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The Speedometer of Life: Stress, Health and Aging

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CHAPTER CONTENTS

INTRODUCTION

The study of stress has long played an important role in the understanding of adult development and health. Over 50 years ago, Hans Selye wrote in the final pages of *The Stress of Life*: "Stress is the sum of all of the wear and tear caused by any kind of vital reaction throughout the body at any one time. That is why it can act as a common denominator of all of the biologic changes which go on in the body; it is a kind of 'speedometer of life'" (1956, p. 274). Indeed, since the pioneering work of Cannon (1932) and Selye (1956), research has focused on the biological and physiological effects of stressor exposure. Biological responses elicited by physical and psychological stressors include the release of catecholamines (e.g., norepinephrine, epinephrine release from the adrenal medulla), sympathetic arousal (e.g., increased blood pressure, elevated heart rate), and hypothalamic-pituitary-adrenal (HPA) axis activation (e.g., cortisol secretion from the adrenal cortex; see Klein & Corwin, 2007; Stratakis & Chrousos, 1995; Taylor et al., 2000 for reviews). Chronic exposure to these stress hormones leads to accumulated wear and tear on the body, referred to as "allostatic load" (McEwen & Stellar, 1993), which, in turn, contributes to an increased risk of illness and mortality (Kiecolt-Glaser et al., 2002). As such, the study of stress and aging are closely aligned. The purpose of this chapter is to describe the links between stress, health, and aging; to identify stress processes that lead to changes in key psychological and physiological indicators; and to examine the associations between biological indicators, health, and well-being.

Our view is grounded in a life-span developmental theory of stress and health that highlights variations and differences within and between individuals as they develop in multidimensional sociohistorical contexts (Baltes et al., 1999; Spiro, 2001). This theory posits that life experiences accumulate throughout the life course, with each stage of life reflecting both the consequences of prior experiences, as well

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as the antecedents for subsequent life experiences. In this chapter we provide a broad framework for incorporating the role of stress into the study of health and aging by describing: (1) concepts and measurement of stress processes, (2) life-span and life course perspectives on stressors and their consequences, (3) biomarkers that provide a link between stressors and health, and (4) emerging research documenting the effects of stress on cognitive health.

CONCEPTS AND MEASUREMENT OF STRESS PROCESSES

There are two prominent ways to think about the links between stress, health, and aging. One approach focuses on the molar impact of major life changes, and is referred to as the life event tradition (e.g., Dohrenwend, 2006; Holmes & Rahe, 1967). This approach examines the discrete, observable, and objectively reportable life changes that are relatively rare (e.g., divorce or job loss) and require significant adjustment on the part of the individual (Wheaton, 1997). The other approach takes a microscopic dynamic approach to stress processes, with a focus on the accumulation of daily stressors (Bolger et al., 2003; Pearlin, 1999). A growing body of research suggests that it is the myriad of these everyday events, or quotidian stressors (Almeida, 2005; Pearlin & Skaff, 1996), rather than the major, but less frequent, life events that most severely impact individual well-being (Lazarus & Folkman, 1984; Pearlin, 1982).

Quotidian stressors are divided into two classes: chronic stressors and daily hassles. Chronic stressors are the persistent or recurrent difficulties of life. One source of chronic stressors is the strains associated with the enactment of certain social roles (Pearlin, 1982, 1999; Pearlin et al., 1981; Wheaton, 1996), such as caregiving for a sick spouse. Another source of chronic stressors may arise from conflicting social roles experienced by individuals, such as being a working parent (Pearlin, 1999). Irrespective of the origin, there is agreement that it is the ongoing and open-ended nature of the stressor that qualifies it as chronic. Furthermore, it is often difficult to identify when the chronic stressor began and when — or even if — it will end (Wheaton, 1996, 1999).

In contrast to chronic stressors, daily hassles are defined as relatively minor events arising from day-to-day living, such as spousal conflict, specific caregiving activities, and work deadlines (Almeida, 2005). An emerging literature has shown that day-to-day stressors play an important part in health and emotional adjustment (Zautra, 2003). Daily stressors represent tangible, albeit minor interruptions that tend to have a more proximal effect on well-being than do major life events. In terms of their physiological and psychological effects, reports of life events may be associated with prolonged arousal, whereas reports of daily stressors may be associated with spikes in arousal or psychological distress during a particular day (Almeida, 2005). Minor daily stressors exert their influence through separate and immediate direct effects on emotional and physical functioning, and by piling up over a series of days to create persistent irritations, frustrations, and overloads that increase the risk of serious stress reactions, such as anxiety and depression (Lazarus, 1999; Pearlin et al., 1981; Zautra, 2003).

Many studies have established an association between health and life events (Dohrenwend, 2006; Brown & Harris, 1978), chronic stressors (Bolger et al., 1996; Eckenrode, 1984; Pillow et al., 1996), and daily hassles (Almeida, 2005; Bolger et al., 1989; Folkman et al., 1987). More recent work has extended this research by examining the linkages between these multiple dimensions of stress and their combined effect on psychological and physical health. Research that includes multiple types of stressors, such as those combining both life events, chronic stressors, and daily hassles, suggests that it is the interaction of stressors and the accumulation over time that leads to poor health outcomes, rather than the immediate and unique effects of a single stressor or type of stressor (Almeida & Wong, 2009; Chiriboga, 1997; Pearlin et al., 1981; Serido et al., 2004; Turner et al., 2000). Prior to being able to make the linkages between stress and health, however, it is necessary to understand developmental processes in stressor exposure and reactivity.

STRESSOR PATHWAYS TO HEALTH AND WELL-BEING

There are two primary pathways through which stressors impact well-being: stressor exposure and stressor reactivity. Stressor exposure is the likelihood that an individual will experience a stressor based on combinations of resilience and vulnerability factors. Experiencing stressors is not simply a matter of chance or bad luck; rather differences in stressor exposure more often emerge from individual sociodemographic, psychosocial, and situational factors (Pearlin, 1993a, 1999; Wheaton, 1997, 1999). There is substantial evidence that stable sociodemographic, psychosocial, and situational factors, such as age (Aldwin, 1990, 1991; Almeida & Horn, 2004; Hamarat et al., 2001), personality (Bouchard, 2003; Penley & Tomaka, 2002), and social support (Brewin et al., 1989; Felsten, 1991) play a significant role in differences in stressor frequency, content, and appraisal. The stressor exposure path contends that stressors and appraisals emerge from a combination

of factors that in turn lead to changes in both self-reports and physiological markers of well-being.

Reactivity is the likelihood that an individual will show emotional or physical reactions to the stressors he or she encounters (Almeida, 2005; Bolger & Zuckerman, 1995; Cacioppo, 1998). In this sense, stressor reactivity is not defined as well-being (i.e., negative affect or physical symptoms), but is operationally defined as the within-person relationship between stressors and well-being. Reactivity, therefore, is a dynamic process that links stressors and well-being over time. Previous research shows that people who are more reactive to daily stressors are more susceptible to physical disease than are people who are less reactive (Cacioppo et al., 1998). Because resources of individuals and their environments (e.g., education, income, chronic stressors) limit or enhance possibilities and choices for coping (Lazarus, 1999), reactivity to stressors is likely to differ across people and across situations. The stressor reactivity path therefore contends that sociodemographic, psychosocial, and other situational factors modify reactivity to daily stressors. To this end, researchers can examine whether the same stressors produce different responses in various people, whether the same person responds differently to disparate stressors, and whether these responses vary according to self-report and physiological measures. A primary purpose of this chapter is to consider how these stress processes inform and are informed by research on aging. The next section articulates how stressor exposure and reactivity changes throughout adulthood, followed by a section on how stress plays an important role in biological and cognitive aging.

CHANGES IN STRESS PROCESSES ACROSS MIDDLE AND LATER ADULTHOOD

One vital component of our life-span perspective is to track intra-individual changes in exposure and reactivity to stressors. Several theoretical perspectives predict *decreases in stressor exposure and reactivity* across adulthood. One set of theories posits that with age, adults exercise more control over their social interactions and avoid situations that are stressful. According to Socioemotional Selectivity Theory (SST; Carstensen, 1987; Carstensen et al., 1999), for example, goals shift across adulthood, from a focus on knowledge-based goals during youth to a focus on emotion-based goals in later adulthood. As a consequence of these shifting goals, the composition of social networks changes, such that young adults broaden the size of their networks, whereas older adults strive to maintain their existing, close networks. These shifting social networks have implications for stressor exposure and reactivity. For example, daily experiences for younger adults may involve meeting new people and putting oneself in novel situations. As people age, however, their daily interactions become more predictable. These differences are reflected in the types of stressors people encounter. Compared to younger adults, older adults report fewer stressors overall and fewer overload stressors (i.e., meeting many demands). They do, however, experience more network stressors (i.e., events occurring to a network of friends and relatives that are experienced vicariously by the individual; Almeida & Horn, 2004). From this perspective, age-related changes in the stress process may emanate from differences in the frequency and type of daily stressors experienced across adulthood.

In a related vein, some theorists argue that adults become less reactive to stressors with age because of the knowledge, resources, and understanding they have accumulated from prior experiences (Whitbourne, 1985, 1986). This adaptation perspective suggests that it is the individual's evaluation of life experience rather than the experience itself that changes across adulthood. Simply stated, when exposed to daily stressors, older adults may interpret their experiences differently based on previous life experiences. Diehl et al. (1996), for example, found that older adults display greater impulse control than do younger adults when dealing with stressors, suggesting that as people age they may cope better with stress and perhaps become less reactive to stressors. These findings are consistent with a growing body of literature suggesting that people regulate emotions more effectively with age. Lawton (1996) suggested that repetition of negative affect states over many years may decrease the likelihood of triggering such states in the future. Such increases in the threshold for experiencing negative affect due to repeated activation are known as "dampening" effects. Certain life-span theories of emotion regulation are also consistent with the idea of lessened emotional reactivity to stress with age (Labouvie-Vief & DeVoe, 1991; Lang et al., 1998). In sum, the association between stress and well-being may shift throughout the life span as a result of reduced stressor exposure with age, reduced stressor reactivity, or a combination of both.

An alternative perspective suggests that the same process (i.e., repeated exposure to stressful events) may actually *increase stressor reactivity with age* in certain people (Mroczek & Almeida, 2004; Sliwinski et al., 2009). In this view, repeated activation may lead to sensitization as opposed to habituation or dampening. Several theoretical perspectives give rise to this idea. Changes in the aging brain alter the way people experience emotion, especially negative affect. The structures that mediate negative affect, the amygdala and limbic system, become more sensitive as we age (Adamec, 1990; Panksepp & Miller, 1996) as does the HPA axis (Seeman et al., 2001). Such heightened sensitivity may

lead to an easier activation of the neural substrates that underlie the experience of negative affect when a stressor is encountered. These neurophysiologic changes make it conceivable that negative affect is more likely to become activated as a consequence of frequent activation. Thus, due to a lifetime of repeated activations of the neural systems that mediate negative affect, reactivity to stressors may increase as people age.

These heightened sensitivities are akin to kindling effects, a process in which repeated exposure to some stimulus causes sensitization (Kendler et al., 2001; van der Kolk, 1997; Woolf & Costigan, 1999). It is believed that kindling effects are a result of neuroplasticity, which refers to the ability of neurons to change and realign themselves in response to repeated exposure. The neural network that governs some processes (e.g., the sensation of pain, an epileptic seizure, and feelings of depression or negative affect) can itself become molded by the stimulus, causing these networks to become even more sensitive to the stimulus (van der Kolk, 1997; Woolf & Costigan, 1999). We suggest that a similar process might be occurring in the affective response to stressful events, especially among individuals who view stressors in a negative light. Indeed, some of our prior work has shown that older adults high in neuroticism display the greatest reactivity to stressors (Mroczek & Almeida, 2004; Mroczek et al., 2006, Sliwinski et al., 2009).

The Theory of Strength and Vulnerability Integration

The theory of Strength and Vulnerability Integration (SAVI) attempts to explain why some research shows increased reactivity to stressors with age and other research shows age-related decreases (Chapter 19, this volume; Charles & Piazza, 2009). According to SAVI, later adulthood is comprised of both strengths and vulnerabilities that affect emotion regulation. Strengths include the motivation to maintain affective well-being due to a shift in time perspective (as described in SST) and the cognitive-behavioral skills to do so, which are acquired over a lifetime of dealing with difficult experiences. These strengths, however, are coupled with age-related physiological vulnerabilities that make it difficult to recover once a stressor has been experienced. Thus, although age may bring with it an improved ability to avoid stressors and/or reappraise them as being innocuous, it also brings with it the vulnerabilities that occur as a result of a less flexible physiological system. For this reason, SAVI posits that age-related changes in emotion regulation are least likely to occur during exposure to a stressor that creates high levels of physiological arousal. At times such as these, physiological cues will largely override emotion regulation strategies. As time from the stressor passes, however, and physiology normalizes, age differences in emotional experience will increase because people's assessments of their emotional states will be less influenced by their physiological experience and more influenced by their appraisals of an event. Although this is hypothesized to be the normative pattern of age-related strengths and vulnerabilities, some life experiences make it difficult to avoid stressors or to modify one's appraisals of an event. Such experiences include loss of social belonging; exposure to chronic, uncontrollable stressors; and neurological dysregulation. Under these circumstances, age-related gains in affective well-being are unlikely to occur, regardless of the time that has passed since the emotion-eliciting event. Thus, although the motivation to maintain affective well-being exists, certain circumstances may impinge upon older adults' ability to do so (Chapter 19, this volume; Charles & Piazza, 2009).

INDIVIDUAL DIFFERENCES IN STRESS PROCESSES

While it is important to document general age trends in daily stress processes, life-span developmental theory asserts that aging does not represent a monolithic predictable trajectory (e.g., Baltes & Baltes, 1990; Rowe & Kahn, 1987, 1997). Thus, researchers should assess group and individual differences in patterns of change in stress processes as a function of resilience and vulnerability factors. Although there is emerging research documenting how factors such as SES, personality, perceived control, and genetic endowment predict exposure and reactivity to daily stressors concurrently, future research should extend this line of inquiry by assessing how stable and changing characteristics of individuals predict changes in stress processes.

Several researchers have asserted that *personal dispositions* interact with stressful situations in determining stressful experiences (Ben-Porath & Tellegen, 1990; Costa et al., 1996; Watson, 1990). For example, people who report high levels of neuroticism also report greater overall psychological distress. However, neuroticism is also associated with elevated distress and negative affect in the context of specific stressful situations (e.g., an argument with a spouse or boss). This tendency is referred to as hyperreactivity (Bolger & Schilling, 1991; Suls, 2001), and is perhaps the central theoretical 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 do those low in this trait (Bolger & Schilling, 1991; Bolger & Zuckerman, 1995; David & Suls, 1999; Gunthert et al., 1999).

The repercussions of hyperreactivity, which refers to an inability (or lessened ability) to regulate back to a more optimal emotional state, are best understood in a developmental framework. In hyperreactivity, the state of distress remains continually activated, which is detrimental if it occurs too frequently, as it does among people high in neuroticism. Kendler et al. (2001) and Wilson et al. (2003, 2004) have hypothesized that neuroticism, and the constant elevated levels of distress that accompany this 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 age, they become more, not less, susceptible to elevated distress. In theoretical terms, earlier experiences influence later experiences, accumulating in a manner suggested by Spiro (2001). This developmental accumulation may potentially result in greater hyperreactivity among certain older adults, namely those who have remained high in neuroticism over time. A study examining the long-term effect of neuroticism on physical health supports the hyperreactivity hypothesis (Charles et al., 2008). In this study, neuroticism was assessed among twin pairs who ranged in age from 15 to 47 in 1973. These individuals were contacted again 25 years later and asked about their physical health conditions. Results indicated that even when controlling for familial influences, those individuals who had a history of neuroticism had a higher likelihood of reporting a physical condition during the follow-up interview. Neuroticism, therefore, appears to have long-lasting physiological consequences.

Socioeconomic factors are also hypothesized to shape patterns of change in stress processes. Individuals with lower levels of socioeconomic status (SES) are at increased risk for major stressful events and chronic difficulties (e.g., violence, discrimination) and are thus more likely to suffer distress (Dohrenwend, 1970, 1973; Ettner, 2000; Marmot et al., 1997; Myers et al., 1972). One mechanism for this association is that lower SES individuals are more emotionally vulnerable to major stressors (Brown & Harris, 1978; Kessler & Cleary, 1980). Our work has shown that this is true for day-to-day stressors as well (Almeida et al., 2005; Grzywacz et al., 2004). On days individuals reported stressors (e.g., arguments, work demands), those with lower levels of education experienced greater psychological distress than did their better educated counterparts. Our cross-sectional analysis found no age differences (from 25 to 74 years) in this effect, but longitudinal analysis would permit testing longer-term effects of this differential reactivity. Over time, for example, people with lower SES may exhibit less optimal affective and physical functioning compared to people with higher SES, a premise consistent with findings of greater morbidity and mortality associated with each incremental decrease in SES (Marmot et al., 1997). In addition, not all people with low SES display poor affective and physical functioning, nor do all people with high SES do well. Using longitudinal data, researchers could examine the wide variability in the profiles of physical and mental health, focusing on people with high levels of functioning despite their lack of access to education, income, and other advantages that accompany higher SES.

Race/ethnicity also plays an important role in stress processes. For example, African Americans are at greater risk for morbidity and mortality compared to European Americans. Although SES and access to health care certainly play a role in these differences (Wagner, 1998), they do not explain why African Americans of the same SES as European Americans tend to be less healthy. Researchers suggest that several factors play a role, including a higher prevalence of risky health behaviors, environmental stressors, and genetic vulnerabilities. In addition, societal factors such as racial discrimination may also result in different health trajectories for African Americans. African American men, for example, are at higher risk for hypertension and experience more rapid progression of end-organ damage from hypertension than European American men (Wagner, 1998), which may be indicative of cardiovascular hyperactivity (Anderson, 1989). In one study, for example, African Americans who attributed mistreatment to discrimination exhibited higher than average diastolic blood pressure reactivity, an effect that was not evident among European Americans. African Americans also exhibited larger increases in mean arterial blood pressure in threat conditions compared to European Americans. Moreover, after exposure to racist stimuli, diastolic blood pressure levels remained elevated, suggesting that even indirect exposure to discrimination elicits significant and long-lasting reactivity (Fang & Myers, 2001). Racial discrimination may thus be a chronic stressor that negatively impacts the cardiovascular health of African Americans through pathogenic processes associated with physiological reactivity (Guyll et al., 2001). Future stress research should investigate linkages between daily psychosocial stressors (e.g., perceived discrimination, perceived inequalities) and biological health (e.g., allostatic load).

Life transition also contributes to stress processes. Changes in stress processes may also be reflected in the timing and transitions into and out of social roles, including role changes in the family and work domains (Almeida & Wong, 2009). These role changes may be precipitated by grown children leaving home (Lowenthal et al., 1975), career transitions (Moen, 2003), and the renegotiation of family relationships (Blatter & Jacobsen, 1993; Kim & Moen, 2002). Role transitions often entail transformations in multiple domains of responsibilities, such as combining work responsibilities and caretaking for one's

aging parents and/or children. Such roles expose adults not only to specific types of major life events, but also to unique daily stressors. For example, in some of our work we examine how timing of retirement shapes daily experiences (Wong & Almeida, 2005, 2006). Although the average retirement age in the United States is rising and there are older workers in the labor force (Bureau of Labor Statistic, 2008), the question of whether delayed retirement is beneficial to the psychological and physical well-being of older workers requires further examination (Moen, 2003). Our analyses indicate that older workers (age 65 and older) report higher levels of daily negative affect as well as greater stressor severity compared to younger workers, and younger and older retirees (Wong & Almeida, 2006). Older workers' reports of higher levels of daily negative mood and greater stressor severity were not attributed to a greater number of total stressors experienced. Rather, older workers appraised stressors as more disruptive and unpleasant than did younger workers and retirees. The area where older workers appraised stressors as having the greatest impact was in the way other people felt about them. These findings suggest that older workers may face issues of discrimination not simply in the work force but in other areas of their lives. It is important to note that these findings were based on a cross-section of workers, and it is necessary to examine social roles longitudinally to assess whether and for whom changes in role status coincide with changes in stress processes.

PHYSIOLOGY OF THE STRESS RESPONSE AND AGE-RELATED CHANGES

Our review thus far has focused on how stressors may influence physical health and how these processes change throughout adulthood. Next, we briefly review two physiological systems that are involved in the stress response: the sympathetic-adrenal-medullary (SAM) axis and the HPA axis. We also discuss normative age-related changes in these two systems and how exposure to psychological stressors may accelerate these changes.

The SAM Axis

Physiological adaptation to stressors is essential for survival. The stress response mobilizes the body's resources to ward off or run from a potential threat, assists in wound repair during times of injury, and motivates an organism during times of challenge (Segerstrom & Miller, 2004). After a stressor is perceived, it is the immediate action of the sympathetic (SNS) branch of the autonomic nervous system that enables an organism to fight or flee. The SNS stimulates the adrenal medulla and the sympathetic neurons to secrete the catecholamines epinephrine and norepinephrine into the blood stream; the process where by the SNS enervates the adrenal medulla to release epinephrine and norepinephrine is known as the SAM axis. Epinephrine and norepinephrine prepare the body against threat by increasing heart rate, blood pressure and perspiration, dilating the pupils and bronchioles, and inhibiting gastrointestinal activity. Although SAM-axis arousal is necessary in the context of acutely stressful situations, continual activation of this system may ultimately arise in damage to the organism. For this reason, cessation of the stress response is imperative to physical health (McEwen & Stellar, 1993).

Age, Exposure to Stressors, and the SAM Axis

There are a number of age-related changes in SAM-axis activity (for a review, see Crimmins et al., 2008), which are oftentimes magnified when an individual is exposed to stressors. After the age of 60, for example, systolic blood pressure tends to increase, whereas diastolic blood pressure tends to decrease (Franklin et al., 2001). Older age is associated with even greater increases in systolic blood pressure in the context of acute psychosocial stressors (Uchino et al., 1999). Age may also be associated with higher levels of norepinephrine (e.g., Barnes et al., 1983) and lower levels of epinephrine (e.g., Esler et al., 1995), both of which may increase in magnitude when older adults are exposed to stressors (Barnes et al., 1983; Ester et al., 1995), though not all studies show these trends (e.g., Lindheim et al., 1992). The associations between age, stress, and SAM-axis activation is further illustrated in a study examining reactivity to a laboratory stressor among spousal caregivers (Aschbacher et al., 2008). In this study, older adult caregivers had higher levels of plasma norepinephrine than did non-caregivers. Moreover, among caregivers, higher perceived role overload was related to higher levels of plasma norepinephrine during recovery from the stressor. Illnesses appear to compound these age differences. For example, resting heart rate is heightened among older adults with cardiovascular disease (Gillum et al., 1991). The associations between age, SAM-axis activity, and stressor exposure may therefore be most pronounced among people with preexisting physical conditions.

The HPA Axis

Stressor-induced SNS activation also stimulates the HPA axis, which begins with the release of corticotrophin-releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus. CRH stimulates

adrenocorticotropin hormone (ACTH) release from the anterior pituitary, and arginine vasopressin (AVP) from the posterior pituitary gland. AVP acts centrally to support the "fight-or-flight" response, whereas ACTH circulates to the adrenal cortex to simulate glucocorticoid release, including corticosteroids such as cortisol (Chrousos & Gold, 1992; for a review see Dickerson & Kemeny, 2004). Corticosteroids regulate HPA-axis function through a negative feedback loop by dampening further release of CRH and ACTH and, consequently, corticosteroids. This negative feedback loop is necessary; although adaptive in the short-term, prolonged and repeated activation of the HPA axis can lead to adverse health outcomes, including the development and progression of chronic disease, dampened immune response, and destruction of hippocampal neurons (McEwen, 1998; Sapolsky, 1996, 2000a,b, 2001).

Age, Exposure to Stressors, and the HPA Axis

There are several changes in the HPA axis that occur with age, both in terms of an altered diurnal pattern and a disruption of the negative feedback loop that essentially stems the tide of cortisol overproduction (for a review, see Epel et al., 2009). With age, for example, the diurnal pattern of cortisol remains relatively flat, with an attenuated awakening response (Almeida et al., 2009b) and a higher evening nadir (Van Cauter et al., 1996). Age is also associated with a greater HPA-axis response to stressors (e.g., Otte et al., 2005; Peskind et al., 1995). As with the SAM axis, psychological stress appears to accelerate the effects of aging on the HPA axis. For example, in a study examining distress and urinary cortisol levels among people of different ages, age was not associated with urinary cortisol levels among people reporting milder distress. Among people experiencing more severe distress, however, urinary cortisol levels increased with age (Jacobs et al., 1984). As with the SAM axis, the associations between age, stressor exposure, and the HPA axis may be strongest among people in poor health (McEwen, 1998).

GENDER DIFFERENCE IN RESPONSES TO STRESS: TEND AND BEFRIEND

Men and women also may differ in their responses to stress as they age. For example, both sexes have similar cortisol levels at rest, yet between puberty and menopause, the HPA-axis response during stress tends to be lower (as indexed by cortisol) in women compared to men (Kajantie & Phillips, 2006). It is hypothesized that this is the time when estrogen levels are higher for women, which may exert a protective effect on some aspects of health functioning (i.e., cardiovascular system), but could increase vulnerability to autoimmune illness. Interestingly, these sex differences may be dependent upon the nature of the stressor. For example, men respond more (increased cortisol) to achievement-oriented challenges whereas women respond more intensely to social rejection (Stroud et al., 2002).

A relatively new theory may help explain the underlying sex difference in reactivity to social stressors. Taylor and colleagues (2000) suggest that although both males and females display the traditional fight-or-flight response to some stressors, a behavioral pattern of "tend-and-befriend" might be a more adaptive response to some stressors for women. Designed to increase the likelihood of survival when faced with a threat, "tending-and-befriending" promotes safety and diminishes distress through creating and maintaining social networks (i.e., befriending), along with nurturing activities that protect the female and her offspring (i.e., tending). The underlying neurobiological system of the tend-and-befriend response differs from the fight-or-flight response in that it appears to be modulated by the posterior pituitary hormone oxytocin (Taylor et al., 2000), which may mediate many of the biobehavioral health effects of stress (e.g., Carter & Altemus, 1997; McCarthy, 1995; McCarthy & Altemus, 1997; Uvnas-Moberg, 1997), including reduced blood pressure (Light et al., 2000), perceived stress levels (Mezzacappa & Katlin, 2002), anxiety (Turner et al., 1999), aggression (Lubin et al., 2003), depression (Anderberg & Uvnas-Moberg, 2000), and, perhaps, improved attention and social memory (e.g., Brett & Baxendale, 2001; Ferguson et al., 2002).

With regard to aging, it is important to note that the effects of oxytocin are enhanced in the presence of estrogen (McCarthy, 1995). Thus, age-related declines in estrogen in women may result in a loss of the stress-buffering benefits of oxytocin and, perhaps, the tend-and-befriend response. In a study of post-menopausal women, Taylor et al. (2006) suggested that higher plasma oxytocin levels signal relationship stress and are associated with elevated cortisol levels. Klein and colleagues (Klein & Corwin, 2002; Klein et al., 2006) suggested that sex differences in stressor reactivity may make women particularly vulnerable to social stressors such as family, work, and social relationships. More specifically, women may be particularly vulnerable to the health consequences (e.g., depression) of social stressors ranging from social isolation and interpersonal conflict to romantic and marital relationships (e.g., Hammen, 2003a,b). Indeed, women are more vulnerable to developing classic signs of depression in response to stressful life events than are men (e.g., Maciejewski et al., 2001; Sherrill

et al., 1997; Weich et al., 2001). Unfortunately, how these sex differences in sensitivity to interpersonal stressors change across the life span is unknown. Our review of the literature suggests that age-related sex differences in tend-and-befriend responses to stress have not been studied and that this is a promising area of research.

STRESS AGE AND COGNITIVE HEALTH

Research has provided considerable evidence of a link between the experience of stressors including major trauma and negative life events, as well as daily hassles and a number of adverse health outcomes (Baum & Posluszny, 1999). An emerging area of inquiry complementing the study of stressors and their links to health and well-being is that of understanding how stressful experiences, as well as self-reported and biological indicators of stress, are linked to cognitive health, including cognitive performance and the structural and functional integrity of the brain. Furthermore, stress has been identified as particularly relevant for understanding cognitive health during old age because of age-related increases in vulnerability to stress (Smith, 2003).

In terms of linking stressful experiences to cognitive health, previous research has shown associations between different types of stressful experiences including major trauma, negative life events, chronic stressors, and daily hassles with compromised cognitive function. Major traumatic experiences including combat and childhood abuse have been associated with both impaired cognitive function and smaller hippocampal volume (Bremner, 1999; Sapolsky, 1996). Similarly, the chronic effects of these major traumatic events, specifically post-traumatic stress disorder (PTSD), has also been linked to impaired memory and attention function in combat veterans (Vasterling et al., 2002). Negative life events have also been adversely associated with cognitive function. Klein and her colleagues (Baradell & Klein, 1993; Klein & Barnes, 1994) showed that younger adults reporting greater numbers of negative life events exhibited poorer decision making, problem solving (Klein & Barnes, 1994), and working memory performance (Klein & Boals, 2001). Similar findings have been observed among older adults. Rosnick et al. (2007) found that older adults reporting greater numbers of stressful life events exhibited poorer memory performance, while Stawski et al. (2006) found that cognitive interference associated with the most stressful life event older adults self-identified was associated with poorer episodic memory, working memory, and processing speed performance. Chronic stressors, such as caregiving, have been shown to be associated with poorer processing speed (Caswell et al., 2003), as well as lower overall general cognitive function (Lee et al., 2004), among older adults. Finally, the experience of daily hassles has been linked to poorer working memory performance among both younger and older adults (Sliwinski et al., 2006), as well as self-reported memory failures in older adults (Neupert et al., 2006). Together, the results of these studies provide evidence that a wide variety of stressful experiences are associated with cognitive health.

The study of biological indicators of stress, as they relate to cognition, has primarily focused on cortisol, which is thought to impair cognitive function by binding to receptors in the hippocampus and frontal lobes and interfering with effective neural function and transmission. Cortisol is thought to have proximal effects on brain function and cognition by interfering with neural function and subsequent behavioral performance (Lupien & Lapage, 2001; Wolf, 2003; however also see Roozendaal, 2002), and more durable and distal effects by causing neuronal death from repeated and prolonged exposure to high levels of cortisol (Sapolsky, 1992; Sapolsky et al., 1986). Consistent with this biological perspective, research has shown that, in old age, higher basal cortisol levels and longitudinal increases in basal cortisol levels are associated with poorer episodic memory performance (Lupien et al., 1994, 1996; Seeman et al., 1997) and smaller hippocampal volume (Lupien et al., 1998). Similarly, McEwen's seminal work on the wear-and-tear hypothesis and the construct of allostatic load (McEwen, 1998) has also been important in motivating the understanding of the biological consequences of stress and their relation to cognitive and brain function. Allostatic load is a multiple-indicator (including cortisol) biological index of the cumulative effects of stressful experiences, permitting the consideration of how multidimensional, multisystemic biological indicators of stress can be used to understand cognitive health. Seeman et al. (2001) showed that higher levels of allostatic load were associated with the most precipitous seven-year declines in global cognitive function among older adults. Together, the results of these studies indicate that biological indicators of stress are predictive of cognitive and brain health.

Psychological indicators of stress, including psychological distress, proneness to distress, and stress-related cognitive interference have all been linked to cognitive performance. The role of these psychological indicators of stress and their link to cognitive function emerged from social psychological perspectives on the stress-cognition link, which suggests that stress competes for limited resources that can otherwise be devoted to information processing (Kahneman, 1973; Mandler, 1979; Wegner, 1988), and also serves to prolong physiological activation in response to stress (Brosschot et al., 2006). Work from Wilson and colleagues has shown that individual differences

in distress and proneness to distress are associated with lower levels of and more precipitous declines in cognitive function, particularly episodic memory performance in both healthy older adults (Wilson et al., 2006) and lower levels of episodic memory among older adults diagnosed with Alzheimer's disease (Wilson et al., 2004). In addition to links between distress and cognitive function, Wilson and colleagues have shown psychological distress and proneness to distress to be associated with an increased likelihood of being diagnosed with Alzheimer's disease, suggesting that proneness to distress may compromise the structural and functional integrity of the brain, rendering older adults more susceptible to age-related pathologies linked to dementia (Wilson et al., 2006, 2007).

Complementing the use of distress as a psychological indicator of stress, cognitive interference, defined as intrusive off-task thoughts and images, and the intentional suppression of such intrusions, has been employed as a useful predictor of cognitive function. Klein and Boals (2001) showed that stress-related cognitive interference, as measured by the Impact of Events Scale (Horowitz et al., 1979), was associated with poorer working memory performance among younger adults. Also using the Impact of Events Scale, Stawski et al. (2006) found that higher levels of stress-related cognitive interference were associated with poorer processing speed, working memory, and episodic memory.

Together, the small portion of the stress-cognition literature reviewed here presents a promising emerging field of research for understanding aging. There is fairly consistent evidence that the experience of discrete and specific stressful events is negatively associated with cognitive health, as are biological and psychological indicators of stress. There are, however, a number of challenges and opportunities that require attention. The existing research linking stressful experiences to cognitive function has not always included and identified relevant mechanisms for explaining the effect of stressor exposure on cognitive health. Similarly, biological and psychological indicators are proxies for exposure to stressors, without demonstrating that stressful experiences are antecedent factors. Finally, stress is a process, as is how stressful experiences are associated with cognitive function. Research linking stressful experiences to level and change in cognitive function, as well as the mechanisms responsible for these links, needs to be thoughtful regarding issues of the appropriateness of study design and the sampling of time for studying these phenomena (Neupert et al., 2008). For instance, the mechanisms linking daily stressors to daily fluctuations in cognitive function may not be the same as linking the chronic stressor of caregiving to longitudinal changes in cognitive function. The field of stress-cognitive health research is growing and is ripe for empirical inquiry. While many challenges for understanding stress-cognition linkages are on the horizon, these challenges present exciting opportunities for melding stress, health, and aging research with research on cognitive aging.

SUMMARY AND CONCLUSIONS

The overarching goal of this chapter was to provide a broad overview on how stress processes play a role in heath and aging. There are multiple dimensions of stressors ranging from major life events to chronic stressors to daily hassles. Adult developmental researchers have examined how stressors affect health by assessing intra-individual change in exposure and reactivity to these types of stressors. Research has also begun to document individual and group differences in changes in these stress processes. Much of the psychological literature on stress and aging has assessed affective reactivity, but emerging work has begun to incorporate biological as well as cognitive outcomes of stress processes. These are all important advances in understanding how stress may speed not only biological aging but also cognitive aging.

Of course the next step in this enterprise is to begin to develop interventions that target stress processes. For example, one intervention designed to reduce stress and its negative effects on health and well-being is the mindfulness-based stress reduction (MBSR) program (Kabat-Zinn, 1990). This program focuses on the cultivation of mindfulness (a form of meditation) and associated practices. Participation has been associated with decreases in medical symptoms (Carmody et al., 2008) and positive changes in brain and immune function in adults (Davidson et al., 2003). In addition, patients recently diagnosed with early stage breast cancer who took part in the MBSR program displayed improved coping, quality of life, and immune function, as well as lower cortisol levels, than those in a control group (Witek-Janusek et al., 2008). Future work could adapt to this approach to other chronic illnesses as well as cognitive outcomes.

Future research on stress and aging will need to consider how multiple dimensions of stressors and their putative outcomes are embedded in one other. Research on the relationship between stressor processes and physical, mental, and cognitive health is rapidly evolving toward perspectives that emphasize the accumulation, associations, and interactions of stressors over time rather than the impact of a single stressor or category of stressors (Almeida et al., 2009a; Chiriboga, 1997; Turner et al., 2000). Stressors often co-occur across multiple levels (Pearlin et al., 1981). The presence of chronic stressors (persistent or recurrent difficulties of life) may increase exposure to daily hassles, such as troubled relationships giving rise to

more frequent arguments. Chronic stressors may also exacerbate emotional and physical reactions to daily stressors, either by increasing negative appraisal (Lazarus, 1999) or by depleting resources such as time or finances needed for successful coping (Serido et al., 2004). Chronic stress or prolonged stressor exposure has profound negative effects on physiological systems including the pathological overproduction of corticosteroids (e.g., cortisol). Similarly, chronic stress exposure can impair immune function, leading to increased susceptibility to infectious disease (e.g., influenza, common cold) and overactivation of the immune response (e.g., allergic and autoimmune responses; Segerstrom & Miller, 2004). Daily stressor exposure also appears to increase risk factors for the development of cardiovascular disease (e.g., Hallman et al., 2001; Twisk et al., 1999). Chronic stressors and daily stressors often share a common etiology rising out of enduring social roles (i.e., worker, parent, or caregiver) that structure demands and create exposure to crises in real life (Eckenrode, 1984; Pearlin et al., 1981; Serido et al., 2004). Future research should track how role transitions, chronic stressors, and daily stressors coalesce to create patterns of vulnerability that impact mental and physical health.

One way to capture multiple levels of stressful experiences is to embed intensive repeated measurements in traditional longitudinal designs (Almeida et al., 2009b). Using data collected over a long period would permit an examination of how major life events and chronic stressors are linked to daily stress processes. Repeated measurement of various domains on the same individuals over the life course allows important questions to be addressed. How do early life experiences shape adult stressor exposure and reactivity? In particular, how does chronic stress such as poverty in childhood, or residing in a high-crime neighborhood affect stressor reactivity and biomarkers in life, net of current SES? What are mechanisms through which such effects might occur, such as a trajectory of low education, poor job, and resulting financial stressors? Importantly, what aspects of one's life circumstances may modify these relationships? Do many severe stressors early in life have additive or multiplicative effects on reactivity? How are such effects dependent upon the characteristics of the life events themselves in terms of content, severity, persistence, and timing? What aspects of life experience (e.g., quality of interpersonal relationships, educational attainment, SES, and health) can reduce stressor exposure and reactivity? And how does stressor exposure and reactivity influence achievement in the future, including SES and educational attainment?

Data obtained over many waves can be used to tease apart the pathways through which economic, social, and psychological factors affect the daily experience of stressor exposure and reactivity. Using the full life course allows further specificity of life cycle variation in these relationships, as well as an exploration of economic and social factors that contribute to alterations in these pathways (Almeida & Wong, 2009). Moreover, going forward in time, the effects of multiple sources of stressor exposure and reactivity on future experiences can also be examined to elucidate how and under what conditions stress processes may speed aging processes.

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