Social Structures, Aging and Self-Regulation (2006), New York, Spring. 69-83

3

Social Influences on Adult Personality, Self-Regulation, and Health

Daniel K. Mroczek, Avron Spiro III, Paul W. Griffin, and Shevaun D. Neupert

There are many definitions of self-regulation, especially conceptualized as a developmental phenomenon (Brandtstadter & Renner, 1990; Heckhausen & Schulz, 1995; Lachman & Burack, 1993), but most theories mention three basic components: mood regulation (with a particular emphasis on the regulation of negative affect), planfulness or impulse control, and control beliefs (as well as actual control; Heckhausen & Schulz, 1995). Although comprehensive theories of self-regulation (e.g., Carver & Scheier, 1982, 1998, 1999) suggest that there may be more to self-regulation than this group of concepts, much of what is meant by the term self-regulation is captured by this trio of individual difference concepts. In many senses, self-regulation is a broad type of personality characteristic, as Bandura (1999) has argued for many years. Moreover, like personality dimensions, people display individual differences in self-regulation in the ways it is manifested and how it functions within their lives. In this chapter, we will first define self-regulation via the three basic personality components we have mentioned. Second, we will discuss how changes in these constituents can influence changes in self-regulatory behavior with an important impact on physical health. Third and last, we will tie these themes to social structure and aging, describing how social or societal factors may impact self-regulation change, which in turn could potentially change self-regulation and even physical health and mortality further down the line.

A previous version of this paper was presented at the Penn State Gerontology Center Conference on Social Structures, Aging, and Self-Regulation in the Elderly, October 4–5, 2004, Penn State University, State College, PA.

SELF-REGULATION AS AN EMERGENT CONSTRUCT FORMED BY PERSONALITY

As mentioned above, we believe three broadly-construed personality dimensions are particularly salient for self-regulation: mood regulation, impulse control, and control beliefs. We contend that these three combine to form self-regulation as an emergent construct (also known as a "formative" construct; Bollen & Lennox, 1991; Edwards & Bagozzi, 2000). In an emergent construct, the defining constructs are distinct, but together they have emergent properties. This definition contrasts with the better-known concept of the latent construct (e.g., the common factor model), which assumes the latent variable causes the observed variables that are used to measure it. However, the personality variables that we believe play a major role in defining self-regulation are distinct constructs in and of themselves; they are not manifestations of an underlying causal, latent variable. Nevertheless, they may be brought together into a common composite or "formative" construct that is integrative in nature (Bollen & Lennox, 1991; Edwards & Bagozzi, 2000).

Mood regulation, the first of these three constructs, refers to a person's ability to govern mood over a period of time (Gross, 1999; Larsen, 2000). Recent theory has suggested that moods fluctuate around a set point (Headey & Wearing, 1989), and that people vary in their capacity to bring their moods back to this set point (Eid & Diener, 1999). Some people are highly variable around their set point, while others exert a greater degree of control over their moods. While not synonymous with mood regulation, individual differences in the personality trait neuroticism vary with individual differences in within-person mood variability (Eid & Diener, 1999; Larsen, 2000). People high in trait neuroticism tend to show greater fluctuations around their set point over short periods of time (Bolger & Schilling, 1991). They are more variable in their moods, leading some to label this trait "emotional stability" (Goldberg, 1992) or "emotionality" (Eysenck & Eysenck, 1968). We believe these forms of mood regulation should be a major constituent of any self-regulation construct.

The second aspect of self-regulation is planfulness. Better regulation implies an ability to control one's impulses and live life in a more organized and planful manner (Roberts & Bogg, 2004). Less planfulness is likely to result in worse health because precautions or preventive steps to maintain

or improve health were not taken, or due to blind impulse-gratification resulting in exposure to grave health risks (e.g., sexually-transmitted diseases or fatal accidents). In fact, the personality trait conscientiousness encompasses many of the planfulness elements of self-regulation. Indeed, prior research has indicated that people low in personality trait conscientiousness and other forms of unplanfulness engage in more risky health behaviors such as drug and alcohol abuse (Caspi et al., 1997) and have higher rates of mortality (Friedman et al., 1993) than those higher in the trait. Planfulness and more effective and organized life management are all key aspects of self-regulation, which in turn lead to less risk-taking and better health behaviors, creating better physical and mental health and lower mortality over the lifespan.

The third and last set of defining variables for our emergent self-regulation construct is control beliefs. Some people believe they can influence events in their lives, giving rise to a sense of mastery and efficacy and the belief that one can achieve goals, including health goals. Others feel less control over their lives and events in their lives, producing the belief that they cannot do much to change the course of their lives or their health (Lachman, Ziff, & Spiro, 1994; Perrig-Chiello, Perrig, & Stahelin, 1999). Control and the sense of mastery it confers is an integral part of developmental regulation (Brandtstadter & Renner, 1990; Heckhausen & Schulz, 1995; Lachman & Burack, 1993). It indexes one of the cognitive elements of self-regulation in that it taps into people's belief systems and provides a window into an important aspect of how people think about and manage (and consciously regulate) their lives.

Each of these three constructs (mood regulation, planfulness, and control beliefs) are well understood, and there is a large nomological net connecting them with other variables (Contrada, Cather, & O'Leary, 1999). Despite this, researchers do not think of the three as connected to one other within a common conceptual structure. The overarching concept of self-regulation, however, provides a broad conceptual structure that allows this trio of empirically and theoretically distinct constructs to be understood together within a framework that has important consequences for behavior. That said, for most of the remainder of this chapter we will focus mainly on only one of the three, mood regulation (often operationalized as neuroticism). We will describe how it influences self-regulation as well as downstream outcomes such as health and mortality.

MOOD REGULATION AND STRESS REACTIVITY: AN ISSUE OF SELF-REGULATION

It is well known that high neuroticism is associated with higher levels of negative affect in general. However, neuroticism is also associated with greatly elevated negative affect during specific types of situations, such as stressful conditions (e.g., during a fight with one's spouse or boss). The tendency of people who are high in neuroticism to react to stressful situations with high negative affect is called the *stress reactivity effect* (Bolger & Schilling, 1991; Suls, 2001), and is perhaps the central concept in understanding individual differences in mood regulation. When people high in neuroticism encounter stressful events, they tend to experience them as more aversive and react with much higher levels of negative affect than those low in this trait (Bolger & Schilling, 1991; Bolger & Zuckerman, 1995; David & Suls, 1999; Gunthert, Cohen, & Armeli, 1999). Suls (2001) calls this process "hyperreactivity," or a large change in negative affect in response to a stressor (Suls, Green, & Hillis, 1998).

The theoretical underpinnings of this hyperreactivity are fourfold (Mroczek & Almeida, 2004), and each one of the four sheds light on why the effect is fundamentally an issue of self-regulation. First, persons high in neuroticism report larger numbers of stressful events in their lives, implying greater exposure to stress or to the creation of stressful situations (Bolger & Zuckerman, 1995; Ormel & Wohlfarth, 1991). Optimal self-regulation requires an ability to identify and avoid situations that may elevate negative affect, and persons high in neuroticism may lack this particular regulatory ability.

Second, persons high in neuroticism are more likely to appraise stressors as threats instead of as challenges, increasing the probability of experiencing negative affect as a response to stressful life events (Lazarus & Folkman, 1984; Suls, 2001). During the appraisal process, persons high in neuroticism also tend to focus more on the negative features of stressful events than people low in neuroticism (Hemenover, 2001). Optimal self-regulation demands healthy appraisal of the events that occur in our lives. If we see everything as a threat, we run the risk of activating threat-response systems, such as Gray's (1991) fight-or-flight response, when they are not necessary. Such over-activation of threat response systems not only results in elevated negative affect or distress when there is no need, but also prolongs their physiological consequences, such as elevated

levels of stress hormones (e.g., cortisol) and immune system parameters (e.g., interleukin-6). Chronic levels of these hormones indicate dysregulation, and can ultimately cause both cardiovascular and neurological damage (Kendler, Thornton, & Gardner, 2001; Wilson, Bienas, Mendes de Leon, Evans, & Bennett, 2003; Wilson, Mendes de Leon, Bienas, Evans, & Bennett, 2004). This physical dysregulation is a consequence of psychological dysregulation.

Third, after a stressful event has occurred, persons high in neuroticism are more likely to remember events as more stressful or traumatic than people low in neuroticism. In essence, they encode life events differently (Larsen, 1990). Again, we believe this a type of regulatory failure. Healthy, optimal self-regulation demands that individuals have a realistic view of events that is not overly negative. If I looked back on any event in my life and focused on only the bad things that happened, discarding the good, then my memories would likely provoke unnecessary feelings of negative affect. Again, this represents an inability (or compromised ability) to maintain a healthy balance of negative to positive or neutral affect. Such imbalance implies dysregulation.

Fourth and last, persons high in neuroticism may employ less productive coping strategies, especially emotion-focused coping (Bolger, 1990; David & Suls, 1999), or utilize strategies that are unproductive (Bolger & Zuckerman, 1995). Suboptimal coping is again a problem of self-regulation, and results in prolonged experiences of negative affect, as opposed to re-regulation back to a state of lessened negative affect.

In each of these four underpinnings of the stress reactivity effect, the end result is elevated negative affect or prolonged periods of heightened negative affect, as well as potential elevations of dangerous stress hormones. Stress reactivity is in many ways an inability (or lessened ability) to re-regulate back to a more optimal emotional state. This is bad enough if it occurs frequently. However, what are the consequences if negative affect is repeatedly activated over long periods of time? What are the potential effects of an inability to self-regulate with respect to negative affect? Kendler et al. (2001) and Wilson et al. (2003, 2004) have hypothesized that neuroticism, and the constant elevated levels of negative affect that accompany that trait over periods of many years or decades, leads to a negative emotion "hair trigger" in older adulthood. They suggest that as people high in neuroticism grow older, they become more susceptible to elevated negative affect. Indeed, neuroticism and prolonged negative affect have

been linked to dysregulation of the hypothalamic-pituitary-adrenal stress axis (HPA) (Kendler et al., 2001).

KINDLING EFFECTS: A SELF-REGULATORY FAILURE

Many studies, including some of our own, have shown that negative affect is lower in older than younger adults (Carstensen, Pasupathi, Mayr, & Nesselroade, 2000; Mroczek, 2001; Mroczek & Almeida, 2004; Mroczek & Kolarz, 1998). More importantly, longitudinal studies using growth-curve models have documented that negative affect declines as we age (Charles, Reynolds, & Gatz, 2001; Griffin, Mroczek, & Spiro, in press). Yet, little is known about older adults who are high in neuroticism. What do their levels of negative affect look like? We mentioned earlier that adults who are high in neuroticism might grow more susceptible to elevated negative affect as they grow older (Kendler et al., 2001; Wilson et al., 2003, 2004). The repeated activation of negative affect over a lifetime may increase reactivity by older adulthood. Repetition of negative affect activation, rather than causing habituation, may lead to its opposite, sensitization. Heightened sensitivity in turn may lead to easier activation of negative affect when a person encounters stressful stimuli. In other words, super-heightened stress reactivity (and concomitant elevated negative affect) may come to characterize people with high neuroticism as they age as a consequence of frequent activation. A lifetime of repeated activations, perhaps of the neural systems that mediate negative affect, may lead to a "hair trigger" in which states of elevated negative affect are prompted by relatively weak stimuli.

These heightened sensitivities are called kindling effects. Kindling occurs when repeated exposure to some stimulus causes sensitization (Gilbert, 1994; Kendler et al., 2001; van der Kolk, 1996, 1997; Woolf & Costigan, 1999). Kindling effects have been observed with respect to chronic pain, drug abuse, epilepsy, traumatic stress, anxiety, and depressive episodes. For example, major depressive episodes are frequently triggered by stressful life events (among those who are predisposed to major depression, of course). However, after repeated depressive episodes, the likelihood increases that these people will spontaneously slip into a depressive episode without the trigger of a stressful life event (Kendler et al., 2001). An individual in a kindled state is also more sensitive to triggering stimuli; depression is more likely to be triggered when a stressful life event takes place. In other words, the stimulus threshold becomes lower. Similarly,

kindling effects occur in chronic pain. Many people become more sensitive to pain rather than developing tolerance over the long term (Woolf & Costigan, 1999). Kindling is a relatively permanent state of heightened susceptibility (Gilbert, 1994), brought on by past experience.

Some contend that kindling effects are a result of neuroplasticity, the ability of groupings of neurons to change and realign themselves in response to repeated exposure to stimuli (Gilbert, 1994; van der Kolk, 1997; Wilson et al., 2003; Woolf & Costigan, 1999). Neural networks that govern some processes (the sensation of pain, an epileptic seizure, feelings of depression, or negative affect) can themselves become molded by stimuli, causing these networks to become even more sensitive to stimuli and to sometimes even react spontaneously (van der Kolk, 1996, 1997; Woolf & Costigan, 1999). We suggest that persons high in neuroticism may become similarly molded over time, resulting in hypersensitivity to stress. Thus, older adults who have been high in neuroticism over their life course should display greater reactivity to stress in older adulthood when the opposite would be more beneficial to their health.

We do have some evidence consistent with this perspective. The 3-way moderator effect shown in Figure 3.1 lends support to the aforementioned perspective. These data are from 1,012 adults (54.5% women, 45.5% men) who are participants in the National Study of Daily Experiences (NSDE; Almeida, 2005; Almeida, Wethington, & Kessler, 2002; Mroczek & Almeida, 2004), one of the few daily experience studies that is national in scope. NSDE respondents completed a questionnaire about daily events, stressors, and affect for eight consecutive days, and these data were analyzed using multilevel models. Figure 3.1 illustrates that older adults high in neuroticism are more reactive to stress than any other group. On stressor days (days when any of seven broad stressful events may have occurred), daily negative affect is extremely elevated among older adults with high neuroticism. Interestingly, older adults with low neuroticism display levels of negative affect much lower than their younger or midlife counterparts low in neuroticism. This may indicate that older adults who are low in neuroticism may do a better job of regulating their moods, or at least their negative affect, than any other age group. Better emotion regulation may be a benefit of older adults who are lucky to have low neuroticism. Yet, older adults who are high in neuroticism have the bad fortune of poorer control or regulation of their negative affect, at least when stress occurs.

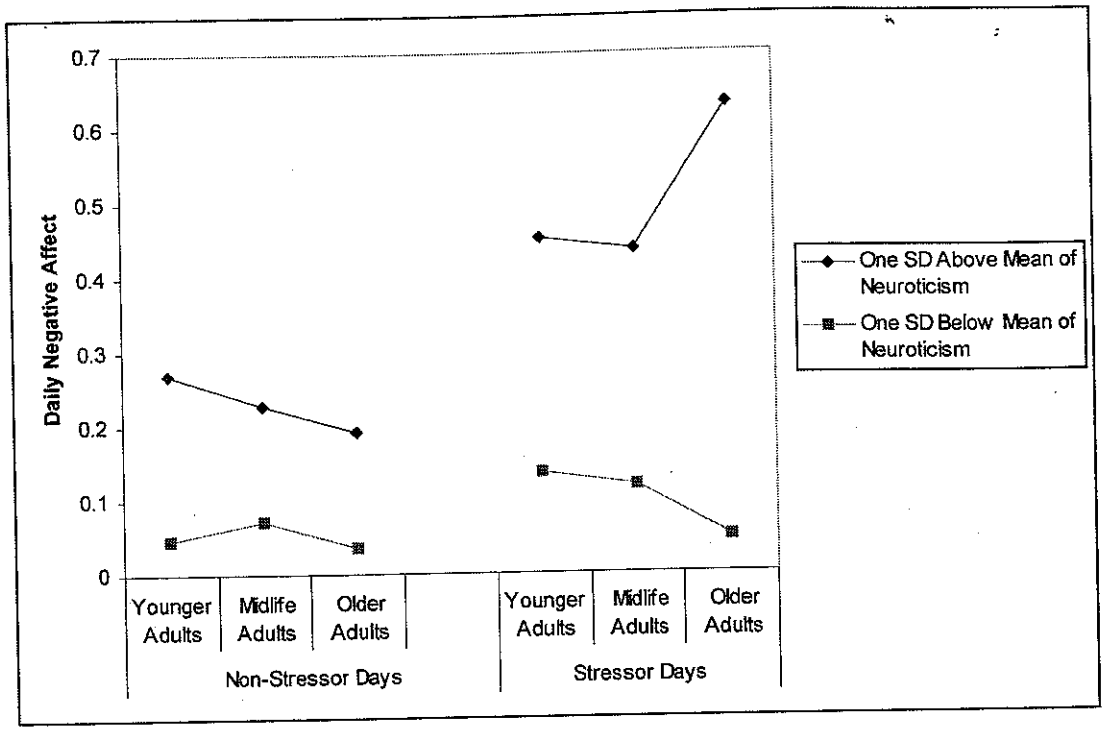

FIGURE 3.1 The stress reactivity effect for older, midlife, and younger adults: Neuroticism and age group moderate the association between daily stress and daily negative affect.

The heightened reactivity among older adults with high neuroticism is consistent with the kindling hypothesis (van der Kolk, 1996, 1997; Woolf & Costigan, 1999). A lifetime of frequent activation of the neural pathways associated with negative affect may bring about such sensitization, causing increased susceptibility to stimuli, including daily stress, that produce negative affect. In essence, the excessive activation of negative affect systems that plagues people high in neuroticism may, over years and decades, make the regulation of negative affect even more problematic. The difficulties in controlling negative affect are one of the key self-regulation problems for people high in neuroticism that may become worse over time. We do not know that this is exactly what underlies the effect in Figure 3.1, but it is one plausible explanation, backed up by recent work in several disciplines on kindling effects.

Tying the kindling hypothesis to the theme of social structure, we can easily imagine people who are high in neuroticism and are exposed to excessive amounts of stress due to social circumstances. For example, there are surely people who were born both high in dispositional neuroticism and to low socioeconomic status. The stressors of poverty combined

with a proclivity toward stress reactivity would presumably put such individuals at a much higher risk for the development of kindling effects and hyperreactivity.

THE HEALTH CONSEQUENCES OF POOR REGULATION OF NEGATIVE AFFECT

It is clear that hypersensitivity to stressful stimuli takes away from the quality of one's life and very likely has implications for mental health. However, there is emerging evidence that such deficits in mood regulation have consequences for physical health as well. Many studies have documented that acute and chronic stressors can elicit cortisol elevation (Dickerson & Kemeny, 2004). Experiencing stress increases corticotrophin-release hormone, activates the HPA-axis, and promotes secretion of glucocorticoids (e.g., cortisol) into blood circulation. Persistent elevated levels of cortisol or non-response of cortisol levels to challenge (blunted sensitivity) are symptomatic of general poor physical health, especially wear and tear on the HPA-axis (Kiecolt-Glaser & Glaser, 1986). Such blunted sensitivity is, in essence, a failure of physical self-regulation brought on by a failure of psychological self-regulation.

Moreover, an elevated level of glucocorticoids in general blood circulation is associated with heart disease and hippocampal cell death, including hippocampal atrophy (Seeman, Singer, McEwen, Horwitz, & Rowe, 1997). Thus, persons who are more effective at managing stress may show less heart disease and hippocampal atrophy than those who have self-regulatory difficulties with respect to stress management (e.g, those high in neuroticism). Self-regulation should moderate the effect of stress on the development of such physical problems as heart disease and hippocampal atrophy, ultimately influencing downstream variables such as mortality. Indeed, recent studies have provided evidence that is consistent with this view. Wilson et al. (2004) documented that high neuroticism in a sample of older adults was associated with a higher risk of mortality, and even earlier mortality, which had been previously linked to other personality traits such as low conscientiousness. These findings highlight the potential damage to physical health that self-regulatory problems can bring about, especially those that are due to personality traits such as high neuroticism and its associated difficulties in managing stress.

CHANGE IN HEALTH RISKS AS A FUNCTION OF CHANGE IN PERSONALITY

Thus far, we have painted a pessimistic picture of problematic self-regulation. We have conjured up images of persons high in neuroticism experiencing difficulty in regulating their moods in response to stress, maximizing negative affect, in turn leading to widespread physical damage in their brains and arteries, culminating in an earlier death. This is perhaps too bleak a picture, considering the evidence that neuroticism can change as we age. If neuroticism changes over time for a given person, it is possible that the health risks associated with high levels of this trait (and perhaps other traits) also change across that person's lifespan.

The debate over change in personality and psychological well-being has quietly evolved over the past several years (Caspi & Roberts, 1999; Roberts & DelVecchio, 2000; Roberts & Wood, in press). The old question of whether or not personality traits are stable has given way to the more subtle perspective that change is an individual difference variable in and of itself (Mroczek & Spiro, 2003a, 2003b). Some people are stable, whereas others change in varying degrees. Recent studies have documented individual differences in rate of change for numerous personality traits (Helson, Jones, & Kwan, 2002; Jones & Meredith, 1996; Jones, Livson, & Peskin, 2003; Mroczek & Spiro, 2003a, 2003b; Mroczek, Spiro, & Almeida, 2003; Small, Hertzog, Hultsch, & Dixon, 2003). These are illustrations of the lifespan developmental tenet of plasticity (Baltes, 1987; Roberts, 1997; Roberts & Wood, in press), which states that developmental constructs remain somewhat malleable throughout the lifespan. Roberts (1997) has argued that personality is an "open system" that remains sensitive to contextual life experiences and socialization processes through the lifespan.

With respect to neuroticism, these studies have generally found that this trait declines in general (across people) as we age, but that there are considerable individual differences around the overall trajectory. Thus, some people go down faster than others, some track the sample trajectory, some remain stable, and still others may see a rise in neuroticism over time. There is a range of change, and it likely has implications for health risk. For example, a person at age 40 who is one standard deviation above the mean on neuroticism has some quantifiable risk for dying younger than someone at the mean of neuroticism, based on the Wilson et al. (2004) study. However, as in most epidemiological studies of risk

factors, Wilson's study used neuroticism as assessed at one point in time. If high neuroticism is associated with higher risk of mortality several years down the road, then what happens to the risk when someone increases or decreases on this trait? Does risk of dying at a given age rise or fall as level of neuroticism changes? We can ask the same question of other findings in the area of personality and mortality. For instance, Friedman et al. (1993) found that lower conscientiousness measured early in life predicted later mortality. Surely, some individuals will remain stable and low in conscientiousness over periods of many years, and these people will remain at high risk for early mortality, but does that risk fall if a person increases on conscientiousness? There is a general (mean-level) decline in neuroticism and a general increase in conscientiousness as people age (Roberts & Walton, 2004). However, imagine the aforementioned person who was a standard deviation above the mean at age 40 experiences a decrease in neuroticism greater than the norm (via psychotherapy or some other intervention) and by age 50 is at the mean level. We may infer that with such a considerable drop in neuroticism, this person's risk of early mortality would drop as well.

This may not be the case if the physical damage (e.g., hippocampal atrophy) done prior to age 40 is extensive enough to keep the risk of an earlier death high even after a substantial drop in neuroticism. However, there is reason to believe that such changes can have a significant impact on health risk. As an analogy, consider high LDL cholesterol and the increased risk of suffering from some event, such as having a heart attack or dying from cardiac failure. LDL cholesterol can drop, like neuroticism, especially with intervention. One's risk of a heart attack declines as the risk factor declines. Getting your LDL back into a specified safe range brings the risk of the adverse health event back to the norm. Just as changes in cholesterol, blood pressure, or body mass index can change the risk of a cardiac event or early mortality, it may be that change in certain personality traits such as neuroticism or conscientiousness may also change the risk of similar undesirable health events.

Speculatively, people who are high in neuroticism may (through intervention) learn to regulate their moods better relatively early in life, perhaps in their 20s or 30s, before the potential damage of kindling effects sets in. Those who changed would presumably be better able to regulate negative affect and its ill effects, and may short-circuit some of the physical damage brought on by chronically high levels of negative emotion. Similarly,

people who are low in planfulness (or high in impulsivity) may improve their ability to plan and control their impulses relatively early in their adult lives (again, through intervention), leading to fewer of the risky health behaviors that are hypothesized to account for the conscientiousness-mortality associations (Friedman et al., 1993). These are interesting possibilities involving the way change in personality leads to change in self-regulatory behaviors (especially health-relevant self-regulation), which leads to change in the risks of health events or mortality.

A similar causal chain may exist among control beliefs, self-regulatory behaviors, and health. A strong feeling of control leads to greater flexibility in the management of one's life goals and choices. It is known that people who feel a greater sense of control over their life, especially their work, tend to be in better health and live longer (Marmot, 2004). However, social factors, such as socioeconomic status, also influence sense of control. Indeed, social influences exert at least some influence over all three of the personality dimensions that impact self-regulation. This finally brings us back to the title of this chapter.

SOCIAL INFLUENCES ON PERSONALITY DIMENSIONS

Families, societies, and cultures each have an impact on the development of neuroticism, conscientiousness, and control beliefs. Earlier, we hinted at proactive methods, such as interventions, that may alter personality trajectories and subsequent health risks. However, larger social forces also shape the level and direction of certain personality trajectories. We acknowledge that genetic factors place limits on the influence of social factors; there is no doubt that biological factors play a strong role in the development of personality characteristics. Yet, recent theory (Caspi & Roberts, 1999; Roberts & Wood, in press) and empirical research (Mroczek & Spiro, 2003a) support the view that personality traits are at least somewhat responsive to environmental forces.

For example, families differ in the extent to which they socialize children and adolescents to deal with stress in an effective and healthy manner. These differences among families likely alter mood regulation trajectories (although within families, shared influences have little impact, as behavior genetics studies have shown). Certainly, genetically-based temperament dimensions create a starting point for where mood regulation trajectories begin, but environmental presses mold them throughout childhood and

adolescence. A person may be predisposed to poor mood regulation, especially a proneness to chronically high negative affect. However, if that person learns ways to minimize negative affect—through family, peer or spouse influence—the potential negative health effects would be blunted. There is other evidence as well that sociocultural factors influence mood regulation. Two recent studies have noted cohort effects in mood regulation (operationalized by neuroticism), indicating that time and place may have some effect on development of this regulatory ability (Mroczek & Spiro, 2003a; Twenge, 2000).

With respect to planfulness and conscientiousness, people are exposed to different social forces that either promote or restrain impulsive behavior. An individual may possess a genetically-based proclivity toward impulsive behaviors, including those that may harm his or her health, but through familial, educational, or cultural forces learns to restrain those impulses. This would alter the course of that person's planfulness and conscientiousness trajectories, having long-term effects on health and health behaviors. Similarly, the influence of getting married or having children may alter conscientiousness trajectories, causing previously low-conscientiousness individuals to check their impulses more than they had before. In other words, they gain a greater degree of self-regulation. Thus, it is not difficult to imagine how family influences impact health via the shifting of planfulness or conscientiousness trajectories and its effect on self-regulation. School and other educational influences may also mitigate the negative impact of impulsivity and low planfulness. Henry, Caspi, Moffitt, Harrington, and Silva (1999) found that impulsive (undercontrolled) boys were less likely to engage in crime the longer they stayed in school.

Finally, families and cultures exert influence over control beliefs as well. The extent to which a person believes he or she is in control of his or her life (external versus internal sense of control) differs across cultures and across families within particular cultures. Yet, peer and spouse influence, as well as the influence of education and work, can potentially alter one's trajectory of control, with downstream effects on self-regulation and health. For example, a person may have come from a family that fostered fatalistic attitudes, creating an initial sense of low control within that person. However, through peer and spouse influence, this person eventually gains a greater sense of control over his or her life, including control over health. Believing that one's actions actually have an effect (internal sense of control) leads that person to take better care of his or her health. Thus,

the environmental influences of peer and spouse shifted this hypothetical person's control trajectories, leading to changes in self-regulatory health behaviors and in turn better health.

THE STRUGGLE AGAINST ONE'S OWN PERSONALITY

In the above examples, we have made the argument that a combination of biological and environmental factors impact personality trajectories, leading to changes in self-regulatory behaviors, in turn influencing health. However, we entertain the possibility that some personality dimensions do not change, but the behaviors associated with them do. That is, someone who is very impulsive (low in planfulness and conscientiousness) learns to restrain impulsive behaviors, but nonetheless still feels the urge to fulfill the impulse. In this case, the underlying, genetically-influenced personality disposition of high impulsivity has not changed, but one's control over the manifest behaviors that flow from the disposition does change. In this case, the person has gained self-regulation abilities. The person gains the ability to *go against his or her own nature,* but the underlying trait may still be there in relatively unaltered form. The person struggles against his or her own personality dispositions, yet in doing so, gains the ability to self-regulate. Again, while this comes naturally to some, others need to actively work for it.

Consequently, it is not clear whether the changes observed in long-term studies of personality, such as our own (Mroczek & Spiro, 2003a), reflect underlying or manifest personality change. This of course goes against the emergent variable model we presented at the beginning of this paper in which mood regulation, planfulness, and control beliefs defined self-regulation. These dimensions change, and by necessity, self-regulation abilities change. Yet, in the underlying versus manifest model, this may not be the case. Only empirical research can verify which model is better.

CONCLUSION

With respect to the health outcomes we have discussed, it almost does not matter whether the underlying self-regulation dimensions change or if only the manifest behaviors change. If people who find it difficult to regulate negative affect gain the ability to regulate it more effectively early in

life, they will likely obtain the health benefits. There may be many paths to improved regulation of negative affect. People may restructure their lives to avoid negative-affect producing situations, or they make seek out cognitive-behavioral therapy, or they may begin meditating regularly. Ultimately, perhaps the underlying disposition does decline for these people who have taken such proactive action. Or it may be that the latent proclivity still lurks, but the manifest behaviors (in this case, feelings of negative affect) are better controlled. Yet, with respect to the health outcomes, it may not matter. A person who possesses a genetic predisposition to produce excess LDL cholesterol never loses that genotypic trait; however, through treatment, the manifest effect (actual excess LDL) is controlled, although the underlying predisposition remains. In this sense, the underlying dispositions may remain stable at a latent level, but if the behaviors that flow from them become better regulated, people will gain health benefits. This is the hopeful message that we wish to convey.

We have focused on health outcomes in this chapter. However, improvements in some of the self-regulatory processes we have described, such as stress reactivity or impulse control, have benefits that go beyond physical health. They can lead to better mental health, enhanced functioning of social relationships, and improved productivity in work. Handling stress without experiencing negative affect or containing impulsive desires comes naturally for some people, who need not change because they already possess functioning self-regulation abilities. Any attempts at intervention need to be targeted at those most at risk. This is where the area of lifespan personality development can be of great use to those who study the development of self-regulation across the life course.

REFERENCES

Almeida, D. M. (2005). Resilience and vulnerability to daily stressors. *Current Directions in Psychological Science, 14–2,* 64–68.

Amaro, H., Russo, N. F., & Johnson, J. (1987). Family and work predictors of psychological well-being among Hispanic women professionals. *Psychology of Women Quarterly, 11,* 505–521.

Andersson, K., Siegel, R., Fuxe, K., & Eneroth, P. (1983). Intravenous injections of nicotine induce very rapid and discrete reductions of hypothalamic catecholamine levels associated with increases of ACTH,

vasopression, and prolactin secretion. *Acta Physiologica Scandinavica, 118,* 35–40.

Aneshensel, C. S., Rutter, C. M., & Lachenbruch, P. A. (1991). Social structure, stress, and mental health: Competing conceptual and analytic models. *American Sociological Review, 56,* 166–178.

Barden, N. (2004). Implication of the hypothalamic-pituitary-adrenal axis in the physiopathology of depression. *Journal of Psychiatry and Neuroscience, 29,* 185–193.

Barrot, M., Marinelli, M., Abrous, D. N., Rouge-Pont, F., Le Moal, M., & Piazza, P. V. (2000). The dopaminergic hyper-responsiveness of the shell of the nucleus accumbens is hormone-dependent. *European Journal of Neuroscience, 12,* 973–979.

Benowitz, N. L. (1988). Pharmacologic aspects of cigarette smoking and nicotine addiction. *New England Journal of Medicine, 319,* 1318–1330.

Benowitz, N. L. (1996). Pharmacology of nicotine: Addiction and therapeutics. *Annual Review of Pharmacology and Toxicology, 36,* 597–613.

Blascovich, J., Spencer, S. J., Quinn, D., & Steele, C. (2001). African Americans and high blood pressure: The role of stereotype threat. *Psychological Science, 12,* 225–229.

Blazer, D. G., Kessler, R. C., McGonagle, K. A., & Swartz, M. S. (1994). The prevalence and distribution of major depression in a national community sample: The National Comorbidity Survey. *American Journal of Psychiatry, 151,* 979–986.

Breslau, N., Kilbey, M. M., & Andreski, P. (1993). Nicotine dependence and major depression: New evidence from a prospective investigation. *Archives of General Psychiatry, 50,* 31–35.

Brown, D. R., Eaton, W., & Sussman, L. (1990). Racial differences in the prevaence of phobic disorders. *Journal of Nervous and Mental Diseas, 178(7),* 434–441.

Browning, C. R., & Cagney, K. A. (2003). Moving beyond poverty: Neighborhood structure, social processes, and health. *Journal of Health and Social Behavior, 44,* 552–571.

Bureau of the Census. (1995). *Statistical brief: Poverty areas.* Washington, D.C.: U.S. Department of Commerce, Economics and Statistics Administration.

Cagney, K. A., & Browning, C. R. (2004). Exploring neighborhood-level variation in asthma and other respiratory diseases. *Journal of General Internal Medicine, 19,* 229–236.

Cannetti, L., Bachar, E., & Berry, E. M. (2002). Food and emotion. *Behavioral Processes, 60*, 157–164.

Centers for Disease Control (CDC). (2002). Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. *Morbidity and Mortality Weekly Report, 51*(14), 300–303.

Centers for Disease Control (CDC). (2003). *Health United States, 2003, with chartbook on trends in the health of Americans.* Hyattsville, MD: CDC, National Center for Health Statistics.

Chatters, L. M., & Taylor, R. J. (1989). Age differences in religious participation among black adults. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences, 44*, S183–S189.

Clark, R., Anderson, N. B., Clark, V. R., & Williams, D. R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist, 54*, 805–816.

Cohen, S., Kessler, R. C., & Gordon, L. G. (1995). *Measuring stress.* New York: Oxford University Press.

Dallman, M. F. (2003). Stress by any other name . . . ? *Hormones and Behavior, 43,* 18–20.

Dallman, M. F., Pecoraro, N., Akana, S. F., la Fleur, S. E., Gomez, F., Houshyar, H., et al. (2003a). Chronic stress and obesity: A new view of "comfort food". *Proceedings of the National Academy of Sciences, 100,* 11696–11701.

Dallman, M. F., Akana, S. F., Laugero, K. D., Gomez, F., Manalo, S., Bell, M. E., et al. (2003b). A spoonful of sugar: Feedback signals of energy stores and corticosterone regulate responses to chronic stress. *Physiology and Behavior, 79,* 3–12.

Danziger, S., & Gottschalk, P. (1997). *America unequal.* Cambridge, MA: Harvard University Press.

De Jong, I.E.M., & De Kloet, R. (2004). Glucocorticoids and vulnerability to psychostimulant drugs: Toward substrate and mechanism. *Annals of New York Academy of Sciences, 1018,* 192–198.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.

Dohrenwend, B. P. (2000). The role of adversity and stress in psychopathology: Some evidence and its implications for theory and research. *Journal of Health and Social Behavior, 41*, 1–19.

Dohrenwend, B. P., & Dohrenwend, B. S. (1974). Social and cultural influences on psychophathology. *Annual Review of Psychology, 25,* 417–452.

Elit, L. M., Levine, M. N., Gafni, A., Whelan, T. J., Doig, G., Streiner, D. L., et al. (1996). Patients' preferences for therapy in advanced epithelial ovarian cancer: Development, testing, and application of a bedside decision instrument. *Gynecolic Oncology, 62,* 329–335.

Epstein, R. M., Alper, B. S., & Quill, T. E. (2004). Communicating evidence for participatory decision making. [The patient physician relationship.] *Journal of the American Medical Association, 291,* 2359–2366.

Essed, P. (1991). *Understanding everyday racism.* Newbury Park, California: Sage Publications.

Etzioni, A. (1993). *The spirit of community: Rights, responsibilities and the communitarian agenda.* New York: Crown Publishers.

Farley, R. (Ed.). (1995). *State of the union: American in the 1990s, economic trends* (Vol. 1). New York: Russell Sage.

Feagin, J. R. (1991). The continuing significance of race: Anti-black discrimination in public places. *American Sociological Review, 56,* 101–116.

Fenton, J. J., & Deyo, R. A. (2003). Patient self-referral for radiologic screening tests: Clinical and ethical concerns. *Journal of the American Board of Family Practice, 16,* 494–501.

Finch, J. F., & Zautra, A. J. (1992). Testing latent longitudinal models of social ties and depression among the elderly: A comparison of distribution-free and maximum likelihood estimates with nonnormal data. *Psychology and Aging, 7,* 107–118.

Fischer, J. (1969). Negroes and whites and rates of mental illness: Reconsideration of a myth. *Psychiatry, 32,* 428–446.

Gallo, L. C., Bogart, L. M., Vranceanu, A., & Matthews, K. A. (2005). Socioeconomic status, resources, psychological experiences, and emotional responses: A test of the reserve capacity model. *Journal of Personality and Social Psychology, 88,* 386–399.

Geronimus, A. T., & Thompson, P. J. (2004). To denigrate, ignore or disrupt: Racial inequality in health and the impact of a policy-induced breakdown of African American communities. *Du Bois Review, 1,* 247–279.

Gottschalk, P. (1997). Inequality, income growth, and mobility: The basic facts. *The Journal of Economic Perspectives, 11,* 21–40.

Green, C. R., Anderson, K. O., Baker, T. A., Campbell, L. C., Decker, S., Fillingim, R. B., et al. (2003). The unequal burden of pain: Confronting racial and ethnic disparities in pain. *Pain Medicine, 4,* 277–294.

Harburg, E., Erfurt, J., Chape, C., Havenstein, L., Schull, W., & Schork, M. A. (1973a). Socioecological stressor areas and black-white blood pressure: Detroit. *Journal of Chronic Disease, 26,* 595–611.

Harburg, E., Erfurt, J. C., Huenstein, L. S., Chape, C., Schull, W. J., & Schork, M. A. (1973b). Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosomatic Medicine, 35,* 276–296.

Harrison, R., & Bennett, C. (1995). Racial and ethnic diversity. In R. Farley (Ed.), *State of the union: America in the 1990's, social trends* (Vol. 2). New York: Russell Sage.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin, 112,* 64–105.

Hayward, M. D., Crimmins, E. M., Miles, T. P., & Yang, Y. (2000). The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *American Sociological Review, 65,* 910–930.

Heisler, M., Rust, G., Patillo, R., & Dubois, A. M. (2004). Improving health, eliminating disparities: Finding solutions for better health care for all. *Ethnicity & Disease, 15,* S2-1–S2-4.

Jackson, J. S. (1991). *Life in Black America.* Newbury Park: Sage Publications.

Jackson, J. S. (1993). African American experiences through the adult years. In R. J. Kastenbaum (Ed.), *The encyclopedia of adult development* (pp. 18–26). Phoenix, Arizona: Oryx Press.

Jackson, J. S. (Ed.). (2000). *New directions: African Americans in a diversifying nation.* Washington, D.C.: National Policy Association & Program for Research on Black Americans.

Jackson, J. S. (2002). Health and mental health disparities among black Americans. In M. Hager (Ed.), *Modern psychiatry: Challenges in educating health professionals to meet new needs* (pp. 246–254). New York: Josiah Macy Jr. Foundation.

Jackson, J. S., Brown, T. B., Williams, D. R., Torres, M., Sellers. S. L., & Brown, K. B. (1996). Racism and the physical and mental health status

of African Americans: A thirteen year national panel study. *Ethnicity & Disease, 6*, 132–147.

Jackson, J. S., Neighbors, H. W., & Gurin, G. (1986). Findings from a national survey of black mental health: Implications for practice and training. In M. R. Miranda, H. L. Kitano (Eds.), *Mental health research and practice in minority communities: Development of culturally sensitive training programs* (pp. 91–115). Rockville, MD: NIMH DHHS Pub. No. (ADM) 86–1466.

Jackson, J. S., Torres, M., Caldwell, C. H., Neighbors, H. W., Nesse, R. M., Taylor, R. J., et al. (2004). The National Survey of American Life: A study of racial, ethnic and cultural influences on mental disorders and mental health. *International Journal of Methods in Psychiatric Research, 13*, 196–207.

Jackson, J. S., Williams, D. R., & Torres, M. (2003). Discrimination, health and mental health: The social stress process. In A. Maney (Ed.), *Socioeconomic conditions, stress and mental disorders: Toward a new synthesis of research and public policy.* Bethesda, MD: NIH Office of Behavioral and Social Research.

James, S. A. (1994). John Henryism and the health of African Americans. *Culture of Medicine and Psychiatry, 18,* 163–182.

James, S. A., LaCroix, A. Z., Kleinbaum, D. G., & Strogatz, D. S. (1984). John Henryism and blood pressure differences among black men: The role of occupational stressors. *Journal of Behavioral Medicine, 7,* 259–275.

Jones, E., & Matsumoto, D. (1982). Psychotherapy with the underserved: Recent developments. In L. Snowden (Ed.), *Reaching the underserved: Mental health needs of neglected populations* (pp. 207–228). Beverly Hills: Sage Publications.

Jones, R. T. (1987). Tobacco dependence. In H. Y. Meltzer (Ed.), *Psychopharmacology: A generation of progress, twenty-fifth anniversary volume, ACNP* (pp. 1589–1595). New York: Raven Press.

Kahng, S. K., Dunkle, R., & Jackson, J. S. (2004). The relationship between the trajectory of body mass index and health trajectory among older adults: Multilevel modeling analyses. *Research on Aging, 26*, 31–61.

Kao, G., & Thompson, J. S. (2003). Racial and ethnic stratification in educational achievement and attainment. *Annual Review of Sociology, 29,* 417–442.

Kawachi, I., Kennedy, B. P., & Glass, R. (1999). Social capital and self-rated health: A contextual analysis. *American Journal of Public Health, 89,* 1187–1193.

Kessler, R. C. (1979). Stress, social status, and psychological distress. *Journal of Health and Social Behavior, 20,* 259–273.

Kessler, R. C., McGonagle, K., Zhao, S., Nelson, C., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51,* 8–19.

Kessler, R. C., & Neighbors H. W. (1986). A new perspective on the relationships among race, social class and psychological distress. *Journal of Health and Social Behavior, 27,* 107–115.

Kirschbaum, C., Wust, S., & Strasburger, C. J. (1992). 'Normal' cigarette smoking increases free cortisol in habitual smokers. *Life Sciences, 50,* 435–442.

Koob, G. F., Roberts, A. J., Schultheis, G., Parsons, L. H., Charles, J. H., Hyytia, P., et al. (1998). Neurocircuitry targets in ethanol reward and dependence. *Alcoholism: Clinical and Experimental Research, 22,* 3–9.

Krieger, N., (1990). Racial and gender discrimination: Risk factors for high blood pressure? *Social Science and Medicine, 30,* 1273–1281.

Krieger, N., & Signey, S. (1996). Racial discrimination and blood pressure: The CARDIA study of young black and white adults. *American Journal of Public Health, 86,* 1370–1378.

Krieger, N., Williams, D. R., & Moss, N. (1997). Measuring social class in U.S. public health research: Concepts, methodologies, and guidelines. *Annual Review of Public Health, 18,* 341–378.

Le, A. D., Quan, B., Juzytch, W., Fletcher, P. J., Joharchi, N., & Shaham, Y. (1998). Reinstatement of alcohol-seeking by priming injections of alcohol and exposure to stress in rats. *Psychopharmacology, 135,* 169–174.

Lender, M. B. (1987). Alcohol, stress and society: The 19th century origins of the tension reduction hypothesis. In E. Gottheil, K. A. Druley, S. D. Pashko, & S. P. Weinstein (Eds.), *Stress and addiction* (pp. 13–23). New York: Brunner/Mazel.

Littell, R. C., Milliken, G. A., Stroup, W. W., & Wolfinger, R. D. (1996). *SAS system for MIXED models.* Cary, N.C.: SAS Institute, Inc.

Magee, W. J., Eaton, W. W., Wittchen, H., McGonagle, K. A., & Kessler, R. C. (1996). Agoraphobia, simple phobia, and social phobia in the National Comorbidity Survey. *Archives of General Psychiatry, 53,* 159–168.

Manning, R. (1998) A care approach. In H. Kuhse, & P. Singer (Eds.), *A companion to bioethics* (pp. 512). Oxford: Blackwell: 98–105.

Marinelli, M., & Piazza, P. V. (2002). Interaction between glucocorticoid hormones, stress and psychostimulant drugs. *European Journal of Neuroscience, 16,* 387–394.

Marmot, M., & Wilkinson, R. G. (1999). *Social determinants of health.* New York: Oxford University Press.

Massey, D. S. (2004). Segregation and stratification: A biosocial perspective. *Du Bois Review , 1*(1), 7–25.

Masten, A. S., & Powell, J. L. (2003). A resilience framework for research, policy and practice. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 1–25). Cambridge, MA: Cambridge University Press.

McCarthy, J., & Yancey, W. (1971). Uncle Tom and Mr. Charlie: Metaphysical pathos in the study of racism and personal disorganization. *American Journal of Sociology, 76,* 648–672.

McEwen, B. S. (1998a). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338,* 171–179.

McEwen, B. S. (1998b). Stress, adaptation and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840,* 33–44.

McLean, D. E., & Link, B. G. (1994). Unraveling complexity: Strategies to refine concepts, measures, and research designs in the study of life events and mental health. In W. R. Avison & I. H. Gotlib (Eds.), *Stress and mental health: Contemporary issues and prospects for the future* (pp. 15–42). New York: Plenum Press.

Messer, N. G. (2004). Professional-patient relationships and informed consent. *Postgraduate Medical Journal, 80,* 277–283.

Miles, T. P. (2005). Correctable sources of disparities in cancer among minority elders. *Medical Clinics of North America, 89,* 869–894.

Mirowsky, J., & Ross, C. (1980). Minority status, ethnic culture, and distress: A comparison of Blacks, Whites, Mexicans and Mexican-Americans. *American Journal of Sociology, 86,* 479–495.

Mirowsky, J., & Ross, C. E. (1989). *Social causes of psychological distress.* New York: Aldine de Gruyter.

Morland, K., Wing, S., & Roux, A. D. (2002). The contextual effect of the local food environment on residents' diets: The atherosclerosis risk in communities study. *American Journal of Public Health, 92,* 1761–1767.

Morland, K., Wing, S., Roux, A. D., & Poole, C. (2002). Neighborhood characteristics associated with the location of food stores and food service places. *American Journal of Preventive Medicine, 22,* 23–29.

Mueller, P. S., Hook, C. C., & Fleming, K. C. (2004). Ethical issues in geriatrics: A guide for clinicians. *Mayo Clinic Proceedings, 79,* 554–562.

Myers, H. F. (1982). Stress, ethnicity, and social class: A model for research with black populations. In E. E. Jones & S. J. Korchin (Eds.), *Minority mental health* (pp. 118–148). New York: Praeger.

Neff, J. (1985). Race and vulnerability to stress: An examination of differential vulnerability. *Journal of Personality and Social Psychology, 49,* 481–491.

Neighbors, H. W. (1984). The distribution of psychiatric morbidity in black Americans: A review and suggestions for research. *Community Mental Health Journal, 20,* 5–18.

Neighbors, H. W., Jackson, J. S., Bowman, P. J., & Gurin, G. (1983). Stress, coping and black mental health: Preliminary findings from a national study. *Prevention in Human Services, 2,* 5–29.

Neighbors, H. W., Jackson, J. S., Broman, C. L., & Thompson, E. (1996). Racism and the mental health of African Americans: The role of self and system blame. *Ethnicity & Disease, 6,* 167–175.

Neighbors, H. W., Jackson, J. S., Campbell, L., & Williams, D. (1989). The influence of racial factors on psychiatric diagnosis: A review and suggestions for research. *Community Mental Health Journal, 25(4),* 201–311.

Nisell, M., Nomikos, G. G., & Svensson, T. H. (1994). Systemic nicotine-induced dopamine release in the rat nucleus accumbens is regulated by nicotinic receptors in the ventral segmental area. *Synapse, 16,* 36–44.

Pearlin, L. I. (1989). The sociological study of stress. *Journal of Health and Social Behavior, 30,* 241–256.

Peele, S., & Brodsky, A. (2000). Exploring psychological benefits associated with moderate alcohol use: A necessary corrective to assessments of drinking outcomes. *Drug and Alcohol Dependence, 60,* 221–247.

Peyser, H. S. (1993). Stress, ethyl alcohol, and alcoholism. In L. Goldberger & S. Breznitz (Eds.), *Handbook of stress: Theoretical and clinical aspects* (2nd ed., pp. 532–549). New York: The Free Press.

Piazza, P. V., & Le Moal, M. L. (1996). Pathophysiological basis of vulnerability to drug abuse: Role of an interaction between stress, glucocorticoids, and dopaminergic neurons. *Annual Review of Pharmacology and Toxicology, 36,* 359–378.

Piazza, P. V., & Le Moal, M. L. (1998). The role of stress in drug self-administration. *Trends in Pharmacological Science, 19,* 67–74.

Pohorecky, L. A. (1981). The interaction of alcohol and stress. *Neuroscience and Behavioral Reviews, 5,* 209–229.

Pohorecky, L. A. (1991). Stress and alcohol interactions: An update of human research. *Alcoholism: Clinical and Experimental Research, 15,* 438–458.

Pomerleau, C. S., & Pomerleau, O. F. (1992). Euphoriant effects of nicotine in smokers. *Psychopharmacology, 108,* 460–465.

Porcu, P., Sogliano, C., Cinus, M., Purdy, R. H., Biggio, G., & Concas, A. (2003). Nicotine-induced changes in cerebrocortical neuroactive steroids and plasma corticosterone concentrations in the rat. *Pharmacology, Biochemistry, and Behavior, 74,* 683–690.

Pritchard, W. S., Robinson, J. H., & Guy, T. D. (1992). Enhancement of continuous performance task reaction time by smoking in non-deprived smokers. *Psychopharmacology, 108,* 437–442.

Raikkonen, K., Keltikangas-Jarvinen, L., Adlercreutz, H., & Hautanen, A. (1996). Psychosocial stress and the insulin resistance syndrome. *Metabolism: Clinical and Experimental, 45,* 1533–1538.

Robins, L., & Regier, D. (1991). *Psychiatric disorders in American: The Epidemiologic Catchment Area Study.* New York: The Free Press.

Rogler, L. H., Malgady, R. G., & Rodriguez, O. (1989). *Hispanics and mental health: A framework for research.* Malabar, Florida: Robert E. Krieger Publishing.

Rosenberg, M. (1979). *Conceiving the self.* New York: Basic Books.

Roux, A. D. (2003). Residential environments and cardiovascular risk. *Journal of Urban Health: Bulletin of the New York Academy of Medicine, 80,* 569–589.

Rutter, M. (1985). Resilience in the face of adversity. Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry, 147,* 598–611.

Rutter, M. (1993). Resilience: Some conceptual considerations. *Journal of Adolescent Health, 14,* 626–631, 690–626.

Ryff, C. D., & Keyes, C. L. (1995). The structure of psychological well-being revisited. *Journal of Personality and Social Psychology, 69,* 719–727.

Saldana, D. H. (1995). Acculturative stress. In D. H. Saldana (Ed.), *Hispanic psychology: Critical issues in theory* (pp. 43–56). New York: Sage Publications.

Salgado de Snyder, V. N. (1987). Factors associated with acculturative stress and depressive symptomatology among married Mexican immigrant women. *Psychology of Women Quarterly, 11,* 475–488.

Sapolsky, R. M. (1998). *Why zebras don't get ulcers: An updated guide to stress, stress-related diseases and coping* (2nd ed.). New York: W. H. Freeman & Company.

Sapolsky, R. M. (2002). Endocrinology of the stress-response. In J. B. Becker, S. M. Breedlove, D. Crews, & M. M. McCarthy (Eds.), *Behavioral Endocrinology* (pp. 409–450). Cambridge, MA: Massachusetts Institute of Technology Press.

Schnittker, J., & McLeod, J. D. (2005). The social psychology of health disparities. *Annual Review of Sociology, 31,* 75–103.

Snyder, C. R. (2002). Hope theory: Rainbows of the mind. *Psychological Inquiry, 13,* 249–275.

Somervell, P. D., Leaf, P. J., Weissman, M. M., Blazer, D. G., & Bruce, M. L. (1989). The prevalence of major depression in black and white adults in five United States communities. *American Journal of Epidemiology, 130,* 725–735.

Stewart, J. (2000). Pathways to relapse: The neurobiology of drug- and stress-induced relapse to drug-taking. *Journal of Psychiatry and Neuroscience, 25,* 125–136.

Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *Proceedings of the National Academy of Sciences of the United States of America, 102,* 6508–6512.

Stolerman, I. P., & Jarvis, M. J. (1995). The scientific case that nicotine is addictive. *Psychopharmacology, 117,* 2–10.

Taylor, R. J., Chatters, L. M., Hardison, C. B., & Riley, A. (2001). Informal social support networks and subjective well-being among African Americans. *Journal of Black Psychology, 27,* 439–463.

Thoits, P. A. (1995). Stress, coping and social support processes: Where are we? What next? *Journal of Health and Social Behavior, (Extra Issue),* 53–79.

Thompson, C. E. & Neville, H. A. (1999). Racism, mental health, and mental health practice. *The Counseling Psychologist, 27,* 155–223.

Turner, R. J., Wheaton, B., & Lloyd, D. A. (1995). The Epidemiology of Social Stress. *American Sociological Review, 60,* 104–125.

U.S. Department of Health and Human Services. (2001). *Mental health: Culture, race, and ethnicity – A supplement to mental health: A report of the Surgeon General.* Rockville: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services.

Vega, W. A., & Rumbaut, R. G. (1991). Ethnic minorities and mental health. *Annual Review of Sociology, 17,* 351–383.

Walsh, S. H. (2004). The clinician's perspective on electronic health records and how they can affect patient care. *BMJ (Clinical Research Ed.), 328,* 1184–1187.

Ware, J. E., Jr., & Sherbourne, C. D. (1992). The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Medical Care, 30,* 473–483.

Ulbrich, P., Warheit, G., & Zimmerman, R. (1989). Race, socioeconomic status and psychological distress. *Journal of Health and Scoial Behavior, 30,* 131–146.

Warheit, G., Holzer, C., & Arey, S. (1975). Race and mental illness: An epidemiologic update. *Journal of Health and Social behavior, 16,* 243–256.

Warner, D. F., & Hayward, M. D. (2002, May). *Race disparities in men's mortality: The role of childhood social conditions in a process of cumulative disadvantage.* Presented at the Population Association of American Annual Meeting, Atlanta, Georgia.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063–1070.

Wesnes, K., & Warburton, D. M. (1983). The effects of cigarettes of varying yield on rapid information processing performance. *Psychopharmacology, 82,* 338–342.

Wheaten, B. (1985). Models for the stress-buffering functions of coping resources. *Journal of Health and Social Behavior, 26,* 352–364.

Wheaton, B. (1994). Sampling the stress universe. In W. R. Avison & I. H. Gotlib (Eds.), *Stress and mental health: Contemporary issues and prospects for the future* (pp. 77–114). New York: Plenum Press.

Wilkinson, R. G., & Pickett, K. E. (2005). Income inequality and population health: A review and explanation of the evidence. *Social Science & Medicine, 62,* 1768–1784.

Williams, D. R. (1990). Socioeconomic differentials in health: A review and redirection. *Social Psychology Quarterly, 53,* 81–99.

Williams, D. R. (1994). The concept of race in Health Services Research: 1966–1990. *Health Services Research, 29*, 261–274.

Williams, D. R. (1995). African American mental health: Persisting questions and paradoxical findings. In R. J. Taylor (Ed.), *African American research perspectives, An Occasional Report of the Program for Research on Black Americans, Ann Arbor: University of Michigan Institute for Social Research. 2(1):8–16.*

Williams, D. R. (1996). Racism and health: A research agenda. *Ethnicity and Disease, 6*, 1–6.

Williams, D. R. (2001). Racial variations in adult health status: Patterns, paradoxes, and prospects. In N. J. Smelser, W. J. Wison, & F. Mitchell (Eds.), *America Becoming: Racial trends and their consequences* (Vol. 2, pp. 371–410). Washington, D.C.: National Academy Press.

Williams, D. R., & Chung, A-M. (in press). Racism and health. In R. C. Gibson & J. S. Jackson (Eds.), *Health in Black America.* Thousand Oaks, CA: Sage Publications.

Williams, D. R., & Collins, C. (1995). Socioeconomic and racial differences in health. *Annual Review of Sociology, 21,* 349–386.

Williams, D. R., & Fenton, B. (1994). The mental health of African Americans: Findings, questions, and directions. In I. L. Livingston (Ed.), *Handbook of Black American health: The mosaic of conditions, issues, policies, and prospects* (pp. 253–268). Westport, CT: Greenwood Publishing.

Williams, D. R. & Jackson, P. B. (2005). Social sources of racial disparities in health: Policies in societal domains, far removed from traditional health policy, can have decisive consequences for health. *Health Affairs, 24*, 325–334.

Williams, D. R., Lavizzo-Mourey, R., & Warren, R. C. (1994). The concept of race and health status in America. *Public Health Reports, 109,* 26–41.

Williams, D. R., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology, 2*, 335–351.

Wilson, W. J. (1996). *When work disappears: The world of the new urban poor.* New York: Knopf.

Winkleby, M. A. & Cubbin, C. (2004). Racial/ethnic disparities in health behaviors: A challenge to current assumptions. In N. B. Anderson, R. A. Bulatao, & B. Cohen (Eds.), *Critical Perspectives on racial and*

ethnic disparities in health in later life (pp. 171–226). Washington, DC: National Research Council.

Zautra, A. J. (2003). *Stress, emotions and health.* New York: Oxford University Press.

Zautra, A. J., Guarnaccia, C. A., & Dohrenwend, B. P. (1986). Measuring small life events. *American Journal of Community Psychology, 14,* 629–655.