



Flatter diurnal cortisol slopes amplify the association between daily stress and somatic symptoms

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ABSTRACT

Background: Daily somatic symptoms, such as fatigue and headaches, are common among adults and can forecast health decline. Flatter diurnal cortisol slopes (DCS), indicating a dysregulated hypothalamic-pituitary-adrenal (HPA) axis, are linked to physical health impairments. Whether DCS moderate the within-person relationship between daily stressors and somatic symptoms, and whether stress buildup further influences this association, remain unclear.

Method: Middle-aged community adults ($N = 1621$) completed daily diary interviews and offered salivary cortisol samples over four days, producing 6484 person-day observations. Three-level generalized linear mixed-effects models using a Tweedie distribution examined main effects, two-way and three-way interactions among DCS, within-person daily stressor exposure, and stress-buildup burden on daily somatic symptom counts. Daily stress was assessed for both stressor frequency and severity; the average stressor severity was assigned *a priori* as the key definition.

Results: Within-person stressor exposure was linked to higher same-day somatic symptoms across all definitions. Flatter DCS were associated with greater somatic symptom burden when stress was defined as the daily stressor count ($\beta = 0.217$). DCS significantly moderated the count-derived stress-symptom relationship ($\beta = 0.019$) but did not moderate the severity-average relationship ($\beta = -0.017$). Moreover, stress buildup moderated this cortisol-related pattern (three-way interaction: $\beta = -0.043$), but this outcome was sensitive to the imputation approach and should be interpreted with caution.

Conclusion: DCS was related to differential somatic reactivity to everyday stress, such that flatter DCS was connected with stress-somatic symptom coupling. Contextual stressors and physiological regulation likely jointly affect daily somatic health among adults.

1. Introduction

Physical or somatic symptoms, such as muscle tension, fatigue, and headaches, are prevalent everyday encounters even among healthy community adults (Leger et al., 2015). If chronic, somatic symptoms could adversely affect focus, sleep quality, work productivity, and psychosocial well-being (Matalon et al., 2011). Longitudinal data indicated that frequent daily somatic symptoms predicted more chronic illnesses and functional impairments across time (Jackson et al., 2006). Given their adverse effects and prevalence, determining the precipitating factors of daily somatic symptoms is key to enhancing well-being and preventing future mental health, physical, and work-related consequences.

It has been proposed that dysregulated hypothalamic-pituitary-adrenal (HPA) axis activity can increase the risk of somatic symptoms (Kuras et al., 2017). Among healthy individuals, cortisol levels rise and peak in the morning after awakening, then decline throughout the day, culminating in low levels in the evening and before bedtime (Adam and Kumari, 2009). This critical daily cortisol decline, referred to as a steep cortisol slope, reflects healthier and more adaptive HPA axis regulation (McEwen, 1998). By comparison, flatter diurnal cortisol slopes, in which evening cortisol levels remain high, indicate atypical stress psychophysiology (Miller et al., 2007). Chronically flat diurnal cortisol slopes are believed to reflect HPA axis dysregulation and a greater allostatic load, i.e., the buildup of physiological wear and tear intimately linked to persistent stress exposure over time (McEwen, 1998). Persistent HPA

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axis dysregulation may thereby be related to heightened somatic symptoms (Brosschot et al., 2005; Geurts and Sonnentag, 2006).

The literature supports the view that flatter diurnal cortisol slope profiles are associated with greater somatic symptoms and related physical illness markers. Adam et al. (2017) reported in a meta-analysis of 80 studies that flatter diurnal cortisol slopes were consistently associated with poorer mental and physical health indices. Likewise, flatter cortisol slope profiles have coincided with greater cardiometabolic susceptibility and more somatic symptoms associated with fatigue. Further, Zilioli et al. (2017) reported that lower socioeconomic status (SES) was associated with flatter diurnal cortisol slope declines and a stronger somatic symptom burden, with cortisol dysregulation partially explaining these patterns. Collectively, this body of research suggests that flatter diurnal cortisol slopes are associated with greater somatic symptom burden. Nonetheless, an important gap remains: how contextual or person-level variables modify the day-to-day relationship between cortisol regulation and somatic symptoms is not well understood.

A viable, theory-driven moderator is the daily stress experience. From a diathesis-stress viewpoint, persistently flatter cortisol slopes can be regarded as a psychophysiological risk factor that is likely to coincide with somatic symptoms when individuals face greater daily stress (Monroe and Simons, 1991). On high-stress days, those with flatter cortisol slope declines may display less effective stress responses and recovery patterns than individuals with steeper cortisol slopes. These processes may be linked to high and sustained muscle tension, proinflammatory activity, and sympathetic nervous system arousal (Brosschot et al., 2006; Cohen et al., 2012). However, it is equally likely that daily stress severity functions as a direct predictor of both somatic symptoms and cortisol regulation independently, rather than only as a context-dependent moderator (Almeida, 2005), which remains an important area for future research. The current study prioritizes the moderation hypothesis, as the diathesis-stress framework specifically posits that the translation of psychophysiological risk into somatic outcomes will depend on stressor load, rather than on mere correlations. Critically, cortisol dysregulation and stress relationships can also unfold over time, with stress on a given day possibly influencing cortisol dysregulation in subsequent days. However, as research on same-day cortisol-somatic symptom coupling in daily diaries is developing, the current study examines simultaneous associations as a preliminary investigation, while recognizing lagged pathways as essential for future studies.

Empirical data support the perspective that higher daily stress exposure is associated with greater same-day somatic symptom burden. Daily diary research found that on days with more stressors, individuals reported more somatic symptoms than on stress-free days (Haight et al., 2023). In addition, previous research demonstrated notable between-person variations in daily stress reactivity (Goldring and Bolger, 2021), implying that stable traits, including biological ones, may moderate this within-person stress-symptom coupling.

The current study adds to this literature in several ways. First, we capitalized on an intensive daily diary approach to capture within-person, day-to-day fluctuations in cortisol and somatic outcomes. This approach facilitates temporal sequencing and the study of perturbations around each individual's baseline while limiting recall biases (Adam and Kumari, 2009; Bolger et al., 2003). Second, most previous cortisol studies relied on clinical or younger samples (Demakakos and Steptoe, 2022); we evaluated predominantly middle-aged adults in the general population using data from the Midlife Development in the United States (MIDUS) project (Almeida et al., 2025). Thus, this endeavor expands outcomes to an underinvestigated age group (Haight et al., 2023). Third, we measured stress concurrently at the everyday and cumulative person levels to evaluate whether a persistent stress burden moderated cortisol-linked risk. Fourth, we evaluated two conceptually distinct definitions of everyday stress: stressor frequency (unique stressor counts) and stressor severity (self-reported intensity), each modeling a different dimension of stressor encounters (Almeida, 2005). Stressor

count reflects exposure to discrete events, while stressor severity indicates their self-appraised intensity; both are conceptually related but can yield differential effects. Studying both *a priori* facilitates a more holistic examination of stress-cortisol-somatic symptoms pathways. The average stressor severity was assigned as the primary definition, as it most directly indicates within-person daily stress intensity; stressor count served as a robustness check. Finally, the diurnal cortisol slope was defined as a person-level average across diary days, consistent with previous daily diary studies that regard the aggregated slope as a marker of stable trait-level differences in HPA axis functioning (Ross et al., 2014; Segerstrom et al., 2014). Moreover, determining moderators of the cortisol-somatic symptom relationship could inform targeted prevention strategies, such as just-in-time adaptive interventions (JITAI; Zainal et al., 2025).

Based on theory and research, we examined three hypotheses, two confirmatory and one exploratory. First, participants with flatter diurnal cortisol slopes would display more daily somatic symptoms across days relative to their counterparts with steeper slopes (Hypothesis 1 [H1]: Cortisol Slope $\rightarrow$ Somatic Symptoms). Second, higher daily stressor exposure would be related to more somatic symptoms, and this within-person relationship would be greater among individuals with flatter diurnal cortisol slopes (Hypothesis 2 [H2]: Cortisol Slope $\times$ Daily Stress $\rightarrow$ Somatic Symptoms). Third, we evaluated the theory-based hypothesis that those with higher stress buildup would display stronger cortisol slope moderation of daily stress exposure effects, but we regarded this as exploratory due to the complexity and nascent nature of this research area (Hypothesis 3 [H3]: Cortisol Slope $\times$ Daily Stress $\times$ Cumulative Stress $\rightarrow$ Somatic Symptoms). Collectively, we anticipated that flatter diurnal cortisol slopes would be related to higher symptom burden (H1), that this risk would be magnified on high-stress days (H2), and that persistent stress would further amplify this conditional relationship (H3).

2. Methods

2.1. Participants

The baseline data were based on the MIDUS 2 (2004–2006) study, which initially included 4963 participants (Ryff et al., 2007). Within this sample, 2022 participants completed the NSDE 2, which comprised daily diary protocols (Almeida et al., 2025). Among NSDE 2 participants, 1735 provided saliva samples for cortisol measurement as part of the NSDE 2 protocol (Dienberg Love et al., 2010). We retained individuals in the family subsample and modeled family membership as a third level to account for non-independence among related participants (e.g., siblings and twins). All participants provided informed consent, and the study procedures were approved by the institutional review boards at all four MIDUS sites. This study was a secondary analysis of de-identified MIDUS data, and thus no additional ethics approval was needed.

2.2. Procedures

Participants completed evening telephone interviews over 8 consecutive days, during which they reported daily stressors and somatic symptoms experienced in the past 24 h (Almeida et al., 2009b). Within the cortisol protocol, four saliva samples were collected at preset times (immediately post-waking, 30 min post-waking, before lunch, and at bedtime) on Days 2 through 5 of the diary period, yielding 16 total samples per participant. Participants received detailed instructions for sample collection, including avoiding food and caffeine, and refraining from brushing teeth for 30 min before each sample collection. Saliva samples were stored in home freezers and returned via prepaid courier. Hours elapsed were computed from self-reported collection times, and samples with missing or implausible times were treated as missing (Dienberg Love et al., 2010).

2.3. Measures

Diurnal cortisol slope. Cortisol levels were determined from saliva samples using a commercially available luminescence immunoassay (IBL, Hamburg, Germany), with intra- and inter-assay coefficients of variation below 5% and a lower detection limit of 0.012 µg/dL (Dienberg Love et al., 2010). Given the typical skewed distribution of cortisol values, we applied a natural log transformation before computing the slope (Miller and Plessow, 2013). Daily slope was calculated as the difference between log-transformed bedtime and waking cortisol values divided by hours elapsed. The 30-minute post-waking sample was excluded from analysis (waking > 100 nmol/L, 30-minute > 150 nmol/L, lunch > 100 nmol/L, bedtime > 50 nmol/L). To enhance reliability, participant-level slopes were aggregated by averaging across all 4 collection days, consistent with recommendations that multiple days are required to obtain stable trait-level estimates (Kuhlman et al., 2019). The resulting person-level slope ($M = -0.118$, $SD = 0.080$) served as a Level 2 predictor in the generalized linear mixed-effects models described in the Analytic strategy subsection. Flatter (less negative and positive) slope values and positive slope values indicated greater HPA axis dysregulation.

Daily stressors. Daily stressors were measured using the Daily Inventory of Stressful Events (DISE; Almeida et al., 2002), administered via telephone interviews. Each evening, participants reported whether they had experienced specific stressors in the past 24 h, including interpersonal arguments, avoided arguments, work or school stressors, home stressors, discrimination, network stressors, and other stressors. We derived a daily stressor count ($M = 0.52$, $SD = 0.74$, range: 0–6), which is less sensitive to individual differences in appraisal than subjective severity ratings and has been widely used in prior NSDE research (e.g., Zainal et al., 2024). It served as a robustness check alongside the average stressor severity, which was the primary exposure variable. Stressors occurred on 39.8% of diary days. To calculate a participant-level index of cumulative stress exposure, the arithmetic mean of daily stressor counts across all diary days was derived (Mean-Stress). The DISE has demonstrated good internal consistency and strong construct validity (Almeida et al., 2009a). In the present study, the internal consistency for this scale was adequate (McDonald's $\omega = .833$; Geldhof et al., 2014), as the seven stressor categories represent distinct event types (e.g., interpersonal arguments, work stressors, discrimination) rather than indicators of a single latent construct.

Daily somatic symptoms. Somatic symptoms were assessed using a 28-item checklist adapted from Larsen and Kasimatis (1991). Participants indicated whether they experienced each symptom that day (e.g., headache, backache, fatigue, nausea). Each item was coded as 0 (*absent*) or 1 (*present*), and the items were summed to yield a daily somatic symptom count ($M = 1.69$, $SD = 2.08$, range: 0–18). We inspected the distributions and observed that somatic symptom counts were zero-inflated. This scale showed high internal consistency and good construct validity (Leger et al., 2015). In the current study, the internal consistency for this measure was high (McDonald's $\omega = .871$).

2.4. Analytic strategy

Missingness, which was present in about 20% of the dataset across the 51 variables, was concentrated mainly in the daily stressor severity items, which were prompted on days when participants reported navigating one or more stressors. Therefore, this missingness was structural and occurred by design. Random forest (RF) imputation was thus well-suited to this structured missingness and was performed using the *missRanger* R package (Mayer, 2024) with predictive mean matching ($k = 5$) and 500 trees. Moreover, RF has the advantage of modeling non-linearities and mixed-variable forms inherent to daily diary data in psychoneuroendocrinology (Shah et al., 2014). We applied RF imputation solely on predictor variables, i.e., no somatic symptom count outcome variables were imputed (Table S1 in the online supplemental

materials [OSM]).

All models were specified using generalized linear mixed-effects models with *glmmTMB* (Brooks et al., 2017; McGillicuddy et al., 2025) in R (version 4.3), with the Tweedie distribution (link = “log”) and maximum likelihood estimation. The Tweedie distribution is well-suited for outcomes exhibiting zero-inflation and overdispersion relative to the Poisson, as it flexibly models exact zeros alongside a right-skewed non-zero portion without requiring a distinct zero-inflation sub-model. The dataset had a three-level structure: daily observations (Level 1) clustered within individuals (Level 2), which were clustered within families (Level 3), with random intercepts at both levels of clustering. Random slopes for within-person daily stress deviation were assessed but excluded due to convergence problems and boundary singularity, which we explicitly reported as a shortcoming. Age and self-reported sex were added as covariates in all models, given their established associations with daily cortisol dynamics and somatic symptom count reporting (Deutschbein et al., 2022).

To differentiate between-person from within-person effects, daily stressor variables were person-mean centered, i.e., $\text{StressDev}_{it} = \text{Stress}_{it} - \text{Stress}_{\bar{i}}$, such that StressDev_{it} modeled day-to-day variations compared to each person's own average level. Moreover, the individual-level average $\text{Stress}_{\bar{i}}$ was retained as a Level 2 predictor, reflecting stress buildup over the daily diary period. Age and diurnal cortisol slope were grand-mean centered to ease interpretation and minimize multicollinearity; self-reported sex was effect-coded ($-0.5 = \text{men}$, $0.5 = \text{women}$). Cortisol slope was defined as an individual-level aggregate (averages across diary days) to indicate stable HPA axis functioning as a trait-level biological moderator, consistent with previous daily diary studies that treated the aggregated slope as a between-person variable (Ross et al., 2014; Segerstrom et al., 2014).

Main-effects and two-way interaction models were estimated under each of three stress definitions (daily stressor count, stressor severity sum, and stressor severity average), and a single three-way interaction model was estimated for the stressor severity average definition, yielding seven models in total. Stressor count and severity models capture important, unique perspectives of daily stress (i.e., frequency vs. intensity), and both were included to facilitate direct contrasts rather than choosing a single definition a priori (Almeida, 2005). Primary results are reported for stressor severity average, which most directly defines average daily stress intensity. The other definitions functioned as robustness checks. Nested models were contrasted using likelihood-ratio tests and the Akaike/Bayesian information criterion (AIC/BIC), with statistically significant interactions probed through Johnson-Neyman regions of significance and simple slopes analyses at $\pm 1 SD$ (Preacher et al., 2006). Key equations are as follows:

- $\log(\mu_{it}) = \gamma_{00} + \gamma_{10}(\text{StressDev}_{it}) + \gamma_{01}(\text{CortSlope}_i) + \gamma_{02}(\text{MeanStress}_i) + \gamma_{03}(\text{Age}_i) + \gamma_{04}(\text{Sex}_i) + u_{0i} + v_{0j}$ (**H1: Main Effects**)
- $\log(\mu_{it}) = \gamma_{00} + \gamma_{10}(\text{StressDev}_{it}) + \gamma_{01}(\text{CortSlope}_i) + \gamma_{02}(\text{MeanStress}_i) + \gamma_{03}(\text{Age}_i) + \gamma_{04}(\text{Sex}_i) + \gamma_{11}(\text{StressDev}_{it} \times \text{CortSlope}_i) + u_{0i} + v_{0j}$ (**H2: Two-Way Interaction**)
- $\log(\mu_{it}) = \gamma_{00} + \gamma_{10}(\text{StressDev}_{it}) + \gamma_{01}(\text{CortSlope}_i) + \gamma_{02}(\text{MeanStress}_i) + \gamma_{03}(\text{Age}_i) + \gamma_{04}(\text{Sex}_i) + \gamma_{11}(\text{StressDev}_{it} \times \text{CortSlope}_i) + \gamma_{12}(\text{StressDev}_{it} \times \text{MeanStress}_i) + \gamma_{21}(\text{CortSlope}_i \times \text{MeanStress}_i) + \gamma_{111}(\text{StressDev}_{it} \times \text{CortSlope}_i \times \text{MeanStress}_i) + u_{0i} + v_{0j}$ (**H3: Three-Way Interaction**)

where Y_{it} indicates the daily somatic symptom count for individual i on day t , $\mu_{it} = E[Y_{it}]$, StressDev_{it} = within-person stress deviation (person-mean centered), CortSlope_i = diurnal cortisol slope (grand-mean centered), MeanStress_i = person-mean stress burden (grand-mean centered), and u_{0i} and v_{0j} indicate random intercepts for individuals and families respectively.

Four prespecified sensitivity analyses were performed to test robustness. First, missing data management was evaluated by contrasting RF-imputed outcomes against per-model complete-case analyses and standard multiple imputation by chained equations (MICE) with

predictive mean matching (Buuren and Groothuis-Oudshoorn, 2011). Missingness diagnostics were also reported. Second, distributional assumptions were tested by refitting all seven models using the negative binomial (NB), zero-inflated Poisson (ZIP), and zero-inflated negative binomial (ZINB) distributional families. We contrasted the fit through AIC and BIC. Third, the reliability of the diurnal cortisol slope was examined using intraclass correlation coefficients (ICCs) from a two-level null model. All models were then re-estimated within a timing-adherent subsample that excluded person-days with data collection times deviating by more than an hour from each person's median, in line with the compliance reference points in cortisol research (Kudielka et al., 2003; Stalder et al., 2016) (OSM Table S2). Fourth, covariate-adjusted models were examined in two sets: (i) including within-person wake-time variability across the entire diary sample, and (ii) including body mass index (BMI), depressive symptoms, prescription medication use, self-rated health, sleep quality, and smoking status within the biomarker sub-sample (OSM Table S3).

3. Results

3.1. Descriptive statistics

Participants were included if they had at least 4 usable cortisol days with $\geq 50\%$ valid cortisol samples per day and at least 4 completed diary interviews with valid stress and symptom data (Ross et al., 2014; Segerstrom et al., 2014). The final analytic sample comprised 1621 participants from 1246 families, contributing 6484 person-day observations (4 days per participant). Participants had an average age of 56.3 years (standard deviation [SD] = 12.02, range = 33–83). Regarding self-reported sex, 56.3% ($n = 912$) were women and 43.7% ($n = 709$) were men. Regarding self-reported race, the majority of participants identified as White (86.4%, $n = 1396$), followed by Black or African American (9.6%, $n = 156$), including 116 participants from the Milwaukee African American oversample, Native American or Alaska Native (1.3%, $n = 21$), Asian (0.2%, $n = 4$), and other racial groups (2.4%, $n = 39$), with race data missing for 5 participants (0.3%); percentages are based on the 1616 participants with valid data.

The final analytic sample ($N = 1621$) contributed 6484 person-days of diary data. Regarding cortisol metrics, diurnal cortisol slopes averaged $-0.12 \log \text{ nmol/L/hr}$ ($SD = 0.08$, range = -0.46 to 0.28), reflecting the anticipated negative decline in cortisol across the waking phase relative to waking onset. Relatedly, daily somatic symptom counts were largely right-skewed, i.e., 2392 person-days (36.89%) exhibited zero somatic symptoms ($M = 1.695$, variance = 4.325 ; variance-to-mean ratio = 2.552). Pearson dispersion descriptives calculated from the fitted Tweedie main-effects models were between 0.602 and 0.609 across all three stressor definitions, suggesting sufficient conditional fit. The maximal observed symptom count was 18. These distributional characteristics (i.e., a high proportion of zeros, marginal overdispersion relative to a Poisson distribution, and right-skew) justified the choice of a Tweedie generalized linear mixed model instead of conventional Poisson regression (Table 1).

3.2. Distribution sensitivity analyses

All seven primary models were examined under the NB, ZIP, and ZINB family distributions and contrasted using AIC. The Tweedie distribution offered a notably better fit across five of the seven models, with AIC values ranging from $-22,011.03$ to $-18,268.36$, which were considerably lower than those of the NB ($19,904\text{--}20,024$), ZIP ($19,881\text{--}19,998$), and ZINB ($19,883\text{--}20,003$) models (Table 2). For the average-stressor severity main-effects and three-way interaction models, Tweedie AICs could not be estimated due to convergence issues, and ZIP yielded the lowest AIC. Pearson overdispersion statistics from the specified Tweedie main-effects models ranged between 0.602 and 0.609, suggesting negligible residual overdispersion. Across specific interaction

Table 1
Distribution diagnostics for daily somatic symptom counts.

Section	Metric	Value
A1: Observed Distribution		
	Total person-days	6484
	Zero observations (n)	2392
	Zero observations (%)	36.89
	Mean somatic symptom count	1.695
	Variance	4.325
	Variance/Mean ratio	2.552
	Maximum count	18
A2: Pearson Overdispersion (Tweedie)		
	Main-1 (Count)	0.602
	Main-2 (Sum)	0.605
	Main-3 (Average)	0.609
A3: Expected vs. Observed Zeros		
	Main-1 (Count): Observation = 2392; Exp(Poisson) = 2340;	
	Ratio = 1.022	
	Main-2 (Sum): Observation = 2392; Exp(Poisson) = 2368;	
	Ratio = 1.010	
	Main-3 (Average): Observation = 2392; Exp(Poisson) = 2411;	
	Ratio = 0.992	

Note. A1: daily somatic symptom count from the MIDUS 2 daily diary. A2: Pearson dispersion $D = \Sigma((y - \mu)^2/\mu)/(n - p)$; $D > 1$ indicates overdispersion. A3: Expected zeros under Poisson = $\Sigma \exp(-\mu)$; ratio > 1 indicates zero-inflation beyond Poisson.

Table 2
Model fit comparison across distributional families (AIC).

Model	Tweedie	NB	ZIP	ZINB	Best (AIC)
Main-1 (Count)	-20,772.8	20,024.0	19,998.1	20,002.7	Tweedie
Main-2 (Sum)	-22,011.0	19,904.0	19,880.7	19,882.7	Tweedie
Main-3 (Average)	—	19,913.0	19,887.8	19,889.8	ZIP
Interaction-1 (Count $\times$ C)	-21,895.6	20,022.2	19,997.2	19,999.2	Tweedie
Interaction-2 (Sum $\times$ C)	-18,268.4	19,904.0	19,882.2	19,884.2	Tweedie
Interaction-3 (Average $\times$ C)	-20,548.4	19,914.0	19,889.5	19,891.5	Tweedie
Three-Way	—	19,911.7	19,886.5	19,888.5	ZIP

Note. AIC = Akaike Information Criterion; NB = Negative Binomial type 2; ZIP = Zero-Inflated Poisson; ZINB = Zero-Inflated Negative Binomial. All models use identical fixed effects, random effects, and BOBYQA optimizer. Lower AIC indicates better fit. — = model did not converge or was not estimable.

terms, three displayed sign reversals compared to NB and three compared to both ZINB and ZIP (OSM Table S4). Six altered statistical significance at $p < .05$ compared to NB and five compared to both ZINB and ZIP.

3.3. Primary multilevel models

Main-effects models (H1) suggested that larger within-person stressor exposure was significantly related to more daily somatic symptoms across all three stress definitions: stressor count ($\beta = 0.059$, $SE = 0.009$, $z = 6.870$, $p < .001$), stressor severity sum ($\beta = 0.043$, $SE = 0.009$, $z = 4.964$, $p < .001$), and stressor severity average ($\beta = 0.122$, $SE = 0.010$, $z = 12.295$, $p < .001$). Across two-way interaction models (H2), diurnal cortisol slopes significantly interacted with stressor count to predict somatic symptom severity ($\beta = 0.019$, $SE = 0.009$, $z = 2.290$, $p = .022$) but was not significant for stressor severity average ($\beta = -0.017$, $SE = 0.009$, $z = -1.852$, $p = .064$), but was non-significant for stressor severitysum ($\beta = 0.011$, $SE = 0.008$, $z = 1.295$, $p = .195$). The three-way interaction model (H3), comprising within-person stressor average, person-mean stressor average, and diurnal cortisol slope, produced a significant three-way term ($\beta = -0.043$, $SE = 0.011$, $z = -4.042$, $p < .001$). This three-way term suggested that the buildup of

stress burden moderated the interaction between cortisol and stressor severity average. Moreover, within the three-way model, the interaction between person-mean stressor severity and cortisol slope was significant ($\beta = 0.165$, $SE = 0.032$, $z = 5.156$, $p < .001$), whereas the within-person stressor severity average by cortisol slope interaction was not ($\beta = 0.011$, $SE = 0.010$, $z = 1.121$, $p = .262$).

3.4. Missing data, stressor-based, and covariate-adjusted sensitivity analyses

First, missingness-based sensitivity analyses were conducted. Little's MCAR test ($\chi^2(120) = 241.39$, $p < .001$) indicated that missingness was not completely at random, consistent with the structural mechanism described above; the test cannot adjudicate between MAR and MNAR. Complete-case analyses comprised 5459 person-days for stressor-count analyses and 2182 for stressor severity-based analyses. Compared with the RF-imputed primary analyses, these models produced 6 sign reversals and 8 changes in statistical significance across key parameter estimates (OSM Table S5). The standard MICE imputation approach, compared with the RF-imputed primary analyses, yielded 4 sign reversals and 12 changes in statistical significance, with the three-way interaction term reversing sign and becoming non-significant ($\beta = 0.005$, $SE = 0.013$, $z = 0.36$, $p = .720$) (Table 3; OSM Table S6).

Next, sensitivity analyses focused on time-compliant participants. On that note, for the cortisol slope definition, ICC = 0.378 for the two-level null model, suggesting that 62.2% of diurnal slope variations indicate within-person fluctuations instead of stable trait-level variations (OSM Table S7). Thus, we re-estimated all models for a subsample ($n = 1542$; 5058 person-days) that excluded days from data collection periods that deviated by more than an hour from each person's median (average waking deviation = 28.32 min, $SD = 38.19$; 21.84% of person-days). Compared with the primary analyses, this time-compliant subsample showed 2 sign reversals and 7 changes in statistical significance across key terms (OSM Table S8).

Table 3

Missing data strategy comparison: RF-imputed vs. complete-case vs. MICE.

Model / Term	RF-Imputed		CCA		MICE		Flags	
	β	p	β	p	β	p	vs CCA	vs MICE
Interaction-1 (Count $\times$ C)								
Cortisol slope	0.089	.003	0.089	.025	0.314	.739		Sig Δ
Count (PMC)	0.041	< .001	0.046	< .001	0.053	.014		
Count $\times$ Cortisol	0.019	.022	0.021	.032	0.018	.495		Sig Δ
Interaction-2 (Sum $\times$ C)								
Cortisol slope	0.313	< .001	0.056	.069	0.067	.721	Sig Δ	Sig Δ
Sum (PMC)	0.069	< .001	0.024	.071	0.053	.013	Sig Δ	
Sum $\times$ Cortisol	0.011	.195	0.027	.102	0.018	.239		
Interaction-3 (Average $\times$ C)								
Cortisol slope	-0.146	< .001	0.118	< .001	0.077	.656	Sign $\leftrightarrow$	Sign $\leftrightarrow$
Average (PMC)	0.055	< .001	0.036	.021	0.009	.660		Sig Δ
Average $\times$ Cortisol	-0.017	.064	0.011	.521	0.000	.978	Sign $\leftrightarrow$	
Main-1 (Count)								
Cortisol slope	0.217	< .001	-0.073	.022	0.061	.858	Sign $\leftrightarrow$	Sig Δ
Count (PMC)	0.059	< .001	0.014	.140	0.053	.058	Sig Δ	Sig Δ
Main-2 (Sum)								
Cortisol slope	0.085	.017	0.043	.150	0.014	.948	Sig Δ	Sig Δ
Sum (PMC)	0.043	< .001	-0.004	.761	0.062	.001	Sign $\leftrightarrow$	
Main-3 (Average)								
Cortisol slope	0.843	< .001	0.133	< .001	0.116	.636		Sig Δ
Average (PMC)	0.122	< .001	0.003	.828	0.014	.700	Sig Δ	Sig Δ
Three-Way								
Cortisol slope	-0.073	.022	0.030	.304	0.084	.435	Sign $\leftrightarrow$	Sign $\leftrightarrow$
Cortisol $\times$ Mean average	0.165	< .001	-0.003	.911	0.025	.801	Sign $\leftrightarrow$	Sig Δ
Mean severity average	0.295	< .001	0.191	< .001	0.160	.054		Sig Δ
Average (PMC)	0.036	< .001	0.008	.606	0.019	.219	Sig Δ	Sig Δ
Average $\times$ Cortisol	0.011	.262	0.020	.246	-0.002	.881		Sign $\leftrightarrow$
Three-way	-0.043	< .001	-0.031	.140	0.005	.720	Sig Δ	Sign $\leftrightarrow$
Average $\times$ Mean average	0.026	.031	0.021	.285	0.031	.024	Sig Δ	

Note. RF-Imputed = missRanger random forest (primary analysis); CCA = per-model complete-case analysis; MICE = predictive mean matching multiple imputation pooled via Rubin's rules ($m = 20$). Sign $\leftrightarrow$ = estimate changed direction vs. RF-Imputed. Sig Δ = significance at $p < .05$ changed vs. RF-Imputed.

reflects a more chronic buildup of demands and hassles that increase ratings on a given day, and that between-person variations in cortisol rhythms are more readily identified in this context. The distinction between stressor severity sum and average definitions also merits discussion. Although severity sum builds up across all stressors indicated on a given day, severity average implies average intensity per stressor event, rendering the two scales differentially responsive to both the frequency and intensity of stressors. Therefore, stressor severity sum and average are not necessarily interchangeable.

Beyond main effects, the interaction outcomes suggest that flatter cortisol slopes may be associated with greater coupling, but the observational design limits mechanistic conclusions. It has been proposed that one mechanism linking cortisol dysregulation to somatic outcomes may involve increased proinflammatory activity, including elevated C-reactive protein, interleukin-6, and fibrinogen (Knight et al., 2021). However, as no proinflammatory markers were assessed in the current study, this pathway remains conjectural. In addition, it is worth considering that the current study treats the diurnal cortisol slope as a stress-reactivity moderator, but an equally plausible account is that daily stressor severity serves as a predictor of both somatic outcomes and cortisol regulation. Future studies should investigate such pathways using intensive longitudinal design approaches (Almeida, 2005).

The current outcome is broadly consistent with the allostatic load model, in which circadian dysregulation reflects the buildup of somatic wear and tear and can culminate in increased somatic symptoms (McEwen, 1998). Through a biobehavioral lens, steeper declines in diurnal cortisol slopes may reflect greater HPA axis flexibility and more efficient psychophysiological recovery following daily stressors (Adam et al., 2017). Comparatively, flatter diurnal cortisol slopes may be linked to regulatory inflexibility and compromised ability to implement suitable, differentiated stress responses, likely increasing somatic symptom counts (Cohen et al., 2012). The moderating function of stress buildup concurs with the diathesis-stress framework (Monroe and Simons, 1991). That is, persistent environmental stressors may compromise the psychophysiological resources required to effectively cope with everyday stressors. Moreover, the outcome that high stress buildup attenuated variations in cortisol slope subgroups aligns with the glucocorticoid receptor resistance frameworks. This framework posits that chronic stress exposure can decrease immune cell sensitivity to cortisol's anti-inflammatory signaling, such that even participants with typical HPA axis output can experience increased somatic risk (Cohen et al., 2012). Together, these findings support a cumulative toxicity pathway in which persistent allostatic burden can compromise the neuroendocrine resources required to protect against the adverse consequences of daily stress (Juster et al., 2010).

The present study had several limitations. First, the observational nature of the study hinders causal inference; unmeasured variables, such as proinflammatory activity levels, can independently affect both the diurnal cortisol rhythm and somatic burden. Second, diurnal cortisol slopes were averaged across diary days and regarded as a between-person, trait-level predictor, aligned with previous daily diary studies of cortisol (Ross et al., 2014; Segerstrom et al., 2014). However, the intraclass correlation analyses indicated that most of the slope variation was attributable to within-person day-to-day fluctuations, implying that averaging may limit the study of state-level cortisol variations that are intimately linked to same-day stress pathways. Future studies should evaluate day-level cortisol slopes as time-varying biobehavioral indices. Third, slopes were calculated only from waking phases and bedtime samples, excluding the cortisol awakening response, a unique HPA axis dimension whose removal may introduce measurement error and reduce assessment accuracy (Stalder et al., 2016). Relatedly, timing-compliance sensitivity analyses revealed that about one in five person-days showed collection-time protocol deviations exceeding an hour, and reanalysis in the timing-compliant subgroup produced 2 sign reversals and 7 changes in statistical significance. Fourth, this study did not examine lagged effects of daily stress on somatic symptoms or cortisol regulation on future

days, so the stress-cortisol coupling may function via carry-over mechanisms (Almeida, 2005). Fifth, we derived the standard errors for the three-way interaction from the Hessian matrix rather than using bootstrap or profile-likelihood approaches, and we observed a boundary singularity in the three-way interaction model's random-effects structure. Thus, the three-way interaction should be interpreted with caution. Sixth, sensitivity analyses suggested that the three-way interaction reversed sign and was no longer statistically significant under standard multiple imputation, indicating limited robustness. Seventh, results differed across stress definitions; the average stressor severity was regarded as the primary definition, as it models within-person mean daily stress intensity, and stressor count functioned as a robustness check rather than a post-hoc replacement. Eighth, the sample was predominantly White (86.4%), and because diurnal cortisol rhythms vary across racial groups, generalizability to other groups is likely limited (DeSantis et al., 2015). Finally, data collection procedures took place between 2004 and 2006, and cohort effects may constrain generalizability to younger populations.

Despite these shortcomings, the current study provides noteworthy strengths. It recruited a large, community-dwelling adult sample with a daily diary design, facilitating stringent decomposition of between- and within-person stress pathways. Distributional diagnostics indicated negligible residual overdispersion under the Tweedie specification, supporting the primary analytic approach. The integration of simultaneous between- and within-person stress indicators also permitted more sophisticated tests of stress-vulnerability pathways than designs relying on a single stress indicator.

If replicated, some translational insights merit consideration. Clinically, detecting diurnal cortisol slope as a plausible stress-risk indicator highlights the importance of HPA axis profiling for vulnerability subgrouping in primary care (Adam et al., 2017). Patients with heightened somatic symptom burden can gain from prevention programs and treatments addressing stress recovery and sleep hygiene to promote healthier cortisol regulation. The buildup of stress underscores the need for population-level strategies, such as community-based lifestyle promotion and occupational wellness programs, to prevent future allostatic burden. JITAIs can serve as adjunctive strategies by providing real-time coping skills embedded in routine contexts on high-stress days, especially for adults displaying psychophysiological dysregulation (Zainal et al., 2025).

To conclude, this project found that flatter diurnal cortisol slopes were associated with greater within-person coupling between daily stressor count and somatic symptoms, a relationship further moderated by stress buildup. These outcomes align with diathesis-stress and allostatic load models and emphasize the interplay between trait-level psychophysiological modulation and both daily and persistent stress exposure in influencing somatic health. Future studies that integrate day-level cortisol slopes, lagged physiological mechanisms, and proinflammatory activity markers would considerably advance mechanistic comprehension.

CRediT authorship contribution statement

Matthew C.H. Lian: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Nur Hani Zainal:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.psyneuen.2026.107984](https://doi.org/10.1016/j.psyneuen.2026.107984).

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