

Socioeconomic Differences in Children's Health: How and Why Do These Relationships Change With Age?

Edith Chen
Washington University

Karen A. Matthews
University of Pittsburgh

W. Thomas Boyce
University of California, Berkeley

The effects of socioeconomic status (SES) on health are well documented in adulthood, but far less is known about its effects in childhood. The authors reviewed the literature and found support for a childhood SES effect, whereby each decrease in SES was associated with an increased health risk. The authors explored how this relationship changed as children underwent normal developmental changes and proposed 3 models to describe the temporal patterns. The authors found that a model's capacity to explain SES–health relationships varied across health outcomes. Childhood injury showed stronger relationships with SES at younger ages, whereas smoking showed stronger relationships with SES in adolescence. Finally, the authors proposed a developmental approach to exploring mechanisms that link SES and child health.

Socioeconomic status (SES) has a profound influence on health. Individuals lower in SES experience higher rates of morbidity and mortality in almost every disease category than individuals higher in SES (see Adler, Boyce, Chesney, Folkman, & Syme, 1993; Adler et al., 1994; N. B. Anderson & Armstead, 1995; Antonovsky, 1967; Haan, Kaplan, & Syme, 1989; Marmot, Kogevinas, & Elston, 1987, for reviews), regardless of whether SES is measured by education (Kitagawa & Hauser, 1973), income (McDonough, Duncan, Williams, & House, 1997), or occupation (Marmot, Shipley, & Rose, 1984). Many explanations for how SES affects health center on factors associated with poverty, such as inadequate living conditions and malnutrition, which would predict a threshold effect for SES. That is, above a certain level of SES, when housing and nutrition are not factors, individuals should all display similar levels of good health. However, epidemiological studies reveal that SES is related to health outcomes in a monotonic fashion (Backlund, Sorlie, & Johnson, 1996; Ecob &

Davey Smith, 1999). That is, each decreasing SES level is associated with an increasing prevalence of disease. Thus, not only do poor people suffer from worse health than nonpoor people, but individuals at each level of SES also enjoy better health than individuals just below them. This finding is challenging to researchers who seek plausible mechanisms to account for the SES effect.

In addition, the majority of research on SES and health has focused on young to middle-aged adults, with the extremes of the age spectrum receiving less attention. Far less is known about the impact of SES on health during childhood, even though childhood health problems constitute a significant proportion of health care costs. For example, estimated lifetime costs of injuries, the leading cause of mortality among children, exceeded \$185 billion in 1985 (Public Health Service, 1990). Reviews have concluded that poverty has a detrimental effect on children's health (Aber, Bennett, Conley, & Li, 1997; Egbuono & Starfield, 1982; Jolly, Nolan, Moller, & Vimpani, 1991; Shah, Kahan, & Krauser, 1987). However, the question remains whether an effect of increasing health problems with decreasing SES exists, similar to the effect found in adult health. In this article, our first objective is to review the evidence for a threshold versus monotonic SES effect on child health.

Understanding the relationship between SES and child health also has developmental implications. For example, the SES and health relationship may change with age; that is, SES may have stronger effects during certain periods of childhood than others. Guided by several possible developmental models proposed below, our second goal is to review how the relationship between SES and health changes across childhood and adolescence for a variety of outcomes, including childhood diseases, risk factors for disease, and health behaviors. Connecting the timing of changes in the SES and health relationship to developmental milestones may

Edith Chen, Department of Psychology, Washington University; Karen A. Matthews, Department of Psychiatry, University of Pittsburgh; W. Thomas Boyce, Department of School of Public Health and Institute of Human Development, University of California, Berkeley.

This research was supported by the MacArthur Foundation Research Networks on Socioeconomic Status and Health and on Psychopathology and Development, National Institutes of Health Grants HL25767 and MH18269, and the Pittsburgh Mind–Body Center (Grants HL65111 and HL65112). We thank Nancy Adler, Sheldon Cohen, David Kupfer, Bruce McEwen, Gregory Miller, and Shelley Taylor for their helpful comments on earlier versions of this article.

Correspondence concerning this article should be addressed to Edith Chen, Department of Psychology, Washington University, Campus Box 1125, One Brookings Drive, St. Louis, Missouri 63130. E-mail: echen@artsci.wustl.edu

provide clues about mechanisms that drive the SES and health relationship in children. That is, if the timing of changes in an SES and health relationship can be shown to coincide with certain developmental changes, these developmental shifts may be candidates for explaining both the existence of an SES and health relationship and why it changes when it does. Thus, our third goal is to discuss potential mechanisms for the childhood SES and health relationship.

Nature of SES Relationship in Childhood and Adolescence

To determine the nature of the relationship between SES and health in children, we searched through the Medline database and used the ancestry method to identify studies that included health outcomes among individuals ranging in age from 0 to 18 years of age. Our Medline search used the following search terms for SES: *socioeconomic status*, *social class*, *education*, *occupation*, *income*, and *poverty*. The following terms were searched for health: *health*, *disease*, *mortality*, and *morbidity*. The goal of this portion of the article is to provide a broad overview of SES and health associations across a spectrum of childhood disorders. This is not an exhaustive review; rather, it is an attempt to discern whether patterns in childhood bear resemblance to those found in adulthood. The descriptions below provide brief summaries of studies covering a wide age range and separated by health outcome. We

review general mortality and morbidity outcomes as well as the primary childhood conditions in which SES effects have been investigated; however, not all major childhood health conditions are discussed. Our next section describing how relationships differ by age contains more detail on a study-by-study basis.

All-Cause Mortality and Morbidity

Table 1 lists large-scale studies that have examined whether SES is associated with all-cause mortality across childhood and adolescence (age range = 0–19). As Table 1 illustrates, a monotonic effect exists such that as SES decreases, all-cause mortality increases (Delamothe, 1991). This occurs regardless of whether SES is measured by parent education, occupation, or income. Morbidity outcomes reveal that ratings made by parents of their children's overall health status also decline in a monotonic fashion with decreasing family income (Federal Interagency Forum on Child and Family Statistics, 1997; L. E. Montgomery, Kiely, & Pappas, 1996).

Cause-Specific Mortality

When mortality rates are broken down by specific causes, similar relationships with SES are found. Children from lower SES backgrounds are more likely to die from chronic conditions such as

Table 1

Summary of Large-Scale Studies That Investigated SES Effects on All-Cause Mortality or Morbidity Measures and Include Wide Age Ranges of Children and Adolescents

Study	Age range (years)	SES measure	Outcome	Finding
Delamothe (1991)	1–15	Occupation	All-cause mortality	Monotonic effect, whereby lower parent occupational class is associated with higher all-cause mortality.
Pharoah & Alberman (1990)	0–14	Occupation	All-cause mortality	Monotonic effect whereby lower parent occupational class is associated with higher all-cause mortality.
Singh & Yu (1996)	1–14	Family income	All-cause & external cause (injury & violence) mortality	Monotonic effect whereby lower family income is associated with higher external cause mortality rates. Those with low family income also have high all-cause mortality rates (but not monotonic).
Mare (1982)	0–19	Family income Maternal education	All-cause mortality	Children of mothers with less than high school education and children with family incomes less than \$10,000 had higher all-cause mortality rates than children from more educated or higher income families.
Nersesian et al. (1985)	0–17	Federal assistance	All-cause mortality	Children from families receiving federal assistance had higher all-cause mortality rates than children from families not receiving federal assistance.
National Health Interview Survey, 1994 (Pamuk et al., 1998)	0–17	Family income	Parent report of child's health status and utilization of medical care	Monotonic effect whereby lower family income is associated with poorer health status, greater limitation from chronic health conditions, fewer doctor visits, and more emergency room visits.
L. E. Montgomery et al. (1996)	0–19	Above or below poverty level	Parent rating of child's health status	Children living below poverty level were more likely to be rated as having fair to poor overall health than children living above poverty level.

Note. SES = socioeconomic status.

asthma and other respiratory disorders (Vagero & Ostberg, 1989; Weiss, Gergen, & Wagener, 1993). Lower SES also is associated with increased mortality rates from less common chronic conditions such as cancers, congenital anomalies, and heart disease (Nelson, 1992; Petridou et al., 1994; Pharoah & Alberman, 1990; Vagero & Ostberg, 1989).

In addition, lower SES children have increased mortality rates due to acute conditions or events. Children from families who receive federal assistance in the United States are more likely to die of pneumonia or influenza compared with families not receiving federal assistance (Nelson, 1992). Lower SES children also have higher mortality rates due to injury, with several studies documenting a monotonic effect (Carey, Vimpani, & Taylor, 1993; Keeling, Golding, & Millier, 1985; Nelson, 1992; Nersesian, Petit, Shaper, Lemieux, & Naor, 1985; Pharoah & Alberman, 1990; Roberts, 1997; Vagero & Ostberg, 1989).

Morbidity From Chronic Conditions

Across all chronic conditions, low SES children are more functionally impaired than high SES children. Children's degree of limitation from chronic health conditions and school absences increases in a monotonic fashion with decreasing family income in the United States (Aber et al., 1997; Pamuk, Makuc, Heck, Reuben, & Lochner, 1998). A higher percentage of U.S. children living in poverty have severe chronic conditions and have to be hospitalized because of these conditions (Newacheck, 1994).

With respect to specific chronic conditions, there is evidence for an SES effect. Education, income, and occupation-based measures of SES all are negatively associated with increased prevalence of asthma, wheezing, and chronic night coughing, with some studies documenting a monotonic effect (Crain et al., 1994; Gortmaker, Walker, Jacobs, & Ruch-Ross, 1982; Halfon & Newacheck, 1993; Schenker, Samet, & Speizer, 1983; Schwartz, Gold, Dockery, Weiss, & Speizer, 1990). Lower SES, whether defined by income or occupation, also is associated in a monotonic fashion with asthma severity outcomes, such as hospitalizations and frequent, limiting asthma attacks (Claudio, Tulton, Doucette, & Landrigan, 1999; Glezen & Denny, 1973; Halfon & Newacheck, 1993; Pamuk et al., 1998; Ray, Thamer, Fadillioglu, & Gergen, 1998; Strachan, Anderson, Limb, O'Neill, & Wells, 1994; Wissow, Gittelsohn, Szklo, Starfield, & Mussman, 1988).

Poor children suffer from higher prevalence rates of other chronic conditions as well. Increased number of vision and hearing disorders has been associated with lower average neighborhood income and greater crowding in the house (Dutton, 1985; Egbuonu & Starfield, 1982). Lower family income also is associated in a monotonic fashion with higher blood lead levels in children (Egbuonu & Starfield, 1982; Pamuk et al., 1998; Starfield, 1989).

Morbidity From Acute Conditions or Events

With respect to acute childhood conditions, families living in more crowded houses are more likely to have infants with infectious disease (Guendelman, English, & Chavez, 1995). Poor children have higher rates of rheumatic fever, meningitis, and parasitic diseases than nonpoor children (Egbuonu & Starfield, 1982). In addition, children from poor families miss more days of school and spend more days in bed as a result of these acute illnesses (Eg-

buonu & Starfield, 1982). Children from lower social classes also report more school absences as a result of upper respiratory or ear infections (Power, 1992). Lower SES, whether defined by education, income, or occupation, also is associated with higher injury rates (Brown & Davidson, 1978; Dougherty, Pless, & Wilkins, 1990; Jolly, Moller, & Volkmer, 1993; Keeling et al., 1985; Morrison, Stone, Redpath, Campbell, & Norrie, 1999; Nelson, 1992; Nersesian et al., 1985; Pharoah & Alberman, 1990; Rivara & Barber, 1985; Roberts, 1997; Roberts, Marshall, Norton, & Borman, 1992; Roberts & Power, 1996; Vagero & Ostberg, 1989).

Health Care and Health Practices

As family income decreases, U.S. children are less likely to be seen by a doctor and more likely to be seen in emergency departments and hospitalized, with patterns revealing a monotonic effect (Aber et al., 1997; Egbuonu & Starfield, 1982; Pamuk et al., 1998). For each \$10,000 decrease in median household income, there is a 9% increase in emergency pediatric intensive care unit admissions (Naclerio, Gardner, & Pollack, 1999). Although this in part reflects more severe health problems among low SES children, it also reflects access to and decisions about health care, whereby low SES families are less likely to have health insurance, less likely to have regular contact with a physician, and more likely to seek care in emergency rooms (Pamuk et al., 1998).

SES also affects health behaviors. Lower SES is associated with increased rates of cigarette smoking, greater exposure to tobacco smoke, and more sedentary lifestyles (see Conrad, Flay, & Hill, 1992, for a review of smoking, and Stephens, Jacobs, & White, 1985, for a review of physical activity; Coombs, Fawzy, & Gerber, 1986; Iscan, Uyanik, Vurgun, Ece, & Yigitoglu, 1996; Pamuk et al., 1998).

Limitations

Although there are substantial data supporting the association of lower SES with increasing health problems, this relationship has not been uniformly documented. In fact, reverse associations of SES and health have been found for a few childhood health problems. Several studies outside the United States have found that prevalence rates of asthma are higher among children whose parents have higher SES (Goh, Chew, Quek, & Lee, 1995; Graham, Rutter, Yule, & Pless, 1967; Hamman, Halil, & Holland, 1975; Peckham & Butler, 1978). Higher SES children are more likely to be diagnosed with Hodgkin's disease and acute lymphoblastic leukemia (Gutensohn & Cole, 1981; see Ross, Davies, Potter, & Robison, 1994, for a review). Allergies and myopia are more common in nonpoor than poor children in the United States (Egbuonu & Starfield, 1982; Newacheck, 1994). One study found an opposite relationship of higher risk of injury among high SES families (Scheidt et al., 1995).

In addition, although the vast majority of studies described above have large sample sizes, some contain other limitations. Some studies rely on dichotomous measures of SES that do not allow tests of monotonic SES effects (Nelson, 1992; Newacheck, 1994). Other studies examine differences only in the lowest and highest SES groups or do not test for linear trends (Delamothe, 1991; Pharoah & Alberman, 1990). In addition, other studies rely on self-report of diseases, uncorroborated by physician diagnosis

(Crain et al., 1994). These approaches can lead to concerns about the validity of the data. For example, reliance on parent interviews about child health may be limited if there are parental biases in symptom reporting.

Summary

Children from lower SES families typically suffer worse health outcomes compared with children from higher SES families. Although many studies are limited in relying on dichotomous measures of SES, there is support for the notion that decreases in SES are associated in a monotonic fashion with increases in health problems, similar to the effect found in adulthood. This effect appears across all causes of childhood mortality, as well as across a number of acute and chronic childhood conditions. Studies have reported these effects to be quite striking in magnitude. For example, the U.S. Census Bureau (1998) estimated that there are 77.75 million children aged 0–19 living in the United States. If this estimate is used, injury rates broken down by neighborhood SES level (e.g., average family income in a neighborhood; Jolly et al., 1993) translate into each decrease in neighborhood SES (on a five-level scale), resulting in an average of an additional 1.5 million U.S. children being injured each year.

Developmental Models of SES and Health

Although the above studies provide strong evidence for a monotonic SES effect during childhood, many collapse across a wide age range. It is possible that this approach misrepresents the true relationship between SES and health throughout childhood. Childhood is a time of dramatic developmental changes, and these changes may result in different SES associations in early childhood compared with late adolescence. As a hypothetical example, low SES is associated with high infant mortality rates (Chamberlain, 1979; Gortmaker, 1979). Low SES children who survive beyond infancy, however, may represent a heartier group, such that beyond infancy, low and high SES children are not different in health (survivor effect). Studies that examine mortality rates from birth until adulthood may document SES differences across childhood that in fact are attributable primarily to the period of infancy. In this case, the conclusion that an SES monotonic effect is evident throughout childhood would be misleading. Thus, it is important to track the SES and health relationship longitudinally from childhood through adolescence.

There are arguably several possibilities for how the relationship between SES and childhood health may change across childhood. Figure 1 shows three developmental models of various ways that this relationship may change. These models are drawn in part from theories proposed by other SES researchers in both the adult and child area.

A childhood–adolescent persistent model (referred to for simplicity as the persistence model in the remainder of the article) posits that SES differences in health are established early in life and remain fairly constant throughout childhood. That is, SES effects on health remain equally strong in spite of developmental changes that children undergo. As a hypothetical example, lower SES children may have more difficulty managing diseases such as diabetes, perhaps because their parents have fewer resources to properly monitor insulin levels and to afford appropriately healthy

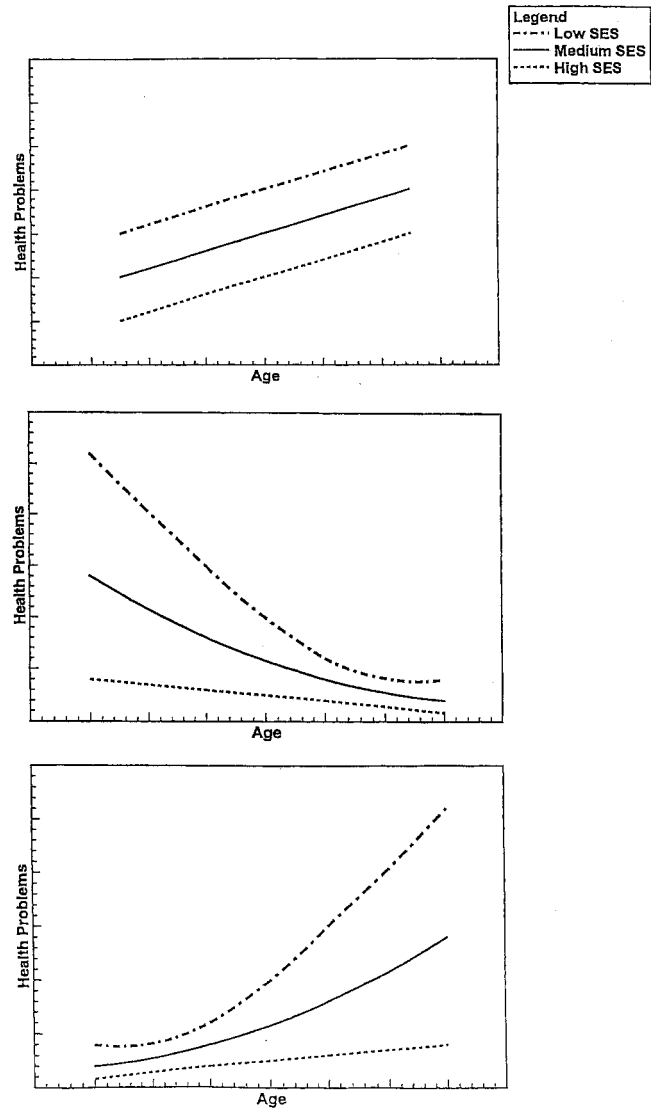


Figure 1. Three possible developmental models of the relationship between socioeconomic status (SES) and child health. The top panel depicts the childhood-adolescent persistent model, showing that the SES and health relationship remains constant over time. The middle panel depicts the childhood-limited model, showing that the relationship between SES and health is strongest in early childhood and weakens with age. The bottom panel depicts the adolescent-emergent model, showing that the relationship between SES and health is weak in early childhood and becomes more pronounced with age.

diets. This disadvantage may result in low SES children with diabetes having more diabetes-related complications than high SES children. If SES differences in parental resources have consistent effects on diabetes-related outcomes throughout childhood and adolescence, a persistence model would emerge. It should be noted that although we have depicted the persistence model as having prevalence rates that steadily increase in childhood, the same model could be depicted with horizontal lines (constant

prevalence rates throughout childhood) or downward-sloping lines (decreasing prevalence rates throughout childhood).

A childhood-limited model suggests that SES effects initially are large, but gradually decrease over time and are most modest during the teenage years. That is, as children develop, other factors play a more important role in determining child health, and the strength of parent SES decreases. One hypothetical example of this model is infectious disease. At young ages, when a child is spending the majority of time at home, poor housing and living conditions may play a large role in spreading infectious illnesses. As a child grows older, however, he or she spends more time in the school environment, which allows for similar exposures to certain viruses among low and high SES children. This suggests that SES differences in infectious illnesses would decrease with age. This type of model has been proposed by West (1990, 1997) as an explanation for the SES and health relationship among youth in Great Britain.

An adolescent-emergent model posits that SES effects initially are modest but gradually increase over time and are most apparent during the teenage years. That is, early childhood SES may start children on a trajectory of increasingly negative health outcomes with increasing age. For example, to the extent that the effect of passive smoking is cumulative and associated with lower SES, one might anticipate that low SES children exposed to passive smoke might begin to show greater susceptibility to bronchial irritation, which when some threshold of irritability is exceeded, may develop into early stages of asthma. With continued accumulation of exposure to smoke and other environmental toxins, the episodes of asthma may become more frequent and severe, leading to debilitating disease.

Power and Hertzman (1997) have proposed an ostensibly similar model to the adolescent-emergent model, in which childhood SES effects appear in adolescence and young adulthood primarily. However, they posit that the accumulation of risk is due to the likelihood of remaining in one's social class of origin as an adult (Hertzman & Wiens, 1996). Another model, the latency model, suggests that early-childhood SES continues to influence health in adulthood, even when adult SES is taken into account (Lynch, Kaplan, & Salonen, 1997; Power, Matthews, & Manor, 1998; Smith, Hart, Blane, & Hole, 1998). These authors suggested that early childhood represents a "critical period," such that low SES children miss or are deprived during certain developmental milestones, which then has negative health consequences that persist into adulthood. Although the latency model and its testing is important for understanding adult health and illness, we do not discuss it further here because of our emphasis on child health outcomes.

The above models are intended as a conceptual and organizational framework from which to interpret the remainder of the studies reviewed below. The models serve as competing predictions about how the relationship between SES and health changes from childhood through adolescence. However, it is possible that different models fit different disease outcomes. Model fit may also vary by culture and/or temporal epoch. That is, a model that describes one disease outcome in the United States may not fit the epidemiology of that same disease in another country. Or a model that describes a particular disease in the United States today may not fit that same disease in the United States 50 years ago, as treatments and societal norms for health behaviors change. Pat-

terns that are described below should be interpreted in light of current health-related trends and treatments.

Evaluating the Models

To evaluate the above models, we selected three common childhood health problems as examples and review evidence for the SES–health monotonic effect by specific age groups. We reviewed SES differences for the most common cause of mortality in childhood, injury (Rodriguez & Brown, 1998); the most common chronic illness in childhood, asthma (Pamuk et al., 1998); and risk factors related to the most common cause of death across the life span, cardiovascular disease (Public Health Service, 1990). A Medline search was conducted of articles from 1966 to June 1999, with search terms including *injury*, *asthma*, *smoking*, *blood pressure*, and *physical activity*. Note that some of the risk factors of cardiovascular disease are also risk factors for other diseases (e.g., smoking and asthma), and thus conclusions drawn from that section have implications for other diseases. The same search terms described earlier were used for SES (*socioeconomic status*, *social class*, *education*, *occupation*, *income*, and *poverty*). The ancestry method was used to identify additional relevant studies. All studies found that investigated one of our specific health outcomes of interest were included in this review, provided they met the following criteria: compared children from different age groups or included children from only one period of childhood; based their SES measure on at least one traditional approach to assessing SES (income, education, occupation); and conducted the study in a non-third-world country.

Why an Enumerative Analysis?

We chose to summarize the available data using an enumerative or descriptive review in this article, as opposed to the more common meta-analytic techniques, for several reasons. First, as we discuss in the following section on methodological issues, existing empirical studies have not sought to directly address the primary question of our review, that is, whether the direction and magnitude of the SES effect varies with age in children. As such, we have been by and large restricted to describing cross-sectional associations within age groups and contrasting them across studies. We have focused this article on summarizing associations by age with the goal of stimulating more definitive, longitudinal research. We think a descriptive approach is especially appropriate at this stage of research as experiments have shown that scientific readers place more confidence in conclusions drawn from meta-analyses than from narrative reviews, and this may be misplaced confidence at this stage (Cooper & Rosenthal, 1980).

Also difficult for meta-analysis is that the research pertinent to our primary question is sparse at the present stage. We would not want to combine all health outcomes (injury, asthma, cardiovascular risk factors) into one quantitative analysis because we predict that SES relationships may differ by outcome. Many studies had different age cutoffs or criteria, and we hypothesized that relationships might change with age. Even within one type of health outcome, there can be quite different approaches to assessment (e.g., incidence vs. mortality rates) that may reveal different SES relationships and mechanisms and thus should not be combined. Although these studies could be pooled initially to test for hetero-

generality across studies, our true interest would lie in the effects within each of these groupings. Once studies are broken down by these groupings, only a handful of studies within many groups are available, which raises questions about the validity of the effect sizes. Meta-analyses that summarize large numbers of studies find different effect sizes than meta-analyses that summarize fewer studies (Cooper, Dorr, & Bettencourt, 1995), and the power to detect small differences in effect sizes between groups will be less with fewer studies (Sackett, Harris, & Orr, 1986).

In addition, a compelling reason for doing meta-analyses often centers on the increased power obtained by combining individual studies with relatively small sample sizes. In this review, however, many of the studies contain large sample sizes (often more than 1,000 participants) and thus should have sufficient power to be judged individually in an enumerative review.

The Findings

Tables 2–6 describe the results of each study included in our review. Information about study design, how SES and health were measured, and prevalence rates for each outcome (when reported) are included. A description of each study's findings is also provided, including odds ratios (when reported) and any variables that were controlled in analyses. When linear trends were statistically tested, these results are included in the description of findings column.

To help the reader compile the findings across various studies, we describe below a tally of the findings by age (younger vs. older children) for each outcome. Given that SES–health relationships may change at different points in time depending on the health outcome, we use the terms *young* and *old* in a relative sense in which the actual age ranges vary by health outcome, depending on when a shift in the relationship occurs. Age ranges for younger children versus older children are specified in each section. Multiple articles published using the same sample are grouped together in the tables and count only once toward the tally. Some studies conducted analyses in which the effects of one SES measure, controlling for all other SES measures, were tested. Given the substantial correlations among SES measures, we did not count a finding as null if it was nonsignificant after other SES measures were controlled. Second, in several studies, SES and health associations are present for some, but not all, SES measures. This is not unusual, as researchers believe that SES measures are not interchangeable and in fact tap different processes (see the *Measures of SES* section below). Thus, one would not expect all measures of SES to always be associated with health outcomes, and findings with different measures often have different policy implications. In our review, we count studies that find associations with at least one of the traditional SES indicators as supportive of an SES–health relationship. Before reviewing the evidence for each health outcome, we first briefly discuss methodological issues related to the studies below.

Methodological Issues

Measures of SES. SES reflects an individual's position within a social system or hierarchy (Adler et al., 1994; see L. E. Montgomery & Carter-Pokras, 1993). The most common measures of SES include parent income, education, or occupation. However,

measures of SES can vary substantially. Income is the most widely used indicator of a family's financial resources, but other measures include car or home ownership, house crowding, and welfare status. Each of the SES indicators, although interrelated, taps different aspects of a person's social position. For example, income relates to the resources or spending power a family has accumulated, occupation relates to a person's prestige in society, and education relates to a person's skills in acquiring economic resources and knowledge about health (Winkleby, Jatulis, Frank, & Fortmann, 1992). Education is the most stable of these measures, whereas income is probably the most fluctuating (see D. R. Williams & Collins, 1995). Each SES measure can have different implications for health.

In addition, measures of SES can be taken at an individual (e.g., family income) or neighborhood (e.g., median family income within a census tract) level. Although individual level SES measures provide information about the characteristics of the family and the family house, neighborhood level variables can provide an indication of community resources and the stressfulness of the neighborhood environment (e.g., crime rates). In the domain of school outcomes, researchers have found both family and neighborhood SES to be independently associated with IQ and school enrollment (Brooks-Gunn, Duncan, Klebanov, & Sealander, 1993). In the review below, we report whether findings with child health outcomes hold for family versus neighborhood SES measures.

Lastly, it should be noted that SES is not a static measure. That is, SES may vary over time, just as health does. It may be that changes in SES or other types of SES trajectories (e.g., persistent poverty) are associated with changes in health. In the mental health literature, researchers have shown that changes in parent occupation affect the family home environment as well as social and behavioral outcomes among children (Menaghan, Kowaleski-Jones, & Mott, 1997; Menaghan & Parcel, 1995; Parcel & Menaghan, 1994). In addition, children who are poor over long periods of time have more behavioral problems and lower IQs (Duncan, Brooks-Gunn, & Klebanov, 1994; McLeod & Shanahan, 1996). Researchers in this domain have advocated that duration and timing of low SES be considered. This approach is certainly an important one for SES research; however, research on changes in SES as it relates to physical health outcomes in childhood and adolescence unfortunately has not been conducted in a manner similar to the mental health area. As a result, we have limited the models and discussion in this article to static SES measures. Investigating the dynamic nature of SES and its differential effects on specific physical health outcomes remains an important direction for future research.

Other methodological issues. There are a number of other caveats that must be raised when interpreting epidemiological data on SES and health. The first issue relates to study design. In seeking to understand how the relationship between SES and health changes across the age span of childhood and adolescence, ideally studies would start with a sample of young children and follow them through their teenage years to track how both SES and health changes in a given individual. In reality, however, this type of design is both time consuming and expensive and thus rarely conducted. The best designed studies included in this review tend to be cross-sectional designs that include a wide age range of children and separate their findings by age. Conclusions that are

(text continues on page 310)

Table 2
 Summary of Studies That Investigated SES Effects on Injury by Specific Age Groups

Study	Age range/ sample size	Study design/ sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Alwash & McCarthy (1988a, 1988b)	0-4 402	All children attending emergency departments with injury during 1 year at one London hospital. Census information used as a denominator.	Parent occupation	-	Monotonic effect, whereby decreases in parent occupational class associated with increases in visits to emergency departments. Relative risk (RR) of lowest SES group (compared to highest) = 4.21. Children of unemployed mothers (but not fathers) had more visits to emergency departments than children of employed mothers. RR = 2.85. Children living in more crowded or rented homes had more visits to emergency departments than children in uncrowded or owner-occupied homes. Average RR = 2.5. Monotonic effect, whereby decreases in parent occupational class associated with increases in severe injuries.
			Parent employment	-	
			Housing	-	
			Visits to emergency departments following injury	-	
Larson & Pless (1988)	3 918	Population based. All children born during 1 year in one part of Montreal	Parent occupation	-	Higher percentage of children of unemployed mothers sustain injuries requiring treatment than children of employed mothers. RR = 1.6. Effect remains significant when controlling for marital status, siblings, child health status. No differences by education, income.
			Maternal education	0	
			Family income	-	
			Lifetime prevalence of medically treated injury	0	
Beautrais et al. (1982)	4 1,092	Population based. All children born during 5 months in one part of New Zealand.	Prevalence = 21.6%	0	Monotonic effect, whereby decreases in maternal education associated with increases in injury rates. Remains significant after controlling for maternal age, sex, ethnicity, life events, family size. No differences by employment. Relative risk of injury in semi- or unskilled occupational classes was 1.5 times the risk in nonmanual occupational classes. Higher percentage of children whose parents belong to lower occupational class were admitted to hospitals for injury, and were more likely to have had accidents than children of high occupation parents. Significant statistical trend, but not significant after controlling for other family factors: mother's education, age, family size.
			Maternal employment	-	
			Maternal education	0	
			Incidence of injuries requiring medical consultation	-	
Bijur et al. (1986)	5 11,966	Population based. All children born during 1 week in England.	Incidence = 49.5%	-	Higher percentage of children with lower educated mothers were admitted to hospitals for injury than children of higher education mothers (not true of injuries warranting medical attention). Not significant after controlling for parent occupation, mother's age, family size. Higher percentage of children from poor urban areas were admitted to hospitals for injury. Higher percentages of children from average urban areas were injured at least once or repeatedly compared with well-to-do. Significant after controlling for other neighborhood factors such as number of household moves. Monotonic effect, whereby increases in disadvantage associated with increases in injury hospitalization. Odds of children from most disadvantaged families being repeatedly injured was 2.3 times greater than for children with no disadvantage. Monotonic effect is evident. Significant after controlling for mother's education, age, child's age.
			Parent occupation	-	
			Maternal education	0	
			Neighborhood type	-	
Stewart-Brown et al. (1986)	13,135		Social index (composite including education, occupation, housing, neighborhood)	-	Higher percentage of children with lower educated mothers were admitted to hospitals for injury than children of higher education mothers (not true of injuries warranting medical attention). Not significant after controlling for parent occupation, mother's age, family size. Higher percentage of children from poor urban areas were admitted to hospitals for injury. Higher percentages of children from average urban areas were injured at least once or repeatedly compared with well-to-do. Significant after controlling for other neighborhood factors such as number of household moves. Monotonic effect, whereby increases in disadvantage associated with increases in injury hospitalization. Odds of children from most disadvantaged families being repeatedly injured was 2.3 times greater than for children with no disadvantage. Monotonic effect is evident. Significant after controlling for mother's education, age, child's age.
			Lifetime prevalence of hospitalization due to injury	-	
			Lifetime prevalence of injury warranting medical attention	-	
			Percentage of children who were accident repeaters	-	
Wadsworth et al. (1983)	12,743		Prevalence rates: ≥ one injury = 43.4%	-	Higher percentage of children from poor urban areas were admitted to hospitals for injury. Higher percentages of children from average urban areas were injured at least once or repeatedly compared with well-to-do. Significant after controlling for other neighborhood factors such as number of household moves. Monotonic effect, whereby increases in disadvantage associated with increases in injury hospitalization. Odds of children from most disadvantaged families being repeatedly injured was 2.3 times greater than for children with no disadvantage. Monotonic effect is evident. Significant after controlling for mother's education, age, child's age.
			Accidents repeaters = 12.1%	-	
			Hospitalizations for injury = 6.3%	-	
				-	
Bor et al. (1993)	5 5,057	Population based. Pregnant women in one area of Australia followed-up for 5 years.	Index of disadvantage (composite including education, income, occupation, welfare status)	-	Higher percentage of children from poor urban areas were admitted to hospitals for injury. Higher percentages of children from average urban areas were injured at least once or repeatedly compared with well-to-do. Significant after controlling for other neighborhood factors such as number of household moves. Monotonic effect, whereby increases in disadvantage associated with increases in injury hospitalization. Odds of children from most disadvantaged families being repeatedly injured was 2.3 times greater than for children with no disadvantage. Monotonic effect is evident. Significant after controlling for mother's education, age, child's age.
			Odds of being injured repeatedly	-	
Langley et al. (1980)	5 991	Population based. All children born during 1 year in one New Zealand hospital	Parent occupation	0	Three injury groups did not differ in terms of parent occupation or education.
Langley et al. (1983)	7 920		Parent education	0	No significant association between any SES measure and number of injuries.
		Housing			
		Group membership: Never injured, one or ≥ two medically treated lifetime injuries			
		Number of lifetime injuries needing medical attention			
		Prevalence rates: one injury = 32.9%			
		≥ two injuries = 16.1%			

(table continues)

Table 2 (continued)

Study	Age range/ sample size	Study design/ sampling strategy	SES measure/outcome	Direction of finding	Description of finding
J. M. Williams et al. (1996)	11, 13, 15 4,710	Population based. Random sampling of school classes from all schools in Scotland.	Parent occupation Family affluence (car ownership, share bedroom) Incidence of medically treated injury Severity of injury, rated by trained coders Incidence = 42.1%	0	No relation between occupation or affluence and either injury or medically rated severity of injury. Child age and sex were controlled.
R. Anderson et al. (1994)	12–16 1,245	Population based. All students from one school district in Pittsburgh.	Neighborhood type (percentage of people living below poverty in each town) Proportion remaining injury-free over 2-year follow-up	0	No township differences in injury risk, either at home or at school.
West et al. (1990)	15–18	Population based. Random stratified sampling of children born during 1 year in one area of Scotland.	Parent occupation Prevalence of lifetime injuries	0	No occupational differences for total number of injuries.
West (1997)	1,009		Total number of lifetime injuries Prevalence of at least one lifetime injury = 54.6%	0 –	No occupational differences for females. Monotonic effect for males, whereby occupational class associated with increases in injury prevalence. Significant linear trend.
Matheny (1987)	1–3, 6–9 172	Subset of children from the Louisville twin study.	Parent occupation Parent education Injury liability (composite representing number of injuries and need for medical attention) Prevalence of high injury liability: Age 0–3 = 31.3% Age 6–9 = 36.8%	– 0	Among 1–3-year-olds, lower parent occupation and lower maternal education was significantly correlated with higher injury liability. Among 6–9-year-olds, no association of occupation or education with injury liability.
Gofin et al. (1993)	0–15 4,347	All children attending two practices in Israel over a 1-year period	Neighborhood type Incidence of injuries	–	Incidence of injuries higher in primary care clinic in lower social class neighborhood compared with clinic in higher social class neighborhood. Differences between the two clinics larger among 0–4- and 5–9-year-olds than 10–15-year-olds.
Nelson (1992)	0–17 3,679	All children who died by 17 years of age in North Carolina over a 3-year period. Census information used as denominator	Eligibility for federal assistance (AFDC) Deaths due to unintentional non- motor vehicle injuries	– 0	Ratio of AFDC/non-AFDC deaths is highest for 1– 4-year-olds (4.2), lower for 5–9-year-olds (3.0), and 10–14-year-olds (2.6), and nonsignificant for 15–17-year-olds.
Pless, Peckham, & Power (1989)	7–11 12–16 11,507	Case control All injured children born during a 1-week period in England, Scotland, or Wales. 35% of noninjured children born during the same week served as controls.	Parent occupation Housing Odds of experiencing a traffic injury	0 0 –	No differences for occupational class. No differences for girls. Odds of boys who lacked basic household amenities at age 7 being injured between ages 8–11 was 1.4 times greater than for boys who did not lack amenities. Not significant after controlling for family problems, crowding, behavioral problems, physical and developmental factors.
Huang et al. (1999)	1–17 191,460	All New York state hospitalizations during 1 year. Census information used as denominator.	Neighborhood type (income inequality, or ratio of highest to lowest income levels) Incidence of injury hospitalizations	– 0	Lacking household amenities at age 11 did not predict injuries from ages 12–16 in boys. Among 1–4-year-olds, greater income inequality is correlated with greater number of injury hospitalizations.
Marsh & Channing (1987)	0–15 456	All poor children who attended one practice in England. These children were matched with same sex and age of wealthier children.	Neighborhood type Lifetime attendance rates at emergency department due to injury Prevalence rates: Age 0–4 = 36.4% Age 5–15 = 54.1%	– – –	No relationship between income inequality and hospitalizations for 5–12 or 13–17-year-olds. Among children under 5, higher percentage of those living in “deprived” areas seen in emergency departments compared with those living in “endowed” areas. Total occurrences higher in deprived areas. Among children 5–15, similar percentages of those living in deprived areas seen in emergency departments compared with those living in endowed areas. However, total number of visits is higher for deprived.
Pless et al. (1987)	0–14 1,004	All children admitted to eight Montreal hospitals because of motor vehicle accidents. Census information used as denominator.	Neighborhood type (based on child poverty levels) Incidence of medically attended motor vehicle accidents	– –	Among boys, injury rates of pedestrians and cyclists for low versus high SES areas increases with age from 3:1 (0–4-year-olds) to 9:1 (10–14-year-olds). Among girls, injury rates of pedestrians and cyclists for low versus high SES areas remains stable (6: 1) across ages. Monotonic effect appears for both boys and girls.

Note. SES = socioeconomic status; 0 = no association of SES and injury; – = negative association of SES and injury (i.e., lower SES children having higher injury rates); AFDC = Aid to Families with Dependent Children program.

Table 3
 Summary of Studies That Investigate SES Effects on Asthma by Specific Age Groups

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Weitzman et al. (1990)	2-5 3,093	Population based. Multistage sampling to provide representative sample of U.S. population of children (National Health Interview Survey)	Maternal education Living in poverty housing Family income Current prevalence of asthma: Caucasians = 3.1% African Americans = 6.5%	-	Higher percentage of children whose mothers had low educational attainment had asthma compared with children whose mothers had high educational attainment. Odds ratio (OR) = 1.7. Significant after controlling for housing status, race, sex, birth weight, mother smoking.
				-	Higher percentage of children living in extreme poverty had asthma compared with those not living in poverty. OR = 1.3. Not significant after controlling for housing status, race, sex, birth weight, mother smoking.
				-	Higher percentage of children living in small houses had asthma compared with those who live in large houses. OR for living in large house = .26. Not significant after controlling for maternal education, race, sex, birth weight, mother smoking.
E. A. Mitchell et al. (1989)	8-9 1,050	Population based. Random sampling of European children in one area of New Zealand.	Paternal occupation Lifetime prevalence of asthma diagnosis Lifetime and 12-month prevalence of wheezing	0	No differences for asthma.
				-	Monotonic effect whereby decreases in parent occupational class associated with increases in wheezing (lifetime and 12 month). Significant after controlling for gender, smokers in the house. Significant linear trend.
Bor et al. (1993)	5 5,057	Population based. Pregnant women in one area of Australia follow-up for 5 years.	Index of disadvantage (composite including education, income, occupation, welfare status) Odds of having asthma	-	Odds of children living in chronic disadvantage having asthma is 1.7 times greater than for children living with no disadvantage. Not significant after controlling for mother's education, mother's age, child's age.
Colley & Reid (1970)	6-10 10,887	Population based. Random sampling of children to fill each age and gender group from areas in England and Wales.	Paternal occupation Indices of respiratory diseases, including prevalence of current chronic cough Lifetime prevalence of bronchitis. Cough prevalence = 14% Bronchitis prevalence = 22%	-	Monotonic effect whereby decreases in parent occupational class associated with increases in current chronic cough or lifetime bronchitis.
Morgan & Chinn (1983)	5-11 5,558	Population based. Random sample of children attending primary schools in specific areas of England.	Paternal occupation Prevalence of any respiratory illness. Prevalence of asthma in past 12 months	-	Higher percentage of children from lower occupational households had respiratory illness relative to children from higher occupational households or higher SES neighborhoods.
Italian Studies on Respiratory Disorders in Childhood and the Environment (1997)	6-7 18,737	Population based. Random sampling of children in two grades in Italy.	Parent education Lifetime prevalence of hospital asthma admission Lifetime prevalence of doctor- diagnosed asthma Lifetime prevalence of wheezing 12-month prevalence of wheezing symptoms Prevalence of asthma hospitalizations = 2.6% Prevalence of doctor-diagnosed asthma = 8.7%	0	No SES difference for asthma.
				-	Monotonic effect whereby decreases in paternal education associated with increases in asthma hospitalization. Significant after controlling for gender, neighborhood urbanization.
				0	No difference in terms of prevalence of asthma diagnosis.
				-	Higher percentage of children of lower education fathers had more than one nocturnal wheezing attack per week compared with those with higher educated fathers. Significant after controlling for gender, neighborhood urbanization.
				0	No consistent differences in lifetime prevalence of wheezing symptoms.
Horwood et al. (1985)	6 1,056	Population based. All children born during 5 months in one part of New Zealand.	Parent education Parent occupation Prevalence of medically diagnosed asthma Prevalence = 10.3%	0	No differences by parent education or occupation in terms of lifetime prevalence of asthma.

(table continues)

Table 3 (continued)

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Peat et al. (1980)	Approximately 9, 13 4,893	Population based. All children from randomly selected schools within one area of Australia.	Parent occupation 5-year prevalence of asthma and bronchitis Young males = 4.8% Young females = 4.7% Older males = 5.9% Older females = 4.1%	+	Monotonic effect whereby increases in parent occupational class associated with increases in asthma among younger and older boys. Among girls, no clear association between parent occupation and asthma.
Cunningham et al. (1996)	9–11 1,416	Population based. All children within selected schools in three areas near Philadelphia	Parent education Family income Housing Health insurance Odds of having doctor-diagnosed asthma or persistent wheeze.	+	No consistent association between parent occupation and bronchitis. Odds of having asthma greater for families who own homes and have higher incomes.
Graham et al. (1967)	9–11 223	Case control. Total population of 9–11-year- olds in Isle of Wight tested. Those with poor achievement performance selected. All with asthma and random sample of controls selected.	Parent occupation Group membership: asthmatics, controls	+	Odds of having persistent wheeze 2.2 times greater for children whose parents are highly educated relative to children whose parents are not well educated. Significant after controlling for race, sex, parent health history.
Mielck et al. (1996)	9–11 4,434	Population based. All 4th graders in Munich	Parent education Prevalence of doctor-diagnosed asthma Severity of asthma Prevalence of asthma = 5.6%	0 –	Higher percentage of children with asthma have parents who belong to higher occupational classes compared with children without asthma.
Silva et al. (1987)	9 952	Population based. All children born in one hospital in 1 year in New Zealand.	Parent occupation Adversity index (composite including occupation, family relationship quality, family size, etc.) Asthma status (severe, mild, past history, no asthma) Prevalence of mild asthma = 10.6% Prevalence of severe asthma = 4.1%	0	No difference in parent education for prevalence of asthma. Monotonic effect whereby decreases in maternal educational attainment associated with increases in severe asthma. OR = 2.4, significant after controlling for child gender.
McNichol et al. (1973)	14 397	Case control. All children in 1/4 of schools in one area of Australia. All children with asthma or wheezy bronchitis selected. 1/20 of remaining children selected as controls.	Parent occupation Paternal occupation Paternal employment Family income Housing Group membership: asthmatics, controls	0	No SES differences by asthma status.
West et al. (1990)	15 1,009	Population based. Random stratified sampling of children born during 1 year in one area of Scotland.	Parent occupation 12-month prevalence of asthma	0	No differences by parent occupation for asthma prevalence. No significant trend.
Goodman (1999)	Approximately 12–18 (grades 7–12) 15,483	Population based. Cluster sampling to provide nationally representative sample of U.S. adolescents.	Parental income Parental education Parental occupation Prevalence of asthma Prevalence = 11.8%	0	No association of any SES measure with adolescent asthma prevalence rate.
Lewis et al. (1995)	5, 16 15,712	Population based. All children born during 1 week in Britain (British Cohort Study).	Social index (composite including occupation, education, income, and housing) Lifetime prevalence of wheezing Prevalence of persistent wheeze Lifetime prevalence = 30.4%	– +	Among 5-year-olds, monotonic effect whereby increases in family disadvantage associated with increases in lifetime wheezing. OR = 1.32, significant linear trend. Not significant after controlling for birth weight, number of children, sex, mother's age, mother smoking. Among 16-year-olds, monotonic effect whereby increases in family disadvantage associated with decreases in persistent wheezing. OR = .52, significant linear trend. Significant after controlling for birth weight, number of children, sex, mother's age, mother smoking.

Table 3 (continued)

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Halfon & Newacheck (1993)	0-17 17,110	Population based. Representative sample of U.S. population of children (National Health Interview Survey).	Living above/below poverty level 12-month prevalence of asthma Prevalence = 4.3%	- 0	Among children 0-5, higher percentage of poor children have asthma compared to non-poor children Among children 6-11 and 12-17-year-olds, no differences by poverty status for asthma prevalence.
Mak et al. (1982)	Approximately 5-6, 11-12 (grades 1, 6) 2,898	School children in Baltimore public schools. School stratified by SES Random sampling of children within each SES group.	Neighborhood type (composite based on median income, education, employment in area) Lifetime prevalence of asthma Prevalence for younger children = 10.6% Older children = 10.3%	0	No relationship between neighborhood SES and prevalence of asthma for either 1st or 6th graders when race and gender are controlled.
H. R. Anderson et al. (1986) Strachan et al. (1996)	7, 11, 16 8,806 0-16	Population based. All children born during 1 week in England, Scotland, or Wales (National Child Development Study).	Housing characteristics (e.g., having to share household amenities) Parent occupation Maternal education Odds of having asthma or wheezy bronchitis	- 0 0	Odds of having asthma or wheezy bronchitis was 1.5 times greater for children who shared household amenities relative to children who shared no amenities. This risk was found for asthma occurring by age 7, but not 11 or 16.
Kaplan & Mascie- Taylor (1985) Peckham & Butler (1978) Kaplan & Mascie- Taylor (1988)	7 14,571 11 13,509 16 11,392		Current and lifetime prevalence of asthma or wheezy bronchitis Prevalence of asthma age 7 = 5.5% New cases of asthma, ages 8-11 = 3.6% New cases of asthma, ages 12-16 = 2.8%	0 0 + 0 0	No association among either 0-7- or 8-16- year-olds of paternal occupation at age 11 with asthma incidence, when controlling for factors such as parent smoking, sex, birth order, and related diseases. Higher percentage of children whose fathers have non-manual occupations have asthma at ages 7, 11, and 16 compared with children whose fathers have manual occupations. Age 16, but not 7, significant when included in discriminant analyses. No differences by parent occupation at age 11 in wheezy bronchitis.
Dawson et al. (1969)	10-15 2,511	Population based. Random sampling of children attending Aberdeen primary schools.	Parent occupation Severity of asthma Prevalence of asthma = 4.8%	-	Monotonic effect whereby decreases in parent occupational class associated with increases in severe asthma.
Huang & Joseph (1999)	1-17 16,491	All New York state hospitalizations for asthma in 1 year. Census information used as denominator.	Neighborhood type (income inequality, or ratio of highest to lowest income levels) Hospitalization rates for asthma	-	Income inequality correlated with higher asthma hospitalization rates for all age groups (1-4, 5-12, 13-17). Percentage variability in asthma hospitalization accounted for by inequality less in older group than in younger group.
Watson et al. (1996)	0-14 10,044	All patients admitted to hospital for asthma during 1 year in one city in England. Census information was used as denominator.	Neighborhood type (composite based on employment, car ownership, housing) Hospital admission rates for asthma: Ages 0-4 = 0.9/100 children Ages 5-14 = 0.3/100 children	-	Significant association, such that lower neighborhood SES is correlated with higher asthma hospitalization rates among 0-4-year-olds and 5-14-year-olds.
Clifford et al. (1989)	7, 11 2,503	Population based. All children in random sampling of first and middle schools in Southampton.	Parent occupation Prevalence (lifetime and 12 month) of wheezing and other respiratory symptoms Lifetime prevalence: Age 7 = 19.14% Age 11 = 18.3%	0	No difference by parent occupation in wheezing prevalence.
H. R. Anderson et al. (1983b)	7-11 376	Case control. All children with wheeze or asthma born during 1 year in London. Randomly selected control group born during same year.	Maternal occupation Car ownership Housing Group membership: controls, children who missed 0-10 days of school because of wheezy illness, and children who missed more than 10 days of school	+ - - 0	Higher percentage of children with wheezy illness who missed > 10 days of school had mothers with nonmanual occupations compared to controls and those who missed 0-10 days. Significant after controlling for home ownership. Higher percentage of children with wheezy illness who missed > 10 days of school lived in rented homes compared with children who missed 0-10 days of school. Significant after controlling for maternal occupation, car ownership, marital status, number of children. Higher percentage of children who missed > 10 days of school lacked access to a car compared with those who missed 0- 10 days of school. Significant after controlling for marital status, number of children, but not car ownership. No differences by education.

Note. Age range is given in years. SES = socioeconomic status; + = positive association of SES and asthma (i.e., higher SES children having higher asthma rates); 0 = no association of SES and asthma; - = negative association of SES and asthma.

Table 4 (continued)

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Greenlund et al. (1997)	Approximately 9–12 (grades 3–6) 933	Population based. Sample of children in four grades in one city in Louisiana (Bogalusa Heart Study).	Maternal education Probability of ever having smoked	0	No association of maternal education with probability of ever having smoked when gender, ethnicity, school grade, family and friend smoking status, and attitudes about smoking controlled.
Elder et al. (1988)	10–14 433	Population based. Students in four out of five San Diego elementary and middle schools that held summer classes.	Paternal employment Composite variable of parent employment, home ownership, and marital status	–	Fathers not being employed associated with greater amount of smoking.
Lowry et al. (1996)	12–17 6,321	Population based. Random sampling of adolescents to provide representative sample of U.S. population (supplement to the National Health Interview Survey).	Amount of smoking in past week/day Parent education Family income Prevalence rate of current smokers: 19.6% are current smokers	–	Children of lower educated parents had higher smoking rates. Significant after controlling for age, sex, race, school status. Monotonic effect apparent. Significant in multiple regression (with years of education).
Petridou et al. (1997)	12–17 547	All children in several low or high SES high schools in Greece.	Paternal education Neighborhood type (low vs. high SES school) Risky behavior composite (smoking, drinking, seat belt use, etc.)	–	Children with lower family incomes had higher smoking rates. Significant after controlling for age, sex, race, school status. Monotonic effect is apparent. Significant in multiple regression (income by \$1,000).
De Vries (1995)	12–16 1,922	Population based. Sample of Dutch students from vocational and high schools. Sampling strategy not specified.	Parent occupation Prevalence of current smokers	–	Lower paternal education was correlated with higher scores on the risky behavior composite. Significant after controlling for neighborhood type, age, gender, school performance.
Donato et al. (1994)	14–15 5,221	Population based. Cluster sample of 9th graders from high schools in 10 Italian towns.	Parent occupation Parent education Smoking status Prevalence of current smokers = 14.3%	0	Living in poorer neighborhoods was correlated with higher scores on the risky behavior composite. Significant after controlling for parent education, age, gender, school performance.
Green et al. (1991)	15 870	Random stratified sampling of children born during 1 year in one area of Scotland. Only two-parent households used in this study.	Parent occupation Prevalence of current smokers 12.5% of children average \geq one cigarette per day	–	Higher percentage of children of parents from lower occupational classes smoked compared with children of parents from higher occupational classes.
Stanton et al. (1994)	15, 18 696	Population based. All children born in one hospital in 1 year in New Zealand. This study included those available for follow-up at ages 15, 18.	Parent occupation Odds of being a daily smoker Prevalence of daily smoking, age 15 = 15% Prevalence of daily smoking, age 18 = 31.4%	–	Higher percentage of adolescents of parents with manual occupations smoked compared with adolescents from nonmanual household. Odds ratio = 1.6, significant after controlling for gender, parent smoking status.
Bergstrom et al. (1996)	14, 17 751	Population based. All students in two grades of selected schools in one city in Sweden.	Parent occupation Prevalence of daily smokers Prevalence, age 14 = 1% Prevalence, age 17 = 8.5%	–	Odds of children of parents with unskilled occupations being a daily smoker was 2.7 times greater than for children of parents of higher occupational class. Significant after controlling for gender, ethnicity, family size, neighborhood, marital status. However, parent occupation did not predict change in smoking status by age 18.
Borland & Rudolph (1975)	Approximately 14–18 (high school) 1,814	Population based. All children in one high school in Pennsylvania.	Paternal occupation Prevalence of current smokers Prevalence = 34.6%	0	Percentage of teenagers who were daily smokers was higher among lower occupational households than higher occupational households. Odds ratio for lowest SES group = 4.5. Not significant after controlling for child education, gender, parent or friend smoking status. No significant differences by education.

(table continues)

Table 4 (continued)

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Tuinstra et al. (1998)	Approximately 14–18 (high school) 1,984	Population based. Random sample of students from 18 schools in four provinces in the Netherlands.	Parent education Parent occupation Smoking status Prevalence of daily smokers = 35.7%	0	No differences in smoking status by parent education or occupation.
Heath et al. (1991)	17–18 2,913	Population based. U.S. adolescents living in households that participated in the National Health Interview Survey.	Child educational status Prevalence of current smokers Prevalence = 15.7%	–	Prevalence of smoking higher among high school dropouts compared with attenders/graduates.
Escobedo et al. (1990)	18–35 17,887	Population based. Multistage design to obtain representative sample of U.S. households (National Health Interview Survey and Hispanic Health and Nutrition Examination Survey).	Child educational status Rate of smoking initiation based on recall of age at which started smoking	–	Individuals with lower educational attainment had higher smoking initiation rates throughout childhood compared with those with higher educational attainment. From age 11 through approximately 17, smoking initiation showed a more rapid acceleration among individuals with low educational attainment.
Ary & Biglan (1988)	Approximately 13–16 (grades 7, 9, 10) 801	Population based. All children in three grades of selected middle and high schools in Oregon.	Parent education Housing Smoking rate (number of cigarettes smoked in past week)	0 –	Middle school teenagers showed no difference in smoking rate by parent education or house crowding. High school teenagers who lived in more crowded houses had higher smoking rates compared with teenagers in less crowded houses. Significant after controlling for family and friend smoking status, other substance use.
Mittelmark et al. (1987)	Approximately 12–17 (grades 7–11) 1,101	Population based. Survey of school children in Minnesota (Childhood Antecedents of Smoking Study).	Parent education Smoking status Prevalence of smokers = 19.4%	0 –	Among 13–14-year-olds, parent education was unrelated to smoking status in discriminant analyses. Among 15–17-year-olds, parent education discriminated whether adolescents smoked during the study period or were nonsmokers throughout study period.
Leino et al. (1996)	9, 12, 15 1,211	Population based. Randomly chosen children in each age group from five cities (and surrounding rural communities) in Finland.	Paternal occupation Prevalence of daily smokers	–	Higher percentage of 9-, 12-, and 15-year-old boys whose parents belonged to lower occupational classes were daily smokers compared with boys whose parents had higher occupational status.
Byckling et al. (1985)	15, 18 1,089		Prevalence of daily smoking for girls = 5.3% Prevalence for boys = 9.8%	–	Among girls, higher percentage of low SES teenagers were daily smokers at age 18, but not at age 15 compared with high SES teenagers.
Pirie et al. (1988)	Approximately 16–18 (grades 11–12) 6,241	Population based. Random sample of 7th graders in four suburban districts in one city in Minnesota. Those available for follow-up 5 years later participated in this study	Child educational status Prevalence of daily smokers Prevalence among females = 27.0% Prevalence among males = 21.7%	0 –	Percentage of daily smokers higher among those who had dropped out of school compared with those who had not. Among boys, differences appeared greater for 18-year-olds than 16-year-olds. Among girls, differences only slightly greater for 18-year-olds than 16-year-olds.
Chassin et al. (1992)	Approximately 12–17 (grades 6–11) 1,937	Nonsmokers from a sample of all children in one midwestern county school system.	Parent education (measured in follow-up study after smoking status evaluated) Change in smoking status over 1-year period Odds of being a current smoker	– –	Cross-sectionally, lower parent education associated with smoking among both middle and high school students. Among those in middle school, higher percentage of children from less educated families started smoking over 1-year period compared with children from well-educated families. Significant after controlling for gender.
Pederson & Lavik (1991)	12–18 1,230	Population based. Longitudinal study of secondary school children in one city in Norway.	Paternal education Odds of being current smoker Change in smoking status over 2-year period Prevalence among females = 37.7% Prevalence among males = 26.4%	– 0 0	Among those in high school, no differences in smoking initiation by parent education. Among 12–15-year-olds who did not smoke at study entry, odds of becoming current smoker was 3.3 times greater for children of lower educated fathers relative to children of highly educated fathers. Significant after controlling for gender, family and friend smoking status. Among 15–18-year-old never smokers, paternal education was unrelated to change in smoking status. No significant cross-sectional associations after parent and peer smoking controlled.

Note. Age range is given in years. SES = socioeconomic status; + = positive association of SES and smoking (i.e., higher SES children having higher smoking rates); 0 = no association of SES and smoking; – = negative association of SES and smoking.

Table 5
 Summary of Studies That Investigate SES Effects on Blood Pressure (BP) by Specific Age Groups

Study	Age range/ sample size	Study design/sampling strategy	SES measure	Direction of finding	Description of finding
Simpson et al. (1983)	7 982	Population based. All children born in one hospital in New Zealand over a 1-year period. Children recontacted at age 7.	Paternal occupation measured at age 5	0	No SES differences in BP.
Boulton et al. (1995)	8 856	Population based. Initial sample of 2,000 children born in one hospital in Australia. Children were recontacted 8 years later for this study.	Parent education Paternal occupation	-(s, D) 0	Children of mothers with lower education had higher SBP and DBP than children of mothers with higher education. No controls, but authors concluded that SES is more important than family history of heart disease in predicting blood pressure. No BP differences by paternal occupation.
Gillum et al. (1985)	7-10 1,505	Population based. All children in three grades in Minnesota public schools targeted initially. Those selected for study included: all children ≤ 5 and > 95 percentile in BP, 1/2 of remaining African Americans, and 1/9 of remaining Caucasians.	Parent education Parent occupation Family income	-(s) 0	Children whose mothers were from lower occupational classes had higher SBP compared with children of high occupation mothers. Significant after controlling for child age, gender, body mass index. No BP differences by education or income.
Walter & Hofman (1987)	Approximately 9 (Grade 4) 2,2591	Population based. All children in one grade within targeted school districts in New York City.	Neighborhood type	-(D)	Children from low SES neighborhood had higher DBP than children from high SES neighborhood. Significant after controlling for child's age, gender, ethnicity, height, triceps, and ponderosity index.
Zuckerman et al. (1989)	Approximately 9-13 (Grades 4-6) 1,041	Population based. Recruited African American children in three grades in District of Columbia public schools.	Neighborhood type (percentage of families eligible for federal assistance)	-(s, D)	Children from lower SES neighborhoods had higher SBP and DBP than children from high SES neighborhood. Significant after controlling for child's age, gender. Also, a higher percentage of low, compared with high SES, children exceeded the risk value for DBP (> 95 percentile).
Whincup et al. (1994)	9-11 1,311	Population based. All children from targeted junior high schools within two cities in Britain.	Parent occupation Maternal education Housing	-(s, D) -(D)	Monotonic effect, whereby decreases in parent occupational class associated with increases in SBP and DBP. Significant linear trend. Significant after controlling for child age, sex. Monotonic effect, whereby decreases in maternal education associated with increases in DBP. Significant linear trend. Significant after controlling for child's age, sex.
Bergstrom et al. (1996)	14, 17 751	Population based. All students in two grades of selected schools in one city in Sweden.	Parent occupation Parent education	0 0	No BP differences by housing status. No BP differences by parent occupation or education.
West et al. (1990)	15 1,009	Population based. Random stratified sampling of children born during 1 year in one area of Scotland.	Parent occupation	0	Among girls, no SBP or DBP differences by parent occupation. Among boys, no significant overall differences by parent occupation for SBP or DBP. However, boys from higher occupational households show a linear trend toward higher SBP compared with those from lower occupational households.

(table continues)

Table 5 (continued)

Study	Age range/ sample size	Study design/sampling strategy	SES measure	Direction of finding	Description of finding
L. B. Wright et al. (1998)	Average age = 11 268	Normotensive children with a family history of essential hypertension in Georgia.	Parent education Neighborhood type (median income, education, etc.)	-(D)	Lower SES 11-year-olds have higher DBP (and higher total peripheral resistance) than higher SES children.
Jackson et al. (1999)	Average age = 13.5 272			0	No resting BP differences by neighborhood SES for 13-year-olds.
Byckling et al. (1985)	3, 6, 9, 12, 15, 18 3,596	Population based. Randomly chosen children in each age group from five cities (and surrounding rural communities) in Finland.	Paternal occupation	0	Among girls, no BP differences by parent occupation for any age group. Boys whose fathers were farmers had highest SBP of any occupational group. This was true for 15-year-old boys, as well as for the sample of 9-, 12-, and 15-year- olds together. No DBP differences.
Leino et al. (1996)	9, 12, 15 1,211				

Note. Age range is given in years. SES = socioeconomic status; 0 = no association of SES and BP; - = negative association of SES and BP (i.e., lower SES children having higher BP); S = systolic; D = diastolic; SBP = systolic blood pressure; DBP = diastolic blood pressure.

drawn on the basis of these types of studies can sometimes be misleading in that they do not represent individual trajectories of change over time.

In addition, the state of research on developmental change in the SES and health relationship is such that many studies do not even include a wide age range of children but rather focus on the relationship between SES and health for a specific age group of children. We chose to include such studies in the present review because of the relative dearth of longitudinal studies or cross-sectional studies that present results for separate age groups. Thus, we assemble multiple studies, each with different age groups, to draw inferences about patterns of change for a particular health outcome. Given the available data, this was the most reasonable approach to exploring the question of how the SES–health relationship differs across childhood and adolescence. However, discrepancies across studies can confound conclusions. For example, differences in the magnitude of the SES and health relationship across studies that appear to be due to age may reflect changes in the prevalence of an illness during the various decades in which these studies were conducted (cohort differences). This is particularly relevant with respect to asthma, whose prevalence has risen dramatically in recent decades (Weitzman, Gortmaker, Sobol, & Perrin, 1992). In addition, differential sampling of participants across studies may also bias SES results. For example, SES differences that appear to be due to age may actually reflect different sampling strategies across age groups (e.g., pediatrician offices for younger children vs. schools for older children). Although this bias is not likely to affect outcomes such as hospitalizations (where data tend to be uniformly drawn from hospital records; e.g., Huang & Joseph, 1999), it could affect studies of prevalence rates. These factors should be considered when interpreting the results below.

A second issue that arises relates to biases that may influence the measured health outcomes. For example, low and high SES individuals may differ in perceptions of symptoms and care-seeking behaviors. If one examines disease onset and if high SES parents are more likely to notice symptoms earlier and take their children to the doctor, then these children may be diagnosed with certain diseases at a higher rate. That is, the true incidence of some diseases may be underestimated in low SES children because of delays in care-seeking behaviors. This may in part account for why some diseases (e.g., myopia, allergies) show a reverse SES and

health effect. These biases, however, should lead to conservative estimates of the magnitude of the SES and health effect.

A third issue relates to the variety of ways in which a particular health outcome can be assessed. Even within the same health outcome, relationships between SES and health may vary depending on how that outcome is assessed. For example, prevalence rates may have different meaning from disease severity measures. As a hypothetical example, SES effects may emerge for only severe asthma because high SES parents have the psychological and tangible resources to manage their child's asthma and thus their children are less likely to develop severe cases of asthma. Or it may be because high SES children are more likely to get diagnosed with mild, but not severe, forms of asthma. Distinguishing among various types of outcomes for the same health problem allows us to be more specific in our models as well as in postulating mechanisms for these relationships. Thus, where relevant, we review SES findings separately for onset versus severity of a disease outcome. It should also be noted that the distinction between prevalence and incidence rates may affect SES relationships. Incidence rates refer to new cases of an illness, whereas prevalence rates refer to all individuals who have an illness at the time of questioning, regardless of when it began. In cases in which SES is thought to play an etiologic role, one might expect an effect for incidence rates. In contrast, in cases in which SES is hypothesized to affect treatment or compliance, one might expect an effect for prevalence rates. Although this distinction is important, we do not emphasize it in the remainder of this review because most studies do not distinguish between the two. One exception is that for injuries (which last very briefly, unlike chronic illnesses), we use the term *incidence* to refer to prospective studies (e.g., of new injuries over the study period) and *prevalence* to refer to retrospective studies (e.g., lifetime number of injuries).

Alternative explanations. Rather than associations being due to the influence of SES on health, it is possible that the opposite relationship is true, that health influences SES. This social selection hypothesis suggests that poorer health may drain one's financial resources, leading to lower SES levels. With respect to SES among children, the burden of childhood illness could lead to lower parental income over time, or adolescents' own SES could drift downward as they become adults. Research in the mental health domain has found that both social causation (SES influenc-

Table 6
 Summary of Studies That Investigate SES Effects on Physical Activity by Specific Age Groups

Study	Age range/ sample size	Study design/sampling strategy	SES measure/outcome	Direction of finding	Description of finding
Kikuchi et al. (1995)	8–9 581	Population based. Random sampling of school children born during 1 year in Britain.	Paternal occupation Maternal education Completion of physical fitness test	0	Neither parent occupation nor education predicted which children were able to complete the test. Controls included height, SBP, skinfold, mother smoking, family history of heart attack.
Zuckerman et al. (1989)	Approximately 9–13 (Grades 4–6) 1,041	Population based. Recruited African American children in three grades in District of Columbia public schools.	Neighborhood type (percentage of families eligible for federal assistance) Recovery from physical fitness test	+	High SES children were less physically fit (showed less recovery after fitness test) than low SES children. Significant after controlling for age and gender.
Slooten et al. (1994)	10–12 56	Randomly selected healthy school boys from Bolivia. Selected from two SES groups and two residential altitudes.	Not specified Ambulatory heart rate: percentage of time spent at 50–85% of heart rate reserve (maximum minus sleeping heart rate)	+	High SES boys were less physically active (spent less time at 50–85% of heart rate reserve) than low SES boys.
Fuchs et al. (1988)	Approximately 12–14 (Grades 7–8) 932	Randomly selected schools within two cities in Germany. Only high or low SES schools were selected.	Neighborhood type (low and high SES school) Time spent for physical activity	– +	Low SES boys reported spending less time engaged in vigorous activities than high SES boys. However, low SES boys spent more time in moderate activities than high SES boys.
Gottlieb & Chen (1985)	12–14 (Grades 7–8) 2,695	Population based. Random sampling of school children in Texas.	Paternal occupation Frequency of exercise (participation in sport activities)	–	Children of low occupation parents had lower frequency of exercise than children of high occupation parents. Significant after controlling for gender and ethnicity.
Leino et al. (1996)	9, 12, 15 1,211	Population based. Randomly chosen children in each age group from five cities (and surrounding rural communities) in Finland.	Parent occupation Composite representing intensity, duration, and frequency of physical exercise	–	Monotonic effect whereby decreases in parent occupational class associated with decreases in physical activity among boys only.
Sallis et al. (1996)	11–19 (Grades 9, 11) 1,871	All children from certain grades in schools in San Diego in high and low SES districts.	Neighborhood type (low and high SES school) Total frequency of vigorous exercise Number of physical education classes per week	–	Low SES Caucasians reported less total frequency of vigorous exercise, and attended fewer physical education classes per week than high SES Caucasians.
Bergstrom et al. (1996)	14, 17 751	Population based All students in two grades of selected schools in one city in Sweden.	Parent occupation Parent education Time on physical fitness test (running) Physical activity (not clearly defined)	– 0	Higher percentage of girls whose parents had low education or occupational status had poorer physical fitness compared with girls of parents with high education or occupations. No differences in physical activity, although this was not clearly defined.
Tuinstra et al. (1998)	Approximately 14–18 (high school) 1,984	Population based Random sample of 18 schools in four provinces in the Netherlands.	Parent education Parent occupation Participation in sports Percentage who do not participate in any sports = 26.1%	– –	Children of parents with lower educational attainment have lower participation rates in sports. Trend appears monotonic. Children of parents with lower occupational status have lower participation rates in sports.
Lowry et al. (1996)	12–17 6,321	Population based Random sampling of adolescents to provide representative sample of U.S. population (supplement to the National Health Interview Survey).	Parent education Family income Prevalence rate of adolescents with sedentary lifestyle (exercising or participating in sports < 3 of last 7 days) 35.6% were sedentary	– –	Children of lower educated parents had higher rates of sedentary lifestyle. Significant after controlling for age, sex, race, school status. Significant in multiple regression (with years of education). Children with lower family incomes had higher rates of sedentary lifestyle. Significant after controlling for age, sex, race, school status. Monotonic effect is apparent. Significant in multiple regression (income by \$1,000).
Aaron et al. (1993)	12–16 1,245	Population based All children within three grades in two school districts in Pittsburgh.	Neighborhood type (percentage of families below poverty) Hours per week spent doing physical activity	0	No significant SES differences in physical activity, although trend toward decreased physical activity with lower SES in girls.

Note. Age range is given in years. SES = socioeconomic status; 0 = no association of SES and physical inactivity; SBP = systolic blood pressure; + = positive association of SES and physical inactivity (i.e., higher SES children having greater physical inactivity); – = negative association of SES and physical inactivity.

ing onset of psychiatric disorders) and social selection (psychiatric disorders causing individuals to drift downward in SES) occur, depending on the outcome of interest. For example, longitudinal studies suggest that social causation plays a more important role than social selection for anxiety, depression, and personality disorders in youths and adults. In contrast, social selection is more important for substance use disorders in youths and for schizophrenia in adults (Dohrenwend et al., 1992; Johnson, Cohen, Dohrenwend, Link, & Brook, 1999). These findings suggest that it is important to conduct longitudinal studies to determine whether certain physical health outcomes fit better with one model or the other.

It is also possible that the relationship between SES and health is actually due to some third variable. As suggested by Adler et al. (1994), genetically based factors may lead to both low SES and poor health. Among children, this could manifest as a genetic vulnerability for a disease that is passed from parent to child. Thus, associations of parent SES with childhood disease prevalence rates could be due to children having their parents' SES as well as an inherited vulnerability for a disease, rather than environmental adversity being causally associated with the onset of a disease. This explanation may be more probable for certain outcomes that have a significant heritable component (e.g., asthma or blood pressure, but not injury; Dewar & Wheatley, 1996; Hunt et al., 1989; R. R. Williams et al., 1991). The complex relationships among genes, environment, and behavior suggest not only that genetic dispositions influence behavior and environmental exposures but also that environmental exposures shape genetic expression (Liu et al., 1997; Plomin & Crabbe, 2000). In addition to relationships with SES and health, genetic factors are likely to affect the psychosocial mediators (e.g., social relationships) that we propose below (Cacioppo, Berntson, Sheridan, & McClintock, 2000). Thus, it is important to recognize that genetic factors likely influence or interact with all aspects of our proposed mechanistic model, although we do not explicitly discuss the role of genes throughout the rest of this review.

Injury

Studies. Injury causes more deaths during childhood than all other causes combined (Rivara & Mueller, 1987). Additionally, injuries are the leading cause of years of productive life lost (Guyer & Eilers, 1990). The studies that we review below emphasize unintentional injuries, as opposed to injuries stemming from violence. However, it is possible that some acts of abuse or violence are unknowingly included in these figures. Higher rates of child abuse have been found in low SES families (Cappelleri, Eckenrode, & Powers, 1993; Jones & McCurdy, 1992); however, it is impossible to estimate the extent to which the injury relationships described below are due to unreported abusive or violent acts.

In early childhood (ages 7 and younger), there appears to be a strong relationship between SES and injury, such that lower SES is associated with higher injury rates (see Table 2). We found associations with a variety of SES measures, including parent occupation, employment, and housing status at an individual level, as well as neighborhood measures of SES. These associations were found both for incidence of any type of injury as well as incidence or prevalence for more serious (medically attended) injuries. Five

studies that included children aged 7 and younger found support for an inverse SES and injury relationship, with only one study finding no relationship between SES and injury at a young age.

In adolescence, there is some evidence that the SES effect is minimal (see Table 2). Among children aged 11–18, two studies found no association between parent occupation, family affluence, or neighborhood type and incidence of injury. One other study also found no SES relationship with number of injuries or lifetime prevalence rates among girls; however, an inverse association was found for boys for lifetime prevalence rates but not number of injuries.

Among studies that reported injury rates by age, five found support for a childhood-limited pattern of SES differences among younger, but not older, children. This pattern was found for both family SES indicators, such as parent occupation and education, and neighborhood SES measures. It applies to both prevalence rates of injuries as well as severity (e.g., death due to injury). Two studies found support for a persistence pattern of inverse SES and injury relationship in both childhood and adolescence. However, one of these studies included only injuries due to motor vehicle crashes (Pless, Verreault, Arsenault, Frappier, & Stulginskas, 1987), which may represent a different cohort of children from other studies. The second study assessed lifetime injury rates for children aged 0–4 and 5–15 (Marsh & Channing, 1987). It is possible that injuries to the 5–15 age group may have happened primarily during their early years of life. If this were true, the negative association of SES with injury for the older group might actually reflect an early childhood phenomenon. No data reported in this article addressed the timing of injuries in either group, and thus it is unclear which type of developmental trajectory more accurately fits this study.

Summary. Overall, these studies indicate preliminary support for an SES and injury relationship that follows the childhood-limited model. A traditional SES monotonic effect is present until approximately age 7. From age 11 on, significant relationships between SES and injury are not typically found. Of those studies of children aged 7 and younger that reported odds ratios, the odds of low SES children being injured ranged from 1.5 to 4.2 times higher than for high SES children. Rates of injury vary by study but on the whole increase with age (see Table 2), suggesting that decreases in the strength of the SES and injury relationship are not due to decreases in the prevalence of injuries with age. Longitudinal studies of injuries throughout childhood and adolescence need to be conducted for more definitive conclusions to be reached.

Note that SES differences by age may reflect changes in common causes of injury across age groups. Injuries due to drowning or fire are more common early in life, whereas injuries due to motor vehicle crashes are more common during adolescence (Rivara, 1995). Thus, it may be that SES differences exist for drowning or fire injuries, but not for motor vehicle crashes because high SES adolescents are more likely to own cars (thus making them more vulnerable to this type of injury). Supporting this, higher SES children have been found to be more likely to experience sports injuries, presumably because they can afford to play more sports (Lyons et al., 2000). In our review on injury, we were not able to elaborate on causes of injuries because many studies either depicted SES differences across all types of injuries or reported

causes of injuries not separated by SES, and thus we have very little information available about SES, age, and cause of injury.

Asthma

Asthma is the most common chronic illness in childhood. It accounts for approximately 25% of all limitations of activity in childhood (Newacheck, Budetti, & Halfon, 1986). In addition, hospitalizations for asthma are becoming more and more common. Between 1979 and 1987, asthma hospitalizations increased by 4.5% per year for children in the United States (Gergen & Weiss, 1990).

Prevalence rates. At younger ages, there is evidence to support the traditional SES relationship with respect to prevalence of asthma and related respiratory illnesses (see Table 3). Among children aged 9 and younger, family SES indicators, including parent occupation, type of housing, and poverty status, are associated with asthma and other respiratory illness prevalence rates. Of study samples containing children primarily aged 9 and younger, five found support for lower SES being associated with higher prevalence rates. Two studies found no association of SES with asthma or wheezing prevalence rates, particularly when parent education was measured (Horwood, Fergusson, & Shannon, 1985; Italian Studies on Respiratory Disorders in Childhood and the Environment, 1997).

Among children aged 9 and older, the traditional SES and health associations are not apparent. In fact, among older children, SES appears to have either a reverse association or no association with prevalence rates of asthma and related respiratory illnesses (see Table 3). Of the eight studies we found that examined asthma prevalence rates in children aged 9 and older, none found an inverse relationship between SES and asthma. Four found no association with asthma prevalence rates when parent occupation, education, or income was measured. Three found a positive association of higher SES being associated with higher asthma or other respiratory illness prevalence rates when parent occupation or education was measured. Note that two studies included a group of children that equally bridged the younger and older age ranges (ages 7–11). These studies reported mixed findings (positive, negative, and no relationship, sometimes within one study), possibly because of the age range the authors recruited (see Table 3).

Lastly, several studies examined the relationship between SES and asthma prevalence rates by age. Two of these studies found support for a childhood-limited pattern, in which an inverse SES and asthma relationship is found at younger ages and no relationship (or a positive relationship) is found at older ages. One of these studies found that children aged 0–5 living below the poverty level had higher asthma prevalence rates than nonpoor children. However, among children 6–11 and 12–17, there were no differences in asthma rates between poor and nonpoor children (Halfon & Newacheck, 1993). A second study found that among 5-year-old children, low SES was associated in a monotonic fashion with a higher odds ratio of wheezing; in contrast, among 16-year-old teenagers, higher SES was associated with a higher odds ratio of wheezing (Lewis, Richards, Bynner, Butler, & Britton, 1995).

One study broken down by age found no relationship between type of neighborhood and asthma prevalence rates among first graders or sixth graders (Mak, Johnston, Abbey, & Talamo, 1982); however, all analyses in this study were conducted controlling for

race. Given that race and SES are often highly correlated, this approach may have led to the null SES findings. One other study examined the relationship between SES and asthma or wheezy bronchitis among 7-, 11-, and 16-year-olds. Several articles were published from this sample, with very mixed findings. Various associations were found, with support for a childhood-limited model apparent when housing amenities were examined, but support for a reverse persistence model (higher SES having higher rates of asthma) present when parent occupation is used (H. R. Anderson, Bland, Patel, & Peckham, 1986; Kaplan & Mascie-Taylor, 1985, 1988; Peckham & Butler, 1978; Strachan, Butland, & Anderson, 1996).

Asthma severity. Although there are fewer studies of asthma severity, those that exist are consistent in their support of a relationship between lower SES and more severe asthma throughout childhood and adolescence (see Table 3). Among 6- to 7-year-old children, lower parent education is associated with greater prevalence of asthma hospitalizations as well as more severe nocturnal wheezing attacks (Italian Studies on Respiratory Disorders in Childhood and the Environment, 1997). Among children aged 9 and older, a similar relationship exists. Whether measured by parent occupation or education, lower SES is associated with more severe asthma in the two studies that examined this age group (Dawson, Horobin, Illsley, & Mitchell, 1969; Mielck, Reitmeir, & Wjst, 1996; R. G. Mitchell & Dawson, 1973).

In addition, two studies examined asthma severity broken down by age (see Table 3). Both of these studies found support for a persistence model, in that an inverse relationship existed between SES and asthma hospitalization rates during both childhood and adolescence. Both studies used neighborhood measures of SES (Huang & Joseph, 1999; Watson, Cowen, & Lewis, 1996).

Summary. The relationship between SES and asthma presents a more complicated picture than that of childhood injury. In regard to prevalence rates of asthma, evidence suggests that the traditional relationship of lower SES and higher asthma prevalence rates holds until approximately age 9 and that this relationship follows a monotonic pattern. Among older children and adolescents, this relationship does not exist, in fact, a reverse SES relationship is evident in some studies. On the basis of this pattern, there is preliminary support for the childhood-limited model for SES and asthma prevalence. Among studies that reported odds ratios for younger children, low SES children are 1.3 to 3.8 times as likely to have asthma as high SES children. Although prevalence rates vary by study, overall the prevalence of asthma does not appear to change much from childhood through adolescence (roughly 7%). This suggests that the changing pattern of the SES and asthma relationship is not likely to be due to changes in asthma prevalence rates with age.

The existence of a childhood-limited or crossover pattern for childhood asthma prevalence rates also may help explain some mixed findings in the literature. A number of asthma studies that have included a wide age range of children have found no association of SES with asthma or wheezing (Charlton, Hartley, Silver, & Holland, 1983; Gergen, Mullally, & Evans, 1988; Higgins & Britton, 1995; Moussa, Skaik, Yaghy, Salwanes, & Bin-Othman, 1996). If, however, the developmental trajectory of SES and asthma follows a crossover pattern, collapsing across wide age ranges might produce such null results. Thus, these studies may

have inadvertently obscured the various relationships between SES and asthma during different periods of childhood.

In contrast with asthma prevalence rates, severe asthma appears to consistently display the traditional SES relationship across childhood. That is, lower SES is associated with higher rates of severe asthma among both children and adolescents. On the basis of this pattern, there appears to be preliminary support for the persistence model for SES and severe asthma. The one study that reported odds ratios for severe asthma found that low SES children are 2.4 times as likely to have severe asthma as high SES children (Mielck et al., 1996). The possibility should be noted that the higher asthma hospitalization rates in lower SES children could reflect lack of health insurance and the use of the emergency room in lieu of a primary care physician, rather than more severe asthma per se. Nonetheless, studies that find higher mortality rates due to asthma in low SES children (Vagero & Ostberg, 1989) provide evidence that cases of asthma in low SES children are indeed more severe. Again, it should be noted that future studies incorporating longitudinal designs will allow more definitive conclusions about model fit to be drawn.

Risk Factors Related to Cardiovascular Disease

Cardiovascular disease causes more deaths across the life span than any other disease. The traditional risk factors for cardiovascular disease include cigarette smoking, high blood pressure, and physical inactivity. There is evidence that the presence of these risk factors during childhood is associated with later indicators of cardiovascular disease (Berenson, et al., 1992, 1998). There is also evidence that exposure to passive smoke increases the risk of coronary heart disease (He et al., 1999). We thus review SES differences related to these risk factors during childhood.

Smoking. Most adults who smoke begin in adolescence, with 89% first smoking before the age of 18 (Giovino, Henningfield, Tomar, Escobedo, & Slade, 1995). Earlier age of onset of smoking has greater risks, as it is associated with increased DNA damage in the lung later in life (Wiencke et al., 1999). In addition, this habit tends to persist from childhood to adulthood: 63% of adolescents who smoked at age 14 continued to smoke at age 24 (Pietila, Hentinen, & Myhrman, 1995). Throughout childhood and adolescence, a traditional SES relationship can be found with respect to both prevalence of childhood smoking as well as passive exposure to tobacco smoke (see Table 4). Among children aged 12 and younger, lower SES is associated with both increased exposure to smoke as well as higher rates of current childhood smoking. These associations hold for a variety of individual-level SES measures, including parent education, occupation, and type of housing. Six studies found support for an inverse relationship of SES and smoking or exposure to smoke. One study found no SES and smoking relationship, although all analyses in this study controlled for race (among other variables), which may have shared a substantial amount of overlap with SES (Greenlund, Johnson, Webber, & Berenson, 1997).

From age 12 on, evidence is also strong that low SES is associated with higher smoking rates (see Table 4). Smoking is associated with individual-level SES measures, including parent education, occupation, and family income. Smoking is also associated with neighborhood SES measures. These associations are found both for being a current smoker as well as for being a daily

smoker. Among studies that investigated adolescents primarily 12 and older, 9 of 11 found support for an inverse SES and smoking relationship.

Among studies that investigate smoking rates in separate age groups, evidence suggests that SES differences may be stronger in older age groups. Adults of low educational achievement report beginning to smoke earlier in childhood than adults of high educational achievement. Moreover, the ratio of low SES to high SES smokers increases steadily from approximately age 11 to 17 in the United States (Escobedo, Anda, Smith, Remington, & Mast, 1990). Lower SES was not associated with smoking status among middle school students but was associated among high school students in the United States (Ary & Biglan, 1988; Mittelmark et al., 1987; for girls only, see Byckling et al., 1985). Differences in percentage of daily smokers between teenagers who had dropped out of school and those who had not appeared greater among 18-year-olds than 16-year-olds (Pirie, Murray, & Luepker, 1988). Among studies that broke their sample down by age, five found support for a pattern of SES differences in smoking prevalence rates that is most apparent at older ages. This would be consistent with an adolescent-emergent model. In contrast, two studies found support for a pattern of differences in early but not late adolescence; however, both of these studies examined changes in smoking status (going from nonsmoker to smoker) over time (Chassin, Presson, Sherman, & Edwards, 1992; Pederson & Lavik, 1991). The discrepancy in findings may make sense if most low SES adolescents start to smoke in the early teenage years. Prevalence rates of smoking reflect numbers of smokers, without regard to when they began smoking; this explains SES differences throughout childhood and adolescence. In contrast, changes in smoking status reflect numbers of new smokers, among the population of adolescents who have never smoked before. Thus, if most low SES adolescents who are going to smoke have already done so in their early teenage years, there may be no differences by SES in smoking initiation rates in the late-teenage years.

Smoking studies: Summary. Taken together, these studies indicate that the SES and smoking relationship is present at an early age and remains during adolescence. Some evidence suggests that the relationship is strongest in late adolescence. On the basis of this pattern, a variant of the adolescent-emergent model in which SES differences appear early in childhood, but may be most pronounced in adolescence, may best represent the findings from these studies. Among studies that examined children younger than 12 years, the odds of low SES children smoking or being exposed to smoke ranged from 1.4 to 2.5 times greater than for high SES children. Among studies that examined children 12 and older, the odds of low SES children smoking ranged from 1.6 to 4.5 times greater than for high SES children. Prevalence rates of passive exposure to smoke are quite high (approximately 45%, all European studies), and prevalence rates of childhood smoking increase with age (Table 4). Thus, it is possible that the pronounced SES and smoking relationship in adolescence is related to the increased prevalence rate of smoking with age.

Blood pressure. High blood pressure in childhood is a risk factor for high blood pressure in adulthood (Lauer, Burns, Clarke, & Mahoney, 1991; Zinner, Margolius, Rosner, & Kass, 1978), and an important marker of subsequent risk of cardiovascular disease (Berenson et al., 1992). Among children 13 years and younger, there is evidence for a traditional SES effect with respect to resting

blood pressure levels (see Table 5). Type of neighborhood shows the most consistent associations with blood pressure. In addition, parent education and occupation also show inverse associations with blood pressure. Five out of six studies in this age range found support for an association of lower SES with higher resting blood pressure.

In adolescence, however, the relationship between SES and resting blood pressure levels is not apparent (see Table 5). Adolescents ranging in age from 14 to 17 show no relationship between parent occupation or education and resting blood pressure levels. This lack of association was demonstrated in two studies.

Of the two studies that broke down findings by age, one study found support for a childhood-limited pattern. A longitudinal study of normotensive children revealed that when children were an average of 11 years old, there was an inverse association between SES and diastolic blood pressure. However, when children were an average of 13.5 years old, there was no association between neighborhood SES and blood pressure (Jackson, Treiber, Turner, Davis, & Strong, 1999; L. B. Wright, Treiber, Davis, Bunch, & Strong, 1998). The other study, conducted in Finland, found no relationship between paternal occupation and blood pressure among 3-, 6-, 9-, 12-, 15-, or 18-year-olds (Byckling et al., 1985; Leino et al., 1996).

Blood pressure: Summary. These studies indicate that the SES and blood pressure effect is present in early childhood, but not during adolescence. Overall, this pattern provides preliminary support for the childhood-limited model. The SES effect appears to be prominent until approximately age 13. During the teenage years, the relationship between SES and blood pressure is likely nonsignificant. We did not include prevalence rates or odds ratios for high blood pressure in Table 5 because these studies were all conducted with healthy children.

The SES pattern for blood pressure raises some intriguing interpretive questions, given that the traditional SES relationship with blood pressure is found in adulthood (Colhoun, Hemingway, & Poulter, 1998). How does one think about a risk factor that is associated with SES in childhood and in adulthood, but not in adolescence? Ideally, one would want to know whether blood pressure in low SES children improves in adolescence or whether blood pressure increases in high SES children during adolescence. Unfortunately, the longitudinal observations needed to answer this question either have not been conducted or have not been reported. Interpreting this SES pattern is further complicated by the fact that childhood blood pressure predicts adult blood pressure 20 to 30 years later (Lauer et al., 1991). Although it is unclear exactly how these various patterns fit together, West (1997) suggested that the general pattern of childhood SES differences that diminish in adolescence may be due to homogenization of youth through a common culture that crosses SES lines in school. This would suggest that the aspects of parental SES that influence blood pressure are present in early childhood, then weak in adolescence because of homogenization, and then strong in adulthood as these adolescents enter the workforce and become redistributed by SES.

Physical inactivity. Physical inactivity is another major risk factor for cardiovascular disease, and activity habits that are established during childhood are thought to continue throughout adult life (Sallis, et al., 1992). Seventy-one percent of adoles-

cents who did weekly physical exercise at age 14 continued to do so at age 24 (Pietila et al., 1995). Among younger children, the relationship of physical inactivity with SES is mixed (see Table 6). Two studies found that among children ranging in age from 9 to 13, lower SES boys were more physically active and fit (Slooten, Kemper, Post, Lujan, & Coudert, 1994; Zuckerman et al., 1989). However, one other study of 8- and 9-year-olds found no associations between parent education, occupation, or employment status and physical fitness (Kikuchi, Rona, & Chinn, 1995).

Among older children, there is evidence for a traditional SES relationship (see Table 6). From age 12 on, lower SES is associated with greater physical inactivity. This relationship holds primarily for parent occupation, with some evidence for relationships with parent education and family income. In addition, the relationship holds for neighborhood SES measures. Most of the associations are with measures of physical activity or exercise; however, one study also found differences in physical fitness among girls (Bergstrom, Hernell, & Persson, 1996). Among studies that tested adolescents aged 12 and older, five found support for an inverse relationship between SES and physical inactivity. One study found no association of type of neighborhood with amount of time spent doing physical activity (Aaron et al., 1993). No studies were found that tested the SES and physical inactivity relationship separately for various age groups.

Physical inactivity: Summary. There is evidence that a traditional SES and physical inactivity relationship exists only in adolescence. On the basis of this pattern, there is preliminary support for the adolescent-emergent model. That is, the relationship between SES and physical inactivity may be nonexistent (or even reversed) in early childhood, but a traditional relationship is present among those aged 12 and older, with lower SES adolescents reporting less physical activity. We did not report prevalence rates or odds ratios for physical inactivity in Table 6 because most studies reported duration or frequency of physical activity as a continuous variable.

Among children 12 and older, the relationship between SES and measures of physical activity is stronger in some studies for boys than for girls (Fuchs et al., 1988; Leino et al., 1996). Boys on average are more active than girls (Aaron et al., 1993), and thus it is possible that lower levels of or variability in activity among girls make it difficult to find statistical differences by SES. It should also be noted that some measures of physical activity are biased toward high SES children. That is, some activities (e.g., horseback riding) are unaffordable to low SES children, thus resulting in high SES children artificially appearing more active in these domains. However, the studies reported above typically contained questions about frequency or duration of any physical activity, which should be less open to bias.

In sum, there is evidence that the relationship of SES with cardiovascular risk factors is not equivalent throughout all periods of childhood. In fact, the strength of the relationship may vary depending on the type of cardiovascular risk factor being investigated. Although the studies we reviewed provide preliminary support for our models, longitudinal studies are needed to test how the SES and cardiovascular risk factor relationship changes within individuals as they age.

Other Factors Influencing the SES and Health Relationship

Other sociodemographic factors, particularly race, may confound the SES and health findings described above. For example, in the United States, African American and Hispanic families, on average, have lower income than Caucasian families. Within minority groups, the relative status of each group may fluctuate over time, depending on societal attitudes and immigration trends. On the whole, however, economic gains of minority groups in recent decades have been minimal compared with Caucasians (Karoly, 1992; D. R. Williams, Yu, Jackson, & Anderson, 1997). This is probably due in part to racism effects that restrict the economic benefits of African Americans, for example, who have the same educational level as Caucasians. These economic patterns are also associated with larger gaps in health and health care access between African Americans and Caucasians (see D. R. Williams, 1999). It is possible, then, that the negative health outcomes associated with SES are due primarily to race.

We explored this possibility by searching for studies of children that included both SES and race. These studies typically compare African American and Caucasian samples, with other minority groups not included or not large enough to make comparisons. Several studies that compared the effects of SES and race found that SES remains significantly associated with child health, even after controlling for race. For example, the odds of U.S. children below poverty being in fair or poor health was not significantly reduced when race was controlled. In contrast, the odds of African American children being in fair or poor health was significantly reduced when poverty status was controlled (L. E. Montgomery, Kiely, & Pappas, 1996). In addition, all-cause mortality rates in childhood are higher among lower income groups, even after race is controlled for. However, racial differences in childhood mortality rates are partially accounted for by income (Singh & Yu, 1996).

Because very few studies directly compare the effects of SES and race, we also explored whether SES and health relationships are similar among African Americans and Caucasians. Overall, studies show that when separated by race, SES effects are still present. Monotonic effects by income and parent education for childhood mortality rates have been found for both African American and Caucasian children (Mare, 1982; Wise, Kotelchuck, Wilson, & Mills, 1985). In addition, for some outcomes (fire-related mortality), differences are found by income but not by race (Wise et al., 1985). Low income and education also are associated with increased number of days missed from school among both African American and Caucasian children (McGaughey & Starfield, 1993).

When specific health outcomes are explored, similar monotonic effects are also found when separated by race. Lower SES Caucasians, African Americans, and Hispanics show higher smoking rates throughout adolescence compared with their respective higher SES counterparts, although the effects appear most pronounced for Caucasians (Escobedo et al., 1990). Blood pressure differences by parent education and neighborhood type are found for both African American and Caucasian children (Hunter, Frerichs, Webber, & Berenson, 1979; Walter & Hofman, 1987). Finally, lower SES children report greater physical inactivity than higher SES children. This relationship is maintained even when race is controlled for (Gottlieb & Chen, 1985).

One unusual race effect is that Mexican American women, despite lower SES, have rates of infant mortality and low-birth-weight children that are comparable with non-Hispanic Whites and are half that of African American women (Becerra, Hogue, Atrash, & Perez, 1991). The reasons for this finding are not entirely clear, although some researchers speculate that acculturation plays a role. That is, those Mexican American women who are less acculturated or integrated into U.S. society may have health behaviors that reflect their country of origin, which in this case are more health promoting (e.g., less fat in their diets, more extensive social support networks), resulting in lower risk of low-birth-weight babies (Cobas, Balcazar, Benin, Keith, & Chong, 1996; Collins & Shay, 1994; Zambrana, Scrimshaw, Collins, & Dunkel-Schetter, 1997). Although this review does not focus on the health of Hispanic families, this finding provokes thought about maternal mediators that may affect health early in a child's life.

Overall, these studies demonstrate that although race is an important factor in children's health, SES is not merely a proxy for race. Those studies in our review that investigated both race and SES effects are cited in Tables 2–6. However, it should be noted that we are not claiming that SES is a more important predictor of health than race. In fact, a number of researchers have documented that race effects persist when SES is controlled for (King & Palmisano, 1992; Winkleby, Robinson, Sundquist, & Kraemer, 1999). Taken together, these studies indicate that the effects of both SES and race are substantial and that each merits attention, both separately and combined, as factors that contribute to children's health. In particular, the question of whether the combination of low SES and minority status has synergistic effects on health needs to be addressed in future research. In addition, questions of the changing status of minority groups as well as the opportunities available to minority groups at each SES level need to be incorporated into future SES and health research. Finally, researchers in industrialized countries that are largely homogeneous in race have a unique opportunity to test SES effects and provide insight into the degree of overlap in SES and race findings for health.

What Are the Potential Mechanisms Linking SES to Health in Children?

A remaining issue is that of psychological and biological mediation and how experiences within SES strata are transduced into pathogenic processes influencing the incidence, severity, or course of childhood morbidities. A more robust understanding of such pathways may also guide the development of specific interventions to diminish social class effects.

In Figure 2, we present a conceptual framework for considering mechanisms influencing the SES and child health relationship. The essence of this model is that although mediational pathways remain similar from childhood to adolescence, normal developmental changes affect the degree of influence of each mediator across time. This dynamic model may help explain how SES developmental trajectories change through childhood and adolescence. For example, mediators that carry more weight during early childhood than adolescence would explain the existence of an SES relationship during early childhood that disappears by adolescence (childhood-limited model). Mediators that are stronger in adolescence would explain an SES relationship that may be present by

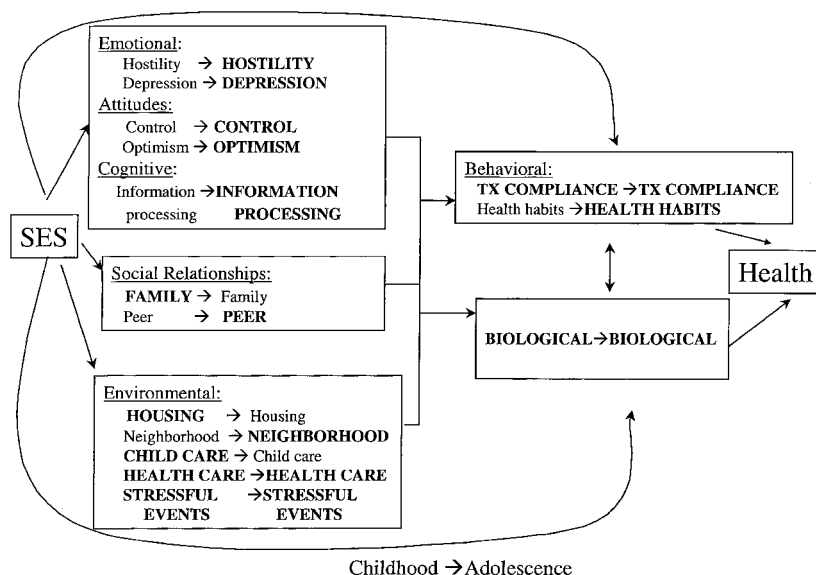


Figure 2. Model of potential mechanisms for the relationship between socioeconomic status (SES) and child health. The strength of each mediator changes over time as children undergo normal developmental changes. Each mediator is depicted twice, once to represent the period of childhood and once to represent the period of adolescence, with an arrow in between indicating developmental change in the mediator. Bolded mediators indicate a period of strong influence of that mediator; nonboldface mediators indicate a period of weaker influence for that mediator. Tx = treatment. It should be noted that, although not depicted, genetic factors likely influence each step in this model (SES, mediator, health outcome).

adolescence but not during childhood (adolescent-emergent model). Finally, mediators that are a strong influence throughout childhood and adolescence would explain a steady SES and health relationship (persistence model). In addition, this type of framework helps to explain why different health outcomes have different developmental trajectories. Health outcomes are influenced by different sets of mediators. If a health outcome were proposed to be influenced by a mediator that was pronounced in early childhood, one would predict a childhood-limited trajectory for that outcome. In contrast, if an outcome were thought to be influenced by a mediator prevalent in adolescence, one would predict an adolescent-emergent trajectory for that health outcome. This model is not meant to depict all possible pathways between SES and health but rather to portray examples of categories of mediators.

Below we discuss examples of each category of psychological and psychobiological mediators. Rather than being a review of the literature, we view this section as a guideline for future research. Very few studies have formally examined mediation; thus, we compile studies that test a factor's association with SES together with other studies that test that factor's association with health. This approach allows us to highlight the most promising factors for future research devoted to mechanism testing. Second, we also provide suggestions for how developmental changes in these factors might help explain the SES trajectories for the health outcomes reviewed above. These suggestions are highly speculative; however, our goal in this section is to lay a foundation for an approach to theoretically driven mediational research in this area. Future researchers would then need to empirically test the merits of this approach as well as the hypotheses below.

Prenatal Factors

Before discussing childhood mediators, we note that maternal factors during pregnancy can also have adverse effects on children's health. For example, lower SES women are more likely to smoke during pregnancy (Gazmararian, Adams, & Pamuk, 1996; Rutter & Quine, 1990; Simpson & Smith, 1986). In turn, smoking during pregnancy has been associated with higher infant mortality rates, lower birth weight, and slower growth in early childhood (Brooke, Anderson, Bland, Peacock, & Stewart, 1989; Elwood, Sweetnam, Gray, Davies, & Wood, 1987; U.S. Surgeon General, 1980). Controlling for smoking reduces the association between SES and low birth weight (Brooke et al., 1980; Kleinman & Madans, 1985). These prenatal factors may lead low SES children to begin life with poorer health. Nonetheless, childhood mediators may still play a role in increasing or decreasing these early-life disparities, along the trajectories suggested above. Our review focuses on mediators that emerge during childhood and adolescence because of our interest in developmental trajectories; as a result, we do not review prenatal or perinatal mediators below.

Emotional/Cognitive Mechanisms

Hostility. Lower SES is associated with higher levels of hostility among adults (Barefoot et al., 1991; Lynch et al., 1997; Scherwitz, Perkins, Chesney, & Hughes, 1991) and African American children (Gump, Matthews, & Raikkonen, 1999). Second, hostility has been linked to coronary heart disease incidence, severity, and mortality among adults (Dembroski, MacDougall, Costa, & Grandits, 1989; Koskenvuo et al., 1988; Siegman, Dem-

broski, & Ringel, 1987) and increased left ventricular mass and heightened cardiovascular reactivity in African American children (Gump et al., 1999). Finally, associations between hostility and coronary heart disease disappear when SES is controlled for, indicating that SES may be a critical link in the relationship between hostility and heart disease (Malkin, Boyle, Milgram, Townsend, & Siegman, 1999).

Depression. Lower SES and economic hardship have been associated with higher levels of both hopelessness and depression among adults (Lynch et al., 1997; Murphy et al., 1991), and children (Adams, Hillman, & Gaydos, 1994; Duncan et al., 1994; Lempers, Clark-Lempers, & Simons, 1989). Second, depression has been linked to diseases such as asthma, arthritis, and ulcers (Friedman & Booth-Kewley, 1987), as well as risk of a first myocardial infarction (MI) and subsequent MI (Barefoot & Schroll, 1996; Frasure-Smith, Lesperance, & Talajic, 1995; Pratt et al., 1996; see Gallo & Matthews, 2001).

Control. Individuals lower in SES report a lower sense of control (Gurin, Gurin, & Morrison, 1978; Levenson, 1981; Operario, Adler, & Ostrove, 1999; Syme, 1990). In turn, partial control of one's social and physical environments is associated with positive developmental outcomes (Weigel, Wertlieb, & Feldstein, 1989) and better health outcomes among adults (Lachman & Weaver, 1998; Langer & Rodin, 1976; Rodin, 1986). Research has also suggested that control is a likely mechanism through which SES affects both physical and psychological health (Operario et al., 1999).

Optimism. SES is associated with optimism, such that higher SES individuals report higher levels of optimism (Kubzansky, Kawachi, & Sparrow, 1999; Schutte, Valerio, & Carrillo, 1996). SES has also been related to positive expectancies and future aspirations among adolescents (Crowley & Shapiro, 1982). Second, optimistic individuals are less likely to develop coronary heart disease (Kubzansky et al., 1999) and recover better after coronary artery bypass surgery (Scheier et al., 1989; Scheier et al., 1999).

Information processing. Lower SES in childhood is associated with greater attributions of hostile intent and anger during ambiguous social situations (Chen & Matthews, 2001). Moreover, this negative tendency in information processing mediates the relationship between low SES and heightened cardiovascular reactivity in children (Chen & Matthews, 2001). In the mental health literature, a similar cognitive construct, hostile attribution biases, is associated with mental health outcomes, such as aggression in childhood (Crick & Dodge, 1996; Dodge, 1980), and with health-related outcomes, such as physical abuse (Dodge, Bates, & Pettit, 1990).

How might developmental changes explain the SES–health relationship? Many of these cognitive and emotional mechanisms do not develop to a significant extent until later childhood. For example, clinical depression occurs at a low rate among children, whereas prevalence rates of depression in adolescence are more similar to adulthood (Hibbs & Jensen, 1996; Kovacs, Feinberg, Crouse-Novak, Paulauskus, & Finkelstein, 1984). Suicidal behaviors steadily increase from age 9 to 17 (Kovacs, Goldston, & Gatsonis, 1993). Similarly, hostility increases with age in childhood and peaks in young adulthood (Barefoot et al., 1991; Woodall & Matthews, 1993), and hostility levels of children are lower than those of adults (Engebretson & Matthews, 1992; Matthews et al., 1992). Optimism levels are also slightly lower among adolescents compared with adults (Malinchoc, Colligan, & Offord,

1996). Thus, one would expect the influence of these mediators on the SES and health relationship to be stronger in adolescence relative to early childhood (see Figure 2).

A health outcome that is associated with these types of mediators would be expected to display an adolescent-emergent pattern of small SES differences in early childhood that grow wider in adolescence. We suggest as one example that depression as a mediator may help to explain the trajectory described for physical inactivity above. Lower SES children experience higher levels of depression (Duncan et al., 1994; Lempers et al., 1989), which is associated with significant decreases in activity level and increased time in bed. Given that depression is more prevalent in adolescence, one would expect SES to have little impact on physical activity through this pathway during childhood and increasing impact on activity levels during adolescence. As stated above, the nature of previous research on SES and health does not allow for us to review the empirical evidence for this model. However, we view the approach of matching mediators to developmental trajectories as important because it allows future researchers to identify a specific set of mediators to test for a given health outcome.

Social Mechanisms

Family–peer relationships. Both family and peer relationships have strong effects on child behavior and health throughout childhood and adolescence. According to Repetti, Taylor, and Seeman (2002), low SES children are more likely to have greater conflict and fewer positive communications with family (Hart & Risley, 1995; McLoyd, 1998), less warmth in parental relationships (Dodge, Pettit, & Bates, 1994), and over- or underregulation of their environment (Bates et al., 1998). Second, these family factors are associated with slow growth, higher illness rates, and more health complaints among children and adolescents (Gottman & Katz, 1989; Mechanic & Hansell, 1989; S. M. Montgomery, Bartley, & Wilkinson, 1997; Wickrama, Lorenz, & Conger, 1997). Poor parental supervision has been related to increased risk for injury (Pless, Verreault, & Tenina, 1989). It should be noted that although SES is related to family relationships (i.e., low SES children being more likely to live in single parent homes), the effects of SES do not appear to be entirely attributable to family structure (McLanahan, 1997; Patterson, Kupersmidt, & Vaden, 1990). For example, studies show that SES is associated with health behaviors such as smoking even after marital status is controlled for (Jarvis, Strachan, & Feyerabend, 1992; Stanton, Oei, & Silva, 1994).

Lower SES children and adolescents also appear to be more drawn to peer contact and susceptible to peer influence. Low SES children initiate more social interactions than middle SES children (Quay & Jarrett, 1988). Adolescents from single-parent families, which tend to be lower in SES, are more easily swayed by peer pressure (Steinberg, 1987). In addition, peer influence tends to be negative in high-risk neighborhoods but positive in low-risk neighborhoods (Leventhal & Brooks-Gunn, 2000). Peer pressure influences health habits, including smoking and alcohol and drug use. For example, having friends who smoke is a more important predictor of adolescent smoking than having a parent who smokes (Meijer, Branski, Knol, & Kerem, 1996; Mittelmark et al., 1987).

Moreover, with age, the ability to resist peer influence decreases (Berndt, 1979; Steinberg & Silverberg, 1986).

How might developmental changes explain the SES–health relationship? Given that peer influence grows stronger during adolescence, one would expect its mediating influence on the SES and health relationship to be stronger in adolescence relative to early childhood (see Figure 2). Thus, a health outcome that is associated with this mediator would be expected to display an adolescent-emergent pattern of small SES differences in early childhood that grow wider in adolescence. We suggest as one example that peer influence may help to explain the trajectory described for smoking behaviors above. Among younger children, the primary influence on smoking may be the parent as a role model. As children age, and if smoking behaviors cluster in low SES schools and neighborhoods, low SES adolescents may be exposed to a greater number of smoking peers, which may make them more likely to try smoking. Thus, by adolescence, both peer and parent influences act together to influence smoking behaviors, creating larger SES effects on smoking rates during adolescence.

Environmental Mechanisms

Housing/neighborhood characteristics/stressful events. Lower SES children live under poorer housing conditions and in neighborhoods that have higher incidences of violence (McLoyd, 1998). In addition, lower SES children report more frequent stressful life events (Attar, Guerra, & Tolan, 1994; Garbarino, Kostelny, & Dubrow, 1991). These factors all relate to child health. Low SES children are more likely to live in older houses with lead paint and to have higher blood lead levels (Pamuk et al., 1998). Living in areas of poverty places children at risk for violent deaths (Christoffel, Anzinger, & Merrill, 1989). Finally, stress is associated with health problems such as asthma and susceptibility to infection (Cohen & Rodriguez, 1999; Cohen, Tyrrell, & Smith, 1991; Wright, Rodriguez, & Cohen, 1998). It is hypothesized that living in poor neighborhoods is detrimental for children because these areas have lower levels of collective efficacy. Collective efficacy relates to the ability neighborhoods have to implement strategies for monitoring their youth. It applies to both children (e.g., intervening when neighbors see children skipping school) and adolescents (e.g., intervening to prevent delinquent or deviant behaviors). Low levels of collective efficacy are associated with higher community violence (Sampson, Raudenbush, & Earls, 1997; see Leventhal & Brooks-Gunn, 2000, for a review) and have implications for health-related outcomes such as substance abuse and injury.

Child care options. Higher SES families are more likely to place their children in high-quality day-care centers. In turn, receiving high-quality child care is associated with positive mental health outcomes, including higher cognitive ability and better socioemotional development (Holloway & Reichhart-Erickson, 1989; NICHD Early Child Care Research Network, 1998; see Leventhal & Brooks-Gunn, 2000, for a review). It is also possible that day-care centers may be associated with physical health-related outcomes. For example, several studies have found that injury rates in day-care centers are lower than injury rates at home (Gunn, Pinsky, Sacks, & Schonberger, 1991; Rivara, DiGuseppi, Thompson, & Calonge, 1989). It may be that higher SES parents are able to afford better quality day care, which is associated with better quality supervision and fewer childhood injuries. However,

note that day-care attendance is thought to be associated with slightly higher rates of certain types of childhood illnesses, such as respiratory infections (Roberts, 1996).

Health care options. Low SES families report fewer visits to the doctor (Pamuk et al., 1998), which may be a function of both lack of access to health care and decisions not to engage in preventive or well-child visits. Even when health care is available, however, lower SES families have been found to receive poorer quality of care than higher SES families (D. R. Williams, 1990). These factors increase the likelihood that health problems will be diagnosed later or that disease complications will arise. Poorer quality of care may mean that low SES children do not receive appropriate diagnostic tests in a timely manner, leading to more severe disease with worse prognosis. Low SES has been associated with greater likelihood of delays in seeking health care (Pamuk et al., 1998) and greater likelihood of cancer being detected at later stages (Bal, Nixon, Foerster, & Brownson, 1995).

School achievement. Lower SES children perform less well on school tests, fail courses more often, and are more likely to drop out of school compared with high SES children (see McLoyd, 1998, for a review; Patterson et al., 1990; White, 1982). In turn, lower school achievement is associated with poorer perceived physical health (Mechanic & Hansell, 1987; Power, Manor, & Fox, 1991; Vingilis, Wade, & Adlaf, 1998). School achievement represents a mediator that is likely an interaction between environmental (e.g., school quality) and individual (e.g., child's ability and motivation) factors.

How might developmental changes explain the SES–health relationship? As children grow older, they tend to spend less time at home and more time at school and outside with their friends. Thus, one would expect the impact of poor housing conditions to be larger in early childhood compared with adolescence. In contrast, the impact of neighborhoods might be larger in adolescence than early childhood (see Figure 2).

A health outcome that is associated with housing-related mediators would be expected to display a childhood-limited pattern of large SES differences in early childhood that decrease in adolescence. We suggest as one example that poor housing conditions may serve as a mediator to explain the pattern described for asthma prevalence above. Low SES children have greater exposure to cockroach allergens in their homes (Rosenstreich et al., 1997; Sarpong, Hamilton, Eggleston, & Adkinson, 1996). Children with greater exposure and sensitivity to cockroach allergens are more likely to miss school and to wake up at night due to asthma symptoms (Sarpong et al., 1996). As children age and spend less time at home and increasing amounts of time at school, the effect of cockroach allergens on asthma attacks should weaken, leading to a childhood-limited pattern. However, that increased time spent outside the home might have opposite effects on other health-related outcomes. That is, although it may decrease asthma prevalence, it could increase the prevalence of other outcomes, such as violence-related injuries.

Behavioral Mechanisms

Health practices. Lower SES families report fewer visits to the doctor (Pamuk et al., 1998) and comply with treatment or medical recommendations less often (Emans et al., 1987; Samora, Saunders, & Larson, 1961). Lack of compliance with treatment

regimens can affect disease progression. For example, asthma hospitalizations and deaths are believed to be avoidable with appropriate medication management (Rand, 1999). Low SES families may engage in poor health practices because of how they weigh the costs and benefits (health belief model; Rosenstock, 1974). As a hypothetical example, families that have children with asthma are encouraged to make environmental changes (e.g., getting rid of carpeting) to alleviate some of the child's symptoms. A low SES parent might decide not to undertake these changes because of barriers (not having money) or a lack of understanding of how these factors contribute to asthma (low perceived benefit).

How might developmental changes explain the SES–health relationship? Although parental SES may increase in an absolute level with age (older parents earning more income than younger parents), the difference in health practices between high and low SES families would be expected to remain similar over time. Thus, a health outcome associated with treatment compliance or other health practices would be expected to display a persistence pattern of similar SES differences from early childhood throughout adolescence (see Figure 2).

We suggest as one example that treatment compliance may help explain the trajectory for severe asthma described above. Despite the fact that severe asthma is more prevalent among lower classes, lower SES children consistently use asthma medications less frequently (H. R. Anderson, Bailey, Cooper, Palmer, & West, 1983a) and are prescribed these medications less frequently (Togias, Horowitz, Joyner, Guydon, & Malveaux, 1997). In addition, some researchers have suggested that asthma symptoms in low SES children go unrecognized more often by physicians (Ray et al., 1998). Low SES children may experience more severe asthma because of improper management of symptoms. Moreover, if SES differences in treatment compliance and physician recognition of symptoms remain similar as children age, so should the relationship between low SES and severe asthma.

Biological Mechanisms

The pathways through which SES affects health likely involve alterations in children's sympathetic, neuroendocrine, and immune activity. The direct effects of SES on such biological outcomes have been demonstrated primarily in animal models, using stressful or deprived environments to represent low SES. For example, early maternal deprivation in rhesus macaque (*Macaca mulatta*) infants produces adult animals with reduced serotonin turnover and behavioral predispositions toward aggression, risk-taking, alcohol preference, and antiaffiliative responses (Higley et al., 1993). Prenatal restraint stress in the pregnant female increases stress-induced corticosterone secretion and attenuates hypothalamic-pituitary-adrenal axis habituation in preweaning and weaning rats (Henry Kabbaj, Simon, Le Moal, & Maccari, 1994; Takahashi, Haglin, & Kalin, 1992), whereas postnatal handling reduces stress-induced corticosterone secretion (Meaney, Aitken, Van Berkel, Bhatnagar, & Sapolsky, 1988). The offspring of prenatally stressed rhesus macaques show decreased birth weights, impaired neuromotor development, attention deficits, delays in achieving object permanence, and disturbed behavior under adverse conditions (Clarke & Schneider, 1993; Schneider, 1992a, 1992b, 1992c). In humans, the biological effects of stress have also been documented. Ongoing life stressors are linked to increases in

cardiovascular reactivity, as well as neuroendocrine and immune changes (Matthews, Gump, Block, & Allen, 1997; Pike et al., 1997).

In addition, the effects of the SES mediators discussed above on these biological outcomes have been demonstrated in humans. For example, depressed individuals have dysregulated immune function compared with psychiatrically healthy individuals (Miller, Cohen, & Herbert, 1999). Uchino, Cacioppo, and Kiecolt-Glaser (1996) found evidence for cardiovascular, neuroendocrine, and immune changes resulting from poor social support. Crowded housing conditions also were related to elevated resting blood pressure in boys (Evans, Lepore, Shejwal, & Palsane, 1998). These studies suggest that SES affects health through a combination of the psychosocial and biological processes described above.

Summary

There is evidence that SES is related to several classes of potential mediators and that these potential mediators are related to health outcomes. However, very few studies have conducted formal tests of mediation among children. In addition, some of the mediators described above may be more compelling than others for explaining a monotonic association. For example, hostility appears to have linear associations with both SES and health (Cohen, Kaplan, & Salonen, 1999). In contrast, health practices such as doctor visits may be tied to health insurance status and may better explain a threshold effect. Another limitation is that evidence for mediation tends to rely on studies conducted with adult populations, and it remains unclear whether the same mediators apply to children.

Despite these limitations, we view this section as important for guiding future research. Relationships between SES and child health have been extensively documented in previous research. Changes in these relationships with age appear likely on the basis of this review and others (West, 1997). The next logical step is a search for mediators; however, a theoretically driven approach is needed so that researchers can begin to narrow the scope of factors to test for each specific health outcome. Figure 2 reflects our conceptual approach to evaluating potential child health mediators. That is, for any given health outcome, its relationship with SES across the developmental trajectory should first be identified. Second, any potential mediator should be explored in terms of its pattern of changes corresponding to normal child development. Third, only mediators that represent a potential fit with the developmental trajectory of a specific SES–health association should be included in future studies devoted to mechanism testing.

Summary and Concluding Comments

Children are born into socioeconomic strata that have immediate and profound effects on their health. Our review shows that as in adulthood, the relationship between SES and health outcomes follows a monotonic pattern in childhood and adolescence. As SES decreases, all-cause mortality and overall morbidity rates increase, as do the prevalence rates of severe asthma; vision and hearing problems; injury; and acute illnesses, such as rheumatic fever and upper respiratory and ear infections. The monotonic effect suggests that the relationship is not simply due to poverty, although poverty does take a great toll on children.

Collapsing data across all ages of children and adolescents obscures an understanding of when and how SES influences their health. Guided by three models, childhood limited, adolescent emergent, and persistence, we evaluated SES–health trajectories for injury, the most common cause of mortality in children; asthma, the most common chronic disease in children; and risk factors related to cardiovascular disease, the most common cause of death across the life span. At a young age, children from lower SES families are at elevated risk for injury, asthma (prevalence and severity), and elevated blood pressure. During adolescence, they are at risk for severe asthma and negative health behaviors including smoking and physical inactivity, although their risk for injury, asthma (prevalence), and elevated blood pressure is no longer present. Preliminary support was found for the various models, suggesting that different mechanisms account for how each health outcome becomes more prevalent among lower SES children.

Based on available, but minimal, data, we speculate that the key mechanisms connecting young children's health and SES include unhygienic home environments; conflictual, inconsistent family relationships; child-care quality; stressful life events; and health care access and quality. In adolescence, the role of peer groups; neighborhood environments; and emotional, attitudinal, and cognitive processes gain prominence. These mechanisms are all subject to empirical investigation. Public policies could then target more precise mechanisms once they are verified.

In closing, we note that the current economic climate is leading to increasing, not decreasing, disparities in family income in the United States (Kawachi & Kennedy, 1997). In part this may be due to the potential for greatly increasing one's wealth through investments among those who have assets, as well as the impact of more women in the workforce potentially leading to greater SES differences when two wage earners' incomes are considered. In addition, the increasing reliance in our society on technology creates an even greater disadvantage for those not technologically skilled. As a result, children born into lower SES families are starting their lives more disadvantaged in health compared with those born into higher SES families. Parents in these families also have a greater burden from their own health problems. Given this trend, health inequalities in children are likely to only increase unless aggressive policies are put into place to invest in children's current and future health.

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Received August 12, 1999
 Revision received June 5, 2001
 Accepted July 11, 2001 ■



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