Emotion

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The temperamental trait of negative emotionality (NE) plays an important role in maladaptation among adults experiencing significant life stress. However, the prospective relation between childhood NE and subsequent interrelated behavioral, emotional, and biological dysregulation in later life has not yet been established among children who experience early adversity. Using a longitudinal sample of youth who experienced parental divorce during childhood (N = 160; 53% male; 83% White), we tested the hypothesis that childhood NE would predict physiological, emotional, and behavioral dysregulation 15 years later. NE was assessed by maternal report when youth were between 9 and 12 years old. Fifteen years later, young adults (mean age = 25.55 years) participated in a psychosocial stress task to assess cortisol reactivity and reported on internalizing symptoms and problematic alcohol use. Structural equation modeling revealed that higher childhood NE predicted significantly greater alcohol use, internalizing symptoms, and total cortisol output during a stress task 15 years later. Importantly, these findings held after adjusting for childhood internalizing symptoms. In addition, problematic alcohol use was associated with greater cortisol reactivity and internalizing symptoms. Findings suggest that childhood NE is a critical risk marker for interrelated forms of dysregulation in young adulthood among at-risk youth.

Keywords: temperament, cortisol, alcohol, internalizing, negative emotionality

Negative emotionality (NE), defined as heightened sensitivity to negative environmental stimuli and a tendency to feel negative emotions (Buss & Plomin, 1984; Rothbart, 2007), plays a critical role in maladaptation among individuals experiencing psychosocial adversity or significant life stress (Galatzer-Levy et al., 2013; Kendler, Kuhn, & Prescott, 2004). NE is a core component of temperament, or individual differences in reactivity and regulation that are apparent early in life and relatively stable across development (Rothbart, 2007; Rothbart & Derryberry, 1981). Temperamental differences in emotionality and reactivity are considered heritable traits that establish a reaction range for biological and behavioral responses, because they influence how children appraise, encode, and respond to events across varied situations and across time (Nigg, 2006; Rothbart, 2007). Consistent with this, aspects of NE have been associated with a range of health risk behaviors and psychopathology in adolescents and adults, suggesting that early emotionality could be an important marker of clinical risk (e.g., Caspi et al., 1997; Kubzansky, Martin, & Buka, 2004; Slutske, Moffitt, Poulton, & Caspi, 2012). However, the prospective relation between NE during childhood and subsequent interrelated behavioral, emotional, and biological dysregulation in later life among children who experience early adversity has not yet been established.

Among the many adverse childhood experiences known to impact mental and physical health, childhood parental divorce is one of the most common, affecting approximately 50% of youths in the United States (National Center for Health Statistics, 2008). There is extensive evidence that parental divorce in childhood predicts greater substance use (Dube, Anda, Felitti, Edwards, & Croft, 2002; Thompson, Lizardi, Keyes, & Hasin, 2008), depressive symptoms (Roubinov & Luecken, 2013), and chronic physical health problems (Larson & Halfon, 2013; Yannakoulia et al., 2008). Identifying early childhood risk factors that prospectively predict physiological and behavioral dysregulation during young adulthood, when the transition out of adolescence poses increased risk for maladjustment, may help interventions to target the high

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rate and co-occurrence of health problems in this population (Nolen-Hoeksema & Watkins, 2011). In the current study, we tested the hypothesis that higher NE in childhood would predict greater internalizing symptoms, more problematic alcohol use, and greater cortisol output during stress 15 years later.

Despite the ubiquity of NE across different types of psychopathology (Watson & Naragon-Gainey, 2014), it is not known whether NE early in life predicts multiple indices of general emotion dysregulation (e.g., mood, anxiety, somatization) in adulthood. Strong associations have been found between childhood NE and depression in adulthood in multiple non-U.S. birth cohorts (Bould et al., 2014; Laceulle, Nederhof, Karreman, Ormel, & van Aken, 2012). Kubzansky and colleagues (2004) found that an observational measure of distress proneness at age 7 predicted greater emotional distress at age 35; however, the measure of distress proneness was most closely related with hostility and externalizing problems, leaving open the question of whether negative affectivity early in life predicts broad-based internalizing problems later on. Among individuals who have experienced family related adversity in childhood, high NE might exacerbate emotional sensitivity to psychosocial stress, increase depressogenic cognitions, and facilitate reliance on emotion-focused coping strategies, all of which are highly associated with depression and anxiety. Indeed, it has been shown that NE in children and adults is related to increased threat perceptions, greater negative expectations for the future, and increased focus on negative aspects of the world and the self, particularly among children from divorced families (Lengua, 2002).

NE also plays an important role in behavioral dysregulation and may be predictive of problematic alcohol use later in life. In their seminal longitudinal study of health and development, Caspi and colleagues (1997) found that high NE at age 18 predicted different but related health-risk behaviors at age 21, including greater problem drinking. However, earlier assessments of temperament are needed because problematic drinking is typically established by young adulthood among those exposed to early life stress (Enoch, 2011). Notably, abuse of alcohol has been related to having a history of early adversity (Dube et al., 2002) and is highly comorbid with other adversity-related sequelae such as anxiety and depression (Swendsen et al., 1998). High negative affectivity in childhood is related to a number of interpersonal and intrapersonal deficits that have been theoretically and empirically linked to a higher risk of alcohol use disorders (Hussong, Jones, Stein, Baucom, & Boeding, 2011). For example, NE in childhood predicts lower social competence in adolescence (Murphy, Shepard, Eisenberg, & Fabes, 2004), which in turn, has been implicated in problematic alcohol use later on, especially among individuals living in more stressful conditions (Zucker, 2008).

Emotional reactivity is one manifestation of heightened stress sensitivity, making NE particularly relevant to the long-term functioning of the stress response system. Cross-sectional studies show that NE may be associated with physiological dysregulation in the form of elevated cortisol reactivity to stress (e.g., Zobel et al., 2004) or higher cortisol throughout the day (Nater, Hoppmann, & Klumb, 2010). In one of the few prospective studies of NE and cortisol activity, Dougherty and colleagues (2013) demonstrated significant positive associations between negative affectivity in infancy and basal cortisol 3 years later. Development and calibration of the stress response system occurs early in life, and children who exhibit high NE may have a highly sensitized system across time or may "self-select" into conflictual environments that contribute to an ongoing sensitization of that system. Yet, it remains unknown whether NE in childhood predicts particular patterns of the physiological response to stress at a subsequent developmental stage. In addition to signaling physiological dysregulation in the context of a specific psychosocial stressor, chronic activation of the cortisol stress response may be one mechanism by which NE increases the risk of health problems. For example, high levels of cortisol have been implicated in a number of mental and physical health disorders in adulthood including but not limited to depression (Burke, Davis, Otte, & Mohr, 2005), atherosclerosis (Dekker et al., 2008), and hypertension (Kidambi et al., 2007). The current study is the first study we are aware of that prospectively tests whether childhood NE predicts a heightened cortisol stress response in young adulthood.

Mood and anxiety symptoms, problematic alcohol use, and physiological responses to stress are also interrelated, and research that examines the impact of childhood NE on biobehavioral dysregulation should ideally include all three. Illustratively, young adults commonly drink alcohol to cope with intense negative affect (Veilleux, Skinner, Reese, & Shaver, 2014), problem drinkers may exhibit greater cortisol reactivity to stress (Starcke, van Holst, van den Brink, Veltman, & Goudriaan, 2013), and heightened cortisol reactivity to stress has been associated with greater internalizing symptoms in some populations (Burke et al., 2005). It may be that high childhood NE undermines the development of more sophisticated emotion regulation skills, thereby increasing vulnerability to internalizing symptoms and increased stress sensitivity along with concomitant faulty behavioral regulation strategies (e.g., self-medication with alcohol) later in life (Kim-Spoon, Cicchetti, & Rogosch, 2013). In sum, early NE may be a shared underlying risk factor for interrelated forms of dysregulation over the long-term, but no research to date has examined this possibility.

The current study tested this hypothesis in children from divorced families, a population that is particularly well suited for examining the predictive power of NE. Children from divorced families experience greater mental and physical health problems in adulthood relative to the general population (Larson & Halfon, 2013). Moreover, parental divorce is often a marker of other potential stressors, such as family conflict, financial hardship, or other negative events. As a result, this population offers the opportunity to examine risk pathways among children exposed to constellations of stressful life circumstances (Lengua, 2002). We hypothesized that higher NE in childhood would predict greater biobehavioral dysregulation in young adulthood, as demonstrated by higher internalizing symptoms, greater problematic drinking, and elevated cortisol during psychosocial stress. To test this hypothesis, we employed structural equation modeling, which allows for simultaneous estimation of the prediction of multiple young adult outcomes from childhood negative emotionality and the magnitude of covariation among the multiple outcomes. Importantly, our models also control for childhood internalizing symptoms, which minimizes the possibility that findings are attributable to early depression or anxiety, rather than negative emotionality during childhood.

Method

Participants

Participants were a subsample of families who participated in a larger, longitudinal study of divorced families that participated in a randomized trial of a prevention intervention. Details of recruitment and eligibility are described in detail by Wolchik and colleagues (Wolchik et al., 2002). Briefly, potential participants were identified by reviewing randomly selected divorce decrees (divorced within 2 years prior to baseline assessment) of families with children between ages 9 and 12. Families were recruited through letters and telephone calls; 20% of the sample was recruited through supplemental methods (e.g., media, referrals). Families were eligible if the primary custodial parent was female, neither child nor mother was in treatment for mental health problems, mother had not remarried, and custody arrangements were expected to be stable. Families were excluded and referred for treatment if the child scored above 17 on the Children's Depression Inventory or 97th percentile on the externalizing subscale of the Child Behavior Checklist or endorsed suicidal ideation.

Although not the focus of the current analyses, the larger study included a randomized controlled trial of a preventive intervention (the New Beginnings Program) designed to reduce mental health problems among children who experienced parental divorce. The original trial included 240 families. This sample size was selected so that small to medium effects of the program could be detected with power equal to or greater than .80. Of the 240 families originally enrolled in the controlled trial, 194 participated in the 15-year follow-up. The current study includes participants who provided saliva samples at the 15-year follow-up, regardless of intervention group assignment. Of the 194 individuals, 12 did not participate in the stressor task or provide saliva samples, and two individuals had a cortisol concentration that was outside normal physiological parameters (>50 nmol/L; Nicolson, 2008), indicating assay interference. Of the remaining 180 participants, 20 individuals were excluded a priori due to pregnancy or breastfeeding ($n = 9$), use of steroidal medications or chronic health conditions ($n = 9$), violation of protocol by smoking within 30 min of the first saliva sample ($n = 1$), or only one viable saliva sample ($n = 1$). The final sample included 160 offspring (53.1% male) between 24 and 28 years old ($M = 25.55$, $SD = 1.22$). Ethnicity was 83% White; 11% Hispanic; 3% African American; 2% Asian American; and 1% American Indian or Alaskan Native. At the 15-year follow-up, 41% of young adults had completed at least some college.

Procedures

The current study includes families who were randomized to participate in a literature control or one of two versions (mother-only program and mother-plus child program) of a preventive intervention for divorced families (Wolchik et al., 2002; Wolchik et al., 2013). Given that neither intervention condition was shown to have direct effects on the outcomes in the current study, the intervention and control groups were combined, and intervention status was included as a covariate in all analyses.

The Arizona State University Institutional Review Board approved all measures and procedures. Six waves of assessment were conducted: baseline, posttest, 3 months later, 6 months later, 6 years later, and 15 years later. Only the baseline and 15-year follow-up assessments were used in the current analyses. All assessments were conducted by trained staff in participants' homes. At each assessment, confidentiality was explained, mothers and children signed consent and assent forms (at baseline), and young adults signed consent forms (at the 15-year follow-up). Families received $45 for participation at baseline, and young adults received $225 for participation in the 15-year follow-up.

Measures

Descriptive statistics for each measure are included in Table 1.

Negative emotionality. At baseline, mothers rated child negative emotionality using the emotionality subscale of the Emotionality, Activity, Sociability, and Impulsive Scale (Buss & Plomin, 1975). This 9-item subscale measures frequency and intensity of negative emotions as well as the threshold of the negative emotional response (e.g., "Your child gets troubled by everyday events."). Items are rated on a 5-point scale from 1 (*very unlike your child*) to 5 (*very like your child*). Buss and Plomin (1984) reported adequate test–retest reliability and validity. In the current sample, Cronbach's alpha was .78.

Internalizing symptoms. At the 15-year follow-up, participants reported on internalizing symptoms experienced over the past 6 months using the internalizing subscale of Adult Self Report (ASR; Achenbach & Rescorla, 2003). The ASR has demonstrated adequate reliability and validity (Achenbach & Rescorla, 2003). The normally distributed total raw score on the internalizing subscale was used in the current analysis. Cronbach's alpha for this subscale was .90.

Alcohol use. Problematic alcohol consumption was assessed at the 15-year follow-up using the ASR form (Achenbach & Rescorla, 2003). The ASR assesses alcohol use with the following item: "In the past 6 months, on how many days were you drunk?" As noted earlier, the ASR has demonstrated adequate reliability and validity (Achenbach & Rescorla, 2003). Given the nonnormality of alcohol use raw scores, the alcohol *t* score was used.

Social stress task and salivary cortisol collection. At the 15-year follow-up, young adults participated in a modified Trier

Table 1
Descriptive Statistics for Main Study Variables

Variable	*N*	Mean (*SD*)	Range
Negative emotionality	151	29.96 (6.51)	12–42
Internalizing-childhood	160	−.10 (.85)	−1.58–1.68
ASR total internalizing symptoms	160	4.66 (3.25)	0–17
ASR alcohol consumption *T* score	160	58.21 (8.35)	50–87
Cortisol			
AUC_G (ug/dl)	154	5.38 (3.41)	1.02–21.84
AUC_I (ug/dl)	154	−.08 (2.10)	−6.07–10.56
Baseline (ug/dl)	159	.088 (.05)	.02–.27
Posttask (ug/dl)	157	.086 (.05)	.01–.36
20 minutes posttask (ug/dl)	159	.090 (.07)	.01–.39
40 minutes posttask (ug/dl)	158	.074 (.05)	.01–.28

Note. Internalizing-childhood = composite of standardized scores on the Child Depression Inventory and Revised Children's Manifest Anxiety Scale; ASR = Adult Self Report; AUC_G = area under the curve with respect to ground; AUC_I = area under the curve with respect to increase.

Social Stress Task (TSST), consisting of mental arithmetic and a videorecorded speech task. The 3-min mental arithmetic involved serial subtraction problems performed out loud with a new starting number provided each minute and adjusted for difficulty in an attempt to hold effort constant across participants. It was conducted under time pressure, with prompting from the research assistant. Immediately following this portion of the task, participants were given 4 min to prepare a speech describing their personal strengths and weaknesses and 4 min to perform the speech. The research assistant was present during the performance, which was also videorecorded. Prior to the performance, the research assistant informed the participant that the video would be evaluated by a team of psychologists and verbally instructed the participant to look into the camera. Participants rated their moods before and after the task with the following items: (a) how angry, irritable, or disgusted do you feel?, (b) how nervous, scared, or jittery do you feel?, (c) how sad, blue, or lonely do you feel? Response choices were from 1 (*not at all*) to 10 (*extremely*). A paired samples *t* test comparing negative mood states before ($M = 4.56$, $SD = 2.58$) and after ($M = 6.85$, $SD = 4.59$) the task was significant, $t(159) = -6.77$, $p = < .001$, indicating negative emotional response to the task.

To aid sample retention, interviews were scheduled at the participants' convenience, between 1 p.m. and 8 p.m. The challenge task began approximately 30 min after arrival at the home. The study design was such that participants provided four samples of cortisol throughout the task at baseline (T1), posttask (T2), 20 min later (T3), and 40 min later (T4). Participants were instructed not to eat, drink, smoke, or exercise during the 2 hrs prior to the first saliva sample. Saliva was collected with a Salivette sampling device (Sarstedt, Rommelsdorf, Germany) held against the participant's inner cheek for 2 min. Saliva samples were then frozen at 20 °C and mailed overnight to Salimetrics, Inc. where they were assayed for cortisol using a high-sensitive enzyme immunoassay. This immunoassay test has a range of sensitivity from .007 to 1.8, and average intra- and interassay coefficients of variation of 4.13% and 8.89%, respectively.

Stress response system activity was measured in two ways: total cortisol output during the psychosocial stress task and magnitude of cortisol reactivity. Total cortisol output was assessed by computing area under the curve with respect to ground (AUC_G) with the trapezoidal formula (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003), a commonly employed parameter for summarizing total concentration of cortisol across a given time period (Fekedulegn et al., 2007). Magnitude of cortisol reactivity was assessed by computing area under the curve with respect to increase (AUC_I), which is calculated with reference to the baseline sample, reflects changes in cortisol across time, and is related to the sensitivity of the stress response system (Fekedulegn et al., 2007). Both formulas take into account minutes between sampling. Average time between samples was 18 min (T1 to T2), 27 min (T2 to T3), and 18 min (T3 to T4). Number of minutes was missing for at least one of the four saliva samples (e.g., T2 only) in 21 individuals; therefore, the average time between the relevant two samples (e.g., 18 min between T2 and T3) was imputed for these individuals. For analyses, AUC_G and AUC_I were log-transformed to correct for deviations from normality. Despite log-transformation, four individuals exhibited AUC_I values more than 3 *SD* above or below the mean. These values were winsorized to 2 *SD* outside the mean. As shown in Table 1, raw cortisol values increased from T1 to T3 on average, but as indicated by some negative AUC_I values, not all individuals showed an increase relative to baseline. Given the diurnal pattern of cortisol output, time of day at which the stress task began was included as a covariate (see below).

Covariates. A number of demographic and health factors known to influence cortisol were evaluated as covariates, including sex, family income during childhood, nicotine intake, caffeine consumption, body mass index, hormonal contraception, and time of day. Per capita income was assessed by maternal report at baseline. At the 15-year follow-up, participants reported on their smoking ("Do you currently smoke cigarettes or cigars?") and caffeine intake ("How many servings of caffeinated beverages do you drink in an average day?"), and females reported on their use of hormonal contraception. Body mass index (BMI) was calculated by dividing participant weight (in kg) by the square of his or her height (in meters). Time of day was calculated by taking the number of hours between midnight and the time at which the baseline cortisol sample was taken, and this was included as a covariate in the analyses.

To demonstrate that NE in childhood prospectively predicted internalizing problems in young adulthood and not concurrent depression and anxiety symptoms in childhood, child-report of internalizing symptoms at baseline were included as a covariate. Internalizing symptoms were assessed using the 27-item Children's Depression Inventory (CDI; Kovacs, 1980/1981; Saylor, Finch, Spirito, & Bennett, 1984; Cronbach's alpha = .75) and the 28-item the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978; Cronbach's alpha = .88). The CDI was designed to assess affective, cognitive, and behavioral symptoms of childhood depression and has been shown to discriminate between clinically depressed versus nondepressed psychiatric patients (Saylor et al., 1984). The RCMAS was designed to assess chronic anxiety in children and has demonstrated temporal stability and validity (Reynolds & Richmond, 1978). In the current study, scores on these two child-report scales were standardized, and an average was taken to form a composite of internalizing symptoms (Cronbach's alpha = .73).

Missing Data and Data Analysis

Across all values on all variables, 2.19% of data was missing. Of the 160 cases, 15 (9.38%) were missing at least one value. All analyses were conducted with Mplus v. 7.1 using full information maximum likelihood (FIML) estimation with robust standard errors to account for missing data. Specifically, structural equation modeling was used to simultaneously estimate and test paths between childhood NE and indicators of behavioral, emotional, and biological dysregulation in young adulthood. Specifically, we modeled the simultaneous effects of negative emotionality in childhood on cortisol AUC_G, cortisol AUC_I, and alcohol consumption and internalizing symptoms, while adjusting for child internalizing symptoms. In addition, we assessed the magnitude and statistical significance of the residual correlations among the de-

pendent variables, to ascertain the extent to which the outcomes were interrelated.

Results

Preliminary Analyses

Neither AUC_I or AUC_G were related to BMI ($r = -.10$, $p = .21$, and $r = .05$, $p = .53$), caffeine consumption ($r = .02$, $p = .81$, and $r = .05$, $p = .56$), nicotine intake ($r = -.08$, $p = .31$, and $r = .08$, $p = .33$), or family income at baseline ($r = -.01$, $p = .92$, and $r = -.07$, $p = .42$). Females taking hormonal contraception ($n = 24$) did not differ in AUC_I or AUC_G from either females not taking hormonal contraception ($n = 51$) or males ($n = 85$). Therefore, these covariates were not considered further. Males exhibited a significantly greater increase in cortisol during the task (AUC_I), $t(152) = 3.16$, $p = .002$; therefore, sex was included as a covariate in all analyses. In addition, to be conservative, we also adjusted for group membership (intervention vs. control); however, group membership was not related to any of the outcomes (r values = .01–.08, p values = .32–.92) nor was it related to childhood NE ($r = .04$, $p = .64$). Childhood internalizing problems were significantly correlated with childhood NE ($r = .18$, $p = .03$) and internalizing problems in young adulthood ($r = .18$, $p = .02$) but were not correlated with the young adult alcohol use ($r = –.01$, $p = .96$), AUC_I ($r = -.01$, $p = .89$), or AUC_G ($r = –.06$, $p = .44$). In sum, participant sex, time of day, childhood internalizing problems, and group membership were included as covariates.[1]

Primary Analyses

The model predicting cortisol AUC_G, AUC_I, internalizing symptoms, and problematic alcohol use in young adulthood from NE in childhood fit the data well: χ^2 ($df = 6$) = 3.084, $p = .80$; CFI = 1.00; RMSEA = 0.00; SRMR = .02. As shown in Figure 1, NE predicted greater problematic alcohol use, $b^* = .15$, $SE = .07$, $p = .031$, internalizing symptoms, $b^* = .18$, $SE = .08$, $p = .029$, and cortisol AUC_G, $b^* = .18$, $SE = .08$, $p = .024$. In addition, internalizing symptoms in childhood were associated with greater internalizing problems in young adulthood, $b^* = .15$, $SE = .08$, $p = .050$. Notably, comparison of the standardized betas indicates that childhood internalizing problems were less strongly related to young adult internalizing problems than childhood NE. Finally, inspection of the residual correlations revealed that greater problematic alcohol consumption was significantly related to higher cortisol AUC_G, $r = .25$, $SE = .09$, $p = .003$, as well as higher cortisol AUC_I, $r = .22$, $SE = .08$, $p = .006$. There was also a positive association between alcohol consumption and internalizing problems, $r = .16$, $SE = .07$, $p = .029$. Number of young adult internalizing symptoms were not related to either AUC_G, $r = -.02$, $SE = .08$, $p = .81$, or AUC_I, $r = -.03$, $SE = .08$, $p = .71$.

Discussion

The present study demonstrated that childhood negative emotionality (NE) predicted multiple indices of biobehavioral dysregulation in young adults who experienced parental divorce during childhood. Children identified by their mothers as being high in NE (e.g., irritability, tendency toward expressing sadness, reactivity to negative stimuli) exhibited higher levels of cortisol output during a stress task, reported greater internalizing symptomatology (e.g., greater mood problems, anxiety, somatization), and engaged in more problematic alcohol use 15 years later. In addition, these indices of emotional, behavioral, and biological dysregulation in young adulthood were significantly interrelated. These findings also held after accounting for childhood internalizing problems, which indicates that links between childhood NE and these three aspects of biobehavioral dysregulation in young adulthood are robust, and not attributable to early depression or anxiety symptoms. In all, the current findings indicate that heightened NE in childhood represents a critical risk marker for multiple forms of dysregulation in young adulthood for children who experience parental divorce.

We found a significant positive association between childhood NE and cortisol output during a stress task. The current study extends previous evidence of cross-sectional relations by demonstrating that NE in late childhood predicted cortisol output during a stress task 15 years later, when the participants were young adults. Interestingly, NE predicted total hormonal output but not change in cortisol levels from baseline. This finding highlights the importance of considering multiple indices of the stress response. Whereas basal levels of cortisol are tightly linked to diurnal cortisol secretion and more "trait-like," cortisol reactivity to a particular stressor might be more "state-like," and therefore more affected by contextual rather than constitutional factors. The different indices of cortisol activity have been correlated with specific health outcomes. Illustratively, greater total cortisol output, rather than the pattern of cortisol release over time, has been implicated in the development of atherosclerosis (Dekker et al., 2008). Our results suggest that children from divorced families who exhibit higher NE may be at greater risk of neuroendocrine dysregulation and concomitant health problems later in life.

The current findings also suggest that heightened NE in childhood represents a risk marker for later problematic alcohol use in children experiencing adverse circumstances. The association between higher NE and more problematic alcohol consumption builds on previous work that identified associations between NE and greater engagement in risky drinking over a shorter time period (e.g., Caspi et al., 1997) and adds to the growing evidence demonstrating the role of negative affectivity in the development of risky drinking behaviors (Hussong et al., 2011). Whereas others have found that an "undercontrolled" temperament (e.g., inattention, impulsivity, negativism, and behavioral withdrawal) at age three predicted risky drinking behavior and disordered gambling in adulthood (Caspi et al., 1997; Slutske et al., 2012), the present finding suggests that NE alone is a precursor to emotional and behavioral dysregulation later in life. Research showing that negative affect and stress sensitivity interact to predict the use of alcohol to cope with distress suggests that the self-medication model of alcohol use is one mechanism by which NE may contribute to this risky health behavior (Colder, 2001).

[1] Given previous findings of an intervention by age interaction on cortisol response in the current sample (Luecken et al., 2015), we also estimated a model controlling for age and this interaction term. Model fit was poor and associations did not change. Therefore, we report the more parsimonious models here.

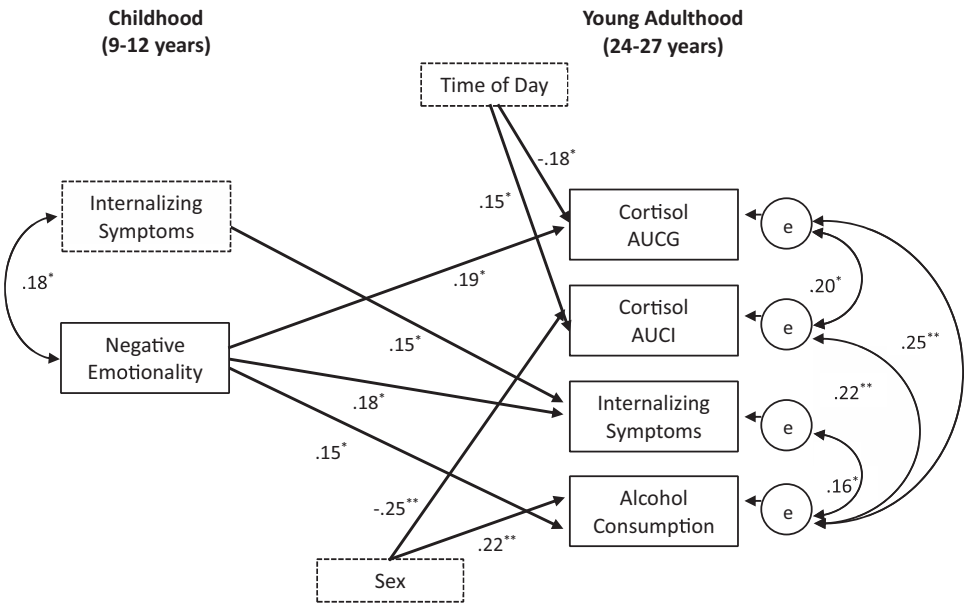

Figure 1. Negative emotionality in childhood predicting cortisol AUC_G, cortisol AUC_I, problematic alcohol use, and internalizing symptoms in young adulthood. Standardized estimates are presented. Covariates are depicted in boxes with dashed lines. Only significant pathways are shown here. Nonsignificant paths not shown here include childhood internalizing → cortisol AUC_G, childhood internalizing → cortisol AUC_I, childhood internalizing → alcohol consumption, sex → young adult internalizing, sex → cortisol AUC_G, negative emotionality → cortisol AUC_I. ** $p < .01$. * $p \leq .05$.

It is noteworthy that mother-rated child NE predicted greater self-rated mood and anxiety symptoms in young adulthood even when adjusting for youth's own report of depression and anxiety symptoms in childhood. This finding attests to the predictive validity of maternal perceptions of their child's level of negative affective reactivity and to the robust relation between NE and young adult outcomes. One possible explanation for these findings is that high levels of NE in childhood may undermine the development of more sophisticated emotion regulation skills. Alternatively, high childhood NE might increase one's exposure to negative contexts and/or negative relationships in adolescence that in turn influence the development of mood and anxiety problems (e.g., Barrocas & Hankin, 2011). For example, it has been postulated that children high in NE elicit less responsive caregiving or more controlling parental behaviors, which in turn accounts for observed relations between NE and subsequent mood problems. However, a meta-analysis of studies focused on temperament and parenting in the early years of life found that associations were weak and moderated by study characteristics (Paulussen-Hoogeboom, Stams, Hermanns, & Peetsma, 2007). Future studies should assess NE and internalizing problems multiple times throughout childhood and adolescence to investigate the processes by which this relation unfolds.

The current study focused on a set of dysregulation indices that cut across behavioral, emotional, and physiological phenomena and demonstrated significant interrelations. Notably, greater alcohol consumption was related to greater overall total cortisol output as well as greater magnitude of reactivity (i.e., change in cortisol levels across the task). The positive association between problematic alcohol consumption and cortisol identified in the current study adds to a growing body of evidence in support of this relation (e.g., Chaplin et al., 2012; Starcke et al., 2013). Specifically, high levels of cortisol have been observed during alcohol intoxication (Adinoff, Ruether, Krebaum, Iranmanesh, & Williams, 2003) and in heavy drinkers in general (Boschloo et al., 2013), raising the possibility of potential bidirectional effects, in addition to the predictive effect of NE on alcohol use found here. We also found a statistically significant positive association between problematic alcohol consumption and internalizing problems in young adulthood. Potential associations between internalizing problems and alcohol use have received relatively little attention (Hussong et al., 2011). Focusing on this association within at-risk populations, such as offspring from divorced families, is critical given that parental divorce in childhood is associated with an increased risk of mood and alcohol use disorders in adulthood (Kessler, Davis, & Kendler, 1997). In contrast to expectations, within this sample of children who experienced parental divorce, internalizing problems were not related to overall cortisol response to the stress task in young adulthood. Associations between internalizing problems and measures of cortisol reactivity to stress have been inconsistent across studies (Spies, Margolin, Susman, & Gordis, 2011), and there has been some indication that this association is evident only in the context of poor parenting or child abuse (e.g., Hagan, Roubinov, Mistler, & Luecken, 2014). Thus, in our sample of children who experienced a range of quality of parenting within the context of family divorce, links between internalizing and cortisol response may be less evident.

Several limitations must be considered when interpreting the present findings. The current study focused on one aspect of

temperament, NE. Other temperamental traits, such as effortful control and behavioral inhibition may also influence later-life biobehavioral regulation (Frenkel et al., 2015). In addition, the present investigation focused on a particular population, youth from divorced families. Although the focus on youth with a specific, documented adverse childhood experience is a strength of the study, findings cannot be generalized to youth from nondivorced families, nor can we determine if the associations documented here are specific to youth from divorced families. Relatedly, generalizability of the current findings to other populations is limited in that participants in the current study were also almost exclusively non-Hispanic White. Additionally, problematic alcohol consumption was assessed by self-report on a single item. This item has been validated and normed such that responses can be compared across same sex and same age peers; however, additional measures of problematic alcohol use, such as daily diaries or information on patterns of use (e.g., binging) could provide a more complete understanding of the long-term behavioral impact of NE.

For many children facing adverse experiences, the mental and physical health consequences are not immediately observable, and early emerging affective vulnerabilities may be overlooked until one or more clinical disorders manifest. Identifying early childhood risk factors that prospectively predict interrelated forms of emotional, behavioral, and biological dysregulation may help interventions to target the high rate and co-occurrence of health problems in this population (Nolen-Hoeksema & Watkins, 2011). The current study demonstrated that greater levels of mother-rated negative emotionality in childhood predicted a constellation of different forms of dysregulation, including greater mood and anxiety problems, greater problematic alcohol consumption, and greater levels of total cortisol output during a stressor task in a sample of individuals who experienced parental divorce during childhood. Future research should aim to further elucidate the pathways by which early negative emotionality increases the risk for multiple forms of dysregulation at a subsequent developmental stage among youth facing stressful circumstances, including youth from divorced families.

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