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The relationship between maternal responsivity, socioeconomic status, and resting autonomic nervous system functioning in Mexican American children

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ABSTRACT

Adversity, such as living in poor socioeconomic conditions during early childhood, can become embedded in children's physiology and deleteriously affect their health later in life. On the other hand, maternal responsivity may have adaptive effects on physiology during early childhood development. The current study tested both the additive and interactive effects of socioeconomic status (SES) and maternal responsivity measured at 1 year of age on resting autonomic nervous system (ANS) function and trajectory during the first 5 years of life. Participants came from a birth cohort comprised of Mexican-origin families living in California. Children's resting ANS functioning (respiratory sinus arrhythmia; RSA; pre-ejection period; PEP; and heart rate; HR) was collected at 1, 3.5, and 5 years of age (N = 336) and modeled across time using Hierarchical Linear Modeling. Consistent with hypotheses, results showed that low SES predicted flatter trajectories of resting HR and PEP over early childhood (i.e., patterns of consistently higher heart rate; shorter PEP), whereas children who experienced positive maternal responsivity had steeper trajectories in RSA and PEP over time (i.e., increasing parasympathetic activation; decreasing sympathetic activation). The interaction between SES and maternal responsivity significantly predicted RSA intercept at age 5, such that among children living in low SES environments, high maternal responsivity mitigated the negative effect of poverty and predicted higher resting RSA at 5 years of age. Results are consistent with the early life programming theory that suggests that environmental influences become biologically embedded in the physiology of children living in socially disadvantaged contexts, and identify increased maternal responsivity as a developmental mechanism that could offset the deleterious effects of low SES.

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1. Introduction

Adversity experienced during sensitive periods of development may "get under the skin" and become embedded in children's physiology and deleteriously affect health outcomes (Boyce and Ellis, 2005; Shonkoff et al., 2009). Poverty experienced in early childhood has been linked to cardiovascular disease, hypertension, impaired immune functioning, as well as mental health disorders such as anxiety, depression, and schizophrenia (Alkon et al., 2012a). One way adversity may impact biological mechanisms affecting health outcomes is by recalibrating the stress response system (Del Giudice et al., 2011). While various biological systems are implicated in the stress response system, the autonomic nervous system (ANS) in particular has received attention because it indexes the calibration of the stress response system to early environmental influences and may represent an etiological pathway to poor cardiac and mental health outcomes (Van den Bergh, 2011; Enlow et al., 2014).

Research has largely focused on the detrimental health effects of environmental influences, such as poverty, on health and ANS functioning (e.g., Waters et al., 2016; Alkon et al., 2012b). However, scarce research has focused on protective factors that could mitigate the effects of poverty. Maternal responsivity is one such protective factor, which has been linked with the development of physiological homeostasis in children (Shonkoff and Garner, 2012; Spangler and Grossmann, 1993; Spangler et al., 1994). Research that identifies modifiable protective factors could help shape treatment strategies for young children and families living in conditions of poverty.

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Biological processes, such as cardiac function assessed via ANS activation, have the potential to yield new information about the way stress associated with poverty becomes engrained in one's physiology. The parasympathetic and sympathetic branches of the ANS flexibly coordinate (via co-active, co-inhibited, reciprocal, or uncoupled patterns of activation (Berntson et al., 1991)) to influence stress responding, cardiac functioning, and dually innervate overall heart rate (Mezzacappa et al., 1997). Given that both branches of the ANS influence heart rate, it is useful to examine more specific indices of parasympathetic and sympathetic activation as well. First, there is vagal tone, a marker of parasympathetic influence on the heart that is non-invasively measured via respiratory sinus arrhythmia (RSA). The parasympathetic branch is responsible for maintaining homeostasis, and serves to calm an individual and decrease heart rate after the experience of an acute stressor. Higher resting levels of RSA are thought to be adaptive and have been positively associated with social interaction, attention, emotional functioning, and behavioral control (Porges, 2007; Eisenberg et al., 1995; Calkins and Keane, 2004; Denver et al., 2007). The second index of specific cardiac function, pre-ejection period (PEP), assesses the influence of the sympathetic nervous system, also known as the fight-or-flight system. The sympathetic branch of the ANS is charged with mounting a response to stress or challenge in the environment, and leads to increases in (and less variability in) heart rate. Activation of the sympathetic branch increases readiness to respond to threat (Alkon et al., 2012a). PEP is a time-based measure of contractility of the heart (milliseconds), with shorter PEP intervals indicating greater sympathetic activation (Bauer et al., 2002; Sherwood, 1995). Thus, studying the patterns of activation of both the parasympathetic and sympathetic branches of the ANS across early life can yield much more precise information regarding the physiological mechanisms by which children's early experiences become biologically embedded. Despite this, most studies of infant and child ANS functioning have focused solely on the parasympathetic branch, given theories surrounding its involvement in early life attention, affect regulation, coping, and communication (e.g., Porges, 1995; Moore et al., 2009).

An extensive literature documents an association between early adversity and subsequent physiological reactivity (see for review Obradovic, 2012; McLaughlin et al., 2013; Blair et al., 2013; Propper and Holochwost, 2013; Sturge-Apple et al., 2016) which suggests that physiological functioning can serve as an indicator of prior exposure to adversity and canalization of stress physiology through experience (Blair and Raver, 2012). Such experiential canalization has implications for subsequent physiological responsivity or adaptability to context (Boyce and Ellis, 2005). While the majority of this research has focused on reactivity as an indicator of early life adversity, it also could be that resting set points are affected by early adversity. Research illuminates the far-reaching implications of early childhood poverty on subsequent child development, regardless of whether there is later upward mobility, and suggests that childhood poverty is linked with increased sympathetic activity indicating increased stress on heart function (Evans and Kim, 2013). However, one longitudinal examination conducted with the present study sample examined the effects of prenatal socioeconomic status on postnatal ANS reactivity and found that socioeconomic adversity marginally ($p = 0.05$) predicted PEP reactivity from 6 months to 5 years of age (Alkon et al., 2014), such that socioeconomic adversity predicted blunted PEP reactivity across time. These results indicate that even assessed prenatally, socioeconomic adversity can affect subsequent ANS trajectories of development. While that examination focused specifically on ANS reactivity, it is likely that socioeconomic adversity also affected children's resting ANS functioning, which could help contextualize the effects on ANS reactivity (e.g., resting ANS levels could reduce the bandwidth available for reactivity, under the law of initial values (Kagan et al., 1987)).

There is reason to focus specifically on the influence of adversities such as low SES on ANS development during the first five years of life. These early years have been identified as a period during which stress physiology circuits can be permanently programmed depending on environmental influences (Van den Bergh, 2011; Lupien et al., 2009). Research demonstrates that during the last trimester of pregnancy and first year of an infant's life, the ANS rapidly changes (Porges and Furman, 2011; Beauchaine, 2001), indicating a time of increased plasticity along the trajectory of development. Given this increased plasticity, both positive and negative environmental influences exerted during this early time frame may become biologically embedded into the physiology of a child and alter trajectory of subsequent ANS development (Del Giudice et al., 2011; Boyce and Ellis, 2005). Low SES represents a cue of unpredictability in the environment which can shift psychobiological set points to become more sensitized to environment (Del Giudice et al., 2011). Such biological embedding can serve to recalibrate physiological set resting or basal activity within an individual as the result of conditional adaptation to environment, or the evolved ability of an organism to modify its developmental trajectory to match the social and physical environment. This recalibration is a part of allostasis, or the process of achieving stability through physiological or behavioral change (McEwen, 1998), which can have subsequent implications for how an organism interfaces with the environment in the future, mediating the individual's adjustment to both positive and negative events (Del Giudice et al., 2011; Boyce and Ellis, 2005).

Positive environmental influences experienced during early childhood also may also alter ANS functioning and subsequent developmental trajectories during this time of plasticity. Both longitudinal and cross-sectional research show that maternal caregiving plays a pivotal role in programming early stress response systems, with more emotionally supportive parenting linked to healthier stress systems (Hostinar et al., 2014). Some of these studies have focused on Hypothalamic-Pituitary-Adrenal (HPA) functioning, a hormonal index of stress functioning (e.g., Moore et al., 2009; Hostinar et al., 2014; Grant et al., 2010), whereas others have investigated the influence of positive factors on ANS functioning.

Many studies have focused specifically on parasympathetic activation in association with maternal behavior, due to its link to emotion and coping (e.g., polyvagal theory (Porges, 1995; Hastings et al., 2008)). Two studies that indexed resting parasympathetic activation found that higher maternal responsivity was a significant predictor of higher resting parasympathetic activation (a more adaptive profile (Kaplan et al., 2008)) and parental conflict was associated with reduced parasympathetic activation (a less adaptive profile (Moore, 2010)). Longitudinal studies of vagal tone have also revealed important associations across childhood. Calkins and Keane (2004) documented extensive stability in children's resting and reactive RSA from 2 to 4.5 years of age, and a second study using data from the present sample showed moderate stability in RSA from six to 60 months in Mexican American children (Alkon et al., 2011). A third large-scale longitudinal investigation examined vagal tone and maternal parenting practices when children were 2–4 years old (Kennedy et al., 2004). This study revealed stability in parasympathetic regulation and parenting, but also showed that less well-regulated child physiology and parenting practices interacted to predict maternal use of restrictive parenting practices across childhood, highlighting the importance of considering proximal effects of parenting in conjunction with physiological development.

Of the fewer studies that have focused on the link between maternal responsivity and profiles of ANS functioning (i.e., sympathetic and parasympathetic function), most have examined ANS reactivity rather than resting ANS. For example, one cross-sectional study found that insensitive caregiving was associated with increased sympathetic activation and less parasympathetic reactivity to the still-face paradigm in 6 month olds (Enlow et al., 2014). Another study examined the role of social support on the co-regulation between parasympathetic vagal reactivity and sympathetic reactivity to stressors in the lab in 4–5 year olds living in poverty (Wolff et al., 2012). This study found that parasympathetic reactivity predicted attenuated sympathetic reactivity, but only in socially supportive contexts, thus highlighting the

importance of social support for children living in poverty. Research is still needed to understand the effects of poverty and maternal responsivity on the development of resting parasympathetic and sympathetic function, especially in early childhood.

Much of the existing knowledge has been derived from predominantly European American samples, and no known research on the relationship between resting ANS functioning, poverty, and maternal sensitivity has been conducted among Mexican-origin children, or any other Latino ethnic group. Mexican American children may represent a particularly vulnerable population, as Latino youth in the U.S. experience poverty at disproportionate rates compared to children from other ethnic groups (Fund CsD, 2014; Brown and Patten, 2014). While the effects of stress appear to be additive in European American populations, such that more stressors predict greater influence over physiological functioning, research with other ethnic minority populations suggests interactive effects exist such that the cumulative stressors of living in low resource environments can cause an exponential effect on physiological functioning (Skinner et al., 2011). For example, prior research has shown that for African American youth residing in impoverished environments, lower or blunted physiological profiles may be the most advantageous so the individual is only responsive to the most extreme or intense stressors in the environment (Skinner et al., 2011). Thus, we may expect to see the effects of low SES and poor maternal responsivity to be compounded (interactive) for Mexican American youth. The addition of poor maternal responsivity with low SES may compound the stress load on physiological mechanisms causing maladaptive resting ANS states (parasympathetic withdrawal, sympathetic activation, and higher heart rate). On the other hand, recent theoretical models emphasize the importance of positive familial interactions to protect children from the effects of stressful environmental influences (Ellis et al., 2011; Shonkoff, 2010), and thus may predict adaptive ANS states (less parasympathetic withdrawal, less sympathetic activation, and lower heart rate) even in the face of low SES. While these interactive effects have been demonstrated in other ethnic minority samples (e.g., African American), the interactive effects of maternal sensitivity and SES have yet to be examined among Mexican origin youth. This research is of upmost importance to public health, as Mexican American children comprise the greatest number of Latino youth in the United States, and therefore, represent an important subpopulation of Latinos for whom poverty and other associated stressors are particularly salient.

The current study examined both the additive and interactive influences of socioeconomic status (SES) and maternal responsivity (measured at 1 year of age as a proxy for early childhood exposure) on ANS functioning at ages 1, 3.5, and 5 years among an impoverished Mexican American sample of children of primarily immigrant parents. We focused on resting ANS functioning, rather than reactivity to challenge tasks. We sought to provide clarity about whether and how SES and maternal responsivity relate to children's resting ANS functioning, which could have direct effects on responsivity to environmental input and subsequent responsivity based on the law of initial values (Kagan et al., 1987). It was hypothesized that low SES would predict a trajectory of higher resting HR, increasing sympathetic activation, and increasing parasympathetic withdrawal across time, with the most divergent effects on these indices by age 5, which is a profile consistent with increased stress on the heart at rest. In contrast, it was expected that maternal responsivity would predict an adaptive trajectory of ANS functioning including lower HR, less sympathetic activation, and more parasympathetic activation, again with the most exacerbated effects by age 5. Finally, it was predicted that there would be an interaction between SES and maternal responsivity, such that the impact of SES on ANS functioning would vary with different levels of maternal responsivity. We expected that low maternal responsivity would be related to a stronger relationship between SES and resting ANS functioning (e.g., high SES relates to better ANS functioning even in the face of low maternal responsivity), while high maternal responsivity will be related to a weakened relationship between SES and resting ANS functioning (e.g., high maternal responsivity will weaken the deleterious impact of low SES on ANS functioning).

2. Methods

Participants were drawn from an ongoing birth cohort study aimed at investigating the association of environmental exposures with children's health and development, called Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS). Participants are primarily Mexican-origin families living in an agricultural community in Monterey County, California. Detailed methods of the CHAMACOS cohort have been described previously (e.g., [omitted for blind review]). Briefly, 1800 pregnant women were screened for eligibility between October 1999 and 2000 at six prenatal clinics serving primarily farmworker families. Pregnant women were eligible if they were 1) 18 years of age or older, <20 weeks gestation, 2) Spanish- or English-speaking, 3) eligible for California's low-income health insurance program (Medi-Cal), 4) receiving prenatal care, and 5) planning to deliver at the county hospital. Of the 1130 eligible women, 601 women initially enrolled, 527 were followed through delivery and 418 of their infants were seen at 1 year of age (336 of whom completed the ANS protocol and had scorable data). Of these infants, 330 (136 had ANS data at 3.5 years) were followed up at 3.5 years of age and 319 (297 had ANS data at 5 years) were seen again at 5 years. This examination utilized data from participants who completed at least one wave of the ANS protocol. Although ANS data were also collected at 6 months of age, this wave of ANS data was not included in our analyses as there were issues of temporal order (our predictor variables were assessed at 1 year of age and after). Our final sample included 336 Mexican American children and their mothers. Analyses indicated no differences between children who were included versus excluded on pertinent study variables.

During the 1-year visit, a 7-min ANS protocol was administered in the office or in a mobile van/recreational vehicle. At the 3.5 and 5-year visit, children completed a 15-minute ANS protocol in the office. Each visit included a home visit, where trained observers completed the Home Observation for Measurement of the Environment (HOME; see measures below). The HOME was also highly correlated across time ($r = 0.34$, $p < 0.001$ comparing 1 and 5-year visits) indicating that using the 1 year wave data was a good proxy for level of maternal responsivity across early childhood.

2.1. Measures

2.1.1. Home observation for measurement of the environment (HOME)

The HOME was created as an objective measure to assess the quality and quantity of stimulation in the home environment within a natural context that is widely used in epidemiological research (Elardo et al., 1975). The Infant-Toddler version of the HOME consists of 45-item scale with response choices that are binary (Yes/No). Higher total HOME scores indicate a more enriched home environment. There is a vast literature linking HOME scores to children's cognitive development, attachment status, medical conditions, psychopathology (of both children and their parents), and parental substance abuse see (Totsika and Sylva, 2004) for review. The Emotional and Verbal Responsivity of Primary Caregiver subscale was used in analyses to measure maternal responsivity. This subscale is comprised of 11 items that are rated by observation of the dyad across the span of an hour in the home. Example items include: “Mother caresses or kisses child at least once during the visit” and “Mother spontaneously vocalizes to child at least twice during the visit.” This measure in particular has been linked to infant attachment status, with mothers rated high on this scale having more securely attached infants (Network NECCR, 2001).

2.1.2. Socioeconomic status (SES)

SES was assessed using a factor that was comprised of three variables assessed at age one. The first variable included in the SES factor was family income-poverty ratio, which was calculated by taking the family's reported monthly household income, divided by number of people supported by that income, and then dividing by the federal poverty threshold ascertained from U.S. Bureau of the Census, Current Population Survey at the time of collection (the year of age 1 data collection in our sample occurred in 2000). The second variable included in the SES factor was household crowding, which was calculated by using the number of people living in the home divided by the number of rooms in the home. Finally, the third variable included in the SES factor was a variable categorizing whether the family received food stamps/Supplemental Nutrition Assistance Program benefits (y/n). A number of other socioeconomic variables were considered for the factor (including father's highest education, mother's highest education, and language spoken at home) but a principal components analysis with varimax rotation indicated that only these three variables (poverty, crowding, food stamps) held together. The factor accounted for 41.57% of variance. Of note, the income-to-poverty ratio was highly correlated across the 1, 3.5, and 5-year visits ($r = 0.35$, $p < 0.001$; comparing the ratio at 1 year to 5 years of age). Thus the 1 year visit was used as a proxy for overall SES during early childhood.

2.1.3. Resting ANS

Children participated in a standardized protocol where continuous measures of impedance cardiography, electrocardiography (ECG), and respirations were measured. Band electrodes were used to collect the ANS data at 1 year of age (for details see [omitted for blind review]) and spot electrodes at 3.5 and 5 years (Alkon et al., 2003). The four impedance spot electrodes were placed on the neck (×2) and trunk (×2) and three electrodes were placed in a lead II configuration to collect ECG measures (Allen and Matthews, 1997). After the bands/spots electrodes were in place, the experimenter verified the quality of the ECG and impedance waveforms for the first 5 min of the protocol during which time no data were analyzed. Data were acquired at 1 year of age using the Minnesota Impedance Cardiograph HIC-2000 and at 3.5 and 5 years of age using the Biopac MP150, as the HIC-2000 was no longer available. Continuous measures of heart rate (HR), ECG, Z_o (basal impedance), and dZ/dt (first derivative of the impedance signal) were collected during the protocol. A 4-mA AC current at 100 Hz was passed through the two outer, current electrode bands/spots and Z_o and dZ/dt signals were acquired from the two inner voltage-recording bands/spots. Data were filtered, extracted, and then scored using the ANS suite software at 1 year (Alkon et al., 2003; Cacioppo et al., 1994) and MindWare Technologies LTD (Gahanna, Ohio) at 3.5 and 5 years of age. The sampling rate was 1000 Hz and the data were scored in 60-second intervals.

Data cleaning procedures involved examining for outliers (>3 SD) minute-by-minute and in summary scores (there were no outliers), and a child's data were deleted if >25% of the task minutes were unscorable and not included in present analyses ($n = 15$ in the current sample). The first 2 min of the ANS protocol at each time point included a resting measure. During the 1-year visit, children listened to a lullaby, and during the 3.5 and 5-year visits, children listened to a non-stressful story read aloud. The 2 min of the rest measure were averaged at each time point and used in longitudinal analyses for the current study.

Respiratory sinus arrhythmia (RSA), a measure of the parasympathetic nervous system, is the periodic oscillation in sinus rhythm occurring at the frequency of respiration and manifested as an increase in heart rate (HR) with inspiration and a decrease in HR during expiration. Respiratory sinus arrhythmia (RSA) was derived in accord with recommendations of the Society for Psychophysiological Research (SPR) committee on heart rate variability (Berntson et al., 1997) (SPR Committee Report) by the MindWare HRV analytical software package. The EKG signal was digitized (1000 Hz) and an interbeat interval (IBI) series was derived by a peak-identification algorithm that identifies the peak of the R wave as the fiducial point. Artifacts were flagged by statistical algorithms, including that of Berntson et al. (1990), and were then checked visually and edited as necessary according to the guidelines of the SPR Committee Report. The IBI series was then converted to a time series by resampling at fixed time intervals with interpolation (Berntson et al., 1995). The time series was linearly detrended to minimize nonstationaries in the data (Litvack et al., 1995). The residual series was then tapered with a Hamming window and submitted to the Fast Fourier Transform (FFT) module of LabVIEW (National Instruments, Austin, TX) to derive the spectral distribution. RSA was quantified as the natural log of the integral power within the respiratory frequency bandwidth (0.24 to 1.04 Hz at 1 year of age and 0.15 to 0.80 Hz at 3.5 and 5 years of age) (Bar-Haim et al., 2000; Bazhenova et al., 2001; Weiner and McGrath, 2016).

PEP, derived from impedance cardiography, is the period between the electrical invasion of the ventricular myocardium (Q wave of the ECG) and the opening of the aortic valve (B point). PEP was quantified as the time interval in milliseconds from the onset of the ECG Q wave to the B point of the dZ/dt wave (Sherwood et al., 1990). The B point was automatically derived by the MindWare analysis software by means of a validated algorithm that used the time interval of the Q point to the maximum point of the dZ/dt wave to estimate the location of the B point (Lozano et al., 2007). For each subject, ECG and impedance data were ensemble averaged for each minute to produce estimates of the PEP.

The interbeat interval (IBI) series was derived by a peak-identification algorithm that identifies the peak of the R wave as the fiducial point. Artifacts were flagged by statistical algorithms, including that of Berntson et al. (1990), and were then visually inspected and edited as necessary according to the guidelines of the SPR Committee Report and previously described methods (Berntson et al., 1990; Lozano et al., 2007). Trained research assistants visually inspected each minute of ECG data to identify the R peaks marked by the MindWare software algorithm as an outlier. If the research assistant determined that a R peak was coded incorrectly or not coded, the research assistant added or deleted a R peak mark.

2.2. Analyses

Main analyses were conducted using Hierarchical Linear Modeling (HLM v6.05) (Raudenbush, 2004) to account for the inherent nesting of the waves of ANS data collected within the individual (level-1) and across individuals (level-2). Hierarchical Linear Modeling (HLM) allows for the simultaneous modeling of ANS levels (intercept) and the changes in ANS functioning across time (slope). Each ANS indicator was modeled separately (i.e., HR, RSA, and PEP), and each model indexed the intercept and slope across three time points (age in months during assessment at wave 1, 3.5, and 5 years) as the outcome of interest (Y_{ANS}). At level-1, ANS intercept was centered at the participant's age in months during the third wave of data (approximately 5 years of age) because, theoretically, we hypothesized that the effects of low SES and maternal responsivity at 1 year would potentiate divergences between individuals on emerging ANS trajectories, resulting in the most striking individual differences at age 5. Thus, the best fitting model also included a variable estimating the slope across time as a function of age in months (β_{1Slope}) for each ANS index (HR, RSA, and PEP). Intercept was a fixed variable, whereas slope was specified to allow for random effects.

Once a level-1 or within-individual equation is established, level-1 predictors can become outcomes-of-interest at level-2. Cross-level interactions capture how individual difference factors impact level-1 associations, specifically ANS trajectories at rest and intercept centered at age 5. Analyses focused on SES and maternal responsivity at age 1 as individual difference predictors capable of impacting ANS. Below is an example of the HLM model.

Level-1 model

$$Y_{ANS} = \beta_0 + \beta_{1Slope}$$

Level-2 model

$$\beta_0 = \gamma_{00} + \gamma_{01}\ (\text{SES,Maternal Responsivity,SES} \times \text{Maternal Responsivity}) + U_0\ (\text{Residual variance and error term})$$

$$\beta_{1Slope} = \gamma_{10} + \gamma_{11}\ (\text{SES,Maternal Responsivity,SES} \times \text{Maternal Responsivity})$$

3. Results

The majority of the participants' mothers were born in Mexico (87.5%). Twenty-five percent of the sample reported receiving food stamps when their children were 1 year of age, and mean housing density was 1.64, SD = 0.74. Mean income to poverty ratio was 0.91 (SD = 0.49). To contextualize these results, the poverty line is at 1, and anything below 1 would be living below the poverty level. Thus, the average family in this sample was living at 9% below the poverty level at 1 year of age. As mentioned, maternal responsivity was highly correlated across time ($r = 0.34$, $p < 0.001$) indicating that using the 1 year wave data likely provided a good proxy for level of exposure across early childhood.

Preliminary analyses investigated the effects of child's sex and mother's country of birth as covariates on the level 1 ANS models. As shown in Table 1, these covariates did not significantly affect the parameters of the base model, and thus were not included in main analyses. Although mother's country of birth trended towards being related to PEP functioning at age 5, inclusion of this covariate in PEP analyses did not alter the pattern of results or model fit and was thus excluded for the sake of parsimony.

The level 1 model demonstrated moderate between-person variability for HR, RSA, and PEP (30%, 32%, and 43%, respectively, and also serves as estimates of interclass correlation) and substantial variability within person (70%, 68%, 57%, respectively). The base model for heart rate demonstrated that resting heart rate tended to decrease over time ($\beta = -0.83$, $p < 0.001$), whereas RSA increased ($\beta = 0.06$, $p < 0.001$) and PEP lengthened with age ($\beta = 0.25$, $p < 0.001$), which are the expected trends with normal development (Alkon et al., 2011). Table 2 lists the beta weights for each predictor of ANS functioning.

3.1. Main effects of SES

Partial support was found for hypotheses predicting lower SES associated with less adaptive ANS functioning across trajectories and intercept age 5. Low SES was associated with a trajectory of high and more slowly decreasing resting heart rate ($\beta = 0.06$, $p < 0.01$; Fig. 1), and a higher and flatter increase in resting PEP across early childhood ($\beta = -0.04$, $p < 0.05$), indicating that children with lower SES demonstrated less change in sympathetic activation at rest over the first few years of life. Low SES was also associated with shorter PEP ($\beta = -1.09$, $p < 0.05$) at age 5. However, SES was not significantly associated with heart rate at age 5, and SES did not significantly impact RSA trajectory from 1 to 5 years of age or intercept at age 5.

Table 1
HLM analyses of potential covariates on the ANS base models (N = 336).

Variables	B0 intercept	B1 slope
HR		
Child's sex	−2.23	0.01
Mother's birth country	2.17	0.05
RSA		
Child's sex	−0.10	−0.01
Mother's birth country	0.07	0.01
PEP		
Child's sex	−0.41	−0.03
Mother's birth country	−2.95+	−0.04

Note: +<0.10.

Table 2
Resting ANS intercept at age 5 and ANS trajectories from 1 to 5 years of age (N = 336).

Variables	B0-Intercept	B1-Slope
HR		
SES	0.77	0.06**
Responsivity	−2.53**	−0.04
SES × responsivity	−1.36+	--
RSA		
SES	−0.06	−0.01
Responsivity	0.11	0.01*
SES × responsivity	0.23**	--
PEP		
SES	−1.09*	−0.04*
Responsivity	0.49	0.06**
SES × responsivity	0.63	--

The interaction effect in prediction of slope was fixed in each model (–) if they were poor predictors of the model and affected model fit.
Note: –fixed, +<0.10.
* $p < 0.05$.
** $p < 0.01$.

3.2. Main (additive) effects of maternal responsivity

Partial support was found for hypotheses predicting higher maternal responsivity would be associated with more adaptive ANS functioning across trajectories and intercept at age 5. High maternal responsivity significantly predicted more steeply increasing trajectories of resting RSA levels, such that the higher the level of maternal responsivity, the steeper the RSA increase up to age 5 ($\beta = 0.01$, $p < 0.05$). Further, higher maternal responsivity predicted a steeper increase in resting PEP trajectory ($\beta = 0.06$, $p < 0.01$), indicating that children with more responsive mothers demonstrated less sympathetic activation at rest across the first years of life (Fig. 2). Maternal responsivity did not relate to heart rate trajectory, but was associated with a lower resting heart rate at age 5 ($\beta = -2.53$, $p < 0.01$). No association was found between maternal responsivity and PEP or RSA intercept at age 5.

3.3. Interactive effects

It was predicted that maternal responsivity and SES would interact to predict the relationship between SES and ANS functioning. Results

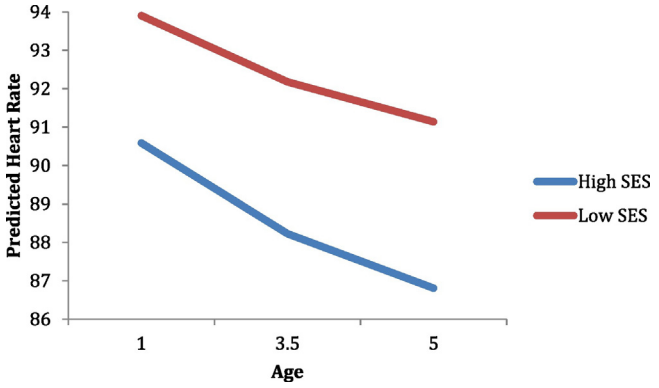

Fig. 1. SES predicting children's resting heart rate trajectory from 1 to 5 years of age ($\beta = 0.06$, $p < 0.01$).

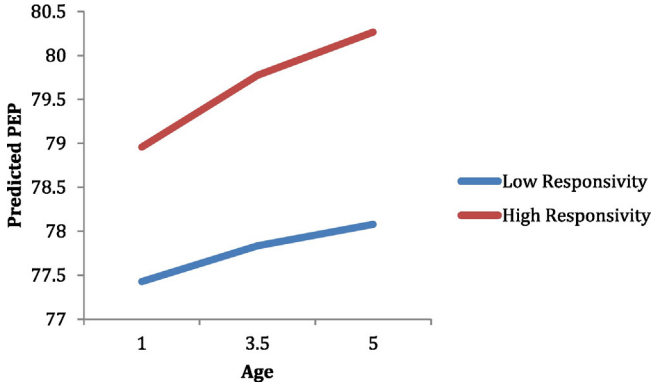

Fig. 2. Maternal responsivity predicting children's resting PEP trajectory from 1 to 5 years of age ($\beta = 0.06$, $p < 0.01$).

suggest only partial support for this hypothesis. A significant interaction between SES and maternal responsivity ($\beta = 0.23$, $p < 0.01$) on RSA intercept at age 5 was found (Fig. 3), but there was no interaction predicting the ANS trajectory. Children with low SES measured at age 1 had higher resting levels of RSA at age 5 if also coupled with high maternal responsivity. The interaction between SES and maternal responsivity was not significant for PEP or heart rate trajectories or intercepts at age 5.

4. Discussion

The current study examined whether low SES in early childhood "gets under the skin" of children to influence their trajectories of resting heart rate, RSA, and PEP (from 1 to 5 years of age) and levels of ANS activation at age 5 (intercept). The protective effects of maternal responsivity were also examined to assess whether high maternal responsivity influences resting heart function, and whether there was an interaction between SES and maternal responsivity. Hypotheses were partially supported. SES related to heart rate and PEP resting trajectories (and PEP at age 5) in the predicted directions, and maternal responsivity related to RSA and PEP resting trajectories, but the interaction between these two environmental variables predicted only parasympathetic activation (resting RSA) at age 5. Higher maternal responsivity appeared to offset the negative effects of low SES on RSA, and this pattern of early experiences was particularly important for children's resting RSA levels at age 5.

The CHAMACOS study provided an optimal test of our hypotheses because our sample is characterized by high poverty, yet had sufficient variability within this high poverty sample to assess the effects of SES on ANS functioning. Moreover, scarce research has examined the psychophysiological effects of poverty on Mexican American youth, and in particular, how low SES "gets under the skin" for this vastly understudied population. Given the large numbers of Mexican-origin youth who are living in poverty, and the growing number of Mexican American youth living in the US, this population merits more attention (Lopez, 2015).

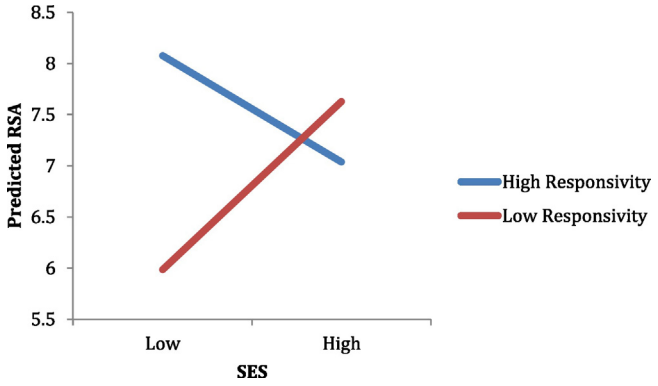

Fig. 3. Interaction between maternal responsivity and family SES predicting children's resting RSA at 5 years of age ($\beta = 0.23$, $p < 0.01$).

The parasympathetic nervous system undergoes momentous shifts during the first year of life (see for review, Beauchaine, 2001). Given the links between vagal tone and emotional and psychological well-being, it is physiologically meaningful that environmental inputs with high emotional value would shape a child's developing resting vagal tone during this developmental window of physiological change, and that the parasympathetic resting set point would have relationships to children's heart and psychological health as they develop. Such adaptive calibration to environmental contexts, whereby environmental inputs biologically embed themselves (for the better in this case) into the child's basal physiology, have implications for future interactions with the social surround and environment (Del Giudice et al., 2011). Indeed, our results fit nicely with prior research showing the important role mothers play during the first year of life in supporting the development of their children's physiological homeostasis, which is associated with healthy socio-emotional and cognitive development (Spangler and Grossmann, 1993; Spangler et al., 1994; Shonkoff, 2012). Such a high quality social environment is likely to foster healthy sensitivity to environmental cues, as well as appropriate physiological response and recovery from daily stressors that are encountered (Del Giudice et al., 2011).

While there are well-known and influential theories about vagal tone development in children (e.g., Porges, 2003), less is generally known about sympathetic development during these young ages and in particular in response to adversities and protective factors. Interestingly, our results showed that SES had a significant effect on both trajectory of change and age 5 resting sympathetic activation, such that low SES at 1 year of age predicted a flatter increase in resting PEP intervals (i.e., PEP remained shorter across time) from 1 to 5 years of age, and also shorter resting PEP at age 5 (i.e., increased sympathetic activation). These results are in line with other research that has found increased sympathetic activity associated with childhood poverty (Evans and Kim, 2013). Given that PEP indexes the "fight or flight" branch of the ANS, it may be more sensitive to the effects of adversity than vagal tone. Given the higher levels of activation across time associated with low SES, it would not be surprising if these children have blunted reactivity to stress given the law of initial values (Kagan et al., 1987). Fortunately, maternal responsivity had an opposing effect on resting PEP trajectory, predicting steeper gains in resting PEP length (i.e., reduced sympathetic activation). The interaction between maternal responsivity and SES was nonsignificant for PEP. More research is needed to assess the effects of prolonged activation of the sympathetic branch as it could be prone to burn-out over time, similar to its' hormonal counterpart, cortisol, which is also released in times of fight-or-flight (e.g., Chida and Steptoe, 2009).

While low SES was not directly related to RSA resting trajectory from one to five years of life, the link between SES and resting RSA levels in our sample at age 5 was differentiated by level of maternal responsivity. In other words, higher maternal responsivity not only predicted how the child's parasympathetic activation developed from 1 to 5 years of age, but also served to offset against the long-term deleterious effects of low SES, at least by 5 years of age. Thus, maternal responsivity appears to have consequences for children's parasympathetic development, in line with other studies relating parasympathetic activity with maternal responsivity (Kaplan et al., 2008; Moore, 2010). This finding supports the notion that the ANS is especially plastic during early childhood, and environmental inputs during this time can continue to influence ANS functioning throughout early development (Van den Bergh, 2011; Lupien et al., 2009).

Finally, it is important to note that the most well-known indicator of ANS functioning, heart rate, is innervated by both the parasympathetic and sympathetic branches and thus, it may be more meaningful to

contextualize our heart rate results in light of what we have discussed above. Maternal responsivity and SES had distinct relationships with which branch of the ANS that they appear to influence. In particular, results of this study demonstrate an RSA-specific set of effects for maternal responsivity, and PEP-specific effects for SES (setting aside heart rate). These effects are related to the chronicity of SES and the potentially more situational experience of maternal responsivity, or related to the proximal nature of maternal responsivity and the more distal effects of SES context elements of the environment (Bronfenbrenner, 1979). If the parasympathetic branch is shaped more by social engagement and can be protected to an extent by positive influences, and sympathetic branch is more responsive to stressful circumstances, then heart rate may be best viewed as an indicator of the overall tax an individual's body experiences as a result of ANS responses in the parasympathetic and sympathetic branches. Regardless, these results give insight into the differential effects of certain kinds of experiences even if the interaction was fairly specific to RSA levels at age 5.

The following limitations should be highlighted. First, this sample was living in moderate to extreme poverty in an agricultural area, and thus these results do not generalize to all Mexican Americans or other racial/ethnic groups. While the sample was optimal for testing the effects of maternal responsivity in low SES environments, future research should attempt to replicate these results in Mexican Americans with more financial resources as well as with other ethnic groups. Second, there are other standardized measures of maternal responsivity that could be employed in future research. However, a large body of epidemiological research has been conducted using the HOME thus allowing for comparison across studies. Third, it should be noted that we changed systems of ANS measurement and data collection after year 1 of the study, which may have impacted our results. However, prior research from this dataset has shown within-person stability of resting ANS measures across these ages despite the changes in equipment (omitted for blind review). Finally, the current study did not model chronicity of poverty or maternal responsivity across the time points. Thus, our model does not disentangle the first year of life from the subsequent years as our measures of SES and maternal responsivity were highly correlated across time and the factor structure limited abilities to decipher individual changes across time due to standardization within each time point. However, our study does provide support for the notion that early poverty and parental responsiveness relate to ANS trajectories of development across the window of early development.

Despite these limitations, this study had a number of strengths. The results of the current study highlight the importance of mother-child interactions, in particular maternal responsivity, in offsetting the deleterious effects of low SES on cardiovascular health and autonomic function from 1 to 5 years of age. These findings provide support for preventative programming aimed at targeting maternal-child interaction among families living in high poverty environments. In particular, programs targeting Mexican American youth should include a strong focus on family interactions, especially maternal responsivity, in order to support resiliency and mitigate the deleterious impact of low SES. As Mexican American children constitute the largest group of youth living in poverty in the U.S. (Fund CsD, 2014), these results have important implications for preventative programming with a potentially wide reach. Further studies are needed to replicate these findings across other ethnic groups at high risk for poverty to better understand if maternal responsivity is an appropriate target for prevention programming more generally. Given that poverty is widespread in the U.S. and is not easily modifiable, prevention efforts to improve the maternal-infant relation could provide a useful pivot point.

Beyond individual-level interventions, policy implications could include allowing for or facilitating more parental involvement with their children early in life, including extending Family Medical Leave Act laws. Parents might further find hope knowing that they have control over something that could buffer the effects of poverty so that it does not “get under the skin” and cause detrimental effects to their children's health. Poverty as a social determinant of health could also be intervened upon, which could reduce health disparities among Mexican-American families who experience poverty at a disproportional rate compared to other groups (Brown and Patten, 2014). For example, the Earned Income Tax Credit creates dramatically different income levels for working families, and changes in this policy over time have been shown to have impacts on child health and development (Evans and Garthwaite, 2014). Future research could assess for change in ANS resting states as a function of such individual and policy-level interventions among high poverty populations.

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