Socioeconomic Status and Health: Mediating and Moderating Factors

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Abstract

Health disparities (differences in health by socioeconomic groups) are a pressing issue in our society. This article provides an overview of a multilevel approach that seeks to understand the mechanisms underlying health disparities by considering factors at the individual, family, and neighborhood levels. In addition, we describe an approach to connecting these factors to various levels of biological processes (systemic inflammation, cellular processes, and genomic pathways) that drive disease pathophysiology. In the second half of the article, we address the question of why some low-socioeconomic-status (low-SES) individuals manage to maintain good physical health. We identify naturally occurring psychosocial factors that help buffer these individuals from adverse physiological responses and pathogenic processes leading to chronic disease. What is protective for low-SES individuals is not the same as what is protective for high-SES individuals, and this needs to be taken into account in interventions aimed at reducing health disparities.

Contents

INTRODUCTION

Pervasive and striking disparities in physical health outcomes exist by socioeconomic status (SES) in our society. For example, those from the lowest-SES groups are 2.5 times more likely to have repeat emergency department visits and 2.7 times more likely to have repeat hospitalizations during a one-year period compared to those from higher-SES groups (Natl. Cent. Health Stat. 2010). Those from the lowest-SES group are also 3.5 times as likely to suffer activity limitations due to disease as high-SES individuals are (32% versus 9%; Braveman et al. 2010). The issue of health disparities has become such a widespread concern that Healthy People 2010, the national health objectives from the US Department of Health and Human Services, labeled the elimination of health disparities as one of two overarching goals (US Dep. Health Human Serv. 2012).

One of the most pressing questions faced by those who are interested in health disparities is why such striking disparities by SES exist across numerous health outcomes. Plenty of hypotheses have been advanced to address this question, but none has provided us with a complete understanding. For example, lack of insurance and access to health care is clearly one reason why low-SES individuals suffer worse health. And yet, countries that have universal health care systems show

the same type of linear, graded relationship (though often less steep) of SES with health as that of countries that do not have universal health care systems, indicating that differential access to care is not the primary explanation for SES disparities (Adler et al. 1993). Low-SES individuals also clearly live under worse physical conditions, with greater exposure to toxicants, pollutants, and other hazards, both in their homes and neighborhoods (Evans 2004). Although these exposures partly account for the SES gradient, they do not explain it entirely. Similarly, low-SES individuals are known to disproportionately engage in health-compromising behaviors such as smoking and inactivity. However, health behaviors account for only a small percentage of the variance in the SES and mortality relationship (Lantz et al. 1998).

Hence, in order to make progress toward the goal of eliminating health disparities, we need a broader understanding of the constellation of contributors to health disparities. In particular, it is important to recognize that psychosocial factors are not contained solely within individuals but also emerge out of the broader family and neighborhood contexts in which individuals are situated. On top of this, in order to create plausible models, research must be able to connect larger psychosocial contexts down to specific biological pathways that are implicated in disease.

In this article, we have two primary aims. The first is to provide an overview of factors at the individual, family, and neighborhood levels that help explain why health disparities by SES exist and to describe an approach to connecting these factors to various layers of biology (e.g., systemic inflammation, cellular responses to infectious stimuli, and genomic pathways that coordinate such responses) that most proximally drive the pathogenesis of disease. The second aim is to address protective factors—that is, to identify naturally occurring psychosocial factors that may buffer individuals who are forced to confront a lifetime under adversity.

MEDIATORS

In describing the social factors that help explain why SES disparities exist, we take an ecological approach. This approach acknowledges that SES creates differences at the individual, family, and neighborhood levels that affect how individuals experience their lives. At the same time, it embraces the view that individuals are nested within larger ecological systems—e.g., family environments, broader social structures—that both shape the individual and are shaped by the individual (Bronfenbrenner 1979). Hence a consideration of the psychosocial factors involved in health disparities must include factors at multiple levels.

We further propose that factors at different levels influence one another in dynamic ways that can alter how each then affects health (Schreier & Chen 2012). That is, factors at different levels should not be conceptualized simply as having independent effects on health (as is implied by studies that examine variables at a single level and their relationship to health), but rather, factors operate in more complex and bidirectional ways to influence health. For example, factors at one level (e.g., the neighborhood) may spill over onto other levels (e.g., the family), in turn, shaping how that factor affects health. Or factors at different levels may have reciprocal effects on each other (e.g., family dynamics affecting child psychological states and vice versa), creating feedback loops that accentuate the effects any one factor can have on health.

Hence, our approach in this section of the review will be first to describe the social context of low SES at the neighborhood, family, and individual levels and their interrelationships, and to discuss connections between these factors and clinical health outcomes (see **Figure 1**). Second, we propose an approach to searching for biological mechanisms that explain how these social factors link to clinical disease outcomes. Our goal with respect to both of the above is not to be exhaustive, but rather to use illustrative studies to articulate the key social characteristics that are implicated in health disparities. In a number of cases, authoritative reviews of certain factors have

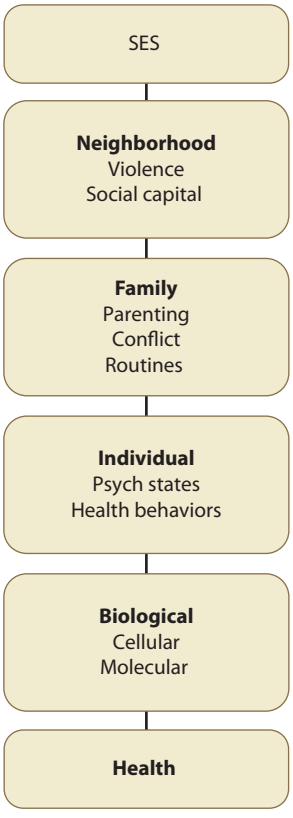

Figure 1

Overview of key pathways at the neighborhood, family, and individual levels linking low socioeconomic status (SES) to poor health.

already been conducted, and where relevant, we point readers to these reviews and use them as a foundation from which to synthesize research across a wide variety of multilevel contributors relevant to health disparities.

NEIGHBORHOOD FACTORS

Violence

The neighborhoods that individuals live in provide a backdrop that shapes the kinds of social exposures that they experience (Diez Roux & Mair 2010). Individuals who live in low-SES neighborhoods are more likely, for example, to witness or be the victims of violence (Buka et al. 2001). An inverse, linear gradient exists between household income and the likelihood of witnessing or experiencing violence (Crouch et al. 2000). Furthermore, violence in low-SES neighborhoods is extremely common, with over 50% of children in these neighborhoods having witnessed severe acts of violence (e.g., shootings, stabbings) across numerous studies (Margolin & Gordis 2000).

In turn, exposure to violence is associated with increased morbidity from a variety of health problems. Greater violence exposure is associated with greater asthma symptomatology (Sternthal et al. 2010, Wright et al. 2004b) and greater risk of cardiovascular disease (Sundquist et al. 2006b).

Experiencing violence is also associated with poorer physical health, greater disability, and more chronic pain (Coker et al. 2000).

Evidence also exists that neighborhood violence serves as one type of environmental exposure—particularly when considered in conjunction with other types of exposures—that helps explain SES and health gradients (Evans & Kim 2010). For example, statistically controlling for cumulative violence exposures reduces the relationship between low household income and poor self-reported health (Boynton-Jarrett et al. 2008). Similarly, exposure to violence (in conjunction with other exposures) has been found to mediate the relationship between poverty and cardiovascular and neuroendocrine biological markers in children (Evans & English 2002, Evans & Kim 2007).

Social Capital

Higher levels of violence in a neighborhood can also alter the social relationships among the members of that neighborhood. Because of concerns about violence, individuals may be less likely to venture outside of their home and hence not know their neighbors as well. Individuals are less likely to trust others in communities where violence is common. In low-SES neighborhoods, research demonstrates that individuals are more likely to believe that people cannot be trusted, that people are not likely to help one another, and that people will take advantage of others if given the opportunity (Sampson et al. 1997). That is, more disadvantaged neighborhoods have lower levels of social capital—lower cohesion and trust among community residents and less willingness to contribute to common community goals (Coleman 1988, Putnam 2000). Neighbors are also less likely to engage in informal social control—that is, to enforce social order by regulating the behaviors of group members (e.g., confronting individuals in the neighborhood who are engaging in deviant behaviors). These two things can then feed into one another, creating a cycle whereby low levels of social control facilitate neighborhood violence and vice versa (Sampson et al. 1997).

In turn, social capital has important implications for health (Kawachi et al. 2008). For example, communities that are higher in social trust (e.g., holding stronger beliefs that others can be trusted) or social capital (social reciprocity, civic participation) have lower mortality rates (Lochner et al. 2003), and residents in communities with high social capital also have better self-reported health (Kawachi et al. 1999). In addition, neighborhoods that are higher in social capital or social cohesion have lower rates of myocardial infarctions and cardiovascular disease (Chaix et al. 2008, Sundquist et al. 2006a).

Social capital also serves as one pathway between low SES and health and well-being (Leventhal & Brooks-Gunn 2000). For example, states that have higher levels of income inequality (greater gap between the rich and the poor) also have lower levels of social trust; in turn, those states with lower levels of social trust also have higher total mortality rates as well as higher mortality rates due to coronary heart disease and malignant neoplasms (Kawachi et al. 1997). In addition, low neighborhood SES is associated with low levels of collective efficacy (residents' willingness to help out for the common good), which in turn is related to an increased likelihood of mortality. Furthermore, collective efficacy mediated the association between low neighborhood SES and all-cause mortality as well as mortality from cardiovascular disease (Cohen et al. 2003).

FAMILY FACTORS

As one example of how family environments matter in health disparities, we focus in this section on the effects that parenting and family dynamics can have on children's health. In this section, we first describe associations of low SES with psychosocial family factors, then describe associations between those same family factors and health outcomes, and finally discuss meditational studies investigating whether family characteristics form one pathway between low SES and poor health outcomes.

Parenting

The characteristics of low-SES neighborhoods can spill over and affect family behaviors. For example, the dangers inherent in low-SES neighborhoods shape parenting behaviors toward children. Parents who live in low-SES neighborhoods are more likely to use controlling and restrictive parenting practices with their children (Furstenberg 1993, Jarrett 1997). They are also more likely to use harsh and punitive parenting strategies, such as corporal punishment (Conger et al. 1994, Dodge et al. 1994). And although there are many reasons why this is adaptive in the context of more dangerous neighborhood environments (e.g., it is better to be harsh and controlling than to have your child be a victim of violence), these types of behaviors are nonetheless generally labeled as negative and associated with poor child outcomes (Repetti et al. 2002).

Parenting approaches may also stem from different philosophies about what is best for one's child in the context of one's life circumstances. Whereas high-SES parents often want to encourage independent thinking and questioning, lower-SES parents often see obedience as critical (Kohn 1977, McLoyd 1990). Obedience helps insure that children do not stray into bad behaviors or fall under the influence of delinquent peers. Thus it is important to understand how differences in parenting styles can stem from the broader neighborhood contexts in which low-SES families are situated.

Nonetheless, certain types of parenting characteristics have been linked to long-term physical health outcomes in children (Repetti et al. 2002). For example, parenting characterized by neglect predicts an increased risk of obesity 10 years later in young adults (Lissau & Sorenson 1994). In the seminal studies conducted on this topic from the Adverse Childhood Experiences (ACE) study, investigators documented that those who grew up in childhood environments involving abusive parenting were more likely as adults to be diagnosed with coronary heart disease (Dong et al. 2004) and to die prematurely (Anda et al. 2009). Conversely, warm, nurturant parenting has been associated with a decreased risk of chronic diseases such as coronary artery disease and hypertension 35 years later (Russek & Schwartz 1997).

Family Conflict

The financial hardships associated with low SES can also take their toll on the quality of family relationships. Low-SES parents often face multiple, competing demands that drain their energy and patience, making family conflict more likely. In order to make ends meet, parents often work multiple jobs and undesirable shifts. In comparison with higher-SES parents, low-SES parents also often have to commute farther to work each day and deal with more frequent problems around the house (e.g., plumbing leaks) because their housing is not as high quality.

These economic pressures cause parents to experience emotional distress and relational problems, which increases the chances they will be distant and irritable with their children, use harsh disciplinary techniques, and depart from their family's typical daily routines (Conger & Donnellan 2007). The multiple demands that parents face, and the reduced time and energy they have for their children, increase the likelihood of conflict. Low-SES families have more frequent conflict and poorer-quality interactions among family members (Conger & Elder 1994). Low-SES parents are more likely to impose rules and to uphold high demands for their children without explaining the reasons behind them (Conger & Elder 1994, McLoyd 1990). Low-SES parents are also more inconsistent in their parenting, punishing one time but not another for the same offense (Conger & Donnellan 2007, McLoyd 1990). These approaches likely stem from the barrage of competing demands that make it difficult for parents to have the time to explain punishments and to apply punishments consistently across situations (McLoyd 1990).

In turn, family conflict and family functioning have established associations with health outcomes (Repetti et al. 2002, Troxel & Matthews 2004). Greater amounts of family conflict are associated with greater health symptoms in youth (Sweeting & West 1995). As well, family conflict and functioning are predictive of the onset of diseases such as asthma (Klinnert et al. 2001) and of metabolic control in youth with diabetes (Miller-Johnson et al. 1994). Also, family conflict during childhood (retrospectively reported) predicts adult illnesses and mortality 13 years later (Lundberg 1993).

Routines

As a result of the multiple competing demands that low-SES parents face in their lives, parents are also less available for their children, which affects families' day-to-day routines. Because of less flexible work schedules, low-SES families are less able to spend time at home with their children (Bradley et al. 2001). They are less available to help their children (McLoyd 1990). And even when there is time, parents have less mental energy because of the toll that life's demands have taken on them. They are more likely to have mental health problems of their own (Conger & Elder 1994), making them less available to provide support when their children need help (Dodge et al. 1994).

In addition, because of competing demands, low-SES children face home lives that are fraught with greater unpredictability (Evans 2004). They are less likely to experience stability in their day-to-day routines (Jensen et al. 1983, Matheny et al. 1995). In particular, when unanticipated demands occur, low-SES parents often have limited resources for addressing them, meaning that effects can spill over onto their children. For example, a bus not showing up results in a parent being late for work and having to make up for missed hours, creating sudden changes to caretaking arrangements for the child. These kinds of experiences can create a sense of chaos in the family environment (Evans et al. 2005), with children experiencing the stresses in their parents' lives through the impact on their own day-to-day schedules.

In turn, family routines affect disease manifestations (Denham 2003). For example, more clearly defined routines within the family are related to youth having fewer asthma symptoms (Sawyer et al. 2000). In addition, in youth with asthma, routines are also related to greater adherence to daily medication and less need for rescue inhalers (Fiese et al. 2005). Similarly, greater daily routines have been linked to better adherence to treatment recommendations among youth with type 1 diabetes (Greening et al. 2007).

Finally, a handful of studies have directly tested family factors such as conflict and routines in mediating the relationship between SES and health. For example, using structural equation modeling approaches, researchers have found that risky family environments (characterized by conflictual, cold families) form one intermediary pathway between low childhood SES and adult metabolic functioning, inflammatory markers, and blood pressure (Lehman et al. 2005, 2009; Taylor et al. 2006). In patient populations, high levels of family conflict (chronic family stress) statistically mediated the relationship between low SES and inflammatory markers in asthma patients (Chen et al. 2003, 2006). Lower levels of family routines (high chaos) also statistically mediated the relationship between low SES and increases over a two-year period in daily output of the hormone cortisol in healthy adolescents (Chen et al. 2010a).

INDIVIDUAL FACTORS

Psychological Characteristics

The broader neighborhood and family environments eventually filter down to shape individual psychological characteristics and behaviors, which have direct implications for health (for a recent

review, see Matthews et al. 2010). For example, low-SES neighborhoods are more fraught with violence, which in turn affects the psychological well-being of those who live in those neighborhoods. When neighborhoods are more violent, both the adults and youth who live in those neighborhoods experience more mental health problems (Stockdale et al. 2007, Xue et al. 2005). In addition, individuals who live in lower-SES neighborhoods (or communities with more unequal distributions of wealth) tend to harbor greater mistrust and cynicism about others, developing personalities characterized by high hostility and pessimism (Barefoot et al. 1991, Heinonen et al. 2006, Kawachi et al. 1997).

In addition, family factors contribute to these types of individual psychological characteristics. Family dynamics affect the psychological states of individual family members, which in turn contribute to future family interaction patterns. For example, low SES is associated with increased family conflict and harsh parenting styles, and these factors in turn are associated with child mental health problems (e.g., depression, anxiety) (Conger & Elder 1994, El-Sheikh et al. 2001). These relationships are also reciprocal, such that mental health problems in one family member feed into greater family conflict in the household. For example, while marital conflict predicts later adolescent depression, adolescent depression also predicts subsequent marital conflict during a two-year period (Cui et al. 2007). Hence, psychological states of individual family members can feed into negative family behaviors that create cyclical and escalating patterns over time (Patterson et al. 1989).

Consistent with all of the above, a large amount of literature has documented that low SES is associated with individual psychological characteristics including negative emotions such as depression and anxiety. These associations are apparent both cross-sectionally and prospectively (for a review, see Gallo & Matthews 2003). As well, low SES has consistent associations with negative emotional and cognitive states such as hostility and pessimism (for a review, see Gallo & Matthews 2003).

In turn, these individual psychological characteristics are associated with poor health outcomes (Kiecolt-Glaser et al. 2002). For example, numerous studies have documented effects of depression on future cardiovascular morbidity and mortality outcomes (for a review, see Rozanski et al. 1999). In addition, a number of prospective studies also find that higher levels of anxiety predict the risk of negative cardiovascular outcomes (for a review, see Rozanski et al. 1999). Finally, those who are higher in hostility are more likely to suffer premature mortality, strokes, myocardial infarctions, and coronary heart disease (Everson-Rose & Lewis 2005, Krantz & McCeney 2002).

A handful of studies have investigated the above types of individual psychological characteristics as mediators of the SES and health relationship. For example, the association between low SES and cardiovascular mortality as well as all-cause mortality was substantially reduced when psychological risk factors (including depression, hopelessness, and social support) were statistically controlled (Lynch et al. 1996). In addition, hostility was found to mediate the relationship between SES and all-cause mortality in men but not women (Nabi et al. 2008). However, in general, researchers have concluded that the evidence is mixed, with some but not all studies finding support for individual psychological characteristics such as negative emotions and personality traits mediating the relationship between SES and health (Matthews & Gallo 2011, Matthews et al. 2010).

Health Behaviors

Neighborhood and family characteristics also shape individual health behaviors, which in turn play an important role in health. For example, when neighborhoods are more dangerous (as low-SES neighborhoods are), parents are more likely to keep their children indoors, meaning that they will be less likely to engage in physical activities such as walking or playing at parks (Carver et al. 2008).

In addition, neighborhood social norms shape the behaviors of individuals within that community (Jencks & Mayer 1990); if, for example, one lives in a neighborhood where deviant behavior such as smoking among peers is more common, youth exposed to those peers will also be more likely to smoke (Alexander et al. 2001).

In addition, the types of family factors that are patterned by SES also play an important role in individual health behaviors. For example, low-SES families have a more difficult time implementing regular family routines; in turn, those families that have less regular routines (e.g., eat together less frequently) have youth who are more likely to be overweight (Taveras et al. 2005). Even years later, those who experienced less regular mealtime family routines in childhood were less likely as adults to eat fruits and vegetables (Larson et al. 2007). In addition, families with less regular mealtime routines have youth who are more likely to drink or smoke (Compan et al. 2002). Negative family relationship factors associated with low SES, such as family conflict, are also predictive of increased smoking and drinking in both youth and adults (Appelberg 1993, Kristjansson et al. 2009).

In turn, these types of health behaviors are important to long-term health. Youth who engage in less physical activity are at greater risk for overweight and obesity (Patrick et al. 2004). Diets that are less healthy are also related to increased risk for overweight and obesity among youth (Delva et al. 2007). Over a lifetime, those who engage in detrimental health behaviors such as smoking, a sedentary lifestyle, and poor diet are at increased risk for cardiovascular disease, cancer, and early mortality (Breslow & Enstrom 1980, Heidemann et al. 2008).

Finally, poor health behaviors form one—though certainly not the only—pathway between low SES and increased morbidity and mortality (Pampel et al. 2010). For example, in one recent study, the association between SES and all-cause mortality in a British civil service population was reduced by 42% when baseline health behaviors were accounted for and by 72% when health behaviors over time were accounted for (Stringhini et al. 2010). The strongest health behavior mediator in this study was smoking. In addition, poor health behaviors such as smoking and physical inactivity increase the effects that stress has on premature mortality among those who are low in SES (Krueger & Chang 2008). However, others have cautioned that health behaviors by themselves cannot fully explain SES and health gradients (Lantz et al. 1998).

PSYCHOSOCIAL MEDIATORS SUMMARY

SES is a broad, distal factor that shapes individuals' health through a variety of pathways at the neighborhood, family, and individual levels. In the above sections, we have attempted to illustrate how these factors are intimately connected with one another and how they simultaneously operate to influence health. Some factors spill over to influence other factors (e.g., neighborhood violence shaping parenting behaviors), whereas others influence each other in reciprocal ways (e.g., family dynamics impacting the psychological states of individual family members and vice versa) that accentuate effects on health. Factors at the neighborhood and family levels affect both individual psychological characteristics (e.g., negative emotions, personality) and individual health behaviors (e.g., smoking), all of which have established relationships with disease outcomes. No one psychosocial characteristic fully explains SES and health gradients, and hence it is important for researchers to acknowledge multiple levels of influences in models of the mechanisms underlying health disparities.

BIOLOGICAL PATHWAYS

The above sections outlined psychosocial mechanisms that help bring SES down to the individual level and connect it to disease outcomes. However, there still remains the "black box" question of

how these social factors operate to alter the pathogenic mechanisms that most proximally affect disease progression. Toward that goal, we recently described a conceptual approach to identifying biological mechanisms that link social factors and disease outcomes (Miller et al. 2009). On the biological end, we argue that it is helpful to target a specific disease and then to understand the basic processes that drive progression of that disease. This then allows researchers to draw on established biomedical understandings of disease pathophysiology and to be able to systematically test which steps within these processes leading to disease are patterned by social factors such as low SES. In this way, researchers can begin to systematically assemble the causal chain of biological mechanisms that underlie the links between SES and disease. The ultimate goal is to lay out a step-by-step mechanistic model of the linear progression from broader social environment to physical health outcome.

To illustrate this approach, we describe below one of our programs of research centered on unpacking the biopsychosocial mediators of childhood asthma disparities. Although numerous other studies have linked SES to biological markers (Cohen et al. 2006, Petersen et al. 2008, Steptoe et al. 2003), and studies have linked psychosocial factors to biological markers in the context of diseases such as asthma (Liu et al. 2002, Marshall & Agarwal 2000, Wright et al. 2004a), the program of research that we describe below is one of the only ones to attempt to systematically unpack the intervening factors between low SES and health outcomes at multiple levels on both the biological and social ends. Hence we focus on it in order to provide an example of how multilevel research can be done across both the biological and social realms. We acknowledge upfront that the biological markers described below are a circumscribed set relevant to asthma. Individuals interested in an overview of the types of biological markers linked to SES should see comprehensive reviews by Seeman et al. (2010) and Matthews & Gallo (2011).

The Example of Asthma

Asthma is a disease involving inflammation of the airways. Chemical messengers of the immune system, known as Th2 cytokines, are important for orchestrating cellular events related to airway inflammation. For example, secretion of the cytokines interleukin (IL)-4 and IL-13 induces B cells to produce immunoglobulin E (IgE) antibodies, which upon binding to allergens trigger a cascade leading to mucus production and constriction of smooth muscle in the airways. This process can result in clinical symptoms of asthma including wheezing, chest tightness, and shortness of breath. Secretion of the cytokine IL-5 recruits cells known as eosinophils to the airways and activates them. These cells orchestrate a more prolonged response that can entail inflammation and obstruction of the airways and result in more persistent clinical symptoms.

Our research has sought to understand the specific types of inflammatory pathways relevant to asthma that are linked to SES, starting with broad, systemic markers and moving progressively toward more specific underlying, causal agents. Biologically, we demonstrated first that children with asthma who came from lower-SES backgrounds had significantly greater eosinophil counts compared to those from higher-SES backgrounds, even after controlling for a variety of medical and demographic characteristics (Chen et al. 2006). This relationship followed a linear pattern, parallel to what the epidemiological studies show for SES and clinical outcomes.

We next investigated the immune processes that foster the production and activation of eosinophils—specifically, the release of IL-5. Here we documented that when the cells of low-SES children with asthma are stimulated, they produce relatively higher amounts of IL-5, at least in comparison to high-SES children. In this case the cells were stimulated in vitro with a mitogen cocktail, roughly modeling what occurs when immune cells encounter allergens in the body (Chen et al. 2003, 2006). Hence, these findings suggest that low SES might prime the immune

cells of children with asthma to respond more aggressively to some allergic stimuli. If so, this response would explain why these children tend to have higher eosinophil counts and worse asthma impairment.

To take this analysis one step deeper, we next explored how SES relates to activity of the molecular signaling pathways that regulate cytokine production as well as other immune cell functions. Here we used DNA microarrays and bioinformatic tools to understand transcription control processes in immune cells. Transcription factors are molecules that relay signals from the external environment of a cell to its nucleus. In the nucleus, they bind to specific segments of DNA called promoters and switch gene expression on or off. Thus, transcription factors regulate the expression of proteins, like cytokines, that are important for the inflammation that contributes to asthma (Busse & Lemanske 2001).

With this in mind, we tested whether SES was related to transcription factor activity in children with asthma. To do so, we conducted genomewide transcriptional profiling of children's T-lymphocytes, which are some of the major cellular players in asthma. The analysis compared activity in children with asthma who came from either low-SES or high-SES families. Based on bioinformatic analyses, low-SES children showed a pattern of increased nuclear factor kappa B (NF-κB) activity and decreased cAMP response element-binding (CREB) activity relative to high-SES children (Chen et al. 2009). NF-κB is a transcription factor that mediates induction of proinflammatory cytokines. CREB is a transcription factor that, among other things, relays signals from the sympathetic nervous system to the immune system, and in doing so regulates cell recruitment, cytokine production, and other processes related to inflammation. These findings suggest that even at the level of transcription factors, activity is patterned by SES. These findings are consistent with other studies that have linked several types of social adversity to gene expression profiles (Cole et al. 2007, Lutgendorf et al. 2009, Miller et al. 2008).

Linking Biology to Psychosocial Mediators

In developing plausible models linking SES to health, it is important to take the next step of formally connecting the presumptive social and biological mediators. To that end, we have found that at the individual level, the associations between low SES and various asthma-relevant biological processes are partially mediated by hostile perceptions of the social world (specifically, a tendency to interpret ambiguous situations in a threatening manner; Chen et al. 2006, 2009).

At the family level, we have found that an experimental manipulation of family conflict results in low-SES youth with asthma showing heightened inflammation in the airways (Chen et al. 2010b). This suggests that the way in which children respond physiologically to family conflict is patterned by SES in children with asthma. Relatedly, we have shown that low levels of family support are associated with higher levels of IgE and eosinophil counts, greater production of various asthma-relevant cytokines, and more resistance to hormones, such as cortisol, that normally slow down the inflammatory process (Chen et al. 2007, Miller et al. 2003). We have also shown that high levels of chronic family stress, when coupled with other negative life events, predispose children with asthma to produce more asthma-relevant cytokines in vitro and express fewer receptors for anti-inflammatory molecules (Marin et al. 2009, Miller & Chen 2006). Together, these findings suggest that children who are experiencing chronic family stress and acute events may be both more vulnerable to inflammation and less responsive to asthma medications (which act through these receptors). Pulling these findings together in more formal analyses, we found that chronic family stress partially mediated the association between low SES and cytokine production (Chen et al. 2006), and furthermore that inflammatory markers partially mediated the relationship between poor family relationships and clinical asthma outcomes (Chen et al. 2007).

BIOLOGICAL MEDIATORS SUMMARY

Through this line of work, we have begun establishing a chain of events that links SES, the broader social environment, and various steps in the pathophysiology of asthma. The latter includes systemic markers of asthma-relevant inflammation (eosinophil counts), cellular processes that contribute to this phenomenon (increased production of IL-5 and other cytokines), and molecular signaling pathways that coordinate these effects in the genome, including activation of relevant transcription control pathways (e.g., NF-κB and CREB). Importantly, all of these relationships are in a direction that is consistent with the clinical phenomenon of children from lower-SES backgrounds experiencing greater asthma impairment.

As is evident, we see much value in documenting mechanisms at multiple levels of analysis, on both the psychosocial and biomedical sides of a research problem. This approach fosters an understanding of where features of the social world intersect with the underlying pathophysiology of a disease. Ideally, research will interweave factors at the individual, family, and neighborhood levels with a multilayered account of disease-relevant biology, and in the process develop a comprehensive causal chain that links the broad social context represented by SES to individual health outcomes.

MODERATORS

Turning now to a different topic, we note that despite the fact that, as we have shown above, low SES is clearly a risk factor for a number of diseases, there are nonetheless a number of disadvantaged individuals who do not show excess vulnerability to medical problems. In the remainder of this review, we explore the question of why some individuals do not get sick even in the face of repeated and severe adversities such as low SES.

FINDINGS FROM PREVIOUS RESEARCH

Much of the previous literature on the notion of resilience (i.e., adaptation in the presence of threat) has focused on psychological adaptation (Luthar 2006). For example, there is a large literature in developmental psychopathology that has discussed resilience from adversities such as abuse, poverty, and war (Garmezy 1985, Masten & Coatsworth 1998, Rutter 1987, Werner 1995). Over the years, this resilience literature has identified key factors at the child (e.g., temperament, cognitive ability, self-efficacy), family (e.g., warm, responsive caregiving), and neighborhood (e.g., effective schools) levels that buffer children facing adversity from behavioral problems and academic failures (Garmezy 1985, Luthar 2006, Masten & Coatsworth 1998, Rutter 1987). However, these studies have largely focused on psychological outcomes, such as social competence, behavioral problems, and academic outcomes (Luthar 2006). Some factors that are clearly important for these outcomes (e.g., effective schools as a way to promote better academic outcomes) may be more tangential to physical health, so our focus has been on identifying psychological characteristics and biological processes that have disease-relevant implications.

Physiologically, the primary buffering concept that has been investigated in the context of low SES or adversity is social support (in particular, maternal warmth). This idea comes in part out of animal models that show that positive prosocial behaviors such as maternal licking and grooming regulate the development of stress response systems in infants, including the hypothalamic-pituitary-adrenal (HPA) axis (review by Caldji et al. 2000). For example, if mothers lick and groom their pups more early in life, their pups show reduced physiological responses to acute stressors as adults, including lower levels of hormones released by the HPA axis (e.g., corticosterone), compared to the offspring of mothers low in licking and grooming (Liu et al. 1997). Moreover, these

effects are clearly due to behavioral experiences early in life rather than genetics. For example, a strain of mice known as BALBc shows reduced HPA responses to stress if they are cross-fostered to C57 mothers, who lick and groom their pups twice as much as BALBc mothers (Zaharia et al. 1996).

Among humans, there is evidence that maternal warmth and related characteristics are beneficial physiologically for those facing adversity. For example, children who have been maltreated show a partial normalization of diurnal cortisol release if their foster parents participate in a program to learn parenting skills (Fisher et al. 2000). In addition, high levels of childhood maternal warmth buffer the effects of poverty on allostatic load (a composite indicator of physiologic risk markers) in children (Evans et al. 2007) and also buffer the effects of low early-life SES on adult metabolic symptoms (Miller et al. 2011). Additionally, among a sample of adults who were all low in childhood SES, those who experienced high childhood maternal warmth exhibited decreased activity of proinflammatory gene networks and decreased inflammatory (IL-6) responses after in vitro microbial stimulation compared to those low in childhood SES and low in maternal warmth (Chen et al. 2011a).

Moreover, long-lasting physiological effects of social support for those confronting adversity can be seen into adulthood. For example, high-quality family relationships during childhood are associated with quicker recovery of blood pressure and heart rate to an acute stressor among adults who experienced childhood adversity (parental loss) (Luecken et al. 2005). In addition, longitudinal studies show that among low-SES (but not high-SES) adults, positive social relationships (high emotional support) were related to decreases in cardiovascular risk and inflammatory activity over an 18-month period (Vitaliano et al. 2001).

SHIFT-AND-PERSIST

The previous studies on the beneficial effects of social support and maternal warmth for low SES/adversity provide a good start. In terms of understanding how these aspects of the social environment could translate into physiologically beneficial effects for an individual, it may be helpful to consider the types of psychological characteristics that support and warmth from others promote in individuals. We recently articulated a model of the psychological characteristics that serve protective functions from a physical health perspective, specifically among those low in SES (Chen & Miller 2012). In particular, we discussed the benefits of a constellation of characteristics labeled "shift-and-persist." The initial premise is that what constitutes "beneficial characteristics" varies depending on the neighborhood and family backdrops that shape daily life. For those low in SES, this typically includes (*a*) the occurrence of repeated, unpredictable, and uncontrollable negative life events such as violence; and (*b*) multiple competing demands (e.g., work demands, financial uncertainties, material deprivation) with limited options for addressing them. Without adequate resources, stressors that arise present themselves as constraints for many low-SES families, rather than solvable problems. And when multiple stressors arise simultaneously, as they tend to do in low-SES families (Hobfoll 2001), this creates competing demands that put additional pressures on low-SES families. For example, a child becomes ill, but the parent will lose her job if she misses work to take care of her sick child. This makes low-SES families vulnerable to "spirals" of resource loss (e.g., parent misses work to take care of sick child, loses job, can't make rent payment, family gets evicted and is now homeless; Hobfoll 2001).

A lifetime of facing constraints with limited options leads some low-SES individuals to value the ability to adjust oneself in response to stressors through emotion-regulation strategies such as reappraisals (shifting). At the same time, in this context, successful adaptation entails enduring adversity with strength, finding meaning in difficult situations, and maintaining optimism in the face of adversity (persisting). We proposed that this combination of approaches to dealing with

adversity reduces physiological responses to stressful situations acutely specifically among those who are low in SES, and over the long term, mitigates the progression of pathogenic processes that lead to physical health problems (Chen et al. 2012). Below we expand on our earlier article by providing an overview of research that describes the benefits of shifting and persisting and their links to physiological outcomes, and then discussing the utility of the combination of shift-and-persist.

Shifting

Shifting entails strategies aimed at adjusting the self to the external environment through tactics such as reframing the meaning of a stressor in less threatening ways. Coping via efforts that emphasize regulating the self may be beneficial because it represents a good fit with the types of constrained situations that are often encountered by those low in SES. That is, given the myriad of day-to-day, largely uncontrollable stressors experienced by low-SES families, their best option may be to control the one thing they can—the self—rather than engage in what may turn out to be futile attempts to control their environment. By controlling the self, they accept that a stressor has occurred and try to change the effect that stressor has on them by reappraising the meaning of an event so that the event evokes less distress in them. They also find other ways to get to their end goal when obstacles arise, thus engaging in behavioral shifts (changing strategies but keeping the goal) that acknowledge the constraints in their lives. As events get reframed as less upsetting, the physiological responses they elicit will be mitigated. Hence we propose that low-SES individuals uphold as an ideal the goal of controlling and adjusting the self when dealing with stress. The ability to successfully do this comprises the "shift" part of our shift-and-persist model.

Adjusting oneself to stressful situations is a coping style that has been referred to in the literature as secondary control coping (Heckhausen & Schulz 1995, Rothbaum et al. 1982). This is in contrast to efforts to change a stressful situation, which has been labeled primary control coping. Previous research has shown that those who are able to adjust themselves specifically during uncontrollable situations have greater psychological well-being. For example, when undergoing uncontrollable painful medical procedures, children with cancer who engaged in secondary control strategies exhibited better behavioral adjustment than children who engaged in primary control strategies (Weisz et al. 1994). Similarly, greater use of secondary control strategies was associated with less distress following the September eleventh terrorist attacks, a presumably uncontrollable stressor (Thompson et al. 2006). Individuals who have passed an opportunity deadline (e.g., women past childbearing age) show more symptoms of depression to the extent that they use primary coping strategies to try to change the situation (Heckhausen et al. 2001). Finally, secondary control strategies such as positive reappraisals are linked to better well-being in older adults, who have fewer remaining life opportunities and future possibilities (Wrosch et al. 2000).

Physiologically, shift strategies such as reappraisal and emotion regulation appear to mitigate pathogenic processes that are implicated in physical health problems. For example, better emotion-regulation abilities are linked to lower allostatic load (a composite measure of physiological risk markers; Kinnunen et al. 2005). Shift strategies can also alter clinical disease outcomes. Shifting in terms of reappraisals (e.g., finding benefit) after a life-threatening event predicts a lower likelihood of having a future heart attack (Affleck et al. 1987). In addition, effectively managing one's emotions (emotional intelligence) is linked to better general indicators of physical health (better self-reported health and fewer illnesses; Goldman et al. 1996, Schutte et al. 2007).

Persisting

Persisting involves enduring adversity with strength by finding meaning in difficult situations and maintaining optimism about the future. This notion has parallels to the literature on resilience

to trauma (Bonanno 2004, Dunkel Schetter & Dolbier 2011), but here we apply it to a low-SES context. For example, the search for meaning allows people to maintain hope, particularly when confronting adversity (Updegraff et al. 2008). Finding meaning increases individuals' sense of security, place, and benevolence in the world, allowing them to understand why adversity has happened to them, facilitating hope and optimism about the future, and enabling adaptation in the face of adversity. Meaning-focused coping is one category of coping that allows individuals to grow in important ways and see value in life, even in the context of adversity (Folkman & Moskowitz 2004).

At the same time as they are seeking meaning, low-SES individuals who engage in persist strategies also are maintaining a steady focus on future, long-term goals. They have ideals and hopes for their future, and they do not let the occurrence or acceptance of day-to-day life stressors detract them from their pursuit of longer-term life goals. Thus they persist in believing that the future can still be bright, and they make active efforts to achieve larger life goals, even as their immediate actions may have to shift somewhat to accommodate the day-to-day stressors that come their way. Consistent with this notion, among a subgroup of low-SES adults who had well-being levels comparable to high-SES adults (resilient low-SES adults), the second most common explanation given for why their life had gone well (after attributions related to upbringing) was about self-development—setting goals and following them, not giving up, and maintaining an effort to achieve the things they wanted in life (Markus et al. 2004).

Although it may seem somewhat inconsistent to accept stressors but yet be proactive in striving for future goals, this profile is possible simultaneously. For example, people are known to activate higher-order goals when faced with an immediate situation that is inconsistent with those goals (e.g., activating larger job-related goals when faced with a temptation to use drugs) (Fishbach et al. 2003). In addition, having a future focus that is positive, such as optimism, makes it easier for individuals to adjust acutely to unsolvable problems (Aspinwall & Richter 1999) and makes individuals more likely to use reappraisal strategies for coping with stressful events (Carver et al. 1993). This type of profile is also consistent with theoretical descriptions of coping and goal pursuit that postulate that secondary control coping can have adaptive value in protecting future goal pursuits (Heckhausen & Schulz 1995).

Physiologically, persistence in terms of optimism, hopefulness, and the maintenance of goal pursuits has been associated with longer-term pathogenic processes relevant to health. For example, optimism is associated with lower levels of inflammatory markers such as IL-6 (Roy et al. 2010), which are implicated in cardiovascular disease. In addition, individuals who report more purpose in life (and hence who may be more persistent in pursuing goals) exhibit lower levels of the soluble IL-6 receptor, a molecule that potentiates the proinflammatory activities of IL-6 itself (Friedman et al. 2007, Ryff et al. 2004). Persisting has also been linked to better clinical outcomes. For example, individuals low in hopelessness or pessimistic explanatory styles live longer and are less likely to suffer myocardial infarctions (Everson et al. 1996, Peterson et al. 1988). In addition, pursuing daily activities related to one's hopes for the future is associated with a decreased risk of mortality over a 10-year follow-up period (Hoppmann et al. 2007). Similarly, those who maintain a greater sense of purpose in life (and hence may persist with long-term goals) have a lower risk of all-cause mortality (Boyle et al. 2009).

IMPORTANCE OF THE COMBINATION OF SHIFT-AND-PERSIST

Given that each of these measures has been the target of investigation in its own right in previous research, is there anything unique about manifesting the combination of shift and persist approaches? We argue that the combination is critical—that is, possessing an approach that values shifting the self in response to stress together with persisting with hopes for one's future will be

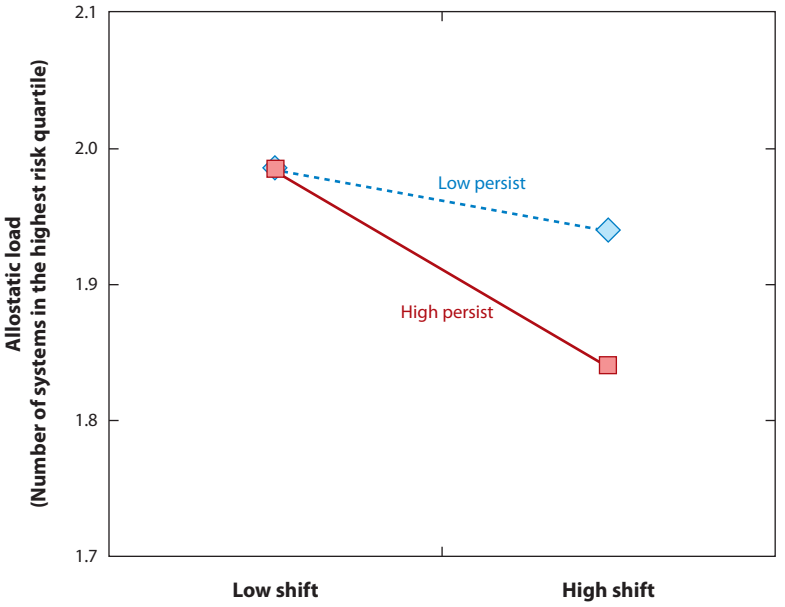

Figure 2

Interaction of shift-and-persist strategies predicting allostatic load among adults from low-childhood-SES backgrounds. Estimated allostatic load scores are plotted at ±1SD of the shift-and-persist variables. No interaction between shift and persist was found among adults from high-childhood-SES backgrounds. Adapted with permission from LWW Journals.

more beneficial than either trait on its own for physiological responses to stress specifically among those low in SES.

We have empirically documented in two studies from our research group the disease-related benefits of shift-and-persist for those low in childhood SES. In the first study, we assessed childhood SES in a national sample of adults. We assessed physiological risk in terms of allostatic load (24 different measures across seven physiological systems were used to form the composite). Shift-and-persist was measured via questionnaires probing coping styles and future orientation. We found a three-way interaction between childhood SES, shift, and persist in predicting cumulative physiological risk in this sample. There was a significant two-way interaction between shift and persist among those from low-childhood-SES backgrounds but not among those from high-childhood-SES backgrounds. The two-way interaction revealed that those low-childhood-SES participants who were high on both shifting and persisting had the lowest allostatic load (see **Figure 2**). In contrast, the combination of shift-and-persist did not predict allostatic load among those from high-childhood-SES backgrounds (Chen et al. 2012).

In a second study using a clinical sample, we investigated the effects of shift-and-persist among children diagnosed with asthma. Among those low in SES, the higher their shift-and-persist scores, the lower their asthma inflammation. Also among low-SES children, higher shift-and-persist scores prospectively predicted less functional impairment (fewer school absences, less rescue inhaler use) six months later, controlling for baseline levels. Low-SES children who were high on shift-and-persist had inflammatory and clinical profiles more similar to high-SES children with asthma than to low-SES children who were low in shift-and-persist. Shift-and-persist was not related to inflammatory or clinical profiles in high-SES children with asthma (Chen et al. 2011b).

Summary of Shift-and-Persist

In sum, low-SES individuals who accept and adapt to life stressors by shifting—i.e., engaging in cognitive reappraisal and emotion regulation—and persisting—i.e., finding meaning and retaining optimism—even in the face of obstacles—appear to have better health outcomes. Specifically, shifting-and-persisting ameliorates some of the inflammatory processes that contribute to asthma impairment, and it also reduces the number of allostatic risk factors that people accumulate by midlife, which might have implications for conditions such as cardiovascular disease.

Furthermore, low-SES individuals may benefit more than other groups from shift-and-persist strategies. For example, among high-SES individuals, a different set of strategies may be beneficial and valued—for example, proactive efforts aimed at eliminating stressors. These proactive efforts are likely to be more effective, given the greater resources, on average, that high-SES individuals possess for engaging in preventive behaviors, resolving situations, and influencing outcomes (Aspinwall & Taylor 1997, Gallo & Matthews 2003, Hobfoll 2001).

Similar to low-SES individuals, older adults are also thought to benefit from secondary coping approaches, given the increasing limitations and constraints they face with age (Heckhausen et al. 2001). However, these life constraints are generally not thought to be reversible. In contrast, low-SES individuals may be constrained currently by their resources, but their futures can still hold promise. Thus, persistence with respect to long-term goals can be beneficial at the same time that secondary coping is useful for addressing immediate circumstances.

COMPARISON OF SHIFT-AND-PERSIST TO OTHER RELATED CONSTRUCTS

A number of constructs overlap with shift-and-persist, and we discuss and differentiate them here. First, as described previously, there is a vast literature on coping and, in particular, constructs related to secondary control coping that parallel the notion of shifting (Heckhausen & Schulz 1995, Rothbaum et al. 1982). However, we propose that it is the specific combination of shifting together with persisting (not just shifting alone) that is beneficial to health among those low in SES. We also contrast shift-and-persist strategies with a style in which one accepts stress but develops expectations that all future stressors will be uncontrollable. This is akin to learned helplessness, which has been associated with increased risk of depression, and in some cases with clinical indicators of disease activity and functional disability in patient populations (Evers et al. 2001, Maier & Seligman 1976). Shift-and-persist incorporates acceptance of stress but maintains a focus on future optimism and future goals.

Similarly, there is resilience literature related to bouncing back from, finding meaning in, and recovering from a life-threatening event (Bonanno 2004, Dunkel Schetter & Dolbier 2011). However, we and others have argued that the adaptive qualities in the context of chronic, enduring stress are different from how one adapts to single occurrences of traumatic events (Dunkel Schetter & Dolbier 2011, Zautra et al. 2008).

The literature on the role of religion in health may also share some parallels with shift-and-persist. That is, many religions emphasize acceptance, having faith in the ability of a higher power to affect life's outcomes, and finding meaning through this. Religion has long been identified as a central, important component, particularly in the lives of many African Americans (who often are also lower in SES) (Mattis & Jagers 2001). In turn, religious involvement has been associated with better health outcomes, such as lower mortality rates (for a review, see McCullough et al. 2000). Religious coping also has been more strongly linked to lower blood pressure among African Americans (who are, on average, lower in SES) than among Caucasians (Steffen et al. 2001). Hence,

although not drawing on the notion of a higher power, shift-and-persist does describe values that overlap with religiosity, and these merit further consideration in future research.

We note that a large literature in developmental psychopathology has discussed a similar phenomenon of resilience (Garmezy 1985, Masten & Coatsworth 1998, Rutter 1987, Werner 1995). These theories have focused on factors ranging from individual temperament and biological traits to developmental processes and social relationships to explain adaptation under adversity (Belsky & Pluess 2009, Caspi et al. 2003, Masten & Coatsworth 1998). However, these articles have largely focused on psychological outcomes, such as social competence, behavioral problems, and psychopathology (Luthar 2006). As such, the constructs in developmental psychopathology that are implicated in resilience differ somewhat from "shift-and-persist" because they do not seek to explain pathogenic mechanisms that are relevant to physical health.

Finally, a number of researchers have framed resilience in terms of the situations to which an individual is exposed. That is, certain types of situational exposures are hypothesized to build resilience. For example, exposure to intermittent stressors has been theorized to create a "toughening" effect physiologically that facilitates subsequent task performance and mitigates emotional reactivity (Dienstbier 1989). And exposure to mild stressors early in life has been argued to be a form of stress inoculation that reduces anxiety, improves cognitive control, and facilitates exploratory behaviors later in life in animals (Parker et al. 2004, 2005). Although this represents a different approach to conceptualizing resilience, we speculate that low-SES individuals who reappraise daily life stressors in less negative ways may in fact experience stressors as milder. The experience of stress, but at less severe levels, may help teach individuals how to successfully navigate stressful situations and produce toughening, rather than damaging, health effects.

CONCLUSIONS AND FUTURE DIRECTIONS

In this article, we sought to provide an overview of multilevel approaches to investigating health disparities and to discuss protective factors that might buffer those who are low in SES from poor health outcomes. We emphasized the importance of considering a variety of factors at the neighborhood (e.g., violence, social capital), family (e.g., parenting, routines, conflict), and individual (e.g., negative emotions, personality, health behaviors) levels and the roles that these factors play in explaining SES and health relationships. We illustrated how these factors are intertwined and hence important to understand simultaneously as part of multilevel models. For example, neighborhood characteristics have spillover effects onto family parenting styles and in turn onto child mental health. Family relationship quality has reciprocal effects with individual mental health, as each affects the other in a cyclical pattern. Without understanding these broader contexts, researchers risk misinterpreting the root causes of health disparities by focusing on an isolated factor at a single level, and hence only partially ameliorating disparities through intervention (for example, targeting child depression but missing the family context—for example, parent marital conflict—that is contributing to both depression and health problems).

In addition to psychosocial factors, we also discussed how one can conceptualize multiple layers of influence biologically leading to disease. Evidence suggests that the influence of social factors such as low SES can be detected at systemic, cellular, and genomic levels. Researchers interested in linking social factors to health will need to conduct more in-depth empirical tests of the specific biological pathways relevant to different diseases that are linked to psychosocial variables of interest. Parallel to the psychosocial side, models should also depict biologically the multiple levels that link psychosocial factors ultimately to clinical disease outcomes.

In sum, future health researchers will need to conceptualize broader multilevel models of health and well-being and to test these empirically. It has become clear that no one factor will fully

explain SES disparities in health, and yet too often in the past, individual variables were studied in isolation, limiting the understanding researchers can have about the contributors to health. Researchers will need to develop broader conceptual models that incorporate a wide array of variables (and their interrelationships) into overarching models and to utilize more sophisticated statistical analysis techniques to test these multilevel models. This will be important both for mechanistic research that seeks to understand why disparities by SES exist in health, as well as for intervention research that seeks to identify what the optimal targets of intervention are for ameliorating health disparities. It will also be important for researchers to specify which types of variables have spillover effects (whereby they influence other factors in a largely unidirectional way) and which variables have reciprocal effects (whereby they and another factor influence each other in cyclical ways). Finally, this research would benefit tremendously from more interdisciplinary work between social scientists (who have complex models of neighborhood and social contexts) and biologists (who have complex models of the biological processes that contribute to disease) that brings these various perspectives together.

The second aim of this article was to provide an overview of protective factors that could help buffer low-SES individuals from poor health. We discussed factors including social support, maternal warmth, and a constellation of psychological traits labeled "shift-and-persist." In investigating protective factors, it is important to consider that factors may not be universally beneficial, but rather there may be factors that are beneficial in one context but not another. Shift-and-persist is one such construct, demonstrating protective effects for low-SES but not for high-SES individuals. These patterns suggest the importance of understanding within-group contexts; that is, in order to better understand and reduce health disparities, we need to identify the factors that are beneficial within low-SES environments and not assume that positive traits identified in the many psychological studies conducted with largely higher-SES populations (e.g., positive emotions, sense of control) would necessarily be effective to promote in disadvantaged groups.

A better understanding of the characteristics that are naturally protective for low-SES individuals raises the possibility that interventions could be developed that would foster these characteristics among other low-SES individuals. If these traits already naturally occur in a subset of low-SES individuals, then presumably they will be feasible to teach in a context-sensitive way to other low-SES individuals. Furthermore, if this type of intervention were effective, this could provide one avenue toward improving health, particularly given the reality within our society of not being able to change the economic circumstances of all low-SES families.

Finally, we raise here the importance of considering more dynamic models in SES and health relationships. Throughout this review, we have implicitly assumed that SES is a static variable in our discussion of its associations with health outcomes. However, it is clear that certain types of SES measures (e.g., income) can fluctuate widely across an individual's lifetime. Hence, we also need to incorporate into the above models a time component in which researchers depict how changes in SES affect neighborhood, family, and individual psychosocial and biological pathways, and in turn trajectories of health over a lifetime.

In sum, the question of the intervening pathways that explain SES disparities is a complex one that requires an understanding across multiple levels spanning from neighborhoods down to the genomic level. In addition, the issue of protective qualities is important to articulate within specific groups and not just generally across the population. Through these approaches, researchers can begin to derive more comprehensive understandings of why health disparities are so pervasive in our society and what can be done to ameliorate them.

SUMMARY POINTS

1. Health disparities mechanistic research requires a multilevel approach that considers factors at the neighborhood, family, and individual levels, and that links this work to biological processes at the systemic, cellular, and genomic levels.
2. Factors at multiple levels are inevitably intertwined and hence important to understand simultaneously. For example, some factors can spill over to affect other factors, whereas other factors will have reciprocal effects with each other, creating dynamic models that impact the magnitude of effect that any given factor will have on health.
3. A subset of individuals is protected from poor physical health despite confronting repeated or prolonged adversities—this is the notion of resilience.
4. Protective qualities that buffer this subset of individuals from poor health are important to articulate within specific groups, as what is protective for one group may not be the same as what is protective for other groups. Understanding what is protective for a particular group requires understanding the broader social context within which a group exists.
5. Characteristics such as shift-and-persist (accepting current stress and reappraising it while holding on to meaning in life and longer-term future goals) may be particularly beneficial for physiological responses to stress and for delaying longer-term pathogenic processes leading to disease in low-SES individuals.

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