Socioeconomic status and health: Do gradients differ within childhood and adolescence?

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Abstract

Socioeconomic status (SES) gradients may not be static across the lifespan, but instead may vary in strength across different life stages. This study examined the periods in childhood when SES and health relationships emerge and are strongest among US children. Data came from the National Health Interview Survey, 1994, a cross sectional, nationally representative sample of 33,911 US children ages 0–18. Parents were asked about family SES and child health status. Global health measures included overall ratings of child health, activity and school limitations. Acute conditions included childhood injuries and respiratory illnesses. For all global child health measures, lower family SES was associated with poorer child health in a gradient fashion (P < .001); these differences did not vary across age. For specific conditions, interaction effects of SES with age were found (P < .05). Interaction effects revealed that for injury and acute respiratory illness, expected SES gradients (lower SES with poorer outcomes) were evident during adolescence. In contrast, respiratory illness had a reverse SES gradient in early childhood. In sum, for global child health measures, associations of lower SES with poorer health throughout childhood suggest that factors that do not change with age (e.g., health care quality) may best explain overall health status. However, for acute conditions, the relationship between low SES and poor child health appears most consistently during adolescence. This suggests that normal development-related changes during adolescence, such as increasing peer group affiliation, may help explain these gradients. These patterns are important to understand for optimally timing interventions to reduce SES disparities in US children’s health.

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Introduction

Socioeconomic status (SES) has a profound association with physical health (see Marmot, Kogevinas, & Elston, 1987; Townsend & Davidson, 1982, for reviews). Individuals lower in SES experience higher rates of morbidity and mortality compared to individuals higher in SES, across many different health outcomes, and across numerous countries, both with and without universal health care (Adler et al., 1994; Adler, Boyce, Chesney, Folkman, & Syme, 1993). Understanding these relationships early in life is critical both for maximizing children’s health and for early prevention efforts to improve health across the lifespan. Children lower in SES also suffer from poorer physical and mental health outcomes compared to children higher in SES (Chen, Matthews, & Boyce, 2002; Duncan & Brooks-Gunn, 1997; Leventhal &
Brooks-Gunn, 2000; Pamuk, Makuc, Heck, Reuben, & Lochner, 1998; Starfield, Riley, Witt, & Robertson, 2002; Starfield, Robertson, & Riley, 2002). However, it remains unclear when socioeconomic gradients emerge in life, and when they have the strongest relationship with child health. These questions have important implications for optimally timing interventions to maximally improve child health in a cost-effective manner.

As has been proposed by a number of lifespan researchers, the relationship between SES and physical health may not be constant across age (Chen et al., 2002; House, Kessler, & Herzog, 1990; Power, Manor, & Matthews, 1999; West, 1997). For example, SES differences may be strong early in life, but weaker during adolescence, as schooling and peer influences lead to greater homogenization among adolescents (West, 1997; West & Sweeting, 2004). Alternatively, SES differences may be weak early in life, but stronger during adolescence and early adulthood as developmental changes occur or as factors accumulate that contribute to health disparities (Chen et al., 2002; Power & Hertzman, 1997). Finally, it is possible that SES differences in health may be similar in magnitude across childhood and adolescence (Chen et al., 2002). This could occur if factors unrelated to child development (e.g., health care availability) play an important role in children’s health.

Furthermore, one model may not capture child health gradients for all outcomes. Different health outcomes may be influenced by different factors, and thus certain models may apply better to certain health outcomes, depending on the timing of when important mediating factors come into play during childhood. Thus, although previous research has demonstrated convincing evidence for SES gradients with respect to child health outcomes, the question of whether and how SES gradients vary by age for different child health outcomes remains. The present study investigated whether SES and health relationships vary by age for (1) global health measures; and (2) specific common acute childhood conditions, among a nationally representative sample of US children. Global health measures reflect more stable indicators of health status (West & Sweeting, 2004), as well as parents’ beliefs about what constitutes good health. In contrast, specific acute conditions are time-limited and often rely on more concrete indicators (e.g., a specific illness episode that warranted medical attention).

We hypothesized that different SES and health relationships by age would emerge for different types of health outcomes. For example, global health status is likely associated with multiple factors, such as families’ access to medical care, parents’ beliefs about health, and persistent or chronic problems that the child has. These factors may not change as a function of child development, and together may have a similar magnitude of overall effect across ages. Thus we predicted that SES gradients for overall health measures would be similar across childhood and adolescence. On the other hand, acute illnesses may be associated with factors that are stronger during certain periods of time. For example, our earlier review paper found support for the notion that lower SES was associated with higher rates of injury during early childhood, but not during adolescence (Chen et al., 2002). With respect to acute respiratory illnesses (not covered in that review), some research has found that early childhood SES is associated with risk of respiratory illness in adulthood (Cohen, Doyle, Turner, Alper, & Skoner, 2004), and there is some suggestion that the difference in hospitalizations for acute respiratory conditions between immigrants and non-immigrants is greater for adolescents than younger children (Geyer, Peter, & Siegrist, 2002). Thus we hypothesized that the SES gradient might be stronger during adolescence for acute respiratory conditions.

Methods
Sample

Data are from the National Health Interview Survey (NHIS) 1994, which is a multistage, cross-sectional household interview survey of the civilian, non-institutionalized population of the United States, conducted by the National Center for Health Statistics. Data from children ages 0–18 were included in these analyses. In most cases, a proxy adult respondent (usually the child’s mother) was used for persons under 19 years old. For simplicity, we refer to these responses as parental report throughout the remainder of the manuscript. Data were extracted from 1994 because this sample contained more than twice as many children (n = 33,911) as any subsequent year of NHIS data. See Table 1 for descriptive information on the sample. Details about the design and methods of the NHIS can be found elsewhere (Massey, Moore,
All research was approved by the University of British Columbia Ethics Board.

Measures

Measures of SES

SES can be measured in multiple ways (e.g., education, income), with each having different implications (Krieger, Williams, & Moss, 1997; Winkleby, Jatulis, Frank, & Fortmann, 1992). For example, prestige-based measures such as level of parent education indicate the status a family has within society. In contrast, resource-based measures such as family income indicate the assets that a family possesses. Because each could have different implications for health, we include measures of both types. Four indicators of SES were used in this study. Years of education of the parents (or responsible adult) in the household was coded in the NHIS dataset from 0 to 18, representing number of years of education (18 = 18 or more years). Family income was coded in the NHIS dataset from 0 to 19.73 (0 = $0–9,999, 19.73 = $19,000–19,999), then in $5000 increments from 20 to 26 ($20,000–24,999, 26 = $50,000 +) (this is the coding system developed by the National Center for Health Statistics). Welfare status was coded as on welfare (1) or not on welfare (0). Poverty was defined in our study as 1.5 times the poverty index, a definition that other researchers have used (Montgomery, Kiely, & Pappas, 1996). Poverty status was coded as below (1) or above (0) poverty.

Measures of health

Global health measures included parent report of child health and functional limitations. Parents were asked whether the child’s health was excellent, very good, good, fair, or poor. This variable was dichotomized into those reporting fair/poor health (1) and those reporting good to excellent health (0). Limitations were assessed by asking whether the child was limited in any activities because of an impairment (physical or mental health condition) or health problem (1 = limited, 0 = not limited); the same question was then asked for limitations in school.

Specific acute conditions included: the leading cause in childhood of deaths (injuries), and the leading cause in childhood of hospitalizations (acute respiratory conditions) (National Center for Health Statistics, 2004). Parents were asked whether the child had experienced an injury that limited activity or resulted in medical attention in the previous 2 weeks. Parents were also asked whether the child had experienced an acute respiratory conditions (including cold, flu, acute bronchitis, pneumonia) with an onset in the 2-week period prior to the interview, and that limited activity or resulted in medical attention. Thus very minor injuries and illnesses are not intended to be included in these rates. Note that acute conditions were distinguished from chronic respiratory conditions (e.g., asthma) in that chronic conditions were defined to have an onset of more than 3 months from the date of the interview. Responses for all conditions were coded as present (1) or absent (0).

Covariates

Child age, sex, and race (coded as Caucasian, African American, or other) were used as covariates, as described below. More information on relationships between SES, race, and child health outcomes in this sample can be found in (Chen, Martin, & Matthews, in press). Note that we also conducted analyses including urban/rural status as a
covariate. None of the pattern of associations below changed, and thus these analyses are not described in detail below.

Data analysis

Analyses were conducted with STATA SE (Stata, 2003), using weighted data to adjust for survey design effects due to clustered sampling and oversampling of certain groups. Logistic regression analyses were conducted predicting dichotomous health outcomes from SES variables. To test whether SES-health relationships change with age, health outcomes were regressed onto SES, age, and the interaction between SES and age simultaneously. Analyses were then repeated controlling for other sociodemographic variables (child sex and race). In addition to the interaction results, we also report main effects of SES. For main effects analyses, both unadjusted odds ratios (ORs), and ORs controlling for sociodemographic factors (child age, sex, and race) are presented. ORs indicate the likelihood of the outcome variable occurring (e.g., a child having fair/poor health) for each unit change in the SES variable.

Results

See Table 1 for the prevalence rates of each child health outcome. With respect to distributions of SES variables, family income exhibited a left skew. The 25th percentile for family income was 17 ($17,000–$17,999), the median was 22 ($30,000–$34,999), and the 75th percentile was 26 ($50,000 and over). For parental education, the 25th percentile was 12 years, the median was 13 years, and the 75th percentile was 16 years. Given the size of the sample, there were still numerous observations even at the lowest income and education levels. We tested whether there were SES differences in demographic variables using the family income variable. Income showed a small correlation with age (r = .06). Girls and boys did not differ in family income. As expected, there were significant differences in family income by racial groups (P < .001). The mean value on the income scale for Caucasian families was 21.08, for African American families was 14.84, and for other racial groups was 17.83. Finally, we tested whether there were differences in response rates to health questions as a function of SES (income). No relationship was found between income and response rate to the overall health or activity limitations questions.

We first tested study hypotheses for global health measures. Logistic regression analyses revealed that all SES variables predicted parent report of child health (P's < .001), such that children from lower SES families were more likely to have fair or poor health. See Table 2. Controlling for race, sex, and age did not change these associations (P's < .001). All SES variables also predicted parent report of child activity limitations (P's < .001), such that children from lower SES families were more likely to be limited in activities. Controlling for race, sex, and age did not change these associations (P's < .001). No interaction effects between SES and age were found for global health measures. As an indicator of the magnitude of these effects, families in the bottom 25th percentile of income had a .031 predicted probability of being in fair/poor health, which was almost twice as high as the predicted probability of those in the 75th percentile of income (.016). Families in the bottom 25th percentile of income had a .073 predicted probability of having an activity limitation, which was almost 1.5 times as high as those in the 75 percentile of income (.051), and a similar magnitude difference existed for school limitations (.091 versus .063 predicted probabilities).

With respect to specific acute conditions, significant interactions were found for income by age (regression coefficient = −.23, P < .05) and for poverty status by age (regression coefficient = .14, P < .01) with respect to childhood injuries. Since estimated coefficients for interactions can be somewhat difficult to interpret, we constructed figures to illustrate the substantive effects estimated by the statistical model. These figures can be read as the predicted probabilities for any age at the minimum, middle, and maximum value of the scales. As Fig. 1 depicts, the effect of income on incidence of injuries was strongest during adolescence. A similar pattern was found for poverty status. Interactions did not change after controlling for race and sex. No significant effects were found for welfare or education.

Significant interactions were found for income by age (regression coefficient = −.15, P < .05) and
marginally for poverty status by age (regression coefficient = .05, \( P < .10 \)) in predicting acute respiratory conditions. As Fig. 2 depicts, an expected gradient of lower income being associated with higher rates of respiratory conditions was found during adolescence, whereas a reverse gradient existed earlier in childhood. A similar pattern was found for poverty status. Interactions did not change after controlling for race and sex. No significant effects were found for welfare.

In addition, for acute respiratory conditions, a main effect of education indicated that parents with higher education were more likely to report respiratory conditions in their children (regression coefficient = 0.08, \( P < .001 \)). Controlling for race, sex, and age did not change this association (\( P < .001 \)).

**Discussion**

The results of this study revealed that associations of SES with global health measures are similar across childhood and adolescence, whereas associations of SES with specific acute conditions vary by age among a sample of nationally representative US children. For global health measures (parent rating of overall child health, activity limitations, and school limitations), an expected gradient was found, such that lower family SES was associated with poorer overall health and greater limitations. These associations were linear, indicating that as family SES decreased, the odds of poor child health increased in a gradient fashion. These findings are consistent with previous research documenting SES gradients in health during childhood as well as adolescence (Goodman, 1999; Newacheck, Hung, Park, Brindis, & Irwin, 2003; Starfield, Riley et al., 2002; Starfield, Robertson et al., 2002); however, by including both children and adolescents, the present study extended previous research by testing for differences in SES gradients across age.

Associations with global health measures also were robust across type of SES measure. This suggests that regardless of whether SES is measured through assets (e.g., family income), or status indicators (e.g., parent education), lower SES is associated with poorer global health throughout childhood and adolescence. In addition, these relationships persisted after controlling for child race, sex, and age, indicating that these associations cannot be better accounted for by other sociodemographic variables linked to SES, such as race.

The fact that global health measures consistently were associated with SES across childhood (no
interactions of SES with age) suggests that developmental factors that are prominent only at certain ages do not constitute the main pathways to global health, as these relationships did not vary by age. This pattern fits with the Persistence model of SES and child health (Chen et al., 2002). This finding also fits with the hypothesis of West and colleagues that changes in the SES gradient with age would be most evident for health states (measures that easily fluctuate over time), but not for health status (longstanding or persistent health measures) (West & Sweeting, 2004).

For global health ratings, it is likely that a variety of factors, unrelated to developmental processes in childhood, contribute to SES and health relationships. These could include factors such as the physical environments that low versus high SES families live in (e.g., exposure to toxins, pollutants) (Lanphear et al., 2001; Pamuk et al., 1998), societal-level variables (e.g., access to and quality of health care) (Andrulis, 1998; Newacheck et al., 2003), characteristics of the neighbourhoods that low versus high SES families live in (e.g., social capital and cohesion) (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Leventhal & Brooks-Gunn, 2000), and family level variables (e.g., social support, family conflict) (Cohen, Kaplan, & Salonen, 1999; Repetti, Taylor, & Seeman, 2002).

In contrast, when we examined specific acute childhood conditions, we found evidence that relationships varied by age. Childhood non-fatal injuries fit an Adolescent-Emergent model (Chen et al., 2002), whereby SES differences were not present during the early childhood years, but significant during the adolescent years. These results suggest that developmental factors that emerge during adolescence may help explain these SES disparities. For example, adolescence is a time of increasing affiliation with peer groups, and declining levels of parental supervision. The transition to adolescence can coincide with increases in risky behaviours that are linked to morbidity outcomes (Green & Palfrey, 2000), and low SES peer groups may be more likely to participate in such risky behaviours. Indeed, previous research has documented that low SES adolescents engage more frequently in behaviours such as drinking alcohol and not wearing seat belts (Droomers, Schrijvers, Casswell, & Mackenbach,
In addition, low SES parents may have less opportunity to supervise their children and to model safe behaviours. These risky behaviours could partially explain the adolescent-emergent relationship for acute childhood non-fatal injuries.

We note that a number of previous studies found SES gradients for injuries during early childhood, but not late adolescence (West, 1997), leading us to conclude in our earlier review paper that the SES and childhood injury gradient might follow a childhood-limited pattern (Chen et al., 2002). However, it should be noted that in that review, we compiled studies that assessed specific age groups of children in order to draw inferences of patterns across childhood and adolescence. Given that these studies used different SES indicators and injury outcomes, the present study in which one sample of children ages 0–18 were studied using consistent measures of SES and injuries across all age groups, provides a stronger test of how SES gradients vary by age. In addition, a number of previous studies have focused on lifetime prevalence rates of injuries, which may be difficult to accurately recall. In contrast, the present study focused on only injuries that had occurred in the previous 2 weeks and that were serious enough to warrant medical attention or limit activity in order to maximize accuracy. Finally, there are differences across studies in the use of parent- versus child-reports. For example, some previous studies that have documented a lack of SES differences in adolescence have relied on adolescents to report injuries and/or family SES (West et al., 2004; Williams, Currie, Wright, Elton, & Beattie, 1996), and thus it is possible the present study’s findings are in part due to the reliance on parental reports. Overall, these differences in methodologies may account for some of the different findings in the literature. We also note that some previous studies have documented expected SES and injury gradients during adolescence (Faelker, Pickett, & Brison, 2000; Nelson, 1992; Nersesian, Petit, Shaper, Lemieux, & Naor, 1985).

For acute respiratory conditions, a crossover effect was found, such that a reverse SES gradient existed in early years, whereas an expected SES gradient appeared during adolescence. Early in childhood, it may be the case that higher SES parents have a lower threshold for noticing respiratory symptoms and taking their child to the doctor. Alternatively, this reverse gradient may be due to the “hygiene hypothesis,” the notion that a decrease in childhood exposure to microbes and infections (often found among higher SES families) may increase the predisposition to respiratory conditions such as allergies and asthma (Strachan, 1989). In contrast, during older ages, differences in parents’ noticing and reporting of respiratory illnesses may become less important, as parents rely more on their children to identify symptoms. Thus others factors may contribute to respiratory illnesses in adolescence. For example, adolescents may spend more time outside the home with peers; if exposures to infectious agents are more likely in low SES neighbourhoods, this could explain the expected SES gradient in adolescence. Alternatively, the accumulation of early environmental exposures or the development of maladaptive health practices in adolescence (e.g., smoking) could mean that expected SES gradients become evident with older age. For example, exposure to tobacco smoke, a factor associated with lower childhood SES (Ben Shlomo & Kuh, 2002), may accumulate over time and contribute to an expected SES gradient for acute respiratory illnesses that is largest during adolescence. This pattern of an expected SES gradient for acute respiratory illnesses appearing later in adolescence is consistent with previous research that has documented effects of childhood SES on adult respiratory illnesses (Cohen et al., 2004), as well as on adult pulmonary functioning (Jackson, Kubzansky, Cohen, Weiss, & Wright, 2004; Lawlor, Ebrahim, & Smith, 2004).

Across the two acute conditions investigated in this paper, the expected SES gradient appears to emerge at similar ages. Although the figures suggest a gradient beginning to appear around the age of 9, statistically, the expected SES gradient is not significant until the teenage years. The timing of this pattern suggests that focusing on the transitions that adolescents undergo as they progress from middle school to high school may help in understanding the reasons why expected SES gradients for acute injuries and respiratory illnesses develop at this time. For example, changes in adolescents’ understanding of their family’s social status and financial circumstances, changes in the types of life stressors adolescents experience, and changes in peer relationships and school cliques may all play a role in the emergence of SES gradients for acute adolescent health outcomes.

Previous research on childhood SES differences has shown some interesting patterns by age.
Recently, Case and colleagues demonstrated that SES and overall health relationships grow stronger with age during childhood, and in particular, that the effect of having a chronic condition and being low in SES is stronger in older children (Case, Lubotsky, & Paxson, 2002). Some researchers have argued that SES effects accumulate from birth onward to predict health through to adulthood (Power et al., 1999). Studies such as the British Birth Cohort Study, which has followed a cohort from birth to age 41, have documented effects of childhood SES on a variety of health measures during different periods of childhood and adulthood (Power, 1992; Power, Manor, & Li, 2002; Power et al., 1999). In addition, childhood SES has been found to predict a number of health outcomes later in life. For example, lower childhood SES has been associated with poorer cardiovascular and dental health in young adulthood (Poulton et al., 2002), increased risk of coronary heart disease (Lawlor, Smith, & Ebrahim, 2004), increased risk of mortality due to stroke and stomach cancer (Galobardes, Lynch, & Smith, 2004; Smith, Hart, Blane, & Hole, 1998), and poorer pulmonary function (Jackson et al., 2004; Lawlor, Ebrahim et al., 2004; Lawlor, Smith et al., 2004), all in adulthood. In contrast, West and colleagues have argued in both empirical and review papers that there is an equalisation of SES effects, such that few SES gradients exist during adolescence (West, 1988; West, 1997; West, MacIntyre, Annandale, & Hunt, 1990; West et al., 2004). The present study adds to some of this previous research by testing children from birth until adulthood for multiple health outcomes, and statistically testing for SES \times age interactions.

Our findings for age differences in SES relationships primarily appeared for asset-based SES measures, such as income and poverty, but not for status-based measures, such as parental education. The findings of interactions between SES and age for health outcomes in this study is consistent with some adult literature that suggests that income by age interactions are more robust than education by age interactions in predicting adult health (House et al., 1994; Mustard, Derksen, Berthelot, Wolfson, & Roos, 1997). It may be the case that asset-based SES measures, which tend to fluctuate more than SES measures such as parental education, are more sensitive to capturing dynamic relationships that vary by age or over time. Alternatively, the measure of parental education in this study may have been limited. For example, the educational level of a particular parent (mother versus father) may be more important to children’s health, and this study was unable to identify whether the educational level reported was that of the mother or father.

Limitations to the present study include the cross-sectional assessment of SES and health. Although differences appeared by age, it is unclear whether an individual child studied over time would show the same health trajectory. Future studies should explore how changes in SES over time affect the physical health of children, similar to studies that have investigated longitudinal effects on adult health outcomes (McDonough, Duncan, Williams, & House, 1997; Power et al., 1999). In addition, health measures were assessed by asking parents about their child’s health, with no physician or biological verification of responses, and it is possible that there are self-reporting biases by SES. Furthermore, health care variables such as type of health insurance, likely play a role in childhood health outcomes and would be important to incorporate into future studies. Future studies would also benefit from interviews of both the target child as well as proxy respondents. With respect to SES, some of the present study’s SES measures were limited (e.g., lack of more fine-grained distinctions in income). In addition, future studies that assess both fatal and non-fatal child health outcomes may produce more robust, or even different, SES by age patterns.

Finally, it should be noted that these patterns were found in a sample of US children. Although SES and health relationships have been documented in numerous countries (Marmot & Bobak, 2000; Wilkinson, 1992), differences across countries in health care systems, cultural beliefs about health practices, and the timing of changes in children’s developmental milestones may result in a different pattern of SES by age gradients in different countries. Thus, for example, our findings differ from that of West et al., who have argued that a pattern of equalisation occurs during adolescence for certain types of health outcomes (West, 1997; West et al., 2004).

One of the two overarching goals of US Healthy People 2010 is the elimination of health disparities (US Department of Health and Human Services, 2000). Understanding the scope of socioeconomic disparities is critical, particularly in childhood when interventions may have the biggest impact. Our findings provide evidence that SES gradients are apparent across all of childhood for global health
ratings, but vary by age for specific acute conditions. These findings have important implications. First, it suggests that in order to better understand the reasons why SES affects these acute conditions, we need to focus on pathways that emerge during adolescence (e.g., changes in peer group affiliations). Understanding the role that such factors play in SES-adolescent health relationships will allow us to develop interventions targeted at appropriate pathways that shape SES-health gradients. Second, the timing of interventions is important; undertaking expensive interventions, such as Moving to Opportunity (Katz, Kling, & Liebman, 2001), when health disparities are not present would unnecessarily utilize precious resources. On the other hand, for outcomes such as global health status, our results suggest the importance of understanding the role of social and environmental factors that are not linked to developmental processes in childhood. Overall, targeting plausible mediators at specific periods of childhood hopefully will help to reduce health disparities in childhood and maximize the health of children across society.

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