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# Overestimating Self-Blame for Stressful Life Events and Adolescents' Latent Trait Cortisol: The Moderating Role of Parental Warmth

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## Conflict of Interest

In the interest of full disclosure, D.A.G. is founder and Chief Scientific and Strategy Advisor of Salimetrics LLC (State College, PA) and SalivaBio LLC (Baltimore, MD). The other authors declare that they have no conflict of interest.

## Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Williams College Institutional Review Board and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

## Informed Consent

Informed consent was obtained from all individual participants included in the study.

## Abstract

Cognitive interpretations of stressful events impact their implications for physiological stress processes. However, whether such interpretations are related to trait cortisol—an indicator of individual differences in stress physiology—is unknown. In 112 early adolescent girls (M age = 12.39 years), this study examined the association between self-blame estimates for past year events and latent trait cortisol, and whether maternal warmth moderated effects. Overestimating self-blame (versus objective indices) for independent (uncontrollable) events was associated with lower latent trait cortisol, and maternal warmth moderated the effect of self-blame estimates on latent trait cortisol for each dependent (at least partially controllable) and interpersonal events. Implications for understanding the impact of cognitive and interpersonal factors on trait cortisol during early adolescence are discussed.

## Introduction

Research suggests that stressful experiences contribute to internalizing psychopathology by altering hypothalamic pituitary adrenal (HPA) axis activity (Stroud et al. 2019). Importantly, individuals' cognitive interpretations of stressful experiences shape their physiological consequences (e.g., Dickerson and Kemeny 2004), and risk for internalizing psychopathology following their occurrence (Alloy et al. 2012). Yet, few studies have tested the physiological effects of interpretations of naturally-occurring stressful life events. Furthermore, prior work has not examined the link between cognitive interpretations of events and individual differences in a trait-like indicator of HPA axis activity. Finally, whether interpersonal factors moderate the physiological effects of such interpretations is unknown. To address these gaps, we tested whether overestimates of self-blame for naturally-occurring stressful life events are related to early adolescent girls' latent trait cortisol, and whether maternal warmth moderates this effect.

Given that adolescence has been identified as a second "sensitive period" for adult health and development (Del Giudice et al. 2011), it may be particularly informative for understanding the etiology of internalizing psychopathology to pursue these questions during adolescence, and in early adolescence in particular, prior to mid-adolescence, a period of high risk for the development of diagnosable depressive (e.g., Rohde et al. 2009) and anxiety (e.g., Costello et al. 2003) disorders. Moreover, early adolescent girls have higher rates of anxiety (e.g., Wittchen et al. 1998) and depressive (e.g., Rohde et al. 2009) symptoms (versus early adolescent boys) in part due to gender differences in cognitive vulnerabilities (e.g., Hankin and Abramson 2002) and in HPA axis functioning (e.g., Gunnar et al. 2009). Thus, further understanding of the interplay of cognitive, interpersonal, and neuroendocrine factors among early adolescent girls may be especially informative for prevention efforts as well as for elucidating their roles in the early trajectory to internalizing psychopathology.

## Cognitive Vulnerability, Stressful Life Events, and HPA Axis Activity

Central to cognitive vulnerability-stress models of depression is the notion that the ways in which individuals appraise stressful life events shape their susceptibility to depression following event exposure (e.g., Abramson et al. 1989). Myriad cognitive vulnerabilities have been identified, such as the tendency to make internal, stable, and global attributions for the causes, consequences, and self-implications of events (i.e., negative cognitive style; Abramson et al. 1989); negative schemas regarding the self, world, and future (Beck 1976); rumination about stressful life events (Brosschot et al. 2006); and the tendency to overestimate self-blame, particularly for uncontrollable events (e.g., Power 2004). Supporting these models, cognitive vulnerabilities predict depressive symptoms when examined alone, and in interaction with stressful life events, among both adolescents (e.g., Lakdawalla et al. 2007) and adults (e.g., Hu et al. 2015). Furthermore, during early adolescence when cognitive vulnerabilities may still be consolidating, they appear to have implications for both depressive and anxiety symptoms (e.g., Alloy et al. 2012).

One potential pathway through which cognitive vulnerabilities confer risk for psychopathology is through alterations in stress physiology (e.g., McEwen 1998a). For example,

appraisal theory posits that stressors activate specific cognitive and affective processes (e.g., negative self-related cognitions and emotions), which mediate the effects of stress on physiological systems (e.g., Dickerson and Kemeny 2004). Indeed, research suggests that various cognitive vulnerabilities are related to HPA axis activity among children (e.g., Hayden et al. 2014), adolescents (e.g., Shapero et al. 2017), and adults (e.g., Ottaviani et al. 2016). For instance, rumination (a perseverative cognitive style; Nolen-Hoeksema 1991) has been linked with flatter diurnal cortisol slopes (Hilt et al. 2017) and lower cortisol awakening responses (CAR; Kuehner et al. 2007). Day-to-day variation in cognitive vulnerabilities also predict diurnal indicators the subsequent day. For example, greater daily appraisals of stressfulness have been linked to reductions in the next day CAR (Gartland et al. 2014). Further, cognitive vulnerabilities are related to youth's reactivity to laboratory-based stressors (Hayden et al. 2014).

Despite the contribution of this work, there are gaps in our understanding of the link between cognitive vulnerabilities and HPA axis activity. First, few studies have tested the physiological consequences of cognitive appraisals of naturally-occurring stressful life events. Instead, most research has focused on one's general tendency to interpret hypothetical events via self-report (e.g., negative cognitive style), rather than on interpretations of specific events, and on physiological responses to laboratory-based, as opposed to naturally-occurring, stressors. Second, few studies have examined the physiological implications of attributing self-blame for events despite evidence that (a) depressed youth exhibit higher levels of self-blame appraisals (e.g., Krackow and Rudolph 2008); and (b) self-blame appraisals predict subsequent internalizing psychopathology (e.g., Lucas-Thompson et al. 2017). The two studies that have examined self-blame appraisals have focused on felt responsibility for parents' marital conflict: self-blame appraisals were associated with flattened cortisol slopes (Lucas-Thompson and Hostinar 2013), which in turn predicted adolescents' internalizing behavior (Lucas-Thompson et al. 2017). Whether self-blame appraisals for other types of stress have implications for HPA axis activity has not been addressed. Third, prior work has not evaluated the accuracy of self-blame appraisals. Thus, although prior work suggests that feeling greater responsibility for stressful circumstances is related to HPA axis activity (e.g., Lucas-Thompson and Hostinar 2013), whether overestimating one's blame for stressful circumstances is related to HPA axis activity is unknown. Importantly, research (e.g., Krackow and Rudolph 2008) and theory (e.g., Beck 1976) indicate that depressed individuals make negatively biased interpretations. To address these gaps, the present study tested the link between self-blame appraisals for naturally-occurring stressful life events and adolescents' HPA axis activity. For each event that occurred over one year, the difference between adolescent's perception of their contribution to the event's occurrence and an objective rating of their contribution (coded by a blind independent coding team) was computed; the differences were then aggregated to index self-blame estimates (adapted from Krackow and Rudolph 2008).

This study also sought to address a final gap in our understanding of the link between cognitive risk and HPA axis activity: no prior investigation has evaluated links between cognitive vulnerabilities and trait cortisol. As such, whether cognitive vulnerabilities are related to enduring differences in HPA axis activity is unknown. Many of the cortisol indices used in prior work examining cognitive factors and HPA axis activity have limited stability over periods

greater than one month (e.g., CAR; diurnal slope; Doane et al. 2015). Further, most of the variance in diurnal indicators over a 9-month period reflects day-to-day variation (CAR; 82.30%; diurnal slope: 81.25%), rather than within-person cross-wave change (CAR: 3.22%; diurnal slope: 0.78%) or stable individual differences (CAR: 14.48%; diurnal slope: 17.97%; Doane et al. 2015). Thus, these indices mostly tap day-to-day or cross-wave fluctuations within individuals, rather than stable individual differences in HPA axis activity.

To quantify stable individual differences, researchers have used modeling approaches that use the correlations among cortisol samples collected across several days in reference to the grand mean to construct latent trait cortisol factors (e.g., Doane et al. 2015). The cortisol samples are used as indicators on a latent trait cortisol factor, which captures individual differences by drawing upon the samples' shared variance. Notably, one study showed that 71–88% of the variability in 3 within-wave LTC indicators (captured over 9 months) was explained by an across-wave latent trait cortisol factor, suggesting that the latent trait cortisol indicators exhibited some stability, and largely reflected individual differences (i.e., between-persons variability) in HPA axis activity (Doane et al. 2015).

Investigating whether self-blame appraisals are related to stable individual differences in HPA axis activity aligns with theoretical models linking cognitive factors and physiological stress processes. Indeed, the allostatic load model posits that allostatic states, which reflect changes in the set points of the HPA axis system, occur as a result of altered or prolonged activity of the HPA axis in response to stress (e.g., McEwen 1998a). That latent trait cortisol mostly taps individual differences in cortisol suggests it may be a more useful indicator of set points, relative to other cortisol indicators in the allostatic load index (e.g., CAR, diurnal slope; Stroud et al. 2016b), which mostly reflect day-to-day variation (Doane et al. 2015). Thus, testing the link between self-blame estimates and latent trait cortisol permits the evaluation of whether cognitive representations of events shape the extent to which events are associated with alterations in the set points regulating the HPA axis. Overestimating self-blame for events may prolong activation of cognitive representations of past events (Brosschot et al. 2006) or alter the physiological consequences of events (e.g., Dickerson and Kemeny 2004), thereby extending their physiological effects, which overtime may result in set point alterations (e.g., McEwen 1998a). Consistent with the allostatic model (e.g., McEwen 1998a), prior work in this sample indicates that various types of stress, including early adversity (Stroud et al. 2016a), and past year acute and chronic stress (Stroud et al. 2016b), are each associated with latent trait cortisol. However, whether individuals' interpretations of stressful experiences are related to latent trait cortisol remains unaddressed.

## The Moderating Effect of Maternal Warmth

Parenting has a lasting impact on youths' stress-response systems, including the HPA axis (e.g., Gunnar and Quevedo 2007). Although many parenting constructs have been associated with youths' HPA axis activity, parental warmth has received substantial support (though see, Hackman et al. 2018), including in adolescent samples (e.g., Luecken et al. 2016). Fewer studies have examined whether parenting is related to trait cortisol, but two studies support a link (Booth et al. 2008; Essex et al. 2011), and one does not (Thompson et al. 2018). In addition to direct

links between parenting and HPA axis activity, research suggests that adaptive parenting can also mitigate the effect of stress on HPA axis activity, consistent with the social buffering model (Hostinar and Gunnar 2013). Indeed, parental warmth buffers the effect of different types of stress on adolescents' cortisol reactivity (e.g., Hagan et al. 2011), daily diurnal cortisol activity (e.g., Lippold et al. 2016), and allostatic load, including measures of HPA axis activity (e.g., Evans et al. 2007). Thus, evidence suggests that parenting characterized by high levels of warmth may assist adolescents in regulating their physiological responses to stress.

Despite support for the moderating effect of parental warmth among adolescents, knowledge gaps highlight the need for further work. First, the social buffering model (Hostinar and Gunnar 2013) posits that repeated buffering may lead to "alterations in the set points of the HPA axis," but this has not been directly tested (p. 566). Second, limited evidence (Hostinar and Gunnar 2013) and contradictory findings (Hostinar et al. 2015) have left questions about whether parents serve as social buffers during adolescence (e.g., Hostinar and Gunnar 2013). As noted (Hostinar and Gunnar 2013), this gap is critical to address as adolescence is characterized by changes in the HPA axis (e.g., Del Giudice et al. 2011) and in the relative importance of parental relationships (Steinberg 1987). Furthermore, early adolescent girls face increasing stress levels (Rudolph and Hammen 1999), and their cognitive interpretations of events are solidifying (e.g., Alloy et al. 2012); thus, it is important to address whether parental warmth buffers the effect of self-blame estimates on latent trait cortisol among early adolescent girls.

## The Present Study

The present study examined whether adolescents' estimates of self-blame for past year stressful life events were associated with latent trait cortisol, and whether maternal warmth moderated this effect. Based upon prior work in this sample demonstrating that lower latent trait cortisol predicts increases in subsequent internalizing psychopathology (Stroud et al. 2019), as well as prior research (e.g., Lucas-Thompson et al. 2017) and theory (e.g., McEwen 1998a), a greater tendency to overestimate self-blame (relative to objectively coded indices) was expected to be associated with lower latent trait cortisol. Based upon the social buffering model (Hostinar and Gunnar 2013) and prior work documenting the protective effect of maternal warmth (e.g., Lippold et al. 2016), maternal warmth was expected to moderate the effect of self-blame estimates on LTC, such that greater self-blame estimates would only be related to lower latent trait cortisol for girls with lower maternal warmth.

The present study also examined whether links between self-blame estimates and latent trait cortisol differed based upon the interpersonal status and controllability of the events. Prior work has identified two dimensions of acute stress that shape its physiological (e.g., Dickerson and Kemeny 2004) and psychological (e.g., Stroud et al. 2011) consequences: controllability and interpersonal status. Prior work in this sample demonstrated that links between past year acute stress and latent trait cortisol varied as a function of event independence (i.e., the degree to which the person contributed to the event's occurrence) and interpersonal status (Stroud et al. 2016b). Researchers have also theorized that the physiological consequences of stress depend upon the stressor type and the way in which it is appraised (Kemeny 2003). Thus, links between

self-blame estimates and LTC were expected to vary according to event independence and interpersonal status, but due to lack of prior work, these analyses were exploratory.

Finally, the present study explored whether links between self-blame estimates and latent trait cortisol remained when accounting for the effects of early adversity, past year chronic stress, and current and lifetime history of depressive symptoms. Prior work has demonstrated that early adversity (Stroud et al. 2016a), chronic stress (Stroud et al. 2016b), and psychopathology (Stroud et al. 2019) are each related to LTC in this sample. Moreover, cognitive vulnerabilities are shaped by prior stress (e.g., Alloy et al. 2004) and depression (e.g., LaGrange et al. 2011). As such, it is important to investigate whether links between self-blame estimates and LTC remain after adjusting for the effects of these potential third variables. These tests were exploratory given lack of prior work.

## Method

### Participants and Procedure

Participants were 122 early adolescent girls who completed the saliva sampling portion of a larger study examining biopsychosocial predictors of psychopathology (N = 132).Footnote1 Participants and their primary female caregivers (herein "mother") were recruited from 2 New England counties via advertisements/flyers; word-of-mouth; and local schools. Participants who completed the saliva collection with low compliance (n = 5; no valid 30 mi post-waking samples; see below) or whose mothers did not complete the measure of warmth measure (n = 5) were excluded. The 112 included participants (M age = 12.39 [SD = 0.77] years, 90.2% White) were mostly middle to upper class (<$40,000 [19.6%]; $41,000–$60,000 [17.9%]; $61,000–$100,000 [25.6%]; >$100,000 [36.6%]).Footnote2

Adolescents and their mothers completed a laboratory visit during which they provided assent and consent (respectively), and each separately completed interviews and questionnaires. Mothers also completed the Five-Minute Speech Sample (Magaña et al. 1986). During the laboratory visit, instructions and materials for saliva collection were also provided. Approximately 1 week after the visit (M = 7.48 days; SD = 8.86), adolescents collected whole saliva by passive drool 3 times per day (waking, 30 min post-waking, bedtime) on 3 consecutive weekdays, avoiding atypical days (e.g., vacations, birthdays). For each sample, adolescents recorded the time and completed a diary assessment (e.g., time of waking; affect; perceived stress, caffeine use, and nicotine use in the hour prior to sampling). Samples were returned via mail; stored at −20 °C; and sent to the Biochemisches Labor at the University of Trier, Germany to be assayed. Compliance was assessed with a MEMS 6TM (Aardex; Aardex Group, Richmond, VA) track cap. On average 8.66 (SD = 0.82) samples were provided per adolescent and 84 (75.0%) used the track cap. All procedures were approved by the Williams College Institutional Review Board.

### Measures

### Cortisol

Samples were assayed in duplicate, using a solid phase time-resolved fluorescence immunoassay with fluorometric endpoint detection (DELFIA; Dressendörfer et al. 1992). Intra-assay coefficients of variation were 4.0–6.7%. Inter-assay coefficients of variation were 7.1–9.0%. Waking samples: Ms = 0.23–0.26 μg/dl, SDs = 0.14–0.16. 30 min past-waking samples: Ms = 0.32–0.39 μg/dl, SD = 0.17–0.19.

**Acute stress**

The UCLA Life Stress Interview (adapted from Rudolph and Hammen 1999; Rudolph et al. 2000) assessed adolescents' past year acute stress (i.e., events with a brief onset and short duration). Mothers and daughters completed separate interviews with the same interviewer, and interviewers were blind to other data. For each event, participants provided information about its surrounding context (e.g., circumstances), duration, and consequences to obtain the degree of impact for a typical individual given the context (i.e., objective impact). Interviewers prepared narrative accounts of each event (detailing the context, but excluding participants' subjective reactions) that were presented to an independent rating team, comprising trained and reliable interviewers who were blind to all other data, including participants' subjective perceptions of events. As in prior work (e.g., Rudolph et al. 2000), when mothers and daughters reported the same event, information from mothers and adolescents was combined into a single narrative. If only one reported the event, the narrative reflected only her report.

For each event, the team rated: (a) objective impact (1 [no negative impact] to 5 [extremely severe negative impact]; half-points permitted); (b) interpersonal status (coded 1/0; rated interpersonal when the primary context involved relations with others or affected the participants' relations); and (c) independence (degree to which the event resulted from the participant's behavior; 1 [fully independent of the person's behavior] to 5 [fully dependent on the person's behavior]; half-points permitted). A second team (blind to original ratings) rerated a set of events n = 132; impact intraclass correlation [ICC] = 0.92; interpersonal status ICC = 0.98; independence ICC = 0.99. Following prior work (Stroud et al. 2011), events were classified as: (1) interpersonal (e.g., break-up); (2) non-interpersonal (e.g., academic failure); (3) independent (i.e., occurred independently of the adolescent's behavior; events rated ≤2.5; e.g., parent's job loss); and (4) dependent (i.e., at least partially due to the adolescent's behavior; events rated ≥3; e.g., conflict).

**Self-blame estimates**

During the Life Stress Interview with adolescent participants, after adolescents described an event and its surrounding context, they were asked to rate their contribution to the event: "On a 1 to 5 scale with 1 being not at all and 5 being completely, how much do you think you contributed to bringing about this event?" (half-points were permitted; adapted from Krackow and Rudolph 2008). For each event, the objective independence rating (coded by the independent rating team) was subtracted from adolescent's subjective rating of their contribution. To index self-blame overestimates, the difference in ratings was aggregated by event type (e.g., interpersonal, non-interpersonal, dependent, independent). Thus, higher scores indicate greater self-blame overestimates. In other words, 4 indexes of self-blame overestimates were created, one for each

event type (e.g., interpersonal, non-interpersonal, dependent, independent; see Krackow and Rudolph 2008 for a similar approach).Footnote3

### Maternal warmth

The Five-Minute Speech Sample assessed maternal warmth (Magaña et al. 1986). Mothers were told that the research assistant (who was not the person who conducted the LSI) would like to hear their thoughts and feelings about their daughter, and asked "to speak for 5 min, telling me what kind of person [child's name] is and how the two of you get along together." The samples were electronically recorded, and later rated by a research assistant who was blind to all other data. Consistent with prior work (e.g., Caspi et al. 2004), tone of voice, enthusiasm, affection, spontaneity, interest in, and enjoyment of the adolescent were used to rate level of global warmth expressed specifically about the adolescent from 1 (no warmth) to 5 (high warmth). A second coder, blind to the original ratings, re-rated 31.7% participants; ICC = 0.86.

## Covariates

### Demographic and health covariates

Potential covariates were: (a) time of waking; (b) race/ethnicity (White = 1; non-White = 0); (c) oral contraceptive use; (d) past hour caffeine use; (e) past hour nicotine use; (f) past hour perceptions of stress (1 [not at all]–5 [very much]); (g) average daily negative affect; (h) average daily positive affect; (i) age; (j) maternal education (less than a Bachelor's degree = 0; a Bachelor's degree or higher = 1; 66.1% coded 1); and (g) pubertal status. Average daily positive and negative affect were assessed using the Positive and Negative Affect Schedule (Watson et al. 1988). Adolescents were asked to report on the degree they felt a list of 10 positive (e.g., excited) and 10 negative (e.g., upset) emotions in the hour prior to sampling from 0 (not at all) to 4 (extremely); mean daily negative and positive affect variables were computed. At the laboratory visit, pubertal status was assessed via adolescent self-report using the Pubertal Development Scale (Petersen et al. 1988). The 5 items, which assess growth spurt in height, skin and body hair changes, breast development, and age at menarche, are rated on a 4-point scale, from no development (1) to development seems completed (4); except for menarche which is rated 1 or 4. The mean was used ($\alpha = 0.70$).

Due to limited frequency, oral contraceptive use (1.8%) and nicotine use (0%) were not included. All other day-level covariates that had significant effect on cortisol levels from corresponding days were retained in the LTC measurement model. In addition, person-level covariates that were significantly correlated with both self-blame estimates and LTC were included (see Table 1).

See Table 1 Bivariate correlations and descriptive statistics for person-level covariates, warmth, and self-blame estimates

See Table 2 Internal moderator analyses examining the effect of self-blame estimates on latent trait cortisol as a function of event status and levels of maternal warmth

### Early Adversity

Mothers and daughters were separately interviewed with the lifetime adversity section of the Youth Life Stress Interview (Rudolph and Flynn 2007) to assess exposure to 10 negative family experiences (e.g., death of a close family member, long separation from caregivers, parental separation/divorce) during adolescents' entire lives (but not the year prior to the interview; for details, see Stroud et al. 2016a). Based upon narratives (i.e., circumstances and the consequences of each adversity), an independent team (blind to all data) rated total adversity from 1 (no adversity) to 9 (extremely severe; inter-rater reliability: ICC = 0.99; n = 60).

### Past and current depressive symptoms

The Schedule for Affective Disorders and Schizophrenia for school-aged children-present and lifetime version (Kaufman et al. 1997) assessed adolescents' current (past month) and lifetime history of depressive symptoms. Symptoms were rated: 0 = no symptoms; 1 = mild symptoms (1–2 symptoms); 2 = moderate, subthreshold symptoms (3–4 symptoms); 3 = diagnosable disorder (e.g., Stroud et al. 2019); ICCs = 1.00 (current), 0.97 (past). The maximum of current and past ratings was used.

### Chronic stress

The Life Stress Interview (Rudolph and Hammen 1999; Rudolph et al. 2000) was also used to assess adolescents' exposure to chronic life stress (i.e., ongoing objective stress) over the past year. Mothers and daughters completed separate interviews with the same interviewer. Probes elicited behavioral descriptions of adolescents' ongoing stress in 7 domains (academics, academic behavior, parent-child relationship, close friends, peer social life, romantic relationships/dating, parents' marital/cohabiting romantic relationship [if applicable]). Using behavioral indicators, interviewers rated chronic stress level in each domain (1-excellent/optimal circumstances to 5-very bad circumstances, half-points permitted). The mean of the domain ratings derived from mothers and daughter interviews ($r = 0.81$, $ps < 0.001$) were combined by taking the mean of the mothers' and daughters' ratings for each domain. Inter-rater reliability: ICCs: M = 0.81 (0.70–0.91).

## Analytic Strategy

Preliminary analyses examined compliance with cortisol sampling. The 30 min post-waking sample was considered compliant if the self-reported time difference between the waking and the 30 min post-waking samples was between 23 and 37 min (Stroud et al. 2019). Samples out of this range were considered non-compliant (60 of 330 samples [18.18%]), and an additional 9 of 627 compliant cortisol values (3.17%) were outliers (i.e., 3 SDs from mean); non-compliant samples and outliers were treated as missing.

Primary analyses were conducted in Mplus 8 (Muthen and Muthen 1998–2018). First, confirmatory factor analysis (CFA) was used to model a latent trait cortisol using the waking and the 30 min post-waking cortisol samples from the three days of collection (e.g., Stroud et al. 2016a). Analyses were conducted with maximum likelihood estimation with robust standard errors (MLR) and full information maximum likelihood (FIML) was used to handle missing data (Savalei and Rhemtulla 2012). Using the correlations between the samples, the latent trait

cortisol indicator was derived by drawing upon the commonalities among cortisol samples in reference to the grand mean. Because the bedtime samples were not significantly correlated with the morning samples, they were not used to construct the latent trait cortisol (e.g., Stroud et al. 2016a).Footnote4 Potential day-level covariates were added to the model one at a time and those that were not significantly associated with the corresponding 30 min post-waking cortisol indicators were trimmed. Based upon the modification indices suggested by Mplus, the errors of some samples were allowed to correlate. Model fit was assessed with the chi-square test (a p-value > 0.05 indicates good fit), the Comparative Fit Index (CFI; >0.90 indicates good fit), and the Root Mean Square Error of Approximation (RMSEA; <0.05 indicates good fit; <0.08 indicates adequate fit; L. Hu and Bentler 1998). Second, bivariate correlations investigated whether the person-level covariates (e.g., pubertal status) were significantly associated with self-blame estimates and latent trait cortisol. None of the covariates were simultaneously correlated with latent trait cortisol and self-blame estimates for different event types, and thus no person-level covariates were included (see Table 1).

Third, the internal moderator approach was used to examine the effect of self-blame estimates on latent trait cortisol for those who reported at least one event versus those who did not report any events (i.e., event status) at varying levels of maternal warmth (Mirowsky 1999). This approach is advantageous as: (1) it retains participants in the analyses regardless of whether or not they have experienced an event, thereby preserving sample size and reducing missing data (e.g., Chen and Jaffee 2018); and (2) it uses a continuous measure of self-blame estimates and maternal warmth (versus categorizing participants as high or low). As in prior work (e.g., Chen and Jaffee 2018), self-blame estimates were standardized so participants with at least one event have a mean of 0 and a standard deviation of 1, and all participants without an event were coded 0 (i.e., event status = 0). To test the effect of self-blame estimates, the interaction term of self-blame estimates and event status, but not the main effect of self-blame estimates, was included. By coding those without an event 0 on self-blame estimates and including only the interaction term, those without an event serve as a comparison group in all models. Thus, the interaction term reflects how much those with and without events differ on latent trait cortisol at the mean level of self-blame estimates. By testing whether the interaction term (self-blame estimates and event status) differed from 0, we examined whether latent trait cortisol differences between those with and without at least one event were conditional on level of self-blame estimates. The main effect of warmth, the interaction between the maternal warmth and event status, and a three-way interaction of self-blame by warmth by event status were also included. The latter coefficient was only estimated using data with non-zero event status, and tests whether the effect of self-blame estimates on latent trait cortisol is moderated by maternal warmth.

To retain all cases, dummy variables for event status (with at least one event = 1, without at least one event = 0) were created, and whether there was a difference in latent trait cortisol (captured by β3 below) between the groups was tested. Separate models were conducted for each dimension of event type (interpersonal, independence). Event status was coded by event type. Thus, for example, in the independence model, there were 2 dummy variables for event status (one for independent events and one for dependent events).

For example, for model 1:

$$Y=\beta_0+\beta_1\times SBest_{Ind}\times Group_{ind}+\beta_2\times SBest_{Dep}\times Group_{Dep}+\beta_3\times Group_{Ind}+\beta_4\times Group_{Dep}+\beta_5\times SBest_{Ind}\times Group_{Ind}\times warmth+\beta_6\times SBest_{Dep}\times Group_{Dep}\times warmth+\beta_7\times warmth+\beta_8\times warmth\times Group_{Ind}+\beta_9\times warmth\times Group_{Dep}+\alpha X+\varepsilon$$

where Y is latent trait cortisol, $SBest_{Ind}$ and $SBest_{Dep}$ are self-blame estimates for independent and dependent events (respectively), $Group_{Ind}$ and $Group_{Dep}$ are independent and dependent event status (respectively), X is a vector of covariates, and α is a vector of parameter estimates for covariates. See appendix for further details.

Significant interactions were probed using the Johnson–Neyman procedure which identified the values of warmth at which self-blame estimates significantly predicted latent trait cortisol. In follow-up robustness tests, chronic stress, early adversity, and depressive symptoms were added to the models to rule out the possibility that these variables were driving the observed effects. Finally, post hoc power analyses using Monte Carlo simulation (Muthén and Muthén 2002) were conducted for models 1 and 2 (without covariates). For both models, we assumed the estimates from the full sample were the population parameters, and we generated 1000 random samples.

## Results

### Measurement Model: Latent Trait Cortisol

The waking and 30 min post-waking samples were used to construct the latent trait cortisol (e.g., Stroud et al. 2016b). None of the day-level covariates were significantly correlated with the corresponding 30 min post-waking indicators and thus, were trimmed. The error covariance between the waking samples on Days 2 and 3 was freely correlated, which significantly improved model fit (initial fit: $\chi2[9] = 16.70$, $p = 0.5$, CFI = 0.92, RMSEA = 0.09, $p = 0.16$; Satorra-Bentler scaled $\Delta\chi2[1] = 7.53$, $p < 0.01$). Final model fit indices were adequate, $\chi2(8) = 11.29$ ($p = 0.19$), CFI = 0.97, RMSEA = 0.06 ($p = 0.36$); all standardized factor loadings were ≥0.45 ($ps < 0.001$; Fig. 1).

*See Figure 1*

### Self-Blame Estimates for Independent and Dependent Events, Maternal Warmth, and Latent Trait Cortisol

In Model 1, for those who experienced at least one independent event, the effect of self-blame estimates for independent events on latent trait cortisol was significant, such that greater self-blame estimates for independent events were associated with lower latent trait cortisol ($\beta1 = -0.21$, SE = 0.11, $p = 0.046$) (Table 2). This effect was not moderated by maternal warmth ($\beta5 = -0.09$, SE = 0.12, $p = 0.431$). In contrast, among those who experienced at least one dependent event, the effect of self-blame estimates for dependent events on latent trait cortisol was not significant ($\beta2 = 0.13$, SE = 0.14, $p = 0.364$), but among those who reported dependent events, the interaction between self-blame estimates for dependent events and maternal warmth was significant ($\beta6 = 0.36$, SE = 0.13, $p = 0.007$). For those with maternal warmth levels of 4 or 5

(28.5% of the 66 participants with at least one dependent event), greater self-blame estimates for dependent events were associated with greater latent trait cortisol. Conversely, for those with maternal warmth levels of 1 or 2 (16.7%), greater self-blame estimates for dependent events were associated with lower latent trait cortisol (Fig. 2a). In a follow-up robustness test, when early adversity, chronic stress, and depressive symptoms were added to the model, findings remained significant, except for the effect of self-blame estimates for independent events on latent trait cortisol among those with at least one independent event ($\beta1 = -0.21$, SE = 0.11, $p = 0.061$). A post hoc power analysis (Muthén and Muthén 2002) indicated that there was 45% power to detect the effect of self-blame estimates for independent events on latent trait cortisol, and 88% power to detect the interaction between self-blame estimates for dependent events and maternal warmth.

*See Figure 2*

### Self-Blame Estimates for Non-Interpersonal and Interpersonal Events, Maternal Warmth, and Latent Trait Cortisol

In Model 2, for those who experienced at least one non-interpersonal event, the effect of self-blame estimates for non-interpersonal events on latent trait cortisol was not significant ($\beta1 = 0.11$, SE = 0.12, $p = 0.380$), and this effect was not moderated by maternal warmth ($\beta5 = 0.09$, SE = 0.09, $p = 0.283$) (Table 2). In addition, among those who experienced at least one interpersonal event, the effect of self-blame estimates for interpersonal events on latent trait cortisol was not significant ($\beta2 = -0.17$, SE = 0.09, $p = 0.065$). However, the interaction between self-blame estimates for interpersonal events and maternal warmth was significant ($\beta6 = 0.256$, SE = 0.10, $p = 0.016$). For those with maternal warmth levels of 3 or below (74.5% of the 94 participants with at least one interpersonal events), greater self-blame estimates for interpersonal events were related to lower latent trait cortisol (Fig. 2b). In a robustness test, when early adversity, chronic stress, and depressive symptoms were added to the model, all findings remained significant. A post hoc power analysis (Muthén and Muthén 2002) indicated that there was 64% power to detect the interaction between self-blame estimates for interpersonal events and maternal warmth.

## Discussion

Considerable evidence indicates that stressful experiences confer risk for subsequent psychopathology by altering the activity of the HPA axis (e.g., Stroud et al. 2019). Moreover, research suggests that cognitive interpretations of such experiences modify their physiological (e.g., Lucas-Thompson and Hostinar 2013) and psychological (e.g., Alloy et al. 2012) consequences. However, no prior study has examined whether cognitive interpretations of naturally-occurring stressful life events are related to trait cortisol, and whether interpersonal factors moderate the physiological effects of such interpretations. In a sample of early adolescent girls, the present study demonstrated that the tendency to overestimate self-blame (relative to objective indices) for past year independent (i.e., uncontrollable) events was related to lower latent trait cortisol—a novel indicator of individual differences in HPA axis activity. Moreover, maternal warmth moderated the effect of self-blame estimates on latent trait cortisol for each

dependent (i.e., at least partially controllable) and interpersonal events. Finally, self-blame estimates for non-interpersonal events were not related to latent trait cortisol, regardless of level of maternal warmth. Collectively, these results help to identify for whom and under what circumstances self-blame estimates confer risk for a pattern of HPA activity that prospectively predicts internalizing psychopathology (Stroud et al. 2019), and highlight maternal warmth and self-blame estimates as potential targets for prevention efforts designed to promote adaptive HPA axis activity among early adolescent girls.

### Overestimating Self-Blame for Independent Events is Associated with Lower Latent Trait Cortisol

The present findings suggest that overestimating self-blame for independent events is associated with lower latent trait cortisol, regardless of maternal warmth level. This finding adds to prior work which has shown that various cognitive vulnerabilities are associated with other indices of HPA axis activity, including cortisol reactivity (e.g., Hayden et al. 2014) and diurnal indicators (e.g., Lucas-Thompson and Hostinar 2013). Given that latent trait cortisol primarily indexes between-person variation in HPA axis activity and exhibits considerable within-person stability over short-term follow-ups (i.e., 9 months; Doane et al. 2015), this suggests that overestimating self-blame for independent events may have longer lasting effects on stress physiology, altering the set points regulating the HPA axis, and thereby influencing future health and psychological well-being (e.g., Juster et al. 2010). Moreover, in examining self-blame estimates (relative to objective indices) for events in adolescents' everyday lives, this finding builds upon prior work by suggesting that biased or inaccurate appraisals are related to HPA axis activity.

Collectively with prior work, results suggest that self-blame estimates shape the nature of the link between acute independent stress and latent trait cortisol. In a prior study in this sample, results indicated that a greater total severity of past year acute independent stress was positively related to adolescents' latent trait cortisol (Stroud et al. 2016b). Importantly, the severity ratings for each event were coded by an independent rating team who was blind to all data, including participants' subjective interpretations of events. Thus, a greater cumulative severity of independent events (coded objectively) is related to greater latent trait cortisol, but greater self-blame for those events is related to lower latent trait cortisol. Though research is needed to identify the mechanisms underlying this pattern of results, it aligns with theory and research. Acute stress exposure initially activates the HPA axis in an adaptive response process which mobilizes energy to cope with the stressor, and regulates other physiological systems (McEwen 1998a). The accumulation of acute stress would thus be reflected in a higher LTC (i.e., hypercortisolism; Stroud et al. 2016b). However, over time or in response to chronically stressful conditions, lower latent trait cortisol (i.e., hypocortisolism) develops (Miller et al. 2007). Indeed, prior research in this sample indicates that chronically stressful conditions, including chronic stress (Stroud et al. 2016b) and early adversity (Stroud et al. 2016a), are each related to lower latent trait cortisol. Drawing upon models of allostatic load and preservative cognition, it may be that attributing self-blame for uncontrollable stressors interferes with adaptive stress responses and/or recovery (McEwen 1998b), prolonging activation of past events, leading to chronic HPA axis activation (Brosschot et al. 2006). In other words, overestimating self-blame may render the

physiological effects of acute stress, akin to those of chronic ones, resulting in lower latent trait cortisol. Given lower latent trait cortisol predicts future internalizing symptoms in this sample (Stroud et al. 2019), these findings suggest that for independent acute stress, it's not the stress exposure—but its interpretation—that confers risk for HPA axis alterations that predict later internalizing psychopathology.

That overestimating self-blame for independent events, but not for other types of events, was associated with lower latent trait cortisol suggests that attributing self-blame for uncontrollable events may have particular implications for individual differences in HPA axis activity during early adolescence. This pattern of findings aligns with models of cortisol reactivity which posit that stressors influence physiological responses by activating specific cognitive and emotional processes; thus, the physiological consequences depend both on the type of stress and on its appraisal (e.g., Kemeny 2003). Drawing upon the allostatic load framework (e.g., McEwen 1998b), it is tempting to hypothesize that the "signature" pattern of cortisol reactivity generated by overestimating self-blame in response to uncontrollable stressors accumulates over time, resulting in lower latent trait cortisol. Future research should aim to elucidate why the link between overestimating self-blame and lower latent trait cortisol only emerged for independent events. One possibility is that this finding was influenced by the early adolescent developmental stage of participants. As in prior adolescent samples, many of the independent events adolescents experienced were focused on their parents (e.g., parental job loss, divorce), and likely would have been coded as dependent in an adult sample (e.g., Harkness et al. 2006). Thus, it may be that overestimating self-blame for these types of family events, over which adolescents have little control, is particularly detrimental for youths' physiological health and well-being. Although speculative, this explanation fits with theory (e.g., Grych and Fincham 1990) and research (e.g., Buehler et al. 2007) highlighting the role of youths' self-blame appraisals as one pathway linking parents' marital conflict (an uncontrollable stressor) and youth psychopathology. Thus, future work should address if this finding replicates across developmental periods. Nevertheless, the results underscore the need for theory and research focused on cognitive biases, HPA axis activity, and perhaps risk for psychopathology to distinguish between different types of stress.

Importantly, after accounting for the effects of early adversity, depressive symptoms, and chronic stress in a robustness test, the link between self-blame estimates for independent events and latent trait cortisol only approached significance. Because the magnitude of the effect remained the same in both models, reduced power in the robustness model may have contributed; thus, replication in a larger sample is needed. However, this may have been driven by the significant correlation between early adversity and latent trait cortisol (see Table 1), perhaps indicating that some of the impact of self-blame estimates for independent events on latent trait cortisol is due to its overlap with early adversity—a prediction supported by research and theory evincing links between early adversity and various cognitive vulnerabilities (for a review, see Alloy et al. 2004).

### The Moderating Effect of Maternal Warmth on the Relation between Self-Blame Estimates and Latent Trait Cortisol

The present results suggest that maternal warmth modifies the effect of overestimating self-blame for each dependent and interpersonal events on latent trait cortisol. Such findings add to prior work that has shown that parental warmth buffers the effect of various risk factors on adolescents' HPA axis activity, including cortisol reactivity (e.g., Hagan et al. 2011) and diurnal indicators (e.g., Lippold et al. 2016), and extends it by demonstrating that warmth can mitigate the effect of risk on enduring differences in HPA axis activity between individuals. Moreover, in a field focused on early childhood (Hostinar and Gunnar 2013), such findings suggest that mothers continue to serve as social buffers at least during early adolescence—a developmental period characterized by changes in HPA axis activity (Gunnar et al. 2009), increased sensitivity to the environment (Del Giudice et al. 2011), and increasing levels of stress (particularly for girls; Rudolph and Hammen 1999). This is important because mid-adolescence is a period of high risk for the development of internalizing psychopathology for girls (e.g., Rohde et al. 2009). Thus, future work should evaluate whether maternal warmth moderates the pathway from self-blame estimates to internalizing psychopathology through latent trait cortisol. Such an investigation would shed light on whether targeting maternal warmth in prevention efforts may protect adolescents from developing alterations in latent trait cortisol and internalizing psychopathology in the face of cognitive risk.

Interestingly, maternal warmth moderated the effect of self-blame estimates for interpersonal—but not non-interpersonal—events: among those with low and moderate maternal warmth (74.5%), greater overestimates of self-blame were associated with lower latent trait cortisol, a pattern of HPA axis activity that prospectively predicts internalizing psychopathology in this sample (Stroud et al. 2019). This pattern of findings fits with prior research and theory underscoring the salience of interpersonal stress for HPA axis activity (e.g., Dickerson and Kemeny 2004) and the development of internalizing psychopathology (e.g., Stroud et al. 2011), and further suggests that the moderating effect of maternal warmth on cognitive risk may vary by event type. Although the mechanisms underlying these results need to be elucidated in future work, meta-analytic evidence from laboratory studies of cortisol reactivity offers hints: stressors that contain social evaluative threat are associated with greater peak reactivity and delayed recovery (as compared to those without such threat; Dickerson and Kemeny 2004). Thus, overtime, this may lead to "wear and tear" on the HPA axis without the protective effect of high maternal warmth. Indeed, warm parents may buffer the effect of overestimating self-blame on latent trait cortisol by teaching and modeling the use of adaptive coping skills, including cognitive restructuring, which may help adolescents reappraise their level of self-blame (e.g., Power 2004), and by creating a family environment characterized by support, acceptance, and security (e.g., Morris et al. 2007). Thus, in the absence of high warmth, overestimating self-blame for interpersonal events may be associated with repeated and prolonged activation of the HPA axis, resulting in set point alterations (McEwen 1998a). Supporting this, prior work suggests that adolescents with lower caregiver warmth may have difficulty physiologically recovering from daily stressors. For example, results of one study showed that adolescents with lower caregiver warmth had flatter cortisol slopes, regardless of their level of daily stressors, whereas those with higher warmth had steeper cortisol slopes on days with lower (but not higher) levels of daily stressors (Lippold et al. 2016). These findings suggest that research evaluating whether maternal warmth buffers day-to-day links between self-blame estimates for

interpersonal events and HPA axis activity may shed light on how maternal warmth modifies the cumulative impact of such estimates on latent trait cortisol over longer time periods.

In addition, maternal warmth moderated the effect of self-blame estimates for dependent, but not independent, events in an unexpected pattern: among those with higher maternal warmth (28.5%), greater overestimates of self-blame for dependent events were associated with higher latent trait cortisol, suggesting that for those experiencing higher warmth, overestimates were associated with a potentially "protective" pattern of HPA axis activity. In contrast, among those with lower maternal warmth (16.7%), greater overestimates of self-blame were associated with lower latent trait cortisol. Because warm parents may promote the development of adaptive coping strategies (e.g., Morris et al. 2007), it may be that overestimating self-blame for dependent events (which were at least in part caused by adolescents' behavior; e.g., exam failure; conflict with a friend) elicits warm mothers to coach their daughters to use primary control coping strategies, such problem-solving and emotion regulation, which aim to directly alter the stressor itself or adolescents' response to the stressor (Connor-Smith et al. 2000). Dependent events by definition are at least partially the result of adolescents' behavior, and thus, they may be more readily resolved by these strategies (versus independent events where there was not find evidence of a moderating effect). Because these strategies have been linked with effective physiological regulation (e.g., Sladek et al. 2016) and may promote physiological recovery following acute stress exposure (e.g., Stewart et al. 2013), over time, if adolescents are adaptively coping with and recovering from dependent events, we might expect to see an adaptive change in the set points regulating their HPA axis, as reflected in higher LTC. Indeed, evidence from laboratory-based studies of cortisol reactivity (Kuhlman et al. 2014) and research examining day-to-day links between stressors and diurnal patterns indicate that warm parenting promotes physiological recovery (Lippold et al. 2016), an effect that has been hypothesized to be mediated by the tendency of warm parents to promote adaptive coping skills (e.g., Lippold et al. 2016). Given the small sample sizes in each region of significance (for those with at least one dependent event [n = 66], 16.7% in lower region and 28.8% in higher region), further research is needed to examine the moderating effect of maternal warmth on the relation between self-blame estimates for dependent events and latent trait cortisol, and if replicated, to elucidate the mechanisms underlying this pattern of findings.

## Limitations

Several limitations merit note. First, generalizability may be limited as the sample was small, self-selected, and comprised early adolescent girls who were mostly White. For example, research in adolescent samples indicates gender differences in the diurnal cortisol rhythm (e.g., Gunnar et al. 2009), as well as in links between cognitive vulnerability and depression (e.g., Hankin and Abramson 2002), and parenting and trait cortisol (Booth et al. 2008). Thus, findings may not replicate in boys. Second, self-report, rather than objective (e.g., actigraphs), measures of waking were used; this may have led to less precise indicators of compliance with the sampling protocol. Third, replication in a high-risk sample of adolescents facing higher levels of stress and greater variance in levels of maternal warmth is needed. Fourth, although it was ensured that stressors occurred prior to the saliva collection, and robustness models adjusted for

the effects of early adversity, past year chronic stress, and lifetime and current depressive symptoms, given the cross-sectional design, other third variables may have driven the results. Fifth, although a gold-standard measure of life stress was used, it was retrospective, which may have introduced recall bias. Fifth, our cortisol sampling protocol was three days at one measurement occasion; thus, the extent to which the trait indicator will exhibit stability over time is unknown. Finally, this investigation focused on only 2 potential moderators of the link between self-blame estimates and latent trait cortisol (i.e., maternal warmth, and event type); future research should aim to delineate other factors that may enhance or mitigate the effect of cognitive risk on adolescents' HPA axis activity. It may be especially fruitful to focus on factors that have been previously shown to influence adolescents' cortisol responses to laboratory-based stressors, such as coping strategies (e.g., distraction, problem-solving; Stewart et al. 2013) and qualities of other interpersonal relationships (e.g., friendships; Calhoun et al. 2014).

## Conclusion

Cognitive interpretations of stressful life events shape individuals' susceptibility to psychopathology following event exposure (e.g., Alloy et al. 2012) in part through alterations in physiological stress processes (e.g., Lucas-Thompson et al. 2017). No prior study has examined whether cognitive interpretations of stressful life events are related to latent trait cortisol—an indicator of individual differences in HPA axis activity (Doane et al. 2015). Moreover, prior work has not investigated whether interpersonal factors and event type modify the effect of such interpretations on HPA axis activity. Using latent trait cortisol to tap individual differences in HPA axis activity, the present findings suggest that overestimating self-blame for independent events is associated with a pattern of HPA axis activity that predicts later internalizing psychopathology (Stroud et al. 2019). Moreover, maternal warmth modified the impact of self-blame estimates on latent trait cortisol for certain types of events (interpersonal, dependent), but not others (non-interpersonal, independent). That maternal warmth emerged as a moderator suggests that even the set points regulating the HPA axis remain responsive to parenting during early adolescence, supporting the use of interventions to modulate HPA axis activity during this developmental period. Given overestimations of self-blame in part contribute to the development of internalizing psychopathology via HPA axis activity (Lucas-Thompson et al. 2017), the present findings highlight the importance of targeting maternal warmth to promote adaptive HPA axis regulation with the goal of interrupting the early trajectory to internalizing psychopathology. Findings also support the use of current evidence-based therapeutic interventions that include strategies to challenge cognitive interpretations of stressful life events as well as to effectively cope with such events (e.g., cognitive behavioral therapy, interpersonal psychotherapy). Moreover, results suggest that prevention efforts should focus on interpretations of, and developing coping strategies for, specific types of events (Stroud et al. 2011), particularly uncontrollable ones. The next step is to examine whether latent trait cortisol serves as a mechanism through which self-blame estimates contribute to internalizing psychopathology, and whether maternal warmth moderates this etiological pathway.

**Notes**

1. There were not significant differences between those who did and did not complete the cortisol assessment on age, income, pubertal status, acute stress, chronic stress, or early adversity (ps > 0.10).
2. Three siblings of participants and two fathers participated in the study. However, all results remained the same when these individuals were excluded from the analyses.
3. Each event is classified as independent or dependent, and interpersonal or non-interpersonal. Thus, for example, some of the independent events are interpersonal and some are non-interpersonal. For analyses, there were not sufficient numbers of events to examine self-blame estimates for events stratified by two dimensions (e.g., independent interpersonal events).
4. In a prior paper in this sample (Stroud et al. 2016a), all 9 samples were used to derive latent trait cortisol. Model fit was unfavorable and none of the factor loadings of bedtime cortisol were significant, suggesting that the bedtime samples were not suitable for constructing the latent trait cortisol. For detailed discussion of why the bedtime samples may not be correlated with the morning samples, see Stroud et al. 2016a, and Doane et al. 2015.

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## Appendix

Because groups were dummy coded for event status, Eq. (1) can be simplified into 4 equations with the parameters estimated based on subsets of participants:

For GroupInd = 0 and GroupDep = 0: $Y=\beta7\times warmth+\alpha X+\varepsilon$.

For GroupInd = 0 and GroupDep = 1:
$Y=\beta2\times SBestDep+\beta4+\beta6\times SBestDep\times warmth+\beta7\times warmth+\beta9\times warmth+\alpha X+\varepsilon$.

For GroupInd = 1 and GroupDep = 0:
$Y=\beta1\times SBestInd+\beta3+\beta5\times SBestInd\times warmth+\beta7\times warmth+\beta8\times warmth+\alpha X+\varepsilon$.

For GroupInd = 1 and GroupDep = 1:

$Y=\beta1\times SBestInd+\beta2\times SBestDep+\beta3+\beta4+\beta5\times SBestInd\times warmth+\beta6\times SBestDep\times warmth+\beta7\times warmth+\beta8\times warmth+\beta9\times warmth+\alpha X+\varepsilon$.

(2)

In Eq. (2), β1 and β2 = the effects of self-blame estimates on LTC for GroupInd = 1 and for GroupDep = 1, respectively; β3 = LTC difference between GroupInd = 1 versus GroupInd = 0; β4= LTC difference between GroupDep = 1 versus GroupDep = 0; β5 and β6 = interaction of self-blame estimates and maternal warmth for independent events and for dependent events, respectively; β7 = the effect of warmth on LTC for those with no events; β8 = difference in the effect of warmth on LTC between those with at least one independent event only (GroupInd = 1 and GroupDep = 0) versus those with no events (GroupDep = 0 and GroupInd = 0); β9 = difference in the effect of warmth on LTC between those with (GroupDep = 1 and GroupInd = 1) versus without (GroupInd = 1 and GroupDep = 0) events. The models for interpersonal and non-interpersonal events were constructed in the same way.

**Table 1 Bivariate correlations and descriptive statistics for person-level covariates, warmth, and self-blame estimates**

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Latent trait cortisol[a] | 1 | | | | | | | | | | | | |
| 2. Self-blame estimates for interpersonal events | −0.22* | 1 | | | | | | | | | | | |
| 3. Self-blame estimates for non-interpersonal events | 0.06 | 0.34** | 1 | | | | | | | | | | |
| 4. Self-blame estimates for independent events | −0.23* | 0.68** | 0.62** | 1 | | | | | | | | | |
| 5. Self-blame estimates for dependent events | −0.02 | 0.49** | 0.32* | 0.06 | 1 | | | | | | | | |
| 6. Maternal warmth | 0.12 | −0.11 | −0.01 | −0.06 | 0.04 | 1 | | | | | | | |
| 7. Maternal education | −0.02 | 0.02 | −0.22 | −0.22* | 0.18 | 0.01 | 1 | | | | | | |
| 8. White | 0.14 | 0.06 | −0.02 | 0.00 | 0.17 | −0.04 | 0.02 | 1 | | | | | |
| 9. Age (in years) | −0.04 | 0.02 | −0.14 | 0.01 | 0.13 | 0.03 | 0.11 | 0.27** | 1 | | | | |
| 10. Pubertal status | 0.04 | 0.02 | 0.06 | 0.10 | 0.14 | −0.03 | −0.10 | −0.05 | 0.40** | 1 | | | |
| 11. Early adversity | −0.23* | 0.05 | −0.15 | 0.08 | 0.00 | −0.06 | −0.36** | −0.15 | −0.05 | 0.17 | 1 | | |
| 12. Past and current depressive symptoms | −0.04 | 0.08 | 0.11 | 0.04 | 0.13 | −0.10 | 0.00 | 0.04 | 0.11 | 0.19* | 0.12 | 1 | |
| 13. Chronic stress | −0.17 | −0.03 | −0.15 | −0.04 | −0.05 | 0.28** | −0.22* | −0.10 | 0.05 | 0.19* | 0.46** | 0.21* | 1 |
| *n* | 112 | 94 | 65 | 95 | 66 | 112 | 112 | 112 | 112 | 112 | 112 | 112 | 112 |

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean | 0 | 0.31 | 0.55 | 0.79 | −0.79 | 3.09 | 0.66 | 0.90 | 12.39 | 2.68 | 4.09 | 0.45 | 1.96 |
| Standard deviation | 0.07 | 0.97 | 1.90 | 1.17 | 1.19 | 0.73 | 0.48 | 0.30 | 0.77 | 0.59 | 2.16 | 0.85 | 0.45 |

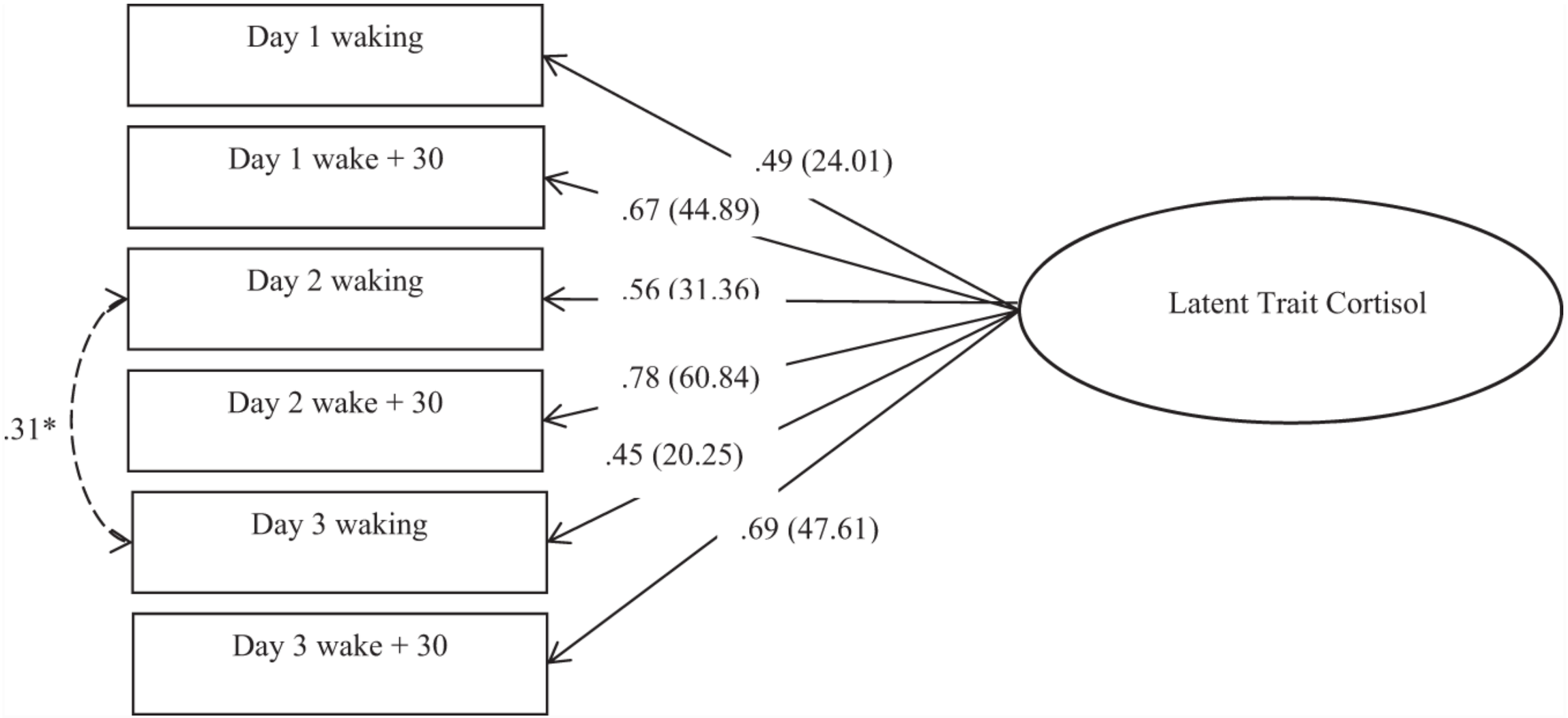
1. [a]Latent trait cortisol is saved output from M*plus* using the Regression Method for factor score determination. *N*s vary for self-blame estimates according to whether or not participants had the target event
2. $^{**}p < 0.01$, $^{*}p < 0.05$

**Table 2 Internal moderator analyses examining the effect of self-blame estimates on latent trait cortisol as a function of event status and levels of maternal warmth**

| | **Model 1 *β*(*SE*) GRP1 = independent GRP2 = dependent** | ***p*** | **Model 2 *β*(*SE*) GRP1 = non-interpersonal GRP2 = interpersonal** | ***p*** |
|---|---|---|---|---|
| Self-blame estimates by GRP1 ($\beta_1$) | **−0.21 (0.11)** | **0.046** | 0.11 (0.12) | 0.380 |
| Self-blame estimates by GRP2 ($\beta_2$) | 0.13 (0.14) | 0.364 | −0.17 (0.09) | 0.065 |
| GRP1 ($\beta_3$) | −0.02 (0.11) | 0.844 | 0.15 (0.10) | 0.136 |
| GRP2 ($\beta_4$) | −0.02 (0.10) | 0.831 | −0.01 (0.11) | 0.937 |
| Self-blame estimates by GRP1 by warmth ($\beta_5$) | 0.09 (0.12) | 0.431 | 0.09 (0.09) | 0.283 |
| Self-blame estimates by GRP2 by warmth ($\beta_6$) | **0.36 (0.13)** | **0.007** | **0.25 (0.10)** | **0.016** |
| Warmth ($\beta_7$) | −0.13 (0.22) | 0.550 | −0.05 (0.23) | 0.824 |
| Warmth by GRP1 ($\beta_8$) | 0.39 (0.23) | 0.092 | −0.12 (0.17) | 0.473 |
| Warmth by GRP2 ($\beta_9$) | −0.06 (0.16) | 0.732 | 0.33 (0.21) | 0.116 |

1. $N = 112$
2. Bolded text indicates significant effects at the $p < 0.05$ level

**Figure 1**




Estimated measurement model of latent trait cortisol. Standardized factor loadings are presented (*p*s < 0.001). The proportion of variance accounted for by the LTC for each sample (derived by squaring the standardized factor loading) is presented in parentheses. Double-headed arrow indicates correlated errors. *$p < 0.01$

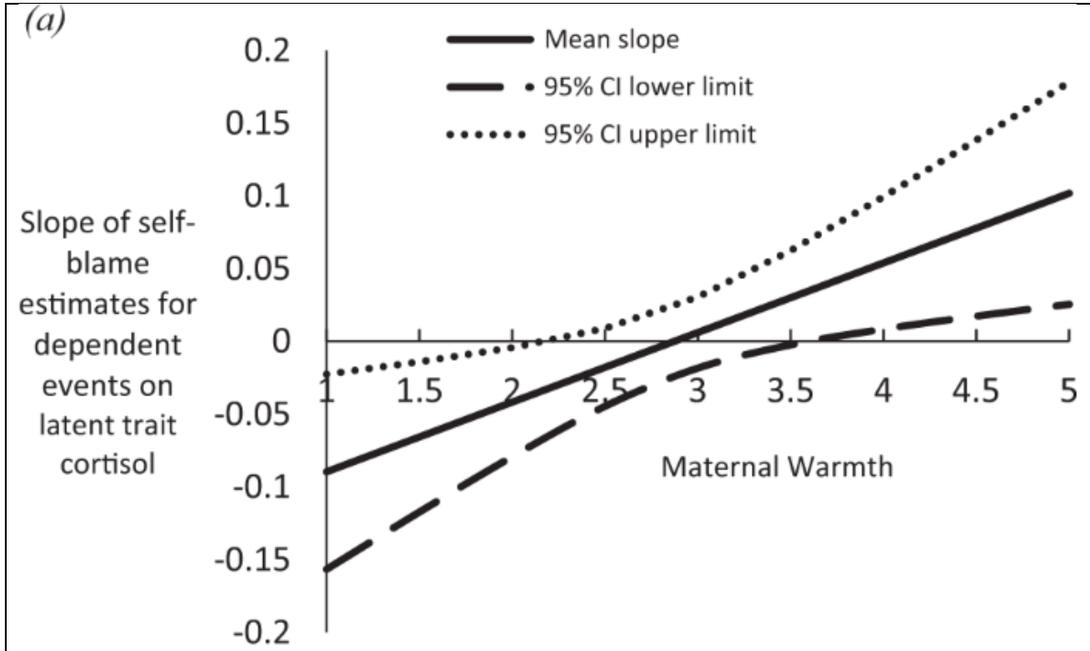



**Figure 2**

Johnson–Neyman regions of significance for the effect of self-blame estimates for dependent (**a**) and interpersonal (**b**) events on latent trait cortisol at varying levels of maternal warmth. The effect of self-blame estimates on latent trait cortisol is significant where the 95% confidence intervals for the slope estimates do not contain 0. For dependent events (**a**), the effect of self-blame estimates on latent trait cortisol is significant among those with maternal warmth levels of 4 or 5 (28.5% of the 66 participants with at least one dependent event), and for those with maternal warmth levels of 1 or 2 (16.7%). For interpersonal events (**b**), the effect of self-blame estimates on latent trait cortisol is significant among those with maternal warmth levels of 1–3 (74.5% of the 99 participants with at least one interpersonal event)