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The Association Between Childhood Emotional Functioning and Adulthood Inflammation Is Modified by Early-Life Socioeconomic Status

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Objective: Identifying interrelationships among childhood social disadvantage, emotional functioning and adult health may help illustrate how health disparities may become embedded early in life, yet few have considered how these factors are associated. We examined whether the association of child emotional functioning and adult health risk was modified by child socioeconomic status (CSES), or whether child emotional functioning mediated the association of CSES and adult health risk. ***Method:*** We studied 430 adult offspring (mean age 42 years) of Collaborative Perinatal Project participants, a cohort of pregnant women enrolled in 1959–1966 (Broman, Nichols, & Kennedy, 1975; Niswander & Gordon, 1972). Child emotional functioning was assessed by psychologist ratings at age 7 and included inappropriate self regulation (ISR) and distress proneness. CSES measures included parental education, household income, and parental occupation. Adult health risk was measured by the inflammatory marker C-reactive protein (CRP). Hypotheses were tested with multiple linear regression. Effect modification was evaluated via interaction terms and stratification of fully adjusted models by CSES. Mediation by child emotional functioning was evaluated via coefficient changes. ***Results:*** There was no evidence that child emotional functioning mediated the association of CSES and CRP. Significant interactions were observed for ISR and low income ($b = 1.67$, $SE = 0.70$, $p < .05$), and distress proneness and low ($b = 3.14$, $SE = 1.47$, $p < .05$) and middle ($b = 3.52$, $SE = 1.46$, $p < .05$) income. Stratified models indicated that associations of child emotion with CRP varied significantly by level of parental education, household income and occupation. ***Conclusion:*** The highest levels of adult inflammation were observed among those with childhood emotional problems who were also exposed to low socioeconomic status as children. This study suggests adulthood disparities in CRP may have developmental origins in childhood adversity.

Keywords: SES, emotional functioning, inflammation, life-course

Reducing and eliminating health disparities is a central goal of public health research and practice today. Researchers are increasingly examining disparities from a life-course perspective as evidence is accumulating that childhood adversity has lifelong consequences for health, with disease occurring often after a lag of years or decades from initial exposure (Shonkoff, Boyce, & McEwen, 2009). Several studies suggest that early-life adversity, and in particular childhood psychosocial stress, may affect later health through the alteration of biological systems during a sensitive period of development and also through the accumulation of risks over the life course (Shonkoff et al., 2009). As such, health disparities patterned by race and socioeconomic status (SES) that are observable in adulthood may be attributable in part to psychosocial adversity and social disadvantage that originated early in life.

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Poor emotional functioning in childhood has increasingly been recognized as a potential early-life risk factor for adverse adult health outcomes. Prior studies have documented associations of child emotional functioning with diverse outcomes such as obesity, migraines, and asthma (Goodwin et al., 2008), with self-rated health and number of illnesses at age 35 (Kubzansky, Martin, & Buka, 2009), and also with biomarkers of inflammation as measured by the C-reactive protein (CRP; Appleton et al., 2011; Danese et al., 2009; Odgers et al., 2007). A significant body of work has also suggested that poor emotional functioning is often exacerbated by exposure to poverty, resulting in significantly worse emotional functioning for children living in low socioeconomic environments (Repetti, Taylor, & Seeman, 2002; Shonkoff et al., 2009). Taken together, these studies suggest that the effects of childhood emotional functioning and early-lifeSES on adult health may be interrelated. To gain greater insight into these relationships, in the present study we examined the joint associations between early-life SES and child emotional functioning with CRP, a marker of inflammation in adulthood.

There has been much recent interest in emotional factors as mediators of SES and health associations. Although several studies provide indirect evidence in support of this idea, few have directly tested emotional factors as mediators and findings have been mixed (Matthews, Gallo, & Taylor, 2010). As such, Matthews, Gallo, and Taylor (2010) recommended building the evidence base by explicitly testing whether emotional factors can explain SES health gradients. However, when considering these factors from a life-course perspective, a mediation model may not be appropriate. Child emotional functioning reflects stable attributes that are somewhat sensitive to the environment in which the child lives (Rothbart & Bates, 1998). As such, it is possible that the strength of the relationship between early-life SES and emotional functioning may be weaker in childhood than in adulthood, when such characteristics have had more time to be shaped by social influences. Therefore, the child emotion-adult health association may be modified by early-life SES. In the current study, we test this effect modification hypothesis, while also examining the more prevalent hypothesis that emotions mediate the effect of SES on health. In examining these alternative models, this study contributes to an important ongoing theoretical discussion as to *how* SES, emotions, and health are jointly associated.

We examined a biomarker of inflammation as the study outcome. Chronic inflammation is associated with risk of age-related diseases, including hypertension, coronary heart disease, and diabetes (Singh & Newman, 2011). As a result, studies among younger populations have begun considering higher levels of inflammation as an early marker of risk for subsequent poor health (Danese et al., 2009). Inflammation is also an important component of the stress response and involves the secretion of cytokines and acute-phase proteins like CRP to promote tissue repair and resist infection (Aiello & Kaplan, 2009). Poor emotional functioning may disrupt stress recovery processes via physiological (e.g., hypothalamic-pituitary-adrenal axis dysregulation) and behavioral (e.g., smoking, obesity) pathways, each of which contribute to increased risk of chronic inflammation (Everson-Rose & Lewis, 2005; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Moreover, recent work has found higher adult CRP to be associated with poor emotional functioning at age 7 (Appleton et al., 2011), as well as with adverse experiences that elicit emotional distress, including early-life social isolation, maltreatment (Danese et al., 2009) and a risky family environment (Taylor, Lehman, Kiefe, & Seeman, 2006). Therefore, we examined how CRP may be influenced by early-life SES and emotional functioning.

Beginning early in life, children display distinct predispositions to certain emotions and ways of responding to the world, and those with highly reactive emotional styles must exert more effort to control and manage emotions effectively than children with less reactive styles (Rothbart & Bates, 1998; Shonkoff & Phillips, 2000). Variation in resources available across child SES groups might help explain why child emotion-adult health associations differ by level of SES. Resources in high SES environments may buffer the deleterious effects of being distress prone, whereas fewer resources available in low SES environments may exacerbate the risk for poor health over the life course. As such, children with reactive or distress-prone emotional attributes who grow up in low SES environments may have worse psychological functioning and subsequent poor health than do children with similar emotion attributes that grow up in high SES environments. Therefore, we hypothesized that the association between child emotional functioning and adult health (as measured by CRP) is modified by early-life SES.

Alternatively, childhood emotional functioning might mediate early-life SES and adult health associations (Matthews et al., 2010). Low SES may influence the development of poor emotional functioning in childhood, thereby laying the foundation for adult emotional functioning, which in turn affects adult health outcomes via behavioral and physiological pathways. Although separate associations for SES and emotions, and emotions with health have been documented (Matthews et al., 2010), formal tests of mediation of the child SES-adult health association by child emotional functioning remains largely untested.

Therefore, the aim of this study was to test whether the association of childhood emotional functioning and adult inflammation was modified by early-life SES, or whether child emotional functioning mediated the association of early-life SES and adult CRP (see Figure 1). We hypothesized that children with poor emotional functioning living in low SES environments would have higher levels of inflammation as adults compared with children with similar emotional problems, but who lived in high SES environments. Because some components of emotion are innate (Rothbart & Bates, 1998), the strength of the relationship between emotional functioning and the social environment may be somewhat weaker in childhood and become stronger as child characteristics are shaped by social influences with age. Thus, as we were examining emotional processes early in life, we favored the modification hypothesis over the mediation hypothesis.

Hypothesized and alternative models are presented in Figure 1. The models are oversimplified to display the variables examined in this study. Associations among early-life SES, emotion, and adult inflammation involve many other life-course factors. For example, in a recent study we examined whether adulthood education, depressive symptoms, smoking, or body-mass index (BMI) mediated the association of child emotional functioning and adult CRP (Appleton et al., 2011). We found that adulthood BMI, but not other adult factors, partly mediated the associations of externalizing domains of child emotional functioning and adult CRP. In the current study, primary hypotheses are examined without adjustment for adulthood factors, as controlling for pathway variables could mask associations with CRP.

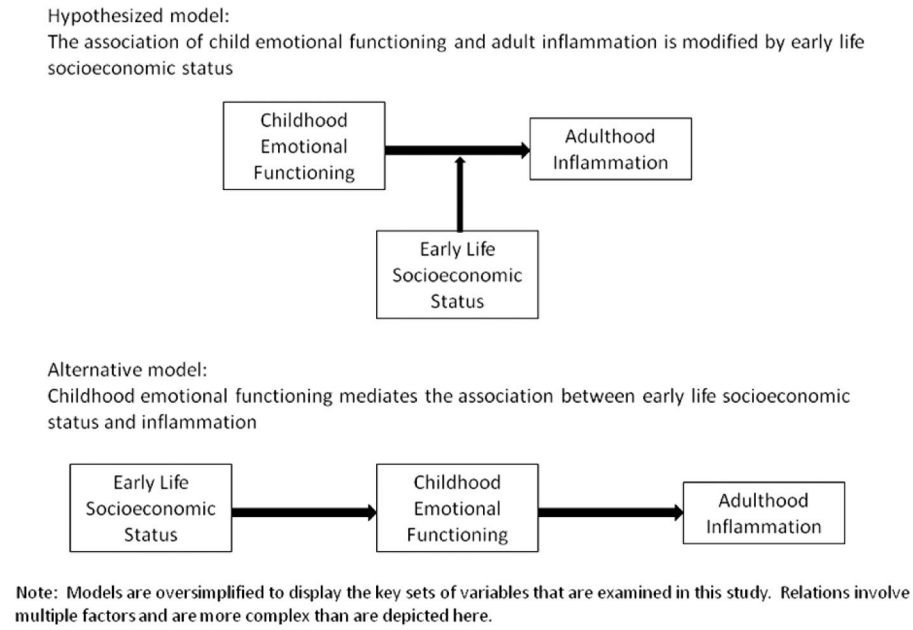

Figure 1. Hypothesized and alternative models for childhood emotional functioning, inflammation and early-life socioeconomic status.

Additional analyses considered the contribution of adulthood factors in the child SES-emotion-CRP associations.

Two measures of childhood emotional functioning (self-regulation, distress proneness) were selected, as previous work has found these and related psychological factors to be associated with CRP (Appleton et al., 2011; Danese et al., 2009; Odgers et al., 2007) and physical health outcomes in adulthood (Goodwin et al., 2008; Kubzansky et al., 2009), and also because related externalizing problems are associated with childhood SES (Shonkoff & Phillips, 2000). We examined three dimensions of early-life SES measured during childhood: parental education, household income, and occupation. Though related, indicators of SES are not interchangeable, as each may affect disease risk in different ways (Braveman et al., 2005), and therefore may have different intervention implications. We control for demographic and childhood risk factors in analysis.

This study contributes to the literature in several ways. First, we provide important theoretical and empirical contributions to an ongoing discussion in the literature as to how emotional functioning, health, and SES may be jointly associated. Also, where prospective information is often unavailable, this study examines associations across a significant portion of life, with childhood factors measured at age 7 and CRP measured 35 years later. Finally, rather than relying on a single indicator to estimate the effects of a dynamic socioeconomic environment, this study considers the differential contributions of education, income, and occupation in the association between child emotional functioning and adult inflammation.

Method

Sample

The study sample comes from the New England Family Study (NEFS), a longitudinal investigation of participants from the Boston, Massachusetts and Providence, Rhode Island sites of the Collaborative Perinatal Project (CPP). Pregnant women enrolled in the CPP between 1959 and 1966 (Broman, Nichols, & Kennedy, 1975; Niswander & Gordon, 1972). The original aims of the CPP were to identify the neurodevelopmental consequences of pregnancy and delivery complications. Women were enrolled during pregnancy, and their offspring were regularly assessed from birth–7 years. Detailed medical and social histories were obtained from mothers at the time of enrollment. Information on birth outcomes, growth, and development, including psychologist ratings of child emotion, were obtained several times during the first year of life, and again at age 7 years.

The NEFS was established to locate and interview the now adult CPP offspring from the Providence and Boston sites. The current sample comes from a recent project examining the pathways linking education and health. Participants were sampled from a larger NEFS study ($N = 1674$). From this pool, there were 914 participants selected, with preference for racial/ethnic minorities and low or high educational attainment, of which 898 were eligible (e.g., living, not incarcerated), and 618 participated. Participants participated in a 3-hr interview in which informed consent, SES, psychological, and health information were collected. Participants provided a blood sample, and anthropomorphic measurements were obtained by trained study personnel. Human subjects committees at the Harvard School of Public Health and Brown University approved the study protocol.

Of the 618 participants interviewed, we excluded 42 participants who were not interviewed in person (and did not complete physiological assessments, including blood samples and anthropometry measures), resulting in 576 eligible participants. Of these, 430 participants provided a blood sample for assay, and 416 had available CRP data. Participants were more likely to be white and have higher education than eligible nonparticipants. There were no significant

differences by race, gender, or education according to whether or not participants provided a blood sample, although those who provided a sample were significantly younger ($M = 0.9$ years). Individuals with plasma CRP levels > 10 mg/L were removed from the sample ($n = 16$), as such levels can be indicative of acute illness or infection (Pearson et al., 2003). In multivariate analyses, sample sizes per model ranged from 388 to 368 depending on the number of covariates included and individuals missing data on those covariates.

Measures

Child emotional functioning. When the CPP went into the field in the late 1950s, no standard measures of child emotional functioning existed for population-based research. Measures of child emotional functioning were derived from psychologist observations of child behavior at the age-7 assessment and included inappropriate-self regulation ($\alpha = .71$) and distress proneness ($\alpha = .50$). Past work has documented the validity of these scales against a contemporary gold standard for child behavior and emotion (Achenbach Child Behavior Checklist; CBCL; Achenbach, 1991) and has found that these scales predict poor emotional functioning in adulthood (Kubzansky, Martin, & Buka, 2004). Detail on scale construction can be found elsewhere (Kubzansky et al., 2004).

Inappropriate self-regulation (ISR) reflects emotional functioning in children whose behavior was unrestrained and impulsive. Distress proneness reflects emotional functioning in children who were emotionally labile and easily frustrated. Both scales positively correlate with CBCL-assessed externalizing problems (Kubzansky et al., 2004), and can be considered domains of externalizing behavior. Scale scores were standardized ($M = 0$, $SD = 1$). Following other work in this area (Gortmaker, Kagen, Caspi, & Silva, 1997; Kubzansky et al., 2004), ISR scores were dichotomized with the top 15% as the indicator category. This cut-off point reflects high-risk individuals with poor emotional functioning. Theory and empirical work by Kagan and colleagues (Woodward, Lenzenweger, Kagan, Snidman, & Arcus, 2000) suggest emotional functioning is not necessarily a continuous construct; domains of emotionality can be grouped into meaningful subgroups at the extreme ends of the distribution. Given this theoretical perspective and because distributions were skewed, yielding fewer people with emotional dysfunction, scale scores were dichotomized. For distress proneness, the distribution was truncated with only the top 3% available for categorization. Given the lower reliability and truncated distribution, results for distress proneness should be interpreted cautiously. Distress proneness is positively correlated with ISR and both are domains of externalizing problems (Kubzansky et al., 2004). Therefore, results for distress proneness can be viewed as underscoring ISR findings. Also, emotion was examined continuously and with fewer extreme cut-off points. Associations with CRP and SES were similar, though less robust. Given the similar pattern of results, and that we wanted to identify a high-risk population, we present results for child emotion using the aforementioned cut-off points. CRP associations for continuous ISR and distress proneness can be found elsewhere (Appleton et al., 2011).

C-reactive protein (CRP). CRP was the outcome variable. The concentration of CRP was determined using an immunoturbidimetric assay on the Hitachi 917 analyzer (Roche Diagnostics; Indianapolis, IN), using reagents and calibrators from DiaSorin (Stillwater, MN). This assay has a sensitivity of 0.03 mg/L. The day-to-day variabilities of the assay at concentrations of 0.91, 3.07 and 13.38 mg/L are 2.81, 1.61 and 1.1%, respectively. In this sample, CRP levels range from 0.07 mg/L to 80.10 mg/L. Those with CRP levels > 10 mg/L were removed ($n = 16$), as such levels may indicate acute illness or infection (Pearson et al., 2003).

Childhood socioeconomic status. Three domains of childhood SES were examined to broadly assess the role of SES in child emotional functioning and adult CRP associations. Parental education was the highest level of education in the household reported by either parent at the age-7 study visit and was categorized as less than high school, high school graduate, and more than high school. This categorization enabled examination of associations across three general levels of education attainment. Childhood household income reflects the total annual household income as reported by the caregiver at the age-7 study visit and was categorized in tertiles: low (< \$5,500), middle (\$5,500–\$8,500) and high (> \$8,500). Tertiles were constructed to enable assessment of child emotion and adult CRP associations across three broad levels of childhood household income. To aid in interpretation, income categories were converted to 2010 dollars using the Consumer Price Index (Williamson, 2011): low < \$36,900, middle \$36,900–\$57,100, and high > \$57,100. Parental occupation reflects the highest occupation level in the household. Both caregivers reported their current occupation at the age-7 visit, which was grouped into four categories: not in the labor force, welfare, manual occupation, and nonmanual occupation. The highest reported occupation grade of the household was used. Due to few responses, not in the labor force, welfare, and manual occupation were combined as the manual group.

Covariates. Demographic and early-life factors that may confound the child emotion and adult CRP associations were controlled for in analysis. Several child health factors were controlled for, as poor early-life physical health may contribute to worse child emotional functioning and also to higher adult CRP. Also, poor child health and IQ are associated with child SES and adult health (Shonkoff & Phillips, 2000). Therefore, we examined the role of SES in child emotion and adult CRP associations, net of early-life co-occurring risks.

Demographics included age at adult follow-up, gender, race (white, not white), and original CPP study site (Boston, MA; Providence, RI). Small for gestational age (SGA) was calculated as whether or not the child's birth weight obtained at birth was less than or equal to the 10th percentile for gestational age at delivery compared to those of the same gestational age within the same CPP study site. BMI at age 7 was calculated as the ratio of weight in kilograms to the square of height in meters (kg/m^2). Weight and height were measured by study personnel at a scheduled visit with standard clinical instruments. Child IQ was assessed at age 7 using the Wechsler Intelligence Scale for Children (Wechsler, 1949). Scores were standardized to have $M = 100$, $SD = 15$. Child health was a summary measure indicating whether or not the child experienced one or more chronic physical health conditions from birth–age 7 as identified by a study pediatrician or maternal report. Categories of conditions included cardiovascular, hematologic (e.g., anemia), abnormalities of the liver, lower respiratory tract (e.g., asthma), neoplastic disease, neurologic abnormality, and prolonged/recurrent hospitalization.

Adulthood factors. Several adulthood factors were included, as past work has documented associations with CRP (Howren, Lamkin, & Suls, 2009; Pearson et al., 2003; Pollitt et al., 2007). Adulthood

BMI was calculated as kg/m^2 using height and weight measurements obtained by study personnel. Depressive symptoms were assessed with the Center for Epidemiologic Studies of Depression scale (CESD; Radloff, 1977; $\alpha = .88$). Education was assessed as total years attained. Current smoker was self-reported yes or no.

Statistical Analyses

Means and frequencies for each variable were generated. Bivariate associations between early-life SES, child emotion, adult CRP and other characteristics were evaluated via independent *t*, chi-square, and ANOVA tests. The effect-mediation hypothesis was evaluated via coefficient changes according to methods described by Baron and Kenny (1986). To test the hypothesis that the association of child emotional functioning and adult inflammation was modified by early-life SES, a series of multiple linear regression models were built. The first model displays the association of child emotional functioning with adult CRP, controlling for demographic and childhood covariates without an early-life SES variable in the model. Three models for each emotion measure were subsequently fit that additionally included early-life SES factors and corresponding interaction terms (emotion × SES). For distress proneness, interactions were fit using dichotomous and continuous emotion measures. Evidence for effect modification was evaluated via significance levels of interaction terms in these models as well as by examination of effects when the first model is stratified by each SES factor. Also, effect sizes were estimated for those with poor emotional functioning and low SES compared with similarly functioning children with higher SES, via mean differences in CRP and Cohen's *d*. Finally, for marginal or significant interactions between child SES and emotion, adulthood factors were added to the model and mediation of the modified effect by adulthood factors was evaluated via coefficient changes (Baron & Kenny, 1986). Models' variance estimates were adjusted for the presence of multiple siblings from the same family in the sample. A study site variable was included as a covariate in all models to adjust for potential differences due to original CPP study location. Statistical significance was determined by *p* values less than 0.05.

Results

Descriptive Statistics and Bivariate Associations

Table 1 summarizes the distribution and bivariate relations of characteristics across levels of child emotional functioning. At the adult follow-up, the sample was on average 42 years old, 80% white, 59% female and had an average CRP level of 1.72 mg/L. At age 7, 33% lived in homes where the highest level of parental

Table 1
Distribution and Bivariate Associations of Childhood Emotional Functioning With Study Variables

	Inappropriate self-regulation			Distress proneness		
Variable	High ($n = 64$)	Low ($n = 336$)	*p*	High ($n = 13$)	Low ($n = 387$)	*p*
Age, mean (*SD*)	42.1 (1.7)	42.2 (1.8)		41.5 (1.2)	42.3 (1.8)	
Race						
Not white, % (*n*)	18.7 (12)	21.0 (70)		23.1 (3)	20.5 (79)	
White, % (*n*)	81.3 (52)	79.0 (264)		76.9 (10)	79.5 (306)	
Gender						
Female, % (*n*)	43.8 (28)	61.3 (206)	**	30.8 (4)	59.4 (230)	*
Male, % (*n*)	56.3 (36)	38.7 (130)		69.2 (9)	40.6 (157)	
Birthweight						
SGA, % (*n*)	10.9 (7)	10.2 (34)		7.7 (1)	10.4 (40)	
Not SGA, % (*n*)	89.1 (57)	89.8 (300)		92.3 (12)	89.6 (345)	
Child health						
Chronic condition present, % (*n*)	17.2 (11)	18.2 (61)		15.4 (2)	18.1 (70)	
No chronic condition, % (*n*)	82.8 (53)	81.8 (274)		84.6 (11)	81.9 (316)	
Child IQ, mean (*SD*)	107.3 (14.0)	100.5 (13.4)	***	106.2 (13.3)	101.4 (13.7)	
Child BMI, mean (*SD*)	16.6 (1.7)	15.9 (1.6)	**	15.9 (1.1)	16.1 (1.6)	
Parental education						
Less than high school, % (*n*)	17.5 (11)	36.3 (119)		30.8 (4)	33.3 (126)	
High school graduate, % (*n*)	50.8 (32)	46.3 (152)	**	38.5 (5)	47.4 (179)	
More than high school, % (*n*)	31.2 (20)	17.4 (57)		30.8 (4)	19.3 (73)	
Child household income						
Low, % (*n*)	25.4 (15)	35.1 (111)		38.5 (5)	33.4 (121)	
Middle, % (*n*)	28.8 (17)	32.9 (104)		23.1 (3)	32.6 (118)	
High, % (*n*)	45.8 (27)	32.0 (101)		38.5 (5)	34.0 (123)	
Parental occupation						
Manual, % (*n*)	32.8 (21)	58.3 (193)	***	38.5 (5)	54.7 (209)	
Non-manual, % (*n*)	67.2 (43)	41.7 (138)		61.5 (8)	45.3 (173)	
Adult C-reactive protein, mean (*SD*)	2.33 (2.4)	1.61 (1.9)	*	2.87 (3.1)	1.69 (1.96)	*
Adult BMI, mean (*SD*)	32.0 (9.8)	28.7 (7.9)	*	32.9 (6.7)	29.1 (7.6)	+
Adult depressive symptoms, mean (*SD*)	1.5 (0.46)	1.6 (0.56)		1.5 (0.48)	1.6 (0.55)	
Adult education attainment, mean (*SD*)	14.4 (3.0)	13.4 (2.5)	*	13.3 (2.5)	13.6 (2.6)	
Adult current smoker, % (*n*)	22.2 (14)	26.5 (88)		30.8 (4)	25.7 (98)	

Note. Cell entries are % (*n*)/means (*SD*) for categorical/continuous variables; *p*-value corresponds to χ^2/ t-tests; SGA = small for gestational age; BMI = body-mass index; IQ = intelligence quotient.
$^{+}p < .10$. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

occupation was less than high school and 54% had a parent with a manual occupation. Significantly higher levels of CRP were observed for those high in childhood ISR and distress proneness. CRP was significantly associated with parental education at age 7, $F(390) = 3.88$, $p = .02$, marginally associated with household income, $F(2, 374) = 2.57$, $p = .08$, but not associated with parental occupation, $t(393) = -1.16$, $p = .25$.

Evaluating Evidence for Effect Mediation

For evidence of mediation following Baron and Kenny (1986), an association between child SES and adult CRP must first be demonstrated in the absence of child emotional functioning (the hypothesized mediator). If such a relation is demonstrated, additional models including the hypothesized mediator would be constructed, and attenuation of the child SES coefficient predicting adult CRP, with child emotional functioning in the model, would suggest effect mediation (Baron & Kenny, 1986). Regressions were performed for CRP with three measures of child SES while controlling for all covariates (data not shown). In separate models, parental education, income, and occupation were not significantly associated with adulthood CRP (p's > 0.10). As such, there was no evidence to pursue further mediation analyses with these factors. Similar results were observed when using alternative methods for evaluating mediation (e.g., product of coefficients; MacKinnon, Fairchild, & Fritz, 2007).

Evaluating Evidence for Effect Modification

Testing for interactions. Table 2 summarizes the main effects and interactions for child ISR and early-life SES with adult CRP. We observed a significant interaction for low-income and ISR problems, marginally significant interactions for low and middle levels of parental education with ISR, and a marginally significant interaction for manual occupation and ISR. Table 3 summarizes the main effects and interactions for childhood-distress proneness and early-life SES with CRP in adulthood. There was a marginal interaction for low-parental education and distress proneness, and a significant interaction for middle levels of parental education and distress proneness. We observed a significant interaction between distress proneness and low- and middle-income levels, and no significant interaction with occupation. Interactions with continuously measured distress proneness underscore primary findings: a marginal interaction with low-parental education ($b = 0.78$, $SE = 042$, $p = .07$), a trend for middle levels of education ($b = 1.00$, $SE = 0.63$, $p = .11$), a marginally significant interaction for low income ($b = 0.71$, se $= 0.42$, $p = .09$), a suggestion of a trend for middle income ($b = 1.35$, $SE = 1.0$, $p = .18$), and no occupation interaction.

Stratified models. Mean CRP levels for ISR stratified by SES factors are depicted in Figure 2. For each child SES indicator, those with ISR problems who also experienced low SES as children had significantly higher CRP as adults. A similar pattern was observed for distress proneness, but stratified findings were not depicted in the figure due to small cell sizes.

Effect size. ISR children from low SES environments had 1.2–1.8 mg/L higher CRP as adults compared with similarly functioning children from higher SES environments (d's $= 0.41$, 0.47, 0.66 for occupation, parental education, and household income, respectively). Likewise, distress-prone children from low-education and low-income environments had 1.0–2.3 mg/L higher adulthood CRP compared with distress-prone children living in higher-education and higher-income environments (d's $= 0.32$, 0.76, respectively). Conversely, distress-prone children whose parents worked a manual occupation had 0.81 mg/L lower adulthood CRP than similarly functioning children whose parents were nonmanual workers ($d = 0.27$). Mean CRP concentrations for poor emotional functioning and low-SES groups were largely greater than 3 mg/L, which suggest significant cardiovascular disease (CVD) risk (Pearson et al., 2003).

Adulthood mediators of child SES-modified associations. Addition of the adulthood factors to the models predicting CRP attenuated the observed interactions for ISR and parental education and ISR and manual occupation (see Table 4). BMI was the only adulthood factor significantly associated with CRP. In separate

Table 2
Multiple Linear Regression Models Examining Effect Modification by Early-Life Socioeconomic Status in the Association of High Versus Low Levels of Childhood Inappropriate Self-Regulation and C-Reactive Protein in Adulthood

Variable	Model 1	Model 2	Model 2a	Model 3	Model 3a	Model 4	Model 4a
Intercept	0.80 (2.71)	−0.16 (2.80)	0.004 (2.78)	1.33 (2.81)	1.24 (2.78)	0.19 (2.77)	0.74 (2.78)
High ISR (versus low)	0.81** (0.28)	0.88* (0.29)	0.02 (0.51)	0.91** (0.29)	0.41 (0.43)	0.85** (0.28)	0.48 (0.35)
Parental low education		0.54+ (0.32)	0.27 (0.34)				
Parental middle education		0.01 (0.28)	−0.26 (0.31)				
High ISR × parental low education			1.44+(0.80)				
High ISR × parental middle education			1.13+ (0.64)				
Low income				0.48+ (0.28)	0.19 (0.31)		
Middle income				0.29 (0.26)	0.22 (0.28)		
High ISR × Low income					1.67* (0.70)		
High ISR × Middle income					0.21 (0.67)		
Manual occupation						0.26 (0.21)	0.09 (0.23)
High ISR × Manual occupation							1.05+ (0.58)

Note. Models control for site, age, race, gender, SGA, child BMI, child IQ, and child health; high education (more than high school) and the high income tertile are the reference categories for education and income analyses; cell entries are b (SE); $n = 64$ for high inappropriate self regulation; ISR = inappropriate self regulation.
+ $p < .10$. * $p < .05$. ** $p < .01$.

Table 3
Multiple Linear Regression Models Examining Effect Modification by Early-Life Socioeconomic Status in the Association of High Versus Low Levels of Childhood Distress Proneness and C-Reactive Protein in Adulthood

Variable	Model 1	Model 2	Model 2a	Model 3	Model 3a	Model 4	Model 4a
Intercept	0.22 (2.73)	−0.59 (2.82)	−0.59 (2.79)	0.71 (2.83)	0.64 (2.81)	−0.31 (2.79)	−0.43 (2.79)
High DP (versus low)	1.26* (0.56)	1.23* (0.56)	−1.01 (1.02)	1.24* (0.56)	−0.78 (0.90)	1.29* (0.57)	1.69* (0.72)
Parental low education		0.46 (0.31)	0.33 (0.32)				
Parental middle education		−0.03 (0.28)	−0.21 (0.29)				
High DP × Parental low education			2.47+ (1.43)				
High DP × Parental middle education			3.79** (1.35)				
Low income				0.44 (0.28)	0.30 (0.29)		
Middle income				0.24 (0.26)	0.11 (0.26)		
High DP × Low income					3.14* (1.27)		
High DP × middle income					3.52* (1.46)		
Manual occupation						0.20 (0.21)	0.23 (0.22)
High DP × Manual occupation							−1.04 (1.15)

Note. Models control for site, age, race, gender, SGA, child BMI, child IQ, and child health; high education (more than high school) and the high income tertile are the reference categories for education and income analyses; cell entries are *b (SE); n* = 13 for high distress proneness; DP = distress proneness.
+ $p < .10$. * $p < .05$. ** $p < .01$.

models, ISR ($b = 3.04$, $SE = 1.23$, $p = .01$), low parental education ($b = 3.32$, $SE = 1.05$, $p = .002$) and manual occupation ($b = 1.24$, $SE = 0.77$, $p = .10$) predicted adulthood BMI. These associations suggest that adulthood BMI may mediate the parental education and occupation-modified effects of ISR on CRP. Although the coefficients for ISR and income, and distress proneness with income and education were somewhat reduced with the addition of the adulthood factors to the model, the interactions remained significant. While adulthood BMI helped to explain the ISR interactions with two of the three SES factors, no adulthood factor explained the ISR and income interaction or the distress proneness findings.

Discussion

This study tested whether the association of child emotional functioning and adult CRP were modified by early-life SES, or whether child emotional functioning might mediate the association of early-life SES and adult CRP. We found evidence in support of the effect modification hypothesis, but not for mediation. Study findings suggest that children with highly reactive emotional styles who grow up in low SES environments may have greater inflammatory risk as adults than do similarly functioning children who grow up in higher SES environments. It is possible that physical and emotional resources available in high SES environments buf-

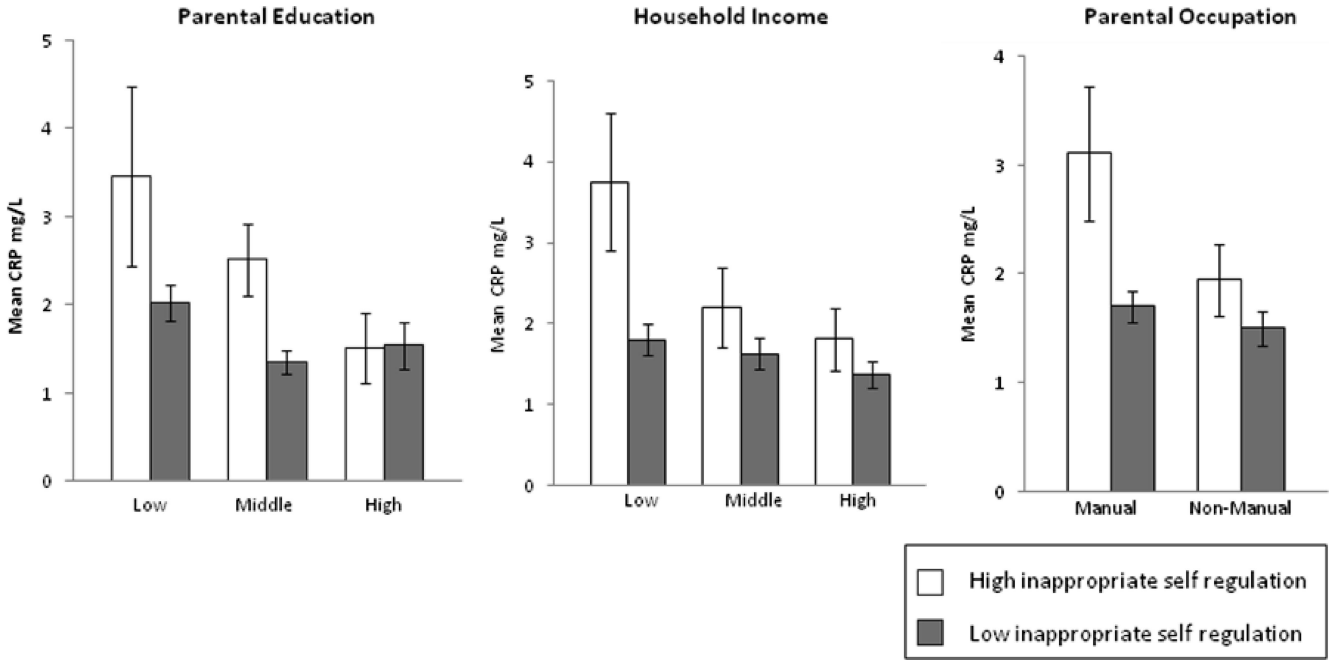

Figure 2. Mean CRP in adulthood by level of inappropriate self regulation and socioeconomic status at age 7 years.

Table 4
Multiple Linear Regression Models Examining Potential Adulthood Mediators of The SES-Modified Associations of Childhood Emotional Functioning and Adult C-Reactive Protein

Variable	High inappropriate self regulation			High distress proneness		
Emotion × low education	0.71 (0.71)			2.39* (1.22)		
Emotion × middle education	0.25 (0.56)			2.40* (1.16)		
Emotion × low income		1.49* (0.62)			2.26* (1.10)	
Emotion × middle income		0.15 (0.59)			1.72 (1.26)	
Emotion × manual occupation			0.42 (0.51)			−0.37 (0.18)
Body-mass index	0.14*** (0.01)	0.14*** (0.01)	0.14*** (0.01)	0.14*** (0.01)	0.14*** (0.01)	0.14*** (0.02)
Education attainment	−0.01 (0.04)	0.03 (0.04)	0.006 (0.04)	−0.005 (0.04)	0.02 (0.04)	0.01 (0.04)
Depressive symptoms	0.04 (0.17)	0.12 (0.17)	0.07 (0.17)	0.04 (0.17)	0.09 (0.17)	0.06 (0.18)
Smoker	0.11 (0.21)	0.10 (0.11)	0.11 (0.21)	0.08 (0.21)	0.06 (0.21)	0.12 (0.22)

Note. Models control for site, age, race, gender, SGA, child BMI, child IQ, child health and the applicable SES and emotion main effects; high education (more than high school) and the high-income tertile are the reference categories for parental education and income analyses; cell entries are *b (SE);* $n = 64$ for high inappropriate self regulation; $n = 13$ for high distress proneness.
* $p < .05$. *** $p < .001$.

fer the deleterious effects of poor emotional functioning, whereas the lack of such resources in low SES environments may exacerbate risk over the life course. These findings are congruent with a growing body of research suggesting that health disparities may have roots in childhood. Moreover, effect sizes were moderate, and mean adult CRP concentrations for those with poor child emotional functioning and low SES were largely greater than 3 mg/L, which is the CDC/American Heart Association's cutoff-point for high risk of CVD (Pearson et al., 2003).

While the magnitude of the interactions varied somewhat, the pattern of relationships was largely consistent and does not suggest that one aspect of SES is more important than another. Two exceptions should be noted. First, distress-prone children with parents working nonmanual occupations (high SES category) had higher CRP. In this sample, the distress-prone group was small ($n = 5$ manual, $n = 8$ nonmanual). This association could be attributable to distribution problems and sample size, rather than indicating high parental SES occupations confers inflammation risk for distress-prone children. Also, while the interactions for ISR and parental education were marginally significant, ISR occurs more frequently among higher levels of parental education. This may make interactions with low levels of education difficult to detect, and education findings difficult to generalize to a minority of those with ISR problems. Future research should examine these relations with larger samples and better distributions of emotion and SES.

We found some evidence that adulthood BMI mediated the parental education- and occupation-modified effects of ISR on CRP. These findings are consistent with our previous study where the association of ISR and CRP was mediated by adult BMI (Appleton et al., 2011). However, adult BMI did not explain other emotion and SES interactions despite being the only adult factor associated with CRP. SES is a multidimensional construct, with each factor influencing health at different times in the life course and through different pathways (Braveman et al., 2005). As such, the independent interaction effects between ISR and income, distress proneness and education and income on CRP may be attributable to setting in motion a range of risk trajectories not examined here. For example, high and low levels of parental education are differentially associated with the development of effective emotion regulation strategies (Shonkoff & Phillips, 2000). Reactive children with highly educated parents may learn effective emotion-regulation skills to tamp down physiological activation associated with emotional arousal, whereas reactive children in low-education environments may not develop such skills. As emotion-regulation strategies tend to be used over the life course (Shonkoff & Phillips, 2000), developing and employing skills to effectively manage emotions may serve as a buffer where the absence of such skills may contribute to inflammatory risk. In contrast, household income may modify the child emotion-adult inflammation association through access to absolute resources. For example, low-income children with poor emotional functioning may lack access to nutritious foods due to cost. As child nutritional deprivation is associated with impeded brain development, behavior problems, and poor physical health (Shonkoff & Phillips, 2000), household income may modify emotion and CRP associations by way of access to healthy foods. Moreover, continued lack of access may lead to the development of poor dietary patterns which may affect inflammation over time. Finally, manual occupations are often characterized by jobs with high psychological demands and low decision latitude, which have significant mental and physical health risks for workers (Karasek, 1979), including higher risk of depression and poor physical health (D'Souza, et al., 2005). Such poor parental health may exacerbate a reactive child's already poor emotional functioning while also inhibiting the parent's ability to obtain supportive services for the child in need. We advise future work to consider the full range of pathways through which SES-modified associations with emotional functioning affects adulthood CRP.

This study has several limitations. When the CPP went into the field in the late 1950s, there were no population-based research tools to assess child emotion. Though the scales used in this study perform moderately well, contemporary measures such as the Achenbach Child Behavior Checklist (Achenbach, 1991) may more accurately assess child emotion. Additionally, the poor distribution of distress proneness and low alpha raise concerns as to the reliability of the distress proneness findings, which should be interpreted cautiously. Also, child SES was examined only once during childhood, leading to potential misclassification. Some may have had low status for a short period of time as parents perhaps

went back to school, obtained new jobs and made more money. Such misclassification would bias estimates toward the null. Also, we did not have a measure of child CRP available to rule out potential reverse causation between early inflammatory processes and child emotional functioning. However, we were able to take account of a comprehensive measure of child health as well as BMI, somewhat mitigating this concern. Finally, though the statistical evidence suggests child SES modifies the child emotion-adult CRP association, the observational study design precludes conclusions of causality.

This study has a number of strengths. First, we examined a biomarker outcome, which is not subject to reporting biases and provides insight into physiological mechanisms through which emotions may influence health. Additionally, emotional functioning and SES were assessed 35 years prior to CRP. Such a lengthy follow up allows for the examination of markers of health risk that emerge in mid life. Also, study variables were collected from multiple sources (e.g., biomarker, psychologist), thereby protecting against common method bias that could result when relying on a single mode of data collection. Finally, this study examined multiple measures of SES measured during childhood. Doing so allows for a broad examination of the role early-life SES plays in the child emotion-adult CRP association.

Evidence is accumulating that some adulthood diseases have developmental origins early in life, particularly in relation to childhood psychosocial adversity (Shonkoff et al., 2009). This study adds to that growing literature and describes one way in which adverse social and psychological environments during childhood may "get inside the body" and affect disease risk decades later. These findings suggest that the benefits of improving the standard of living and mental health of children may not be limited to childhood psychosocial well being. Such benefits could potentially confer a lifetime of physical health protection as well.

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Correction to Ramo, Hall, and Prochaska (2011)

In the article "Reliability and Validity of Self-Reported Smoking in an Anonymous Online Survey With Young Adults" by Danielle E. Ramo, Sharon M. Hall, and Judith J. Prochaska (*Health Psychology*, 2011, Vol. 30, No. 6, pp. 693–701), the below funding information was omitted from the author note.

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