Socioeconomic Adversity, Negativity in the Parent Child-Relationship, and Physiological Reactivity: An Examination of Pathways and Interactive Processes Affecting Young Children's Physical Health

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ABSTRACT

Objective: We tested the hypothesis that socioeconomic status (SES) would predict children's physical health problems at the end of kindergarten among children whose parent reported greater parent-child relationship (PCR) negativity and/or who exhibited greater parasympathetic (RSA) reactivity. We also tested whether RSA and PCR negativity mediated the SES-health association.

Methods: Data were collected from 338 children (mean [SD] age, 5.32 [.32] years) and their primary caregivers (87% biological mothers) during the fall and subsequent spring of kindergarten. In the fall, parents reported income and education level (SES) and PCR negativity, and RSA reactivity was assessed via a standardized challenge protocol for young children. In the fall and then spring, parents reported children's chronic medical conditions and physical health impairments. Multivariate regression was conducted within a structural equation-modeling framework to test hypotheses.

Results: Significant interactions were found between SES and PCR negativity ($b = -0.074$, $p = .035$) and between SES and RSA reactivity ($b = 0.169$, $p = .019$) as predicts children's spring health impairment, adjusting for health in the preceding fall. Lower SES was associated with greater health impairment among children whose parents reported more PCR negativity ($b = -0.110$, $p = .024$) and children who showed greater RSA reactivity ($b = -0.106$, $p = .011$). Socioeconomic status was unrelated to physical health at low PCR negativity or RSA reactivity. Mediation models were not supported.

Conclusion: Parent-child relationship quality and individual differences in stress reactivity may modulate the influence of SES on physical health in childhood.

Key words: SES, early childhood, physical health, parenting, parasympathetic reactivity.

INTRODUCTION

As aptly summarized by Shonkoff et al (1), "adult disease prevention begins with reducing early toxic stress" (*p*. 2256). Socioeconomic adversity in childhood is a particularly powerful form of early-life stress that contributes to poor global physical health and greater health-related impairment throughout the lifespan (2–5). Illustratively, lower socioeconomic status (SES) is associated with greater rates of respiratory illness, obesity, and cardiovascular conditions in childhood and adolescence, and greater morbidity and mortality in adulthood (6–8).

Despite national public health campaigns to reduce health disparities (e.g., *Healthy People* 2010), a strong, inverse association between SES and physical health continues (9).

The negative association between SES and physical health is found across the entire range of the socioeconomic continuum in adulthood (10). Robust evidence for these social disparities in health at the population level may mask important variation at the individual level.

PCR = parent-child relationship, **RSA** = respiratory sinus arrhythmia, **SES** = socioeconomic status

Inconsistent findings have been observed with regard to the steepness of the gradient across childhood (9), raising questions about individual differences that may determine which children are most likely to show poor health in the context of low SES. Indeed, the identification of moderators is essential for early determination of risk as well as the design of well-targeted interventions tailored to those most likely to benefit. It has also been suggested that the origins of SES-related health issues may be traced to developmental processes that are compromised early in life (11). As such, identifying explanatory mechanisms that mediate the negative effects of SES on physical health at a young age would improve understanding of how adversity becomes biologically embedded to affect health (12). Surprisingly, few investigations into the link between SES and physical health outcomes have focused on the first 5 years of life, and even fewer have examined putative moderators or mediators of this association in childhood. This may be partially explained by a reliance on cross-sectional designs; prospective investigations are better suited to examine how risk factors work together to produce disorder (13). The current study addresses this gap by examining under what conditions (i.e., moderation) and how (i.e., mediation) SES is associated with changes in young children's physical health.

The quality of the parent-child relationship is one of the most critical influences on health early in life and represents an important, microlevel dimension of the larger social ecology within which children develop. Socioeconomic adversity may be more likely to "get under the skin" and increase physical health problems in children whose relationship with a primary caregiver is compromised by negativity. There is some evidence that maternal sensitivity can buffer the effects of poverty on adult health (14). However, research evaluating the role of parent-child relationship quality in the association between SES and children's physical health is limited (15), most likely owing to an assumed divide between the socioemotional lives of children and their biomedical well-being. Lower-quality parenting has been shown prospectively to predict poorer health among offspring during early childhood (16). Few if any investigations have examined the potential for parent-child relationship quality to moderate the link between SES and physical health in early childhood. This gap is notable given that, for young children, their perceptions of stressful circumstances are largely filtered through the parent-child relationship, with children relying on their parent to signal when there is threat or stress in the environment. Conflict or negativity in this relationship is likely to exacerbate a child's vulnerability to the negative effects of economic adversity. Alternatively, parenting practices have been described as "socially patterned," and as such, parent-child relationship quality may also mediate the association between SES and physical health (6).

Developmental models of vulnerability to stress have long emphasized the importance of individual physiological reactivity and regulation in the development of psychopathology (17). A burgeoning body of literature suggests that high physiological reactivity to stress may signal heightened susceptibility to environmental influences (11,18). For example, multiple researchers have reported that high physiological reactivity to stress increases vulnerability to maladjustment among children exposed to family stress (19,20). These studies focused on changes in heart rate variability (HRV), or rapid changes in the beat-to-beat timing of systole. Although both sympathetic and parasympathetic branches of the autonomic nervous system (ANS) influence HRV, psychophysiological theories of HRV emphasize the role of respiratory sinus arrhythmia (RSA) in emotion regulation (21). Respiratory sinus arrhythmia, the oscillation in heart rate produced by the respiratory cycle, indexes parasympathetically mediated HRV. During a challenging task, RSA decreases (parasympathetic withdrawal), and the magnitude of this decrease reflects reactivity to the task. Several investigations have found that physiological reactivity moderates the association between quality of the family environment and physical health (22,23) and that reactivity may play a similar sensitizing in the prediction of physical health in the context of socioeconomic adversity. Alternatively, adversity-induced alterations in biological systems may play a mechanistic, explanatory role in the SES-health connection such that physiological reactivity mediates the relation between SES and physical health (24). Evans and Kim's (25) finding that greater durations of poverty in childhood predicted blunted blood pressure responses during adolescence provides some evidence in support of such early-life biological embedding of SES-related stress, but additional work is needed in this area, particularly with longitudinal designs (26).

In sum, much remains to be learned about the prospective effects of low SES on children's *physical* health in early childhood and under what conditions or by which pathways these effects are most likely to occur. Comprehensively addressing such questions is best achieved by examining risk and explanatory factors that exist at multiple levels and across physiological and socioemotional domains (27). It is important to consider the temporal order of variables when developing hypotheses of moderation and mediation (13). The pattern of relations between socioeconomic factors, family environment, and children's health are likely to be complex and transactional, making several different orderings of relations among SES, parent-child relationship quality, and children's stress reactivity plausible. Our primary hypothesis was that parent-child relationship quality and children's parasympathetic reactivity would each moderate the association between SES and children's physical health. Given the importance of examining the

longer-term effects of SES across children's transition to kindergarten, we predicted that this finding would be present for children's physical health in the spring, adjusting for physical health in the preceding fall. Inclusion of children's baseline physical health at the beginning of the school year allows for potential conclusions to be drawn regarding the prospective effects of SES, rather than its correlates. Specifically, we expected that low SES would predict significantly poorer health in the spring among children whose parent reported high negativity in the parent-child relationship and who exhibited elevated physiological reactivity to stress in the preceding fall. Although evidence in young children is limited, in light of the advancing theories of allostatic load and biological embedding, as well as evidence that parenting can be affected by SES, we also examined whether parasympathetic reactivity and parent-child relationship quality mediated the association between SES in the fall and physical health in the subsequent spring.

METHODS

Participants

The current study comprised 338 children (163 females and 175 males) who participated in a longitudinal study of social status, biological responses to adversity, and child mental and physical health. Participants were recruited in 3 waves from 29 kindergarten classrooms within 6 public schools in the San Francisco Bay Area (Oakland, Albany, and Piedmont Unified school districts) during the fall seasons of 2003, 2004, and 2005. The schools were selected to represent the sociodemographic and racial/ethnic characteristics of the metropolitan area. Families were recruited through home mailings, presentations at kindergarten parents' welcome nights, and in-person recruitment during school drop-off and pick-up times. Although every family with a child in a participating classroom was invited to take part in the study, families who were not fluent in either English or Spanish were excluded to ensure adequate comprehension of study questionnaires. Schools were provided with $20 per child enrolled in the study.

Participants' characteristics are displayed in Table 1. The mean (SD) age of the children at the kindergarten entry was 5.32 (0.32) years (range, 4.75–6.28). The sample was racially and ethnically diverse, and among families that provided demographic information (n = 322), children were identified as African American, 19%; Asian, 11%; European or White, 43%; Latino, 4%; multiethnic, 22%; and "other" race/ethnicity, 22%. Primary caregivers were biological mothers, 87%; biological fathers, 9%; adoptive mothers, 2.5%; biological grandmothers, 0.6%; and other relations, 0.9%. As most of the primary caregivers were parents, these terms are used interchangeably here.

Procedures

Data for this study were collected in the fall (T1) and spring (T2) during the kindergarten year. Parents' informed consent and children's assent were obtained before the start of data collection. Parental reports of parental education, family income, parent-child relationship negativity, and children's physical health were collected through an extensive series of home mailings, and families were compensated with $50 for each completed time point. In addition, children participated in a 20-minute, in-school reactivity protocol during which time their autonomic activity was assessed.

TABLE 1. Participants' Characteristics

Sex	
Female	163 (48%)
Male	175 (52%)
Participants' ages, mean (SD), range	5.32 (.32), 4.75–6.28
Ethnicity (n = 322)	
White, non-Hispanic	43%
Hispanic/Latino	4%
Black/African American	19%
Asian	11%
Multiethnic/Multiracial	22%
Other	2%
Family income (n = 322)	
Less than $10,000	13 (4%)
$10,000–$14,999	8 (3%)
$15,000–$19,999	7 (2%)
$20,000–$29,999	15 (5%)
$30,000–$39,999	22 (7%)
$40,000–$49,999	15 (5%)
$50,000–$59,999	16 (5%)
$60,000–$79,999	40 (13%)
$80,000–$99,999	50 (16%)
> $100,000	125 (40%)
Highest education level completed in household (n = 322)	
Less than high school	8 (2%)
High school	18 (6%)
Some college or 2-year degree	55 (17%)
4-year college graduate	57 (18%)
Some graduate or professional school beyond college	39 (12%)
Professional or graduate degree	145 (45%)

Measures

Socioeconomic status (SES)

Assessing multiple dimensions of SES allows for greater understanding of how social determinants contribute to an outcome under study, thus, we assessed both family income and education. Total household annual income, including all sources, was assessed on an 11-point scale that ranged from <$10,000 to >$200,000 (mean, $60,000–79,999; median, $80,000–99,999). Highest level of educational attainment for any caregiver in the household was assessed using a 6-point scale that ranged from less than a high school diploma to a professional or graduate degree, with 75% of caregivers having at least a college degree, a level commensurate with the surrounding community (Table 1). Socioeconomic status was calculated as the average of standardized highest household education of adults in the home and standardized total annual household income.

Negativity in the Parent-Child Relationship

The quality of the parent-child relationship was assessed using 3 items from the Child-Rearing Practices Report (28), which were rated on a scale from

1 (extremely true) to 7 (extremely untrue). Items were the following: "I often feel angry with my child," "There is a good deal of conflict between my child and me," and "I feel that my child is a bit of a disappointment to me." These items have been used together in numerous prior studies to capture parent-child relationship negativity and have shown the expected associations with multiple aspects of child functioning (29–32). The items were reverse-scored and averaged to create a composite of negativity in the parent-child relationship, with higher scores indicating greater negativity in the parent-child relationship. Internal consistency was low ($\alpha = 0.59$) but reasonable given the small number of items (33).

Respiratory Sinus Arrhythmia (RSA) Reactivity

Children's autonomic reactivity was assessed by computing changes in RSA (34) during a developmentally challenging reactivity protocol. This kindergarten "stress reactivity" protocol is detailed extensively elsewhere (29,35) and only briefly summarized here. After a 2-minute baseline period during which the experimenter read a calming story aloud, children were challenged with 4 age-appropriate tasks designed to elicit moderate stress responses to social, cognitive, sensory, and emotional challenges (22). To measure the psychophysiological response to the 4 challenges without confounding by motor activity, each challenge task in the reactivity protocol was preceded by a nonchallenging "control task" that paralleled the motor and engagement demands of the corresponding challenge task (35). The challenge tasks included a structured interview (social challenge), a digit-span recitation task (cognitive challenge), taste identification (sensory challenge), and watching an emotion-evoking movie clip (emotional challenge).

After the child was familiar with the equipment, 4 spot electrodes were placed in the standard tetrapolar configuration on the child's neck and chest, and electrocardiogram electrodes were placed on the right clavicle and lower left rib. A 4-mA AC current at 100 kHz was passed through the 2 current electrodes. Respiratory sinus arrhythmia was monitored continuously during the protocol. Data were acquired using the Biopac MP150 (Biopac Systems, Santa Barbara, CA) interfaced to a PC-based computer. Analog data were continuously monitored on the computer for signal and noise, and digitized data were stored for offline analysis. Respiratory sinus arrhythmia was derived in accordance with recommendations of the Society for Psychophysiological Research committee on heart rate variability (36). The sampling frequency was 1 kHz. Before analyses, each waveform was verified, interbeat intervals were visually checked, and artifacts were identified using an algorithm (37) created for the MindWare software program (http://www.mindwaretech. com). Respiratory sinus arrhythmia was estimated as the natural logarithm of the variance of heart period within the high-frequency band pass associated with respiration at this age (i.e., 0.15–0.80 Hz) (38). Outlier data were checked and verified minute by minute if they were >3 SD from the group mean. Missing ANS data were due to acquisition or scoring problems such as equipment malfunction, research assistant error, extraneous movement, and electrode displacement; 96% of the 338 children had scorable ANS reactivity data (n = 326). Mean RSA magnitude was calculated for each 1-minute interval and averaged within task. Respiratory sinus arrhythmia levels during each of the control tasks were used as baseline reference to create 4 task RSA reactivity scores (task minus baseline). These 4 difference scores were then averaged to create one index of RSA reactivity. Negative scores, indicating a decrease in RSA and vagal withdrawal in response to the challenge, represented greater parasympathetic reactivity. Conversely, positive scores, reflecting an increase in RSA and vagal augmentation in response to the challenge, constituted low parasympathetic reactivity. Average control-task RSA was significantly correlated with RSA change ($r = 0.19$, $p < .01$), but control-task RSA was not correlated with any of the physical health indices, negativity in the parent-child relationship, or SES (p values ranged from .42 to .74).

Child Physical Health Outcomes

Children's physical health was assessed using parent report on 2 subscales from the MacArthur Health and Behavior Questionnaire (HBQ) (39). The 5-item Global Physical Health subscale measures children's general physical health, including physical health-related distress and impairment. The Chronic Medical Conditions (CMC) subscale is a checklist of 22 chronic medical conditions (e.g., asthma, diabetes, hearing problems, etc.). The latter subscale is scored by summing the number of conditions endorsed. Higher scores on Global Physical Health or CMC indicate greater health-related impairment or more chronic health conditions, respectively. These MacArthur Health and Behavior Questionnaire subscales have demonstrated good test-retest reliability and rank-order stability, good cross-informant agreement, and independence from demographic factors (39,40). Furthermore, scores on these subscales have been shown to relate positively to objective health measures, such as body mass index (41).

Statistical Analysis

All analyses were conducted using Mplus with full information maximum likelihood estimation and bias-corrected bootstrapped standard errors; these estimation techniques enable the use of all available data points, are robust to multivariate non-normality, and, as noted by Fritz and MacKinnon (42), maximize statistical power. Path models with observed variables were estimated within a structural equation modeling framework, which has many advantages over other statistical methods, including the ability to examine multiple mediator and outcome variables simultaneously, to estimate both individual coefficients and overall model fit, and to model error terms rather than pooling error across variables (43–45). In addition, this method allowed for the examination of specific indirect effects even if there was an absence of significant total or direct effects in the mediation model (44).

Two path models were tested, and adequacy of model fit was determined by examining the following fit indices were examined: RMSEA, SRMR, and CFI. Specifically, we first tested a multiple moderation model (model 1) in which the association between SES at T1 and physical health outcomes at T2 was hypothesized to be moderated by both T1 parent-child relationship negativity and T1 RSA reactivity, adjusting for T1 physical health outcomes. If the estimates associated with either interaction term were significant, we examined the nature of the interactions by computing and testing the statistical significance of the simple slopes for the association between T1 SES and T2 physical health impairment at 1 SD above and below the mean of the moderating variables, following guidelines outlined by Aiken and West (46). Next, we tested a multiple mediation model (model 2) in which T1 parent-child relationship negativity and T1 RSA reactivity were hypothesized to mediate the association between T1 SES and T2 physical health outcomes, adjusting for T1 physical health outcomes. Finally, based on research indicating differences in the association between SES and health depending on race/ethnicity, we also tested 2 multiple group models (model 1 and model 2 by racial/ethnic minority status) to ascertain whether relations among our variables of interest were specific to or different across white children and children from other racial/ethnic groups.

RESULTS

Zero-order correlations for all study variables are included in Table 2. As shown, chronic medical conditions in the fall and spring were not significantly related to parent-child relationship negativity, RSA, or SES; however, T1 and T2 physical health-related impairment were positively associated with parent-child relationship negativity and negatively associated with SES at T1.

TABLE 2. Zero-Order Correlations Among Study Variables

	1	2	3	4	5	6
1. SES						
2. PCR (n)	−0.03 (297)					
3. RSA (n)	−0.06 (310)	0.002 (286)				
4. CMC-F (n)	−0.07 (294)	0.04 (289)	0.017 (284)			
5. PHI-F (n)	−0.14* (293)	0.24** (287)	0.06 (282)	0.39** (290)		
6. CMC-S (n)	−0.01 (246)	0.04 (241)	0.04 (237)	0.77** (236)	0.31** (237)	
7. PHI-S (n)	−0.18**(279)	0.30** (297)	−0.08 (269)	0.28** (268)	0.50** (264)	0.33** (245)

PCR = Parent-child relationship negativity; RSA = respiratory sinus arrhythmia reactivity; CMC = chronic medical conditions (Fall or Spring);
PHI = physical health-related impairment (Fall or Spring).

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

Multiple Moderation Model

Model fit was adequate, according to recommendations for cutoff points (47,48): RMSEA, 0.074 [90% confidence interval, 0-0.149]; CFI, 0.989; χ^2, 5.694, $p = .058$; SRMR, 0.019. As shown in (Table 3 and Fig. 1), the positive association between SES and T2 chronic medical conditions did not reach statistical significance ($p = .078$), and there was no evidence of moderation by either parent-child relationship negativity or RSA. In contrast, lower SES ($p = .041$), greater parent-child relationship negativity ($p = .001$), and greater RSA reactivity ($p = .012$) predicted greater health-related impairment at T2; however, these main effects must be examined within the context of 2 statistically significant interactions: both parent-child relationship negativity ($p = .035$) and RSA reactivity ($p = .019$) moderated the effect of T1 SES on T2 physical health-related impairment. To test whether these associations varied by race/ethnicity, an unconstrained multiple group (white and nonwhite) model was estimated, followed by a constrained multiple group model in which associations among variables were constrained the be equal. A χ^2 difference test indicated there was no improvement in model fit ($p = .549$); thus, there was no evidence that associations among the variables varied by race/ethnicity.

As shown in Figure 2, our continuous measure of SES was negatively related to T2 health-related impairment for children with higher levels of negativity in the parent-child relationship (unstandardized estimate, −0.110; SE, 0.049; $p = .024$); however, there was no association between SES and health at lower levels of negativity (unstandardized estimate, 0.016; SE, 0.033; $p = .63$). As shown in Figure 3, SES was negatively related to T2 health-related impairment for children exhibiting higher RSA reactivity (unstandardized estimate, −0.106; SE, 0.042; $p = .011$); however, there was no association between SES and health at lower levels of RSA reactivity (unstandardized estimate, −0.006; SE, 0.034; $p = .86$).

Multiple Mediation Model

A multiple mediation model was tested in which the effect of SES on the 2 physical health outcomes was mediated by negativity in the parent-child relationship and RSA reactivity. Model fit was excellent: RMSEA, 0.029 [90% confidence interval, 0–0.10]; CFI, 0.997; χ^2, 3.829; $p = .28$;

TABLE 3. Unstandardized Estimates in the Multiple Moderation Model Predicting T2 Physical Health Outcomes

	T2 Chronic Medical Conditions			T2 Physical Health Impairment		
	Coefficient	SE	p Value	Coefficient	SE	p Value
T1 CMC	.736	.047	<.001	—	—	—
T1 PHI	—	—	—	.443	.081	<.001
T1 RSA	−.071	.164	.663	−.152	.060	.012
T1 PCR	.001	.046	.986	.093	.028	.001
SES	.093	.053	.078	−.054	.026	.041
SES × RSA	.070	.217	.747	.169	.072	.019
SES × PCR	−.070	.057	.218	−.074	.035	.035

CMC = Chronic medical conditions; PHI = physical health impairment; RSA = RSA reactivity; PCR = Parent-child relationship negativity;
SES = socioeconomic status.
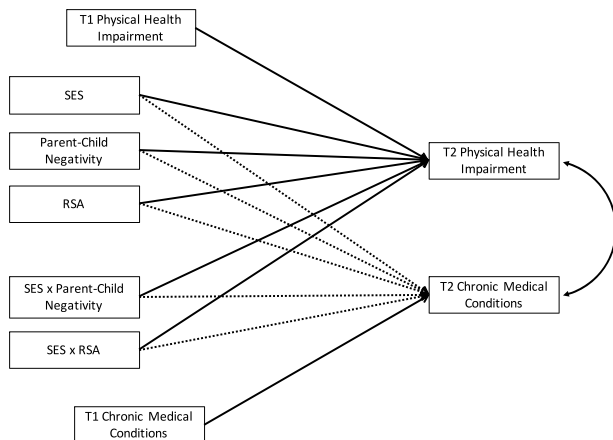
FIGURE 1. Hypothesized associations between SES, parent-child relationship negativity, RSA reactivity, and physical health outcomes. Bold lines are significant at $p < .05$.

SRMR, 0.020. However, consistent with the zero-order correlations (Table 2), there was no association between SES and either parent-child relationship negativity ($p = .88$) or RSA reactivity ($p = .35$), and there was no indirect effect of SES on either measure of physical health at T2 (specific indirect effect p values between .44 and .99, and total indirect effect p values between .10 and .11), indicating no mediation. A multiple group model was estimated to test whether mediation was evident in either white or nonwhite participants. The model would not converge, suggesting poor fit, and was not examined further.

DISCUSSION

Socioeconomic status is a robust epidemiologic predictor of health, yet there is substantial heterogeneity in adult physical health outcomes among those exposed to early adversity (1). Much remains to be learned about the conditions under which lower SES environments predict greater physical health problems in childhood and beyond (26). Studies of
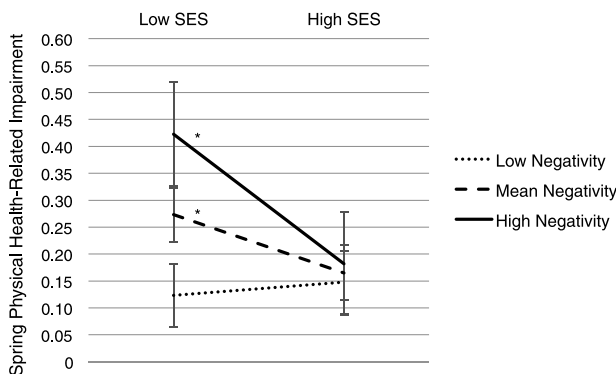
FIGURE 2. Prospective association between SES and spring physical-health impairment in children at low (−1 SD), mean, and high (+1 SD) levels of parent-child relationship negativity. *$p < .05$. Error bars reflect 95% confidence interval.
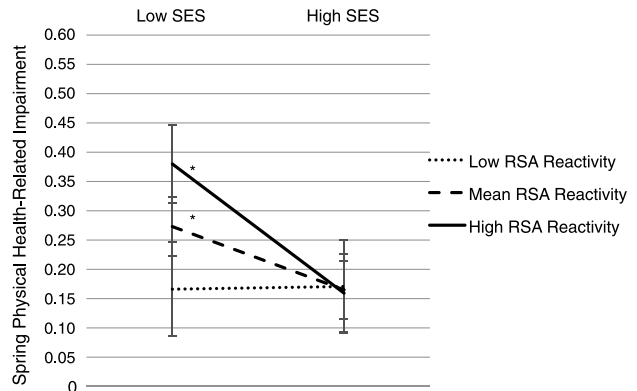
FIGURE 3. Prospective association between SES and spring physical-health impairment in children at low (+1 SD), mean, and high (−1 SD) levels of RSA reactivity.

mediation and moderation examine possible "third variables" (47) posited to shape the relation of economic disadvantage to children's health. The current study examined the prospective relation between family SES and child physical health as well as factors postulated to influence or underlie the SES–physical health association during the transition to kindergarten. Lower SES predicted greater spring physical health impairment, over and above fall physical health impairment, but not chronic medical conditions. In addition, this association was strongest among children who experienced high parent-child relationship negativity or heightened physiological reactivity to stress. There was no evidence of mediation by parent-child relationship quality or stress reactivity. Findings suggest that the effects of socioeconomic adversity, one of the most potent and persistent contributors to physical health problems, can be buffered by more proximal social environments (e.g., the parent-child relationship) and individual differences (e.g., parasympathetic reactivity).

There is extensive evidence that the quality of the parent-child relationship plays a critical role in the mental health of young children. As hypothesized in the current study, we observed detrimental associations between low SES and children's physical health, but only among children reared in environments characterized by high levels of parent-child relationship negativity. It has been suggested that low SES is associated with greater stress and more chaotic home environments (49). The present findings suggest that the physical health correlates of this adverse context can be *diminished* by a family environment with lower levels of parental harshness and parent-child conflict or *exacerbated* by heightened negativity in the parent-child relationship. In the former circumstance, the stressors of socioeconomic disadvantage may be tolerable and amenable to coping strategies, whereas in the latter condition, such stressors may become more toxic and pathogenic (1). Although a reading of the current study should not conflate positive parenting with low levels of parental negativity, the likelihood

of positive parent-child interactions is presumably greater when negativity is lower and may operate as a buffer against the consequences of SES adversity. Cumulative risk research also offers a perspective on the combined detrimental effects of low SES and harsh parenting, as such studies have consistently observed negative consequences of co-occurring environmental adversities on children's well-being (50).

Prior studies of poverty exposure and physical health in childhood have focused on the role of proinflammatory processes (51), elevated HPA-axis activity (52), and blood pressure responses to stress (25). To our knowledge, the role of parasympathetic reactivity in the connection between socioeconomic disadvantage and physical health outcomes has not yet been examined. Challenge-induced reductions in parasympathetic activation may represent a particularly relevant indicator of physiological reactivity to psychosocial stress. As described in Porges's polyvagal theory, the vagal system exerts a critical role in regulating adaptive social and emotional responding to environmental challenges (21). Dysregulated vagal responses have implications for psychopathology related to poor emotion regulation (53) and may extend to an influence on the development of physical health problems. The present findings indicated that in the context of socioeconomic disadvantage, physical health impairments at the end of kindergarten were highest among children with greater RSA withdrawal and lowest among less parasympathetically reactive children. Although there have been well-supported findings that greater RSA reactivity may act as a protective factor (54), the current findings are consistent with previous research showing that physiologically reactive children exposed to adverse early family environments are *more* susceptible to mental health problems relative to their less reactive peers (55,56). The current study extends such literature, suggesting that heightened RSA reactivity may be viewed as a vulnerability factor that places children at increased risk for experiencing harmful effects of socioeconomic adversity on physical health.

There was no association between any of the variables of interest and chronic medical conditions across the school year. The absence of these relations was surprising given the extensive literature documenting associations between SES and asthma and cardiovascular conditions (57). As might be expected within a community sample of 5-year-old children, approximately half of the children did not have chronic health conditions, and relatively few had more than one condition. The current study used statistical methods robust to non-normal data; however, SES relations with health might be better detected in a population with a greater number of conditions. Furthermore, SES has most often been implicated in associations with the *severity* of a chronic condition in childhood, such as asthma, and it is reasonable to surmise that the link between SES and

number of different medical conditions might not arise until later in life when comorbidity is a more common occurrence. In addition, previous research suggests the effects of SES on health may differ among racial groups. Indeed, prior study within this sample observed differences in the associations between SES and children's daily cortisol between minority and nonminority children (58). However, in the current investigation, associations between parenting, RSA reactivity, and physical health outcomes were not moderated by racial/ethnic minority status. Future studies with larger diverse samples spanning childhood are needed to comprehensively examine the complex relations among SES, race/ethnicity, and children's health.

Neither the parent-child relationship nor RSA reactivity were associated with SES, precluding those variables from serving as mediators of the association between SES and health within these analyses. Low SES is associated with a multitude of environmental challenges and uncertainties, with effects on domains of parenting and the family environment that were not assessed here, including family stress, parental investment, parent psychopathology, and interparental conflict (49,59,60). The absence of a relation between SES and the measure of parent-child negativity used in the current study suggests that the affective quality of the parent's perception of their relationship with the child operates independently from SES-related stress to influence the connection between family SES and child physical health. Given this, interventions focused on improving or protecting physical health in children exposed to socioeconomic adversity should incorporate supports for the parent-child relationship. Although it has been proposed that the biological embedding of early life stress can occur through effects on the autonomic nervous system (12), given the ongoing development of RSA regulatory functioning during early childhood, an association between SES and RSA reactivity may not be easily detectable by age 5. Additional research of other indices of biological reactivity (e.g., sympathetic nervous system), and future longitudinal studies will be important for determining whether these theories apply to early-life SES effects on health.

A number of limitations must be considered when interpreting the findings. First, although the sample size was significantly larger than most samples reported in psychological studies examining mediation (42), there may have not been adequate power to detect small indirect effects in the multiple mediation model. Second, SES can be measured in multiple ways, including objective and subjective assessments. We used a "traditional" measure of SES that encompassed family income and highest level of educational attainment in the household. Other research suggests the importance of "contextual" (e.g., neighborhood-based) approaches to capturing socioeconomic disadvantage, since community resources can vary widely even among families with similar SES (61). Third, we cannot rule out

the possibility that lower SES parents might overestimate their children's health impairments, and findings would be strengthened by an objective measure of physical health (i.e., physical examinations and medical record review). Fourth, the current study used a brief parent-report measure of parent-child negativity that has previously demonstrated relations with children's mental and physical health (31,32). However, the internal consistency of this scale was low in the current sample, which may be attributed to its limited number of items (33). The present study also focused on negative qualities of the parent-child relationships, and supportive parenting is likely also an important protective factor in the association between low SES and health. Observational measures of parent-child relationship quality that capture positive and negative attributes might provide a stronger test of the role of parenting in mitigating the effects of economic adversity on child physical health.

Finally, it is important to consider the temporal order of variables when conducting statistical tests of moderation and mediation (13). However, given the dynamic nature of parent-child relationship quality, physiological reactivity, and socioeconomic risk early in life, the temporality of each measure relative to another is not easily established. Randomized controlled trials of interventions designed to improve parent-child relationship quality (62) and children's physiological regulation (63) in the context of family-related stress represent the criterion standard for establishing causality. Thus, the most powerful test of the associations observed here would involve improvements in physical health as a result of intervention-induced changes in parenting and children's regulatory capacity in children exposed to socioeconomic adversity. Bolstered by the results of the current study, such examinations are apt for future research.

The present investigation contributes to extant research on the relation between economic adversity and physical health in childhood. The association between SES and physical health varies with age and is particularly strong for some outcomes during early childhood (8). The transition to kindergarten represents a critical developmental period in the life of a child. It is a time of increased expectations, greater independence and responsibility, and new demands for social competence (64). Physical health is an important component of school readiness and children's potential for learning (65). In short, the first school year lays a foundation for a child's academic and social development during the subsequent years, and children who experience physical health problems face greater academic challenges and lower SES later in life (66). Identifying the environmental, familial, and individual qualities that influence children's physical health during this time can have a substantial impact on their course of development.

In addition to focusing on a sensitive developmental period, the use of physical health measures collected at the beginning and end of the school year offered a unique opportunity to examine the prospective effects of SES on physical health outcomes. By adjusting for baseline health, we were able to ascertain whether SES contributed uniquely to health outcomes at the end of a school year, above and beyond physical health conditions at the time of SES assessment. Findings suggest that intervention need not occur before associations between SES and physical health arise to be effective. Furthermore, in examining 2 putative moderators, we gave concurrent consideration to both family-level (e.g., parenting) and individual-level (e.g., physiological) factors, thereby advancing research on the conditions under which economic disadvantage influences children's health. Finally, by using structural equation modeling, we were able to test these relations simultaneously, avoiding the problems of increased error probability associated with conducting multiple statistical tests and improving our ability to understand the complex, multilayered predictors of health.

In sum, a robust and growing literature supports the pervasive, detrimental effects of low SES on health across the lifespan. Socioeconomic status–related health disparities have increased over the course of the past several decades (4), elevating the salience and importance of such research. The complexity of this association calls for attention to factors that explain or shape the relation between economic adversity and health outcomes, particularly during the rapid developmental period of early childhood. The current study examined the role of negativity in the parent-child relationship and individual differences in RSA reactivity in the association between SES and physical health outcomes among children during the kindergarten year. Socioeconomic adversity was associated with greater physical health impairment among children exposed to higher levels of parental negativity and among those with elevated RSA reactivity, but was unrelated to health impairment at low levels of negativity or reactivity. Support for a mediating role of either variable was not found. The moderating influence of such individual- and family-level variables may inform public policy and early childhood interventions to tailor programmatic efforts to those most vulnerable to the costs of socioeconomic adversity on health and development.

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