Published in final edited form as:

Int J Psychophysiol. 2012 May ; 84(2): 172–179. doi:10.1016/j.ijpsycho.2012.02.003.

Hostile behavior links negative childhood family relationships to heart rate reactivity and recovery in young adulthood

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Abstract

Prior research has demonstrated that vulnerability to stress is influenced by early life experiences. This study evaluates the impact of negative childhood family relationships on cardiovascular stress reactivity in young adulthood. Participants (age 18–22) from families characterized by negative ($n = 39$) or positive relationships ($n = 36$) engaged in a role-play conflict task. Hostile/aggressive verbal behaviors during the task were observed, and blood pressure (BP) and heart rate (HR) responses were measured before, during, and after the task. Participants from negative families engaged in more hostile/aggressive verbal behavior during the task and showed attenuated HR reactivity. Hostile/aggressive verbal behavior predicted attenuated HR reactivity and recovery. Path analyses linked negative family relationships to more hostile verbal behavior during the task, and attenuated HR reactivity and recovery. These results support the development of hostile/aggressive behavior in social situations as a pathway linking childhood adversity to stress vulnerability across the lifespan.

Keywords

childhood adversity; hostility; cardiovascular reactivity

1. Introduction

Exposure to adverse childhood family environments increases the risk for negative psychological and physical health outcomes over the lifespan (Weich et al., 2009). A recent 45-year epidemiological study found that psychopathological consequences of childhood adversity can persist through adolescence, young adulthood, and into middle-age (Clark et al., 2010). Reports emanating from the Adverse Childhood Experiences study link early life adversity to a wide range of psychological and physical health outcomes, including obesity, depression, smoking, substance use, chronic obstructive pulmonary disease, chronic bronchitis and emphysema, and ischemic heart disease (Anda et al., 2006). The strength and frequency of these associations has prompted research into the mechanistic pathways by which early family risk factors exert their influence. An understanding of these processes may offer important insight into intervention strategies to reduce the long-term health impact of adverse childhood experiences.

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Research into patterns of physiological stress reactivity has proven informative as one possible pathway linking childhood adversity to negative mental and physical health outcomes. Although the majority of research has focused on the hypothalamic-pituitary-adrenal (HPA) stress response system, childhood adversity has also been associated with dysregulation of the sympathetic nervous system. For example, dysregulated patterns of cardiovascular activity (Davies et al., 2009), elevated blood pressure (Ballard et al., 1993), and higher skin conductance level reactivity (El-Sheikh, 2005) have been observed among children from homes characterized by high conflict. Notably, these relations may persist later in life; poor cardiovascular stress reactivity and recovery patterns have been observed among young adults reporting poor family relationships in childhood (Luecken, 1998; Luecken et al., 2005). Elevated blood pressure and greater cardiovascular disease risk have also been observed among adults reared in early family environments characterized by low socioeconomic status (SES) and conflict (Lehman et al., 2009) and those maltreated in childhood (Batten et al., 2004).

Little is known about mechanisms underlying the relation between childhood adversity and cardiovascular stress responses. Lovallo and Gerin (2003) suggest that specific patterns of physiological stress reactivity may arise from individual differences at a cognitive-emotional level, as people evaluate events and formulate habitual behavioral responses. Thus, characteristic patterns of appraising and responding to stressful situations represent an important pathway linking stress to long-term disease outcomes. Bearing associations with both negative childhood family environments and physiological reactivity, hostility and hostile/aggressive behavioral responses to stress represent a particularly promising pathway for exploration.

Smith (1994) defines hostility as “a devaluation of the worth and motives of others, an expectation that others are likely sources of wrongdoing, a relational view of being in opposition toward others, and a desire to inflict harm or see others harmed” (p. 26). Negative parental behaviors, including rejection, low affection, conflict, and strict control, are associated with trait hostility in children and young adults (Matthews et al., 1996). The behavioral component of hostility, aggression, has been observed among children exposed to poor quality family environments (Andreas and Watson, 2009) and “destructive” conflicts (Cummings et al., 2004). Modeling of aggressive tactics in the home has been hypothesized to provide children with a framework by which to respond to other conflicts, and studies have observed direct effects of parental marital aggression on children’s aggressive behaviors at home and school (Erath and Bierman, 2006). Researchers have also theorized that negative interactions with caregivers disrupt children’s ability to self-regulate their emotions, leading to aggressive behavior (Cummings et al., 2004). Importantly, conditions of conflict early in life may contribute to a persistent hostile/aggressive interpersonal style, setting the stage for more hostile interpretations and aggressive behavioral responses to later experiences of stress. Larkin and colleagues (1998) provide evidence that adults reared in an early family environment characterized by low cohesion and adaptability utilize more negative verbal behavior during a stressful role-play task.

A hostile and aggressive orientation towards others has been associated with long-term adverse cardiovascular outcomes, including cardiovascular disease, hypertension, and abnormal cholesterol levels (Miller, Chen, & Cole, 2009). It is commonly theorized that exaggerated or prolonged cardiovascular reactivity is a mechanism linking hostility to long-term health outcomes (Das and O’Keefe, 2006; Rozanski, Blumenthal, & Kaplan, 1999). However, the literature is marked by inconsistency regarding the impact of self-reported trait hostility on the cardiovascular stress response among adults. Positive associations between adult hostility/aggression and blood pressure and heart rate reactivity to stress have been reported (Chida and Hamer, 2008), as well as negative associations (Kline et al., 2008), and

contrasting effects of hostility on blood pressure and heart rate (Hernandez et al., 2009). High hostility has been associated with delayed recovery of blood pressure after stress (Brydon et al., 2010; Anderson et al., 2005), while others have found no differences in recovery between participants high and low in hostility (Vella and Friedman, 2007).

Similarly, existing literature on childhood adversity, hostility/aggression and cardiovascular reactivity presents a complex picture of hyper-reactivity in some contexts and hypo-reactivity in others. Although it seems somewhat consistent that children demonstrate elevated blood pressure and heart rate in the context of adverse family environments, the implications for stress responsivity in young adulthood are less clear, as both exaggerated and blunted patterns of cardiovascular activity have been reported. Further, as the aforementioned studies indicate, empirical evidence links hostile/aggressive states and behaviors among adults to both higher and lower cardiovascular reactivity. A plausible conclusion to be reached from these seemingly contradictory findings is that departures from normative cardiovascular activity and reactivity represent dysregulation that is potentially health-damaging. Such an interpretation is consistent with theories of allostatic load, suggesting that chronic stress can promote dysregulated physiological reactivity, as evidenced by responses that are exaggerated, prolonged, or blunted, contributing over time to the etiology of heart disease and infectious illnesses (McEwen and Wingfield, 2003). Exaggerated or prolonged cardiovascular arousal has been associated with the development of hypertension and preclinical and clinical cardiovascular disease (Treiber et al., 2003), while blunted cardiovascular reactivity has been linked to poorer self-rated health, depression, addictions, and obesity (Phillips, 2011).

Although theoretical models have linked early life stress to health and disease via a pathway of hostility that influences behavior and biological responsivity (Cohen et al., 2007; Repetti et al., 2002), empirical evidence has mainly evaluated discrete associations between individual components in such a pathway; comprehensive models remain to be tested (Matthews and Gallo, 2011). Moreover, prior studies of adult hostility have relied primarily upon limited self-report measures. The assessment of observable hostile behavior provides a different perspective that may advance understanding of the relations between hostility and cardiovascular activity. The current study extends prior literature by testing an integrative model of childhood adversity, hostile verbal behavior, and cardiovascular stress responses. It was hypothesized that young adults from negative childhood family environments would display more hostile/aggressive verbal behavior during a social stress interaction. It was further predicted that those from negative families would have poor cardiovascular recovery after the interaction. Given the preliminary state of this research area, we take an empirical approach to the evaluation of cardiovascular reactivity, and do not put forth a directional hypothesis. Finally, a path model was hypothesized suggesting that group differences in cardiovascular reactivity and recovery could be explained by hostile/aggressive verbal behavior during the task.

2. Material and methods

2.1. Participants

Participants were 75 students recruited from Introductory Psychology classes after completing a large screening survey that included the Family Relationships subscales (FR; conflict, cohesion, expressiveness) of the *Moos Family Environment Scale* (FES; Moos and Moos, 1994; $\alpha = .90$). Respondents were asked to complete the FES in reference to their family environment prior to age 16. The three subscales were combined and scored such that higher FR scores reflect more positive relationships. Respondents age 18–22, raised in continuously married families by both biological parents, and who scored in the highest (positive relationships) or lowest (negative relationships) quartiles of FR on the screening

survey were invited to participate. Respondents who experienced parental death or divorce were not eligible. Eighty-one potential participants were invited to a lab session 1–3 months after completing the screening survey, at which time they again completed the FES. Only participants who scored within the same positive or negative group on both administrations were included in analyses ($n = 75$; 39 from the lowest quartile and 36 from the highest quartile). Test-retest reliability on the FES was high, $R = .91$. Sample characteristics are displayed in Table 1.

2.2. Procedure

Participation occurred between 1–5 PM, Monday-Friday. After providing informed consent and measuring height and weight, participants stood in the location of the upcoming role-play task while 10 minutes of pre-task cardiovascular measures were taken. Participants were instructed on and completed a 10-minute role-play task, after which 10 more minutes of cardiovascular readings were taken while participants remained standing. Participants completed questionnaires after the role-play.

2.2.1. Role-play task—The role-play task was chosen to reflect a naturalistic interpersonal stressor and has elicited angry emotional and behavioral responses in prior research (Frazer et al., 2002; Semenchuk and Larkin, 1993). Conflictual role-play tasks are believed to enhance the generalizability of cardiovascular responses to stress observed in the laboratory (Waldstein et al., 1998), especially among individuals high in hostility (Suls and Wan, 1993). For 10 minutes, participants role-played a challenging social situation (requesting a neighbor to turn down loud music so he/she can study for an important exam) with a same-sex research assistant. The interaction was videotaped, and the “neighbor” maintained a neutral expression and posture while following an ordered series of scripted responses indicating a refusal to cooperate. Experimenters and research assistants were blind to the participant’s family relationship history.

2.3. Hostile/aggressive verbal behavioral responses

Participant verbalizations during the role-play task were coded for spoken expressions of hostility/aggression. Categories of behavioral hostility/aggression were chosen in accordance with Smith’s (1994) definition, and included Patronize/Sarcasm/Mock (e.g., mimicking the research assistant), Blame/Insult (e.g., calling the research assistant “stubborn” or “inconsiderate”), Demand/Insist (e.g., “Shut the music off *now*”), Threat of Revenge (e.g., “Next time you have a big exam I’m going to turn *my* music up”), and Threat of Violence (e.g., “I’m going to make both you and your music be quiet”). Each interaction was divided into 30-second intervals and coded for the absence/presence (coded as 0 or 1) of each strategy in each interval. Two members of the research team, blind to the participant’s family relationship history, coded a small subset of videotapes collaboratively until multiple examples of each strategy were observed and agreed upon. Coders then worked independently until 10% of the videotapes had been coded consecutively with a reliability (κ) ≥ 0.85 . The remaining videotapes were then divided among coders for completion, with a random 10% of the remaining videos coded collaboratively to verify continued interrater reliability. Interrater reliability was high for all categories, ranging from 0.85 to 0.98. A composite hostile/aggressive verbal behavior variable was created by summing the frequency of occurrence of each type of behavior.

Confirmatory analysis of the composite variable was conducted with structural equation modeling (SEM; Amos 18.0; Maximum Likelihood Estimation), by forming a latent variable with the 5 categories (patronize, blame, demand, revenge, violence) as indicators. The model represented a good fit to the data, $\chi^2(5) = 2.3$, $p = .80$, CFI = 1.0, RMSEA = 0.0, {0.0; 0.098}. All factor loadings were significant at $p < .05$.

2.4. Cardiovascular Measures

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were measured using an Omega 5600 adult blood pressure monitor (Invivo Research; Orlando, FL), with the cuff positioned on the participant's non-dominant arm. The Omega 5600 uses an oscillometric method of measurement to provide SBP, DBP, and HR. Readings were taken every two minutes during ten minute pre-task, task, and recovery periods, and were then averaged for the pre-task, task, and recovery experimental periods to create three repeated measures.

2.5. Anxiety and depressive symptoms

Because of the potential confounding effects of current anxiety or depressive symptoms on the relation between family relationship history and current cardiovascular activity, participant reports of recent symptoms of anxiety were assessed with the Beck Anxiety Inventory (BAI; Beck, 1990; $\alpha = .91$), and recent symptoms of depression were assessed with the Beck Depression Inventory II (BDI; Beck, 1996; $\alpha = .84$).

2.6. Data Analyses

Repeated measures GLM were used to evaluate the impact of childhood family relationships and hostile/aggressive behavior on SBP, DBP, and HR responses to the role-play task. Greenhouse-Geisser corrections were used to correct for sphericity. Family relationships group (coded with the negative relationships group = '0' and the positive relationships group = '1') or hostile/aggressive verbal behavior (entered as a continuous variable) served as independent variables, and three repeated cardiovascular readings (pre-task, task, and recovery) served as dependent variables. Participant sex and BMI were covariates in models predicting SBP and DBP (see preliminary analyses for details of covariate selection). Models predicting HR included BMI and anxiety as covariates. Analyses predicting hostile/aggressive verbal behavior included participant sex as a covariate. BMI and anxiety were centered at the sample mean. Following significant omnibus tests of differences in the overall pattern of responses, planned within-subjects contrasts evaluated reactivity (the change from pre-task to task measures) and recovery (the change from task to recovery measures). Partial eta-squared (η_p^2) is reported as a measure of effect size.

AMOS 18.0 was used to evaluate the proposed structural model shown in Figure 1. Standardized residualized change scores were calculated to represent reactivity and recovery. The residualized change score, which represents the difference between the observed HR and the predicted HR, is used as an alternative to calculating difference scores because it adjusts for baseline but avoids some of the reliability concerns with difference scores (MacKinnon, 2008). This strategy does not fully remove baseline differences between the groups, but is used to minimize their effect on change scores. A standardized residualized change score for reactivity was created by predicting task HR from pre-task HR. A standardized residualized change score for recovery was created by predicting recovery HR from task HR. Anxiety symptoms were included as a covariate in the path model. Because men exhibited more hostile/aggressive verbal behavior, sex was also included as a covariate. Maximum Likelihood Estimation was used, and model fit was examined using Chi-square Goodness of Fit Test, as well as Cumulative Fit Index (CFI) and Root Mean Square Error of Approximation (RMSEA), as recommended for smaller sample sizes (Fan et al., 1999).

3. Results

3.1. Preliminary Analyses

3.1.1. Demographic comparisons—The family relationships groups were compared for equivalence on demographic variables and covariates potentially associated with cardiovascular activity. Chi-square and t-tests found no group differences in sex ($p = .73$), ethnicity ($p = .72$), family income ($p = .36$), age ($p = .36$), body mass index (BMI; $p = .51$), waist/hip ratio ($p = .60$), hormonal contraceptive use ($p = .42$), medications ($p = .38$), or smoking status ($p = .21$). On the day of testing, the groups did not differ on the time of day of participation ($p = .84$), number of cigarettes smoked ($p = .46$), or caffeine/energy drinks ($p = .39$). Higher BMI predicted elevated overall SBP ($p < .01$) and SBP reactivity ($p = .049$). BMI did not predict overall DBP ($p = .09$), DBP reactivity ($p = .65$), overall HR ($p = .91$), or HR reactivity ($p = .09$). Because BMI predicted SBP and was near-significant in the prediction of overall DBP and HR reactivity, it was included as a covariate in all statistical models predicting cardiovascular outcomes.

3.1.2. Current anxiety and depression—Participants from families characterized by negative relationships reported elevated symptoms of anxiety, $t(71) = 2.2$, $p = .032$, and depression, $t(71) = 3.4$, $p = .001$, relative to those from families characterized by positive relationships (see Table 1). Anxiety predicted elevated HR reactivity to the challenge task, $F(2, 126) = 3.4$, $p = .050$, but did not predict SBP or DBP reactivity, p 's $.26$, or overall HR, SBP, or DBP, p 's $.29$. Depressive symptoms did not predict HR, SBP, or DBP reactivity, p 's $.46$, or overall HR, SBP, or DBP, p 's $.28$. Therefore, anxiety was included as a control variable in statistical analyses of HR. Neither anxiety ($p = .59$) nor depressive symptoms ($p = .30$) were significantly correlated with hostile behavior during the task.

3.1.3. Gender differences—Women had lower SBP and DBP across the task than men (SBP $p < .001$; DBP $p = .017$). Women did not differ in SBP reactivity ($p = .25$), but did show less DBP reactivity than men ($p = .001$). Because of these gender differences, participant sex was a covariate in models predicting SBP or DBP. Men and women did not differ in overall HR ($p = .14$) or HR reactivity ($p = .57$). Men and women did not differ on reports of family relationships ($p = .87$). Men displayed more hostile/aggressive verbal behavior ($M = 7.5$, $SD = 7.4$) than women ($M = 4.3$, $SD = 4.6$; $p = .04$).

3.1.4. Reactivity to the role-play task—Significant reactivity (change from pre-task to task level) to the role-play task was observed across the sample for SBP, $F(1, 67) = 140.7$, $p < .001$, DBP, $F(1, 66) = 145.4$, $p < .001$, and HR, $F(1, 67) = 14.0$, $p < .001$. Significant recovery (change from task to recovery level) was also observed across the entire sample for SBP, $F(1, 67) = 86.2$, $p < .001$, DBP, $F(1, 66) = 66.4$, $p < .001$, and HR, $F(1, 67) = 9.5$, $p = .01$.

3.1.5. Examination of outliers—Outliers were examined using the measures of influence DFFITS and DFBETAS (SPSS 17.0; Chicago, IL). For small to moderate sample sizes, DFFITS and DFBETAS greater than an absolute value of 1 suggest an influential case and require further analyses (Cohen et al., 2003). Three cases were identified as potentially influential (2 from the negative family group, 1 from the positive family group). Upon closer inspection, all three reported elevated anxiety ($M = 26.3$; sample $M = 7.0$). When analyses were repeated excluding those 3 data points there were no significant changes in the pattern or significance of findings, with the exception that anxiety symptoms were no longer predictive of HR reactivity, $p = .55$. Thus all cases were retained for analyses.

3.2. Family relationships and cardiovascular responses to the task

We first evaluated the influence of a negative childhood family environment on SBP and DBP responses to the roleplay task. A repeated measures GLM predicting SBP from family relationships group, controlling for sex and BMI was not significant in the prediction of SBP response to the task ($p = .31$) or mean SBP across the task ($p = .88$). Similarly, family relationships group was not significant in the prediction of DBP response to the task ($p = .24$) or mean DBP across the task ($p = .63$).

We next evaluated the influence of a negative childhood family environment on HR responses. Family relationships group was a significant predictor of HR response to the task, $F(2, 124) = 4.2$, $p = .018$ (Greenhouse-Geisser adjusted $p = .027$), $\eta_p^2 = .063$ (see Figure 2). Tests of within-subjects contrasts were significant for the change from pre-task to task levels, $F(1,62) = 6.4$, $p = .014$, $\eta_p^2 = .093$, such that the negative relationships group had attenuated reactivity. The change from task to recovery did not significantly differ between the groups, $p = 0.20$. Although the groups did not differ in overall HR ($p = .24$), the negative relationships group showed a non-significant trend towards higher pre-task HR than the positive relationships group, $F(1,66) = 3.6$, $p = .063$.

3.3. Family relationships and hostile/aggressive behavior

Analyses comparing family relationships groups on hostile/aggressive verbal behavior during the task found that the negative relationships group displayed significantly more hostility/aggression during the task than the positive relationships group, $F(1,55) = 8.0$, $p = .007$ (see Table 1).

3.4. Hostile/aggressive behavior and cardiovascular responses to the task

Next, we evaluated if hostile/aggressive verbal behavior during the task would be associated with cardiovascular responses. Hostile/aggressive behavior was not a significant predictor of SBP responses to the task ($p = .11$), DBP responses to the task ($p = .69$), overall SBP ($p = .33$) or overall DBP ($p = .59$). However, a repeated measures GLM predicting HR from hostile/aggressive behaviors was significant, $F(2,108) = 4.07$, $p = 0.020$ (Greenhouse-Geisser adjusted $p = .029$), $\eta_p^2 = .070$ (see Figure 3). Analyses of reactivity were significant for the change in HR from pre-task to task levels, $F(1,54) = 5.1$, $p = 0.028$, $\eta_p^2 = .087$, showing attenuated reactivity among those displaying more hostile/aggressive verbal behaviors. The change from task to recovery was also significant, $F(1,54) = 4.5$, $p = 0.038$, $\eta_p^2 = .078$, such that more hostile/aggressive participants had attenuated HR recovery. Hostile/aggressive behaviors did not predict overall HR ($p = .91$).

3.5. Path model

The proposed path model shown in Figure 1 evaluated a path linking negative childhood family relationships to more hostile/aggressive verbal behavior during the conflict task, which then would predict attenuated reactivity and recovery. Because the prior analyses were only significant for HR, the model was only evaluated predicting HR reactivity and recovery. The model represented a good fit to the data (criteria by Hu and Bentler, 1999): $\chi^2(6) = 2.8$, $p = .84$, CFI = 1.0, RMSEA = 0.00, {0.0; 0.089}, AIC = 44.8, all paths significant at $p < .05$. More negative family relationships predicted higher hostile/aggressive behavior, and higher hostile/aggressive behavior predicted attenuated reactivity and attenuated recovery. Higher anxiety predicted higher reactivity and stronger recovery. A comparison model evaluating if elevated pre-task HR may have contributed to hostile/aggressive behavior included a path from pre-task HR to hostile verbal behavior. This model was a poor fit to the data, $\chi^2(9) = 33.0$, $p < .01$, CFI = .697, RMSEA = 0.19, {0.12; 0.26}, AIC = 85.0, and the path from pre-task HR to hostile verbal behavior was not significant, $p = .$

39. Results suggest that the more parsimonious original model is a better fit to the observed data.

4. Discussion

The present study examined the influence of negative family relationships in childhood on behavioral and cardiovascular responses to a conflict interaction task in young adulthood, and evaluated a behavioral pathway linking childhood adversity to cardiovascular reactivity in young adulthood. Attenuated HR reactivity to the task was observed among young adults reporting more negative family relationships relative to those who experienced more positive relationships. This finding supports a growing research literature linking early adversity to attenuated biological stress response systems (Miller et al., 2007). Exposure to chronic stress, as is the nature of consistently negative family relationships, has been theorized to lead to reduced physiological reactivity to new stressors over time (Musante et al., 2000). Existing literature has primarily assessed neuroendocrine systems; however attenuation may also be apparent in sympathetic nervous system responses to stress. For example, childhood adversity has been associated with lower vascular resistance in childhood (Gump et al., 1999), as well as lower daily blood pressure (Luecken et al., 2009) in young adulthood. Consistent with the results of the current study, attenuated heart rate reactivity has also been observed among young adult men reared in stressful childhood family environments (Torres et al., 2001). Although research often focuses on the negative health consequences of increased reactivity, attenuated HR reactivity to stress may also put individuals at higher long-term risk for poor health. Responses that do not sufficiently prepare the body to respond to stress or facilitate recovery are recognized as maladaptive, and have been associated with risk for addiction, obesity, cardiovascular disease, poor self-rated health, and depression (Carroll et al., 2009; Phillips, 2011; Salomen et al., 2009).

The current study also evaluated the relation between negative childhood family relationships and behavioral concomitants of hostility. As theorized, we observed greater hostile verbal behavior during the role-play task among young adults reared in negative early family environments. Developmental contextual approaches to social learning describe the family environment as a “training ground” through which parents model and support social skills that influence children’s interpersonal relationships (Cui et al., 2002). Perceptions of the primary caregivers as unsupportive, argumentative, and uncaring may contribute to the development of the hostile behaviors observed among individuals exposed to negative family relationships in childhood. A general interpretive style characterized by heightened vigilance and suspicion of others may develop in the context of stressful and unpredictable childhood environments (Chen et al., 2004); our findings suggest that such an interpersonal orientation may persist later in life. In a recent study of implicit affect, attributions of anger and fear to one’s early childhood family environment were associated a greater tendency to perceived threat in response to ambiguous cues among adults (Chan et al., 2011).

Our results further suggest that hostile behaviors during interpersonal stressors can significantly impact cardiovascular activity. More hostile/aggressive verbal behavior during the role-play task predicted attenuated HR reactivity and recovery. Discrete findings linking childhood adversity to hostile behavior and attenuated HR responses support prior research, however the current study advances the literature by evaluating hostile behavior as a potential mechanism linking adversity to attenuation. A path analytic model of the relations between childhood adversity, contemporaneous hostile/aggressive behavior, and HR stress responses provided a good fit to the data, and suggested that negative family-of-origin relationships can exert a lasting effect on heart rate responses to stress into adulthood via a pathway of hostile verbal behavior during challenging interpersonal interactions. These

results are partially supportive of findings by Larkin et al. (2010), who similarly observed more negative verbal behavior during a stressful role-play task among young adults reared in negative family environments. However, their findings of elevated DBP reactivity in response to stress diverged from the current study which found no effects of hostile verbal behavior on DBP reactivity or recovery.

One strength of this study is in the use of observational measures of hostility and aggression during a simulated social conflict task. Behavioral assessments of hostility have been primarily conducted among children; within adult samples, self-reports have predominated. Smith (2003) recommended that studies inducing stress responses use an experimental stressor relevant to the population or psychosocial risk factor being studied. The construct of hostility is, in part, understood in terms of its consequences on social interactions and social relationships, suggesting that an interpersonal conflict stressor may provide an ecologically valid approach to the study of the impact of childhood adversity and hostility on cardiovascular reactivity. By observing and coding verbal behavior during the role-play, we assessed hostile responses as they occurred, minimizing potential biases in self-reports, and more immediately tying hostile behavior to cardiovascular responses. The observed pattern of aggressive verbal behavior and cardiovascular consequences in participants from negative families may simulate the responses that occur in a variety of social interactions in participants' natural environments, increasing the potential long-term health risks for those exposed to early family adversity. Although self-reported trait hostility in adulthood has been associated with patterns of higher and lower cardiovascular activity, a meta-analysis across 40 studies related childhood aggressive behavior to lower heart rate during a stressful task (Ortiz & Raine, 2004). Thus the method through which hostility is assessed, as well as developmental stage, may be important considerations when evaluating the relation of hostility to cardiovascular activity.

The finding that hostile/aggressive behavior predicted attenuated HR recovery further emphasizes the importance of considering recovery from stress, in addition to reactivity, in gaining a comprehensive understanding of the physiological responses of young adults exposed to adverse childhood family relationships. Delayed cardiovascular recovery after the termination of a stressor may emerge after prolonged stress, and has been linked to future hypertension risk (Steptoe and Marmot, 2005). Prolonged cardiovascular activity resulting from poor recovery may predispose individuals to heart problems in addition to the risks imposed by elevated reactivity (Curtis and O'Keefe, 2002; Stewart and France, 2001).

Although significant reactivity to the task was observed across the sample for SBP, DBP, and HR measures, the relations among family environment, hostility/aggression, and attenuated reactivity and recovery responses found in the present study were only observed among measures of HR. Prior studies of negative family environments have observed an effect on measures of HR (Torres et al., 2003) or BP (Larkin et al., 2010) only, without significant findings on other indices of cardiovascular functioning. These nuanced patterns of stress responsivity highlight the importance of examining multiple physiological indices through which adverse childhood environments may influence health. Individual differences in appraisals of the task were not assessed in the current study, but may help future studies understand differences between HR and SBP findings. For example, "active" stressors are expected to elicit elevations in both SBP and HR, while "passive" stressors may elicit elevated SBP but minimal HR responses (Gregg et al., 1999). Passive and withdrawn orientations toward stressors may develop among individuals consistently exposed to family adversity early in life (Santiago and Wadsworth, 2009; Repetti et al., 2002). Thus, a speculative explanation for our findings is that participants from negative families may have been more likely to approach the conflict task in a passive manner, resulting in similar SBP reactivity but a blunted HR response relative to those from positive families. However, it

should be noted that heart rate and blood pressure responses may appear discrepant when examining any one association within a discrete measurement period, but bear greater relation when multiple measurements are aggregated across tasks or settings (Kamarck and Lovallo, 2003).

Because participants from more negative family environments also reported higher current symptoms of anxiety and depression, it was important to consider whether the heart rate findings were explained by current distress. Higher anxiety predicted greater reactivity to the role-play task across the sample, but the relation of negative family relationships to reduced heart rate reactivity remained significant after controlling for anxiety. Although inclusion in the statistical model does not fully remove the effect of anxiety, it suggests anxiety does not explain the effects of family group on heart rate reactivity. Hostile behavior was uncorrelated with anxiety, and was not predicted by pre-task HR. Further, adjusting for anxiety did not alter the observed behavioral pathway linking adversity to HR reactivity. Nonetheless, additional time for relaxation and acclimation to a novel laboratory environment may help minimize effects of anxiety on cardiovascular reactivity. Levels of depressive symptoms were not significantly related to any of the cardiovascular measures. In short, we find little evidence that current distress explains the relation of a negative childhood family environment to heart rate reactivity in young adulthood.

Results of the current study must be evaluated in light of its limitations. Results may not generalize to adults in a different developmental stage or to the general population. The characteristics of childhood family relationships were assessed retrospectively, and although accuracy cannot be independently verified, high test-retest reliability of the measure was obtained. In addition, experimenters and coders were blind to participants' family environments, and we considered current distress in our analyses. All participants were raised by two continuously married biological parents. The sample may not have captured individuals exposed to more severe family relations or adversity for whom the model of hostility, aggression, and stress reactivity may be especially relevant. Previous research has highlighted the multidimensional nature of aggression (e.g., proactive, reactive, overt, relational, indirect, etc.; Vaillancourt et al., 2007), a distinction not made by the current study. Our observational measure included a composite of multiple types of hostile verbal behavior (e.g., patronizing, blaming, demanding, threatening). It would be interesting for future studies to attempt to link specific types of hostile behavior to particular patterns of HR arousal and recovery.

The measured heart rates in the sample were higher than typical resting heart rates among young adults, potentially because participants were standing and the room was warm. The heart rates in the sample should not be taken to represent clinical measures of resting blood pressure/heart rate. One effect of an adverse childhood environment on HR may be via negative emotionality; it is possible that the negative family group reacted with more arousal upon entering the novel environment or reading the Informed Consent. Chronically elevated HR or HR increases upon entering the lab could contribute to a ceiling effect in reactivity to the interaction task, potentially contributing to reduced reactivity in the negative family group. The groups did not significantly differ in pre-task or overall HR, but it is possible that the sample size limited power to find significant differences. Future studies of HR in natural environments will be important in order to address these possibilities.

There are several of limitations associated with path modeling. Results from SEM analyses support the theorized path model, but the non-experimental design does not allow for conclusions about causality. Alternative models may also fit the data, but the model is strengthened by theoretical support for causal relations and temporal precedence in the ordering of the variables. An alternative model in which pre-task HR predicted hostile

verbal behavior was a poor fit to the data and the path from pre-task HR to hostile behavior was not statistically significant. Finally, the sample size was relatively small for SEM. We used model evaluation indices appropriate for small sample sizes but the results should be considered exploratory until replicated with a larger sample size.

The present study advances research on the health impact of childhood adversity by evaluating a potential behavioral mechanism linking adversity to cardiovascular activity in young adulthood. Negative childhood family relationships were associated with more hostile/aggressive verbal behavioral responses to stress in young adulthood as compared to participants reporting positive childhood family relationships. Additionally, previously observed relations between hostility/aggression and attenuated heart rate reactivity and recovery were extended to young adults with a history of family adversity. In combination, support was found for a path model in which negative family relationships in childhood contributed to behavioral aggression in young adulthood in the context of a challenging interpersonal interaction, which predicted attenuated heart rate reactivity and recovery. Over time, aggressive behavioral responses to recurring interpersonal conflict may contribute to maladaptive cardiovascular activity, increasing long-term health risk. These results provide evidence for the development of hostility as an important psychosocial mechanism of risk for dysregulated cardiovascular responses to stress in young adults exposed to an adverse childhood family environment.

Acknowledgments

Role of the funding source

This research was supported by NIMH R03 MH069804-1 Luecken (PI). The funding source had no role in study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

ABBREVIATIONS

BMI	body mass index
BP	blood pressure
FR	family relationships
HPA	hypothalamic-pituitary-adrenal
HR	heart rate
SAM	sympathetic-adrenomedullary
SBP	systolic blood pressure

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Highlights

- We examined the impact of early family adversity on cardiovascular stress reactivity in young adulthood.
- Young adults from negative families had attenuated heart rate reactivity during a conflict role-play task.
- Young adults from negative families displayed more hostile behavior during the conflict task.
- Hostility during the task was associated with attenuated heart rate reactivity and recovery.
- Negative childhood family environments may impact adult cardiovascular functioning via a pathway of hostile responses to stress.

Figure 1.

A path model linking childhood family relationships to HR responses to a conflict task via hostile/aggressive verbal behavior^a

^aAll paths significant at $p < .05$. Regression weights and standard errors shown. Higher scores on Childhood Family Relations indicate more positive relationships. HR reactivity and recovery are represented by residualized change scores. Standardized error terms not shown.

Figure 2.

HR responses to roleplay task by the quality of childhood family relationships^a

^a“Neg FR” = negative family relationships group; “Pos FR” = positive family relationships group; Error bars are standard errors.

Figure 3.

Hostile/aggressive behavioral responses to the task and HR reactivity^a

^a“low aggr/ho”: set at 1 SD below the mean on hostile/aggressive responses during the task.

“high aggr/ho”: set at 1 SD above the mean on hostile/aggressive responses during the task.

Table 1 Sample demographics and study variables^a

	Total sample	Neg FR	Pos FR
Age (M, SD)	18.9 (.98)	18.8 (.90)	19.0 (1.1)
Gender (N, %)			
Male	38 (50%)	19 (49%)	19 (51%)
Female	37 (50%)	20 (51%)	17 (49%)
Family Income (N, %)			
\$0 – 29,999	3 (4%)	3 (8%)	0
\$30,000 – 44,999	8 (11%)	3 (8%)	4 (11%)
\$45,000 – 59,999	6 (8%)	4 (10%)	2 (5%)
\$60,000 – 79,999	7 (9%)	4 (10%)	3 (8%)
\$80,000 – 99,999	16 (21%)	9 (23%)	7 (20%)
\$100,000 +	34 (45%)	16 (41%)	18 (51%)
No answer	2 (3%)	0	2 (5%)
Ethnicity (N, %)			
White non-Hispanic	57 (76%)	28 (72%)	29 (80%)
Hispanic	12 (16%)	7 (18%)	5 (14%)
African-American	2 (3%)	2 (5%)	0
Asian	2 (3%)	1 (3%)	1 (3%)
Other	2 (3%)	1 (3%)	1 (3%)
BMI (M, SD)	23.5 (3.8)	23.8 (4.1)	23.2 (3.4)
FR (M, SD) **	16.1 (6.7)	10.6 (3.7)	22.2 (2.6)
Anxiety symptoms (M, SD) *	7.8 (7.1)	9.4 (8.2)	5.9 (5.1)
Depressive symptoms (M, SD) **	8.2 (5.6)	10.1 (6.0)	5.9 (4.2)
Hostile/aggressive behavior (M, SD) **	5.9 (6.3)	8.1 (7.9)	3.9 (3.3)
Cardiovascular measures (M, SD) ^b			
SBP			
Pre-task	115.5 (10.7)	117.3 (11.8)	113.7 (9.4)
Task	127.3 (12.2)	127.2 (12.6)	127.4 (11.9)
Recovery	118.0 (10.3)	118.0 (10.5)	118.0 (10.4)
DBP			
Pre-task	76.2 (6.3)	76.7 (6.9)	76.8 (5.7)
Task	86.9 (9.7)	86.0 (10.3)	87.7 (9.2)
Recovery	78.3 (7.3)	78.0 (6.7)	78.6 (7.9)
HR			

	Total sample	Neg FR	Pos FR
Pre-task	91.2 (14.3)	94.6 (16.6)	87.8 (11.3)
Task	94.9 (12.9)	96.4 (14.4)	93.4 (11.3)
Recovery	92.9 (14.0)	95.4 (16.6)	90.4 (10.3)

^a“Neg FR” = negative family relationships group; “Pos FR” = positive family relationships group

^b unadjusted means

* group difference $p < .05$

** group difference $p < .01$