical health outcomes—and, thus, partially adjusting for active and evocative selection effects—the current study estimates the physical health correlates of a general factor of cumulative stress, dubbed "s-factor" (Caspi et al., 2014; Lahey et al., 2012; Lubinski, 2004), as well as specific dimensions of cumulative stress, before and after adjusting for potential genetic confounds.

2. Methods

2.1. Sample

The present study analyzed data from the Study of Midlife Development in the United States (MIDUS) (Ryff and Krueger, 2018). Data are available on the MIDUS Colectica portal (http://midus.colectica.org).

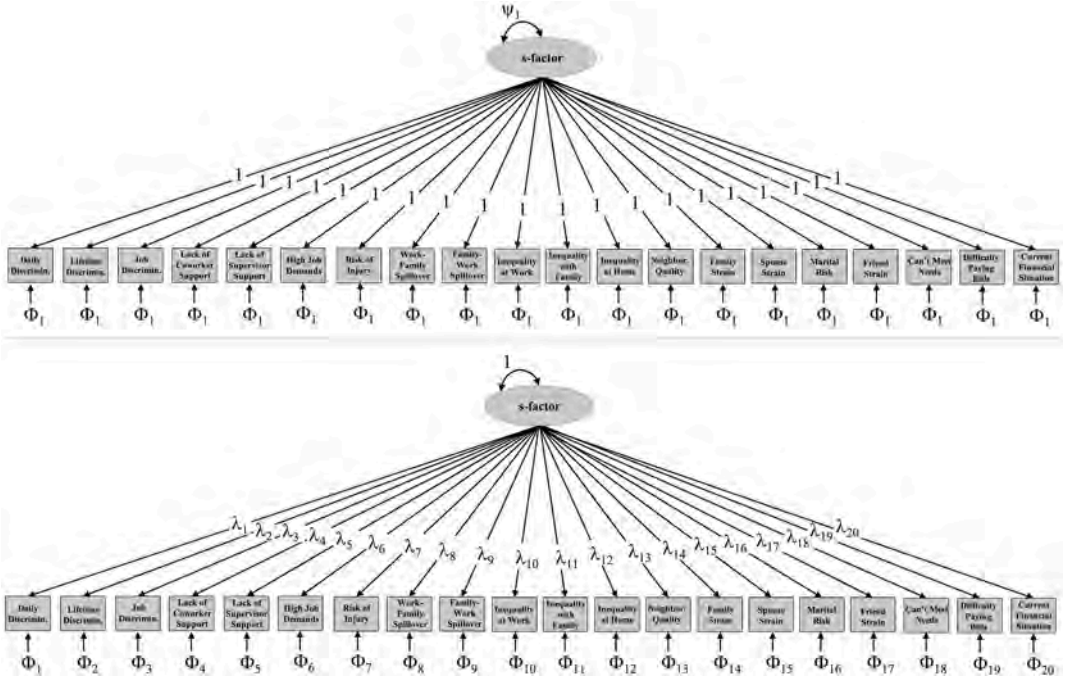

Fig. 1. Unidimensional confirmatory factor analysis models of cumulative stress.**Note.** A measurement model with factor scores that are a linear transformation of unit-weighted sum scores is depicted on the top panel. An unrestricted unidimensional measurement model is depicted on the bottom panel.

Analysis scripts are available on the Open Science Framework (BLIND). For information regarding participant recruitment and data collection, see (Ryff and Krueger, 2018). MIDUS implemented a multiple cohort, cross-sequential design. The present study analyzes data from the MIDUS-II, MIDUS Refresher, and MIDUS III cohorts, where data were available for measures of social, relational, and financial stress, polygenic risk scores, and physical health outcomes. Data from the MIDUS-II cohort was collected from 2004 to 2005 during a time of considerable economic growth in the U.S., while data collection for the MIDUS Refresher cohort began in 2011 during a period of economic unease, following the 2008–2009 recession. Consequently, the MIDUS-Refresher cohort provides an excellent resource to not only test the replicability of findings but also the generalizability of findings across different economic periods.

To conduct exploratory and confirmatory analyses, data from the MIDUS-II cohort were randomly split into approximately equally sized training (n = 2007) and hold-out samples (n = 2008). To assess the generalizability of findings, confirmatory analyses were also conducted using data from the MIDUS-Refresher cohort (n = 2577). Cohort differences and demographic correlates were estimated using data from the MIDUS-II and Refresher cohorts (n = 6592). Models that included the effects of polygenic risk scores on cumulative stress and physical health were estimated using data from the Biomarker cohort (n = 1281), which includes a subsample of participants from the MIDUS-II and Refresher cohorts. Prospective associations between cumulative stress and physical health measured approximately a decade later were estimated using data from the MIDUS-II (n = 4015) and MIDUS-III cohorts (n = 2700; ~67% retention), as well as a subsample of participants from the MIDUS-III cohort who were genotyped in the biomarker cohort (n = 725). After removing cases with >50% missing data for demographic variables and measures of stress, full-information maximum likelihood was used in *Mplus*(Muthén and Muthén, 2017) to estimate models using all available observations. Sensitivity analyses were also conducted using diagonally weighted least squares with mean and variance adjustments (see Open Science Framework for results). Sample characteristics were similar across cohorts and subsamples, including age, sex, race/ethnicity, and level of education (Table S1).

2.2. Measures

Demographic Factors and Polygenic Risk Scores. Demographic factors included chronological age, self-reported sex (female and male), level of education, and self-reported race/ethnicity (White, Black, and Other Race/Ethnicity). A dozen polygenic risk scores (PRSs) were included in analyses that are putatively relevant to psychological and physical health and of interest to clinicians and epidemiologists. PRSs for the following phenotypes were included: Alzheimer's disease, autism spectrum disorder, body mass index (BMI), educational attainment, high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides, type-II diabetes, neuroticism, anxiety, post-traumatic stress disorder, and schizophrenia. Polygenic risk scores were calculated using all available single nucleotide polymorphisms (SNPs), including the additive effects of all measured and imputed SNPs. Details on genotyping, imputation, and polygenic risk scoring are reported elsewhere (Martin, Mann and Krueger, 2020) and can be found in documentation on the MIDUS Colectica portal.

Physical Health. Four physical health outcomes were included in the analyses: self-reported number of chronic physical conditions, body mass index (BMI), basic activity of daily living (e.g., "Bathing or dressing yourself"), and intermediate or moderate activity of daily living (e.g., "Vigorous activities [e.g., running, lifting heavy objects]). Basic and moderate activity of daily living were coded such that higher values reflect greater difficulty completing activities.

Environmental Stress. Twenty self-reported environmental and psychosocial stressors were included in analyses: daily discrimination, lifetime discrimination, job discrimination, lack of coworker support, lack of supervisor support, high job demands, risk of accident or injury at work, work-family spillover, family-work spillover, inequality at work, inequality with family, inequality at home, poor neighborhood quality, family strain, spouse strain, marital risk, friendship strain, not enough money to meet one's needs, difficulty paying monthly bills, and a subjective assessment of one's current financial situation. Descriptive statistics for study variables are reported in supplemental material (Tables S1 & S2), as are detailed descriptions including sample items and references. Additional information can also be found on the MIDUS

Colectica portal, including Cronbach's alpha (*range* of $\alpha = 0.64$ to 0.92, *mean* = 0.78, *median* = 0.78).

2.3. Data analytic procedures

Data analytic procedures are summarized in Figures S1 & S2 in supplemental material. Data was imported into R Studio version 1.3.1056 (Allaire, 2012) processed and then exported using the 'MplusAutomation' package version 0.7.1 (Hallquist and Wiley, 2018). Analyses were conducted using Mplus version 8.1 (Muthén and Muthén, 2017) and using the 'EFAtools' package in R Studio (Steiner and Grieder, 2020). MIDUS includes a subset of siblings and twin-pairs. Therefore, a family identification number was included as a cluster variable when conducting analyses in Mplus. This implements a sandwich estimator to adjust standard errors for the non-independence of observations that results from subsets of siblings being nested within the same family. Using the full analytic sample ($n = 6592$), we calculated parameter and non-parameter zero-order correlations between stressors (Table S3). Next, using the training sample ($n = 2007$), we calculated Bartlett's test of sphericity and the Kaiser-Meyer-Olkin (*KMO*) criterion. A statistically significant Bartlett's test ($p < .05$) and a *KMO* index greater than 0.60 indicate that the data are generally suitable for factor analysis.

2.4. Exploratory factor analysis

To examine dimensions of covariation among measures of stress, an exploratory factor analysis (EFA) was conducted in MPlus using the default oblique Geomin rotation, and also using the 'EFAtools' package in R Studio. Consistent with recent methodological recommendations (Auerswald and Moshagen, 2019), the best-fitting factor solution was determined using sequential model tests, the empirical Kaiser criterion (Braeken and Van Assen, 2017), and by examining scree plots from the EFA and parallel analysis (Horn, 1965). A scree plot is a line plot of the eigenvalues for the sample correlation matrix ordered from largest to smallest. Traditionally, the number of latent factors retained in an EFA is equal to the number of eigenvalues before the scree plot breaks or plateaus, although this criterion is subjective and sometimes difficult to discern. In a parallel analysis, an EFA is conducted on samples of random data with the same numbers of variables and observations that were used to estimate the sample correlation matrix. The suggested number of factors to retain is equal to the number of eigenvalues for the sample correlation matrix that exceed the average or 95th percentile of eigenvalues for matrices of random data (Auerswald and Moshagen, 2019; Montanelli and Humphreys, 1976). We also consulted model fit statistics to determine the optimum number of factors to retain. For each factor solution, we relied primarily on root mean squared error of approximation (*RMSEA*) and comparative fit index (*CFI*), whereby $RMSEA \leq 0.05$ and $CFI \geq 0.90$ indicative good model fit (Hu and Bentler, 1999).

2.5. Confirmatory factor analysis

Informed by the results of the EFA, confirmatory factor analysis (CFA) models were used to test the relative fit and replicability of different hierarchical models (higher-order and bifactor), compared to an unrestricted unidimensional model and to a unit-weighted summary score (McNeish and Wolf, 2020), similar to those used in contemporary research to operationalize cumulative stress. These models are depicted in Fig. 1. In the "sum score equivalent" model, all factor loadings were fixed to one and the residual variances of indicators were constrained to equality. In the less restrictive unidimensional model, all factor loadings and residual variances were freely estimated with no equality constraints. The higher-order and bifactor models, on the other hand, do not assume that cumulative stress is unidimensional. Instead, the selection of indicators for multiple latent factors was theoretically and empirically motivated. Empirically, the pattern and strength of standardized factor loading from the preferred EFA model were used to inform the selection of factor indicators. Theoretically, the face validity and conceptual relations between measures were also considered.

As reviewed by Markon (2019), higher-order and bifactor models are examples of hierarchical models that yield different interpretations of a general "s-factor" of cumulative stress. These models are depicted in Fig. 2. With the higher-order model, the general s-factor represents the tendency for multiple dimensions of cumulative stress to correlate. With the bifactor model, the general s-factor is theoretically primary to subordinate dimensions of cumulative stress, which capture residual variance in measures of stress that are not explained by the general factor (Markon, 2019). So, with the bifactor model, multiple dimensions of cumulative stress are orthogonal to each other and the general s-factor, capturing distinct dimensions of covariation among stressors. To capture the general tendency for dimensions of cumulative stress to co-occur, on the other hand, a higher-order model provides a suitable interpretative framework to operationalize cumulative stress. Nonetheless, at the request of a reviewer, bifactor models were fit to the data and included in comparisons as an alternative hierarchical model.

2.6. Concurrent and predictive validity

After testing the replicability and generalizability of a hierarchical model of cumulative stress, concurrent and predictive validity was assessed with a series of extended CFA models using data from the full analytic sample, including data from both MIDUS-II and MIDUS-refresher cohorts ($n = 6592$). Not only does combing data across cohorts increase power to detect demographic differences but also facilitates an additional test for the validity of a hierarchical model, as cumulative stress at the population-level should be higher during a period of economic uncertainty, compared to a period of economic growth. Therefore, a CFA model was estimated, whereby the general "s-factor" was regressed on demographic factors including a dummy-coded variable for cohort. Next, using data from the biomarker subsample ($n = 1281$), this model was extended to include additional predictors of the general s-factor, including the first five genetic principal components and multiple polygenic risk scores for an array of physical and mental health outcomes. These regressions tested whether demographic factors and genetic liability for psychological and physical health outcomes were associated with cumulative stress.

Next, using data from the MIDUS-II cohort ($n = 4015$), CFA models were used to test whether general and subordinate dimensions of cumulative stress were associated with physical health outcomes, both concurrently and prospectively. In these models, physical health outcomes (i.e., number of chronic conditions, BMI, and basic and moderate levels of physical activity) were regressed on the general s-factor and then on subordinate factors of stress in a subsequent model. Finally, using data from the biomarker subsample, the same associations were estimated, while adjusting for the effects of multiple polygenic risk scores. To control for longitudinal stability, physical health outcomes at Wave 3 were residualized for prior-levels at Wave 2 before estimating associations with stress.

The precision of estimated associations was evaluated using 95% bias-corrected bootstrapped confidence intervals calculated from 500 replicate samples using the 'BCBOOT' option in Mplus (Muthén and Muthén, 2019), and we report p-values from models with robust standard errors (MLR; Muthén and Muthén, 2019). Given the number of tests and sample sizes involved in the current study, it may be wise to place greater emphasis on the direction, size, and precision of estimated effects, instead of a binary decision to reject or retain a null hypothesis. Nevertheless, we report *p*-values for all tests, noting when effects are statistically significant ($p < .05$) after implementing a conservative Bonferroni-correction for multiple testing.

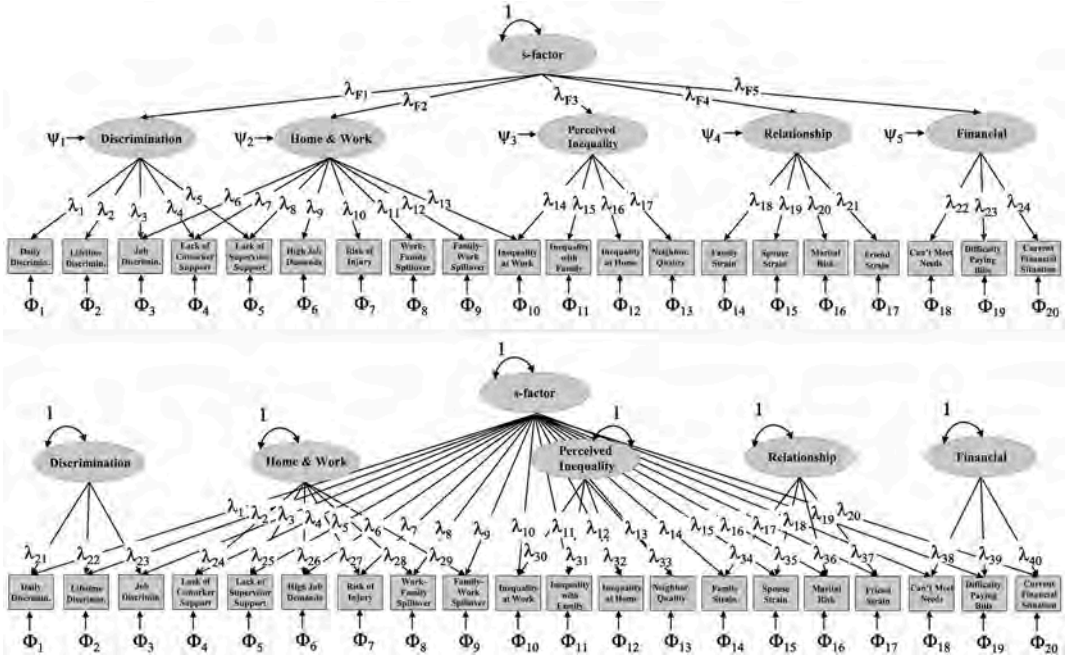

Fig. 2. Hierarchical confirmatory factor analysis (CFA) models of cumulative stress.**Note.** Path diagrams of higher-order and bifactor models are depicted on the top and bottom panels, respectively.

3. Results

3.1. Exploratory factor analysis

Bartlett's test ($\chi^2 = 11{,}488.00$, $df = 190$, $p < .001$) and *KMO* index (0.83) indicated that measures of stress were suitable for factor analysis. Depicted in supplemental material, according to the scree plot from the EFA, five factors were needed to adequately capture observed patterns of covariation among measures of stress. Similarly, the empirical Kaiser criterion indicated that five factors should be retained. According to the parallel analysis, a five-factor solution was also preferred, as the first five eigenvalues for the sample correlation matrix exceeded the 95% percentiles of eigenvalues for 100 matrices of random data (Figure S3 in the supplement).

Model fit statistics provided mixed support for a five-factor solution. Although sequential model tests based on $\Delta\chi^2$ and Akaike information criteria (*AIC*) suggested that 11 or 10 factors should be retained, these fit statistics can be overly sensitive in large samples. The lower bounds of the 90% confidence intervals for *RMSEA* suggested that 7 factors should be retained, while point estimates for *RMSEA* suggested that a minimum of 5 factors should be retained (*RMSEA* = 0.058, *CFI* = 0.913), as a more parsimonious four-factor solution exhibited poor fit to the data (*RMSEA* = 0.071, *CFI* = 0.853). Crucially, the pattern of factor loadings for the five-factor solution yielded interpretable dimensions of cumulative stress. Considering all of the above, a 5-factor solution was selected as the preferred model.

Geomin rotated loadings and correlations between latent factors from the preferred EFA model are reported in Table 1. The strongest loadings on each factor are emphasized in the interpretation of results and guided the specification of confirmatory models. The first factor predominantly captured the tendency to experience discrimination, including daily (λ = 0.67), lifetime (λ = 0.53), and job-related discrimination (λ = 0.49). The second factor captured the tendency to experience home and work-related stress, including negative family-work spillover (λ = 0.85), high job demands (λ = 0.63), and perceived inequality at work (λ = 0.55). The third factor captured the tendency to experience stress related to personal financial hardship, including difficulty paying monthly bills (λ = 0.83), not having enough money to meet one's needs (λ = 0.80), and reporting an overall poor financial situation (λ = 0.76). The fourth factor captured the tendency to report high levels of perceived inequality, including inequality at home (λ = 0.88), poor neighborhood quality (λ = 0.51), and inequality with family members (λ = 0.31). Finally, the fifth factor captured the tendency to

Table 1
Factor loadings and correlations from the preferred exploratory factor analysis (EFA) model.

Measure	Factor				
	1	2	3	4	5
Daily Discrimination	**.67**	-.10	.02	.00	.07
Life Discrimination	**.53**	-.07	.04	-.14	.02
Job Discrimination	**.49**	.23	.03	.03	.01
Lack of Coworker Support	.26	.12	.04	.11	-.10
Lack of Supervisor Support	**.35**	.25	-.09	.06	-.15
High Job Demands	.13	**.63**	-.05	-.10	.02
Risk of Accident or Injury at Work	.18	.08	.11	-.03	-.03
Negative Work-Family Spillover	.34	.07	.18	.23	-.01
Negative Family-Work Spillover	-.01	**.85**	.03	.00	.01
Inequality at Work	-.01	**.55**	.08	.04	.20
Inequality with Family	.11	-.08	.18	**.31**	.11
Inequality at Home	-.02	.00	.01	**.88**	.00
Poor Neighborhood Quality	.08	.00	-.02	**.51**	.06
Family Strain	**.35**	.08	.01	.02	.28
Spouse Strain	.02	.00	-.02	.00	**.86**
Marital Risk	.00	.01	.12	.03	**.62**
Friend Strain	**.35**	.06	-.07	.01	**.30**
Not Enough Money to Meet Needs	.01	-.02	**.80**	.00	-.05
Difficulty Paying Monthly Bills	.00	.07	**.83**	-.02	.03
Current Financial Situation	.01	-.01	**.76**	.07	.03
Factor	Correlations				
	1	2	3	4	5
1	1.00	.44	.35	.39	.36
2		1.00	.20	.23	.24
3			1.00	.43	.29
4				1.00	.32
5					1.00

Note. Geomin rotated factor loadings and correlations are reported with factor loadings ≥0.30 printed in bold font.

experience interpersonal stress, including strain with one's spouse (λ = 0.86) and marital risk (λ = 0.62), as well as strain with friends (λ = 0.30) and family (λ = 0.28). Notably, a positive manifold emerged among factor correlations (range of r = 0.20 to 0.44), indicating that when individuals score high on one dimension of stress, they tend to score high on other dimensions as well.

3.2. Confirmatory factor analysis (CFA)

Model fit statistics for CFA models are reported in Table S4. In the hold-out sample and refresher cohort, fit statistics indicated that unidimensional models were not supported by the data, including a measurement model with factor scores that are a perfect linear transformation of unit-weighted sum scores (*RMSEA* = 0.306, *CFI* = 0.000). These results are consistent with the results of the EFA, which indicated that a minimum of five factors was needed to account for observed patterns of covariation among stressors. Despite strong and statistically significant loadings (*ps* < .001) on a general factor, the higher-order model only approached conventional standards for good fit in the hold-out and validation samples. Fit statistics were slightly lower for the bifactor model. However, simulations have shown that model comparisons tend to favor bifactor models even when samples of data are generated using an alternative model (Morgan et al., 2015). Moreover, the bifactor model produced a non-positive definite residual covariance matrix in the hold-out sample, specifically a negative residual variance. In the refresher cohort, the bifactor model failed to converge, exceeding the default number of iterations (1000), with final estimates including negative residual variances. Therefore, for the higher-order model, modification indices were consulted, adding residual covariances until statistics met conventional standards for good fit (Mueller and Hancock, 2008). This resulted in the specification of five residual covariances depicted in Figure S4. These covariances indicate that the relations between these stressors were not fully accounted for by their respective latent factors. For the bifactor model, negative residual variances were fixed to zero to eliminate Heywood cases and facilitate convergence.

After adjusting hierarchical models as described above, model fit statistics indicated that the higher-order CFA model including residual covariances was preferred over alternative solutions in both the hold-out sample (*RMSEA* = 0.051, *CFI* = 0.903) and the validation sample (*RMSEA* = 0.047, *CFI* = 0.917). As the higher-order model is also more parsimonious than the bifactor model, it was selected as the preferred model and final parameter estimates from the hold-out sample, including factor loadings, intercepts, residual variances and covariances were saved for subsequent analyses. Factor loadings onto subordinate dimensions and the general s-factor were moderate-to-strong and similar in magnitude across the hold-out and validation samples. These results are reported in Table 2. The cumulative s-factor accounted for significant variance in subordinate dimensions of stress (range of R^2 = 0.26 to 0.64, *ps* < .001). After accounting for the general s-factor, there was also significant residual variance in the five subordinate dimensions of stress (range of σ^2 = 0.36 to 0.74, *ps* < .001).

Final estimates from the preferred model were carried forward to subsequent analyses for two reasons. First, by including the final estimates from the hold-out sample as fixed parameters in the validation sample, a fully saturated model with no free parameters is fit to the data, performing a highly restrictive confirmatory test. Second, when testing for concurrent and predictive validity, the specification of fixed rather than free parameters ensures that the measurement and interpretation of cumulative stress is consistent across models and samples. Strikingly, despite not estimating a single parameter, the highly restrictive higher-order model approached conventional standards for good fit in the validation sample (*RMSEA* = 0.056, *CFI* = 0.826), providing evidence for the replicability of the higher-order model across cohorts.

3.3. Concurrent and predictive validity

Reported in Table 3, a number of demographic factors were associated with the cumulative s-factor. Older adults, being male, and higher levels of education were associated with lower levels of cumulative stress, while self-identifying as Black was associated with higher levels of cumulative stress, as was identifying as another non-White race/ethnicity. There was also a notable cohort effect, such that enrollment in the MIDUS-Refresher cohort was associated with higher levels of cumulative stress. A more nuanced pattern of demographic correlates emerged for subordinate dimensions of stress. For example, compared to

Table 2
Standardized factor loadings from the preferred hierarchical confirmatory factor analysis (CFA) model.

Measure	Hold-Out Sample (n = 2008)					Validation Sample (n = 2577)				
	F1	F2	F3	F4	F5	F1	F2	F3	F4	F5
Daily Discrimination	.80	–	–	–	–	.81	–	–	–	–
Life Discrimination	.57	–	–	–	–	.58	–	–	–	–
Job Discrimination	.35	.38	–	–	–	.27	.49	–	–	–
Lack of Coworker Support	.23	.13	–	–	–	.17	.15	–	–	–
Lack of Supervisor Support	.12	.34	–	–	–	.08	.35	–	–	–
High Job Demands	–	.67	–	–	–	–	.67	–	–	–
Risk of Injury at Work	–	.25	–	–	–	–	.30	–	–	–
Negative Work-Family Spillover	–	.82	–	–	–	–	.83	–	–	–
Negative Family-Work Spillover	–	.62	–	–	–	–	.61	–	–	–
Inequality at Work	–	.12	.44	–	–	–	.22	.44	–	–
Inequality with Family	–	–	.54	–	–	–	–	.53	–	–
Inequality at Home	–	–	.70	–	–	–	–	.78	–	–
Poor Neighborhood Quality	–	–	.61	–	–	–	–	.65	–	–
Family Strain	–	–	–	.70	–	–	–	–	.56	–
Spouse Strain	–	–	–	.53	–	–	–	–	.57	–
Marital Risk	–	–	–	.56	–	–	–	–	.57	–
Friend Strain	–	–	–	.50	–	–	–	–	.44	–
Not Enough to Meet Needs	–	–	–	–	.84	–	–	–	–	.84
Difficulty Paying Monthly Bills	–	–	–	–	.77	–	–	–	–	.78
Current Financial Situation	–	–	–	–	.79	–	–	–	–	.81
	General s-Factor					General s-Factor				
	λ_{F1}	λ_{F2}	λ_{F3}	λ_{F4}	λ_{F5}	λ_{F1}	λ_{F2}	λ_{F3}	λ_{F4}	λ_{F5}
	.72	.54	.73	.80	.55	.68	.51	.77	.80	.66

Note. All estimates >0.10 are statistically significant at p < .001. Dashes indicate that the factor loading was omitted or fixed to zero.

Table 3
Effects of demographic factors on dimensions of cumulative stress.

	General s-factor			Discrimination Stress		
	β	CI.95%	*P*	*β*	CI.95%	*p*
Cohort (Refresher)	0.22	0.16, 0.28	<.001	0.10	0.04, 0.16	.001
Sex (Male)	−0.07	−0.11, −0.01	.010	0.01	−0.04, 0.06	.780
Race/Ethnicity (Black)	0.88	0.75, 1.02	<.001	1.26	1.10, 1.40	<.001
Race/Ethnicity (Other)	0.37	0.26, 0.49	<.001	0.41	0.29, 0.53	<.001
Level of Education	−0.01	−0.02, −0.01	<.001	−0.01	−0.02, −0.01	<.001
Age (years)	−0.02	−0.02, −0.02	<.001	−0.01	−0.01, −0.01	<.001
Age^2	0.00	0.00, 0.00	.429	−0.00	−0.00, −0.00	.001
	Home & Work-Related Stress			Perceived Inequality		
	β	CI.95%	*P*	*β*	CI.95%	*p*
Cohort (Refresher)	0.15	0.09, 0.21	<.001	0.28	0.21, 0.34	<.001
Sex (Male)	0.08	0.03, 0.14	.005	−0.06	−0.11, 0.00	.038
Race/Ethnicity (Black)	−0.39	−0.55, −0.21	<.001	0.63	0.50, 0.78	<.001
Race/Ethnicity (Other)	−0.09	−0.24, 0.04	.186	0.37	0.25, 0.48	<.001
Level of Education	0.00	−0.00, 0.01	.263	−0.03	−0.03, −0.02	<.001
Age (years)	−0.03	−0.04, −0.03	<.001	−0.01	−0.01, −0.01	<.001
Age^2	−0.00	−0.00, −0.00	<.001	0.00	0.00, 0.00	<.001
	Relationship Stress			Financial Stress		
	β	CI.95%	*P*	*β*	CI.95%	*p*
Cohort (Refresher)	−0.02	−0.09, 0.04	.483	0.30	0.25, 0.35	<.001
Sex (Male)	−0.15	−0.20, −0.09	<.001	−0.04	−0.09, 0.01	.083
Race/Ethnicity (Black)	0.38	0.23, 0.53	<.001	0.59	0.47, 0.72	<.001
Race/Ethnicity (Other)	0.16	0.04, 0.29	.017	0.24	0.14, 0.35	<.001
Level of Education	0.01	0.01, 0.02	<.001	−0.02	−0.03, −0.01	<.001
Age (years)	−0.02	−0.02, −0.02	<.001	−0.01	−0.01, −0.01	<.001
Age^2	−0.00	−0.00, −0.00	<.001	0.00	0.00, 0.00	.022

Notes. β = standardized multiple regression coefficient. CI.95% = 95% bias-corrected bootstrapped confidence interval. P-values are reported from models that were estimated using maximum likelihood with robust standard errors (MLR); Effects are statistically significant ($p < .05$) after a Bonferroni-correction if $p < .001$.

self-identifying as White, self-identifying as Black was associated with higher levels of discrimination and lower levels of home and work-related stress. Being male as opposed to female was associated with higher levels of home and work-related stress and lower levels of relationship stress.

Controlling for demographic factors, the first five genetic principal components, and an array of polygenic risk scores for mental and physical health outcomes, polygenic risk scores for autism spectrum disorder were associated with higher levels of cumulative stress (β = .09 [0.03, 0.15], $p = .002$). On the other hand, polygenic risk scores for elevated high-density lipoprotein (HDL) cholesterol were marginally associated with lower levels of cumulative stress (β = −0.07 [-0.14, −0.02], $p = .024$), but this association was not statistically significant after implementing a Bonferroni-correction for multiple testing. Polygenic risk scores for educational attainment were associated with fewer difficulties with basic activities (β = −0.11 [-0.18, −0.05], $p < .001$) and intermediate activities of daily living (β = −0.09 [-0.15, −0.04], $p = .002$), and polygenic risk scores for BMI (β = 0.22 [0.16, 0.27], $p < .001$) and type-II diabetes (β = 0.07 [0.02, 0.13], $p = .012$) were associated with higher BMI. However, the effect of the polygenic risk scores for type-II diabetes on BMI did not "survive" a Bonferroni-correction for multiple comparisons. The effects of the remaining polygenic scores on cumulative stress and physical health outcomes were estimated with comparatively low precision, such that 95% bootstrapped confidence intervals included zero. These results are depicted in the supplement (Figures S5 & S6). Together, after accounting for the effects of demographic factors, the percent of variance in BMI explained by genetic principal components and polygenic risk scores was limited ($\Delta R^2 = 0.066$), similar to number of chronic conditions ($\Delta R^2 = 0.017$) and difficulty with basic activities ($\Delta R^2 = 0.024$) and intermediate activities of daily living ($\Delta R^2 = 0.017$).

Associations between the cumulative s-factor and physical health outcomes are reported in Table 4. Adjusted for the effects of demographic factors, the cumulative s-factor was concurrently related to more chronic conditions, higher BMI, and greater difficulty performing basic and intermediate activities of daily living. Controlling for the same demographic factors, as well as prior levels of physical health to adjust for longitudinal stability, the cumulative s-factor *prospectively* predicted, nearly a decade later, more chronic conditions and greater difficulty performing basic and intermediate activities of daily living. Controlling for the effects of demographic factors on physical health, subordinate dimensions of stress were differentially associated with physical health outcomes.

Reported in Table 4, accounting for the common variance among multiple dimensions of stress, the discrimination and financial factors were concurrently associated with higher BMI and greater difficulty completing basic and intermediate activities of daily living. Moreover, the residual variance in relationship stress prospectively predicted greater difficulty with activities of daily living nearly a decade later. Finally, although estimated with comparatively less precision, given the smaller subsample of participants with genotype data, the associations between dimensions of cumulative stress and physical health were similar in magnitude after adjusting for potential genetic confounds by regressing physical health outcomes on the first five genetic principal components and polygenic risk scores. These results are reported in supplemental material (Table S5).

4. Discussion

The present study tested a hierarchical model of cumulative stress. Results indicate that, similar to other human individual differences, cumulative stress can be understood in a hierarchical fashion, whereby multiple correlated dimensions of stress can be captured by a single higher-order factor, which accounts for the general tendency of these dimensions to correlate. Notably, even after implementing controls for demographic factors and potential genetic confounds using multiple

Table 4
Effects of cumulative stress on physical health outcomes.

	Concurrent Chronic Conditions			Prospective Chronic Conditions		
	β	CI.95%	*p*	*β*	CI.95%	*p*
General s-factor	0.37	0.33, 0.42	<.001	0.18	0.12, 0.24	<.001
Discrimination	0.11	0.04, 0.17	.001	0.06	−0.02, 0.14	.107
Home & Work	0.13	0.06, 0.19	.001	−0.01	−0.09, 0.08	.782
Perceived Inequality	0.09	0.02, 0.16	.015	0.05	−0.03, 0.14	.237
Relationship	0.06	−0.02, 0.14	.083	0.05	−0.04, 0.14	.279
Financial	0.08	0.04, 0.13	<.001	0.05	0.00, 0.11	.058
	Concurrent Body Mass Index			Prospective Body Mass Index		
	β	CI.95%	*p*	*β*	CI.95%	*P*
General s-factor	0.21	0.16, 0.26	<.001	0.02	−0.04, 0.08	.505
Discrimination	0.14	0.06, 0.20	<.001	−0.01	−0.10, 0.06	.719
Home & Work	0.02	−0.03, 0.08	.491	−0.02	−0.10, 0.06	.583
Perceived Inequality	0.04	−0.02, 0.11	.274	−0.08	−0.15, −0.01	.061
Relationship	−0.04	−0.11, −0.03	.256	0.09	−0.00, 0.17	.057
Financial	0.10	0.06, 0.14	<.001	0.04	−0.01, 0.09	.107
	Concurrent Basic Activity			Prospective Basic Activity		
	β	CI.95%	*P*	*β*	CI.95%	*P*
General s-factor	0.32	0.28, 0.36	<.001	0.13	0.07, 0.18	<.001
Discrimination	0.11	0.05, 0.17	<.001	−0.06	−0.13, 0.00	.086
Home & Work	0.13	0.06, 0.21	.001	0.01	−0.07, 0.09	.839
Perceived Inequality	0.12	0.06, 0.18	<.001	0.05	−0.03, 0.12	.163
Relationship	−0.07	−0.14, 0.00	.066	0.09	0.02, 0.19	.044
Financial	0.12	0.07, 0.16	<.001	0.06	0.01, 0.11	.010
	Concurrent Intermediate Activity			Prospective Intermediate Activity		
	β	CI.95%	*p*	*β*	CI.95%	*P*
General s-factor	0.35	0.31, 0.38	<.001	0.11	0.05, 0.16	<.001
Discrimination	0.12	0.07, 0.17	<.001	−0.06	−0.12, 0.01	.119
Home & Work	0.15	0.09, 0.21	<.001	−0.00	−0.07, 0.07	.903
Perceived Inequality	0.09	0.04, 0.15	.001	0.04	−0.05, 0.10	.335
Relationship	−0.06	−0.12, 0.00	.076	0.10	0.00, 0.18	.020
Financial	0.14	0.10, 0.18	<.001	0.05	−0.00, 0.10	.080

Notes. β = standardized multiple regression coefficient. CI.95% = 95% bias-corrected bootstrapped confidence interval. P-values are reported from models that were estimated using maximum likelihood with robust standard errors (MLR); Effects are statistically significant ($p < .05$) after a Bonferroni-correction if $p < .001$.

polygenic scores and genetic principal components, the present study found that a cumulative factor of stress, dubbed "s" factor, was related to physical health outcomes, including BMI, number of chronic conditions, and difficulty performing activities of daily living. Moreover, the hierarchical s-factor predicted physical health outcomes almost a decade later, even after accounting for demographic factors and prior levels of physical health.

Conversely, results did not support a unit-weighted summary score or unrestricted unidimensional model of cumulative stress. Instead, five dimensions of environmental stress were identified—stress related to discrimination, home and work, perceived inequality, interpersonal relationships, and personal financial hardship. Results indicate that distinguishing common from residual variation using a hierarchical model can reveal differential associations between dimensions of cumulative stress and physical health outcomes. For example, after accounting for the general s-factor of cumulative stress, the discrimination factor was concurrently associated with myriad physical health outcomes, while relationship stress prospectively predicted greater difficulty with intermediate activities of daily living. After accounting for the cumulative s-factor, home and work-related stress were only associated concurrently with number of chronic conditions and activities of daily living, while stress related to personal financial hardship was also associated with BMI. On the other hand, relationship stress was marginally associated with both basic and moderate activities of daily living nearly a decade later.

As reviewed by Slavich (2016), "there is little agreement on what features of stressors are most important to measure." Stress can be characterized as having a uniform physiological response pattern (i.e., alarm, resistance, and exhaustion), regardless of the type of stressor (Selye, 1976) Alternatively, stress can be viewed as the amount of change and readjustment a person experiences in life; that is, the more life changes, the more stress (Holmes, 1967). Other researchers have posited that stress is a function of intrapersonal factors, such as perceived controllability (Maier and Watkins, 2005), disruption to the pursuit of personal goals (Brown and Tirril, 1978), or exacerbation of existing vulnerabilities (Clark et al., 1999). The present study forwards a novel perspective by focusing on the co-occurrence of stressors, both acute and chronic, to empirically derive dimensions of stress that might be the most deleterious for health outcomes.

4.1. Limitations and future directions

There are several limitations to the current study. For example, it may be argued that polygenic scores provide only limited controls for gene-environment correlations, as polygenic scores only capture additive and common genetic liability. Consequently, the current study was not able to control for potential confounds due to the influence of rare variants and non-additive genetic effects. Moreover, although a number of polygenic risk scores were marginally associated with cumulative stress and more strongly with physical health outcomes, the percent of variance in cumulative stress and health outcomes explained by polygenic risk scores was relatively small (~2%–7%). It remains an open question whether the estimated links between cumulative stress and health will wax or wane as genome-wide association studies continue to

grow in size and, in turn, increase the predictive potency of resulting polygenic risk scores.

Environmental and psychosocial stressors were also measured using self-reports. Therefore, the present study cannot rule-out contributions of method variance to factors of cumulative stress. However, implementing genetic controls for anxiety and neuroticism, as well as other psychological variables, helps account for potential self-evaluation bias. Nevertheless, future studies should implement a multi-trait multi-method approach to advance research on a hierarchical model of cumulative stress. For example, future studies would benefit from measuring chronic stress exposure not only using self- *and* informant-reports, but also by using detailed interviews, automated stress inventories, like the Stress and Adversity Inventory (STRAIN), and laboratory-based measures of acute stress reactivity (e.g., the Trier Social Stress Test and the Cold pressor test).

The correlational design of the present study also precludes drawing causal conclusions about the relations between study variables, as causal inference is not warranted without conducting a true experiment. The present study focused on cumulative stress in the sense of simultaneous exposure to multiple stressors. However, cumulative stress can also be conceptualized as repeated exposures to the same stressors. Future studies may benefit from focusing on repeated exposures over time, as well as simultaneous exposure to multiple stressors.

Although the current study documented concurrent and prospective associations with physical health outcomes, a more fine-grained longitudinal analysis of stress would allow one to evaluate whether absolute levels of stress or changes in stress over time are more strongly related to mental and physical health. It is also important not to discount the potential adverse health effects of other stressors not examined in the current study, as well as how other stressors may fit within the hierarchical structure of stress documented in the current study. Associations with demographic factors and physical health may differ when focusing on a different array of stressors. The current study relied on existing data, and the measures that are available for secondary analysis often vary across national-level datasets, which prevents researchers from directly replicating findings across existing datasets. Nevertheless, future studies should extend findings along with other environmental stressors to improve the predictive utility of a cumulative factor of stress and enhance our understanding of the number of subordinate dimensions of stress that have predictive validity. Future research also stands to benefit from testing the generalizability of findings across other populations and cohorts, though the current study found that a hierarchical model of stress replicated across distinct economic periods, when the average levels of cumulative stress significantly differed.

5. Conclusions

Although the present study is not without limitations, results provide a first-step toward empirically organizing and operationalizing the multidimensional, correlated structure of psychosocial and environmental stress. Results provide evidence for the concurrent and predictive validity of a hierarchical "s-factor" of cumulative stress. In addition, results highlight the potential predictive value of distinguishing common from residual variation when evaluating the impact of multiple stressors on physical health. In the current study, we expounded arguments for and tested a hierarchical model of cumulative stress. We found that a hierarchical "s-factor" of cumulative stress was higher following a recession, compared to period of relative prosperity, and associated with physical health outcomes. Moreover, the s-factor of cumulative stress predicted physical health outcomes almost a decade later. Five distinct lower-level dimensions of stress— discrimination exposure, stress related to home and work, stress related to perceived inequality, interpersonal stress, and personal financial hardship —exhibited varied associations with physical health outcomes. These findings bear potentially important implications for how researchers aggregate information across multiple indicators to measure cumulative stress.

Author contributions

F.D.M. developed the idea for the study, conducted analyses, and drafted the methods and results. In discussion, F.D.M. and A.G.C. selected the variables for analyses. A.G.C. and F.D.M. drafted the introduction and discussion. R.F.K. helped obtain funding for the study, advised F.D.M., contributed to the design of the study, and provided critical project framing, as well as revising the manuscript throughout its construction. All authors provided critical revisions and approved a final version of the manuscript.

Data accessibility

Data are available on the MIDUS Colectica portal (http://midus.colectica.org). Analysis scripts are available on the Open Science Framework (https://osf.io/yht6b/).

Declaration of competing interest

The authors have no conflicts of interest to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2021.114405.

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