HHS Public Access

Author manuscript

Child Psychiatry Hum Dev. Author manuscript; available in PMC 2018 April 01.

Published in final edited form as:

Child Psychiatry Hum Dev. 2017 April ; 48(2): 225–234. doi:10.1007/s10578-016-0635-6.

The Interactive Effects of Stressful Family Life Events and Cortisol Reactivity on Adolescent Externalizing and Internalizing Behaviors

Christine M. Steeger[1], **Emily C. Cook**[2], and **Christian M. Connell**[1]

[1]Department of Psychiatry, Division of Prevention and Community Research, Yale University School of Medicine, New Haven, CT, USA

[2]Department of Psychology, Rhode Island College, Providence, RI, USA

Abstract

This study investigated the associations between stressful family life events and adolescent externalizing and internalizing behaviors, and the interactive effects of family life events and cortisol reactivity on problem behaviors. In a sample of 100 mothers and their adolescents (*M* age = 15.09; *SD* age = 0.98; 68% girls), adolescent cortisol reactivity was measured in response to a mother-adolescent conflict interaction task designed to elicit a stress response. Mothers reported on measures of family life events and adolescent problem behaviors. Results indicated that a heightened adolescent cortisol response moderated the relations between stressful family life events and both externalizing and internalizing behaviors. Results support context-dependent theoretical models, suggesting that for adolescents with higher cortisol reactivity (compared to those with lower cortisol reactivity), higher levels of stressful family life events were associated with greater problem behaviors, whereas lower levels of stressful family life events were related to fewer problem behaviors.

Keywords

Stressful family life events; Externalizing behaviors; Internalizing behaviors; Cortisol reactivity

Research suggests that both chronic psychosocial stressors and stressful life events are associated with greater child and adolescent psychopathology, including externalizing and internalizing problem behaviors [1, 2]. Although a large body of research demonstrates this well-established link between stress and emotional and behavioral problems, there continues to be a need for understanding both individual differences in how stress affects child and adolescent adjustment outcomes, and how different stress contexts may affect outcomes. In particular, individual differences in the relation between stressful family life events (e.g., financial problems, moving residences) and externalizing and internalizing problems may be accounted for by variations in physiological reactivity of the stress hormone cortisol, which

Correspondence concerning this article should be addressed to: Christine Steeger, Social Development Research Group, School of Social Work, University of Washington, 9725 3rd Ave NE, Suite 401, Seattle, WA 98115. Contact: csteeger@uw.edu; Phone (206) 221-3195; Fax (206) 543-4507.

is produced by the hypothalamic-pituitary-adrenocortical (HPA) axis. Some studies have examined cortisol reactivity as a moderator among children or emerging adults in stressful contexts [e.g., 3, 4]; however, less is known about HPA axis functioning during adolescence.

The examination of how cortisol reactivity may function as a moderator between stress and problem behaviors may be especially important during adolescence, a developmental time period characterized by biological, cognitive, and psychosocial transitions [5-7]. Adolescence represents a particularly sensitive period to stress, and is marked by heightened stress responsivity and emotional reactivity [8-10]. Moreover, less is known about potential interactions between cortisol reactivity and the cumulative effect of recent stressful family life events during adolescence, rather than chronic stressful environments such as poverty and marital conflict, which have a greater research base. Thus, the primary goal of this study was to investigate how potential interactions between recent family stressful life events and cortisol reactivity affect adolescent externalizing and internalizing behaviors.

Stressful Family Life Events and Adolescent Problem Behaviors

A large body of literature has linked stress in various contexts to child and adolescent externalizing and internalizing behaviors [1]. For example, several studies have highlighted the deleterious effects of poverty-related stress [11, 12], marital conflict [13, 14], family aggression [15], and child maltreatment [16] on child and adolescent emotional and behavioral outcomes. Specific to the family context, research has shown that measures of chronic family stress and cumulative stressful family life events (sometimes referred to as *adversity* or *adverse life events*) are also related to child and adolescent problem behaviors. Several studies have examined this link during childhood [17, 18], but fewer studies have focused on adolescence and the family domain [for exceptions see 2, 19, 20]. For example, a large-sample longitudinal study found that stressful life events (including family stress) were reciprocally related to adolescent externalizing and internalizing behaviors [2]. Another study using a large African American adolescent sample showed that stressful life events in family, peer, and individual domains predicted youth depressive symptoms over time [20]. Additionally, other research has also found associations between family life events and problematic outcomes such as adolescent depressive symptoms and frequent drinking behaviors among Finnish adolescents [19].

Collectively, these studies described above show a robust connection between family stress and youth problem behaviors, though more research examining these relations should be conducted with adolescents and in specific stressful life event domains for greater specificity of contexts [19, 20], rather than more global measures of stress. Stressful life events in the family context may affect adolescent emotional and behavioral responses differently than child responses, given maturation in cognitive functioning, reorganization of the family structure, and changes in parent-adolescent dyadic relations (e.g., increased conflict) during adolescence [5, 6]. The current study addresses this research need by examining associations among cumulative family stressful life events and adolescent problem behaviors.

The Human Stress Response and Cortisol Reactivity

Several physiological systems are involved in the human stress response. Two major stress response systems are the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis. The quicker-responding SAM system is primarily responsible for sympathetic activation in the “fight or flight” response, which includes increased circulation of catecholamines (e.g., epinephrine, norepinephrine), as well as increased blood flow to skeletal muscles, enhanced cardiovascular and respiratory rates, and elevating blood glucose [21, 22]. The HPA axis, which produces steroid hormones called glucocorticoids (cortisol in humans), is a complex and slower-acting stress response system, in that it may take approximately 25 minutes to reach peak levels of glucocorticoids [22, 23]. Main functions of the HPA axis include preparing the body for chronic exposure to stress by suppressing systems that do not promote immediate coping, and increasing the available energy through conversion of proteins and fats into glucose via glucocorticoid secretion [24]. Cortisol has been used in several studies as a marker of individuals’ response to acute stressors, and provides an important measure of individual variability in how children’s and adolescents’ bodies handle stressful life events [for reviews see 22, 24-27].

When an individual is exposed to chronic stressful contexts (e.g., poverty, marital conflict) for a prolonged period of time, or to cumulative stressful family life events (e.g., loss of income, parental divorce, death of a family member), chronic physiological activation requires increased demands from the individual, which is termed “allostatic load or overload” [27-29]. Chronic and cumulative stress may cause frequent activation of the stress response without adequate recovery or resources, which, in turn causes dysregulated responses such as hyper- or hypo-reactivity or arousal [22, 30]. Subsequently, dysregulated stress responses may place an individual at an increased risk for behavioral and physical problems, particularly when youth encounter new stressful events in their lives [22, 27].

Interactive Effects of Stressful Family Life Events and Cortisol Reactivity

To investigate how family life events may interact with cortisol reactivity on adolescent problem behavior outcomes, several stress response theories provide a collective framework to account for individual differences in physiological reactivity. Well-known diathesis stress models [e.g., 31] show that high physiological reactivity may be a marker of diathesis or vulnerability, whereas lower reactivity may be perceived as a marker of resilience. More recent evolutionary-based theories that extend diathesis stress models have received support from several empirical studies using physiological measures [24]. For example, reconceptualizations of physiological reactivity, such as Belsky and colleagues’ differential susceptibility theory (DST) [32, 33] and Boyce and Ellis’s biological sensitivity to context theory (BSCT) [34, 35] have received considerable attention and empirical support, particularly for children.

DST and BSCT are context-dependent theories and converge on the hypothesis that high reactivity (i.e., physiological, behavioral, or emotional arousal) is not inherently maladaptive. Rather, highly reactive individuals may be more susceptible to both positive (development-enhancing) and negative (risk-promoting) environments, as well as more

responsive to interventions [36, 37]. For example, Boyce and Ellis suggested that children and adolescents may thrive more in positive and supportive contexts (e.g., cohesive family environment), but may also be more vulnerable to the adverse effects of negative contexts (e.g., chronic marital conflict, harsh parenting) [34, 35, 38]. In fact, several studies have found that a heightened stress response may be associated with maladaptive behaviors or adaptive functioning outcomes among youth [e.g., 5, 27, 34, 39, 40]. In the current study, DST and BCST are useful theories for conceptualizing how contextual factors, such as stressful family life events, may interact with adolescent cortisol reactivity to affect problem behavior outcomes.

Purpose of Study

Although recent studies have examined relations between stressful contexts such as marital and family conflict [14, 15], child maltreatment [16], poverty [11, 12], and youth emotional and behavioral outcomes, much less is known about the role of cortisol reactivity in moderating the association between stressful life events in the family context and problem behaviors during adolescence. Most prior research has focused on early childhood and adulthood, leaving an important gap in understanding relations among family stress, HPA axis stress response, and emotional and behavioral health outcomes during adolescence [30].

Growing empirical support for both DST and BSCT motivates our hypotheses in the current study [e.g., 4, 14, 16, 24, 35, 41]. Consistent with context-dependent, person-environment theories, we hypothesized that heightened adolescent cortisol reactivity will moderate the relations between stressful family life events and both externalizing and internalizing behaviors. Specifically, we posited that adolescents with high cortisol reactivity and high levels of stressful family life events will have higher levels of problem behaviors, whereas those with high cortisol reactivity and low stressful family life events will have lower levels of problem behaviors. Conversely, we hypothesized that adolescents with lower levels of stressful family life events and lower cortisol reactivity will have lower levels of externalizing and internalizing problem behaviors. This study aimed to: (1) examine whether recent stressful life events in the family predict externalizing and internalizing problem behaviors, and (2) determine potential interaction effects of stressful family life events and cortisol reactivity on adolescent problem behaviors.

Methods

Sample

One hundred adolescents and their maternal caregivers (91% biological mother), living in an urban area in the Northeastern United States were recruited through flyers, a commercial mailing list, community events, and handing out information at willing private schools in the area. The adolescent participants primarily self-identified as European American (78%), with fewer youth identifying as other races/ethnicities (9% more than one race, 8% Black or African American, 3% Hispanic, 2% Asian). Adolescents were in grades 9^{th} – 11^{th} (*M age* = 15.09 years, *SD age* = .98) and were predominately female (68%). The majority of mothers reported being married (74%) and that this was their first marriage (64%). Mothers on average held a college degree (41%), which is higher than the percent within the state

reporting the same (18.6%; U.S. Census, Table S1501). The median level household income for the family was reported as falling between $80,000 - $89,999 US Dollars, which is similar to married-couple families within the state ($88,243; U.S. Census, Table S1901).

Procedures

Data were collected during home visits that occurred between 3:00-4:30pm to minimize the amount of cortisol variation due to time of day [42]. During the first part of the visit adolescents relaxed for 30-minutes by watching a video of their choice, either a G-rated cartoon (21%) or a nature DVD (79%). Choice of video was unrelated to measures of stress response. The relaxation period allowed us to obtain baseline levels of stress (cortisol); so that changes in cortisol could be attributed to the parent-adolescent interaction used as a stressor in this study. Mothers filled out surveys during the first part of the visit to assess key study constructs.

Following the relaxation period, adolescents engaged in two 8-minute interactions (order counter-balanced) that involved mothers and adolescents discussing an issue of conflict within the home and discussing an issue that adolescents were having outside of the home. These interactions were used to induce a stress response in adolescents that would reflect conflict with parents in their day-to-day lives [40, 43]. The Family Issues Checklist was used to identify a conflict issue in the home; mother and adolescents each identified topics of conflict that occurred within the last month, such as doing homework and coming home on time [44]. Mothers and adolescents separately marked the conflict topics and then rated how they felt while discussing that task on a scale from 1 (*calm*) to 5 (*angry*). The highest rated conflict for mothers and adolescents was chosen for discussion. During the other 8-minute interaction adolescents reported on an issue of conflict outside the home by identifying an issue (e.g., problems with a friend) that they were experiencing, which did not involve their home life. To obtain peak measures of stress response, cortisol was taken 15 minutes following both tasks to account for the delayed response in cortisol [23]. Following the interaction task, adolescents were again asked to relax while watching their chosen video and recovery measures of stress response were taken throughout the following 30 minutes. Mothers and adolescents were compensated $40 each for participation in the study. Consent and assent were obtained prior to the home visit. All data collection procedures were approved by the University's Institutional Review Board.

Measures

Adolescent problem behaviors—Mothers reported on adolescent externalizing and internalizing behaviors using the Child Behavior Checklist (CBCL) [45]. Responses ranged from 0 (*not true*) to 2 (*very/often true*), with higher scores indicating greater externalizing and internalizing problems now or within the past six months. Externalizing behaviors were items from the aggression and delinquency scales of the CBCL. Sample items included, "My child has a hot temper" and, "My child steals at home." Internalizing behaviors included items that measured anxiety, depressive symptoms, and somatic complaints [45]. Sample items of internalizing behaviors were, "My child feels unhappy, sad, or depressed" and, "My child worries a lot." Internal consistency, as measured by Cronbach's α, was .88 for externalizing problems and .89 for internalizing problems.

Stressful family life events—Mothers reported on stressful life events affecting the family using a Cumulative Adversity scale [46, 47]. The original list of 41 life experiences [46] was reduced to a 22-item survey to reduce participant burden; items most relevant to family stress were retained in the questionnaire, but items related to witnessing violence and other life traumas/traumatic news were not included. For example, consistent with the original Lloyd and Turner [46] measure of life adversity, items pertaining to major life events affecting families such as moving or changing family residence, financial problems, increases in number of arguments with child or children, serious injury or illness of a family member, and death events of relatives or close friends were most relevant to this study. Mothers were asked if each event occurred, and a count of how many events occurred represented the cumulative number of stressful family life events. Responses were coded as 0 (*did not happen in the past 6 months*) or 1 (*happened in the past 6 months*) to identify stressors that recently occurred.

Stress responses—Adolescent cortisol stress response was measured at five points in time during data collection procedures: twice during baseline (once 25 minutes into the session and following the relaxation period 40 minutes into the session), directly following both parent-adolescent interaction tasks (65 minutes into the visit), and at two points spaced 20 minutes apart toward the end of data collection (80 minutes into the visit and 100 minutes into the visit). A peak-baseline score was calculated using cortisol baseline following the relaxation period and cortisol peak roughly 15 minutes after the interaction task to obtain an indicator of HPA axis response as a measure of stress, which is a common measure of stress response in several cortisol reactivity studies [e.g., 15, 16]. Salivary samples were obtained non-invasively using a cotton strip that adolescents were asked to stick between their back molars and the inside of their cheek for approximately two minutes. Samples were stored at −40C. Saliva was assayed in duplicate at the Laboratory for Biological Health Psychology (Brandeis University, Waltham, MA) using a competitive chemiluminescence immunoassay (CLIA; IBL-International, Toronto, ON, Canada). The intra-assay coefficients of variation for the assay kits ranged from 3.9 to 6.6%, and the inter-assay coefficients of variation ranged from 2.6 to 5.5%, which were in the standard range for saliva assays.

Results

Preliminary Analyses

Sample descriptive characteristics and bivariate correlations among study variables are presented in Table 1. Adolescent gender was related to internalizing behaviors ($r = -.32$), with girls showing a greater association with internalizing than boys. This pattern of findings is consistent with other studies that reported gender differences in associations between stressful life events and depressive symptoms [e.g., 20]. Gender was subsequently controlled for in analyses with internalizing behaviors as the outcome variable. Adolescent age was not related to study variables and was therefore not included in any analyses. Additionally, correlations were examined among externalizing and internalizing outcomes and time of data collection, time of last food eaten, medication use, caffeine use on day of data collection, date of menstruation for females, and youth report of a puberty scale (Pubertal

Development Scale) [48]. All *ps* > .05; thus, no additional study variables were included as covariates in subsequent analyses.

Regression Analyses

Analyses were conducted with SPSS (version 21.0) software. In separate hierarchical regression models per outcome, we examined the effect of stressful family life events on externalizing and internalizing problems (model 1), as well as including cortisol reactivity and potential interactive effects of family life events and cortisol reactivity on externalizing and internalizing problems (model 2). Moderator models were tested following the procedures of Aiken and West [49], which included computing interaction terms after centering the independent variable (stressful family life events) and the potential moderator (cortisol reactivity). To examine the simple slope of interactions, we compared the effect of low (i.e., one *SD* below the mean) and high (i.e., one *SD* above the mean) levels of stressful family life events and cortisol reactivity. Simple slopes determined whether the effect of stressful family life events on externalizing and internalizing problems varied across levels of cortisol reactivity. Two cases (2% of the total sample) were missing cortisol observation data in the present study. Due to the small percentage of missing data, we used listwise deletion for the two cases. Therefore, a total of 98 mother-adolescent dyads were available for all analyses.

Externalizing problems—To test the hypothesis that adolescent externalizing problems were a function of high stressful family life events in the context of high cortisol reactivity, we conducted a hierarchical multiple regression analysis. In model 1, family life events were included, which accounted for a significant amount of variance in externalizing behavior problems, $R^2 = .114$, $F(1, 96) = 12.30$, $p < .01$. Results showed that family life events were significantly related to externalizing problems ($b = 1.24$, SE = .36, $p < .01$) (see Table 2). Next, in model 2, cortisol reactivity and the interaction term between stressful family life events and cortisol reactivity were added to the model. The main effect of stressful family life events was qualified by a significant family life events × cortisol reactivity interaction. Results showed that although cortisol was not associated with externalizing, the interaction term accounted for a significant proportion of the variance in adolescent externalizing problems, $R^2 = .160$, $\Delta R^2 = .047$, $F(3, 94) = 5.98$, $p < .05$ ($b = .43$, SE = .19, $p < .05$) (see Table 2). Simple slope tests showed that the slope of family life events on externalizing problems varied for different levels of cortisol reactivity. That is, there was a stronger relationship between stressful family life events and externalizing behaviors for youth high in cortisol reactivity ($b = 1.74$, $t(94) = 4.23$, $p < .001$) than for youth low in cortisol reactivity ($b = .89$, $t(94) = 2.34$, $p < .05$). Examination of the interaction plot showed that for adolescents with high cortisol reactivity, higher levels of stressful family life events were related to higher externalizing problems, whereas lower levels of family life events were related to lower externalizing problems (see Figure 1).

Internalizing problems—We tested internalizing problems with the same procedure as described above and showed similar results to externalizing problems. In model 1, stressful family life events and adolescent gender accounted for a significant amount of variance in internalizing behavior problems, $R^2 = .234$, $F(2, 95) = 14.54$, $p < .001$. Results showed that

both family life events and adolescent gender were associated with internalizing problems (b = 1.52, SE = .37, $p < .001$) and ($b = -5.11$, SE = 1.41, $p < .001$), respectively (see Table 2). Next, in model 2, cortisol reactivity and the interaction term between stressful family life events and cortisol reactivity were included in the model. The main effect of stressful family life events was qualified by a significant family life events × cortisol reactivity interaction, while controlling for adolescent gender. Results indicated that cortisol was not related to internalizing. However, the interaction term accounted for a significant proportion of the variance in adolescent internalizing problems, $R^2 = .274$, $\Delta R^2 = .040$, $F(4, 93) = 8.79$, p < .001 ($b = .43$, SE = .20, $p < .05$) (see Table 2). Simple slope tests showed that the slope of family life events on internalizing problems varied for different levels of cortisol reactivity. More specifically, there was a stronger relation between stressful family life events and internalizing behaviors for high cortisol reactivity ($b = 2.03$, $t(93) = 4.66$, $p < .001$) than for low cortisol reactivity ($b = 1.17$, $t(93) = 2.89$, $p < .01$). The interaction plot illustrated that for adolescents with high cortisol reactivity, higher levels of stressful family life events were related to higher internalizing problems, whereas lower levels of family life events were related to lower internalizing problems (see Figure 2).

Discussion

This study investigated the effect of stressful family life events on adolescent problem behaviors, and potential interactions between stressful family life events and adolescent cortisol reactivity on problem behavior outcomes. Results showed family life events predicted both externalizing behaviors and internalizing behaviors, even after controlling for adolescent gender in the internalizing behaviors model. These effects of family life events on problem behaviors are consistent with literature on relations among life stress, adversity, marital conflict, family disruption, and child problem behaviors [4, 14, 20]. The main effects of family life events on problems behaviors were qualified by significant interactions between family life events and cortisol reactivity on problem behaviors, such that high cortisol reactivity moderated the relation between higher levels of stressful family life events and high adolescent externalizing and internalizing problems. More specifically, we found that adolescents with high cortisol reactivity and high levels of stressful family life events showed increases in both externalizing and internalizing problem behaviors. In other words, at high levels of cortisol reactivity, more family life events were related to higher externalizing and internalizing problems, whereas fewer family life events were related to lower levels of problem behaviors. However, at low levels of cortisol reactivity, externalizing and internalizing problems were similar across both low and high levels of stressful family life events. The pattern of findings in this study contributes to our understanding of how context and individual vulnerabilities may work in concert to affect adjustment during adolescence.

Our results are consistent with other research supporting context-dependent perspectives (i.e., DST and BSCT), in which highly reactive adolescents show differential levels of problem behaviors when raised in adverse environments or contexts (e.g., high levels of stressful family life events), but low levels of problem behaviors in lower-stress and supportive environments [34, 35, 50]. Our findings correspond to prior studies such as Obradovic et al. [4], which found that high stress reactivity was associated with poor

adjustment outcomes in the context of high family adversity, but with better adjustment in the context of low adversity. Similarly, Saxbe et al. [15] found that for youth with higher cortisol reactivity, aggressive family environments were related to greater posttraumatic stress symptoms and antisocial behaviors over time, whereas youth in low-aggression family environments may exhibit fewer behavioral problems. Some literature has referred to these types of context-sensitive children as an “orchid child,” who may have adaptive or maladaptive outcomes depending on whether the environment or context is nurturing or neglectful/adverse [35]. In contrast, youth with low cortisol reactivity have been referred to in the literature as a “dandelion child,” who may show adaptive outcomes regardless of the adverse environment [35]. Indeed, our study results showed that for adolescents with low cortisol reactivity, levels of problem behaviors did not significantly vary across the context of low or high stressful family life events. Overall, findings of the current study contribute to our understanding of individual differences in how context affects development at a critical developmental time period.

Although our results were consistent with several empirical studies that found support for DST or BSCT in understanding relations among stressful contexts, cortisol response, and emotional or behavioral outcomes [e.g., 4, 15, 41], other research shows inconsistencies in low and high cortisol reactivity to stressors. Several methodological and developmental factors may explain discrepant findings across studies, though one main factor may be that cortisol reactivity and child adjustment outcomes are often sample-dependent. For example, compared to studies which used clinical samples of children or youth [39, 51], non-clinical, normative samples often show increased reactivity to stressors, particularly on externalizing behaviors [e.g., 7, 16, 43]. Moreover, some prior research is divided on heightened or blunted stress responses on behavioral outcomes across studies [15, 52, 53]. Further research should attend to sample variability and methodological factors (e.g., cross-sectional or longitudinal study design, specific stress induction task, patterns among age groups) when interpreting discrepant findings among studies.

In addition to potential variability across samples, another important finding to note is that only approximately 20% of our sample increased in baseline to peak cortisol reactivity levels. This rate is similar to levels found in several other studies that examined changes in salivary cortisol levels after mild-to-moderate stressors (e.g., conflict-discussion interaction tasks) [3, 4, 54]. In other research, Gunnar and colleagues [40] reviewed six studies which used either simulated parent-parent or parent-child conflict discussion tasks, and none produced significant elevations in cortisol. The use of other stress response interactions or tasks might elicit higher stress reactivity for a greater number of adolescents. For example, others have found that the Trier Social Stress Task (TSST) [55] and other public speaking tasks may be effective in eliciting a stress response, although there is evidence that the pubertal transition period from childhood to adolescence may be a time when elevations in stress response are more difficult to provoke [40]. However, the mother-adolescent interaction task used in this study was selected due to its relation to and appropriateness with both the predictor of stressful family life events and adolescent problem behaviors, and use and comparability with other studies [51, 56]. Nonetheless, the use of particular stress response tasks and specific physiological indicators of stress are important for study designs and outcomes, given recent discussions showing that physiological reactivity is highly

context-specific across different laboratory challenges (e.g., levels of alpha amylase may be particularly affected by interpersonal stress) [24, 40, 54].

Limitations and Future Directions

Although this study makes notable contributions in examining cortisol reactivity as a moderator of stressful family life events on problem behaviors during adolescence, some limitations should be noted. The current study used cortisol reactivity of the HPA axis as its primary indicator of stress reactivity. To gain a better understanding of the effects of stressful events and environments on physiological indicators of stress, additive effects and interactions within and between multiple stress systems should be examined [24, 57, 58]. For example, use of stress response indicators in the autonomic nervous system (ANS), a branch of the SAM stress response system, should also be examined, as well as potential additive and interaction effects among the ANS and HPA axis stress response systems [4, 24, 53]. ANS indicators may include alpha amylase, skin conductance levels (SCL), cardiac pre-ejection period (PEP), and respiratory sinus arrhythmia (RSA). Overall, the concurrent examination of multiple stress response systems will add to a more complete understanding of the complexities of biological reactivity during adolescence [30].

Moreover, the use of cumulative measures of stress reactivity (e.g., allostatic load) [27, 28, 59-61] may be particularly informative during adolescence, a sensitive period of development when individuals undergo changes in psychological, psychosocial, and biological systems [6, 40]. Given the changes in contextual stressors during adolescence, it will also be important for future research to examine multiple stress response systems and allostatic load longitudinally, across the transition from childhood to adolescence. Discrepancies in findings between cross-sectional [52] and longitudinal [15, 40] stress reactivity studies underscore the need for additional prospective longitudinal research. Studies that include a developmentally-focused and process-oriented framework will be useful in gaining a greater understanding of the complex mechanisms of stress reactivity across development [38]. Furthermore, interactions among gender differences in physiological reactivity, contextual factors, and acute stressors should also be tested in longitudinal studies, as some reviews have indeed found gender differences in physiological responses among adolescents [62, 63].

Other limitations of the current study include the use of only mother reports, which contribute to possible shared method variance. Although a mother's report of family life events and problem behaviors is useful, future studies may utilize both parent and adolescent reports of constructs. Additionally, we note that the current sample is predominantly European American, middle class, and well-educated. It will be important for future research to include participants with more diverse sociodemographic characteristics to increase the generalizability of findings. We also acknowledge that our predictors and interaction term explained only a modest amount of variance in the problem behavior outcomes. Although our R^2 and ΔR^2 are similar in magnitude to other cortisol reactivity studies [e.g., 4, 14, 53], our findings highlight the need to examine additional predictors and characteristics of parents and adolescents that may also explain how adolescent biological reactivity and behavioral outcomes are related. Lastly, and consistent with recommendations from

Obradovic et al. [4], future studies will also benefit from measuring positive environmental contexts, as fewer family life events and problem behaviors do not necessarily correspond with adaptive functioning in other domains.

Summary

The primary purpose of this study was to test the effects of stressful family life events and the potential interaction effects of stressful family life events and adolescent cortisol reactivity on problem behaviors. Results showed that high cortisol reactivity moderated the relation between stressful family life events and externalizing and internalizing problem behaviors in a normative adolescent sample. We examined both externalizing and internalizing behaviors as separate outcomes in this study and found the same patterns of results at low and high levels of cortisol reactivity. The current study provided further support for context-dependent theories that highlight the importance of person-environment interactions during development [32-36]. Our findings using an adolescent sample are consistent with results from prior work with young children [4], which suggests support for context-dependent theories across developmental age groups. Individual differences in high cortisol reactivity levels may not be inherently beneficial or detrimental, but are dependent on characteristics of the environment. DST and BSCT illustrate differential sensitivity to both potentially harmful and protective environmental contexts [35, 50]. Adolescents with low reactivity were unresponsive to environmental stress, such that they had similar levels of problem behaviors across both low and high levels of stressful family life events. Additionally, Ellis and Boyce [35] discussed that by taking into account individual differences in stress response phenotypes, results such as those shown in the current study may have implications for shaping youth intervention development. High biological sensitivity (e.g., cortisol reactivity) may serve as a biomarker to tailor intervention programs designed to prevent maladaptive outcomes for children and adolescents [35]. Furthermore, preventive interventions may also modify emotional and behavioral outcomes of biological processes [64, 65]. Overall study findings highlight the importance of examining specific environmental contexts, such as family stressful life events, paired with high and low physiological reactivity profiles to determine combination needs for tailored preventive interventions.

References

1. Grant KE, et al. Stressors and child and adolescent psychopathology: Evidence of moderating and mediating effects. Clinical Psychology Review. 2006; 26(3):257–283. [PubMed: 16364522]
2. Kim KJ, et al. Reciprocal influences between stressful life events and adolescent internalizing and externalizing problems. Child Development. 2003; 74(1):127–143. [PubMed: 12625440]
3. Hagan MJ, Roubinov DS, Mistler AK, Luecken LJ. Mental health outcomes in emerging adults exposed to childhood maltreatment: the moderating role of stress reactivity. Child Maltreatment. 2014; 19(3-4):156–167. [PubMed: 24920249]
4. Obradović J, et al. Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socioemotional behavior and school readiness. Child Development. 2010; 81(1): 270–289. [PubMed: 20331667]
5. Dahl RE, Gunnar MR. Heightened stress responsiveness and emotional reactivity during pubertal maturation: implications for psychopathology. Development and Psychopathology. 2009; 21(01):1–6. [PubMed: 19144219]

6. Steinberg L, Morris AS. Adolescent development. Journal of Cognitive Education and Psychology. 2001; 2(1):55–87.
7. Susman EJ, et al. Cortisol and alpha amylase reactivity and timing of puberty: Vulnerabilities for antisocial behaviour in young adolescents. Psychoneuroendocrinology. 2010; 35(4):557–569. [PubMed: 19819639]
8. Romeo RD. Adolescence: a central event in shaping stress reactivity. Developmental Psychobiology. 2010; 52(3):244–253. [PubMed: 20175102]
9. Spear LP. Heightened stress responsivity and emotional reactivity during pubertal maturation: Implications for psychopathology. Development and Psychopathology. 2009; 21(01):87–97. [PubMed: 19144224]
10. Steinberg L. A social neuroscience perspective on adolescent risk-taking. Developmental Review. 2008; 28(1):78–106. [PubMed: 18509515]
11. Conger RD, et al. Economic pressure in African American families: a replication and extension of the family stress model. Developmental Psychology. 2002; 38(2):179. [PubMed: 11881755]
12. Wadsworth ME, et al. Testing the adaptation to poverty-related stress model: Predicting psychopathology symptoms in families facing economic hardship. Journal of Clinical Child & Adolescent Psychology. 2011; 40(4):646–657. [PubMed: 21722035]
13. Cummings EM, Davies PT. Effects of marital conflict on children: Recent advances and emerging themes in process-oriented research. Journal of Child Psychology and Psychiatry. 2002; 43(1):31–63. [PubMed: 11848336]
14. Obradović J, Bush NR, Boyce WT. The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors. Development and Psychopathology. 2011; 23(01):101–114. [PubMed: 21262042]
15. Saxbe DE, et al. Does dampened physiological reactivity protect youth in aggressive family environments? Child Development. 2012; 83(3):821–830. [PubMed: 22548351]
16. Cook EC, et al. The stress response and adolescents' adjustment: The impact of child maltreatment. Journal of Youth and Adolescence. 2012; 41(8):1067–1077. [PubMed: 22359225]
17. Campbell SB, et al. Boys' externalizing problems at elementary school age: Pathways from early behavior problems, maternal control, and family stress. Development and Psychopathology. 1996; 8(04):701–719.
18. Flouri E, Tzavidis N, Kallis C. Adverse life events, area socioeconomic disadvantage, and psychopathology and resilience in young children: the importance of risk factors' accumulation and protective factors' specificity. European Child & Adolescent Psychiatry. 2010; 19(6):535–546. [PubMed: 19820985]
19. Fröjd S, et al. Significance of family life events in middle adolescence: A survey on Finnish community adolescents. Nordic Journal of Psychiatry. 2009; 63(1):78–86. [PubMed: 19012055]
20. Sanchez YM, Lambert SF, Ialongo NS. Life events and depressive symptoms in African American adolescents: do ecological domains and timing of life events matter? Journal of Youth and Adolescence. 2012; 41(4):438–448. [PubMed: 21706385]
21. Cannon, W. Bodily Changes in Pain, Hunger, Fear, and Rage. CT Branford Co.; Boston: 1929. Inc.
22. Gunnar M, Quevedo K. The neurobiology of stress and development. Annu. Rev. Psychol. 2007; 58:145–173. [PubMed: 16903808]
23. Gunnar, M., Adam, E. Physiological measures of emotion from a developmental perspective: State of the science. 2012. What can neuroendocrine measures reveal about emotion. Monographs of the SRCD
24. Obradović J. How can the study of physiological reactivity contribute to our understanding of adversity and resilience processes in development? Dev Psychopathol. 2012; 24(2):371–387. [PubMed: 22559120]
25. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychological Bulletin. 2004; 130(3):355. [PubMed: 15122924]
26. Gunnar, MR., Vazquez, D. Stress neurobiology and developmental psychopathology. 2006.
27. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. European Journal of Pharmacology. 2008; 583(2):174–185. [PubMed: 18282566]

28. McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Annals of the New York Academy of Sciences. 1998; 840(1):33–44. [PubMed: 9629234]
29. McEwen BS, Stellar E. Stress and the individual: mechanisms leading to disease. Archives of Internal Medicine. 1993; 153(18):2093–2101. [PubMed: 8379800]
30. Hostinar CE, Gunnar MR. Future directions in the study of social relationships as regulators of the HPA axis across development. Journal of Clinical Child & Adolescent Psychology. 2013; 42(4): 564–575. [PubMed: 23746193]
31. Monroe SM, Simons AD. Diathesis-stress theories in the context of life stress research: implications for the depressive disorders. Psychological Bulletin. 1991; 110(3):406. [PubMed: 1758917]
32. Belsky J, Bakermans-Kranenburg MJ, Van IJzendoorn MH. For better and for worse differential susceptibility to environmental influences. Current Directions in Psychological Science. 2007; 16(6):300–304.
33. Belsky J, Pluess M. Beyond diathesis stress: differential susceptibility to environmental influences. Psychological Bulletin. 2009; 135(6):885. [PubMed: 19883141]
34. Boyce WT, Ellis BJ. Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. Development and Psychopathology. 2005; 17(02): 271–301. [PubMed: 16761546]
35. Ellis BJ, Boyce WT. Biological sensitivity to context. Current Directions in Psychological Science. 2008; 17(3):183–187.
36. Ellis BJ, et al. Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. Development and Psychopathology. 2011; 23(01):7–28. [PubMed: 21262036]
37. Klein Velderman M, et al. Effects of attachment-based interventions on maternal sensitivity and infant attachment: differential susceptibility of highly reactive infants. Journal of Family Psychology. 2006; 20(2):266. [PubMed: 16756402]
38. Obradović J, Boyce WT. Individual differences in behavioral, physiological, and genetic sensitivities to contexts: Implications for development and adaptation. Developmental Neuroscience. 2009; 31(4):300–308. [PubMed: 19546567]
39. Granger DA, et al. Reciprocal Influences among Adrenocortical Activation, Psychosocial Processes, and the Behavioral Adjustment of Clinic-Referred Children. Child Development. 1996; 67(6):3250–3262. [PubMed: 9071780]
40. Gunnar MR, Talge NM, Herrera A. Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. Psychoneuroendocrinology. 2009; 34(7):953–967. [PubMed: 19321267]
41. Bradley RH, Corwyn RF. Infant temperament, parenting, and externalizing behavior in first grade: A test of the differential susceptibility hypothesis. Journal of Child Psychology and Psychiatry. 2008; 49(2):124–131. [PubMed: 18211274]
42. Blackhart GC, Eckel LA, Tice DM. Salivary cortisol in response to acute social rejection and acceptance by peers. Biological Psychology. 2007; 75(3):267–276. [PubMed: 17485157]
43. Chaplin TM, et al. Parent–adolescent conflict interactions and adolescent alcohol use. Addictive Behaviors. 2012; 37(5):605–612. [PubMed: 22341765]
44. Robin, A., Foster, S. Negotiating adolescence: A behavioral family systems approach to parent-adolescent conflict. Guilford; New York: 1989.
45. Achenbach, TM. Integrative guide for the 1991 CBCL/4-18, YSR, and TRF profiles. Department of Psychiatry, University of Vermont; 1991.
46. Lloyd DA, Turner RJ. Cumulative adversity and posttraumatic stress disorder: evidence from a diverse community sample of young adults. American Journal of Orthopsychiatry. 2003; 73(4): 381. [PubMed: 14609400]
47. Turner RJ, Lloyd DA. Lifetime traumas and mental health: The significance of cumulative adversity. Journal of Health and Social Behavior. 1995:360–376. [PubMed: 8719054]
48. Shirtcliff EA, Dahl RE, Pollak SD. Pubertal development: correspondence between hormonal and physical development. Child Development. 2009; 80(2):327–337. [PubMed: 19466995]
49. Aiken, LS., West, SG., Reno, RR. Multiple regression: Testing and interpreting interactions. Sage; 1991.

50. Ellis BJ, Boyce WT. Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. Development and Psychopathology. 2011; 23(01):1–5. [PubMed: 21262035]
51. Granger DA, Weisz JR, Kauneckis D. Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic-referred children and adolescents. Journal of Abnormal Psychology. 1994; 103(2):267. [PubMed: 8040496]
52. Daughters SB, et al. Gender specific effect of psychological stress and cortisol reactivity on adolescent risk taking. Journal of Abnormal Child Psychology. 2013; 41(5):749–758. [PubMed: 23338478]
53. Gordis EB, et al. Asymmetry between salivary cortisol and α-amylase reactivity to stress: Relation to aggressive behavior in adolescents. Psychoneuroendocrinology. 2006; 31(8):976–987. [PubMed: 16879926]
54. Granger DA, et al. Focus on methodology: salivary bioscience and research on adolescence: an integrated perspective. Journal of Adolescence. 2012; 35(4):1081–1095. [PubMed: 22401843]
55. Buske-Kirschbaum A, et al. Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. Psychosomatic Medicine. 1997; 59(4):419–426. [PubMed: 9251162]
56. Klimes–Dougan B, et al. Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. Development and Psychopathology. 2001; 13(03):695–719. [PubMed: 11523855]
57. Bauer AM, Quas JA, Boyce WT. Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. Journal of Developmental & Behavioral Pediatrics. 2002; 23(2):102–113. [PubMed: 11943973]
58. Chen FR, et al. Interaction of Adrenocortical Activity and Autonomic Arousal on Children's Externalizing and Internalizing Behavior Problems. Journal of Abnormal Child Psychology. 2015; 43(1):189–202. [PubMed: 24943055]
59. Blair C, et al. Allostasis and allostatic load in the context of poverty in early childhood. Development and Psychopathology. 2011; 23(03):845–857. [PubMed: 21756436]
60. Evans GW, et al. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. Developmental Psychology. 2007; 43(2):341. [PubMed: 17352543]
61. El-Sheikh M, Hinnant JB. Marital conflict, respiratory sinus arrhythmia, and allostatic load: Interrelations and associations with the development of children's externalizing behavior. Development and Psychopathology. 2011; 23(03):815–829. [PubMed: 21756434]
62. Kudielka BM, Kirschbaum C. Sex differences in HPA axis responses to stress: a review. Biological psychology. 2005; 69(1):113–132. [PubMed: 15740829]
63. Ordaz S, Luna B. Sex differences in physiological reactivity to acute psychosocial stress in adolescence. Psychoneuroendocrinology. 2012; 37(8):1135–1157. [PubMed: 22281210]
64. O'Neal CR, et al. Understanding relations among early family environment, cortisol response, and child aggression via a prevention experiment. Child Development. 2010; 81(1):290–305. [PubMed: 20331668]
65. Cicchetti D, Gunnar MR. Integrating biological measures into the design and evaluation of preventive interventions. Development and Psychopathology. 2008; 20(03):737–743. [PubMed: 18606029]

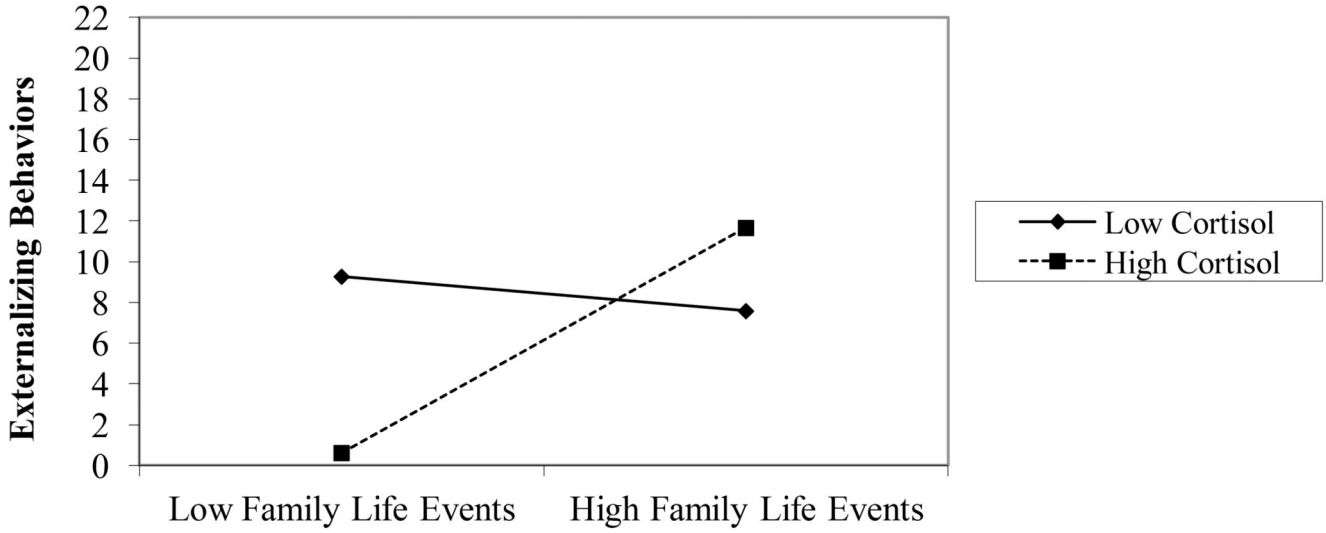

Figure 1. Externalizing behaviors as a function of stressful family life events and cortisol reactivity

Note. Mothers reported on all constructs. Low and high family stress are depicted at 1 *SD* below and above the mean.

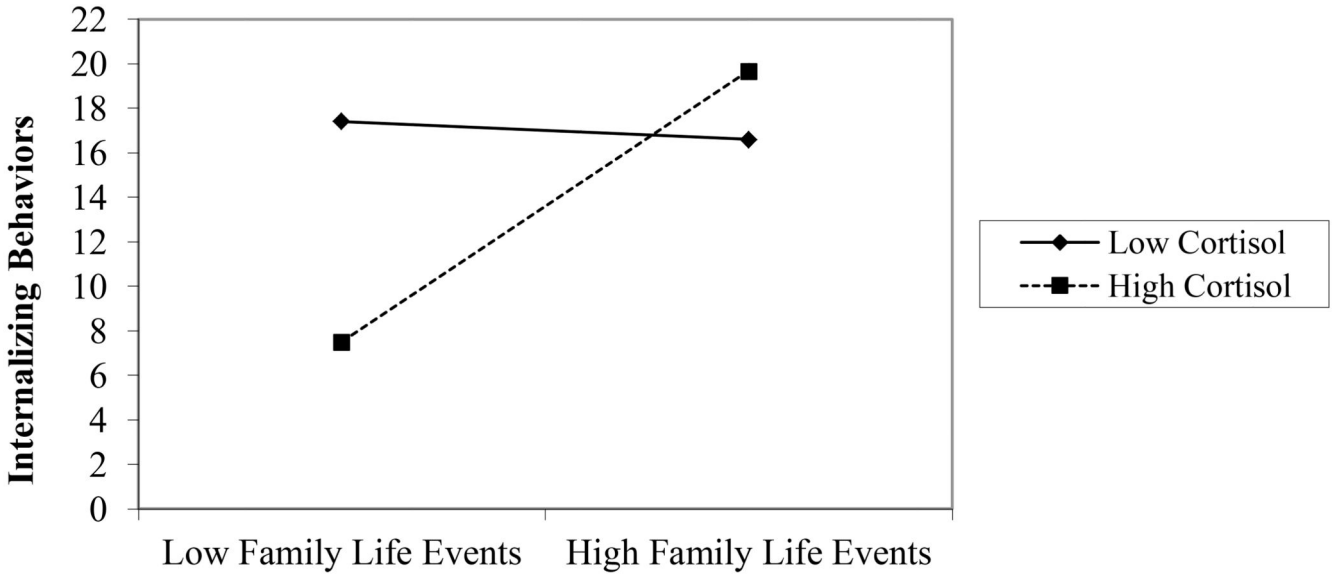

Figure 2. Internalizing behaviors as a function of stressful family life events and cortisol reactivity

Note. Mothers reported on all constructs. Low and high family stress are depicted at 1 *SD* below and above the mean.

Table 1
Descriptive Statistics and Intercorrelations Between Study Variables

Measure	1	2	3	4	5	6
1. Adolescent gender	--					
2. Adolescent age	.11	--				
3. Family life events	.02	−.03	--			
4. Externalizing behaviors	−.10	−.13	.34*	--		
5. Internalizing behaviors	−.32*	−.05	.36**	.60**	--	
6. Adolescent cortisol reactivity (peak-baseline)	−.10	−.06	.00	.02	−.02	--
M	--	15.07	1.96	7.29	8.77	−0.92
SD	--	0.98	1.78	6.56	7.40	4.22
Range	--	13-17	0-7	0-25	0-29	−8.96-24

Note. N = 98.

Females were coded as “1” and males were coded as “2”.

* $p < .01$.

** $p < .001$.

Table 2

Hierarchical Ordinary Least Squares Regression Analyses for Externalizing and Internalizing Behavior Outcomes

	Externalizing			
	Model 1		Model 2	
	***b* (SE)**	**B**	***b* (SE)**	**B**
Constant	7.26 (.63)		7.26 (.62)	
Family life events	1.24 (.36)	.36*	1.32 (.35)	.36***
Cortisol			−.28 (.20)	−.18
Family stress × cortisol			.43 (.19)	.30*
Unadjusted R^2	11.4% (6.20); $F(1, 96) = 12.30$**		16.0% (6.10); $F(3, 94) = 5.98$*	
ΔR^2			4.7%	
	Internalizing			
	Model 1		Model 2	
	***b* (SE)**	**B**	***b* (SE)**	**B**
Constant	15.51 (1.98)		15.26 (1.97)	
Family life events	1.52 (.37)	.37***	1.60 (.37)	.38***
Cortisol			−.41 (.21)	−.24
Gender	−5.11 (1.41)	−.33***	−4.92 (1.40)	−.31**
Family stress × cortisol			.43 (.20)	.27*
Unadjusted R^2	23.4% (6.55); $F(2, 95)=14.54$***		27.4% (6.44); $F(4, 93) = 8.79$***	
ΔR^2			4.0%	

Note. N = 98.

* $p < .05$;

** $p < .01$;

*** $p < .001$