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Issue: *The Biology of Disadvantage***Are psychosocial factors mediators of socioeconomic status and health connections?****A progress report and blueprint for the future**Karen A. Matthews,¹ Linda C. Gallo,² and Shelley E. Taylor³¹University of Pittsburgh, Pittsburgh, PA. ²San Diego State University, San Diego, CA. ³University of California at Los Angeles, Los Angeles, CA

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The association between socioeconomic status (SES) and physical health is robust. Yet, the psychosocial mediators of SES-health association have been studied in relatively few investigations. In this chapter, we summarize and critique the recent literature regarding negative emotions and cognitions, psychological stress, and resources as potential pathways connecting SES and physical health. We discuss the psychosocial origins of the SES-health links and outline how psychosocial factors may lead to persistently low SES. We conclude that psychosocial resources may play a critical mediating role, and the origins of the SES-health connection are apparent in childhood. We offer a blueprint for future research, which we hope contributes to a better understanding of how SES gets under the skin across the life span.

Keywords: socioeconomic status; emotions; stress; resources; development

Introduction

It is well established that socioeconomic status (SES) is a strong correlate of health across the lifespan. Those who are less educated, earn less income, or hold less prestigious occupations are at greater risk for a wide variety of diseases and premature death (chapters 3 and 4, this volume).¹ By and large these relationships are sustained in men and women, in diverse ethnic groups, and in children and young, middle-aged, and elderly adults. The associations are graded, such that with every decrease in SES, there is increasing risk, although extreme poverty can be particularly health-damaging. A variety of factors may account for the health effects of low SES, including exposure to environmental toxins; employment in jobs that have a high risk of injury or disability; lack of health insurance or access to high quality and preventative health care; poor nutrition; and adverse health behaviors, such as smoking, excessive alcohol intake, and physical inactivity. These factors do not work individually but are likely to accumulate in low SES environments.²

This chapter focuses on psychosocial factors that may serve as pathways connecting low SES and poor

health. From a psychosocial perspective, the leading candidate pathways are the frequency and intensity of exposure to stress and related emotional responses. As shown in Figure 1, low SES environments are thought to be associated with greater exposure to frequent and intense harmful or threatening situations and to fewer rewarding or potentially beneficial situations (Arrow A). Indeed, studies show that individuals with lower SES encounter more frequent negative life events and chronic stressors^{3,4} and interpret even ambiguous events as more stressful,^{5,6} relative to those with higher SES. Exposure to chronic and acute stressors, in turn, has a direct negative impact on emotional experiences (Arrow B). However, studies that account for initial differences in stress exposure suggest that at every level of stress, individuals with lower SES report more emotional distress than those with higher SES.^{3,7}

Why might individual residing in low SES environments be more reactive to stress? Our framework suggests that low SES individuals maintain a smaller bank of resources—tangible, interpersonal, and intrapersonal—to deal with stressful events compared to their higher SES counterparts

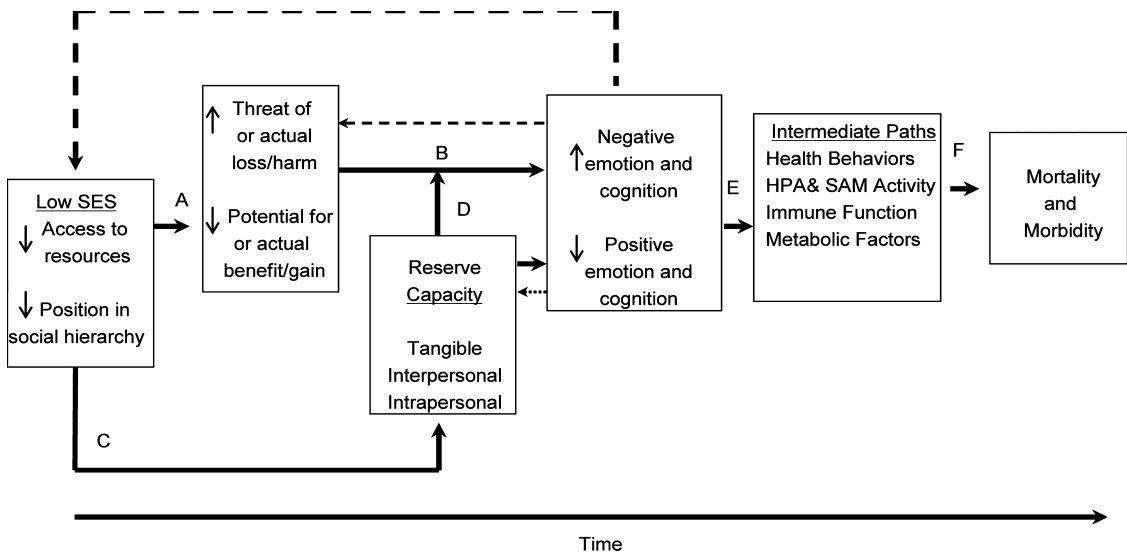


Figure 1. The reserve capacity model for the dynamic associations among environments of low socioeconomic status (SES), stressful experiences, psychosocial resources, emotion and cognition, and biological and behavioral pathways predicting morbidity and mortality over time. *Dashed lines* depict possible reciprocal influences. *Arrow A* depicts the direct influence of SES on exposure to stressful experiences. *Arrow B* indicates the direct impact of stressful experiences on emotion and cognition. *Arrow E* shows the effects of stress on intermediate pathways hypothesized to affect health outcomes. *Arrow C* shows that socioeconomic environments condition and shape the bank of resources (*i.e.*, the reserve capacity) available to manage stress. *Arrow D* shows that the reserve capacity represents a potential moderator of the association between stress and cognitive–emotional factors. *Arrow E* indicates the direct impact of cognitive–emotional factors on intermediate pathways and *Arrow F* on intermediate pathways to illness and death. HPA: hypothalamic-pituitary-adrenocortical axis; SAM: sympathetic adrenal-medullary axis.

(Arrow C).⁸ Resources tend to occur in aggregate, or be absent in the aggregate, suggesting the existence of a generic protective influence or resource bank.⁹ Borrowing a concept from the aging literature, we label this reserve capacity. Reserve capacity to deal with stressful environments may be diminished in circumstances of low SES for two reasons: (a) low-SES individuals are exposed to more situations that require they use their resources and (b) low-SES environments prevent the development and replenishment of resources to be kept in reserve. Indeed, having few resources exacerbates the effects of stressful events on outcomes such as depression.¹⁰ Furthermore, once an individual has been exposed to stress, resources tend to deteriorate, leaving individuals more vulnerable to future strains (arrow D; *e.g.*, Ref. 11). Hence, having fewer stress-dampening resources, which are further reduced by more stress exposures, individuals of low SES are likely to show greater responsiveness when faced with stress (Ar-

row E). According to this model, elevated negative emotions and cognitions and reduced positive emotions and cognitions then lead to intermediate physiological pathways (Arrow E) and eventually to poor health (Arrow F). Finally, this model takes a life-course perspective in that the processes it proposes are thought to start early in life and may also lead to lowering of SES over time (dashed lines).

Ten years ago there were few studies testing these proposed relationships. In fact, through 2001, we found only seven articles that simultaneously examined SES, cognitive or emotional factors, and a physical health outcome defined broadly (this review did not include stress as a mediator).⁸ These studies, taken together, did not provide conclusive evidence for the mediating role of negative emotions. The major objectives of the current chapter are (1) to summarize and critique the recent literature regarding negative emotions and cognitions, psychological stress, and resources as pathways

connecting SES and physical health; (2) to discuss the psychosocial origins of the SES-health links; and (3) to outline the psychosocial factors may lead to persistent low SES. Suggestions for future psychosocial research are offered throughout the chapter.

Review of new literature on psychosocial pathways

The criteria for articles to be in the review were inclusion of (1) a measure of SES (with title search words SES, social status, socioeconomic position, education (not patient education), income, occupation, occupational status, disparity, inequality, gradient); (2) an objective physical health outcome (with search keywords CVD, CHD, atherosclerosis, hypertension, blood pressure, diabetes, metabolic syndrome, asthma, cancer, infectious disease, HIV, and mortality); and (3) a candidate psychosocial factor (negative emotions/attitudinal factors, with search keywords anxiety, depression, hostility, anger, negative emotions, emotional factors; intrapersonal and interpersonal resources, with search keywords mastery, perceived control, optimism, self-esteem, social support, social competence, social integration, social network, and resources; and environmental stress, with search keywords stress, discrimination, life events, and coping). The final criterion was the use of statistical methods that would permit an evaluation of mediation. We did not include papers that focused only on general self-rated health due to the very subjective nature of these measures and their overlap with psychosocial pathways of interest (*e.g.*, negative emotions). Moreover, we did not include papers that focused exclusively on individual health risk factors (*e.g.*, obesity, cardiovascular reactivity, immune functioning, health behaviors). PsychINFO, PsychArticles, and Medline were searched and results were limited to articles published in English, between 1990 and 2008, in peer-reviewed journals. One thousand and ninety-seven articles were obtained in the search. After scanning titles for potential relevance, 53 abstracts were further examined; 23 met criteria and were not previously discussed by Gallo and Matthews.⁸ Several additional articles were identified via inquiries to colleagues, or from scanning reference lists, for a total of 27 studies. Data on each study's design, population sampled, measurement of SES and psy-

chosocial factors, and results are summarized in Table 1.

Studies examining stress as a mediating pathway

A number of researchers have suggested that the association between SES and health may, in part, reflect the psychosocial pathway of "stress."^{12–14} This assertion is based on a large body of indirect research connecting SES to various types of stress on the one hand (*e.g.*, Refs. 4,15–19), and connecting stress with health and disease processes on the other hand (for reviews, see Refs. 20,21). The community, work, and home environments that individuals with low SES inhabit are often characterized by frequent and repeated challenges and negative events, potentiating physiological and behavioral dysregulation that, in turn, increases the risk of negative health outcomes.^{22,23} Our review uncovered nine studies that had examined a measure of stress as a possible explanatory mechanism in SES-health gradients.^{24–32} Specific health outcomes varied across studies, with four examining mortality,^{26–28,32} one examining incident stroke,²⁴ three examining the metabolic syndrome or metabolic functioning,^{29–31} and one study focusing on subclinical coronary artery disease.²⁵ Most of the studies examined either life events^{24,29,32} or perceived stress,^{25,26,28,31} one study used five different stress measures,²⁷ and one focused on stressful aspects of the early environment.³⁰ Five studies found little or no evidence for a mediating role of stress.^{24,25,28,29,31} In fact, three studies reported no significant relationship between SES and stress^{25,29,31} and one found that individuals with higher SES reported *more* stress.²⁸ On the other hand, van Oort *et al.*³² found that accounting for variability in life events (simultaneous with locus of control) led to a 21 to 48% reduction in the excess mortality risk attributed to low versus high education. Much of the effect of these factors in that study appeared to be indirect, via pathways from material factors, to psychosocial factors, to mortality. Khang and Kim²⁶ found an 11% reduction in the excess mortality risk for low versus high income (16% reduction in models not controlling for baseline health) with simultaneous control for depression and perceived stress. Lantz *et al.*²⁷ showed that statistical control for five measures of stress led to a 35 and 45% reduction in the excess

mortality risk for lower income groups relative to the highest income group, with life events appearing to have the greatest explanatory role. Finally, Lehman *et al.*³⁰ found that a risky, early family environment represented part of a significant, indirect pathway from SES to poorer metabolic functioning.

Several additional studies focused specifically on the contribution of “job strain” to socioeconomic health disparities.^{33–36} Job strain is often defined as a combination of job high demands and low levels of control in the work environment, and is associated with elevated CVD risk, particularly in men.³⁷ Marmot and colleagues³⁶ found that job control explained about 64 and 51% of the excess risk for CHD (self-reported) associated with low versus high occupational class, in men and women, respectively, enrolled in the Whitehall study. In a case control study of CHD in women, statistical control for job strain reduced the excess risk associated with lower occupational grade relative to the highest grade by 8–14%.³⁴ Of the components of job strain, perceptions of control seemed to be the more important factor in explaining the occupational grade-CHD gradient. In the same sample, accounting for job strain combined with social risk factors (*e.g.*, low support) reduced the excess CHD risk for low education by 57%.³³ In contrast, Kuper *et al.*³⁵ found no evidence that job stress contributed to the association between education and stroke risk. Importantly, job strain can be conceptualized as a type of chronic stress, but it is also closely related to SES, since individuals with lower SES are more likely to hold jobs that are higher in demands and, particularly, lower in control. Thus, whether job strain represents an independent stress pathway from low SES to health, or whether it represents one of the toxic components of low SES itself, is unclear (*e.g.*,³⁸).

Studies examining emotional factors as a mediating pathway

High levels of negative emotions, such as depression, anxiety, and anger, are a common correlate of stress that have been related to health outcomes (particularly CVD) in substantial prior research.^{39–42} In addition, individuals with lower SES are at greater risk for negative emotions and emotional disorders relative to those with higher SES.^{8,43} Therefore, these variables may represent another psychosocial pathway contributing to SES-health gradients.

In updating our prior review,⁸ we identified 12 studies that examined emotional factors as possible explanatory mechanisms in the association between SES and health.^{24–26,29,30,44–50} These studies were widely varied with respect to emotional indicator/s and health outcomes. Three studies identified little or no support for a mediating role of emotional factors, and specifically, depression and anxiety in the association between SES and CHD incidence,^{46,50} and positive and negative emotional styles in the association between SES and vulnerability to infectious illness.⁴⁴ In contrast, Avendano *et al.*²⁴ found that control for depression reduced the association between income and education with incident stroke by approximately 23 and 30%, respectively. Moreover, two studies identified support for aggregate negative emotions as a pathway in the association between SES and the metabolic syndrome.^{29,30} Schnittker *et al.*⁴⁹ showed that neuroticism (*i.e.*, a trait-like propensity to frequently experience negative emotions) and depression explained 15 and 37%, and 26 and 16% of the association of income and education, respectively, with the aggregate number of self-reported chronic health conditions. Nabi *et al.*⁴⁸ found relatively strong evidence for a mediating role of hostile personality traits in the association between SES and mortality in men (with 28–29% attenuation in SES effects after statistical control for hostile personality traits), but very little explanatory role for personality of any type in explaining mortality risk in women. Gallo *et al.*²⁵ showed that controlling for depression and anxiety attenuated the association between education and aortic calcification (an indicator of atherosclerosis) by 5 and 16%, respectively, in a sample of healthy women. Hostility and anger did not relate to atherosclerosis and were not tested as mediators in this study.

The contributing role of emotional factors is more difficult to ascertain in the remaining studies because multiple mediators were considered simultaneously. Khang *et al.*²⁶ found that concurrent control for depression and perceived stress reduced the association between SES and mortality by 11%; Maty *et al.*⁴⁷ found that control for depression along with many other risk factors (*e.g.*, baseline health, health behaviors) reduced the excess diabetes risk associated with lower education by 47%; and Gorman *et al.*⁴⁵ found that, after controlling for social and demographic factors, accounting for depression and

Table 1. Research addressing the whether psychosocial factors mediate the association between socioeconomic status (SES) and objective health outcomes

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Alter <i>et al.</i> (54)	3138 patients hospitalized for myocardial infarction, mean age 64 years, 30% women	Prospective, 2 years f/u	Income	Social support (if participants lived alone and if they had "someone to talk to about private feelings and personal decisions")	Mortality	Age, sex, race	Higher income significantly predicted lower mortality risk, at 30 days and at 2 years f/u. Adjustment for social support had very little effect on HRs for income.	Social support assessment was quite limited. Short- follow-up	–
Avendano <i>et al.</i> (24)	2524 men and women form the EPESE study, aged 65 and older	Prospective, 12 years f/u	Education Income	Depression (Validated measure) Social Integration (Validated measure) Stressful life events	Incident stroke	Age, sex, race	Education and income related significantly and inversely with stroke risk. A reverse pattern was observed in persons aged 75 and older. After adjusting for race, inclusion of depression and social integration attenuated the HR for income and education by 23% and 30% and 37% and 27%, respectively. Adjustment for life events had little effect on SES- stroke relationship. Adjustment for all psychosocial factors reduced HRs by about 50%.	Additional analyses showed that adjustment for conven- tional risk factors reduced the effect of education by 22% and of income by 43%.	±
Bosma <i>et al.</i> (57)	2462 participants from the Dutch GLOBE study, 50% men, average age 51 years	Prospective, 6-year f/u	Education Occupation Income	Perceived control (Dutch version of Rotter's Locus of Control Scale)	Mortality	Age, sex, baseline health	Lower education and occupation predicted higher mortality risk, whereas income did not relate significantly to mortality. Statistical control for control perceptions reduced the education AMRs by 54–57%, occupation AMRs by 41 to 50%, and income AMRs by 37 to 65%.	Additional analyses showed that low SES in adulthood predicted adverse changes in control perceptions across the 6-year follow-up.	+

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Bosma <i>et al.</i> (58)	3888 men and women from the Groningen Longitudi- nal Aging Study, 57 years and older	Prospective, 5-year f/u	Composite of education, occupation, income	Control beliefs: General self efficacy and Mastery (validated scales)	Incident CHD (myocardial infarction and congestive heart failure)	Sex, age, traditional risk factors	Low SES was associated with a 45% higher rate of heart disease (prior to risk-factor adjustment). Inclusion of control beliefs reduced the HR by 29%, after control for traditional risk factors.		+
Chaix <i>et al.</i> (55)	498 participants from the Swedish "Men born in 1914" study	Prospective, 10 year f/u	Income Income change	Support from family and friends Neighborhood support	CHD Incidence CHD Mortality	None	Low income 10 years before retirement predicted increased CHD and mortality risk. Income change was not predictive. Lower income related to less neighborhood support, but not support from friends and family. After control for risk factors, neighborhood support led to an additional 7 and 8% decrease in the income effect on CHD incidence and mortality, respectively. 16% and 14% of the risk for low income in CHD and mortality that was explained by risk factors was attributable to neighborhood support.	Marriage and social support from friends and family were not found to be protective. The authors speculate that neigh- borhood support may gain importance in older ages, given social network changes associated with retirement.	±

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Cohen <i>et al.</i> (44)	95 men and 98 women, aged 21 to 55 years, volunteer sample	Cross sectional	Subjective SES (MacArthur ladder measure). Education Income	Positive and negative mood (adjective checklist administered on each of 14-days preceding the experiment). Mastery, optimism, self-esteem, purpose in life, and extraversion (all validated measures)	Infection, signs and symptoms of the common cold, and clinical illness, after exposure to virus.	Immunity to the virus as assessed by pre-challenge antibody titer, age, BMI, race, sex, virus-type, and season of exposure.	Neither income nor education related to illness. Lower subjective SES predicted a higher risk for developing a cold. The association between subjective SES and colds remained statistically significant in analyses controlling for negative emotional, style, positive emotional style, self-esteem, mastery, purpose, optimism, extraversion, and all of these variables examined in a single model.	Volunteer sample is a limitation of the research. Additional analyses suggested that poorer sleep duration and efficiency associated with lesser subjective status might contribute to observed findings.	–
Gallo <i>et al.</i> (59)	145 healthy Latinas (primary Mexican-American) (mean age 47 years) recruited from health clinics along the California–Mexico border	Cross-Sectional	Educational Attainment	Reserve Capacity – composite of perceived social support, optimism, self esteem, and perceived control (validated measures used for all constructs)	Risk factors that comprise the metabolic syndrome	Age, menopausal status	Education showed a significant, inverse relationship with most components of the metabolic syndrome, and women with lower SES also reported lower psychosocial resources. Approximately 1/3 of the relationship between SEP and waist was due to an indirect effect through psychosocial resources (<i>P</i> for indirect effect < .05).	Limitations include convenience sample, cross sectional design, and the fact that some risk factors were available only in a subset of participants. Range of SES was restricted (sample was low income).	±

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Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^a
Gallo <i>et al.</i> (25)	308 women from the Healthy Women Study, average age 67 years old, 90% non- Hispanic white	Prospective	Educational Attainment	Hostility, anger, depression, anxiety, social support, job satisfaction, perceived stress (all validated measures)	Calcification of the coronary arteries and aortic valve, measured by electron beam computed tomography	Age	A significant linear trend was observed in the association between education and coronary and aortic calcification (<i>P</i> < .05). Education also showed a positive association with hostility, depression (<i>P</i> < .10), anxiety, and an inverse association with social support. Coronary calcification did not relate to psychosocial risk factors. Only depression and anxiety related to aortic calcification, and were tested as mediators. Regression coefficients for the education trend for aortic calcification were reduced by 5% with control for depression and 16% with control for anxiety.	Control for individual traditional risk factors also had a small impact on the magnitude of the association between education aortic calcification. No risk factor met statistical criteria for mediation.	±
Gorman & Siva- ganes (45)	29,816 US adults 25 years and older, from the 2001 National Health Interview Survey	Cross- sectional	Education Family income to poverty ratio Working status	Social support (1-item) Social integration Depression (validated measure) Life Satisfaction (1-item) Happiness in past 30 days (1-item)	Self-reported hyperten- sion	Age Ethnicity Nativity (Age only and emotional factors)	Working status was related to self-reported hypertension, whereas education and income did not predict this outcome. In age-adjusted (only) models, greater emotional support and integration, life satisfaction, and happiness related to lower hypertension risk, and depression related to higher risk. Control for social factors had little impact on SES effects. Additional control for both health behaviors and emotional factors further reduced the OR for unemployed versus employed by approximately 24%.	Limitations include the self-reported physical health outcome, cross- sectional framework, use of 1-item measures or non-validated assessments of some psychosocial constructs, testing emotional factors together with health behaviors, and lack of consistent SES effects.	±

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Khang & Kim(26)	5437 participants from Korean NHANES study	Prospective 5-year f/u	Income	Marital status Feelings of depression/ sadness past year (1-item) Perceived stress past year (1-item)	Mortality	Age, gender, urbaniza- tion of residence, number of family members, baseline health status	Income showed an inverse association with mortality risk. Control for psychosocial factors created a 11.2% reduction in RR for the low income group compared to the high income group (16.5% change in RR in models that did not control for baseline health).	In compari- son, control for health behaviors and biological risk factors created a 14.3 and 15.3%, respective, reduction in RR.	±
Kittleson <i>et al.</i> (46)	1131 male medical students from the Johns Hopkins Precursors Study	Prospective, median f/u 40 years	Childhood SES (father's occupation)	Depression (incidence of clinical depression, assessed via self-report)	Incident CHD (myocardial infarction, sudden death, angina pectoris, chronic IHD, other coronary disease requiring bypass or percuta- neous interven- tion) before age 50.	CVD risk factors, in some analyses	Risk of CHD on or before age 50 was higher with low SES. HR was largely unchanged with control for depression.	Self-report of diagnosed depression may be a limitation, given the sample.	—
Kuper <i>et al.</i> (35)	47,942 women from the Women's Lifestyle and Health Cohort Study (Sweden), aged 30 to 50 yrs	Prospective, average f/u 11 years	Education	Job demands, job control, and social support at work (validated measures) General social support	Incident stroke	Age	Lower education significantly predicted higher stroke risk. Work characteristics did not relate significantly to stroke risk, whereas lower general support related to higher risk. Adjustment for support and work characteristics had very little effect on the education and stroke association.	Age of participants was relatively young to examine stroke, and only 200 incident cases were observed.	—

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Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Lantz <i>et al.</i> (27)	3,617 men and women from the Americans' Changing Lives survey, 25–65 years, 90% non- Hispanic white	Prospective, 8-years f/u	Education Income	Five measures of stress: financial stress, parental stress, marital/ domestic relationship stress, lifetime events, and life events past 3 years. Administered at baseline and at a 3-year f/u.	Mortality	Age, sex, race, baseline health	Income related significantly to mortality, after controlling for education, whereas education did not relate to mortality risk beyond the effects of income. In models that included SES and all stress measures, only wave 1-assessed total life events related to mortality risk. HRs for the lowest and middle income groups relative to highest income group were reduced by 45% and 35%, respectively, but remained statistically significant.	Inclusion of financial stress is problematic given conceptual overlap with income.	±
Lehman <i>et al.</i> (30)	3255 participants from CARDIA, aged 33 to 45 years, 44% male, 45% black, 55% white	Cross Sectional	Childhood SES (composite of mother's and father's education) Adult SES (composite of subjective SES, measured with the MacArthur ladder measure, income, and education)	Early family environment (7 item scale measuring factors such as abuse, supervision, chaos, and feeling loved and cared for) Summary psychosocial measure of depression, hostility (validated scales), and social contacts (supportive and unsupportive)	Metabolic Functioning (fasting glucose, insulin, LDL and HDL- cholesterol, triglyc- erides)	None reported	Structural equation models showed a significant direct path from SES to metabolic functioning, and significant indirect paths from SES to risky family environment, to psychosocial factors, to metabolic functioning. Additional pathways were identified from childhood SES to adult SES to psychosocial functioning. Adult SES did not relate directly to metabolic functioning.	Additional analyses in separate sex/race groups found poor model fit for black men, and variations in the model among other groups.	+

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Table 1. *Continued*

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Liu <i>et al.</i> (56)	4,049 participants (42% women; Mean age 68.19 years) from the Taiwan Survey of Health and Living Status of the Elderly	Prospective, 4-year f/u	Education	Social Participation (attends any activities or none) Emotional Support (2 item measure)	Mortality	Gender Age Ethnicity Marital Status	Education related significantly to mortality. About 83% of this effect was indirect, via influences of education on baseline health status, social factors, and health behaviors. Social participation and emotional support related significantly to mortality, and accounted for about 26% of the indirect effect ($P < .01$).	In compar- son, health behaviors explained about 9% of the relationship between education and mortality.	+
Macleod <i>et al.</i> (28)	5232 men, 35 to 64 years, recruited from various companies in Scotland	Prospective, 25 years follow up	Occupation of participant and father Area deprivation Education Height Car access Subjective occupa- tional status	Perceived stress (Reeder Stress Inventory) Job satisfaction	Mortality	Age Risk factors (smoking, drinking, blood pressure, cholesterol, BMI, fitness)	All SES indicators were associated significantly with all-cause mortality. Adjusting for stress and job satisfaction had little or no impact on the magnitude of the RR associated with low SES, after control of risk factors.	Additional analyses showed that higher SES was associated with <i>greater</i> stress perceptions. The validity of the Reeder inventory is unknown.	-
Marmot <i>et al.</i> (36)	7372 men and women from Whitehall II	Prospective, 5 year f/u	Occupational grade	Job Control (decision authority/skill discretion) Social support (validated measure)	CHD Incidence (self- reported: angina pectoris from the Rose ques- tionnaire, report of severe, prolonged chest pain, or report of doctor- diagnosed angina or myocardial infarction)	Smoking, serum cholesterol, BMI, hyper- tension, and physical activity, height.	Lower occupational status was associated with higher incident CHD risk in men and women. In men, adjustment for job control and social support reduced the OR for low versus high grade by 64% and 14%, respectively. In women, adjustment for job control reduced the OR for the occupation effect by 51%, whereas control for social support <i>increased</i> the magnitude of the occupation effect.	Self report of CHD and inclusion of angina (a subjective endpoint) are limitations of the current study. Oc- cupational grade trends were relatively weak.	+

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Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Matthews <i>et al.</i> (29)	401 women from the Healthy Women's Study (Pittsburgh, PA), 42–50 at study entry, >90% non- Hispanic white	Prospective, 12-years follow-up	Education	Negative emotions (depression symptoms, trait anxiety, trait anger). Reserve Capacity (optimism, social support, self-esteem). Stressful life events All validated measures.	Incident metabolic syndrome		Structural equation models (SEM) showed that SES had a significant indirect pathway to metabolic syndrome through reserve capacity and negative emotions, and a significant direct pathway to metabolic syndrome. Indirect pathways from SES to negative emotions through reserve capacity, and from reserve capacity to metabolic syndrome through negative emotions, were also significant.	Education showed no association with stress and including stress in SEMs did not significantly alter the factor loadings or paths among SES, reserve capacity, negative emotions, and the metabolic syndrome.	±
Maty <i>et al.</i> (47)	6147 men and women from the Alameda County Study	Prospective, 43 years f/u	Education Income Occupation	Depression	Incident diabetes	Age, gender, race, marital status	Only education related significantly and consistently to incident diabetes. With control for behavioral risk factors, body composition (BMI, waist), blood pressure, insurance status, access to care, and depression, the excess incident diabetes risk associated with lower education was reduced by nearly 47%.	The indirect effect of SES through depression cannot be determined due to si- multaneous control for multiple risk factors.	±

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Nabi <i>et al.</i> (48)	14 445 participants from the French GAZEL study (employees of France's national gas and electricity companies), aged 39–54 at enrollment	Prospective, 12.7 years f/u	Father's social class Education Occupa- tional grade, Income	Type A behavior pattern (Validated measure) Hostility (Validated Measure) Personality types (Grossarth- Maticcek and Eysenck- Personality- Stress- Inventory)	Mortality	Age, sex, marital status, alcohol, smoking, BMI. Also, depression in analyses involving personality.	Results for men: Father's social class did not relate to mortality, whereas education, occupational grade, and income were inversely, significantly associated with mortality. In analyses adjusting for personality factors, neurotic hostility attenuated SES effects by 28–29%, 'CHD-prone' by 13–16%, and 'antisocial' by 12–22%. Adjustment for 'ambivalent' personality type attenuated SES effects by only 3–5%. With adjustment for all personality characteristics, SES effects were reduced by 28 to 34%. Results for women: Father's social class and income were significantly, inversely associated with mortality, whereas occupation and education were not. Controlling for TABP reduced the SES effects by 3–11%. Controlling for CHD-prone personality type and 'healthy' personality <i>increased</i> the magnitude of SES effects.	Evidence concerning the validity of the Grossarth- Maticcek and Eysenck personality type measure is mixed. In the current study, alphas for some scales were <.70.	±

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Prescott <i>et al.</i> (31)	5801 participants (57% women), aged 20–97 years from the Copen- hagen City Heart Study	Cross- Sectional	Educational attainment Occupation Income	Perceived Stress (single item) Vital exhaustion (17-item measure of depression and fatigue) Social integration (3 items, examined separately)	Metabolic syndrome index	Age and sex (in sex- combined models)	All SES indicators were inversely related to the metabolic syndrome. Education was most strongly related and was used in further analyses. Control for psychosocial factors had little to no impact on the association between education and MS prevalence.	Behavioral factors (smoking, alcohol, physical activity) were also tested as mediators, and did not account for associations between education and MS prevalence.	–
Schnittker (49)	Americans’ Changing Lives Study, Waves 1–III (1986– 1994) (3,617 participants at Wave 1)	Cross- Sectional	Income Education	Mastery (3 items from Pearl’s scale) Self Esteem (3 items from Rosenberg’s scale) Depression (11-item version of CESD) Neuroticism (5-item scale)	Total number of self- reported chronic conditions in past year (arthritis, lung disease, hy- pertension, heart disease, diabetes, cancer, foot problems, loss of urine beyond one’s control)	Age Sex Race	At wave 1, higher income and education predicted fewer chronic conditions. With control for self-esteem, mastery, neuroticism, and depression, the coefficients for low income (relative to highest income) were reduced by 13%, 12%, 15%, and 37%, respectively, and the coefficients for low education (relative to highest education) were reduced by 5%, 4%, 26%, and 16% respectively. Effects of (baseline) psychological factors and their mediating roles were diminished at subsequent waves of assessment.	The use of self-report assessments and inclusion of subjective conditions is a limitation of the current study. The cross sectional framework and brief measures of some psy- chological factors are additional limitations.	±

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Thurston <i>et al.</i> (50)	6265, 25–74 years, 46% men, from NHANES I and follow-up studies	Prospective, 15.1 years f/u	Educational attainment	Depressive and anxious symptoms (Validated measure)	Incident CHD	Age, gender, race/ ethnicity, marital status, smoking status, leisure time physical activity, alcohol use, BMI, diabetes, hyperten- sion	In fully adjusted models, education related significantly to CHD incidence. Depressive symptoms accounted for 4.8% and anxious symptoms < 1% of the education effect in fully adjusted models.	The authors also tested moderation, and found no significant education by depression or anxiety interaction effect in relation to incident CHD.	–
Van Oort <i>et al.</i> (32)	3979 men and women, aged 15–74, from the GLOBE study, the Netherlands	Prospective, 7 years f/u	Education	Life events (9 possible) Long lasting difficulties Emotional support Active and avoiding coping styles Locus of control	Mortality	Age, gender	Education predicted mortality. Coping, chronic stress, and support were not considered further as mediators, due to a lack of consistent association with education and mortality. With control for life events and locus of control, the HRs for the lower education groups compared to the highest education group were reduced by 21 to 48%.	Additional analyses showed that the inde- pendent effect of psychosocial factors was relatively small (0 to 11%), but in part, material factors (insurance, financial difficulties, housing) had an indirect role in the association between education and mortality <i>through</i> psy- chosocial factors (10% to 48% of all variance was explained by these indirect effects).	+

Continued.

Table 1. Continued

Study	Population/ Source	Design	Indicator/s of SES	Indicator/s of Psychosocial Variable/s	Health Outcomes	Controls	Results	Comments	Evidence ^d
Wamala <i>et al.</i> (33)	292 CHD patients and 292 age-matched controls (all women, 65 years and younger)	Case-Control	Education	Job strain (validated measure) Social isolation Capacity for coping	CHD	Age	Education related significantly and inversely to CHD risk, and control factors reduced this association by 57%.	In comparison, control for behavioral factors reduced the education effect by 48%. The design is a limitation, given the possible influence of recall and survivor biases.	+
Wamala <i>et al.</i> (34)	200 CHD patients and 212 age-matched controls (all employed women, 65 years and younger)	Case-Control	Occupation	Job strain (validated measure)	CHD	Age	Women with lower occupational status had significantly higher CHD risk. Adjustment for job stress reduced the excess risk of semi/unskilled versus professional workers by 14%, and other groups by 8% to 14%. The linear trend for occupational class remained significant.	The case control design is a limitation of the study, given the possible influence of recall and survivor biases.	+

^dEvidence for a mediational role of psychosocial factors in the SES-health association. Rated as “–” if little or no support, “±” if mixed or limited support “+” if clear support.

health behaviors reduced the excess (self-reported) hypertension risk associated with not working by approximately 24%.

Studies examining psychosocial resources as a mediating or moderating pathway

Finally, we examined studies that evaluated the mediating role of social resources, such as social support or integration, and psychological resources, such as self-esteem and mastery, in the association between SES and health. Such factors have been related to health in prior research, although more evidence supports their roles in psychological than in physical health outcomes.^{51–53} Likewise, levels of psychosocial resources are inconsistently

distributed across the SES spectrum, and therefore, these variables have been proposed as another possible explanatory pathway.^{8,51}

We identified 17 studies that examined the contribution of one or more psychosocial resource variables to the association between SES and physical health. Ten studies^{24,25,31,33,35,36,45,54–56} focused explicitly on the independent contribution of social factors (*e.g.*, social support, integration). Of these, four studies found no evidence that social support or integration contributed to SES-gradients in health outcomes including mortality,⁵⁴ self-reported hypertension,⁴⁵ incident stroke,³⁵ and the metabolic syndrome.³¹ Moreover, in the study by Gallo *et al.*,²⁵ social support did not relate to the outcome of subclinical atherosclerosis, and, was not tested as a

mediator. In contrast to these null findings, Averdano *et al.*²⁴ found that statistical control for social integration led to a 37 and 27% reduction in excess stroke risk associated with low education and income, respectively. Liu *et al.*⁵⁶ showed that most (83%) of the association between education and mortality occurred indirectly through a variety of pathways, and moreover, that social resources accounted for approximately 26% of this indirect effect. In a study by Marmot *et al.*,³⁶ accounting for social support reduced the excess CHD risk associated with a low occupational grade by approximately 14% in men, but control for support actually strengthened the occupational effect in women. Another study showed that neighborhood social support reduced the excess risk of CHD incidence and CHD mortality experienced by individuals with low income by 16 and 14%, respectively.⁵⁵ However, support from friends and family was not related to mortality and was not tested as a mediator in this study. Finally, as noted earlier, a study by Wamala *et al.*³³ found that simultaneous control for job strain and social isolation reduced the excess risk associated with low versus high education by 54%.

Four studies examined intra-personal resources as mediating pathways. In one study, accounting for variations in locus of control reduced the SES-mortality associations by 37–57%.⁵⁷ General control beliefs also contributed significantly to explaining the association between SES and incident CHD,⁵⁸ with statistical control for these factors reducing the excess CHD risk for low SES by nearly 30%. As noted earlier, simultaneous statistical control for locus of control and life events reduced the excess mortality risk of less educated participants by 21–48% in a study by van Oort *et al.*³² Finally, Schnittker *et al.*⁴⁹ found that accounting for mastery reduced the association between income and education and total number of chronic conditions by 12% and 4%, respectively, and self-esteem reduced the income and education associations by 13% and 5%.

Finally, three studies examined the aggregate influence of psychosocial resources in explaining SES health disparities. Cohen *et al.*⁴⁴ found no evidence that psychosocial factors including positive and negative emotional style, self-esteem, mastery, purpose in life, optimism, and extraversion contributed to explaining the association between subjective SES

and vulnerability to infectious illness. In contrast, Matthews *et al.*²⁹ found that educational attainment related to incident metabolic syndrome risk both directly, and indirectly, via pathways from low SES, to lower reserve capacity (aggregate of social support, self-esteem, and optimism), to higher negative emotions, to the metabolic syndrome. Similarly, Gallo *et al.*⁵⁹ found that lower SES related to waist circumference (a primary factor underlying the metabolic syndrome) in part through an association with reserve capacity (aggregate of social support, self-esteem, mastery, and optimism).

Conclusions

Our review of the literature examining psychosocial mediating pathways leads to two clear conclusions. First, despite widespread discussion of the possible roles of psychosocial factors in SES-health disparities, evidence is insufficient to draw strong conclusions about the hypothesized relationships. Second, the limited existing literature has produced mixed findings. In general, the evidence seems least supportive of stress as a mediational pathway, even though exposure to stress is believed to be a primary explanation for the association between low SES and poor health.^{12–14,60} The one exception may be job strain, which appears to be promising. The role of psychosocial resources, and particularly intra-personal resources, such as control, mastery, and self-esteem, seems clearer. Because the empirical base is still rather small, it is difficult to ascertain differences in study results due to differences in populations, health outcomes, or SES marker. Finally, no studies have found that psychosocial resources moderate the effects of stress on health, a proposed pathway in the reserve capacity model, although few studies have tested a moderator role. At this point, resources appear to play a role of direct mediation, as opposed to moderation. Overall, additional, and carefully designed research is needed to better test psychosocial factors as possible mediating mechanisms or moderating factors. Later we discuss specific limitations of the literature and directions for future research to continue to evaluate psychosocial explanations for health disparities.

Limitations of the literature

As noted, a primary limitation of the literature is the small number of studies that have directly addressed

psychosocial pathways contributing to SES-health gradients. For example, although stress is frequently mentioned as a pathway linking SES with health, very few studies have examined this hypothesis explicitly. Thus, a primary recommendation is that researchers should take advantage of available datasets collected on a national or local level to better test mediational hypotheses. More studies are needed examining all types of psychosocial mediators—stress, negative emotions, and resource variables, as well as the exploratory pathways discussed further.

A limitation the literature that is available is the use of self-reported health outcomes in some cases.^{45,47,49,61} This is problematic for a number of reasons. First, asking participants to report physician-based diagnoses introduces biases related to access to medical care, because discrepancies in access to quality health care are a well known factor contributing to health disparities.^{62,63} Thus, the link between SES and health outcomes may be attenuated when self-reported diagnoses are assessed, given confounding of prevalence and awareness, especially among people with low SES who are less likely to come into regular contact with the medical system, or who may receive fewer diagnostic tests by nature of inadequate insurance, etc. Furthermore, even assuming equivalent healthcare, individuals with lower SES (particularly lower education) may have difficulty in accurately answering questions about their health status, due to the link between SES and health literacy.⁶⁴ The resulting increased error variance can attenuate power to identify direct or indirect pathways from SES to health. The use of self-reported measures is also problematic when more subjective indexes of illness are administered. For example, the assessment of total chronic conditions used in the study by Schnittker⁴⁹ asked about the presence of a number of health problems that require a judgment call on the part of the respondent. Given the well-known associations between negative emotionality and distress with somatic symptoms—sometimes in the absence of objective underlying disease^{65,66}—including subjective outcomes such as these may lead to biased estimates of the possible roles of psychosocial factors. Thus, additional studies that include objective endpoints (*e.g.*, mortality, or objectively measured indications of disease) in particular are needed.

A further shortcoming of the available literature is the reliance on statistical techniques with lim-

ited potential to clearly elucidate the roles of psychosocial factors. For example, it is typical for epidemiological studies to include blocks of variables in analyses that attempt to “explain” SES-health gradients. Often, these combinations of variables are not clearly conceptually related and moreover, the practice leads to an inability to determine the specific contributions of psychosocial factors as unique from other influences. This approach also results in missed opportunities to identify theoretically meaningful indirect pathways. For instance, the study by Gorman and colleagues⁴⁵ simultaneously examined the contribution of depression and health behaviors to SES-disparities in (self-reported) hypertension prevalence. This approach disallows evaluation of the independent impact of depression; in addition, the influence of depression on hypertension risk may occur indirectly in part, via associations with health behaviors such as smoking and physical activity.⁶⁷ Similarly, Chaix *et al.*⁵⁵ report the additional reduction in the SES–CHD association with control for social support after accounting for other risk factors, but it is probable that, in part, support exerts effects on distal endpoints via proximal influences on clinical and behavioral risk factors. A better strategy would be to perform path analyses that can accommodate multi-step links among psychosocial and other types of mediating mechanisms (*e.g.*, Refs. 32,59). Optimally, structural equation modeling can be used to test conceptually driven hypotheses concerning direct and indirect pathways of interest (for example, Refs. 29,30; for further discussion, see Ref. 1). In addition to considering alternative analytic strategies, it is recommended that researchers report more complete information about the results of tests for mediation. A number of studies concluded that there was “little” or “no” evidence for mediation based on the fact that SES effects remained statistically significant after statistical control for psychosocial pathways. However, in general, the contribution of psychosocial factors (and any single pathway) to SES-health gradients is likely to be small. Because these factors certainly represent only one of many relevant mechanisms accounting for the link between SES and health, even minor contributions in terms of variance explained may be informative and meaningful on a population level. Finally, a further analytic limitation of the available literature is the failure to consider non-linearity in the associations among SES, psychosocial pathways,

and health. That is, psychosocial factors could potentially have a greater impact at certain levels of SES than at others, or different psychosocial factors may be important for determining health of lower versus higher SES individuals.

Another limitation is the quality of measurement. Large studies are needed to understand and isolate the many contributing factors. However, the trade-off of large, nationally based studies is that they often include very limited measures of psychosocial functioning. For example, several studies evaluated in the current review used single-item measures of stress and other psychosocial constructs,^{26,31} or administered brief measures with unknown psychometric properties.²⁸ Even in cases where more complete measures were included, generally only one type of stress (*e.g.*, life events) was examined. Yet, the association between SES and stress is complex, and a multi-dimensional assessment of stress may be necessary to fully understand status-based differences that contribute to health.⁶⁸ For example, a prior study showed that lower SES was associated with the experience of fewer, but more severe, daily-life hassles.⁶⁹ Another study suggested that stressor domain, severity, timing, and perceived risk influenced the nature of the relationship between SES and stress.⁷⁰ Further, over time, consistent exposure to disadvantage may lead to stress habituation,⁷¹ attenuating related appraisals in some individuals. Studies that include multiple measures of stress and other psychosocial variables²⁷ may allow a clearer understanding of the roles that these factors have in SES-health gradients. Moreover, studies that include a smaller number of participants but examine psychosocial experiences at a more refined level (*e.g.*, via approaches of ecological momentary assessment)^{4,19,69} represent important complements to large, epidemiological studies, which have the advantage of adequate power to examine relatively rare objective health outcomes (*e.g.*, mortality, stroke). Likewise, since psychosocial factors could contribute differentially to disparities based on diverse socioeconomic indicators, optimally, more than one measure of SES would be examined.

Population diversity is another important consideration when evaluating the nature of SES-health disparities, and identifying psychosocial and other contributing pathways. Although SES and ethnic disparities are often considered separately, given the closely confounded nature of socioeconomic and

ethnic minority status, a better strategy would be to consider their joint impact. In support of this assertion, some studies reviewed here showed that the link between SES and health largely dissipated with control for other demographic characteristics, and in particular, race and ethnicity (*e.g.*, Ref. 24). In addition, it is possible that SES and ethnic minority status may operate in a synergistic manner, requiring tests of interaction effects. Specifically, individuals with low SES who are also ethnic minorities may be at especially high given the added impact of discrimination and other unique stressors, or, ethnic minority groups may experience “diminishing returns” on health with higher SES, for example, due to factors such as racism in the workplace, “glass-ceilings,” or residential segregation, when compared with non-Hispanic whites.^{72–74} In addition, the association between SES and health is frequently observed to be attenuated in studies of Hispanics and in immigrants. These trends may reflect immigration patterns, or they may indicate resilience to the health implications of low SES as a consequence of health-protective behavioral or social factors.^{73,75} Inasmuch as the SES-related distribution of stress and other psychosocial factors differs according to race/ethnicity,⁷⁶ their roles in explaining SES-health gradients may differ across ethnic groups.

Psychosocial origins of SES-health connections across the life span

This chapter has reviewed the psychosocial pathways connecting SES and health. The populations under study were all adults. Yet, we know that SES in childhood is associated with some indices of adult health, especially mortality from hemorrhagic stroke and stomach cancer, and prevalent cardiovascular disease.^{77,78} Although there are a number of methodological deficiencies in this literature that preclude strong conclusions (Cohen *et al.* this volume), the body of evidence does suggest the low SES in childhood confers risk in adulthood. Furthermore, among children and adolescents, there are strong associations between parental SES and indicators of health status, *e.g.* severity of asthma, physical limitations due to health, and physical inactivity.⁷⁹ These findings suggest that the SES-health connections begin early in life and have long-lasting health effects. We next review what has been learned

about the psychosocial origins of the SES-health connections.

Early family environment

From the psychosocial perspective, an obvious potential contributor to the pattern of psychosocial risk associated with low SES in adulthood is the quality of the early family environment. Relevant family characteristics include overt family conflict, manifested in recurrent episodes of anger and aggression, and deficient nurturing, especially family relationships that are cold and unsupportive or neglectful. Families with these characteristics are conceptualized as “risky” because they may leave their children vulnerable to a wide array of emotional and physical disorders.⁸⁰ Specifically, risky families fail to provide children with the experiences they need to develop effective socioemotional skills.⁸⁰ Instead, offspring from risky families exhibit a propensity to experience and display chronic negative affect and have difficulty developing or maintaining supportive social networks.⁸¹ Consistent with the allostatic load model,⁸² these effects can contribute to the experience of chronic or recurring stress, which, in interaction with genetic predispositions and acquired risks, such as poor health habits, lead to accumulating damage to biological systems. Ultimately, these processes can lead to the increased health risks associated with low childhood SES and an adverse early family environment.

Risky families are more likely to emerge in low SES than high SES families. In both longitudinal and retrospective studies, low SES has been related to all of the “risky family characteristics,” including familial conflict, neglect, and cold, non-nurturant behavior.^{2,80,83} Low SES children are at heightened risk for physical mistreatment or abuse^{84,85} and exposure to family violence,⁸⁶ and they are at greater risk of being in family relationships lacking in warmth and support (*e.g.*, Ref. 87). Both sustained poverty and decline into poverty move parenting into more harsh, punitive, irritable, inconsistent, and coercive directions.^{2,84,88}

A model complementary to and consistent with the risky family model emphasizes the role of *marital* conflict and dissolution, specifically. Troxel and Matthews⁸⁹ suggest that marital conflict leads to decreased monitoring of children, less expressed warmth and affection, and changes in communica-

tion and discipline style. These parenting practices, in turn, are posited to lead to children’s inability to learn how to regulate negative emotions and to develop a sense of security with and attachment to important figures in their lives. Emotional dysregulation and insecurity in childhood may lead to mistrust of others, poor social and coping skills, and feelings of depression, anxiety, and anger. To the extent that marital conflict leads to marital dissolution, then the family also declines in SES, with the consequent additional stress of financial insecurity and insufficient resources. Indeed, adults who experience parental divorce in childhood are less likely to attend college, more likely to be unemployed and on welfare, and less likely to have sufficient financial resources than their counterparts raised in intact families. They also have more problems in relationships with parents and siblings, and in forming and maintaining other intimate relations, including marriage.⁹⁰

Low SES and a risky family environment, including those marred by marital conflict, in turn, have been related to the intermediate pathways such as outlined in Figure 1. A harsh family environment has been tied to poor health behaviors,^{80,91} high levels of depression, hostility, and anxiety,^{80,81} elevated autonomic and cortisol responses to threatening circumstances (*e.g.*, Ref. 92), and poor health (*e.g.*, Ref. 83). Research shows that marital dissolution specifically leads to greater risk for physical health problems in adolescence, including substance abuse and sexual promiscuity,⁹⁰ and maternal-marital dissatisfaction is associated with adolescent offspring’s poor physical health 5 years later.⁹³ Interestingly in an analysis of middle and high school students, adolescents from high conflict intact families had more physical symptoms than adolescents from low conflict divorced families.⁹⁴ Finally, parental divorce in childhood is associated with early mortality among men.^{91,95}

Three investigations have explicitly tested the risky families model in the Coronary Artery Risk Development in Young Adults (CARDIA) study, an epidemiological investigation of risk factors for atherosclerosis and hypertension in young and middle-aged black and white adults. In year 15 of CARDIA, 3225 participants completed measures of childhood SES, early family environment, and adult psychosocial functioning, a latent factor assessed by depression, hostility, and positive and negative

social contacts.³⁰ The model was then tested using these measures in relation to metabolic functioning, C-reactive protein, an inflammatory marker, and high blood pressure. Structural equation modeling showed that low childhood SES was associated with a harsh family environment, which, in turn, was related to the latent factor composed of negative affect and inadequate social support. Results showed that the latent factor was related to poor metabolic functioning in the full sample, although the fit was better for white women and men, and black women, than it was for black men. A similar model characterized the relationships between a harsh family environment and elevations in C-reactive protein⁹⁶ and prevalence of high blood pressure and blood pressure increases across 5 years (this model did not include social support).⁹⁷ Unlike the evidence for metabolic functioning, the model fit the blood pressure data in all four CARDIA subsamples of white and black men and women.

It is clear, however, that not all the variance in relations between childhood SES and health outcomes can be explained through a pathway implicating a risky early family environment. In the blood pressure study, for example, there was a significant direct path from low childhood SES to blood pressure, independent of the relation to early family environment and the psychosocial variables.⁹⁷ Thus, a risky early family environment is best conceived of as one of several potential pathways by which low SES in childhood contributes to adverse health outcomes in adulthood.

Intergenerational transmission of SES

Developed democratic countries offer their citizens the promise of improvement in standard of living with hard work and adequate preparation. Yet, in general, in the United States and in other developed countries, low SES families produce by and large children who grow up to be low SES adults and high SES families produce children who grow up to be high SES adults. Why should low SES be persistent across generations?

In part, this channeling of low SES across generations may reflect the fact that a harsh early family environment leads to poor psychosocial functioning in offspring. Individuals who are high in negative emotions and low in positive emotions may lack the coping skills necessary to attain a higher

educational degree, may be employed but passed over for career advancement, thereby earning lower incomes, when compared to individuals who are low in negative emotions and high in positive emotions. Indeed, data from CARDIA show that among employed individuals, those who had elevated depressive symptoms lost substantial annual income over the next 5 years.⁹⁸

A more direct way that low parental SES may impact the SES of offspring concerns the educational environments associated with low SES communities. Children from low income families have a lower sense of being connected to their schools, have less qualified teachers, and have parents who are less involved in school activities.² Females from lower SES families are more likely to be pregnant as teenagers, thus reducing the likelihood of completing high school or attending college.

Low income children also experience less cognitive stimulation and enrichment at home than do higher income children. In a provocative study by Hart and Risley,⁹⁹ parent-child verbalizations were observed monthly from 6 months to 3 years of age among 42 families that were defined by occupational status (welfare, lower/middle, and professional families). Lower SES children heard fewer words and proportionally more orders or commands and fewer positive comments, relative to higher SES children. These differences occurred even when the child was preverbal at age 9 months to a year: infants from the welfare families heard about 500 words, whereas infants from professional families heard about 1500 words during the observation session. Similar findings have been reported by others.^{100,101} The differences in speech production observed by Hart and Risley⁹⁹ were strongly correlated with higher childhood scores in vocabulary and linguistic complexity more generally (see also Ref. 102). Thus, lower SES children are less prepared to succeed in formal educational environments.

In consequence, it is not surprising that children's academic achievement is correlated with family income¹⁰³ and that the effects may be cumulative over childhood. In the NICHD Early Child Care Network study,¹⁰⁴ the longer that a child lived in poverty from the ages of birth to 9 the more likely the child was to have behavioral problems and low cognitive performance. Being poor later in childhood tended to be more detrimental than early poverty in this sample.⁸⁴ Consistent with the importance of parenting

practices, the link between income and child outcomes was in part due to less positive parenting.

There may also be structural impediments to upward mobility that even highly motivated lower SES children experience. For example, the lack of counseling about higher educational alternatives, the reluctance to provide or to accept educational loans to families with no savings or no income, and the lack of public transportation to travel the necessary distances to search for and obtain good jobs would make advancement difficult. These impediments undoubtedly vary widely by state and region.

Finally, there are some data suggesting that education attainment and to a lesser extent earned income are heritable. For example, based on a nationally representative sample of full and half siblings 28–35 years old, heritabilities due to a common genetic factor were estimated to be 0.52 for education and 0.12 for income, with environmental influences due to a common shared environmental factor being 0.018 for education and 0.08 for income.¹⁰⁵ These analyses, while provocative, do not indicate what heritable traits lead people to attain different levels of social status or which heritable traits might respond differently to life stressors correlated with SES.

Promising future directions

This chapter has concentrated on the role of three classes of psychosocial factors—stress, negative emotions, and resources—in relation to SES and health. There are several other psychosocial pathways at a different level of analysis that may inform the links between SES and physical health. Chen and Matthews⁵ suggest that low SES individuals may be more likely to perceive threat in ambiguous situations in compared to high SES individuals facing the same ambiguous situation. Thus, low SES individuals not only experience more objective stress but also subjectively experience the same situation as more stressful, a pattern of results consistent with the reserve capacity model. Support for this perspective has been obtained in a number of studies of children and adolescents. For example, Chen and Matthews⁵ found that the extent of children's perceiving threat in ambiguous situations was correlated with parental SES based on education and occupation, and that the association between SES and cardiovascular reactivity to stress was mediated

in part by perceptions of threat. Similarly, Chen *et al.*¹⁰⁶ found that in children with asthma, lower family SES, as measured by home rental versus ownership, was associated with heightened production of IL-5 and IL-13 and higher eosinophil counts, and family SES was associated with higher chronic stress and perceived threat. Statistical mediation tests revealed that chronic stress and threat perception represented statistically significant pathways between family SES and immune processes. It is noteworthy that a similar emphasis on threat interpretation has been the focus of studies on poor attachment and divorce: individuals from divorced families are more vigilant to threat cues in contrast to those from intact families who are more avoidant of threat cues.^{107,108}

Low brain serotonergic activity correlated with impulsivity and aggression comprises another candidate pathway connecting SES with health. Manuck *et al.* have suggested that low brain serotonergic activity predisposes to impulsive aggression in both adult humans and primates.¹⁰⁹ Their extensive review of the literature documents that heightened aggressiveness, whether indexed by violent criminal activity, lifetime history of aggression, or aggressive behavior observed in a laboratory setting, is associated with diminished or dysregulated central serotonergic activity (see also Ref. 110). These associations are apparent using several measures of serotonergic function, including CSF 5-HIAA concentrations, neuroendocrine challenges, and responsiveness to serotonergic stimulation of frontal brain regions thought to modulate aggressive behavior. Interestingly, the component of impulsivity that is most highly associated with brain serotonergic activity is the nonplanning component, which is also associated with SES.¹¹⁰ Diminished serotonergic function is associated with individual and neighborhood level measures of SES, adverse health behaviors, metabolic syndrome, and blood pressure in nonpsychiatric samples of middle-aged men and women (*e.g.*, Refs. 111–114).

As we gain understanding on the key psychosocial variables that may connect SES to health, it will be helpful to chart fine-grained models of how brain function and volume may be related to health outcomes (see McEwan & Gianaros, this volume). For example, lower perceived SES is related to smaller hippocampal brain volume and greater amygdala reactivity to threat cues as measured by functional magnetic resonance imaging (fMRI) in several

studies.^{115,116} Individual differences in impulsivity are related to greater activity in the bilateral ventral amygdala, parahippocampal gyrus, dorsal anterior cingulate gyrus, and bilateral caudate, and to lower activity of the ventral prefrontal cortex and dorsal amygdala.¹¹⁷ Adults who report having harsh family upbringing, consistent with the risky families model,¹¹⁸ showed little amygdala reactivity to observing fearful/angry faces and a strong positive association between amygdala activation while labeling emotions in the face and activation of right ventral prefrontal cortex, suggesting a dysregulation in the neural systems involved in responses to emotional stimuli. In contrast, adults from nonrisky families showed the expected amygdala reactivity to observing the faces and activation of right ventrolateral prefrontal cortex while labeling emotions. Thus, adults who grew up in risky families exhibit atypical responses to emotional stimuli that are evident at the neural level.

Identifying and integrating genetic risks may further clarify the pathways connecting SES and health. For example, children who had physician-diagnosed asthma were identified as high or low SES based on parent education and income.¹¹⁹ Genome-wide transcriptional profiles from T lymphocytes were measured. Results showed that children with asthma from a low SES family showed overexpression of genes regulating inflammatory processes, including those involved in chemokine activity, stress responses, and wound responses, compared to high SES children with asthma. Bioinformatic analysis suggested that decreased activity of CREB and NF- κ B, and increased NF- κ B, transcriptional signaling mediated these effects. These pathways are known to regulate catecholamine and inflammatory signaling in immune cells.

Another example is from an analysis of the relation of the serotonin transporter gene to depressive symptoms.¹²⁰ Among young adults with the *s/s* genotype, those who came from risky families were at enhanced risk for depressive symptoms but those who came from nurturant families were at significantly reduced risk for depressive symptoms, relative to those with other genotypes (see also References 121, 122). Thus, early environment importantly moderated the phenotypic expression of the genotype. Childhood SES and physical health was not examined in those studies. However, in another sample, adult SES was associated with low brain

serotonergic function, but only among those with at least one short allele¹²³ also suggesting that SES may serve as a moderator of the phenotypic expression of the genotype. Integrating evidence at the SES level with neural and genetic evidence, psychological data, and assessments of family environment can provide richly detailed information regarding exactly how these variables may inform an overarching model of the relations of childhood SES to adult health outcomes.

Final comments

We began this chapter with a framework regarding how SES may impact health through the emotional/cognitive sequelae of high levels of psychosocial stress, and how SES may reduce resources or reserves for dealing with stress, due to underdeveloped or overused reserves. This framework—called the reserve capacity model—has been tested empirically only a few times. The literature review contained herein suggests that psychosocial resources may be important but not because they impact stress responses. Rather, they may have a direct and mediating effect on health outcomes. The surprising aspect of the review is how much there is yet to be learned regarding the psychosocial pathways connecting SES and health. In that regard, we have suggested a number of design and measurement issues that need to be addressed in future research. We may be approaching the measurement of stress in too simplistic a manner, not taking into account more micro-levels of analysis and domains of stress relevant to different social strata. Similarly, more fine grained analyses of psychosocial processes, such as sensitivity to threat, impulsivity, and correlated brain function, along with measures of SES and health should be fruitful. Even though much is yet to be learned, evidence does suggest that origins of SES-health connections can be traced to psychosocial processes in childhood. Children who are exposed to parental conflict, neglect, and cold, non-nurturant behavior in the family are particularly likely to be at risk for emotional dysregulation, sense of insecurity, and their physiologic sequelae. This suggests that early interventions directed toward children and their parents/guardians may not only impact well being in childhood but have lasting health benefits into adulthood. Given the potential importance of

psychosocial resources, enhancing resources, as opposed to decreasing stress, in high-risk groups may also be worthwhile. We hope that the blueprint for future research provided in this chapter contributes to a better understanding of how SES gets under the skin across the life span.

Conflicts of interest

The authors declare no conflicts of interest.

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