Socioeconomic status inequalities in low-grade inflammation during childhood

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ABSTRACT

Background Family socioeconomic status (SES) is an important source of child health disparities in the USA. Chronic stress is one way SES may impact children's physiology with implications for later health inequalities. These processes may work differently across childhood due to differences in exposure and susceptibility to stressors at different ages. We assess associations between family SES and one biomarker of chronic stress exposure—low-grade inflammation detected by elevated C reactive protein (CRP)—and evaluate differences in the associations by child age.

Methods We used nationally representative data from the National Health and Nutrition Examination Survey and Tobit regression models to estimate SES associations with CRP and the moderating effects of age for children age 2–18 years. Our sample was limited to CRP ≤10 mg/l to focus on low-grade inflammation (N=13 165).

Results Children whose parent had less than a high school degree had 35% higher CRP than those with a college graduate parent; and, poor children had 24% higher CRP than those with high family income, net of controls. When children's body mass index was accounted for, low education and poverty associations were reduced to 19% and 15%, respectively. Child age interactions were negative and significant for both parental education and family income.

Conclusions This study provides new evidence that SES is associated with low-grade inflammation in children, and that these associations may be particularly strong during early and mid-childhood. Future research should further our understanding of stressors related to low family SES that may lead to immune system dysregulation during childhood.

What is already known on this topic?

- Chronic stress exposure can increase low-grade inflammation as measured by C reactive protein
- Low family socioeconomic status (SES) is one potential source of chronic stress exposure that may increase inflammation in children.

What this study adds?

- Low parent education and family income are associated with higher inflammatory marker concentrations in children.
- Much of the socioeconomic status (SES) associations are mediated by children's body mass index (BMI).
- Net of BMI and recent illness, the lowest SES categories are significantly associated with low-grade inflammation in children, with particularly strong effects in young children.

INTRODUCTION

A large body of literature documents socioeconomic status (SES) disparities in child health.[1 2] Researchers are now challenged with understanding how SES may 'get under the skin' to produce these disparities. One potentially important pathway from low SES to poor child health is through exposure to chronic stress and related biological changes.[3–6] Biological research suggests that exposure to chronic stress may result in dysregulation of the neuroendocrine and immune systems; and, these systems may be particularly sensitive during childhood.[7 8]

In this study we assess how family SES is associated with one biomarker of chronic stress exposure—low-grade inflammation[9]—in US children. Psychosocial stress may affect inflammation levels by stimulating the production of proinflammatory cytokines, which increase acute phase proteins (such as C reactive protein, CRP) circulating in the blood.[10] This inflammatory response may be further enhanced if repeated activation of the autonomic nervous system and hypothalamic-pituitary-adrenal axis due to chronic stress has resulted in glucocorticoid resistance; so that, immune cells lose their sensitivity to glucocorticoids that normally would stop the inflammatory process.[11] Chronic stress could thus result in long-term circulation of low levels of CRP in the blood, an indicator of low-grade, systemic inflammation. Low-grade inflammation has been associated with depression[12 13] and cardiovascular risk factors in children;[14 15] and, may increase the risk for adult inflammation and chronic disease.[16 17]

Our study builds on existing research[18] by using nationally representative data and multivariable regression analysis to assess how two measures of family SES—parental education and family income—are associated with low-grade inflammation (indicated by CRP) in children age 2–18 years. We consider the mediating role of body mass index (BMI) due to evidence that low family SES is associated with higher BMI in children,[19] and that adipose tissue can increase the production of CRP.[20] Importantly, we assess whether child age conditions the SES-CRP associations, given past findings that the SES-health gradient differs by child age.[21 22] To date, research on age as a modifier of SES-child health associations has relied on parental reports of child health, with little attention to biomarkers such as CRP.

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METHODS

Data

Our data come from the continuous National Health and Nutrition Examination Surveys (NHANES), a nationally representative data set on the health and nutrition of children and adults collected by the National Center for Health Statistics (NCHS) every 2 years. We use physical health exam and interview data across six waves spanning 1999–2010. Blood samples were collected from all participants >1 year in waves 2, 3 and 4; from participants >3 years in waves 1, 3 and 6; and from children age 3–5 and 12+ years in wave 5. In all waves an adult household respondent (a parent or guardian for children under 16 years) was interviewed to gather SES, child illness and household demographic information. We concatenated the survey waves to obtain stable estimates for children age 2–18 years. Each wave of NHANES was approved by the NCHS Research Ethics Review Board.

Sample

Our sample consists of children age 2–18 years who provided blood samples, excluding 1% of children with a household respondent who was <17 years older than the child (to ensure that the household respondent was not a sibling or the index child).[23] Due to our interest in low-grade inflammation as a response to chronic stress we limited our sample to children with CRP ≤10 mg/L.[9 24] Past research indicates that CRP values >10 mg/L likely reflect acute inflammation in response to an infection or injury and should not be considered in studies of low-grade inflammation.[25]

CRP was missing for 25% of our sample; however, we found no substantial difference in child or family characteristics between those with and without CRP, with the exception of child age. Children missing CRP were younger than those in our sample (mean of 7 years vs 11 years). This may be due to NHANES age restrictions in some waves and higher refusal rates for blood collection in younger children. In our analyses we apply survey weights to account for the non-response.

Our sample includes children with undetectable CRP levels, indicated in NHANES as <0.1 mg/L. These cases are considered censored (non-missing, but unknown true value) and require Tobit regression analysis. Since Tobit models cannot be estimated with imputed data, we dropped cases missing independent variable data (19%), resulting in a final sample of 13 165 children. Median CRP for missing cases was similar to our sample median, 0.4 and 0.3, respectively. The top quarter of the sample had a CRP range of 1.2–9.7 among those missing and 1.0–10 for those not missing independent variable data.

Measures

Our outcome variable is a continuous measure of CRP in mg/L. For regression analyses, we logged CRP (base 10) due to its skewed distribution.

We measured family SES as two categorical variables: parental education and family income-to-poverty ratio (IPR). Household respondents provided their education level as <high school degree, high school graduate, some college or associates degree, and graduated college (reference category in regression models). We refer to this as 'parental' education because for 83% of the sample children (those under age 16 years) a parent/guardian was the household respondent. While we could not confirm relationship of the household respondent for the 16–18 year olds, limiting the sample to children whose household respondent was ≥17 years older than the child ensured that the respondent was likely a parent or guardian. Household respondents over age 60 (possible grandparent) occurred in 4% of the sample cases—the findings were robust to models excluding these cases.

Our second SES measure was family IPR, calculated as total family income divided by the federal poverty level (FPL) for a given family size and year, capped at 5×FPL by NHANES. We created the following categories: poor (<1×FPL), low income (1–2×FPL), moderate income (2.1–3.9×FPL), and high income (≥4×FPL) (reference group in regression models).

Statistical methods

We weighted both the descriptive statistics and regression analyses with individual medical exam weights computed for the 12-year period. Our multivariable analyses used Tobit regression of log CRP on family SES. Tobit models are estimated with maximum likelihood and take into account the censored nature of our dependent variable.[26]

Our first regression model included the family SES variables and all control variables. Child-level controls were: age, illness (recent cold, infection or stomach illness), gender (male=1) and race (black, Hispanic, other race vs non-Hispanic white). Household respondent (parent) controls included: age, gender, US born, and marital status (single, cohabiting, separated/divorced, and widowed vs married). Household size and dummies for NHANES survey waves were also included.

Our second model added child BMI z-scores, calculated using measured height and weight and 2000 for Disease Control and Prevention growth charts as the reference population. We entered BMI z-scores sequentially to assess changes in our SES associations when controlling for BMI, which along with supplemental analyses, allowed us to evaluate potential mediation of family SES by child BMI. Our final models included interaction terms between child age and the SES variables to test for moderating effects of child age. All analyses were performed using Stata V.13 and significance was assessed through two-tailed z-tests of $p<0.05$.

RESULTS

Key characteristics of the sample children are shown in table 1. Median CRP was 0.3 mg/L. Median child age was 10 years and 38% had a recent illness, with colds being the most common. The sample median BMI z-score was 0.4, just over half the sample was male and 61% of the children were reported as non-Hispanic white.

The SES measures indicated that 44% of children had a parent with a high school degree or less, 30% had some college or an associate's degree, and 26% had a bachelor's degree or higher. Approximately a fifth of children lived below the poverty line, 24% were low income (1–2×FPL), 29% moderate income (2.1–3.9×FPL) and 26% were in high-income families.

Table 2 provides the regression results (not all controls were shown for brevity). Since our dependent variable is logged, the coefficients are interpreted as per cent increase in CRP between the SES category of interest and the reference category. Results from model 1 indicated strong associations between low education and family income with log CRP. Children whose parent had a lower high school degree had 35% higher CRP than those with a college graduate parent, net of control variables. Parent education levels of high school degree and some college were associated with 29% and 15% higher child CRP, respectively, than children with a college graduate parent. Children living in poverty had, on average, 24% higher CRP than those in high-income families. Children in families with low and

Table 1 Weighted descriptive statistics

Variable	Median	Per cent	Min.	Max.
C reactive protein (mg/L)*	0.3		0.1	10
Family socioeconomic status				
Parental education				
Less than high school degree		20	0	1
High school degree		24	0	1
Some college		30	0	1
College graduate		26	0	1
Family income-to-poverty ratio†	2.3		0	5
Poor (<1×FPL)		21	0	1
Low income (1–2×FPL)		24	0	1
Moderate income (2.1–3.9×FPL)		29	0	1
High income (≥4×FPL)		26	0	1
Child characteristics				
Age in years	10		2	18
Recent head or chest cold		23	0	1
Recent influenza, pneumonia, ear infection		6	0	1
Recent stomach or intestinal illness		9	0	1
BMI z-score	0.4		−4.9	4.6
Male		52	0	1
Non-Hispanic white		61	0	1
Hispanic		19	0	1
African-American		13	0	1
Other race		7	0	1
Household respondent characteristics				
Age	40		20	80
Male		52	0	1
US born		81	0	1
Married		70	0	1
Cohabiting		4	0	1
Single		7	0	1
Divorced/separated		17	0	1
Widowed		2	0	1
Household size	4		2	7

Children age 2–18 years with CRP ≤10 mg/L, NHANES 1999–2010. N=13 165.
*Those missing CRP values did not differ from the analytical sample by family socioeconomic status, race/ethnicity or other social characteristics. Missing CRP children did have a significantly lower age (median 7 years) and slightly lower BMI z-score (median 0.3) than sample children.
†Capped at 5.
BMI, body mass index; CRP, C reactive protein; FPL, federal poverty line; NHANES, National Health and Nutrition Examination Surveys.

moderate income were estimated to have 14% and 18% higher CRP, respectively, than those in the highest family income category.

In model 2, we show the association between children's BMI z-scores and log CRP, and compare the results with model 1 to assess mediation of family SES by BMI. Results indicated a 63% increase in CRP with each point increase in BMI z-score (table 2, model 2). Including BMI z-score reduced the parent education coefficients, with a decrease from 0.35 to 0.19 for <high school degree, 0.29 to 0.17 for high school degree, and a now insignificant coefficient on parent with some college (see models 1 and 2). The family poverty effect declined from 0.24 to 0.15 when accounting for child BMI, while low-income and moderate-income coefficients became small and insignificant. Additional evidence of mediation was found in supplementary multivariate ordinary least square regression models (not shown here), which indicated significant positive associations between the education and income categories and BMI z-scores, net of control variables.

Finally, we assessed the moderating role of child age by including *child age×parent education* (model 3) and *child age×family IPR* (model 4) interaction terms. Results from both models indicated significant negative interaction effects, suggesting larger associations between family SES and log CRP in younger children. To illustrate the impact of the interaction effects, figure 1 shows the predicted per cent differences in CRP for children between low versus high SES categories by child age. Among the youngest children in the sample (age 2 years), having a parent with less than a low high school degree was associated with 52% higher CRP than for those with high parental education. Each additional year of age reduced the association by approximately 4%; so that, by age 13 years, the predicted per cent difference in CRP for children with low versus high parent education was reduced to insignificance. Also shown in figure 1, the youngest children had a predicted 50% higher CRP when living in poverty compared with similar children in high-income families. The poverty effect was reduced to insignificance by age 12 years.

DISCUSSION

In this study we assessed family SES inequalities in a biological marker of low-grade inflammation, elevated CRP, in a representative sample of US children. Our results indicated that both low parental education and family income were independently associated with higher child CRP net of potential confounders. We found that child BMI partially mediated the family SES associations; however, significant associations between the lower SES categories and CRP remained. Our child age-SES interaction effects, although somewhat small, provided evidence of strong associations between SES and CRP in early childhood even when controlling for BMI.

The strengths of our study include: assessment of two aspects of children's SES (parental education and family income), the use of regression techniques and inflammation parameters consistent with previous research for comparisons across studies, and the use of national-level data with children spanning the SES spectrum and full childhood age range. Further, our analysis of CRP levels ≤10 mg/L and controlling for children's recent illnesses allowed us to focus on the associations between SES and psychosocial stress-related inflammation. Finally, we provided a more nuanced understanding of SES-CRP associations, with evidence that SES works both through and apart from BMI, and new findings of child age as a moderator of SES-CRP associations.

The main limitation of our study is the lack of longitudinal data to assess how family SES may impact inflammation as children age, and whether the effects are long term. Cross-sectional data also limit the study to associational rather than causal findings. Further, we were unable to assess SES-related psychosocial stressors that may explain our findings.

Despite these limitations, this study increases our understanding of SES as an aspect of children's family contexts that may induce inflammation related to chronic stress exposure. Our income findings corroborate with another national-level study that found significant positive associations between family income and inflammation in children 3–16 years.[18] Our results for parental education are consistent with studies of older children in Los Angeles,[27] Ohio,[28] and 10-year olds in the UK.[29] Thus, we add to the growing literature that suggests low SES may be an important risk factor for low-grade inflammation in children.

The finding that children's BMI mediates the SES effect is also consistent with past studies of income[18 30] and education[29] effects on children's CRP. Findings across studies, including ours, indicate that low SES works to affect CRP, in part, through

Table 2 Tobit regression results of family SES associations with log CRP

	Model 1		Model 2		Model 3		Model 4	
Variables	**ß**	**95% CI**	**ß**	**95% CI**	**ß**	**95% CI**	**ß**	**95% CI**
Family SES								
Parent<high school degree*	0.35**	(0.21 to 0.50)	0.19**	(0.05 to 0.32)	0.60**	(0.28 to 0.93)	0.18**	(0.05 to 0.31)
Parent high school degree*	0.29**	(0.15 to 0.42)	0.17**	(0.05 to 0.30)	0.50**	(0.16 to 0.84)	0.17**	(0.04 to 0.29)
Parent some college*	0.15*	(0.023 to 0.27)	0.061	(−0.05 to 0.18)	0.36*	(0.04 to 0.68)	0.056	(−0.06 to 0.17)
Poor†	0.24**	(0.09 to 0.39)	0.15*	(0.01 to 0.28)	0.14*	(0.01 to 0.28)	0.58**	(0.25 to 0.91)
Low income†	0.14*	(0.002 to 0.28)	0.09	(−0.04 to 0.21)	0.09	(−0.04 to 0.21)	0.40*	(0.06 to 0.74)
Moderate income†	0.18**	(0.06 to 0.31)	0.11	(−0.01 to 0.23)	0.11	(−0.005 to 0.23)	0.19	(−0.16 to 0.54)
Child age and health								
Age in years	0.07**	(0.06 to 0.08)	0.05**	(0.04 to 0.06)	0.08**	(0.06 to 0.10)	0.070**	(0.05 to 0.09)
Recent cold	0.58**	(0.49 to 0.68)	0.60**	(0.51 to 0.69)	0.59**	(0.50 to 0.68)	0.59**	(0.50 to 0.68)
Recent influenza, pneumonia, ear infection	0.35**	(0.19 to 0.52)	0.21**	(0.05 to 0.36)	0.21**	(0.05 to 0.36)	0.20**	(0.05 to 0.36)
Recent stomach illness	0.14	(−0.01 to 0.28)	0.09	(−0.05 to 0.22)	0.09	(−0.06 to 0.22)	0.09	(−0.05 to 0.22)
BMI z-score			0.63**	(0.59 to 0.66)	0.63**	(0.59 to 0.66)	0.63**	(0.59 to 0.66)
Child age×family SES interactions								
Age×parent<high school degree					−0.04**	(−0.06 to −0.01)		
Age×parent high school degree					−0.03*	(−0.06 to −0.002)		
Age×parent some college					−0.03*	(−0.05 to −0.001)		
Age×poor							−0.04**	(−0.06 to −0.01)
Age×low income							−0.03*	(−0.05 to −0.001)
Age×moderate income							−0.01	(−0.03 to −0.02)

Children age 2–18 years with CRP ≤10 mg/L, NHANES 1999–2010. N=13 165.
*p<0.05, **p<0.01.
*Ref: Parent is a college graduate.
†Ref: High family income (income-to-poverty ratio ≥4×FPL).
Control variables included in models but not shown for brevity: child gender, child race, parent age, parent gender, parent foreign born, parent marital status, household size, survey wave dummy variables.
BMI, body mass index; CRP, C reactive protein; FPL, federal poverty level; NHANES, National Health and Nutrition Surveys; SES, socioeconomic status.

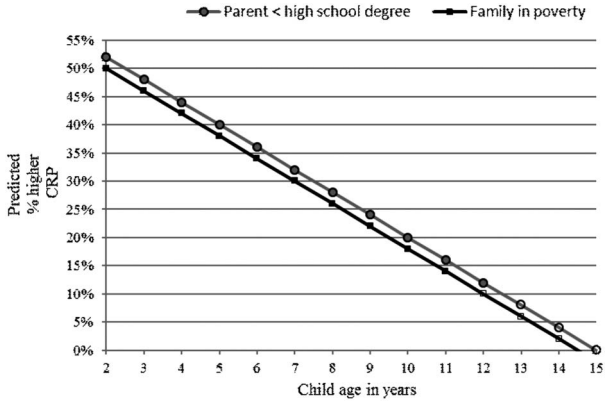

Figure 1 Associations between low family socioeconomic status and C reactive protein (CRP) by child age. Calculated based on results from models 3 and 4.

higher BMI in low-SES children. This may indicate another 'stress' pathway, whereby psychosocial stress in low-SES family contexts may produce biological effects that increase body fat.[31 32] However, our finding that the lowest SES categories had significant associations with CRP net of BMI suggests a direct link between low SES and inflammation. The child age interaction models, which included BMI z-scores, indicated that the associations between SES and low-grade inflammation may be particularly important in early childhood.

Our finding that child age moderated the SES-inflammation relationship is consistent with child health research indicating SES associations with other, non-biological measures of child health are stronger in early childhood.[33 34] Our study contradicts other research that finds family income effects on health to be more pronounced during adolescence.[21] It may be that low-grade inflammation, unlike global measures of parent-reported child health, is more sensitive to developmental stage rather than cumulative effects of children's social contexts. Since the moderation effect of child age has not been reported in other studies of SES and CRP, this remains to be further studied.

In addition to increasing our understanding of inequalities in inflammation during childhood, our findings have potentially important implications for adult health. Low-grade inflammation has been shown to increase the risk of cardiovascular and other diseases in adulthood.[35–38] A growing body of literature suggests that this process of disease onset due to chronic low-grade inflammation may begin during childhood.[15 39] Thus, it will be important to further explore low SES during childhood, and related stressors, as potential precursors to adult inflammation.

More research is needed in this area, with particular attention to how biosocial processes related to inflammation (and other biomarkers) unfold over time during childhood and continue into adulthood. The use of biomarkers in child health research allows for the detection of physiological changes that may not be perceptible to parents, uncovering inequalities in children's biological systems that may lay the foundation for later health disparities. The collection of longitudinal family, social and economic data, along with inflammation and other stress-related biomarkers, will be important for future research to improve our understanding of social contexts, psychosocial stress and health across the life course.

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Notes