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“Shift-and-Persist” Strategies: Why Being Low in Socioeconomic Status isn’t Always Bad for Health

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Abstract

Some individuals, despite facing recurrent, severe adversities in life such as low socioeconomic status (SES), are nonetheless able to maintain good physical health. This article explores why these individuals deviate from the expected association of low SES with poor health, and outlines a “shift-and-persist” model to explain the psychobiological mechanisms involved. This model proposes that in the midst of adversity, some children find role models who teach them to trust others, better regulate their emotions, and focus on their futures. Over a lifetime, these low SES children develop an approach to life that prioritizes shifting oneself (accepting stress for what it is and adapting the self to it) in combination with persisting (enduring life with strength by holding on to meaning and optimism). This combination of shift-and-persist strategies mitigates sympathetic-nervous-system and hypothalamic-pituitary-adrenocortical responses to the barrage of stressors that low SES individuals confront. This tendency vectors individuals off the trajectory to chronic disease by forestalling pathogenic sequelae of stress reactivity, like insulin resistance, high blood pressure, and systemic inflammation. We outline evidence for the model, and argue that efforts to identify resilience-promoting processes are important in this economic climate, given limited resources for improving the financial circumstances of disadvantaged individuals.

Keywords

resilience; socioeconomic status; health; cardiovascular

One of the most striking and robust social factors to influence physical health is socioeconomic status (SES). Those from the lowest SES group are 2.5 times more likely to be hospitalized or visit the emergency department, and 3.5 times as likely to suffer activity limitations due to disease compared to those from high SES groups (Braveman et al., 2010; National Center for Health Statistics, 2010). By age 25, those from the lowest SES group have a life expectancy of 6 fewer years compared to those in the highest SES group (Braveman et al., 2010). Moreover, the effects of SES on health can be seen across a variety of diseases, regardless of whether one investigates prevalence, severity, or mortality rates (Adler et al., 1994; Anderson and Armstead, 1995; Chen et al., 2002). The effects of low SES persist across both countries with and without universal health care (Adler et al., 1993). And they have been demonstrated across the lifespan, from children to older adults (Kubzansky et al., 1998; Starfield et al., 2002).

While these observations have been useful for shaping research into the contributors to disease, they have often left unanswered an important question: Why do some individuals *not* get sick despite facing persistent and severe adversity? For example, in one study, individuals of different social class backgrounds were intentionally exposed to a virus while quarantined and followed clinically to track symptoms of the common cold. Low SES individuals were three times more likely to develop colds than high SES individuals. Despite this relative increase in colds, about 55% of those in the lowest SES category remained cold-free, even though they had all been exposed to the virus (Cohen et al., 2004). A number of researchers have argued that such resilience is more the norm than an anomaly, occurring in anywhere between 35–55% of individuals who confront severe or traumatic stressors (Bonanno, 2005; Masten, 2001). What is it, then, that can protect the health of a subset of individuals who face adversity?

In this paper, we attempt to explain why some individuals do not experience the chronic disease costs of adversity. We focus on a specific kind of adversity - low SES - that is quite common in the United States today, affecting nearly 20 percent of children (Chou et al., 2010). Low SES is defined as poverty, or being from a family with low levels of education, occupational status, or income relative to others in the population. Taking a psychosocial perspective, we present a model of how broader social contexts early in life shape children's views about and responses to the world. Applying this model to physical health, we discuss how these world views shape individuals' response to stressors in ways that, over a lifetime, have effects on physiology and chronic diseases into adulthood. Given our ultimate interest in whether people develop disease later in life, we focus on how broader social contexts are registered at the level of the individual and translated into physiological responses, and why some low SES persons embrace a style we label "shift-and-persist."

On the physical health end, the goal of this paper is to explain how shift-and-persist strategies can alter an individual's long-term disease trajectory across the lifespan. Hence we focus on risk for chronic diseases of aging, such as cardiovascular disease (CVD), stroke, diabetes, autoimmune disorders, and cancer in adulthood. Evidence suggests that there are common biological mechanisms, such as systemic inflammation and insulin resistance, which are relevant to all of these conditions (Chung et al., 2009). Thus, toward our goal of developing mechanistic models, we will discuss processes such as these that represent common precursors to chronic diseases of aging. In addition, much of the emphasis in this paper will be on CVD, both because biologically, there is a strong understanding of the pathophysiological processes underlying CVD, and because the strongest evidence linking both SES and psychosocial shift-and-persist constructs to biological mechanisms revolves around CVD-related processes (Everson-Rose and Lewis, 2005; Kaplan and Keil, 1993; Krantz and McCeney, 2002).

We note that there are a number of other literatures that discuss notions of resilience—that is, adaptation in the presence of threat (Masten and Coatsworth, 1998; Masten, 2001). This includes literature from developmental psychopathology about the predictors of competence (socially, academically, behaviorally) in the face of adversity (Luthar, 2006; Rutter, 1987; Werner, 1995), literature on coping in the face of chronic illnesses such as cancer or HIV/AIDS (Bower et al., 1998; Helgeson et al., 2006; Taylor et al., 2000a; Taylor, 1983), and literature on recovery following acute traumas (e.g., natural disasters, terrorist attacks) (Bonanno et al., 2006; Bonanno, 2004; Bonanno, 2005; Updegraff et al., 2008). While we discuss links to these literatures in more detail later in the paper, we point out here that the vast majority of these literatures deal with psychological adaptation and adjustment, whereas our focus is on chronic diseases of aging, especially CVD, and the psychobiological mechanisms that are involved in their pathogenesis.

In this article, we begin by providing an overview of the basic tenets of the shift-and-persist model. We explain why, over the long term, low SES is a risk factor for many chronic diseases of aging by describing the family and neighborhood environments associated with low SES, and how these environments affect disease risk. Next, we articulate how shift-and-persist strategies can vector low SES individuals away from trajectories of chronic disease. In the final section, we explore the origins of shift-and-persist strategies by discussing the broader social contexts that shape children's development and describing why some low SES children are able to adopt shift-and-persist strategies despite ongoing experiences with adversity.

Overview of Model

In order to develop a model that identifies the psychobiological mechanisms linking low SES to chronic diseases of aging, we focus on the role of stress. That is, we know that those who come from low SES backgrounds face a variety of stressors from their family and neighborhood environments (Leventhal and Brooks-Gunn, 2000; Repetti et al., 2002; Troxel and Matthews, 2004). These stressors typically evoke appraisals of threat (Lazarus and Folkman, 1984) and acute physiological activation of the sympathetic nervous system (SNS) and the hypothalamic pituitary adrenocortical (HPA) axis. If maintained over time, these responses can promote long-term pathogenic mechanisms that result in disease years later (Miller et al., 2009b). The nature of the physiological effects of stress in humans has been described both theoretically and empirically in other reviews (Dickerson and Kemeny, 2004; Gunnar and Quevedo, 2007; Krantz and McCeney, 2002; Miller et al., 2007; Sapolsky et al., 2000; Schneiderman et al., 2005; Segerstrom and Miller, 2004).

To explain, then, how some individuals, despite experiencing these adverse environments, remain free of chronic disease, we discuss individual psychological processes that mitigate perceptions of stress. The idea here is that by identifying the specific psychological processes that ameliorate stress in a low SES context, we can get a handle on how and why some disadvantaged individuals avoid the biological cascades to disease. Developmentally, our model proposes that if in the midst of adversity, children find positive role models, who teach them that the world has others that they can depend on and trust, this allows them to more positively reappraise future stressful situations. Role models also teach children adaptive emotion regulation behaviors. And as children mature, role models orient youth toward their future potential, facilitating optimism about the future and helping them to find meaning in life.

Experiencing this positive foundation during childhood allows some disadvantaged children to develop a "shift-and-persist" approach, which we contend is adaptive specifically for dealing with the circumstances of low SES life. This approach balances acceptance of and adaptation to stress by shifting oneself (adjusting oneself to stressors through reappraisals and emotion regulation), while at the same time, persisting in life (enduring adversity with strength by finding meaning and maintaining optimism). This combination of approaches to dealing with adversity reduces physiological responses acutely, and over the long-term, mitigates the progression of pathogenic processes leading to chronic diseases such as CVD. See Figure 1 for an overview of this model.

In the next section, we provide a background about why low SES – under typical circumstances – is a risk factor for disease. We do this by briefly describing the family and neighborhood environments that are associated with low SES, and then explaining how these environments affect risk for chronic diseases of aging.

The Characteristics of Low SES Environments

The reality of day-to-day life for many low SES families is a barrage of stressors that are not only frequent and recurring, but also largely uncontrollable (Brady and Matthews, 2002). For example, within in the family domain, low SES children have home lives that are unpredictable and chaotic (Evans et al., 2005; Evans, 2004). They are less likely to experience stability in their day-to-day routines (Jensen et al., 1983; Matheny et al., 1995), in part because of demands, over which low SES families have little control, that disrupt these routines. For example, low SES parents often have to work multiple jobs with late shifts that restrict their ability to be at home with their children (Presser and Cox, 1997). In addition, when events occur that present unanticipated changes to daily routines (e.g., bus not showing up to take parent to work, roof suddenly leaking), low SES parents often do not have adequate resources to accommodate these demands, leading to negative consequences that spill over onto their children's day-to-day lives.

Low SES children also experience the interpersonal aspects of family life as more uncontrollable. On average, they experience more frequent conflict and poorer quality family interactions (Conger and Elder, 1994). They are more likely to be the recipients of harsh and punitive parenting strategies (Bradley et al., 2001; Conger and Elder, 1994; Dodge et al., 1994; McLoyd et al., 1994) as well as inconsistent parenting (e.g., punishing one time but not another for the same offense) (Conger and Donnellan, 2007; McLoyd, 1990). These types of family environments have been characterized as 'cold' and 'neglectful' (Repetti et al., 2002), although it is important to note that while the descriptive labels may be accurate, they largely stem from the difficult life circumstances - the multiple demands and constraints - that low SES parents face, rather than being intentional parenting styles.

Within the neighborhoods in which they live, low SES children are also more likely to experience uncontrollable events such as violence. For example, over 50% of inner-city adolescents reported witnessing a violent attack in the past year (Attar et al., 1994). Almost 50% of low income youth reported having witnessed a murder, and close to 75% reported having seen someone shot or shot at (Fitzpatrick and Boldizar, 1993). These types of violence are much more prevalent in low SES communities (Buka et al., 2001).

In turn, these experiences within the broader family and neighborhood social environments provide a framework that shapes the ways in which individuals perceive and respond to stressors (Bronfenbrenner, 1979). For example, low SES individuals are more likely to experience negative emotions including depression, anger, and anxiety (Barefoot et al., 1991; Lynch et al., 1997). Furthermore, these types of negative emotions have robust associations with disease outcomes (Everson-Rose and Lewis, 2005; Kiecolt-Glaser et al., 2002), and have been proposed as one important pathway linking low SES to poor health (Gallo and Matthews, 2003; Matthews et al., 2010). To delve more specifically into the biological mechanisms involved in these associations, in the next section, we describe the types of physiological responses that are elicited in the face of stress and their implications for the chronic diseases of aging.

Physiological Responses to Stress and Links to Disease

Stressors are known to evoke both psychological appraisals of threat as well as acute physiological responses (Lazarus and Folkman, 1984; Selye, 1950; Selye, 1955). The hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) become activated with many stressors, releasing hormones such as cortisol, epinephrine, and norepinephrine (though different stressors elicit somewhat distinct profiles of HPA and SNS activation) (Cannon, 1932; Dickerson and Kemeny, 2004; Kemeny, 2003). These molecules bind to receptors in many different bodily tissues, including those that make up the heart and

blood vessels, and those that carry out metabolic and immune functions. With repeated, prolonged exposure to these stress hormones, the structure and function of these tissues can be altered. Ultimately these changes can give rise to pathogenic processes that drive a variety of chronic diseases of aging. Our focus in this paper is largely on CVD, given its links to SES (Galobardes et al., 2006). Within that context, persistent exposure to higher levels of HPA and SNS products can drive forward a number of the pathogenic mechanisms that ultimately contribute to CVD, including visceral fat accumulation, insulin resistance, systemic inflammation, high blood pressure, endothelial dysfunction, and platelet activation (Brotman et al., 2007; Everson-Rose and Lewis, 2005; Rozanski et al., 2005). These ideas are encapsulated in the concept of “allostatic load,” that is the long-term physiological wear and tear that results from the body’s efforts to maintain stability in response to change (McEwen, 1998), and which has been found to predict CVD later in life (Seeman et al., 1997; Seeman et al., 2001).

Thus the model depicted in Figure 1 proposes that low SES environments in childhood entail a variety of stressors that evoke HPA and SNS activity. If these acute responses are triggered repeatedly, over the long-term they can alter functions of the heart, vasculature, and metabolic and immune systems, fostering pathogenic processes, such as insulin resistance, high blood pressure, and systemic inflammation, that ultimately set the stage for CVD.

Consistent with these ideas, there is burgeoning evidence that links low childhood SES with vulnerability to CVD at much later stages of the lifecourse. Independent of traditional risk factors, individuals raised in low SES families have a 2–3 fold increased risk of experiencing the clinical manifestations of CVD, particularly hemorrhagic stroke and myocardial infarction (Galobardes et al., 2004; Galobardes et al., 2006; Galobardes et al., 2008). Controlling for adult SES does not eliminate these relationships, suggesting that childhood exposures to poverty have a direct influence on later disease risks, which is not simply a function of continuing disadvantage across the lifecourse.

In the upcoming sections we evaluate the evidence relevant to the model’s assertions. First, we review literature that highlights psychologically adaptive strategies for dealing with the stressors commonly encountered in low SES contexts. Second, we review what is known about the linkage between these psychological approaches and biological processes involved in CVD. Given what is specified in the model, we consider outcomes that include biological responses to stress (by the SNS, HPA axis, and cardiovascular system), which are predictive over time of disease risk (Krantz and Manuck, 1984; Linden et al., 1997; Linden et al., 2003; McEwen, 1998; Schwartz et al., 2003). We also consider longer-term pathogenic mechanisms that are more proximal to the development of CVD, such as the accumulation of visceral fat, insulin resistance, high blood pressure, systemic inflammation, and endothelial dysfunction that drive CVD (Brotman et al., 2007; Everson-Rose and Lewis, 2005; Hashimoto et al., 2001; Pradhan et al., 2002; Ridker et al., 2000; Rozanski et al., 2005; Seeman et al., 1997; Vasani et al., 2001).

Shift-and-Persist Strategies in the Context of Low SES

We propose that the socioeconomic context in which individuals reside determines, in part, whether a particular strategy for dealing with stress proves to be adaptive. Through accumulated life experience, low SES children come to embrace certain approaches to dealing with stress that are uniquely suited to meeting the particular challenges of their environment, and doing so given the constraints on resources under which they typically operate. In particular, a lifetime of facing constraints with limited options leads those living in a low SES context to place value on the ability to adjust oneself in response to stressors

through reappraisals and emotion regulation (what we call *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 (what we call *persisting*). In contrast, among high SES individuals, proactive efforts aimed at eliminating stressors 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 and Taylor, 1997; Gallo and Matthews, 2003; Hobfoll, 1989; Hobfoll, 2001).

Shifting

“Life is accepting what is and working from that.”

-Gloria Naylor, novelist who grew up low in SES

Shifting entails strategies aimed at adjusting oneself to the external environment. It can involve reframing the meaning or implications of a stressor in a less threatening manner, and not allowing a stressor to evoke negative emotional reactions in an individual. The benefits of shifting in a low SES context have been articulated in literatures across developmental psychopathology, lifespan development, and cultural psychology. Developmental psychopathology has a long history of theorizing about the contributors to resilience – that is, positive developmental outcomes for children in the face of adversity. One of the key child-level characteristics implicated in this literature is the ability to engage in self-regulation, that is the ability to control one’s interpretations of, and emotional and behavioral responses to, stressful situations (Eisenberg et al., 1997; Luthar, 2006). Specifically, among low income children, better self regulation is associated with fewer behavioral problems and higher emotional wellbeing (Buckner et al., 2003; Fine et al., 2003).

Coping via efforts to regulate the self may be beneficial because it represents a good fit with the types of situations often encountered by those low in SES. Lifespan development theories of coping postulate two types of control efforts, primary control coping (efforts to influence the external world) and secondary control coping (efforts to adjust the self to fit into the world) (Heckhausen and Schulz, 1995; Heckhausen et al., 2010; Rothbaum et al., 1982). (A similar construct of accommodative (akin to primary) and assimilative (akin to secondary) coping also exists (Brandtstadter and Renner, 1990; Brandtstadter and Rothermund, 2002)). Efforts to exert primary control are considered a fundamental human motivation, but because doing so is not always possible, this necessitates an alternative approach of secondary control coping. Secondary control coping is thought to be beneficial during circumstances in which primary control coping is not possible (e.g., uncontrollable events), and is beneficial to the extent that it preserves motivation for future primary control efforts with respect to other goals. Because economically disadvantaged individuals have fewer opportunities to select or modify their life situations (alternative forms of emotion regulation; (Gross, 1998; Gross, 2001)), reappraisals represent a realistic approach to emotion regulation for those low in SES. Thus for those low in SES, an approach that emphasizes adjusting the self (through reappraisals and emotion regulation) should be a feasible and beneficial strategy for dealing with the multitude of day-to-day stressors presented by life in a disadvantaged environment.

However, our model departs somewhat from lifespan developmental theories, which assign primary control strategies higher status in the hierarchy of coping responses than secondary control strategies. These models assume that the human motivation for primary control is not only fundamental, but universal. Our model suggests a somewhat different take. We postulate that as a result of accumulated life experience, low SES individuals come to embrace secondary control coping strategies as an ideal. This hypothesis is informed by

social class theories of agency, which state that there are different ways to be agentic – that is, to act in and respond to the world – and that different cultural groups prefer different types of agency, or control, with no one type being necessarily better (Markus et al., 2004). For example, low SES individuals emphasize controlling the self over and above controlling a situation (Snibbe and Markus, 2005). They accept not having much choice in life, without letting it affect their likes and dislikes (Snibbe and Markus, 2005). They also tend to respond to major life stressors by accepting and enduring them (Stephens et al., 2009). Furthermore, low SES individuals are more likely to explain the causes of life events through external, contextual factors, rather than individual, dispositional factors (Kraus et al., 2009), suggesting that they accept that the broader social environment exerts control over their lives. In contrast, high SES individuals prefer to exert control over situations and to be able to work toward eliminating or mitigating stressors (Stephens et al., 2009).

Hence we propose that low SES individuals uphold as an ideal the goal of controlling and adjusting the self when confronting stressors through reappraisals and emotion regulation. The ability to successfully do this comprises the “shift” part of our shift-and-persist model.

Persisting

“You have to be so strong-minded to survive. You do the best you can do, and if you fail, you get up again. That’s all you can do.”

-Hurricane Katrina survivor, low SES (Stephens et al., 2009)

Drawing on the resilience to trauma literature (Bonanno, 2004; Dunkel Schetter and Dolbier, in press), we suggest that in a low SES context, persistence involves enduring adversity with strength, holding oneself steady and finding meaning in difficult situations, and maintaining optimism about the future. Of particular importance to adjustment is finding meaning. The search for meaning has been argued to be a basic human motivation that allows people to maintain hope, particularly when confronting adversity (Frankl, 1963). Finding meaning increases individuals’ sense of security, place, and benevolence in the world, allowing them to maintain hope and optimism, and enabling adaptation in the face of adversity. Finding meaning has been found to be beneficial in the context of personal traumas (e.g., spinal cord injury (Bulman and Wortman, 1977)), as well as during collective traumas such as terrorist attacks (e.g., 9/11 (Updegraff et al., 2008)). Consistent with these notions, meaning making in the developmental psychopathology literature has been identified as one important factor in how vulnerable children cope with challenges (Luthar, 2006). Similarly, coping researchers have identified meaning-focused coping – the use of cognitive strategies to manage the meaning of a situation – as a primary category of coping (Folkman and Moskowitz, 2004), and have theorized that the search for meaning is useful for reconciling the occurrence of major stressors with one’s beliefs about the world and self (Folkman and Moskowitz, 2004; Janoff-Bulman, 1989). Meaning allows individuals to perceive benefits from stressors, and to grow in positive ways and experience positive affect, despite the occurrence of major stressful life events (Folkman and Moskowitz, 2000).

Finding meaning may also be beneficial for facilitating the ability to endure adversity with strength. Experiences with adversity can sometimes promote “toughness” (Dienstbier, 1989), a characteristic that has particular value among low SES individuals. For example, low SES individuals who faced a life threatening event (Hurricane Katrina) more frequently mentioned themes related to maintaining strength and not giving up, compared to high SES individuals, who emphasized taking control of the situation (Stephens et al., 2009). Similarly, when low SES individuals discussed the characteristics of a good life or their hopes for the future, they were more likely than high SES individuals to mention themes related to “hanging tough” and endurance (Markus et al., 2004).

Hence we propose that low SES individuals also uphold as an ideal values related to successfully enduring adversity by drawing meaning, maintaining optimism, and persisting with strength in the face of adversity. The ability to do this comprises the “persist” part of our shift-and-persist model.

In addition, we argue that there is something critical to the combination – that is, shifting plus persisting, which reduces risk for chronic diseases of aging specifically among those low in SES. The two components have some known reciprocal effects; for example, optimism facilitates shifting, making it easier for individuals to adjust acutely to unsolvable situations (Aspinwall and Richter, 1999), and making individuals more likely to use reappraisal strategies for coping with stressful events (Carver et al., 1993).

Hence the label that we use, “shift-and-persist,” is intended to connote a combination of characteristics, including shifting oneself in the face of stressors by adjusting appraisals and emotional reactions, together with persisting by maintaining optimism and drawing meaning out of adversity. We argue that this “shift-and-persist” approach will be beneficial specifically to low SES individuals not only for psychological well-being, but also physically over the long-term, in terms of chronic diseases such as CVD. In the next section, we discuss the implications of shift-and-persist strategies for such diseases, and the empirical evidence supporting these links.

Are Shift-and-Persist Strategies Related to Disease-Relevant Processes?

Above we outlined a rationale for why it would be beneficial, psychologically, to possess a shift-and-persist approach for dealing with many of the daily life stressors that arise in a low SES context. However, the question remains of whether benefits would also extend to the biological processes that drive chronic diseases of aging like CVD. Theoretically, an approach that values acceptance, adapting the self, and maintaining strength and optimism should mitigate negative appraisals and negative emotions in response to many of the challenges associated with low SES life. This should, in turn, dampen stress reactivity. Over the long term, this would reduce tissue exposure to hormonal products of the HPA and SNS and, in doing so, forestall the development of pathogenic processes that low SES normally sets into motion.

Empirical evidence links components of shift-and-persist strategies to stress reactivity, intermediate pathogenic mechanisms, and clinical outcomes. For example, shift strategies of reappraisals are more beneficial than other emotion regulation strategies (e.g., emotional suppression) at reducing cardiovascular reactivity to acute laboratory stressors (Gross, 1998). More benign appraisals of threat have been associated with reduced blood pressure reactivity during acute stressors in both children and adults (El Sheikh and Harger, 2001; Maier et al., 2003), and lower ambulatory blood pressure in adolescents during daily life social interactions (Chen et al., 2007). Individuals high in the ability to reappraise stressful situations or regulate their emotions show reduced vascular reactivity during acutely stressful tasks (Mauss et al., 2007). In addition, better emotion regulation abilities are linked to more favorable allostatic load profiles, whose components include CVD risk factors like higher HDL cholesterol, lower triglycerides, and lower basal systolic blood pressure (Kinnunen et al., 2005).

Similarly, persistence, in terms of optimism and meaning is also associated with dampened reactivity to stress, more favorable risk profiles, and fewer adverse health outcomes. For example, higher levels of optimism are associated with lower blood pressure during daily life (Raikkonen et al., 1999), better in vitro natural killer cell cytotoxicity, a type of immune response involved with infectious disease (Segerstrom et al., 1998), less systemic inflammation (Roy et al., 2010), slower progression of carotid atherosclerosis over a 10–13

year period (Matthews et al., 2004), reduced likelihood of developing coronary heart disease (CHD), and better chances of quick recovery after treatment for CHD (Giltay et al., 2004; Kubzansky et al., 2001; Maruta et al., 2000; Scheier et al., 1989; Scheier et al., 1999). Similarly, individuals who report strong purpose in life (related to finding meaning) have less cortisol secretion across the day (Lindfors and Lundberg, 2002; Ryff et al., 2004), lower basal levels of the IL-6 receptor (which serves to amplify the effects of the pro-inflammatory cytokine IL-6) (Friedman et al., 2007; Ryff et al., 2004), and a lower risk of all-cause mortality (Boyle et al., 2009). Those who believe that the world is just and fair (one consequence of meaning making) are less likely to appraise an acute stressor as threatening and show less vascular reactivity during that stressor (Tomaka and Blascovich, 1994). Finally, finding meaning (benefit) after a life threatening or traumatic event also predicts a lower likelihood of having a future heart attack (Affleck et al., 1987), and a lower likelihood of mortality from AIDS (Bower et al., 1998).

Taken together, these data provide support for the notion that both the “shift” strategies of reappraisal and emotion regulation, as well as the “persist” strategies related to optimism and meaning are plausible candidates for promoting physical health resilience because they have been linked in previous literature to biological processes involved with CVD pathogenesis and clinical outcomes like morbidity, mortality, and treatment recovery. In the next section, we review evidence that these factors are beneficial specifically for low SES individuals.

Shift-and-Persist Strategies Among Low SES and Links to Disease Processes

We argue that shifting is beneficial in terms of reducing risk for the chronic diseases of aging among those low in SES. Consistent with this line of reasoning, research shows that when low SES individuals make active efforts to control situations – the opposite of shifting – a toll is exacted on some bodily systems involved with CVD. For example, low SES adults who are high on John Henryism (the tendency to use active coping efforts for dealing with stressors that are largely uncontrollable) show higher blood pressure, greater total peripheral resistance, and increased risk of hypertension compared to those who are low in SES and low in active coping, or those who are high in SES and high on active coping (James et al., 1983; James et al., 1987; James et al., 1992; Wright et al., 1996).

In a different study of adolescents that reached similar conclusions, participants were randomized to receive one of two psychosocial interventions for reducing blood pressure reactivity. Among low SES adolescents, experimentally providing control over an acutely stressful situation did not reduce blood pressure or heart rate responses during the stressor; in contrast, an intervention that involved resources being provided by a supportive other was more effective at reducing cardiovascular reactivity among low SES adolescents (Chen, 2007).

A different line of work suggests that the consequences of stress may fall along an inverted U-shaped curve. According to this work, health benefits accrue with exposure to modest stress, and this phenomenon may be similar to the effect that shifting has on low SES individuals. For example, there is animal literature on resilience that has documented that the experience of mild stressors is adaptive (and in fact, better than not experiencing stress) for cognitive, anxiety, and cortisol profiles (Parker et al., 2004; Parker et al., 2005). This parallels findings in the human literature that a moderate amount of cumulative lifetime adversity is associated with less functional impairment and distress compared to no adversity (Seery et al., 2010). We extrapolate from this work to hypothesize that low SES individuals who are able to reappraise daily-life stressors in less negative ways are in fact

experiencing these stressors as less severe. The experience of stress, but at mild or moderate levels, is thought to be beneficial because it creates ‘toughening’ experiences (Dienstbier, 1989) that help teach individuals how to successfully navigate stressful situations. Thus low SES individuals who “shift” themselves in response to stress may be experiencing daily life as a series of less severe stressors that have toughening, rather than damaging, health effects.

Hence, the above findings suggest that active efforts to control or eliminate stressors are ineffective for dampening reactivity among those low in SES, and that a more successful approach involves efforts to control the self, rather than situations. One other study provides somewhat ambiguous evidence with respect to control and self-reported health outcomes. In that study, individuals who were low in SES but high in perceived control had profiles of self-reported health, acute health symptoms, and functional limitations similar to high SES individuals, and better than those of low SES individuals with low perceived control (Lachman and Weaver, 1998). However, it is important to note that the distinction between primary and secondary control was not made in this study, and hence the control items may have encompassed both types of control.

A separate set of studies provides evidence that shifting in terms of emotion regulation is beneficial for long-term susceptibility to CVD. These studies collectively show that heightened expression of negative emotions, particularly anger, has stronger associations with cardiovascular disease risk (carotid artery intima-media thickness, risk of myocardial infarction) among those low in SES compared to those high in SES (Mendes de Leon, 1992; Merjonen et al., 2008; Mittleman et al., 1997). This suggests that the ability to effectively regulate the expression of such negative emotions might be more beneficial to cardiovascular health among those low in SES.

With respect to the notion that persisting is beneficial among those low in SES, previous research has examined disease-relevant correlates of meaning and optimism. For example, one study recruited a nationally representative sample of mid-life U.S. adults. Among those individuals who were low in SES (low levels of education), higher purpose in life (similar to meaning in life) was associated with lower levels of IL-6, a marker of systemic inflammation that predicts, and likely has a role in, CVD pathogenesis. By contrast, purpose in life was not associated with IL-6 in high SES adults. Levels of IL-6 among those who were low in SES but high in purpose in life were similar to those of high SES individuals (Morozink et al., 2010).

A similar pattern was found with respect to optimism. In a study of mid-life women, among those who were low in SES (based on low education and income), those who reported high pessimism (or conversely, low optimism) had higher ambulatory blood pressure readings, and a higher likelihood of hypertension compared to those who were low SES and low in pessimism, those who were high SES and high in pessimism, and those who were high SES and low in pessimism. The latter three groups were all similar to one another in terms of ambulatory blood pressure readings and likelihood of hypertension (Grewen et al., 2000). These findings suggest that low pessimism/high optimism specifically buffers low SES women from detrimental cardiovascular risk profiles.

Finally, we address the notion that the combination of shift-and-persist is important for biological processes involved in chronic diseases. In two studies from our research group, we document disease-related benefits of shift-and-persist for those low in SES. In the first study, we utilized a nationally representative sample of U.S. adults, and assessed the childhood circumstances from which they came. We assessed shifting utilizing measures of secondary control coping (positive reappraisals) and emotion regulation (emotional reactivity to stress), and assessed persisting using a measure of future thinking. Biological

assessments consisted of 24 different measures across seven physiological systems, from which a composite measure of allostatic load was created. We found a significant 3-way interaction among childhood SES, shift, and persist. Shift and persist scores interacted to predict allostatic load among adults from low childhood SES backgrounds. However, there was no association between shift-and-persist and profiles of allostatic load among those from high childhood SES backgrounds. The nature of this interaction was such that among those from low childhood SES backgrounds, the combination of high shift and high persist was associated with the lowest allostatic load scores. In addition, there were no significant main effects of shifting or persisting on allostatic load in either the low or high SES groups (Chen et al., under submission). These findings suggest that the combination of shift and persist is beneficial for allostatic load only among those who grew up in low SES households, whereas shift and persist strategies are not beneficial to those who grew up in high SES households.

In a second study, we investigated the effects of shift-and-persist in a clinical sample of children diagnosed with asthma. SES in this study was measured as family resources (e.g., family savings). Shifting was measured based on positive reappraisals, and persisting was measured in terms of optimism. At baseline, asthma-related inflammation measures were obtained, and children were also followed over a six month period in terms of clinical indicators (rescue inhaler use, school absences due to asthma). We found significant 2-way interactions between SES and shift-and-persist, such that among those low in SES, higher shift-and-persist scores (in this case, a total score reflecting the use of both shift and persist strategies) were associated with lower levels of asthma inflammation at baseline. In addition, among low SES children, higher shift-and-persist scores prospectively predicted less asthma impairment (fewer school absences, less rescue inhaler use) six months later, controlling for baseline asthma impairment. In fact, low SES children with high shift-and-persist scores looked similar to high SES children in terms of asthma impairment. In contrast, shift-and-persist did not benefit high SES children with asthma, either in terms of inflammation or clinical impairment scores (Chen et al., in press).

In sum, the literature described above demonstrates that shift-and-persist strategies are beneficial with respect to chronic disease risk among low SES individuals, dampening acute physiological responses to stress, retarding longer-term pathogenic processes like allostatic load, and as well, reducing clinically relevant disease outcomes. In contrast, no benefits of shift-and-persist strategies have been found among high SES individuals.

Other counter-regulatory processes

We note that the above evidence focuses around the idea that shift-and-persist strategies dampens stress-evoked responses of circuits like the SNS and HPA axis. This is a useful starting point, because the bulk of the existing data address stress reactivity in these systems. However, it is also possible that shift-and-persist strategies could activate different biological processes that serve to counteract the effects of stress. This would be consistent with the notion of enhanced allostasis (Bower et al., 2009), which occurs when people find benefits in traumatic experience, and as a result show “more efficient, circumscribed, and tightly regulated (p. 229)” physiological responses to later stressors (Bower et al., 2008). There are several biological processes that could function in this capacity.

Oxytocin (OT) is peptide hormone implicated in a broad array of interpersonal processes, including social affiliation, nurturing behavior, and emotion recognition (Ross and Young, 2009). Evidence suggests that peripheral OT levels increase when people experience feelings like warmth, trust, and security (Grewen et al., 2005; Light et al., 2005). In humans and animals, intranasal OT administration significantly diminishes the magnitude of HPA and SNS responses to acute stress (Ditzen et al., 2009; Gutkowska and Jankowski, 2008;

Heinrichs and Gaab, 2007; Heinrichs et al., 2003; Taylor et al., 2000b). On a more chronic basis, peripheral OT could retard the pathogenic mechanisms leading to disease. OT is increasingly recognized as having anti-inflammatory properties (Clodi et al., 2008; Nation et al., 2010). Animal models also suggest that peripheral OT action counters obesity, facilitates glucose control, and improves insulin sensitivity, processes that are centrally involved in the CVD precursor condition of metabolic syndrome (Camerino, 2009). Hence to the extent that low SES individuals develop trust in others, based on early life experiences with positive role models, not only will this facilitate engagement in shift-and-persist strategies, this may also increase availability of OT, which in turn could counter many of the health-damaging biological processes that low SES normally initiates.

Second, the parasympathetic nervous system (PNS) could also function in a similar capacity to attenuate acute stress responses and slow down resulting pathogenic mechanisms. Studies have found that aspects of shift-and-persist, such as flexible coping, emotion regulation, and benefit finding, are associated with greater heart rate variability (HRV) (Appelhans and Luecken, 2006; Bower et al., 2008), which is viewed as an index of PNS control over the heart rhythm. HRV has also been proposed as one biological component of resilience in the developmental psychopathology literature (Calkins et al., 2007; Shannon et al., 2007). PNS vagal fibers innervate the heart and the vasculature, and in general function to counter the influence of the SNS. There is some evidence to suggest the PNS is capable of regulating HPA outflow via its influence on the central autonomic network (Thayer and Sternberg, 2006). The PNS also seems to play a role in regulating CVD-related pathogenic mechanisms, such as glucose control (Thayer and Lane, 2007). And a “vagal anti-inflammatory reflex” has been described in mice (Pavlov and Tracey, 2005), which enables the PNS to regulate macrophage production of inflammatory mediators via cholinergic signaling. Hence, there is evidence that the PNS has regulatory influences on a number of allostatic systems (Thayer and Sternberg, 2006). To the extent, then, that shift-and-persist strategies engage the PNS, this may serve to regulate some of the physiological responses typically seen in response to stress.

Finally, some authors have suggested that anabolic hormones might also have a role in fostering resilience to stress (Bower et al., 2009). In a handful of studies, constructs such as active coping, benefit finding, and positive emotion have been linked to levels of anabolic hormones, including growth hormone, dehydroepiandrosterone, and insulin-like growth factor (Bower et al., 2008; Pressman and Cohen, 2005). As these hormones promote growth and recovery of tissues and organs, they might help to counter stress-related wear and tear on systems involved in CVD. However, these ideas are still preliminary and will need to be evaluated more fully before any definitive conclusions about their role can be rendered.

Role of Health Behaviors

Finally, another important pathway by which low SES circumstances may lead to poor health is via effects on health behaviors (see Figure 1). Evidence indicates that individuals from lower SES backgrounds are more likely to engage in unhealthy behaviors, such as smoking, sedentary lifestyles, high fat diets, and in some cases, substance use (Hanson and Chen, 2007; Jacobsen and Thelle, 1988; Lowry et al., 1996). This may be in part because low SES environments contain barriers to healthy behaviors. For example, low SES neighborhoods are more likely to lack physical activity-related facilities (Gordon-Larsen et al., 2006), and fresh, affordable produce in local grocery stores (Moore and Diez Roux, 2006). Furthermore, the stress of life in a low SES environment may trigger certain unhealthy behaviors (e.g., smoking) as coping mechanisms (Stead et al., 2001). Thus as depicted in Figure 1, the stressors associated with low SES contexts may also lead to more

detrimental health behaviors, which in turn will have negative consequence for disease pathogenesis and progression.

In addition, shift-and-persist strategies may also have implications for health behaviors. In particular, persist strategies related to maintaining hope about the future may help with promoting positive health behaviors. Individuals who are more future-oriented are less likely to engage in detrimental health behaviors, such as smoking and using drugs or alcohol (Keough et al., 1999; Robbins and Bryan, 2004; Robbins and Bryan, 2004; Wills et al., 2001). To the extent that low SES individuals utilize shift-and-persist strategies (in particular, focusing on the future) to refrain from engaging in behaviors that are pleasurable in the moment (e.g., smoking), but detrimental in the long term, this may create an additional pathway mitigating the chronic pathogenic processes leading to CVD.

Summary of Shift-and-Persist Model & Links to Other Literatures

In sum, among low SES individuals whose lives often entail recurrent and uncontrollable stressors, accepting and adapting to life by shifting oneself - engaging in reappraisals and emotion regulation - acutely reduces the magnitude of physiological responses to stress, and over the longer term slows down pathogenic disease processes. In addition, persistence – that is, enduring adversity with strength by holding on to meaning and optimism in life- is associated with more favorable cardiovascular, neuroendocrine, and immune profiles among those low in SES.

We propose that the combination of both shift and persist (more so than either one on its own) is important for reducing risk over the long-term of chronic diseases of aging, and furthermore, that this constellation is beneficial specifically to low SES individuals. Shifting without persisting has the potential to lead to learned helplessness (passive acceptance of all stressors), 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 and Seligman, 1976). Furthermore, shift-and-persist is postulated to be uniquely adaptive among low SES individuals because it represents a good fit with their environmental conditions. In contrast, among high SES individuals, proactive efforts at coping that are aimed at reducing or eliminating a stressor may 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 and Taylor, 1997; Gallo and Matthews, 2003; Hobfoll, 1989; Hobfoll, 2001). Of course, there will be instances in which low SES individuals can exert primary control, and instances in which high SES individuals will need to exert secondary control, and consistent with the coping flexibility literature (Cheng, 2001), it will be useful for individuals in both groups to calibrate their coping responses to a specific situation's demands. Nonetheless, our theory postulates that low and high SES groups come to value different types of coping strategies based on their cumulative life experiences, and preferentially use the strategies that are more valued by their group.

We also note that there is a large and burgeoning literature on positive psychological features and their association with physical health outcomes (Fredrickson, 2001; Pressman and Cohen, 2005; Seligman and Csikszentmihalyi, 2000; Taylor et al., 2000a). While clearly important to the field, this literature has largely taken a 'main effects' approach – that is, identifying the positive psychological characteristics that are beneficial to health across populations. In contrast, in this paper we have sought to articulate qualities that are uniquely beneficial to a certain group. This is consistent with literature in sociology, for example on neighborhood effects, that argues that the qualities that are beneficial in one context (e.g., high SES neighborhoods) may not be beneficial in other contexts (e.g., low SES

neighborhoods). For example, attachment to one's neighborhood – a positive characteristic in high SES neighborhoods - can have detrimental effects on behaviors such as peer deviance when present in low SES neighborhoods (Moore et al., 2009).

Many of the ideas advanced here come out of rich literatures on traumatic stress and developmental psychopathology. Here, we extend some of this thinking to physical health and the psychobiological mechanisms that contribute to it. For example, there is a large literature in developmental psychopathology that has discussed resilience (Garmezy, 1985; Masten and Coatsworth, 1998; Masten, 2007; Rutter, 1987; Werner, 1995; Yehuda et al., 2006; Yehuda, 2004). Over the years, this resilience literature has identified key factors at the child (e.g., temperament, cognitive ability, emotion and behavior regulation, self-efficacy), family (e.g., warm, responsive caregiving, parent mental health), and neighborhood (e.g., effective schools, connections to adults and peers) levels that buffer children facing adversity from behavioral problems and academic failures (Garmezy, 1985; Luthar et al., 2000; Luthar et al., 2006; Luthar, 2006; Masten and Coatsworth, 1998; Masten and Obradovic, 2006; Masten, 2001; Rutter, 1987; Rutter, 1993; Werner and Smith, 1992). However, these studies have largely focused on psychological outcomes, such as social competence, a lack of behavioral problems, and academic outcomes (Luthar et al., 2006; Werner and Smith, 1992; Yehuda et al., 2006). Some factors that are clearly important for these domains (e.g., cognitive ability, effective schools) are more tangential to the development of chronic disease, and hence not the focus of this paper. Rather, we extend this work by focusing on individual-level constructs that have established links to stress reactivity, the longer-term pathogenic mechanisms it promotes, and actual disease outcomes. Of particular interest here are constructs like self-regulation (i.e., emotion regulation and coping (Eisenberg et al., 1997)). In order to impact disease outcomes, broader social factors must be registered at the level of the individual in ways that could alter HPA and SNS reactivity. Thus, although we recognize that there are a myriad of factors at the neighborhood and family level that affect children, the focus on this paper ultimately coalesces around an individual-level set of characteristics (admittedly shaped by neighborhood and family factors) that are applicable and proximal to the pathophysiology that drives chronic diseases of aging.

More recently, there has also been discussion about incorporating biology into resilience research in developmental psychopathology (Charney, 2004; Cicchetti and Curtis, 2006; Curtis and Cicchetti, 2003; Feder et al., 2009). Most of this work has focused on intermediary biological measures as endpoints (e.g., salivary cortisol (Cicchetti and Rogosch, 2007), or has sought to understand brain systems or genetic polymorphisms that moderate risk for psychopathology in vulnerable populations (Caspi et al., 2003; Caspi et al., 2005; Cicchetti and Curtis, 2006). Our paper extends this works by connecting resilience constructs with specific biological pathways whose functions are directly linked to the pathogenesis of chronic diseases of aging. In doing so we situate resilience more squarely within a biomedical disease framework.

In addition, there is a second source of resilience literature on adaptation to traumatic or life-threatening events (e.g., cancer, bereavement, terrorist attacks) (Bonanno, 2004; Bonanno, 2005; Dunkel Schetter and Dolbier, in press). This literature discusses the factors that promote recovery from traumatic events, and establishes a foundation for the role of finding meaning in response to life stressors (Bower et al., 1998; Taylor, 1983; Updegraff et al., 2008). However, much of this literature is focused around psychological adaptation, rather than the physical health consequences of adaptation to traumatic events. In addition, these types of stressors represent a single occurrence of a major life event, and while they can certainly evolve into long-lasting events, the framework for conceptualizing resilience remains centered around bouncing back from, finding meaning in, and recovering from an

event that has changed one's life and potentially one's view of the world. In contrast, we argue that low SES typically represents a much more chronic and pervasive stressor, in which a lifetime of adversity may lead to quite a different mindset from a lifetime of relative well-being, upon which a traumatic event is suddenly imposed. This notion is consistent with other researchers who have argued that resilience in the context of chronic, enduring stress may be different from resilience in the context of acute traumatic events (Dunkel Schetter and Dolbier, in press), and that resilience in terms of recovery (returning to baseline function after a major stressor) is distinct from sustainability (the ability to continue forward and maintain values in the face of adversity) (Zautra et al., 2008; Zautra et al., 2010). Thus while we draw on this literature on life-threatening and traumatic events, we focus our discussion around individual characteristics that would be beneficial to health in the context of a lifetime of chronic, enduring stress. As such, we did not discuss studies that either examined resilience characteristics (e.g., ego-resilient personality) unconnected to adversity (Block and Block, 1980; Hart et al., 2005; Simeon et al., 2007), or that investigated resilience characteristics in samples experimentally exposed to acute laboratory stressors, but without background adversity (Smeekens et al., 2007; Souza et al., 2007; Tugade and Fredrickson, 2004).

The Broader Social Context

Having described a set of strategies that help low SES individuals to remain physically healthy under adverse circumstances, we next turn to the question of where these traits arise from. In particular, what would lead some low SES individuals to be able to reappraise stressful situations and see meaning in adversity, given the persistent day-to-day difficulties they face?

To understand how shift-and-persist strategies develop, we move back to the broader social contexts that shape individuals. We propose that key to the development of shift-and-persist strategies in low SES individuals is the presence of role models (see Figure 1). In the midst of difficult life circumstances, role models allow low SES children to develop beliefs that others can be trustworthy and dependable, which in turn shape reappraisals of stressful situations. They also model adaptive emotional and behavioral responses to stressors, teaching effective emotion regulation to children. Finally, they help orient youth toward their futures, promoting optimism and meaning-making. By role model, we refer generally to any individual who provides a stable, regular presence and serves as an attachment figure for a child – e.g., parent, extended family member, teacher. Below, we discuss evidence that these types of supportive others buffer individuals from adversity, and then discuss why role models would be helpful in terms of shift-and-persist strategies. Because we are interested in how social contexts shape the development of beliefs and behaviors, we focus on research in children. We note that this literature has primarily focused on psychological outcomes, and hence we start with this before discussing implications for pathogenic mechanisms and disease.

The Benefits of Role Models

The single, most robust factor that protects children exposed to adverse life circumstances is nurturant parenting (Luthar, 2006). Children who are in a supportive family environment, despite facing poverty or other serious adversities, show better psychological adjustment and social competence, fewer behavioral problems, and lower levels of depression (Bradley et al., 1994; Klein and Forehand, 2000; Rutter, 1979; Wyman et al., 1999; Yates et al., 2003). Importantly, these effects also extend to individuals outside of the immediate family (Garmezy, 1987; Masten, 2004; Masten, 2007). In a birth cohort study of the predictors of resiliency into adulthood (Kauai Longitudinal study), one-third of infants who were considered at risk (because of being born into poverty or other adversity) developed into

adults free of behavioral or mental health problems through age 32. These resilient individuals all had at least one person in childhood with whom they developed a close bond and who accepted them unconditionally (Werner, 1993). Often, this was from a substitute caregiver, such as a grandparent or older sibling (Werner, 1995), as well as neighbors, teachers, or community elders (Werner, 1993; Werner, 1995). For example, all resilient adults from the Kauai study identified at least one teacher who had been a significant source of support for them (Werner, 1995).

The question that arises, however, is what the relevance of role models is for shift-and-persist strategies. We propose that role models work in at least two ways to promote shifting-and-persisting: 1) by facilitating secure attachment relationships that promote positive beliefs about others and the world (e.g., trust and optimism, which serve as a foundation for the development of a shift-and-persist approach); and 2) by modeling behaviors that teach children reappraisals and emotion regulation strategies (the “shift” component).

Attachment

Attachment refers to the bond that a child forms with another individual. Interactions with significant others who are available, sensitive, and responsive to one’s needs allow infants in their early years to develop a sense of attachment security (Bowlby, 1969). Attachment security in turn shapes social transactions throughout life and the development of secure adult attachment relationships later in life (Hazan and Shaver, 1987; Smith et al., 2004).

Because of our interest in how attachment relationships shape qualities relevant to shift-and-persist, we focus on how attachments foster: 1) world views, and 2) emotion regulation. Moreover, while the infant attachment literatures largely deals with parental (or early caregiver) attachment, we argue that shift-and-persist strategies can still develop out of positive attachment relationships with others, even if they are not the primary caregiver. For example, attachment to teachers buffers toddlers exposed to difficult life circumstances from behavioral problems (Howes and Ritchie, 1999), and attachment to one’s neighborhood buffers inner-city children from delinquency problems (Gorman-Smith and Tolan, 2003).

With respect to shift-and-persist strategies, attachment is relevant because it shapes underlying beliefs about trust in others. Secure attachments promote beliefs about the availability and dependability of others (Bowlby, 1988). Individuals who are securely attached anticipate positive reactions from others, believe in others’ good intentions, and maintain a sense that the world is a safe place (Allen et al., 1996; Bowlby, 1988; Hazan and Shaver, 1994; Mikulincer et al., 2003; Shaver and Mikulincer, 2002). In contrast, insensitive caregiving fosters beliefs in children about the world as unstable, unpredictable, and insecure (Minuchin, 1967; Yates et al., 2003). Children who were able to rise above adversity were more likely to hold beliefs that adults could be turned to for nurturance and support (Musick et al., 1987), and to have established trust with another person and to believe that life has meaning (Werner, 1989; Werner, 1993; Werner, 1995). Hence, to the extent that a child develops positive attachment relationships, that child will be more likely to develop beliefs about others as trustworthy and dependable as well as optimism that good things can happen in the world. These beliefs form the starting point for shift-and-persist strategies.

Second, attachment relationships help children learn how to regulate their own emotions. From young children through adolescents, securely attached individuals show better emotion regulation in terms of experiencing fewer trait negative emotions, engaging in more problem-solving strategies, and displaying greater control over one’s behaviors (Carlson and Sroufe, 1995; Gilliom et al., 2002; Kobak and Sceery, 1988; Kobak et al., 1993; Mikulincer

et al., 2003). In contrast, insensitive caregiving does not provide children with the foundation they need for regulatory development (Yates et al., 2003). Thus attachment to a role model is important for facilitating both the underlying beliefs as well as the emotion regulation abilities that form components of shift-and-persist strategies.

Socialization of behaviors

Role models also help in concrete ways by teaching children appropriate emotion regulation behaviors, and by helping to focus youth on their futures. For example, from a young age, parents (or other role models) help children learn how to understand their emotions, and enact appropriate response strategies to emotional experiences (Denham et al., 1995; Dunn and Brown, 1994). They do this by serving as coaches who help shape children's experiences with emotions and emotion regulation (Halberstadt, 1991). And in fact, parents who engaged in more coaching of their children's emotions had children with better emotion regulation years later (Gottman et al., 1996).

Second, role models teach children through their reactions to children's emotional expressions (Eisenberg et al., 1996). For example, parents who responded to children's anger with calm neutrality had children who then were less likely to be angry or respond negatively in future situations, even when their mother was absent (Denham, 1993). Third, when role models express their own emotions, they are modeling to children how emotions should be managed (Halberstadt, 1991). For example, mothers who were more affectively positive even during difficult times had children who showed better affect balance with peers (Denham et al., 1997). Overall, these findings suggest that role models, such as parents or other significant attachment figures, provide a scaffolding from which children learn adaptive emotion regulation strategies.

Finally, as children mature, role models help by keeping youth focused on their futures. For example, among a sample of inner-city teens, the most important factor (after receiving education and job opportunities) that they endorsed as helping them to achieve a more positive future was greater opportunity for adult-adolescent interactions and adult role models (Ginsburg et al., 2002). Role models serve as someone youth can aspire to be like and imitate, allowing them to see potential for their own future, and promoting positive academic outcomes and psychological well-being (Bryant and Zimmerman, 2003).

Taken together, the studies above suggest that role models serve as attachment figures that facilitate the development of shift-and-persist strategies by: 1) fostering trust in others and positive world views; 2) teaching reappraisal and emotion regulation strategies; and 3) encouraging youth to focus on their futures. The studies above, however, all focus on children's psychological well-being and development. To bring the discussion back to physical health, below we discuss preliminary evidence that the functions that role models serve can affect processes implicated in chronic diseases like CVD.

Role Model Characteristics and Disease-Relevant Processes

Research demonstrates that attachments and positive caregiving experiences early in life buffer against the physiological consequences of stress. This evidence comes from both human and animal work. For example, infants who are securely attached are less likely to show elevations in hormones such as cortisol during times of stress (Ahnert et al., 2004; Gunnar and Donzella, 2002; Gunnar and Quevedo, 2007; Gunnar et al., 1996; Hertzgaard et al., 1995; Nachmias et al., 1996). The animal equivalent – high levels of maternal licking and grooming in infancy – has similar effects (review by (Caldji et al., 2000). For example, the offspring of high licking and grooming mothers had smaller HPA responses to acute stressors as adults, compared to offspring of low licking and grooming mothers (Liu et al.,

1997). Moreover, the effects are due to behavioral experiences early in life, rather than genetics (Francis et al., 1999). For example, BALBc mice show 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).

In addition, there is evidence in the human literature that the qualities characteristic of role models can be beneficial physiologically for those facing adversity. For example, among children who have been maltreated, a program to teach parenting skills to foster parents produced greater declines in cortisol compared to maltreated children in foster care whose parents did not receive the program (Fisher et al., 2000). Similarly, the effects of cumulative adversity (e.g., poverty) on measures of allostatic load were mitigated among children who experienced high maternal warmth (Evans et al., 2007). Conversely, hostile maternal attitudes toward parenting are associated with more insulin resistance only among low SES, but not high SES, girls (Pulkki et al., 2003). Taken together, these findings suggest that the positive qualities of role models related to attachment and positive parenting may help disrupt the effects that low SES has on pathogenic mechanisms leading to chronic disease.

Moreover, these effects appear to last into adulthood. Among those who experienced adversity in childhood (e.g., parental loss), those with high quality family relationships in childhood displayed quicker recovery of cortisol, blood pressure, and heart rate to an acute stressor in adulthood (Luecken and Appelhans, 2006; Luecken et al., 2005; Luecken, 1998; Luecken, 2000). Longitudinal studies reveal that among low SES adults, high levels of emotional support from social relationships predict decreases in cardiovascular risk and inflammatory activity over an 18 month period. These relationships were specific to low SES individuals, and not evident among those high in SES (Vitaliano et al., 2001). Similarly, among older adults, greater negative interactions in social relationships were associated with a greater risk of heart disease only among low SES, but not high SES, individuals (Krause, 2005). These findings suggest that the benefits of supportive relationships may be more important for disease-relevant processes and outcomes among low SES individuals.

Work from our own group has investigated whether characteristics related to attachment might be able to buffer the effects of childhood adversity on long-term inflammatory profiles into adulthood. In an initial study using genome-wide microarray technology, we found that healthy adults who came from low early life SES backgrounds showed indications of increased activity of pro-inflammatory gene networks, combined with resistance to signaling that involved cortisol, compared to adults who came from high early life SES backgrounds (Miller et al., 2009a). Moreover, these relationships were independent of current SES, current health behaviors, and current perceived stress.

We subsequently showed that high levels of childhood maternal warmth could mitigate these effects. In a follow-up study of adults who were all low in early life SES, those who experienced high levels of maternal warmth in childhood displayed significant downregulation of genes with response elements for nuclear factor-kappa B (NF- κ B), the central transcription factor responsible for orchestrating inflammation, compared to those who were low SES and who experienced low childhood maternal warmth. In addition, the white blood cells of low early life SES participants who experienced high maternal warmth showed decreased IL-6 responses after in vitro microbial stimulation compared to those who were low in early life SES and low in maternal warmth (Chen et al., 2011).

Taken together, the above studies provide some intriguing suggestions that the types of role model characteristics posited to be important for shift-and-persist, that is, attachment and socialization of behavior through positive parenting practices, can diminish the effects of

childhood adversity on cardiovascular, neuroendocrine, and inflammatory profiles. Moreover, the impact of such support appears to persist into adulthood. However, we acknowledge that much of this evidence is preliminary, and hence we close by outlining some suggestions for future research to better understand role models, shift-and-persist strategies, and the pathogenic processes that contribute to chronic diseases of aging such as CVD.

Future Directions

Future research will need to consider the dynamic nature of SES. This paper has assumed that SES is quite stable over a lifetime. While this is largely true (McDonough et al., 2005), there will of course be examples of individuals who change in SES, and researchers will need to clarify whether changing social environments require different sets of psychosocial characteristics for optimal health. We also acknowledge that the relationship between individuals and their environment is not static. That is, while we have largely framed this paper around how the environmental circumstances of low SES life affect individuals, it is also true that individuals shape their environments, both by seeking out environments that fit with their personalities, and by creating patterns of interactions in their social world that serve to reinforce their beliefs about others (Caspi et al., 1989). Hence future research will need to incorporate more dynamic, interactive models of individuals together with their environments.

In this paper, we have focused on one specific kind of life adversity, low SES. We took this approach because we believe that the stressors associated with low SES are fairly unique in terms of their pervasiveness across domains, their chronicity, and the constraints they impose on life choices. Other types of adversities studied in the resilience literature tend to affect a single domain (e.g., illness), or are limited in time (e.g., natural disaster). However, there are clearly other sources of adversity (e.g., childhood maltreatment) that may produce similar effects to low SES, and which would be important to understand in their own right. In addition, one often mentioned overlapping characteristic with low SES is race/ethnicity. Members of minority groups also experience worse health than those in the majority group (National Academy of Sciences, 2002; Williams, 2002), and some of the stressors experienced by minority groups may have similar characteristics to those experienced by low SES individuals. That said, it will be important for future research to study the contributors to physical health resilience separately in different ethnic groups, as there may be different cultural factors (e.g., communalism, the role of extended family members) that play important protective roles in specific ethnic groups (Castro and Murray, 2010; Ruggles, 1994).

In addition, future studies will need to incorporate other contributors to physical health—for example, availability of health care, physical environment characteristics such as pollution, and genetic susceptibilities to disease, in order to develop more comprehensive models of the contributors to physical health resilience. Finally, with respect to measuring disease processes, future researchers will need to consider various conceptualizations of health (e.g., the absence of all diseases, or the absence of a specific disease such as CVD that is the target of inquiry), and will need to explore alternative biological pathways beyond those involved in stress reduction.

Finally, on the psychological end, future research will need to elaborate in greater detail the links between role models and shift-and-persist strategies. For example, longitudinal data are needed to establish whether the presence of a stable role model during childhood predicts over time the development of shift-and-persist strategies in low SES children. In addition, low SES children may face difficulties within their families, schools, and neighborhoods,

and it is unclear whether one positive role model can be sufficient to override the effects of adversity across all these domains, or whether role models are needed within specific domains in order to buffer the physiological effects of stressors within that life domain. Thus research about the critical characteristics of role models and how they shape shift-and-persist approaches to life are much needed and important next steps. In addition, our model makes assumptions about the frequency and value of shift-and-persist strategies amongst low SES individuals. Future research that can empirically document the prevalence of use of shift-and-persist strategies across those from different SES backgrounds, as well as the differences in prioritizing shift-and-persist strategies among those from different socioeconomic groups will be important in order to help more firmly ground the links in this model empirically.

Conclusions

In sum, we have focused here on the question of why it is that some individuals, despite facing adverse life circumstances (e.g., low SES), are nonetheless able to maintain, over the long-term, a low risk of chronic diseases. Our model proposes that early in life, in the midst of difficult family and neighborhood life circumstances, some children find positive role models, who teach them that the world has others that they can depend on and trust. This positive view of others allows these children to develop a “shift-and-persist” approach to life that balances shifting oneself by accepting and adapting to life’s day-to-day demands (e.g., by trying to reappraise stressors more benignly) with persisting in life through endurance and holding on to meaning and optimism. This shifting-and-persisting mitigates SNS and HPA responses to day-to-day stressors for those who are low in SES, and over time, forestalls the accumulation of pathogenic processes including obesity, insulin resistance, systemic inflammation, high blood pressure, endothelial dysfunction, and platelet activation, that eventually give rise to chronic diseases of aging, such as CVD.

Understanding the psychosocial qualities that can buffer low SES individuals from chronic disease risk is important for providing a fresh perspective toward reducing health disparities. Despite the increasing attention that has been focused on tackling health disparities in recent years, eliminating them has remained an intractable problem in our society. It is often argued that to reduce health disparities, policies need to address inequalities in resources, as well as structural impediments to social mobility. We agree that such changes are needed. However, in the current economic climate with limited resources, an approach like ours that seeks to understand resilience among low SES individuals is an important one, because it has the potential to identify targets for intervention that could complement simultaneous efforts to change economic inequities in our society. Moreover, if we can identify characteristics that some low SES families naturally possess that promote positive disease trajectories, these should be ones that in theory would be possible to alter through intervention in other low SES families. The long-term goal would be to make a significant dent in reducing health disparities by understanding the psychosocial processes that would be adaptive to foster among those who are forced to confront the difficulties of a life in poverty.

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References

- Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, et al. Socioeconomic status and health: The challenge of the gradient. *American Psychologist*. 1994; 49:15–24. [PubMed: 8122813]

- Adler NE, Boyce WT, Chesney MA, Folkman S, Syme SL. Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association*. 1993; 269:3140–3145. [PubMed: 8505817]
- Affleck G, Tennen H, Croog S, Levine S. Causal attribution, perceived benefits, and morbidity after a heart attack: an 8-year study. *J Consult Clin Psychol*. 1987; 55(1):29–35. [PubMed: 3571655]
- Ahnert L, Gunnar MR, Lamb ME, Barthel M. Transition to child care: associations with infant-mother attachment, infant negative emotion, and cortisol elevations. *Child Dev*. 2004; 75(3):639–650. [PubMed: 15144478]
- Allen JP, Hauser ST, Borman-Spurrell E. Attachment theory as a framework for understanding sequelae of severe adolescent psychopathology: an 11-year follow-up study. *J Consult Clin Psychol*. 1996; 64(2):254–263. [PubMed: 8871409]
- Anderson NB, Armstead CA. Toward understanding the association of socioeconomic status and health: A new challenge for the biopsychosocial approach. *Psychosomatic Medicine*. 1995; 57:213–225. [PubMed: 7652122]
- Appelhans BM, Luecken LJ. Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*. 2006; 10:229–240.
- Aspinwall LG, Richter L. Optimism and self-mastery predict more rapid disengagement from unsolvable tasks in the presence of alternatives. *Motivation and Emotion*. 1999; 23:221–245.
- Aspinwall LG, Taylor SE. A stitch in time: self-regulation and proactive coping. *Psychol Bull*. 1997; 121(3):417–436. [PubMed: 9136643]
- Attar BK, Guerra NG, Tolan PH. Neighborhood disadvantage, stressful life events, and adjustment in urban elementary-school children. *Journal of Clinical Child Psychology*. 1994; 23:391–400.
- Barefoot JC, Peterson BL, Dahlstrom WG, Siegler IC, Anderson NB, Williams RB. Hostility patterns and health implications: Correlates of Cook-Medley Hostility scale scores in a national survey. *Health Psychology*. 1991; 10:18–24. [PubMed: 2026126]
- Block, JH.; Block, J. *The Minnesota Symposia on Child Psychology*. Vol. 13. Hillsdale, NJ: Erlbaum; 1980. The role of ego-control and ego-resiliency in the origination of behavior. In W. A. Collings (Ed.); p. 39-101.
- Bonanno GA. Loss, trauma, and human resilience: have we underestimated the human capacity to thrive after extremely aversive events? *Am Psychol*. 2004; 59(1):20–28. [PubMed: 14736317]
- Bonanno GA. Resilience in the face of potential trauma. *Current Directions in Psychological Science*. 2005; 14(3):135–138.
- Bonanno GA, Galea S, Bucciarelli A, Vlahov D. Psychological resilience after disaster: New York City in the aftermath of the September 11th terrorist attack. *Psychol Sci*. 2006; 17(3):181–186. [PubMed: 16507055]
- Bower J, Low CA, Moskowitz JT, Sepah S, Epel E. Benefit finding and physical health: Positive psychological changes and enhanced allostasis. *Social and Personality Psychology Compass*. 2008; 2:223–244.
- Bower J, Moskowitz JT, Epel E. Is benefit finding good for your health? Pathways linking positive life changes after stress and physical health outcomes. *Current Directions in Psychological Science*. 2009; 18:337–341.
- Bower JE, Kemeny ME, Taylor SE, Fahey JL. Cognitive processing, discovery of meaning, CD4 decline, and AIDS-related mortality among bereaved HIV-seropositive men. *Journal of Consulting and Clinical Psychology*. 1998; 66(6):979–986. [PubMed: 9874911]
- Bowlby, J. *Attachment*. Vol. 1. New York, NY: Basic Books; 1969. Attachment and loss.
- Bowlby, J. *A secure base: Clinical applications of attachment theory*. London, England: Routledge; 1988.
- Boyle PA, Barnes LL, Buchman AS, Bennett DA. Purpose in life is associated with mortality among community-dwelling older persons. *Psychosomatic Medicine*. 2009; 71(5):574–579. [PubMed: 19414613]
- Bradley RH, Whiteside L, Mundfrom DJ, Casey PH, Kelleher KJ, Pope SK. Early indications of resilience and their relation to experiences in the home environments of low birthweight, premature children living in poverty. *Child Dev*. 1994; 65(2 Spec):346–360. [PubMed: 8013226]

- Bradley RH, Corwyn RF, Mcadoo HP, Coll CG. The home environments of children in the United States part I: Variations by age, ethnicity, and poverty status. *Child Development*. 2001; 72(6): 1844–1867. [PubMed: 11768149]
- Brady SS, Matthews KA. The effect of socioeconomic status and ethnicity on adolescents' exposure to stressful life events. *Journal of Pediatric Psychology*. 2002; 27:575–583. [PubMed: 12228329]
- Brandtstadter J, Renner G. Tenacious goal pursuit and flexible goal adjustment: explication and age-related analysis of assimilative and accommodative strategies of coping. *Psychol Aging*. 1990; 5(1):58–67. [PubMed: 2317302]
- Brandtstadter J, Rothermund K. The life-course dynamics of goal pursuit and goal adjustment: A two-process framework. *Developmental Review*. 2002; 22:117–150.
- Braveman PA, Cubbin C, Egerter S, Williams DR, Pamuk E. Socioeconomic disparities in health in the United States: What the patterns tell us. *American Journal of Public Health*. 2010; 100:S186–S196. [PubMed: 20147693]
- Bronfenbrenner, U. *The ecology of human development*. Cambridge, MA: Harvard University Press; 1979.
- Brotman DJ, Golden SH, Wittstein IS. The cardiovascular toll of stress. *Lancet*. 2007; 370(9592): 1089–1100. [PubMed: 17822755]
- Bryant AL, Zimmerman MA. Role models and psychosocial outcomes among African American adolescents. *Journal of Adolescent Research*. 2003; 18:36–67.
- Buckner JC, Mezzacappa E, Beardslee WR. Characteristics of resilient youths living in poverty: the role of self-regulatory processes. *Dev Psychopathol*. 2003; 15(1):139–162. [PubMed: 12848439]
- Buka SL, Stichick TL, Birdthistle I, Earls FJ. Youth exposure to violence: prevalence, risks, and consequences. *Am J Orthopsychiatry*. 2001; 71(3):298–310. [PubMed: 11495332]
- Bulman RJ, Wortman CB. Attributions of blame and coping in the “real world”: severe accident victims react to their lot. *J Pers Soc Psychol*. 1977; 35(5):351–363. [PubMed: 874739]
- Caldji C, Diorio J, Meaney MJ. Variations in maternal care in infancy regulate the development of stress reactivity. *Biol Psychiatry*. 2000; 48(12):1164–1174. [PubMed: 11137058]
- Calkins SD, Blandon AY, Williford AP, Keane SP. Biological, behavioral, and relational levels of resilience in the context of risk for early childhood behavior problems. *Dev Psychopathol*. 2007; 19(3):675–700. [PubMed: 17705898]
- Camerino C. Low sympathetic tone and obese phenotype in oxytocin-deficient mice. *Obesity (Silver Spring)*. 2009; 17(5):980–984. [PubMed: 19247273]
- Cannon, WB. *The wisdom of the body*. New York, NY: Norton; 1932.
- Carlson, EA.; Sroufe, LA. Contribution of attachment theory to developmental psychology. In: Cicchetti, D.; Cohen, D., editors. *Developmental psychopathology, Vol. 1: Theory and methods*. New York, NY: Wiley; 1995. p. 581-617.
- Carver CS, Pozo C, Harris SD, Noriega V, Scheier MF, Robinson DS, et al. How coping mediates the effect of optimism on distress: a study of women with early stage breast cancer. *J Pers Soc Psychol*. 1993; 65(2):375–390. [PubMed: 8366426]
- Caspi A, Moffitt TE, Cannon M, McClay J, Murray R, Harrington H, et al. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene × environment interaction. *Biol Psychiatry*. 2005; 57(10):1117–1127. [PubMed: 15866551]
- Caspi A, Bem DJ, Elder GH. Continuities and consequences of interactional styles across the lifecourse. *Journal of Personality*. 1989; 57:375–406. [PubMed: 2769561]
- Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, et al. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*. 2003; 301:386–389. [PubMed: 12869766]
- Castro, F.; Murray, KE. Cultural adaptation and resilience: Controversies, issues, and emerging models. In: Reich, JW.; Zautra, AJ.; Hall, J., editors. *Handbook of Adult Resilience*. New York, NY: Guilford Press; 2010. p. 375-403.
- Charney DS. Psychobiological mechanisms of resilience and vulnerability: implications for successful adaptation to extreme stress. *American Journal of Psychiatry*. 2004; 161(2):195–216. [PubMed: 14754765]

- Chen E, Miller GE, Kobor MS, Cole SW. Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Mol Psychiatry*. 2011; 16(7): 729–737. [PubMed: 20479762]
- Chen, E.; Miller, GE.; Lachman, ME.; Gruenewald, TL.; Seeman, TE. Protective factors for adults from low childhood socioeconomic circumstances: The benefits of shift-and-persist for allostatic load. (under submission)
- Chen E, Strunk RC, Trethewey A, Schreier HMC, Maharaj N, Miller GE. Resilience in low socioeconomic status children with asthma: Adaptations to stress. *Journal of Allergy and Clinical Immunology*. (in press).
- Chen E. Impact of socioeconomic status on physiological health in adolescents: An experimental manipulation of psychosocial factors. *Psychosomatic Medicine*. 2007; 69(4):348–355. [PubMed: 17510293]
- Chen E, Matthews KA, Boyce WT. Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin*. 2002; 128:295–329. [PubMed: 11931521]
- Chen E, Matthews KA, Zhou F. Interpretations of ambiguous social situations and cardiovascular responses in adolescents. *Annals of Behavioral Medicine*. 2007; 34:26–36. [PubMed: 17688394]
- Cheng C. Assessing coping flexibility in real-life and laboratory settings: a multimethod approach. *J Pers Soc Psychol*. 2001; 80(5):814–833. [PubMed: 11374752]
- Chou, M.; Thampi, K.; Wight, VR. Basic Facts about Low-Income Children, 2009. New York, NY: National Center for Children in Poverty, Mailman School of Public Health, Columbia University; 2010.
- Chung HY, Cesari M, Anton S, Marzetti E, Giovannini S, Seo AY, et al. Molecular inflammation: underpinnings of aging and age-related diseases. *Ageing Res Rev*. 2009; 8(1):18–30. [PubMed: 18692159]
- Cicchetti D, Rogosch FA. Personality, adrenal steroid hormones, and resilience in maltreated children: a multilevel perspective. *Dev Psychopathol*. 2007; 19(3):787–809. [PubMed: 17705903]
- Cicchetti, D.; Curtis, WJ. The developing brain and neural plasticity: Implications for normality, psychopathology, and resilience. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental Psychopathology*. 2. Vol. 2. Hoboken, NJ: John Wiley & Sons; 2006. p. 1-64. *Developmental Neuroscience*
- Clodi M, Vila G, Geyeregger R, Riedl M, Stulnig TM, Struck J, et al. Oxytocin alleviates the neuroendocrine and cytokine response to bacterial endotoxin in healthy men. *Am J Physiol Endocrinol Metab*. 2008; 295(3):E686–91. [PubMed: 18593851]
- Cohen S, Doyle WJ, Turner RB, Alper CM, Skoner DP. Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine*. 2004; 66(4):553–558. [PubMed: 15272102]
- Conger RD, Donnellan MB. An interactionist perspective on the socioeconomic context of human development. *Annual Review of Psychology*. 2007; 58:175–199.
- Conger, RD.; Elder, GH. *Families in troubled times*. New York, NY: Aldine de Gruyter; 1994.
- Curtis WJ, Cicchetti D. Moving research on resilience into the 21st century: theoretical and methodological considerations in examining the biological contributors to resilience. *Dev Psychopathol*. 2003; 15(3):773–810. [PubMed: 14582940]
- Denham SA. Maternal emotional responsiveness and toddlers' social-emotional competence. *J Child Psychol Psychiatry*. 1993; 34(5):715–728. [PubMed: 8340440]
- Denham SA, Mason T, Couchoud EA. Scaffolding young children's prosocial responsiveness: preschoolers' responses to adult sadness, anger, and pain. *International Journal of Behavior Development*. 1995; 18:489–504.
- Denham SA, Mitchell-Copeland J, Strandberg K, Auerbach S, Blair K. Parental contributions to preschoolers' emotional competence: Direct and indirect effects. *Motivation and Emotion*. 1997; 21:65–86.
- Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*. 2004; 130(3):355–391. [PubMed: 15122924]

- Dienstbier RA. Arousal and physiological toughness: Implications for mental and physical health. *Psychol Rev.* 1989; 96(1):84–100. [PubMed: 2538855]
- Ditzen B, Schaer M, Gabriel B, Bodenmann G, Ehlert U, Heinrichs M. Intranasal oxytocin increases positive communication and reduces cortisol levels during couple conflict. *Biol Psychiatry.* 2009; 65(9):728–731. [PubMed: 19027101]
- Dodge KA, Pettit GS, Bates JE. Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development.* 1994; 65:649–665. [PubMed: 8013245]
- Dunkel Schetter C, Dolbier C. Resilience in the context of chronic stress and health in adults. *Social and Personality Psychology Compass.* (in press).
- Dunn J, Brown J. Affect expression in the family, children's understanding of emotions, and their interactions with others. *Merrill-Palmer Quarterly.* 1994; 40:120–137.
- Eisenberg N, Fabes RA, Murphy BC. Parents' reactions to children's negative emotions: relations to children's social competence and comforting behavior. *Child Dev.* 1996; 67(5):2227–2247. [PubMed: 9022240]
- Eisenberg, N.; Fabes, RA.; Guthrie, IK. Coping with stress: The roles of regulation and development. In: Wolchik, SA.; Sandler, IN., editors. *Handbook of Children's Coping: Linking Theory and Intervention.* New York, NY: Plenum; 1997. p. 41-70.
- El Sheikh M, Harger J. Appraisals of marital conflict and children's adjustment, health, and physiological reactivity. *Developmental Psychology.* 2001; 37(6):875–885. [PubMed: 11699760]
- Evans GW. The environment of childhood poverty. *American Psychologist.* 2004; 59(2):77–92. [PubMed: 14992634]
- Evans GW, Gonnella C, Marcynyszyn LA, Gentile L, Salpekar N. The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science.* 2005; 16(7):560–565. [PubMed: 16008790]
- Evans GW, Kim P, Ting AH, Teshler HB, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology.* 2007; 43(2):341–351. [PubMed: 17352543]
- Evers AW, Kraaimaat FW, van Lankveld W, Jongen PJ, Jacobs JW, Bijlsma JW. Beyond unfavorable thinking: the illness cognition questionnaire for chronic diseases. *J Consult Clin Psychol.* 2001; 69(6):1026–1036. [PubMed: 11777106]
- Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. *Annu Rev Public Health.* 2005; 26:469–500. [PubMed: 15760298]
- Feder A, Nestler EJ, Charney DS. Psychobiology and molecular genetics of resilience. *Nature Reviews Neuroscience.* 2009; 10(6):446–457.
- Fine SE, Izard CE, Mostow AJ, Trentacosta CJ, Ackerman BP. First grade emotion knowledge as a predictor of fifth grade self-reported internalizing behaviors in children from economically disadvantaged families. *Dev Psychopathol.* 2003; 15(2):331–342. [PubMed: 12931831]
- Fisher PA, Gunnar MR, Chamberlain P, Reid JB. Preventive intervention for maltreated preschool children: impact on children's behavior, neuroendocrine activity, and foster parent functioning. *J Am Acad Child Adolesc Psychiatry.* 2000; 39(11):1356–1364. [PubMed: 11068890]
- Fitzpatrick KM, Boldizar JP. The prevalence and consequences of exposure to violence among African-American youth. *J Am Acad Child Adolesc Psychiatry.* 1993; 32(2):424–430. [PubMed: 8444774]
- Folkman S, Moskowitz JT. Positive affect and the other side of coping. *Am Psychol.* 2000; 55(6):647–654. [PubMed: 10892207]
- Folkman S, Moskowitz JT. Coping: pitfalls and promise. *Annual Review of Psychology.* 2004; 55:745–774.
- Francis D, Diorio J, Liu D, Meaney MJ. Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science.* 1999; 286:1155–1158. [PubMed: 10550053]
- Frankl, V. *Man's search for meaning.* New York, NY: Washington Square Press; 1963.
- Fredrickson BL. The role of positive emotions in positive psychology. The broaden-and-build theory of positive emotions. *Am Psychol.* 2001; 56(3):218–226. [PubMed: 11315248]

- Friedman EM, Hayney M, Love GD, Singer BH, Ryff CD. Plasma interleukin-6 and soluble IL-6 receptors are associated with psychological well-being in aging women. *Health Psychology*. 2007; 26(3):305–313. [PubMed: 17500617]
- Gallo LC, Matthews KA. Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*. 2003; 129(1):10–51. [PubMed: 12555793]
- Galobardes B, Lynch JW, Smith GD. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. *J Epidemiol Community Health*. 2008; 62(5):387–390. [PubMed: 18413449]
- Galobardes B, Lynch JW, Smith GD. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiologic Reviews*. 2004; 26:7–21. [PubMed: 15234944]
- Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Annals of Epidemiology*. 2006; 16(2):91–104. [PubMed: 16257232]
- Garnezy N. Stress, competence, and development: continuities in the study of schizophrenic adults, children vulnerable to psychopathology, and the search for stress-resistant children. *Am J Orthopsychiatry*. 1987; 57(2):159–174. [PubMed: 3296774]
- Garnezy, N. Stress-resistant children: The search for protective factors. In: Stevenson, JE., editor. *Recent Research in Developmental Psychopathology*. Oxford: Pergamon; 1985. p. 213-233.
- Gilliom M, Shaw DS, Beck JE, Schonberg MA, Lukon JL. Anger regulation in disadvantaged preschool boys: strategies, antecedents, and the development of self-control. *Dev Psychol*. 2002; 38(2):222–235. [PubMed: 11881758]
- Giltay EJ, Geleijnse JM, Zitman FG, Hoekstra T, Schouten EG. Dispositional optimism and all-cause and cardiovascular mortality in a prospective cohort of elderly dutch men and women. *Arch Gen Psychiatry*. 2004; 61(11):1126–1135. [PubMed: 15520360]
- Ginsburg KR, Alexander PM, Hunt J, Sullivan M, Zhao H, Cnaan A. Enhancing their likelihood for a positive future: the perspective of inner-city youth. *Pediatrics*. 2002; 109(6):1136–1142. [PubMed: 12042555]
- Gordon-Larsen P, Nelson MC, Page P, Popkin BM. Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics*. 2006; 117(2):417–424. [PubMed: 16452361]
- Gorman-Smith, D.; Tolan, PH. Positive adaptation among youth exposed to community violence. In: Luthar, SS., editor. *Resilience and vulnerability: Adaptation in the context of childhood adversities*. New York, NY: Cambridge University Press; 2003. p. 392-413.
- Gottman JM, Katz LF, Hooven C. Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology*. 1996; 10:243–268.
- Grewen K, Girdler SS, West SG, Bragdon E, Costello N, Light KC. Stable pessimistic attributions interact with socioeconomic status to influence blood pressure and vulnerability to hypertension. *J Womens Health Gend Based Med*. 2000; 9(8):905–915. [PubMed: 11074957]
- Grewen KM, Girdler SS, Amico J, Light KC. Effects of partner support on resting oxytocin, cortisol, norepinephrine, and blood pressure before and after warm partner contact. *Psychosomatic Medicine*. 2005; 67(4):531–538. [PubMed: 16046364]
- Gross JJ. Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*. 2001; 10:214–219.
- Gross JJ. Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J Pers Soc Psychol*. 1998; 74(1):224–237. [PubMed: 9457784]
- Gunnar M, Quevedo K. The neurobiology of stress and development. *Annual Review of Psychology*. 2007; 58:145–173.
- Gunnar MR, Brodersen L, Nachmias M, Buss K, Rigatuso J. Stress reactivity and attachment security. *Dev Psychobiol*. 1996; 29(3):191–204. [PubMed: 8666128]

- Gunnar MR, Donzella B. Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*. 2002; 27(1–2):199–220. [PubMed: 11750779]
- Gutkowska J, Jankowski M. Oxytocin revisited: It is also a cardiovascular hormone. *J Am Soc Hypertens*. 2008; 2(5):318–325. [PubMed: 20409913]
- Halberstadt, AG. Socialization of expressiveness: Family influences in particular and a model in general. In: Feldman, RS.; Rime, R., editors. *Fundamentals of emotional expressiveness*. Cambridge, England: Cambridge University Press; 1991. p. 106-162.
- Hanson MD, Chen E. Socioeconomic status and health behaviors in adolescence: a review of the literature. *Journal of Behavioral Medicine*. 2007; 30(3):263–285. [PubMed: 17514418]
- Hart D, Burock D, London B, Atkins R, Bonilla-Santiago G. The relation of personality types to physiological, behavioural, and cognitive processes. *European Journal of Personality*. 2005; 19:391–407.
- Hashimoto H, Kitagawa K, Hougaku H, Shimizu Y, Sakaguchi M, Nagai Y, et al. C-reactive protein is an independent predictor of the rate of increase in early carotid atherosclerosis. *Circulation*. 2001; 104(1):63–67. [PubMed: 11435339]
- Hazan C, Shaver P. Romantic love conceptualized as an attachment process. *J Pers Soc Psychol*. 1987; 52(3):511–524. [PubMed: 3572722]
- Hazan C, Shaver PR. Attachment as an organizational framework for research on close relationships. *Psychological Inquiry*. 1994; 5:1–22.
- Heckhausen J, Schulz R. A life-span theory of control. *Psychol Rev*. 1995; 102(2):284–304. [PubMed: 7740091]
- Heckhausen J, Wrosch C, Schulz R. A motivational theory of life-span development. *Psychol Rev*. 2010; 117(1):32–60. [PubMed: 20063963]
- Heinrichs M, Baumgartner T, Kirschbaum C, Ehler U. Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biol Psychiatry*. 2003; 54(12):1389–1398. [PubMed: 14675803]
- Heinrichs M, Gaab J. Neuroendocrine mechanisms of stress and social interaction: implications for mental disorders. *Curr Opin Psychiatry*. 2007; 20(2):158–162. [PubMed: 17278915]
- Helgeson VS, Reynolds KA, Tomich PL. A meta-analytic review of benefit finding and growth. *J Consult Clin Psychol*. 2006; 74(5):797–816. [PubMed: 17032085]
- Hertsgaard L, Gunnar M, Erickson MF, Nachmias M. Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Dev*. 1995; 66(4):1100–1106. [PubMed: 7671652]
- Hobfoll SE. Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist*. 1989; 44(3):513–524. [PubMed: 2648906]
- Hobfoll SE. The influence of culture, community, and the nested-self in the stress process: Advancing Conservation of Resources Theory. *Applied Psychology: An International Review*. 2001; 50(3):337–370.
- Howes C, Ritchie S. Attachment organizations in children with difficult life circumstances. *Dev Psychopathol*. 1999; 11(2):251–268. [PubMed: 16506533]
- Jacobsen BK, Thelle DS. Risk factors for coronary heart disease and level of education. The Tromso Heart Study. *American Journal of Epidemiology*. 1988; 127(5):923–932. [PubMed: 3258732]
- James SA, Hartnett SA, Kalsbeek WD. John Henryism and blood pressure differences among black men. *Journal of Behavioral Medicine*. 1983; 6(3):259–278. [PubMed: 6663614]
- James SA, Keenan NL, Strogatz DS, Browning SR, Garrett JM. Socioeconomic status, John Henryism, and blood pressure in black adults. The Pitt County Study. *American Journal of Epidemiology*. 1992; 135(1):59–67. [PubMed: 1736661]
- James SA, Strogatz DS, Wing SB, Ramsey DL. Socioeconomic status, John Henryism, and hypertension in blacks and whites. *American Journal of Epidemiology*. 1987; 126(4):664–673. [PubMed: 3631056]
- Janoff-Bulman R. Assumptive worlds and the stress of traumatic events: Applications of the schema construct. *Social Cognition*. 1989; 7:113–136.

- Jensen EW, James SA, Boyce WT, Hartnett SA. The Family Routines Inventory: Development and validation. *Social Science & Medicine*. 1983; 17(4):201–211. [PubMed: 6844952]
- Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: A review of the literature. *Circulation*. 1993; 88:1973–1998. [PubMed: 8403348]
- Kemeny ME. The psychobiology of stress. *Current Directions in Psychological Science*. 2003; 12:124–129.
- Keough KA, Zimbardo PG, Boyd JN. Who's smoking, drinking, and using drugs? Time perspective as a predictor of substance use. *Journal of Basic and Applied Social Psychology*. 1999; 21:149–164.
- Kiecolt-Glaser JK, McGuire L, Robles TF, Glaser R. Emotions, morbidity, and mortality: new perspectives from psychoneuroimmunology. *Annual Review of Psychology*. 2002; 53:83–107.
- Kinnunen ML, Kokkonen M, Kaprio J, Pulkkinen L. The associations of emotion regulation and dysregulation with the metabolic syndrome factor. *J Psychosom Res*. 2005; 58(6):513–521. [PubMed: 16125518]
- Klein K, Forehand R. Family processes as resources for African American children exposed to a constellation of sociodemographic risk factors. Family Health Project Group. *J Clin Child Psychol*. 2000; 29(1):53–65. [PubMed: 10693032]
- Kobak RR, Cole HE, Ferenz-Gillies R, Fleming WS, Gamble W. Attachment and emotion regulation during mother-teen problem solving: a control theory analysis. *Child Dev*. 1993; 64(1):231–245. [PubMed: 8436031]
- Kobak RR, Sceery A. Attachment in late adolescence: working models, affect regulation, and representations of self and others. *Child Dev*. 1988; 59(1):135–146. [PubMed: 3342708]
- Krantz DS, Manuck SB. Acute psychophysiological reactivity and risk of cardiovascular disease: A review and methodological critique. *Psychological Bulletin*. 1984; 96:435–464. [PubMed: 6393178]
- Krantz DS, McCeney MK. Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology*. 2002; 53:341–369.
- Kraus MW, Piff PK, Keltner D. Social class, sense of control, and social explanation. *J Pers Soc Psychol*. 2009; 97(6):992–1004. [PubMed: 19968415]
- Krause N. Negative interaction and heart disease in late life: exploring variations by socioeconomic status. *J Aging Health*. 2005; 17(1):28–55. [PubMed: 15601782]
- Kubzansky LD, Sparrow D, Vokonas P, Kawachi I. Is the glass half empty or half full? A prospective study of optimism and coronary heart disease in the normative aging study. *Psychosomatic Medicine*. 2001; 63(6):910–916. [PubMed: 11719629]
- Kubzansky LD, Berkman LF, Glass TA, Seeman TE. Is educational attainment associated with shared determinants of health in the elderly? Findings from the MacArthur Studies of Successful Aging. *Psychosomatic Medicine*. 1998; 60:578–585. [PubMed: 9773761]
- Lachman ME, Weaver SL. The sense of control as a moderator of social class differences in health and well-being. *Journal of Personality and Social Psychology*. 1998; 74:763–773. [PubMed: 9523418]
- Lazarus, RS., Folkman, S. *Stress, appraisal, and coping*. New York: Springer Publishing Company; 1984.
- Leventhal T, Brooks-Gunn J. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*. 2000; 126(2):309–337. [PubMed: 10748645]
- Light KC, Grewen KM, Amico JA. More frequent partner hugs and higher oxytocin levels are linked to lower blood pressure and heart rate in premenopausal women. *Biol Psychol*. 2005; 69(1):5–21. [PubMed: 15740822]
- Linden W, Earle TL, Gerin W, Christenfeld N. Physiological stress reactivity and recovery: conceptual siblings separated at birth? *J Psychosom Res*. 1997; 42(2):117–135. [PubMed: 9076640]
- Linden W, Gerin W, Davidson K. Cardiovascular reactivity: status quo and a research agenda for the new millennium. *Psychosomatic Medicine*. 2003; 65(1):5–8. [PubMed: 12554811]
- Lindfors P, Lundberg U. Is low cortisol release an indicator of positive health? *Stress and Health*. 2002; 18:153–160.

- Liu D, Diorio J, Tannenbaum B, Caldji C, Francis D, Freedman A, et al. Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science*. 1997; 277(5332):1659–1662. [PubMed: 9287218]
- Lowry R, Kann L, Collins JL, Kolbe LJ. The effect of socioeconomic status on chronic disease risk behaviors among us adolescents. *JAMA*. 1996; 276(10):792–796. [PubMed: 8769588]
- Luecken LJ. Childhood attachment and loss experiences affect adult cardiovascular and cortisol function. *Psychosomatic Medicine*. 1998; 60(6):765–772. [PubMed: 9847038]
- Luecken LJ. Parental caring and loss during childhood and adult cortisol responses to stress. *Psychology and Health*. 2000; 15:841–851.
- Luecken LJ, Appelhans BM. Early parental loss and salivary cortisol in young adulthood: the moderating role of family environment. *Dev Psychopathol*. 2006; 18(1):295–308. [PubMed: 16478564]
- Luecken LJ, Rodriguez AP, Appelhans BM. Cardiovascular stress responses in young adulthood associated with family-of-origin relationship experiences. *Psychosomatic Medicine*. 2005; 67(4): 514–521. [PubMed: 16046362]
- Luthar, SS. Resilience in development: A synthesis of research across five decades. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental psychopathology*. 2. Vol. 3. New York, NY: John Wiley & Sons; 2006. p. 739-795. Risk, disorder, and adaptation
- Luthar SS, Cicchetti D, Becker B. The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*. 2000; 71(3):543–562. [PubMed: 10953923]
- Luthar SS, Sawyer JA, Brown PJ. Conceptual issues in studies of resilience: Past, present, and future research. *Annals of the New York Academy of Sciences*. 2006; 1094:105–115. [PubMed: 17347344]
- Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviors and psychosocial characteristics by stages of the socioeconomic lifecourse. *Social Science and Medicine*. 1997; 44:809–819. [PubMed: 9080564]
- Maier KJ, Waldstein SR, Synowski SJ. Relation of cognitive appraisal to cardiovascular reactivity, affect, and task engagement. *Annals of Behavioral Medicine*. 2003; 26(1):32–41. [PubMed: 12867352]
- Maier SF, Seligman MEP. Learned helplessness: Theory and evidence. *Journal of Experimental Psychology: General*. 1976; 105:3–46.
- Markus, HR.; Ryff, CD.; Curhan, KB.; Palmersheim, K. In their own words: Well-being at midlife among high school and college educated adults. In: Brim, OG.; Ryff, CD.; Kessler, RC., editors. *How healthy are we? A national study of well-being at midlife*. Chicago, IL: University of Chicago Press; 2004. p. 273-319.
- Maruta T, Colligan RC, Malinchoc M, Offord KP. Optimists vs pessimists: survival rate among medical patients over a 30-year period. *Mayo Clinic Proceedings*. 2000; 75(2):140–143. [PubMed: 10683651]
- Masten AS. Ordinary magic: Resilience processes in development. *American Psychologist*. 2001; 56:227–238. [PubMed: 11315249]
- Masten AS. Regulatory processes, risk, and resilience in adolescent development. *Annals of the New York Academy of Sciences*. 2004; 1021:310–319. [PubMed: 15251901]
- Masten AS. Resilience in developing systems: Progress and promise as the fourth wave rises. *Development and Psychopathology*. 2007; 19(3):921–930. [PubMed: 17705908]
- Masten AS, Coatsworth JD. The development of competence in favorable and unfavorable environments. *American Psychologist*. 1998; 53:205–220. [PubMed: 9491748]
- Masten AS, Obradovic J. Competence and resilience in development. *Annals of the New York Academy of Sciences*. 2006; 1094:13–27. [PubMed: 17347338]
- Matheny AP, Wachs TD, Ludwig JL, Phillips K. Bringing order out of chaos: Psychometric characteristics of the Confusion, Hubbub, and Order Scale. *Journal of Applied Developmental Psychology*. 1995; 16(3):429–444.
- Matthews KA, Gallo LC, Taylor SE. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. *Annals of the New York Academy of Sciences*. 2010; 1186:146–173. [PubMed: 20201872]

- Matthews KA, Raikkonen K, Sutton-Tyrrell K, Kuller LH. Optimistic attitudes protect against progression of carotid atherosclerosis in healthy middle-aged women. *Psychosomatic Medicine*. 2004; 66(5):640–644. [PubMed: 15385685]
- Mauss IB, Cook CL, Cheng JY, Gross JJ. Individual differences in cognitive reappraisal: experiential and physiological responses to an anger provocation. *Int J Psychophysiol*. 2007; 66(2):116–124. [PubMed: 17543404]
- McDonough P, Sacker A, Wiggins RD. Time on my side? Life course trajectories of poverty and health. *Social Science & Medicine*. 2005; 61(8):1795–1808. [PubMed: 16029777]
- McEwen BS. Protective and damaging effects of stress mediators. *New England Journal of Medicine*. 1998; 338(3):171–179. [PubMed: 9428819]
- McLoyd VC, Jayaratne TE, Ceballos R, Borquez J. Unemployment and work interruption among African American single mothers: effects on parenting and adolescent socioemotional functioning. *Child Dev*. 1994; 65(2 Spec):562–589. [PubMed: 8013240]
- McLoyd VC. The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. *Child Development*. 1990; 61:311–346. [PubMed: 2188806]
- Mendes de Leon CF. Anger and impatience/irritability in patients of low socioeconomic status with acute coronary heart disease. *Journal of Behavioral Medicine*. 1992; 15(3):273–284. [PubMed: 1625339]
- Merjonen P, Pulkki-Raback L, Puttonen S, Keskivaara P, Juonala M, Telama R, et al. Anger is associated with subclinical atherosclerosis in low SES but not in higher SES men and women. The Cardiovascular Risk in Young Finns Study. *Journal of Behavioral Medicine*. 2008; 31(1):35–44. [PubMed: 17940862]
- Mikulincer M, Shaver PR, Pereg D. Attachment theory and affect regulation: The dynamics, development, and cognitive consequences of attachment-related strategies. *Motivation and Emotion*. 2003; 27:77–102.
- Miller GE, Chen E, Fok AK, Walker H, Lim A, Nicholls EF, et al. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences of the United States of America*. 2009a; 106(34):14716–14721. [PubMed: 19617551]
- Miller G, Chen E, Cole SW. Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology*. 2009b; 60:501–524.
- Miller GE, Chen E, Zhou E. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*. 2007; 133:25–45. [PubMed: 17201569]
- Minuchin, S. *Families of the slums*. New York, NY: Basic Books; 1967.
- Mittleman MA, Maclure M, Nachnani M, Sherwood JB, Muller JE. Educational attainment, anger, and the risk of triggering myocardial infarction onset. The Determinants of Myocardial Infarction Onset Study Investigators. *Arch Intern Med*. 1997; 157(7):769–775. [PubMed: 9125009]
- Moore LV, Diez Roux AV. Associations of neighborhood characteristics with the location and type of food stores. *Am J Public Health*. 2006; 96(2):325–331. [PubMed: 16380567]
- Moore S, Daniel M, Gauvin L, Dube L. Not all social capital is good capital. *Health Place*. 2009; 15(4):1071–1077. [PubMed: 19482506]
- Morozink JA, Friedman EM, Coe CL, Ryff CD. Socioeconomic and psychosocial predictors of interleukin-6 in the MIDUS national sample. *Health Psychol*. 2010; 29(6):626–635. [PubMed: 20954777]
- Musick, JS.; Stott, FM.; Spencer, KK.; Goldman, J.; Cohler, BJ. Maternal factors related to vulnerability and resiliency in young children at risk. In: Anthony, EJ.; Cohler, BJ., editors. *The invulnerable child*. New York, NY: Guilford Press; 1987. p. 229-252.
- Nachmias M, Gunnar M, Mangelsdorf S, Parritz RH, Buss K. Behavioral inhibition and stress reactivity: the moderating role of attachment security. *Child Dev*. 1996; 67(2):508–522. [PubMed: 8625725]

- Nation DA, Szeto A, Mendez AJ, Brooks LG, Zaias J, Herderick EE, et al. Oxytocin attenuates atherosclerosis and adipose tissue inflammation in socially isolated ApoE^{-/-} mice. *Psychosomatic Medicine*. 2010; 72(4):376–382. [PubMed: 20368478]
- National Academy of Sciences. Unequal treatment: Confronting racial and ethnic disparities in health care. National Academy of Sciences; 2002. <http://www.nap.edu/openbook/030908265X/html/3.html>
- National Center for Health Statistics. Health, United States, 2009. Hyattsville, MD: 2010.
- Parker KJ, Buckmaster CL, Justus KR, Schatzberg AF, Lyons DM. Mild early life stress enhances prefrontal-dependent response inhibition in monkeys. *Biol Psychiatry*. 2005; 57(8):848–855. [PubMed: 15820705]
- Parker KJ, Buckmaster CL, Schatzberg AF, Lyons DM. Prospective investigation of stress inoculation in young monkeys. *Arch Gen Psychiatry*. 2004; 61(9):933–941. [PubMed: 15351772]
- Pavlov VA, Tracey KJ. The cholinergic anti-inflammatory pathway. *Brain, Behavior, and Immunity*. 2005; 19(6):493–499.
- Pradhan AD, Manson JE, Rossouw JE, Siscovick DS, Mouton CP, Rifai N, et al. Inflammatory biomarkers, hormone replacement therapy, and incident coronary heart disease: Prospective analysis from the Women's Health Initiative observational study. *Jama-Journal of the American Medical Association*. 2002; 288(8):980–987.
- Presser HB, Cox AG. The work schedules of low-educated American women and welfare reform. *Monthly Labor Review*. 1997; 120(4)
- Pressman SD, Cohen S. Does positive affect influence health? *Psychol Bull*. 2005; 131(6):925–971. [PubMed: 16351329]
- Pulkki L, Keltikangas-Jarvinen L, Ravaja N, Viikari J. Child-rearing attitudes and cardiovascular risk among children: moderating influence of parental socioeconomic status. *Preventive Medicine*. 2003; 36(1):55–63. [PubMed: 12473425]
- Raikkonen K, Matthews KA, Flory JD, Owens JF, Gump BB. Effects of optimism, pessimism, and trait anxiety on ambulatory blood pressure and mood during everyday life. *Journal of Personality and Social Psychology*. 1999; 76(1):104–113. [PubMed: 9972556]
- Repetti RL, Taylor SE, Seeman T. Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*. 2002; 128:330–366. [PubMed: 11931522]
- Ridker PM, Rifai N, Stampfer MJ, Hennekens CH. Plasma concentration of interleukin-6 and the risk of future myocardial infarction among apparently healthy men. *Circulation*. 2000; 101(15):1767–1772. [PubMed: 10769275]
- Robbins RN, Bryan A. Relationships between future orientation, impulsive sensation seeking, and risk behavior among adjudicated adolescents. *J Adolesc Res*. 2004; 19(4):428–445. [PubMed: 16429605]
- Ross HE, Young LJ. Oxytocin and the neural mechanisms regulating social cognition and affiliative behavior. *Front Neuroendocrinol*. 2009; 30(4):534–547. [PubMed: 19481567]
- Rothbaum F, Weisz JR, Snyder S. Changing the world and changing the self: A two-process model of perceived control. *Journal of Personality and Social Psychology*. 1982; 42:5–37.
- Roy B, Diez-Roux AV, Seeman T, Ranjit N, Shea S, Cushman M. Association of optimism and pessimism with inflammation and hemostasis in the Multi-Ethnic Study of Atherosclerosis (MESA). *Psychosomatic Medicine*. 2010; 72(2):134–140. [PubMed: 20100888]
- Rozanski A, Blumenthal JA, Davidson KW, Saab PG, Kubzansky L. The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice: the emerging field of behavioral cardiology. *Journal of the American College of Cardiology*. 2005; 45(5):637–651. [PubMed: 15734605]
- Ruggles S. The origins of African-American family structure. *American Sociological Review*. 1994; 59:136–151.
- Rutter, M. Protective factors in children's responses to stress and disadvantage. In: Kent, MW.; Rolf, JE., editors. *Primary prevention of psychopathology*. Vol. 3. Hanover, NH: University Press of New England; 1979. p. 49-74. Social competence in children
- Rutter M. Psychosocial resilience and protective mechanisms. *Am J Orthopsychiatry*. 1987; 57(3): 316–331. [PubMed: 3303954]

- Rutter M. Resilience: some conceptual considerations. *J Adolesc Health*. 1993; 14(8):626–31. 690–6. [PubMed: 8130234]
- Ryff CD, Singer BH, Love GD. Positive health: Connecting well-being with biology. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*. 2004; 359(1449): 1383–1394.
- Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*. 2000; 21(1):55–89. [PubMed: 10696570]
- Scheier MF, Matthews KA, Owens J, Magovern GJ, Lefebvre RC, Abbott RA, et al. Dispositional optimism and recovery from coronary artery bypass surgery: The beneficial effects on physical and psychological well-being. *Journal of Personality and Social Psychology*. 1989; 57:1024–1040. [PubMed: 2614656]
- Scheier MF, Matthews KA, Owens JF, Schulz R, Bridges MW, Magovern GJ, et al. Optimism and rehospitalization following coronary artery bypass graft surgery. *Archives of Internal Medicine*. 1999; 159:829–835. [PubMed: 10219928]
- Schneiderman N, Ironson G, Siegel SD. Stress and health: psychological, behavioral, and biological determinants. *Annu Rev Clin Psychol*. 2005; 1:607–628. [PubMed: 17716101]
- Schwartz AR, Gerin W, Davidson KW, Pickering TG, Brosschot JF, Thayer JF, et al. Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine*. 2003; 65(1):22–35. [PubMed: 12554813]
- Seeman TE, McEwen BS, Rowe JW, Singer BH. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America*. 2001; 98(8):4770–4775. [PubMed: 11287659]
- Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation - Allostatic load and its health consequences: MacArthur studies of successful aging. *Arch Intern Med*. 1997; 157(2259): 2268.
- Seery MD, Holman EA, Silver RC. Whatever does not kill us: cumulative lifetime adversity, vulnerability, and resilience. *J Pers Soc Psychol*. 2010; 99(6):1025–1041. [PubMed: 20939649]
- Segerstrom SC, Miller GE. Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*. 2004; 130(4):601–630. [PubMed: 15250815]
- Segerstrom SC, Taylor SE, Kemeny ME, Fahey JL. Optimism is associated with mood, coping, and immune change in response to stress. *Journal of Personality and Social Psychology*. 1998; 74(6): 1646–1655. [PubMed: 9654763]
- Seligman ME, Csikszentmihalyi M. Positive psychology. An introduction. *Am Psychol*. 2000; 55(1): 5–14. [PubMed: 11392865]
- Selye H. Stress and disease. *Science*. 1955; 122(3171):625–631. [PubMed: 13255902]
- Selye, H. *Physiology and pathology of exposure to stress*. Montreal, Canada: Acta; 1950.
- Shannon KE, Beauchaine TP, Brenner SL, Neuhaus E, Gatzke-Kopp L. Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. *Dev Psychopathol*. 2007; 19(3):701–727. [PubMed: 17705899]
- Shaver PR, Mikulincer M. Attachment-related psychodynamics. *Attachment and Human Development*. 2002; 4:133–161. [PubMed: 12467506]
- Simeon D, Yehuda R, Cunill R, Knutelska M, Putnam FW, Smith LM. Factors associated with resilience in healthy adults. *Psychoneuroendocrinology*. 2007; 32(8–10):1149–1152. [PubMed: 17913377]
- Smeekens S, Marianne Riksen-Walraven J, van Bakel HJ. Cortisol reactions in five-year-olds to parent-child interaction: the moderating role of ego-resiliency. *J Child Psychol Psychiatry*. 2007; 48(7):649–656. [PubMed: 17593145]
- Smith TW, Glazer K, Ruiz JM, Gallo LC. Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of Personality*. 2004; 72(6):1217–1270. [PubMed: 15509282]
- Snibbe AC, Markus HR. You can't always get what you want: Educational attainment, agency, and choice. *Journal of Personality and Social Psychology*. 2005; 88(4):703–720. [PubMed: 15796669]

- Souza GG, Mendonca-de-Souza AC, Barros EM, Coutinho EF, Oliveira L, Mendlowicz MV, et al. Resilience and vagal tone predict cardiac recovery from acute social stress. *Stress*. 2007; 10(4): 368–374. [PubMed: 17853065]
- Starfield B, Robertson J, Riley AW. Social class gradients and health in childhood. *Ambulatory Pediatrics*. 2002; 2(4):238–246. [PubMed: 12135396]
- Stead M, MacAskill S, MacKintosh AM, Reece J, Eadie D. “It’s as if you’re locked in”: qualitative explanations for area effects on smoking in disadvantaged communities. *Health Place*. 2001; 7(4):333–343. [PubMed: 11682332]
- Stephens NM, Hamedani MG, Markus HR, Bergsieker HB, Eloul L. Why did they “choose” to stay? Perspectives of Hurricane Katrina observers and survivors. *Psychol Sci*. 2009; 20(7):878–886. [PubMed: 19538433]
- Taylor SE. Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist*. 1983; 38:1161–1173.
- Taylor SE, Kemeny ME, Reed GM, Bower JE, Gruenewald TL. Psychological resources, positive illusions, and health. *American Psychologist*. 2000a; 55(1):99–109. [PubMed: 11392870]
- Taylor SE, Klein LC, Lewis BP, Gruenewald TL, Gurung RAR, Updegraff JA. Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*. 2000b; 107(3):411–429. [PubMed: 10941275]
- Thayer JF, Lane RD. The role of vagal function in the risk for cardiovascular disease and mortality. *Biol Psychol*. 2007; 74(2):224–242. [PubMed: 17182165]
- Thayer JF, Sternberg E. Beyond heart rate variability: vagal regulation of allostatic systems. *Annals of the New York Academy of Sciences*. 2006; 1088:361–372. [PubMed: 17192580]
- Tomaka J, Blascovich J. Effects of justice beliefs on cognitive appraisal of and subjective, physiological, and behavioral responses to potential stress. *Journal of Personality and Social Psychology*. 1994; 67(4):732–740. [PubMed: 7965617]
- Troxel WM, Matthews KA. What are the costs of marital conflict and dissolution to children’s physical health? *Clinical Child and Family Psychology Review*. 2004; 7(1):29–57. [PubMed: 15119687]
- Tugade MM, Fredrickson BL. Resilient individuals use positive emotions to bounce back from negative emotional experiences. *J Pers Soc Psychol*. 2004; 86(2):320–333. [PubMed: 14769087]
- Updegraff JA, Silver RC, Holman EA. Searching for and finding meaning in collective trauma: results from a national longitudinal study of the 9/11 terrorist attacks. *J Pers Soc Psychol*. 2008; 95(3): 709–722. [PubMed: 18729704]
- Vasan RS, Larson MG, Leip EP, Evans JC, O’Donnell CJ, Kannel WB, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *New England Journal of Medicine*. 2001; 345(18):1291–1297. [PubMed: 11794147]
- Vitaliano PP, Scanlan JM, Zhang JP, Savage MV, Brummett B, Barefoot J, et al. Are the salutogenic effects of social supports modified by income? A test of an “added value hypothesis”. *Health Psychology*. 2001; 20(3):155–165. [PubMed: 11403213]
- Werner EE. Children of the garden island. *Scientific American*. 1989; 260:106–111. [PubMed: 2928762]
- Werner EE. Risk resilience, and recovery: Perspectives from the Kauai Longitudinal Study. *Development and Psychopathology*. 1993; 5:503–515.
- Werner EE. Resilience in development. *Current Directions in Psychological Science*. 1995; 4:81–85.
- Werner, EE.; Smith, RS. *Overcoming the odds: High risk children from birth to adulthood*. Ithaca, NY: Cornell University Press; 1992.
- Williams DR. Racial/ethnic variations in women’s health: The social embeddedness of health. *American Journal of Public Health*. 2002; 92:588–597. [PubMed: 11919058]
- Wills TA, Sandy JM, Yaeger AM. Time perspective and early-onset substance use: a model based on stress-coping theory. *Psychol Addict Behav*. 2001; 15(2):118–125. [PubMed: 11419227]
- Wright LB, Treiber FA, Davis H, Strong WB. Relationship of John Henryism to cardiovascular functioning at rest and during stress in youth. *Annals of Behavioral Medicine*. 1996; 18:146–150.

- Wyman PA, Cowen EL, Work WC, Hoyt-Meyers L, Magnus KB, Fagen DB. Caregiving and developmental factors differentiating young at-risk urban children showing resilient versus stress-affected outcomes: a replication and extension. *Child Dev.* 1999; 70(3):645–659. [PubMed: 10368913]
- Yates, TM.; Egeland, B.; Sroufe, LA. Rethinking resilience: A developmental process perspective. In: Luthar, SS., editor. *Resilience and vulnerability: Adaptation in the context of childhood adversities.* New York, NY: Cambridge University Press; 2003. p. 243-266.
- Yehuda R. Risk and resilience in posttraumatic stress disorder. *Journal of Clinical Psychiatry.* 2004; 65(Suppl 1):29–36. [PubMed: 14728094]
- Yehuda R, Flory JD, Southwick S, Charney DS. Developing an agenda for translational studies of resilience and vulnerability following trauma exposure. *Annals of the New York Academy of Sciences.* 2006; 1071:379–396. [PubMed: 16891584]
- Zaharia MD, Kulczycki J, Shanks N, Meaney MJ, Anisman H. The effects of early postnatal stimulation on Morris water-maze acquisition in adult mice: genetic and maternal factors. *Psychopharmacology (Berl).* 1996; 128(3):227–239. [PubMed: 8972542]
- Zautra AJ, Arewasikporn A, Davis MC. Resilience: Promoting well-being through recovery, sustainability, and growth. *Research in Human Development.* 2010; 7:221–238.
- Zautra AJ, Hall JS, Murray KE. Resilience: A new integrative approach to health and mental health research. *Health Psychology Review.* 2008; 2:41–64.

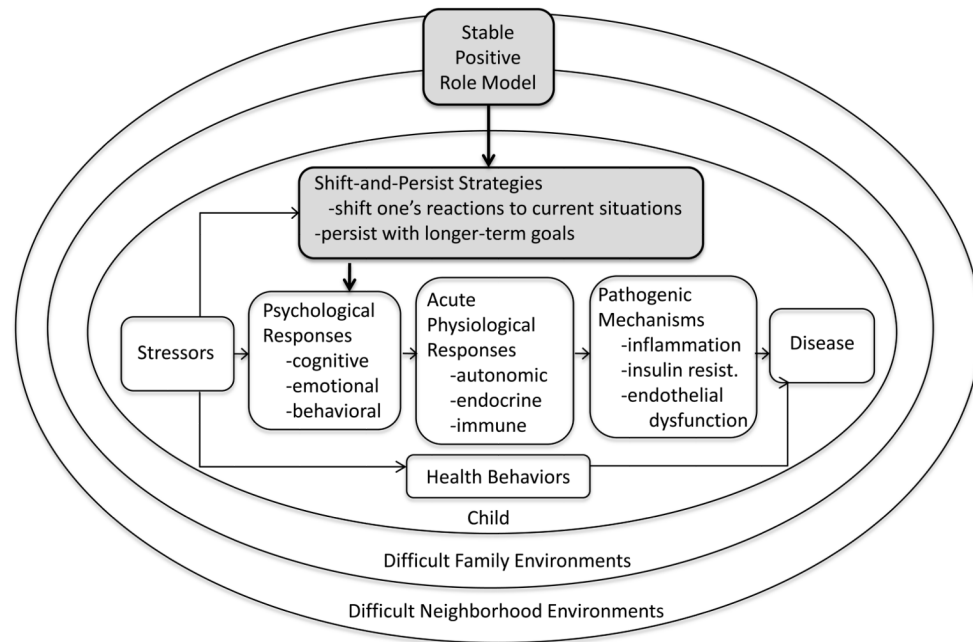


Figure 1.

Model of how “shift-and-persist” strategies lead to health benefits among low socioeconomic status (SES) individuals. The model depicts how low SES children are embedded within broader family and neighborhood contexts which entail exposure to recurrent, often uncontrollable stressors. Traditional models (middle row in model) state that these stressors evoke a constellation of psychological responses, including cognitive threat appraisals, negative emotions, and coping behaviors, as well as health behaviors that can contribute to disease. These responses in turn elicit acute physiological activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, which if maintained over time, promote long-term pathogenic mechanisms that result in disease years later. However, in the midst of this adversity, some low SES children are able to find positive role models, who provide stable attachment relationships that promote positive beliefs about others and the world (e.g., trust and optimism), and who socialize children about appropriate emotion regulation behaviors. In turn, attachment and socialization of behaviors allows children to more easily shift themselves in the face of immediate stressors (reappraise stressors and regulate their emotional reactions), while at the same time, persisting with life by holding on to meaning and optimism. Together, this set of strategies makes low SES children less likely to acutely display negative psychological and physiological responses to stressors. Over a lifetime, these tendencies can offset the pathogenic processes for chronic diseases of aging that low SES normally sets into motion.