A Longitudinal Study of the Effects of Child-Reported Maternal Warmth on Cortisol Stress Response 15 Years After Parental Divorce

Linda J. Luecken, PhD, Melissa J. Hagan, PhD, MPH, Sharlene A. Wolchik, PhD, Irwin N. Sandler, PhD, and Jenn-Yun Tein, PhD

ABSTRACT

Objectives: The experience of parental divorce during childhood is associated with an increased risk of behavioral and physical health problems. Alterations in adrenocortical activity may be a mechanism in this relation. Parent-child relationships have been linked to cortisol regulation in children exposed to adversity, but prospective research is lacking. We examined maternal warmth in adolescence as a predictor of young adults' cortisol stress response 15 years after parental divorce.

Methods: Participants included 240 youth from recently divorced families. Mother and child reports of maternal warmth were assessed at 6 time points across childhood, adolescence, and young adulthood. Offspring salivary cortisol was measured in young adulthood before and after a social stress task. Structural equation modeling was used to predict cortisol response from maternal warmth across early and late adolescence.

Results: Higher child-reported maternal warmth in early adolescence predicted higher child-reported maternal warmth in late adolescence (standardized regression = 0.45, standard error = 0.065, \( p < .01 \)), which predicted lower cortisol response to a challenging interpersonal task in young adulthood (standardized regression = −0.20, standard error = 0.094, \( p = .031 \)). Neither mother-reported warmth in early adolescence nor late adolescence was significantly related to offspring cortisol response in young adulthood.

Conclusions: Results suggest that for children from divorced families, a warm mother-child relationship after divorce and across development, as perceived by the child, may promote efficient biological regulation later in life.

Trial Registration: ClinicalTrials.gov Identifier: NCT01407120.

Key words: parental divorce, cortisol, mother-child relationship, warmth.

INTRODUCTION

Mounting evidence demonstrates that adverse experiences early in life are associated with a number of health problems later in life. Stress that occurs during a developmentally sensitive period is theorized to promote dysregulation of biological stress responses systems, increasing vulnerability to stress-related illnesses during childhood and across the life span. The development and functioning of the neuroendocrine system is commonly posited as a mechanism by which childhood adversity can affect long-term health. For example, animal studies demonstrate that early life stress associated with low maternal care can alter hippocampal gene expression and result in exaggerated hypothalamic-pituitary-adrenal (HPA) reactivity to later stress (1). Dysregulation of components of the HPA axis, in particular the stress hormone cortisol, has been identified in children, adolescents, and adults exposed to childhood adversity (2–4) and can be evident in the form of exaggerated or attenuated reactivity to challenge, higher or lower basal levels, or flattened diurnal slopes (5,6). It has been proposed that severe adversity sustained across development may initially be associated with hypersecretion that down-regulates over time to result in hyposecretion (5,7). Dysregulated HPA activity has been linked to a wide range of poor mental and physical health outcomes in childhood and later in life, including depression, anxiety disorders, inflammatory disorders, coronary heart disease, hypertension, and substance abuse (8–14).
Although the health risks of childhood adversity are well documented, there are considerable individual differences in consequences, with many children demonstrating better-than-expected outcomes. Theory and empirical data consistently indicate that a supportive, warm relationship with the primary caregiver promotes psychosocial adaptation and may prevent or reduce hormonal dysregulation associated with adversity (2,9,15). Concurrent and short-term associations have been found between positive parenting and cortisol responses to stress in infancy, childhood, and adolescence (16–20). Retrospective studies indicate that a positive parent-child relationship may have a lasting protective effect. Maternal warmth predicts lower biological risk in adulthood after exposure to significant childhood adversities including maltreatment, poverty, and parental loss (9,21–23). For example, Luecken (23) found that retrospectively reported warmth from the surviving parent buffered cortisol stress responses in young adults who experienced childhood parental loss. Retrospective reports of parental warmth in childhood also predicted lower allostatic load in adults abused as children (21) and lower risk of metabolic syndrome at midlife (24).

Few prospective studies have examined whether the neuroendocrine benefits of positive parenting are observed at later developmental stages. In a small sample of predominantly white, middle- or high-income families, Kuhlman et al. (25) found that mother-reported warmth when children were 5 years old predicted a lower child cortisol stress response at age 7 years. In contrast, a recent investigation by Hackman and colleagues (26) with a small sample of low-income African American families found that low levels of parental responsivity, assessed when children were 4 years old, predicted blunted cortisol reactivity (increase from baseline) to a social stress task in late adolescence. In a sample of parentally bereaved children (age 8–16 years), Hagan et al. (27) found that mother- and child-reported positive parenting in the first 2 years after the death moderated the impact of recent stressful events on cortisol output 6 years later: among youth with a history of low positive parenting, recent negative life events were associated with elevated cortisol. Despite small sample sizes, attrition, and/or the measurement of parenting at a single time point, these studies nonetheless suggest the potential for caregiver warmth in childhood to impact neuroendocrine regulation at a subsequent developmental stage.

Parental divorce is a stressful and emotionally challenging process for many affected children, who may experience a number of secondary adversities including economic stress, relocation, disrupted parenting, and conflict between parents and among extended family members. Although most children adapt well after parental divorce (28) and some even show improved well-being (29), numerous studies document that children from divorced families are at higher short- and long-term risk of internalizing and externalizing problems, poor health behaviors such as smoking and alcohol abuse, general health problems, and decreased longevity (28–33). Roustit et al. (34) reported that the relation of childhood parental divorce to poor self-rated health in adulthood was mediated by the quality of parent-child relationships. The wide-ranging stressors associated with parental divorce may make children particularly vulnerable to negative long-term biological consequences. However, stress response system functioning in children who experienced parental divorce has received little research attention relative to other adversities. Lower HPA stress responses have been documented in young adults from divorced families relative to those from two-parent families (35,36). Although not specific to parental divorce, studies have also noted exaggerated or attenuated patterns of cortisol activity after adversities commonly associated with parental divorce, including poor marital functioning (37), interpersonal conflict (38–40), poor parent-child relationships (41), and maternal depression (42).

There are a number of limitations in existing research that we sought to address in the current study. First, because much of the research on the long-term impact of childhood adversity has relied on retrospective reports of parenting

<table>
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<th>TABLE 1. Sample Characteristics</th>
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<tr>
<td>Age at Wave 1 ( (n = 240) ), M (SD)</td>
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<td>Age at Wave 6 ( (n = 194) ), M (SD)</td>
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<td>Age at parental divorce ( (n = 240) ), M (SD)</td>
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<tr>
<td>Sex, ( n (%) )</td>
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<td>Female</td>
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<td>Maternal Ethnicity, ( n (%) )</td>
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<td>Regular smoker, ( n (%) )</td>
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<td>Use of medications, ( n (%) )^a</td>
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<td>Pretask cortisol ( (n = 160) ), M (SD) ( \mu g/dl )</td>
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<td>Posttask cortisol ( (n = 159) ), M (SD) ( \mu g/dl )</td>
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<td>20-min postcortisol ( (n = 161) ), M (SD) ( \mu g/dl )</td>
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<td>40-min postcortisol ( (n = 159) ), M (SD) ( \mu g/dl )</td>
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<td>Cortisol AUCg ( (n = 155) ), M (SD), ( \mu g/dl )</td>
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M = mean; SD = standard deviation; AUCg = area under the curve with respect to ground.

^a Includes allergy, antidepressant, antianxiety, or other medications that could potentially affect cortisol.

Nontransformed data.
and adversity, it is not known whether later biological indices are affected more by current perceptions or perceptions during childhood. Prospective longitudinal studies in which parental warmth is assessed at multiple time points during childhood as well as in adulthood, in a sample with documented childhood adversity, are critical for addressing this limitation. Second, existing longitudinal studies with children at risk due to a variety of adversities have not examined prospective relations between parental warmth and cortisol regulation over prolonged periods (e.g., into adulthood). Third, theoretical and empirical accounts suggest that parental divorce may pose a risk to developing biological regulation, and positive mother-child relationships are consistently associated with improved child psychological adjustment after divorce (43), but research has not yet prospectively evaluated maternal warmth as a predictor of cortisol regulation in children from divorced families. Finally, studies of the impact of parental warmth on children's health primarily rely on caregiver reports, whereas retrospective research in adulthood primarily relies on offspring ratings. A better understanding of the process may be achieved by separately examining child and caregiver reports of caregiver warmth.

The current study addresses these limitations with a prospective, longitudinal study that followed mothers and children at 6 time points across 15 years after parental divorce. Both mother and child reports of maternal warmth were assessed at each time point, and offspring cortisol response to a challenging speech task was assessed at the final time point (offspring age 23–27 years). We hypothesized that higher maternal warmth during childhood and adolescence would predict lower cortisol response to the task in adulthood. Because parenting quality during childhood has been shown to exert a unique impact on young adult functioning above and beyond parenting in young adulthood (44), we predicted that the effect of maternal warmth in childhood would be independent of current reports of maternal warmth. By separately evaluating mother and offspring reports of warmth, we also addressed whether mother's or child's perspective has a stronger effect on cortisol regulation following parental divorce.

**METHODS**

**Participants**

Participants were 240 youth from divorced families who participated in a larger study of an experimental trial for families of divorce. Participants were 9 to 12 years old at the beginning of the study and 23 to 27 years old at the final time point. Most (80%) were recruited from randomly selected court records of divorce decrees granted within 2 years of the trial's start; the remainder responded to media advertisements. The primary eligibility criteria included the following: a) primary residential parent was female, b) at least one 9- to 12-year-old child who resided at least 50% of the time with the mother, c) neither mother nor any child was in treatment for mental health problems, and d) mother had not remarried, did not plan to remarry, and did not have a live-in boyfriend (45). Demographic information is displayed in Table 1. The study was approved by the Internal Review Board at Arizona State University and carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki). Children signed informed assent forms; parents and youths older than 18 years signed informed consent.

Assessments were conducted in participants' homes at 6 time points across 15 years: W1 (baseline; May 1992–February 1994) was conducted when children were 9 to 12 years old; Waves 2–4 were conducted approximately 3, 6, and 9 months after W1; W5 was conducted 6 years later (ages 15–19 years; 1998–2000); and W6 was conducted 15 years later (ages 23–27 years; April 2007–February 2009). At W1, families were randomly assigned to an intervention (New Beginnings Program; n = 164) or Literature Control group (n = 76). Waves 2-6 were conducted after the intervention. Because intervention effects are not the focus of the current analyses and prior analyses indicated that participation in the intervention did not predict cortisol output at the 15-year follow-up (46), the groups are combined for the current analyses.

Of the 240 mother-child dyads who participated at W1, 194 (89.6% of the families) completed the 15-year follow-up. Cortisol samples were not obtained from 12 youth (8 refused, 4 were living outside the country). Two participants had cortisol levels outside normal physiological levels (i.e., > 50 nmol/L), indicating interference in the assay. Nineteen were
TABLE 2. Zero-Order Correlations Between Study Variables (Pearson \(r/n\))

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<td>1.0</td>
<td>0.56** 233</td>
<td>0.53** 231</td>
<td>1.0</td>
<td>0.42** 229</td>
<td>0.60** 229</td>
<td>1.0</td>
<td>0.31** 199</td>
<td>0.35** 193</td>
<td>0.33** 193</td>
<td>1.0</td>
<td>0.20** 183</td>
<td>0.12 177</td>
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\(W = \text{wave; AUCg = area under the curve with respect to ground.}

\(* p < .05, ** p < .01.\)

excluded a priori from analyses due to pregnancy (\(n = 8\)), current use of thyroid or other medications known to affect glucocorticoids (\(n = 9\)), or cortisol levels higher than 4 standard deviations (SDs) from the mean (\(n = 2\)).\(^1\) Five participants did not have at least three viable samples for calculation of area under the curve with respect to ground (AUCg). Thus, cortisol AUCg was available from 155 participants.

Internal and external validity was evaluated by testing if attritors differed from nonattritors on baseline demographics or maternal warmth at Waves 1–5. Attrition status was not associated with participant age (\(p = .75\)), ethnicity (\(p = .82\)), sex (\(p = .41\)), months since parental divorce (\(p = .53\)), or family income (\(p = .51\)). Attrition status also was not associated with child- or mother-reported maternal warmth at any of the first five waves (\(p \text{ values } = .21–.85\)).

Procedure

To aid sample retention, interviews were scheduled at the participants’ convenience, between 1 PM and 8 PM. Saliva samples were collected before, immediately after, and 20 and 40 minutes after a modified Trier Social Stress task (TSST; described later) which began approximately 30 minutes after arrival at the home.

Measures

Maternal Warmth

Maternal warmth was assessed with the acceptance subscale of the Child Report of Parental Behavior Inventory (48), completed by mothers and children at W1 to W6. The full Acceptance subscale (16 items; \(\alpha \text{ values } = .83–.94\)) was completed by youth at W3 and W4 (\(\alpha = .86\) and \(\alpha = .86\), respectively). Mothers completed the full Acceptance subscale at all waves (\(\alpha \text{ values } = .81–.88\)).

Cortisol Response to the TSST

Saliva samples were collected from participants before, immediately after, and 20 and 40 minutes after a video-recorded modified TSST. Three 1-minute trials of mental arithmetic (difficulty adjusted based on performance and conducted under time pressure) were followed by 2 minutes of preparation and 4 minutes of a speech about personal strengths and weaknesses. To increase the social-evaluative aspect of the task, participants were told that the speech would be graded and evaluated by a panel of psychologists. 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 (mean [SD] = 4.62 [2.65]) and after (mean [SD] = 6.92 [4.68]) the task was significant (\(t(161) = −6.8, p < .001\), indicating negative emotional response to the task.

Saliva samples were obtained with the Salivette (Sarstedt, Rommelssdorf, Germany) and shipped on dry ice to Salimetrics (State College, PA) for analysis of free cortisol using high-sensitive enzyme immunoassay. The test has a range of sensitivity from 0.007 to 1.8 \(\mu G/dl\), and mean intrassay and interassay coefficients of variation of 4.13% and 8.89%. Cortisol values were log transformed to correct for deviations from normality. Total cortisol across the task was the primary outcome variable, quantified by AUCg, a summary measure reflective of the magnitude of cortisol response over a specified time (49).

Data Analyses

Preliminary Analyses

\(T\)-tests and correlations were used to evaluate potential covariates for cortisol AUCg, including participant demographics (e.g., age, sex, W1 family income, ethnicity, and time since parental divorce) and information from the day of testing (time of day, recent meals, exercise, caffeine, medications, or hormonal contraceptive use). Only time of day of testing was a statistically significant correlate of cortisol AUCg (\(r = −0.17, p = .035\)).
Primary Analyses
The primary study analyses were conducted using the general analysis program MPlus 7.0 (50), which uses full-information maximum likelihood to manage missing data. Reports of maternal warmth from W1 to W4 were modeled with a latent variable to capture warmth during childhood/early adolescence, which was used to predict warmth in late adolescence (W5) and cortisol AUCg in young adulthood (W6; see Fig. 1). Model fit was examined using Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Square Residual (SRMR), using criteria by Hu and Bentler (51) to interpret fit indices. The statistical significance of the indirect path from maternal warmth in early adolescence to cortisol AUCg in young adulthood via maternal warmth in late adolescence was evaluated using the multivariate delta method (52,53). The proposed model was evaluated separately for child- and mother-reported maternal warmth. Time of day was covaried with cortisol AUCg. The models were repeated adjusting for W6 child- and mother-reported maternal warmth to address the hypothesis that the effects would be independent of current maternal warmth.2

RESULTS

Preliminary Results
Pearson correlations were computed for mother and child reports of maternal warmth at each wave (see Table 2). Across Waves 1–5, child reports were significantly and positively correlated with each other and mother self-reports of warmth were significantly correlated with each other. Correlations between child and mother reports, however, ranged from nonsignificant to significant (p values = .001–.69).

Primary Results

Child Report of Maternal Warmth
The first model evaluated child reports of maternal warmth across early (W1-W4) and late (W5) adolescence in the prediction of cortisol AUCg in young adulthood (W6). The model was of good fit (CFI = 0.986, χ²(11) = 15.8, p = .15, RMSEA = 0.043, [0.0; 0.086], SRMR = 0.025). Maternal warmth in early adolescence positively predicted warmth in late adolescence (p < .01). Higher warmth in late adolescence significantly predicted lower cortisol AUCg in young adulthood, p = .031. Warmth in early adolescence did not directly predict cortisol AUCg (p = .58), but the indirect path to AUCg from early adolescence through late adolescence was significant (standardized estimate = −0.092, standard error = 0.045, p = .041). When warmth in young adulthood (W6) was included in the model, overall model fit declined slightly (CFI = 0.970, χ²(15) = 26.1, p = .04, RMSEA = 0.056, [0.014; 0.090], SRMR = 0.031), but the path from late adolescent maternal warmth to AUCg remained significant (p = .036; see Fig. 1).

Mother Report of Maternal Warmth
The second model evaluated mother reports of maternal warmth in the prediction of offspring cortisol AUCg in young adulthood. Although the overall model was of adequate fit (CFI = 0.98, χ²(11) = 20.1, p = .04, RMSEA = 0.059, [0.01; 0.10], SRMR = 0.030), neither of the paths predicting cortisol AUCg from mother-reported maternal warmth was statistically significant (early adolescence, p = .58; late adolescence, p = .42). Inclusion of mother-reported warmth at W6 improved model fit (CFI = 0.987, χ²(15) = 22.24, p = .11, RMSEA = 0.045,
DISCUSSION

Although rates are decreasing, parental divorce remains a common stressor for children, with nearly 50% of first marriages in the United States ending in divorce (54). Parental divorce is a well-established risk factor for a number of short- and long-term mental and physical health problems. However, the presence of a responsive, supportive caregiver has consistently emerged as a protective influence for children exposed to varied forms of adversity, including children from divorced families (43). The current longitudinal study evaluated maternal warmth as a protective influence on biological regulation among young adults who experienced parental divorce during childhood. Results suggested that higher child-reported maternal warmth, assessed at multiple time points in late childhood and adolescence, was associated with lower cortisol response assessed at a challenging interpersonal task in young adulthood, even after including maternal warmth in young adulthood. Mother-reported maternal warmth, however, did not predict cortisol response in offspring.

This study advances research on the long-term impact of childhood adversity on cortisol regulation in several respects. First, although other forms of adversity (e.g., maltreatment) are well studied, limited research has evaluated long-term biological correlates of childhood parental divorce. Second, rather than relying on retrospective reports of maternal warmth or a single self-report of maternal warmth, the current study prospectively examined both mother and child reports of maternal warmth at 5 time points across late childhood and adolescence. Children's perceptions of higher maternal warmth in early adolescence predicted higher maternal warmth in late adolescence, which predicted lower cortisol response in young adulthood, even after including maternal warmth in young adulthood in the model. These findings contribute to a growing literature documenting the critical influence of warm, sensitive caregiving during childhood on biological regulation later in development. The results also extend previous research using retrospective reports to demonstrate relations of childhood caregiver warmth with adult cortisol stress responses.

Third, this study assessed both child and mother reports of caregiver warmth. Only child-reported maternal warmth predicted later-life cortisol response. In combination with a relatively low degree of correlation between mother and child reports of warmth, not uncommon in developmental research (55), these results suggest that children’s perceptions of the quality of their relationships with their mothers may be better predictors of long-term biological consequences than maternal perceptions. The potential for social desirability bias in caregiver self-reports of their parenting behavior calls for research with a broader perspective that can be obtained by collecting child reports (56). The current results support the importance of considering the child's perspective when evaluating the long-term biological correlates of childhood adversity.

There are several limitations of the study. First, we did not assess cortisol activity at study entry, and we cannot determine the developmental timing or changing pattern of influence of maternal warmth on HPA axis regulation. Second, assessments were conducted in the home, which likely reduced the magnitude of cortisol reactivity to the task relative to laboratory-based protocols. Third, because all participants experienced parental divorce, we cannot determine whether the prospective influence of maternal warmth on cortisol in adulthood applies broadly to childhood adversity or is unique to divorce. Finally, we did not evaluate the impact of paternal warmth on offspring cortisol regulation. The father-child relationship is typically more disrupted after divorce than the mother-child relationship (57), and the quality of paternal parenting is independently related to children’s postdivorce adjustment (43). A growing research literature recognizes the importance of father-child relationships for developing biological regulatory systems as well (e.g., Ref. (58)), and it will be important to consider paternal warmth in future research.

CONCLUSIONS

Compelling evidence demonstrates the potential mental and physical health risks associated with childhood exposure to parental divorce. However, there are considerable individual differences in outcomes, with many children demonstrating resilience after parental divorce. Consistent research has suggested the power of positive parenting to promote child mental and physical health in the face of adversity, but how positive parenting promotes resilience has not been fully identified (59). The current study provides support for biological regulation as a pathway by which caregiver warmth promotes positive offspring adjustment after parental divorce. This study advances the literature by prospectively examining both mother and child reports of maternal warmth at multiple time points across development in a sample of recently divorced families. Child reports of maternal warmth in adolescence predicted lower cortisol response across a challenging interpersonal task in young adulthood, but mother reports were not significantly associated with later offspring cortisol response. The results demonstrate the importance of children’s perceptions of parenting and suggest that for children, the presence of a supportive caregiver during a stressful period in development may promote efficient biological regulation during moderately challenging situations later in life.

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