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Childhood Parental Warmth and Heart Rate Variability in Midlife: Implications for Health

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Abstract

The current study investigated high-frequency heart rate variability (HF-HRV) as a potential mediator between childhood parental warmth and later health and mortality outcomes. Participants were 1,255 adults (56.9% female). Childhood parental warmth was reported retrospectively at mean age 46; resting HF-HRV was measured at mean age 57; cardiovascular health and self-evaluated health were assessed at mean age 57 and 63, and mortality records extracted at mean age 63. Results revealed a positive association between childhood parental warmth and resting HF-HRV, as well as associations between higher HF-HRV and reduced risk of having a later cardiovascular health problem and of mortality by age 63. Mediation analyses revealed a small significant indirect effect of parental warmth, through HF-HRV, on cardiovascular health.

Keywords

heart rate variability; parental warmth; cardiovascular; mortality; MIDUS

The quality of the social environment during childhood has enduring repercussions for offspring health and wellbeing (Chen, Brody, & Miller, 2017). In particular, a growing body of research has found associations between characteristics of the parent-child relationship and offspring physiology, with parenting that is high in warmth generally predicting a more moderate and flexible physiological profile (Flannery, Beauchamp, & Fisher, 2017; Gunnar & Quevedo, 2007; Repetti, Taylor, & Seeman, 2002). However, most prior research has focused on links between parenting and the hypothalamic-pituitary-adrenal (HPA) axis, and much less is known about the potential association between parenting and the autonomic nervous system (ANS). Understanding the relations between childhood parental warmth and ANS physiology has important implications for public health promotion, as more moderate and flexible ANS physiology has been associated with several beneficial health outcomes (Thayer, Yamamoto, & Brosschot, 2010). The present study aimed to (1) address this research gap by testing the association between childhood parental warmth and ANS physiology, indexed through resting high frequency heart rate variability (HF-HRV); (2) add

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to the growing body of evidence linking HF-HRV to lifelong health and mortality outcomes; and (3) test HF-HRV as a mediator between parental warmth and health and mortality.

Parental Warmth and Health

Parental warmth refers to parenting that is affectionate, consistent, and marked by sensitive responding to the child's needs (Zhou et al., 2002). Past mediation studies attempting to explain the association between parental warmth and health have typically focused on two mechanisms: (1) health behaviors, whereby warmer parenting is associated with healthier behavior patterns –e.g., diet, physical activity (Davids, Roman, & Leach, 2017; Graves, Wang, Mead, Johnson, & Klag, 1998); and (2) stress physiology and the immune system, such that warmer parenting predicts more moderate stress reactivity and more adaptive inflammatory processes, which in turn are associated with better health outcomes (Chen, Miller, Kobor, & Cole, 2011; Chen et al., 2017; Miller & Chen, 2010; Uchino & Way, 2017). However, most studies investigating the link between parental warmth and biological processes have focused on the HPA axis and the immune system, and the relatively fewer studies on parental warmth and the ANS are largely limited to youth, precluding the testing of ANS physiology as a mediator between parental warmth and long term health outcomes, such as cardiovascular disease and mortality.

Parental Warmth and Offspring Physiology

Research with children and adolescents has shown that warm parenting can dampen neuroendocrine stress responses (Hostinar, Sullivan, & Gunnar, 2014) and attenuate the effects of adversity on youth physical health (Chen et al., 2017; Farrell, Simpson, Carlson, Englund, & Sung, 2016). Warmer parenting may therefore help dampen the offspring's general physiological reactivity during early-life, helping to maintain the offspring's physiology within more moderate set points, potentially "programming" the development of physiological profiles that are more moderate and adaptive (Miller, Chen, & Parker, 2011).

Parenting may also influence the development of offspring physiology by shaping the development of emotion regulation. During early life, parents act as primary regulators of their offspring's affect and physiology (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Because of this, parents who sensitively respond when their child is in distress, providing scaffolding assistance to the child's attempt to regulate their own physiological reaction, help create internalized patterns of regulatory processes (Thompson, 1994), potentially leading to the development of more moderate and well-regulated ANS physiology. While most studies investigating early life "programming" of offspring biology have focused on the HPA axis, there is growing evidence supporting the need to better understand how early life environmental characteristics, particularly parenting, may predict the development of ANS physiology (Propper & Moore, 2006).

Heart Rate Variability

A common technique for investigating individual differences in ANS physiology is to measure heart rate variability (HRV) (Laborde, Mosley, & Thayer, 2017). HRV is a

measurement of the beat-to-beat changes in heart rate, and high frequency HRV (HF-HRV) is often used as an index of parasympathetic influence, via the vagus nerve, over cardiovascular activity (Porges, 2007). High resting HF-HRV is believed to represent ANS physiology that can flexibly adapt to changing environmental demands, increasing heart rate during times of threat, then quickly returning to a calm resting state once the threat has subsided (Appelhans, & Luecken, 2006).

Previous research on the association between parenting characteristics and resting HF-HRV is somewhat limited. Nevertheless, the evidence so far suggests an association between positive parenting characteristics and high resting HF-HRV. For example, infants of mothers who warmly responded to their infant's communication cues had higher resting HF-HRV (Porter, 2003). In addition warmer parenting (e.g., high involvement, high support) has been shown to predict higher resting HF-HRV in adolescents (Graham, Scott, & Weems, 2017), and reduced decreases in resting HF-HRV over a one-year period (Fox, Aldrich, Ahles, & Mezulis, 2018). Experimental evidence from randomized controlled trials has also revealed associations between (1) increased sensitivity following parenting intervention and higher resting HF-HRV in preschoolers (Bell, Shader, Webster-Stratton, Reid, & Beauchaine, 2018), and (2) reductions in negative parenting following intervention and higher resting HF-HRV in 9-year olds (Tabachnik, Raby, Goldstein, Zajac, & Dozier, 2019).

There is also evidence of associations between parenting quality and HRV change during challenge, sometimes referred to as vagal withdrawal, though findings have been mixed, with warmer or more sensitive parenting sometimes predicting increased HRV change (Calkins, Graziano, Berdan, Keane, & Degnan, 2008; Perry et al., 2012), and sometimes predicting decreased HRV change (Hastings et al., 2018). While these mixed results highlight the complexity of HRV change research, it suggests that parenting quality may influence child state-level ANS physiology in addition to the trait-level ANS physiology captured by resting measures.

HF-HRV and Health

High resting HF-HRV has been previously associated with better physical health and reduced mortality risk. Low resting HF-HRV has been linked to hypertension (Singh et al., 1998), high cholesterol (Christensen, Toft, Christensen, & Schmidt, 1999), and coronary heart disease (Liao, Carnethon, Evans, Cascio, & Heiss, 2002). In addition, low resting HF-HRV has also been previously associated with increased risk for diabetes (Liao et al., 1995) and all-cause mortality (Gerritsen et al., 2001; Tsuji et al., 1994). However, some studies have failed to find associations between HRV and cardiovascular risk factors (Klutting, Kuss, & Greiser, 2010). Given this mixed evidence, it is important to further test the relation between HRV and health outcomes using large samples.

Current Study

The present study aimed to (1) expand our current understanding of the association between childhood parental warmth and adult ANS physiology, (2) investigate the relations between ANS physiology and long-term health and mortality outcomes in older adulthood, and (3)

test HF-HRV as a potential mediator of the relation between parental warmth and health and mortality. Considering some of the previously reported associations between warm parenting and high resting HRV, and high resting HRV and better health and reduced mortality risk, we developed three hypotheses: (1) we hypothesized that adults who reported retrospectively to have received warmer parenting during childhood would exhibit higher resting HF-HRV in midlife; (2) we hypothesized that higher resting HF-HRV would predict better physical health outcomes (cardiovascular health, self-evaluated health) and reduced risk of mortality, and (3) we hypothesized that resting HF-HRV would mediate the relation between childhood parental warmth and health and mortality outcomes in older adulthood.

Methods

Participants

Data for this study were drawn from the Midlife Development in the United States (MIDUS) longitudinal study. The first MIDUS wave involved phone and mail surveys to a nationally representative sample of adults (N = 7,108). In 2004-2009, a subset of this sample (n = 1,255) were recruited for a follow up study, the Biomarker Project of MIDUS 2, involving assessments of biological markers. The third wave, MIDUS 3 began in 2013, and involved phone survey assessments similar to the first wave. Complete details on the MIDUS study are available at www.midus.wisc.edu.

Participants in the current study include individuals who participated in all three MIDUS waves. Of the 1,255 participants who took part in the MIDUS 2 Biomarker Project, 1,148 had technically acceptable resting HF-HRV data. Of the individuals who returned for MIDUS 3, 938 had data on cardiovascular health, and 943 had data on self-evaluated health. Participants included in the current study were: middle-aged, with a mean age at MIDUS 2 of 57.3 (range = 35 to 86); predominantly Caucasian (78.7%), though a large subset of the sample were African American (17.2%), 1.4% were Native American, 0.2% Asian, and 2.5% Other (please see Table 1 for complete sample demographics). On average, participants had moderate childhood socioeconomic status (SES), with 41.3% of participants reporting that at least one of their parents completed some college. Just over half of participants were female (56.8% female; 43.2% male).

Measures

Childhood Parental Warmth.—Childhood parental warmth was measured retrospectively at MIDUS 1 (mean age 46) using a validated questionnaire (Rossi, 2001). Participants were asked to reflect on their childhood experiences, and answer seven questions regarding the quality of their relationship with each parent (participants completed separate 7-item scales for their mother and their father). Questions assessed how much that parent understood the participant's worries; how much attention, effort, and affection that parent provided; as well as the participant's subjective interpretation of the overall quality of the relationship (e.g., *"How would you rate your relationship with your mother/father during the years you were growing up?"*). Responses were coded on a 4-point Likert-type scale, ranging from "*Not at all*" to "*A lot*"; or, regarding quality of the relationship, from "*Poor*" to "*Excellent*"; such that higher scores reflected more parental warmth. Both maternal and

paternal warmth scales showed high internal consistency (Cronbach's alpha of .91 and .92, respectively). The maternal and paternal scores were averaged together to provide a measure of combined parental warmth.

High-Frequency Heart Rate Variability.—High-frequency heart rate variability (HF-HRV) was measured during MIDUS 2, using a 3-lead electrocardiogram (ECG) attached to the chest in Einthoven's triangle configuration. ECG recordings took place in the morning, after an overnight clinic stay, following breakfast, with no caffeine consumption permitted. ECG data were recorded during an 11-minute seated, resting baseline period, as part of a larger ECG protocol involving cognitive tasks. The current study utilized data from the resting baseline measure. ECG data were digitized at 500 Hz by a 16-bit National Instruments analog-to-digital board. ECG wave R peaks were visually inspected and cleaned. Arrhythmias were handled using interpolation. Interbeat-intervals (IBIs) were processed through a Fast Fourier transformation algorithm, with a high frequency band filter set at 0.15 – 0.50 Hz, providing mean HF-HRV from two 300-second epochs, which were then averaged together. If uncleanable noise precluded the collection of a minimum of 180 continuous seconds, data from that participant were not calculated.

Cardiovascular Health.—To create a measure of cardiovascular health, we used participants' answers to ten questions, during MIDUS 3 (mean age 63), regarding the presence or absence of heart problems as diagnosed by a physician. Participants were asked if they had ever been diagnosed by a physician with any of the following: stroke, heart attack, high blood pressure, valve disease, blocked artery, irregular heartbeat, heart murmur, or heart failure; participants were also asked if they had ever had a major heart procedure, and if they currently experience chest pain while walking. Participant responses to these questions were used to create a dichotomous variable, such that individuals who answered "*No*" to all questions were coded as "0" (excellent cardiovascular health), and individuals who answered "*Yes*" to at least one question were coded as "1". We also created a count variable by summing the total number of "*Yes*" responses, such that higher scores represented more cardiovascular health problems.

Self-Evaluated Health.—Self-evaluated health was measured during MIDUS 3 via phone surveys. This measure was indexed using one item, answered on a 5-point Likert-type scale: *"In general, would you say your physical health is excellent, very good, good, fair, or poor?"* Responses to this question were reverse coded from the original coding scheme so that higher scores indicated better self-evaluated health (e.g. "*poor*" = 0; "*excellent*" = 4). This question has been found in previous studies to be predictive of objective health outcomes such as mortality (Benjamins, Hummer, Eberstein, & Nam, 2004).

Mortality.—Mortality data were collected through October 2015, using three methods: (1) tracing conducted by the University of Wisconsin Survey Center, which used a variety of database searches to confirm participant identity and status, (2) formal searches through the National Death Index (NDI), and (3) longitudinal sample maintenance procedures, including regular participant outreach.

Covariates.—Additional demographic and health data were collected during MIDUS 2, when the ECG protocol was conducted. Demographic information included: age, sex, race, and childhood SES. Race was entered as binary code (Caucasian = 0, Non-Caucasian = 1) given that other racial categories had very low frequencies and did not allow sufficient statistical power for inclusion. Childhood SES was indexed through parental education level, as an ordinal variable with 12 categories ranging from "*Some primary school"* = 1 to "*Doctoral or professional degree"* = 12, in line with prior publications from MIDUS (Fuller-Rower, Curtis, Chae, & Ryff, 2018). When parents had different education levels, the highest education level between parents was used. Health information included: binary coded exercise habits (*engages in regular exercise* = 1), binary coded smoking status (*current smoker* = 1), and whether or not the participant was taking medication that may influence HF-HRV. Medication regimen was coded into two dummy variables representing whether or not the participant was currently taking (1) medications known or believed to increase HF-HRV (*parasympathomimetic agents, beta-blockers*), and (2) medications known or believed to decrease HF-HRV (*anticholinergic agents, anti-depressants, sedatives, anti-arrhythmic agents, cardiac drugs, antipsychotics*), a method previously used in research on HF-HRV with the MIDUS cohort (Sloan et al., 2017). Menopause status, collected during MIDUS 2, was dummy coded such that both females who had yet to go through menopause and males were assigned a "0", and females who had gone through menopause were assigned a "1", in line with previous MIDUS research on HF-HRV (Sloan et al., 2017). Health data (cardiovascular health, self-evaluated health) were also collected during MIDUS 2 and included as covariates in analyses predicting respective MIDUS 3 health outcomes.

Data Analysis

Bivariate correlations and regression analyses were calculated using IBM SPSS Statistics version 25, and mediation analyses were performed using structural equation modeling in Mplus version 6.12. To address Aim 1 examining the relation between childhood parental warmth and HF-HRV, a multiple linear regression was performed. Linear regression analysis was performed in 2 steps: first, resting HF-HRV was regressed on parental warmth, then demographic and health related covariates (age, sex, race, childhood SES, exercise, smoking, medication regimen, menopause status) were added to the model. HF-HRV data were positively skewed, so in accordance with standard methods (Malik, 1996), we performed a natural log transformation prior to analyses. Due to the well documented correlation between HF-HRV and heart rate (HR), we followed recent recommendations (de Geus, Gianaros, Brindle, Jennings, & Berntson, 2018) and calculated HF-HRV adjusted for concurrent HR (aHF-HRV), using the following equation: aHF-HRV = 100*(HF-HRV/$(IBI)^2$). Initial analyses were performed using unadjusted HF-HRV; post hoc sensitivity analyses were performed for all analyses using aHF-HRV. Positively skewed aHF-HRV data were log transformed prior to analyses. Participants excluded from regression analysis due to missing one or more variables were on average 6.3 years older, thus we adjusted for age statistically in all analyses. Participants with missing data did not significantly differ from those included in regards to any other variable. Sample size varies across analyses depending on data availability. In order to correct for multiple comparisons we utilized the Benjamini-Hochberg False Discovery Rate (FDR). With this method an estimate is significant if the p-value is smaller than the corresponding FDR q-value.

To address Aim 2, testing the relation between resting HF-HRV and health and mortality outcomes, four separate regression models were performed. The association between HF-HRV and cardiovascular health was investigated through two models: (1) a binary logistic regression predicting the dichotomous cardiovascular health problem variable, and (2) a generalized linear model with a Poisson distribution predicting the total number of cardiovascular health problems. A Poisson distribution allows for better modeling of the distribution of count data, which is characterized by the absence of values below zero and a positive skew (Atkins & Gallop, 2007). To test the association between HF-HRV and self-evaluated health, we performed a multiple linear regression with resting HF-HRV as a predictor of self-evaluated health. Finally, to test the association between HF-HRV and mortality we performed a binary logistic regression. All models, with the exception of the model predicting mortality, were run in three steps: (1) first without covariates, then (2) controlling for respective health measured during MIDUS 2, then (3) with demographic and health covariates (age, sex, race, childhood SES, exercise, smoking, medication regimen, menopause status) added to the model. The model predicting mortality was tested in two steps: (1) first without covariates, then (2) with demographic and health covariates.

To address Aim 3, testing HF-HRV as a mediator between childhood parental warmth and later health and mortality outcomes, we performed four mediation analyses using bias-corrected bootstrapping methods (Hayes, 2009), with 1000 samples, in Mplus software. With this method an indirect effect is significant if the 95% bootstrapped confidence interval does not span zero. In order to address missing data in our mediation analysis, Full Information Maximum Likelihood (FIML) was used. Mplus FIML employs Ecker-Huber-White estimation, which accounts for non-normality and the non-independence of data clustered within families, a method previously used with this data set (Donoho, Seeman, Sloan, & Crimmins, 2015; Wiley, Gruenewald, Karlamangla, & Seeman, 2016).

Results

Preliminary Analysis

Table 1 provides characteristics of participants included in the analysis. We highlight some of the significant correlations (for complete bivariate correlational results, please see Table 2; note that Spearman rank correlations were used for this correlation table given that some of our outcome variables were skewed). Parental warmth was positively correlated with HF-HRV ($r_s = .06$, $p = .04$), such that warmer parenting predicted higher HF-HRV. Higher HF-HRV was associated with better cardiovascular health and reduced mortality risk: HF-HRV was negatively correlated with having a cardiovascular health problem ($r_s = -.13$, $p < .001$), total number of cardiovascular health problems ($r_s = -.13$, $p < .001$), and mortality ($r_s = -.09$, $p = .002$). Cardiovascular health and self-evaluated health were moderately correlated ($r_s = -.32$, $p < .001$), suggesting that these two measures represent different aspects of physical health outcomes. Mortality was negatively correlated with self-evaluated health ($r_s = -.08$, $p = .02$).

The Relation Between Childhood Parental Warmth and HF-HRV

Results from a multiple linear regression revealed a significant positive relation between childhood parental warmth and HF-HRV at the univariate level, β = .06, p = .04. Results from the final model, presented in Table 3, which included covariates, also revealed a significant positive association between childhood parental warmth and HF-HRV, β = .07, p = .01 (*FDR* q = .025). Raw data are presented in Supplemental Figure S1.

HF-HRV and Cardiovascular Health

To test the relation between HF-HRV and cardiovascular health we conducted two models. First, using a dichotomous dependent variable representing presence or absence of any cardiovascular health problems, we conducted a binary logistic regression. Results revealed a significant association between HF-HRV and cardiovascular health both at the univariate level, B = −.24, SE = .06, p < .001, OR = .79, 95% CI [0.71, 0.89], and after including demographic covariates, B = −.14, p = .02, OR = .87, 95% CI [0.77, 0.98]. The final model, including covariates related to HF -HRV, again revealed a significant association between HF-HRV and cardiovascular health, B = −.17, SE = .06, p = .007 (*FDR* q = .0125), OR = .84, 95% CI [0.74, 0.95], such that greater HF-HRV predicted lower odds of having a cardiovascular health problem at mean age 63. Raw data for this association are presented in Supplemental Figure S2. In order to test the robustness of this relation we also conducted a generalized linear model (GLM) to see if HF-HRV could predict total number of cardiovascular health problems. Results from this model again revealed a significant association between HF-HRV and total number of cardiovascular health problems (please see Table 4 for GLM results).

HF-HRV and General Self-Evaluated Health in Midlife

Results from this model revealed no significant association between HF-HRV and self-evaluated health. This outcome was consistent at the univariate level, β = .03, p = .31, after controlling for previous self-evaluated health, β = .04, p = .27, and in the final model with the addition of covariates, β = .05, p = .18 (*FDR* q = .05).

HF-HRV and Mortality

Results from a binary logistic regression revealed a significant association between HF-HRV and mortality at the univariate level, B = −.36, SE = .11, p = .001, OR = .70, 95% CI [0.57, 0.86]. The final model, presented in Table 5, which included all covariates, also revealed a significant association between HF-HRV and mortality, B = −.23, SE = .11, p = .035 (*FDR* q = .037), OR = .79, 95% CI [0.64, 0.99], such that greater HF-HRV predicted decreased odds of mortality.

Testing the Mediation Models

Mediation analysis revealed a significant indirect effect of childhood parental warmth, through HF-HRV, on cardiovascular health, B = −.02, SE = .01, 95% CI [−.043, −.003], presented in Figure 1. Results from our robustness check, using sum of cardiovascular problems, again revealed a significant indirect effect of parental warmth, through HF-HRV, on sum of cardiovascular health problems, B = −.01, SE = .005, 95% CI [−.022, −.002].

Results from the mediation model predicting mortality revealed that the indirect effect of parental warmth, through HF-HRV, on mortality was not significant, $B = -.002$, 95% CI [−.01, .00]. The indirect effect of parental warmth, through HF-HRV, on self-evaluated health was also not significant, $B = .11$, 95% CI [−.002, .01].

Post-Hoc Sensitivity Analysis

Adjusted HF-HRV.—Using HF-HRV adjusted for concurrent HR (aHF-HRV), controlling for the same covariates in the initial models, results were unchanged: parental warmth was positively associated with aHF-HRV ($B = .16$, $p = .014$); higher aHF-HRV was associated with better cardiovascular health, using both a dichotomous variable ($B = -.17$, $p = .006$), and a count variable ($B = -.09$, $p = .004$); aHF-HRV predicted reduced odds of mortality ($B = -.14$, $p = .014$); and aHF-HRV did not significantly predict self evaluated health ($B = .02$, $p = .48$). In addition, mediation results were unchanged, with aHF-HRV only significantly mediating the relation between parental warmth and cardiovascular health.

Moderation by Age.—Due to the large age range of this sample, and the retrospective methods used for measuring childhood parental warmth, a sensitivity analysis was conducted to test age as a moderator of the relation between parental warmth and resting HF-HRV. Without covariates the interaction term between age and parental warmth was significant, $B = -.01$, $SE = .005$, $p = .049$. A Johnson-Neyman region of significance test revealed that the relation between parental warmth and HF-HRV was only significant for individuals younger than 59 years old. After including covariates, the interaction term was no longer significant, $B = -.007$, $p = .17$.

Discussion

Parenting that is affectionate, consistent, and sensitive, or warm parenting, has often been associated with better physical health outcomes for offspring (Miller et al., 2011; Newland, 2015). A comprehensive understanding of the mechanisms involved in this association, including biological mechanisms, is still needed. The present study set out to investigate the associations between childhood parental warmth, resting high-frequency heart rate variability (HF-HRV), and health and mortality outcomes. Furthermore, we aimed to test whether ANS physiology may mediate the relation between parental warmth and health indices in older adulthood. Findings from the current study suggest that (1) retrospective measures of childhood parental warmth can predict resting HF-HRV in midlife, (2) resting HF-HRV can predict cardiovascular health over a nine year period, controlling for baseline cardiovascular health, and can also predict mortality, and (3) resting HF-HRV may act as a mediator between childhood parental warmth and health in older adulthood, though the indirect influence of parental warmth through HF-HRV on health may depend on the specific physical health outcomes measured.

Consistent with our first hypothesis, we found that individuals who reported greater parental warmth during childhood exhibited higher HF-HRV during rest. This is consistent with previous research on parental warmth and HRV with infants and adolescents (Graham et al., 2017; Porter, 2003). In general, these results shed light on the potential influence of parenting on ANS physiology (Propper & Moore, 2006). Warm parenting may influence the

offspring's lifelong autonomic physiology by providing stable, predictable environments that keep their physiological reactivity within certain set points (Flannery et al., 2017). This has important implications, as HF-HRV has been associated with several significant health outcomes (Thayer & Lane, 2007).

The effect size was relatively small within our sample, compared to previous research in infants and adolescents (Graham et al., 2017; Porter, 2003). This may be attributed to a weakening association across development, as additional variables (e.g., health behavior, age) continue to influence ANS physiology into midlife. Alternatively, given the large sample used in the current study, this may reflect a true small effect. Nevertheless, a small effect of parental warmth on lifelong offspring physiology may have significant consequences at the population level and the individual level, particularly if it co-varies with early-life adversities, such as poverty. Results from our unadjusted model examining moderation by age provide some evidence that parental warmth may exhibit reduced influence on HF-HRV with age, though these results should be interpreted with caution as they were post-hoc and did not hold up to the inclusion of covariates. Alternatively, moderation by age results may reflect increasing memory bias with age, such that older individuals' retrospective reports may have had more measurement error. Further research using longitudinal designs and multiple measures of retrospective accounts of childhood experiences could help clarify these moderation results.

For the second aim of this study, we hypothesized that higher resting HF-HRV would predict better health outcomes, indexed through participants' reports of physician diagnosed cardiovascular health problems and self-evaluated health. Our findings were generally supportive of this hypothesis, and the effect was specific for cardiovascular health rather than a general effect on overall self-evaluated health. Specifically, we found that higher resting HF-HRV was associated with better cardiovascular health, indexed through (1) absence or presence of any diagnosed cardiovascular disorders, and (2) total number of cardiovascular health disorders. This association remained significant after statistically controlling for prior cardiovascular health, indicating that higher resting HF-HRV may protect against worsening in cardiovascular health over a 9-year period. These findings add to the large body of evidence suggesting an association between low resting HF-HRV and cardiovascular disease. While low resting HF-HRV has been associated with other health diagnoses, such as diabetes (Carnethon, Golden, Folsom, Haskell, & Liao, 2003), the majority of past associations between low resting HF-HRV and physical health have focused on cardiovascular health, including: hypertension (Singh et al., 1998), high cholesterol (Christensen, Toft, Christensen, & Schmidt, 1999), and coronary heart disease (Liao, Carnethon, Evans, Cascio, & Heiss, 2002). It has been proposed that vagal activity has beneficial effects for cardiovascular health through multiple pathways, including a vagal anti-inflammatory pathway (Sloan et al., 2007; Tracey, 2007) that may reduce risk of atherosclerosis (Libby, 2002). Furthermore, low HF-HRV may also lead to poor health through its association with poor psychosocial functioning (Appelhans & Luecken, 2006; Kemp & Quintana, 2013).

The large and nationally representative sample of adults and the longitudinal design of the MIDUS study provided a unique opportunity for also investigating the relation between HF-

HRV and mortality. Previous research on HRV and mortality suggests that higher resting HF-HRV predicts reduced all-cause mortality risk (Thayer et al., 2010); the results were consistent with these past findings. While the association between HRV and cardiovascular disease may partially explain these findings, the all-cause mortality data included causes of death not directly related to cardiovascular health, such as cancer. However, considering the previously mentioned associations between low resting HRV and both (1) inflammatory processes (Tracey, 2007) and (2) poor psychosocial functioning (Kemp & Quintana, 2013), it is possible that the mechanisms underlying the relation between low resting HF-HRV and cardiovascular disease are similar to those that drive the association between low resting HF-HRV and all-cause mortality.

The absence of a significant association between HF-HRV and global self-evaluated health suggests that HF-HRV may be more strongly related to specific health conditions, such as cardiovascular disease. It is possible that this global measure aggregates across many different aspects of health (e.g., mental health, physical limitations) that have weaker connections to high-frequency heart rate variability.

For the third aim of this study, we hypothesized that resting HF-HRV would mediate the relation between childhood parental warmth and health outcomes. Our findings were generally supportive of this, and the effect was specific for cardiovascular health rather than a general effect on overall self-reported health. Specifically, we found that childhood parental warmth was indirectly associated with better cardiovascular health, indexed through both absence of any diagnosed cardiovascular health disorders and fewer cardiovascular health disorders, and that this indirect association was explained through (1) the association between high childhood parental warmth and high resting HF-HRV, and (2) the association between high resting HF-HRV and better cardiovascular health. These results are consistent with the model proposed by Repetti et al. (2002) whereby early-life family-environmental characteristics are theorized to affect offspring life-long health outcomes through their influence on offspring physiology. However, considering the weak indirect effect it is not likely that HF-HRV fully mediates the relation between parental warmth and cardiovascular health, and instead may work in addition to, or in an interaction with, other biological mediators (e.g., inflammation, the HPA axis).

While we did not observe a direct, bivariate relation between childhood parental warmth and cardiovascular health among this sample, contemporary mediation analysis no longer requires a direct association to be significant as a prerequisite to mediation analysis (MacKinnon, Krull, & Lockwood, 2000; Shrout & Bolger, 2002; Zhau, Lynch, & Chen, 2010). Using simulation models, quantitative researchers have shown that mediation can occur even when an initial direct effect is not found (Hayes, 2009; Rucker, Preacher, Tormala, & Petty, 2011). Such a phenomenon has been explained through (1) suppression, whereby unmeasured mediators express an indirect effect in the opposite direction, or (2) unbalanced statistical power, such as when the mediator has less measurement error than the independent or dependent variables, and therefore significant relations between either the independent or dependent variable and the mediator are easier to find than a significant relation between the independent and dependent variables (Rucker et al., 2011). Considering the expansive literature on the association between warmer parenting and better health

(Chen, Brody, & Miller, 2017), suppression is an unlikely explanation of our results. Instead, for the current study, the greater precision in the measurement of the biological mediator relative to the self-report methods employed for both the independent and dependent variables may have contributed to unbalanced statistical power, which allowed us to detect significant associations between parental warmth and HF-HRV, as well as HF-HRV and cardiovascular health outcomes, but not between self-reported parental warmth and cardiovascular health outcomes.

Limitations and Future Directions

While the current study addressed an important gap in the literature and has a number of strengths, there are some limitations that should be considered for future research. The first limitation is the use of a retrospective measure of parental warmth, which may be subject to memory bias (Hardt & Rutter, 2004). However, due to the logistical constraints associated with longitudinal designs that span decades from childhood into middle/older age when chronic diseases begin to manifest, retrospective measurements are frequently utilized for investigating the long-term outcomes associated with parenting characteristics (Rothrauff, Cooney, & An, 2009; Wright, Turner, & McCarty, 2017). Stability of retrospective reports over time (Silva & Maia, 2013) and modest but significant associations between observational and later retrospective measures of parenting (Newbury et al., 2018) provide some support for the validity of retrospective methods. In addition, the limitations associated with retrospective measures may be particularly important to consider within the current study due to the large age range, considering memory bias may increase with age. Concerns over this limitation may be partially alleviated by the inclusion of age as a covariate in our analyses.

Lastly, future research could benefit from employing an experimental design, for instance, by randomly assigning parents to receive parental warmth training; and from measuring HF-HRV at more than one time point, in order to directly observe change. The correlational design utilized in the current study limits our ability to interpret causality or direction of effects; it is possible that offspring autonomic physiology may influence parental warmth, as some have suggested (Hastings, Grady, & Barrieau, 2018; Kennedy, Rubin, Hastings, & Maisel, 2004), or that the relation is bidirectional. It is also possible that the association between childhood parental warmth and HF-HRV may be in part explained by shared genetics related to autonomic physiology. For example, it is possible that parents who themselves have high resting HF-HRV may be more likely to engage in warmer parenting, and pass on a genetic predisposition to high resting HF-HRV to their offspring. However, previous research has shown both intraindividual change in HRV across early years of development (Bornstein & Suess, 2000), and discordant HRV among monozygotic twin pairs (Healy, 1992), suggesting substantial environmental contributions to HRV that need to be clarified.

Despite these limitations, the current study adds to the limited literature on the relation between parental warmth and ANS physiology, and is the first to investigate ANS physiology as a mediator between parental warmth, and health and mortality. Among a nationally representative sample of adults, we found that retrospective measures of

childhood parental warmth were positively associated with resting HF-HRV, and that higher resting HF-HRV predicted better cardiovascular health and decreased all-cause mortality risk. We also found that HF-HRV may partially mediate the relation between parental warmth and health, and that this relation may be specific to cardiovascular health. A better understanding of the biological mechanisms behind the association between family-environmental factors and health contributes important evidence of potentially lasting beneficial implications of warm parenting, and provides unique opportunities for intervention, such as biofeedback training.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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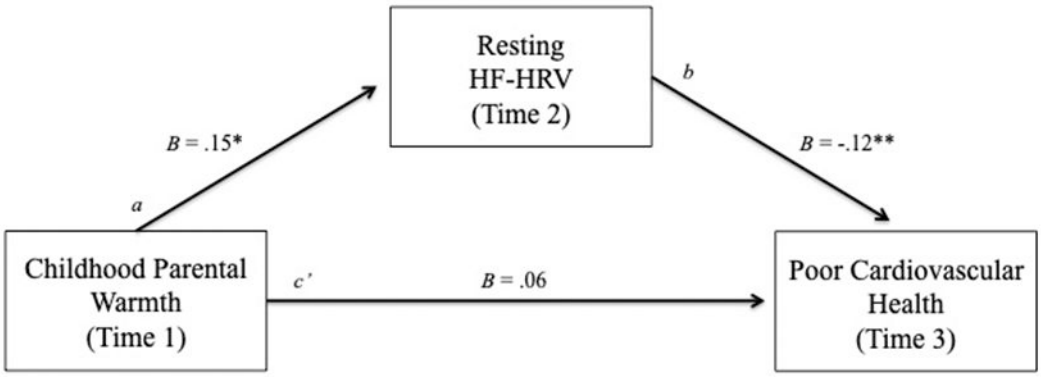

Figure 1.

Model of resting high-frequency heart rate variability (HF-HRV) as a mediator between parental warmth and presence of a cardiovascular health disorder.

*$p < .05$. **$p < .01$.

Total effect (c): $B = .04, SE = .08, p = .58$

Indirect effect($a*b$): $B = -.02$, $SE = .01$, 95% Bootstrapped CI [−.043, −.003]

Table 1.

Major Variable Descriptives and Sample Characteristics

Variable	*N*	*M*	*SD*	Range
HF-HRV	1,148	4.92	1.28	0.90–9.66
Heart Rate	1,148	72.84	10.85	44.4–109.8
aHF-HRV	1,148	0.97	1.45	−3.84–5.95
Parental Warmth	1,223	2.92	0.66	0.96–3.96
Number of CV Problems	938	0.79	0.90	0–5
Number of CV Problems (MIDUS 2)	1,227	0.79	0.97	0–6
Self-Evaluated Health[a]	943	2.54	1.02	0–4
Self-Evaluated Health[b] (MIDUS 2)	1,010	7.84	3.08	0–10
Age (years)	1,255	57.32	11.55	35–86
Childhood SES[c]	1,217	5.82	2.86	1–12
	N	**Level**	**Frequency**	**Percent**
Cardiovascular Problems	938	At least one	524	55.9
		None	414	44.1
Cardiovascular Problems (MIDUS 2)	1,244	At least one	647	52.0
		None	597	48.0
Deceased	1,255	Yes	84	6.7
		No	1171	93.3
Sex	1,255	Female	713	56.8
		Male	542	43.2
Race	1,251	Caucasian	985	78.5
		African American	215	17.2
		Native American	17	1.4
		Asian	3	0.2
		Other	31	2.5
Engages in Regular Exercise	1,255	Yes	960	76.5
Current Smoker	1,255	Yes	187	14.9
Medication that Increases HF-HRV	1,255	Yes	186	14.8
Medication that Decreases HF-HRV	1,255	Yes	195	15.5
Menopause Status	1,255	Post	248	19.8

Note. HF-HRV = high-frequency heart rate variability. aHF-HRV = HF-HRV adjusted for heart rate. CV = cardiovascular.

[a]Self-evaluated Health during MIDUS 3 ranged from: 0 = *poor* to 4 = *excellent*.

[b]Self-Evaluated Health during MIDUS 2 ranged from: 0 = *worst* to 10 = *best*.

[c]Childhood SES was indexed through highest parental education level, and ranged from: 1 = no school/some grade school to 12 = doctoral degree.

Table 2.

Bivariate Correlations Between Study Variables and Covariates

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. HF-HRV	.06*	−.13**	−.13**	.02	−.09*	−.28**	.07*	.18**	.00	.00	.14**	−.01	−.09**	−.04	−.06*	−.06*	.00	−.47**	.98**
2. Parental Warmth	–	.04	.01	.11**	.01	.04	−.11**	.16**	−.01	.04	−.01	.02	−.06*	−.03	.02	.03	.14**	−.07*	.07*
3. CV Problems (1 or more)		–	.93**	−.32**	.05	.28**	−.04	.04	−.08*	−.08*	.00	.31**	.05	−.01	.56**	.57**	−.20**	−.01	−.10**
4. Number of CV Problems			–	−.35**	.06	.28**	−.03	.04	−.11**	−.12**	.02	.36**	.08*	−.01	.56**	.60**	−.23**	−.04	−.09**
5. Self-Evaluated Health				–	−.08*	.01	.00	−.13**	.11**	.15**	−.15**	−.16**	−.13**	.07*	−.22**	−.25**	.40**	.00	.17
6. Deceased					–	.23**	−.05	−.04	−.09*	−.05	.00	.10**	.11**	−.01	.16**	.18**	−.06	.04	−.09**
7. Age						–	−.03	−.16**	−.19**	−.03	−.17**	.23**	.07*	.26**	.29**	.33**	.07*	−.14**	−.21**
8. Sex (female)							–	.08**	.02	−.03	−.04	−.02	.11**	.43**	.05	.05	.04	.17**	.03
9. Race (non-Caucasian)								–	−.19**	−.15**	.18**	−.01	−.05	−.01	.09**	.09**	−.04	.04	.15**
10. Childhood SES									–	.11**	−.11**	−.08**	−.02	−.01	−.11**	−.14**	.07*	.04	.00
11. Exercise										–	−.07**	−.11**	−.07**	−.02	−.10**	−.12**	.02	−.05	.00
12. Smoking											–	−.04	.02	−.06*	−.03	−.02	−.11**	−.03	.13**
13. Medications that Increase HF-HRV												–	.07*	.00	.35**	.39**	−.08	−.26**	.06*
14. Medications that Decrease HF-HRV													–	−.01	.13**	.15**	−.08**	.01	−.08**
15. Menopause Status (post)														–	.03	.03	.02	.03	−.04
16. CV Problem (MIDUS 2)															–	.94**	−.16**	−.02	−.04
17. Number of CV Problems (MIDUS 2)																–	−.18**	−.06	−.04
18. Self-Evaluated Health (MIDUS 2)																	–	−.02	.00
19. Heart Rate																		–	−.65**
20. aHF-HRV																			–

Note. HF-HRV = high frequency-heart rate variability. CV = cardiovascular. aHF-HRV = HF-HRV adjusted for heart rate. Correlations calculated using Spearman's rho.

*$p < .05$

** $p < .01$.

Table 3.

Multiple Linear Regression Predicting Resting HF-HRV

	Step 1			Step 2		
Predictor	***B***	***SE***	**β**	***B***	***SE***	**β**
Constant	4.53	.18		6.04	.30	
Parental Warmth	.12	.06	.06*	.14	.06	.07*
Age				−.03	.004	−.27**
Sex (female)				.17	.08	.06*
Race (non-Caucasian)				.34	.10	.11**
Childhood SES				−.01	.01	−.02
Exercise				.04	.09	.01
Smoking				.25	.11	.07*
Medication that Increases HF-HRV				.25	.11	.07*
Medication that Decreases HF-HRV				−.28	.10	−.08**
Menopause Status				.01	.11	.002

Note. HF-HRV = high-frequency heart rate variability.

* $p < .05$

** $p < .01$.

Table 4.

Generalized Linear Model Predicting Total Sum of Cardiovascular Health Problems

	Step 1			Step 2			Step 3		
Predictor	***B***	***SE***	**95% CI**	***B***	***SE***	**95% CI**	***B***	***SE***	**95% CI**
Constant	0.28	.16		−0.28	.17		0.28	.43	
HF-HRV	−.12**	.03	[−.19, −.06]	−.10**	.03	[−.17, −.04]	−.10**	.04	[−.17, −.03]
CV Problems (MIDUS 2)				.52**	.03	[.45, .59]	.44**	.04	[.36, .52]
Age							.01	.004	[−.002, .01]
Sex (female)							.08	.09	[−.11, .26]
Race (non-Caucasian)							−.27	.14	[−.55, .01]
Childhood SES							−.01	.02	[−.04, .02]
Exercise							.10	.10	[−.09, .29]
Smoking							−.20	.12	[−.45, .04]
Medication that Increases HF-HRV							−.45**	.10	[−.65, −.25]
Medication that Decreases HF-HRV							−.05	.11	[−.27, .16]
Menopause Status							−.02	.12	[−.25, .21]

Note. HF-HRV = high-frequency heart rate variability. CV = cardiovascular. 95% CI = [lower limit, upper limit] of 95% Wald confidence interval of *B*.

* $p < .05$

** $p < .01$.

Table 5.

Binary Logistic Regression Predicting Mortality by the MIDUS 3 assessment

	Step 1			Step 2		
Predictor	***B***	***SE***	***OR***	***B***	***SE***	***OR***
Constant	−1.20	.48		−6.36	1.24	
HF-HRV	−.36**	.11	.70	−.23*	.11	.79
Age				.08**	.01	1.08
Sex (female)				−.53	.34	.59
Race (non-Caucasian)				.44	.38	1.55
Childhood SES				−.04	.06	.96
Exercise				−.11	.32	.90
Smoking				.54	.41	1.71
Medication that Increases HF-HRV				.43	.33	1.54
Medication that Decreases HF-HRV				1.06**	.32	2.88
Menopause Status				.05	.43	1.05

Note. HF-HRV = high-frequency heart rate variability. *OR* = odds ratio.

* $p < .05$

** $p < .01$.