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Handbook of Cognitive Aging: Interdisciplinary Perspectives

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Considerations for Sampling Time in Research on Aging: Examples From Research on Stress and Cognition

Aprocess is defined as a series of actions or events that are part of development and is often characterized by gradual changes leading to a particular result (Merriam-Webster, 2005). In psychology research, processes are often characterized as explanations or mechanisms for phenomena that unfold within individuals over time. Although there has been a renewed interest in assessing within-person designs (e.g., Molenaar, 2004), in this chapter we highlight the importance of sampling time as it is linked with process. In particular, we highlight research on physiological and psychological processes that account for the association between stress and cognition.

Collecting data at a single point in time, multiple times, and the length between multiple time points all have different consequences for examining an underlying process that is assumed to be captured. When considering processes of change, Nesselroade (1991) identified two kinds of systematic intraindividual (within-person) change that longitudinal researchers should consider. The first is *intraindividual variability* that identifies short-term, relatively reversible changes or fluctuations. For example, fluctuations in mood would be considered intraindividual variability because people vary around their own average. In contrast, *intraindividual change* designates long-term changes that are usually less reversible. These concepts are especially salient for aging researchers because the characterization of a given individual at any point in time involves his or her status on intraindividual change (e.g., trait change, ontogenetic) and his or her status on intraindividual variability (e.g., state, microgenetic; Nesselroade, 1991). Therefore, differences among persons reflect at least three sources of variance: (1) stable individual differences, (2) intraindividual changes, (3) and intraindividual variability. Combining intraindividual variability and intraindividual change leads to a better representation of the developing individual (Baltes, Reese, & Nesselroade, 1977; Nesselroade, 1991; Nesselroade & Ghisletta, 2000). We assert that the elucidation or detection of mechanisms linking processes may be inextricably linked to the choices of sampling time. One aim of this chapter is to show that daily designs can elucidate short-term processes and mechanisms. A second aim is to show that the repetition of the daily approach (i.e., measurement burst design) can help understand micro-level (daily) effects on behavior as well as contextual (e.g., burst-to-burst) and cumulative/long-term effects. We cast our comments within the context of stress and

cognition, an area that has received considerable attention from many disciplines and has great importance for the aging process (e.g., McEwen, 1999)

In laboratory-based studies, stress is typically associated with poorer cognitive performance (e.g., Lupien & Lepage, 2001; Sapolsky, 1996) and has been implicated as a component that may accelerate age-related cognitive decline (McEwen & Sapolsky, 1995; Sapolsky, 1996). Similarly, people who report higher levels of self-reported stress outside the laboratory also tend to experience poorer memory performance (e.g., Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000; VonDras, Powless, Olson, Wheeler, & Snudden, 2005). This has led researchers to propose various explanations for the negative effects of stress on memory. Cortisol, a hormone released in response to stress, has been advanced as one important mechanism for explaining the effects of stress on cognition (Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Wright, Kunz-Ebrecht, Iliffe, Foese, & Steptoe, 2005). Longitudinal studies have provided some of the most compelling evidence of a cortisol-cognition link (Lupien et al., 1994, 1998; Seeman, McEwen, Singer, Albert, & Rowe, 1997). These findings suggest that, over time, repeated exposure to heightened levels of cortisol irreversibly damage the hippocampus, one of the consequences of a phenomenon called *allostatic load* (McEwen, 1999)

The evidence supporting the cortisol-cognition link has served to motivate research investigating the effects of short-term acute stressful events on cognitive function. What is not clear, however, is whether the mechanisms that are assumed to underlie the relationship between stressors and long-term changes or differences in cognition are the same mechanisms responsible for short-term changes or decrements in cognition. The long-term effects of cortisol on the hippocampus have been used as a working model for studying the effects of short-term, acute stressful experiences on cognitive function. We propose that the mechanisms that pertain to effects transpiring over longer periods of time (e.g., years), may not be the same as those that operate over shorter periods of time (e.g., days, hours, or even minutes). In this chapter, we highlight the usefulness of daily diary (Bolger, Davis, & Rafaeli, 2003) and measurement burst designs (an extension of a daily diary design; Nesselroade, 1991) for examining both shorter and longer term processes.

The Importance of Sampling Time

Within the existing stress and cognition literature, time plays an important role for both constructs. Stressors can be short, acute events with short-term effects or ongoing, chronic events with more long-term effects. For example, stressors can take the form of life events (discrete, observable events that stand for significant life changes with a relatively clear onset and offset; Wheaton, 1999), chronic stressors (slowly developing prob-

lematic conditions in social environments or roles that typically have a longer time course than life events; Wheaton, 1999), or daily hassles (the irritating, frustrating, distressing demands that to some degree characterize everyday transactions with the environment; Kanner, Coyne, Schaefer, & Lazarus, 1981, p. 3). Also, time is important for cognitive aging researchers who may be interested in examining changes in cognition over a long period of time (e.g., Colsher & Wallace, 1991; Evans et al., 1993; Wilson, Beckett, Bennett, Albert, & Evans, 1999), as well as fluctuations in memory ability over shorter periods of time such as from one day to the next (e.g., Neupert, Almeida, Mroczek, & Spiro, 2006; Sliwinski, Smyth, Hofer, & Stawski, 2006). It is important to note that cross-sectional designs do not permit an assessment of the nuances of time, such as the duration of the stressor itself, the duration of its impact, and the changes in cognition. For these reasons, studies that can combine repeated micro-longitudinal designs within the context of a more traditional longitudinal design may be the best suited to address questions of mechanisms between stress and cognition over varying time frames. For example, mechanisms linking an argument on a given day to memory performance the next day could be very different from the mechanisms that link the stress of a chronically toxic relationship to long-term changes in encoding deficits.

The timing of the constructs themselves is important, but the timing in terms of the life course is also crucial to consider. Older adults have been exposed to more stressors throughout their lifetimes compared to younger adults (Pearlin & Mullan, 1992), so the long-term accumulation of stressful events could be an additional context for consideration in the link between a stressful event and cognition in aging. Evidence suggests that older adults, in addition to experiencing more stressors throughout their lifetimes, have poorer memory for activities (e.g., Earles & Coon, 1994; Kausler & Hakami, 1983), slower processing speed (Madden, 1985; Salthouse, 1996), decreased working memory ability (Salthouse, Babcock, & Shaw, 1991), and poorer episodic memory ability (Foos & Sarno, 1998) than younger adults. Longitudinal studies have shown that, on average, cognition declines in old age (e.g., Colsher & Wallace, 1991; Evans et al., 1993; Wilson et al., 1999). Furthermore, there is evidence of age-related atrophy of the hippocampus and frontal lobes (Rosch, 1997). Loss of recent memory, as well as impaired learning and concentrating skills in older adults, is often attributed to smaller hippocampi (Rosch, 1997). Therefore, stress and cognition are important constructs within the aging process. Many of the studies we review in this chapter have been conducted with samples of older adults, further underscoring the importance of changes in stress and cognition and the link between them for aging individuals.

It is also important to consider multiple time frames of stress-cognition processes. For example, the context of micro (daily) events often occurs in the context of more macro (enduring) life situations. It is often the case

that daily hassles or stressors occur as a chronic stressor is also unfolding (Serido, Almeida, & Wethington, 2004). Therefore, relatively short-term processes can take place within a context of accumulation, which could compound their effects. For example, continual daily fights with one's spouse could eventually lead to separation and divorce. The ability to concentrate on a day-to-day basis may be impaired by intrusive thoughts about domestic problems, thus impairing cognitive functioning (Sarason, Pierce, & Sarason, 1996). It is also possible that the chronicity of this stress may be associated with impaired cognition because of prolonged stress responses (e.g., Sapolsky, 1992). Daily stressors affect well-being not only by having separate, immediate, and direct effects on emotional and physical functioning, but also by piling up over a series of days to create persistent irritations and overloads that may result in more serious stress reactions, such as anxiety and depression (Lazarus, 1999; Zautra, 2003). We assert that the mechanisms underlying the process of short-term stressors may be distinct from and interact with those related to accumulation and that it is important to design studies that take both into account. In the following sections, we review the literature of chronic and acute stressors and the mechanisms that may tie them to cognition, and then we outline the benefits of daily diary and measurement burst designs.

Chronic Stress

Much of the research linking the effect of chronic stress to cognition has done so by linking cortisol, a hormone released in response to stress, to impaired cognitive function. Glucocorticoids (e.g., cortisol and hydrocortisone) are secreted by the adrenal gland in response to a wide variety of stressors (Heffelfinger & Newcomer, 2001; Sapolsky, 1992). In addition, glucocorticoids are known to regulate various brain functions, including human cognitions (Heffelfinger & Newcomer, 2001). Excessive exposure to glucocorticoids can damage the brain and make neurons more vulnerable to insults (Sapolsky, 1992), and increased glucocorticoid exposure in humans at levels associated with stress can decrease memory and learning function (Heffelfinger & Newcomer, 2001)

In Selye's initial studies (e.g., Selye, 1936), he conceptualized the stress response as having distinct components. In the initial, *alarm* stage, the stressor was noted or experienced. The second stage, *resistance*, consisted of successfully dealing with the short-term physical insult. The third stage was where disease started, when the stressor became chronic. This final stage was termed *exhaustion*, because there were no longer sufficient glucocorticoids to combat the stressor. In Selye's view, then, stress-related disease was due to the

stressor itself attacking the undefended body. However, there is little empirical evidence of such a global exhaustion of the hormones of the stress response (Sapolsky, 1992). Chronic stress is not damaging because the body's defenses fail but because, with enough chronic stress, those defenses themselves (i.e., glucocorticoids) become damaging (Sapolsky, 1992, 1996). During an acute stressor, the costs of the stress response can be contained, but with chronic activation they exact a toll on the body (Sapolsky, 1992, 1996)

Cortisol and Cognition: Chronic and Acute Effects

There has been considerable interest in understanding how stress affects cognitive function because long-term exposure to cortisol impairs the structural (Brown et al., 2004; Lupien et al., 1998; McEwen, 1999; Sapolsky, 1996; Starkman, Giordani, Gebarski, & Schteingart, 2003) and functional (Brown et al., 2004; Lupien et al., 1994, 1998; Seeman et al., 1997; Starkman, Giordani, Berent, Schork, & Schteingart, 2001) integrity of the hippocampus (Jelicic & Bonke, 2001) and frontal lobe (Lupien & Lepage, 2001). Some of the most compelling evidence suggesting a cortisol-cognition link comes from studies documenting the deleterious effects of long-term exposure to high levels of cortisol (Jelicic & Bonke, 2001). Lupien et al. (1994, 1996) examined how change in older adults basal cortisol levels over a 4-year period was related to cognitive functioning at the end of that same period. Lupien et al. (1994) examined performance on two tasks as a function of subgroups based on cortisol slope trajectories. Three distinct groups were identified: (1) high increasing slope, (2) moderate increasing slope, and (3) no slope. The increasing-slope groups exhibited reliably poorer recall and slower search times compared with older adults exhibiting no change in cortisol. Furthermore, the older adults exhibiting no change in cortisol did not differ in cognitive performance from healthy young adults. This suggests that longitudinal increases, over the long term, in cortisol are a sign of hypothalamic-pituitary-adrenal axis dysfunction and that increasing levels of cortisol may impair cognitive performance. Thus, behavioral deficits may be mediated by cortisol-related changes in brain structure (McEwen, 1999; Sapolsky, Krey, & McEwen, 1986)

Seeman et al. (1997) found that change in cortisol over 2.5 years reliably predicted change in delayed recall performance in women, but not in men. Furthermore, changes in recall were found to vary as a function of the change in cortisol. Women who exhibited decreases in cortisol exhibited improved recall over that same time period, whereas women with increasing cortisol levels exhibited a substantial decline in recall performance. Similarly, Seeman et al. (2001) found that baseline levels of allostatic load, of which cortisol is one marker,

predicted 7-year changes in general cognitive function. Together, these studies suggest that cortisol is related to changes in cognitive function and that allostatic load may be a risk factor for impaired hippocampal and general cognitive functioning.

Although there is considerable evidence to suggest that cortisol is related to cognitive and brain function, there is reason to suspect it is not the sole mechanism underlying acute stress-related cognitive impairments. First, the evidence suggesting a relationship between changes in endogenous cortisol and cognitive function is inconsistent. Although some studies have shown that stress-related increases in cortisol are associated with lower memory performance (Kirschbaum et al., 1996; Wolf, Schommer, Hellhammer, McEwen, & Kirschbaum, 2001), others have shown no association between cortisol reactivity and memory performance (Domes, Heinrichs, Reichwald, & Hautzinger, 2002; Jelicic, Geraerts, Merckelbach, & Guerrieri, 2004). A number of studies have shown that an increase in cortisol over the course of years is predictive of memory (Lupien et al., 1994, 1998), intellectual deficits (Seeman et al., 1997, 2001), and smaller hippocampal volume (Lupien et al., 1998). Acute increases in cortisol, however, are less predictive of cognitive function (Domes et al., 2002; Jelicic et al., 2004). With longitudinal increases in cortisol linked to the occurrence of stressful events, one cannot rule out factors other than stressful experiences causing such increases in cortisol.

Although it is important to understand how the physiological mediators of stressors impact cognition, it may be equally important to understand the contexts of the triggering stressors themselves. In other words, secretion of cortisol needs to be considered within the context or domain of the stressor. For example, Dickerson and Kemeny (2004) postulated a *social self-preservation theory*: Individuals who might be expected to experience greater threat due to a highly valued ability (e.g., cognitive performance) were the ones who secreted higher amounts of cortisol during the threat. Neupert, Miller, and Lachman (2006) found that older adults with high levels of education secreted the most cortisol over time (i.e., had the steepest slopes of cortisol reactivity) in response to laboratory-based cognitive stressors. In their study, cognitive tests were used as the impetus for changes in cortisol; that is, cognitive testing served as the stressor, and then slopes of change in cortisol were predicted by age and education. In line with Dickerson and Kemeny's ideas, it is possible that cognitive testing is more stressful among individuals with higher education relative to those with lower education because the former are more concerned about decline and have a greater stake in their cognitive performance. In addition, these stressors may be more salient for older adults who could be experiencing some age-related cognitive decline (Neupert, Miller, & Lachman, 2006). This is also consistent with Lazarus's (1999) notion of *primary appraisal*, in which a situation or event must be considered valuable or salient before it can become stressful.

In addition to the domain or context of the stressor, time is also crucial to consider with respect to cortisol studies. Sapolsky, Romero, and Munck (2000) suggested that cortisol can take approximately 1 hour, if not longer, to exert effects on hippocampal function. Thus, the time course for the effects of cortisol on neural function may be at odds with the hypothesis that acute stress-related increases in cortisol mediate cognitive performance decrements observed very proximal to the stressor. Cortisol is typically measured within 10 to 20 minutes after cessation of a laboratory stressor, whereas cognitive assessments are typically conducted within minutes of the stressor. Therefore, cortisol may not have had sufficient time to manifest its effects on the brain, but other components of the physiological stress response could (Sapolsky et al., 2000). Taken together, the results of previous research suggest that cortisol is likely to be implicated in the long-, but not necessarily the short-term effects of stress on cognition. However, as Sapolsky et al. (2000) and Lupien and McEwen (1997) have pointed out, the exact time course for the effects of cortisol on cognition is not known. Future studies examining cortisol and cognition could focus on the time course of the effects and whether they depend on certain situations (e.g., naturalistic vs. laboratory based) and/or individual characteristics.

The sheer accumulation of events and experiences is an important element to understanding time and the stress-cognition links. The cumulative effects of stress hormones are associated with smaller hippocampi (e.g., Heffelfinger & Newcomer, 2001; Starkman, Gebarski, Berent, & Schteingart, 1992; Uddo, Vasterling, Brailey, & Sutker, 1993; Vasterling et al., 2002). Therefore, older adults who have been exposed to more stressors throughout their lifetime compared with younger adults (Pearlin & Mullan, 1992) may have fewer resources (i.e., smaller hippocampi) to combat the negative effects of stressors, thereby resulting in heightened cognitive reactivity (i.e., reduced cognitive performance in response to stressors). It is also possible that resources may never fully develop when traumatic stressful events occur early in life (e.g., Vythilingam et al., 2002). Anderson and Craik (2000) suggested that reductions in hippocampal volume lead to reduced attentional resources and cognitive slowing. Subsequently, these deficits result in reduced cognitive control that then negatively impacts memory performance. Thus older adults, when faced with a stressor, may be more likely to experience lowered cognitive performance because they are unable to tap the same resources available to younger adults. Older adults who have more stressors than younger adults (interindividual difference) may be at greater risk, but the accumulation of stressors across the life course (intraindividual change) is also important.

Stress and Cognition: Psychological Perspectives

Although the physiological view of cortisol has been the predominant view for explaining the effects of stress on cognition, the evidence we have reviewed thus far calls into question whether cortisol is the sole mechanism underlying this relationship. This is particularly important to underscore when examining the short-term effects of stressors, because cortisol appears to exact its toll over extended periods of time (e.g., Heffelfinger & Newcomer, 2001) but may be less influential immediately after a stressor (e.g., Domes et al., 2002). A complementary view of short-term effects is rooted in psychological processes, where mental control, cognitive interference, and resource capacity may be important mechanisms. Mandler (1979) suggested that the experience of stress draws attention away from primary task processing and redirects it toward changes in physical states and the conditions causing such changes. Similarly, Wegner (1988) argued that stress impairs mental control, specifically the ability to concentrate. Consistent with these perspectives, Sarason and colleagues (Sarason et al., 1996; Sarason, Sarason, Keefe, Hayes, & Shearin, 1986), as well as Eysenck and Calvo (1992), have advanced cognitive interference as a mechanism responsible for effects of stress on cognition. Cognitive interference, according to Sarason and Eysenck, can be task-oriented worries (e.g., worrying about performance quality) and off-task thoughts (e.g., thinking about a negative event that may have just happened). The premise of the cognitive interference perspective is that stress-related cognitions create a dual-task situation whereby attention is divided between task performance and coping with the stress. These cognitions in turn limit the capacity to process and store information. Reduced attention to primary-task processing results in decreased performance (e.g., Eysenck & Calvo, 1992). Sarason et al. (1996) stated that this cognitive interference can exist as task-unrelated thoughts (e.g., thoughts about a stressful event that was just experienced) and task-related worries (i.e., self-initiated performance evaluation because of a belief that performance quality is poor after stress). This perspective, then, is concerned with the context of the cognitive testing situation as it pertains to thoughts within the individual at the timing of testing.

Much of the evidence supporting the cognitive-interference hypothesis has come from Klein and colleagues, who have examined how negative life events stress is related to cognitive function. In an interindividual-differences design Klein used negative life events stress as a proxy for a person's proclivity to experience stress-related cognitive interference, such that individuals with higher levels of life stress were (implicitly) those who experience more recurring thoughts about those events (i.e., cognitive interference). Thus, Klein used life events stress to serve as an indirect index of stress-related cognitive interference. Baradell and Klein (1993) and Klein and Barnes (1994) examined how the impact of self-reported negative life events predicted per-

formance on a decision-making task in a sample of college students. Baradell and Klein observed that individuals who reported greater negative life events stress and were more sensitive and aware of bodily states (e.g., heart rate) made more errors, used less efficient decision-making strategies, and made more decisions without examining all options compared with individuals who had low life stress. Similarly, Klein and Barnes observed that high-life-stress individuals who were keenly aware of bodily states and anxious at the time of testing made more errors on a complex reasoning task. These individuals were less efficient in their solving of the analogies, and this inefficiency mediated the effect of life stress on overall problem-solving performance. Klein and Barnes took these findings as evidence that individuals with high life event stress have more (intrusive) thoughts about their previous stressors and focus on bodily states, subsequently devoting less attention to information processing and task performance. One limitation of these studies is that no measure of cognitive interference was included; therefore life events stress is at best an indirect indicator of cognitive interference.

In subsequent studies also using interindividual differences designs, both life events stress and cognitive interference were found to be predictive of cognitive performance. Yee, Edmonson, Santoro, Begg, and Hunter (1996) demonstrated that individuals with higher levels of negative life events stress also exhibited the poorest performance on an analogical reasoning task. Furthermore, individuals reporting higher levels of cognitive interference during the reasoning task took longer to complete the task. Klein and Boals (2001b) conducted three studies examining the relationship among negative life events stress, stress-related cognitive interference, and working memory. They observed reliable negative relationships between negative life events stress and working memory and between stress-related cognitive interference and working memory. Furthermore, the observed relationships between stress and working memory increased as a function of capacity and processing demands, suggesting that cognitive interference is increasingly detrimental when processing and capacity demands are high. Given that these results are correlational, direction of causality cannot be determined. An alternative interpretation is that thought suppression or inhibition is governed by working memory and that individuals with lower working memory capacity are poorer at inhibiting off-task and distracting thoughts (Brewin & Beaton, 2002; Engle, 2002; Rosen & Engle, 1998; Stoltzfus, Hasher, & Zacks, 1996). Furthermore, because event severity and cognitive interference were not examined in the same statistical model, it is not clear whether the experience and impact of stress and the psychological reactivity (intrusive thoughts) about stressful experiences are unique predictors of working memory performance.

Although previous studies cannot provide evidence of directionality or causality, Klein and Boals (2001a) used a within-person design to demonstrate that reducing cognitive interference actually improves working mem-

ory. Using a stress reduction technique, they showed that expressive writing (Smyth, 1998) about a stressful life event increased working memory 7 weeks later, compared with writing about a neutral topic. Furthermore, the increase in working memory was mediated by a reduction in stress-related cognitive interference. This study provides the first evidence that stress-related cognitive interference impairs cognitive function, and it is consistent with theories positing such effects when cognitive capacity and attentional resources are limited (Eysenck & Calvo, 1992). In addition, Stawski, Sliwinski, and Smyth (2006) showed that cognitive interference can be especially salient within the context of aging. They found that stress-related cognitive reactivity was predictive of poorer working memory, episodic memory, and processing speed in older adults.

Taken together, the results of these studies indicate that the impact of life experiences and stress-related cognitive interference are important predictors of cognitive functioning in both young and old populations. Furthermore, the impact of negative life experiences may serve as an indirect indicator of stress-related cognitive interference. Given that these studies have demonstrated that a cognitive/psychological component of the stress response is an important factor related to cognitive performance, theories such as processing efficiency theory (Eysenck & Calvo, 1992) and the cognitive interference hypothesis appear tenable. Thus, stress-related cognitive interference may act as a cognitive load and create a situation in which limited capacity and processing resources are divided between task-oriented processing and stress-related cognitive interference. It is possible that cognitive interference could have both acute and long-term effects on cognition. Future studies that incorporate measures of cognitive interference within short time intervals as well as longer time intervals could examine whether cognitive interference acts across varying time frames, much like cortisol. Additionally, because the accumulation of stress can compound the negative effects of daily stressors (e.g., Zautra, 2003), repeated assessments could elucidate whether the effects of stress on cognition are a function of individual trait-like characteristics (e.g., a high-stress individual or someone with high cognitive interference) or a function of the time when the individual was assessed (e.g., a time of particularly high stress or high cognitive interference), or both (e.g., individuals who are assessed at a time of high stress but who typically experience few stressors may react the most)

Limitations of the Cognitive Interference Perspective

Although the literature supporting the cognitive interference hypothesis is small, the results indicate that stress-related cognitive interference is a factor reliably related to cognitive function. However, there are a

number of reasons these studies cannot conclusively substantiate the cognitive interference hypothesis. One limitation of the studies providing support for the cognitive interference hypothesis is that cognitive interference is not assessed during concurrent cognitive processing. The cognitive interference hypothesis, and similarly processing efficiency theory (Eysenck & Calvo, 1992), posit impaired cognitive performance when stress-related cognitive interference is experienced simultaneously. Each of the studies reviewed here demonstrated a relationship between stress-related cognitive interference and cognitive performance but did so without evidence of an online effect. Thus, at best, these results suggest that people with higher levels of stress-related cognitive interference also exhibit poorer performance across a number of cognitive domains. As stated previously, individuals with a lower level of cognitive function may be increasingly susceptible to the experience of stress-related cognitive dysfunction because of an inability to inhibit off-task thoughts (Hasher & Zacks, 1988; Rosen & Engle, 1998). However, Yee et al. (1996) provided evidence indicating that individuals reporting higher levels of cognitive interference immediately after cognitive assessment exhibited poorer performance. Their findings were observed independent of any experience of stress, so the cause of the cognitive interference cannot be solely attributed to an acute stressful event but may be associated with a dispositional trait (cf. Pierce et al., 1998). To tease apart the influence of states and traits, study designs need to incorporate indicators of intraindividual variability and interindividual differences (Nesselrode, 1991). As we assert in more detail below, designs with frequent repeated assessments over time could be particularly beneficial for examining person- versus context-specific effects.

A second limitation of these studies is that cognitive interference was not measured with respect to a single specific event, proximal to performance on a cognitive task. Therefore, there is no indication that any interference was experienced at the time of, or even proximal to, the cognitive assessment. These assessments of stress-related cognitive interference were inefficient for examining the effect of stress-related cognitive interference during concurrent cognitive performance. Studies that examine both cognitive performance and cognitive interference immediately or even shortly after the experience of a stressful event would provide a better test of the cognitive interference hypothesis as well as provide evidence of direction of causality in the stress-cognition link. It is possible that cognitive interference may have a dynamic effect on memory, but it is also important to note that there could be interindividual trait-like differences in cognitive interference that may act as moderating effects in a dynamic process. We assert that daily diary designs could be beneficial for examining these processes as they occur (or shortly thereafter)

Strengths of the Daily Diary Design for Examining Short-Term Effects

The daily experience paradigm allows researchers to examine within-person covariation between components of daily well-being and daily stressors over time, thereby establishing temporal links between daily stressors and well-being (Shiffman & Stone, 1998; Tennen, Suls, & Affleck, 1991). By studying within-person through-time covariation between daily stressors and well-being one can more precisely establish the short-term effects of concrete daily experiences (Almeida & Kessler, 1998; Bolger, DeLongis, Kessler, & Schilling, 1989; Larson & Almeida, 1999; Lewinsohn & Talkington, 1979; Stone, Reed, & Neale, 1987). Also, the daily diary design reduces retrospective recall bias because participants are asked to recall events that occurred over the previous 24-hour period as opposed to a week or even a year (Kessler, Mroczek, & Belli, 1999). Therefore, a more accurate picture of individuals daily lives can be captured with this design, and more accurate assessments of stressors and cognitive interference are possible. When conclusions are drawn between people about the relationship between the predictors and outcomes, the covariation that occurs through time is lost. In a within-person design, conclusions can be made about the simultaneous effects of within-person covariation as well as between-person differences. This is especially important when many interindividual differences (e.g., trait-level cognitive interference) may exist in the within-person relationship between stressors and cognition.

In a daily diary study of older adults that examined daily stressors and memory failures over 8 consecutive days, Neupert, Almeida, et al. (2006) found that there was significant within-person covariation between concurrent day stressors and memory failures as well as between stressors and change in memory failures from one day to the next. These associations remained even after controlling for the effects of neuroticism, life event stressors, and health. Life event stressors were also related to the number of memory failures, such that people who reported more life event stressors also reported more memory failures. The general trend in the association between stressors (life events and daily events) and memory failures supports the findings of many laboratory-based studies (e.g., Vedhara et al., 2000) and extends previous work to a more naturalistic setting. Although life event stressors and daily stressors are different types of events (Wheaton, 1999), they are both important when one is examining everyday memory failures. This finding not only shows the association between stressful life events and memory but also underscores the deleterious effects of seemingly minor stressors that most people experience frequently on a daily basis. Therefore, even if someone does

not experience any major life event stressors, day-to-day stressors can still negatively affect his or her memory. When participants experienced interpersonal stressors, they experienced more memory failures on that same day as well as an increase in memory failures from one day to the next. It is possible that interpersonal stressors are especially distracting for older adults, who tend to be solution oriented when faced with an interpersonal conflict (Bergstrom & Nussbaum, 1996) and therefore place more effort and attention on finding a solution for emotionally salient goals (Carstensen, Isaacowitz, & Charles, 1999). It is also possible that interpersonal stressors are related to memory failures through cognitive interference, because stressful social situations have been linked with more intrusive cognitions (Sarason et al., 1996). Because effort and attention are directed toward the interpersonal conflict, less attention may be available for tasks requiring memory.

Sliwinski et al. (2006) examined age differences in the effects of naturally occurring daily stressors on cognitive performance. Participants completed a daily diary of stressful events (Almeida, Wethington, & Kessler, 2002) as well as two performance-based cognitive tasks used to assess working memory (the *n*-back [McElree, 2001; Verhaeghen & Basak, 2005] and *n*-count [Garavan, 1998]) for 6 days over the course of 14 days. Participants completed two versions of each task, one with high attentional demands and one without. On days when stress was greater than usual, performance on the attentionally demanding versions of each task was significantly poorer, whereas performance on the nonattentional demanding versions remained unaffected. Furthermore, the stress effect was significantly larger for the older adults on the *n*-count task, suggesting that the effects of stress on cognition may be more detrimental to older adults. These results indicate that stress impairs attentionally demanding cognitive performance and that older adults may be more susceptible to these effects. This study is the first to link naturally occurring stressors to laboratory-based indicators of cognition, but future research that is able to examine stress and cognition on consecutive days could test for lagged effects of the link between stress and cognition from one day to the next. This method and analysis would allow researchers to determine whether the cumulative buildup of stressors over a short period could be important for short-term changes in cognition. It is important to note that daily diary designs could include assessments of cortisol and cognitive interference to examine the saliency of both stress mechanisms. Daily diary studies conducted to date have not empirically determined a mechanism for the observed acute effects of stress on cognition. In addition to identifying possible single mechanisms, future studies could examine whether multiple mechanisms are acting in concert in the dynamic interplay between stress and cognition.

Strengths of the Measurement Burst Design for Examining

Short-Term and Long-Term Effects

Just as a single assessment of an attribute does not convey information about intraindividual variability, a design with 1 week or 2 weeks of daily assessments does not convey information about change in intraindividual variability transpiring over longer intervals. As Nesselroade (1991) noted, the accurate assessment of intraindividual variability requires repeated assessments over short time intervals, and the assessment of intraindividual change requires repeated measurements over intervals suitable to the subject of inquiry. He proposed that longitudinal research designs be planned around successive “bursts” of measurements rather than just successive measurements. Specifically, each time of measurement within a longitudinal design should provide estimates of intraindividual variability. Researchers have begun to use these suggestions by conducting repeated daily diary assessments over the course of months (e.g., the Cognition, Health, and Aging Project at Syracuse University), years (The VA Normative Aging Study), and even decades (the ongoing National Study of Daily Experiences study within the Midlife in the United States survey). The measurement burst design could be particularly valuable for the study of stress and cognition, because questions regarding the unfolding of short-term effects within longer time periods as individuals and contexts change could be addressed. Specifically, the dynamic processes that occur at and interact over difference cadences (e.g., days, years), between stressors and cognition, such as cortisol, cognitive interference, and developmental changes, could be teased apart with the implementation of this design.

Conclusion

We assert that sampling time should be taken seriously, because it has tremendous implications for the underlying process being examined. As others (e.g., Martin & Hofer, 2004) have noted, sampling time will influence analysis and interpretation of intraindividual variability and short-term change. The measurement burst design provides a novel approach for examining the short- and long-term cognitive effects of stressors, but this design can also be applied to other areas of cognitive aging. It is important to note that measurement burst designs can capture short-term processes as they unfold within a more long-term context. By extending the daily diary design to a measurement burst design, researchers will be able to examine long-term changes in short-term covariation within the changing contexts of the individual.

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- cortisol
- stressors
- interference
- memory failure
- cognition
- life events
- glucocorticoids

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