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Global Perceived Stress Predicts Cognitive Change among Older Adults

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Abstract

Research on stress and cognitive aging has primarily focused on examining the effects of biological and psychosocial indicators of stress with little attention provided to examining the association between perceived stress and cognitive aging. We examined the longitudinal association between global perceived stress (GPS) and cognitive change among 116 older adults ($M_{age} = 80$, $SD = 6.40$, range: 67–96) in a repeated measurement burst design. Bursts of six daily cognitive assessments were repeated every six months over a two-year period with self-reported GPS assessed at the start of every burst. Using a double-exponential learning model, two parameters were estimated: 1) asymptotic level (peak performance), and 2) asymptotic change (the rate in which peak performance changed across bursts). We hypothesized that greater GPS would predict slowed performance in tasks of attention, working memory, and speed of processing and that increases in GPS across time would predict cognitive slowing. Results from latent growth curve analyses were consistent with our first hypothesis and indicated that level of GPS predicted cognitive slowing across time. Changes in GPS did not predict cognitive slowing. This study extends previous findings by demonstrating a prospective association between level of GPS and cognitive slowing across a two-year period highlighting the role of psychological stress as a risk factor for poor cognitive function.

Keywords

Stress; global perceived stress; cognition; aging; retest effects

An important public health goal of cognitive aging research has been to identify modifiable risk-factors of cognitive decline. Identifying factors that contribute to cognitive impairments in old age is imperative for designing interventions that assist people in maintaining their cognitive health and functional independence (e.g., Tomaszewski Farias et al., 2009). Stress represents one potentially modifiable risk-factor of cognitive decline and impairment in old age (McEwen, 2000). Although much research has focused on the biological markers of stress and stressful life events, less is known about the association between the experiential

aspects of stress (i.e., perceived stress) and cognition. The purpose of this study is to examine the longitudinal association between perceived stress and cognitive function in older adults.

Stress and Cognitive Function

Two lines of research provide support for the association between stress and cognitive functioning. One line examines putative biomarkers of stress such as cortisol and inflammation. Research in this area suggests that chronic activation of the physiological stress response leads to cumulative “wear and tear” of the brain and body through overexposure to glucocorticoids (e.g., Lupien, McEwen, Gunnar, Heim, 2009; McEwen, 1998) and circulating inflammatory cytokines (e.g., Black, 2002; Yaffe, 2004). Elevated levels of stress biomarkers (e.g., cortisol) are associated with atrophy of brain structures, such as the hippocampus and prefrontal cortex that are essential for cognitive functioning (Lupien & Lepage, 2001). This line of research presupposes that these biomarkers are the end result of events and experiences that elicit psychological stress in individuals, but the psychological component of the stress response is not examined in these studies (e.g., Lupien et al., 1997). Furthermore, examining associations among biomarkers of stress alone is confounded with their association with other health conditions (e.g., cardiovascular disease; Williamson, Mangos, & Kelly, 2005) that are also associated with impaired cognitive function (e.g., Knopman et al., 2001). It is therefore necessary to examine the effects of the psychological aspects of stress to establish a direct connection between experienced stress and cognitive impairment.

A second line of research has established associations between exposure to stress events and cognitive function in adults. Greater reports of life events or chronic difficulties, for example, are associated with declines in cognitive performance among adults with already compromised cognition (i.e., those who initially exhibited symptoms of mild cognitive impairment; Peavey et al., 2009). Although there have been inconsistent findings in the literature regarding life events and cognitive function (e.g., Comijs, van den Kommer, Minnaar, Penninx, Deeg, 2011), adverse effects of these events on functioning appear to be dependent upon the type of stressor and the perceived implications of the event on an individual’s well-being (Cohen, Kessler, & Gordon, 1995; Rosnick, Small, McEvoy, Borenstein, & Mortimer, 2007; Tschanz et al, 2013). This indicates that exposure to events alone does not predict loss of cognitive function.

Although individuals who report experiencing a greater frequency of stressful life events may not exhibit cognitive impairment, they may exhibit poorer cognitive performance at times proximal to reporting having experienced a negative event. For instance, on days when participants reported having experienced a negative stress event, they were more likely to perform worse on attention-demanding cognitive tasks compared to non-stress days and these decrements were more pronounced among older compared to younger adults (Sliwinski, Smyth, Hofer, & Stawski, 2006). Reports of daily negative events have also been associated with memory failures and self-reported changes in memory (Neupert, Almeida, Mroczek, & Spiro, 2006; Stawski, Mogle, & Sliwinski, 2013). Although daily negative experiences may exert immediate effects on cognitive function, long-term ramifications for

cognition have not been established. It is possible that the accumulation of these negative experiences have long-term consequences for cognition as has been demonstrated for mental and physical health outcomes (Charles, Piazza, Mogle, Sliwinski, & Almeida, 2013; Piazza, Charles, Sliwinski, Mogle, & Almeida, 2012).

Consistent with this perspective, there is evidence that individuals under chronic stress associated with their social roles are at risk for loss of cognitive function. Individuals who are in a chronically stressful social role, such as an uncontrollable work environment or caring for a family member with Alzheimer's disease, may experience burnout (Maslach, Schaufeli, & Leiter, 2001; Takai et al., 2009). Burnout is a combination of psychophysiological symptoms, including emotional exhaustion, reported as a result of chronic role-related stress (Linden, Keijsers, Eling, & Schaijk, 2005). Indeed, individuals exhibiting symptoms of work-related chronic burnout perform more poorly on tasks of sustained attention as well as immediate and delayed recall compared to controls (Linden et al., 2005; Öhman, Nordin, Bergdahl, Birgander, & Neely, 2007; Sandström, Rhodin, Lundberg, Olsson, & Nyberg, 2005). Middle-aged adults who reported greater job strain, characterized by having low control in ones' occupation, were also at greater risk of cognitive impairment in older adulthood (Anel, Crowe, Kareholt, Wastesson, & Parker, 2011). Caregiving for a spouse with Alzheimer's disease is a source of chronic role stress among older adults that involves exposure to numerous long-term stressors, which are often unpredictable and uncontrollable and have been prospectively associated with cognitive decline (Vitaliano et al., 2005).

Overall these studies show that experiences of stress and being in a demanding social role are associated with poorer cognitive function. However, it is likely that not all individuals in a particular social role or who experience a potentially stressful event become psychologically stressed (e.g., Aldwin, Sutton, Chiara, & Spiro, 1996). Chronic exposure to demanding situations and occupying difficult social roles certainly contribute to psychological stress but there are individual differences in how people experience stress in response to similar difficult situations. Additionally, psychological stress can emerge even in the absence of major life events and in persons who do not occupy normatively stressful social roles. Although individual differences in subjective appraisals of stress likely play a critical role in determining the effects of stress, they have seldom been explored in the cognitive aging literature. The current study aims to address this lack of knowledge on the association between individual differences in perceived stress and cognitive function. We examine the longitudinal association between perceived stress and cognitive function in a sample of older adults followed prospectively for two years as part of a measurement burst study (Nesselrode, 1991; Sliwinski, 2008).

Perceived Stress and Cognitive Function

A key component of the stress process is that events increase risk for adverse health outcomes if they are perceived to be stressful (Cohen et al., 1995; Lazarus & Folkman, 1987). Therefore, the psychological perceptions of environmental demands or events are important to examine in relation to health outcomes. Consistent with this view, some evidence indicates that distress resulting from negative events, as opposed to the number of

events experienced, has implications for cognitive function. Stawski and colleagues, for example, found that greater affective responses to daily hassles, rather than the frequency of exposure to hassles, were associated with fluid cognitive ability (Stawski, Almeida, Lachman, Tun, & Rosnick, 2010; Stawski, Mogle, Sliwinski, 2011). These results may indicate that those with greater fluid abilities are better able to regulate their affective reactions to stressors. Stawski's results may also indicate that greater emotional reactions to daily hassles are associated with poorer cognitive abilities. This is in line with Rosnick and colleagues' (2007) findings, which indicated that it was not the total count but the self-reported severity of the events that had implications for task performance.

Perceived stress is a key component of the stress process that triggers emotional, physiological or behavioral responses associated with increased risk of cognitive impairment (Cohen, Kamarck, & Mermelstein, 1983). In a recent longitudinal study, Rönnlund and colleagues examined the association between self-reported stress and cognitive function every five years over three time points among middle aged adults (Rönnlund, Sundström, Sörman, & Nilsson, 2013). Participants responded to one item at each measurement time point: "Do you feel stressed in general?" and were categorized as reporting consistently high levels of stress across each of the measurement occasions or consistently low levels. Responses for the single stress item were consistent with scores in a perceived stress questionnaire such that those who consistently responded yes to the stress item also tended to exhibit greater perceived stress scores on a longer perceived stress measure. Middle aged adults who were categorized as reporting consistently high levels of stress across each of the three assessment occasions, reported greater declines in subjective cognition compared to those who were categorized as reporting consistently low levels of stress (Rönnlund et al., 2013). Among older adults, Aggarwal and colleagues (2014) reported that greater baseline perceived stress was associated with impaired cognitive function as well as with cognitive decline across a six year period.

Consistent with Aggarwal and colleagues (2014), the current study assesses global perceived stress (GPS) measured via the Perceived Stress Scale (Cohen et al., 1983). This scale measures psychological stress by capturing individuals' feelings of overload, unpredictability, and uncontrollability over the previous month (Cohen, Kessler, & Gordon, 1995). We refer to this as "global" perceived stress, because this measure reflects the chronic influence of ongoing life circumstances, concerns about the future, and reactions to events (Cohen et al., 1983).

GPS may result from acute and more common everyday hassles as well as major life events, thus it may be sensitive to the changing demands an individual perceives (Sliwinski, Almeida, Smyth, & Stawski, 2009; van Eck, Nicolson, & Berkhof, 1998). This implies that GPS is not constant or fixed, but will change within people over time to reflect changes in life circumstances and coping resources. Indeed, fluctuations in GPS within people were found to be associated with greater negative affect reactivity to daily events such that older adults tended to report greater negative affect in response to daily events during measurement occasions when they reported greater GPS (Sliwinski et al., 2009). Despite the apparent dynamic nature of GPS, previous work has only examined how GPS status at baseline predicts subsequent change in cognition in older adults (i.e., Aggarwal et al., 2014);

no study to date has examined how changes in GPS predict changes in cognitive function. Assessing the extent to which changes in GPS are associated cognition is important in order to understand the process through which stress affects cognition through fluctuations in perceived demands or chronic and consistent levels of them. In all, it is still unclear what it is about GPS that results in poorer cognitive function. That is, is it the overall amount of burden an individual experiences (i.e., level) or is it the adjustment necessary as a result of increases in environmental demands (i.e., change) that puts individuals at greatest risk for cognitive impairment?

Present Study and Hypotheses

GPS may be associated with poor cognitive function in at least two ways. First, greater levels of GPS may, over time, increase the likelihood of cognitive decline through neurocognitive impairments that result from exposure to stress-related dysregulation of hormones and cytokines such as cortisol and inflammation (Lupien et al., 1998; Sapolsky, Krey, & McEwen, 1986). Second, individuals who experience an increase in their GPS level may display decrements in cognitive function perhaps due to secondary effects of stress such as fatigue, negative mood, and worry (Smyth, Zawadzki, & Gerin, 2013). These two possible pathways lead to two predictions for this study. First, we predict that higher overall levels of GPS reflect chronic or enduring burden and will be associated with greater cognitive slowing in attention, working memory, and speed of processing tasks (Hypothesis 1). Second, we predict that increases in GPS indicate changes in psychological and environmental demands and will be associated with increased rates of cognitive slowing (Hypothesis 2). We account for depressed mood and physical symptoms as they may be potentially confounding variables. Depressed mood is robustly associated with both psychological stress (Öhman, Bergdahl, Nyberg, & Nilsson, 2007; Watson & Pennebaker, 1989; Watson, 1988) and cognitive function (McBride & Abeles, 2000), and health related functional limitations have also been linked with increased perceived stress (Vasunilashorn, Lynch, Gleit, Weinstein, & Goldman, 2014).

Previous studies that have examined the prospective effects of stress on cognition have not considered the influence of potential practice or retest effects (e.g., Wilson, Li, Bienias, & Bennett, 2006). Failure to model retest effects may result in confounding the effects of stress on practice (or learning of the task due to repeated exposure to the stimuli) and its adverse effects on cognition. In the current study, we use a measurement burst design (Nesselroade, 1991; Sliwinski, 2008) to assess cognition repeatedly (6 assessments) every 6 months for 2 years (30 measurements across 2 years per individual). Practice effects are ubiquitous in longitudinal cognitive research (e.g., Salthouse, Schroeder, & Ferrer, 2004) and may be amplified in intensive repeated measurement designs. Indeed, a previous publication analyzing data from this sample showed retest related performance improvements across sessions within the baseline measurement burst that follow a negative exponential learning function (Sliwinski et al., 2006). Due to the clustered assessments in a measurement burst, conventional approaches to accounting for retest effects, such as dropping the first assessment occasion or statistically adjusting for the number of retest occasions, are not appropriate for addressing retest effects in measurement burst designs (Sliwinski, Hoffman, & Hofer, 2010).

Because measurement bursts consist of clusters of closely spaced measurements (e.g., daily) repeated over longer intervals (i.e., biannually), retest effects likely reflect different processes occurring across these different timescales. Sliwinski, Hoffman, and Hofer (2010) described the double-exponential learning model which captures two retest processes: continuous learning, and a recovery or 'warm-up' effect. Continuous learning effects accrue across all the sessions of a measurement burst; in the case of this present study the maximum number of sessions is 30. However, because there are six month 'gaps' between bursts, performance decrements for initial sessions of follow-up bursts might be worse than peak performance after practice during the previous burst. This implies there might be some initial 'warm-up' effects during follow-up bursts that reflect a quick return to previous, highly-practiced performance levels, overlaid on continuous processing of learning. That results in a rate of retest improvement during follow-up bursts that is faster than could be predicted by a single learning function. Such warm-up effects are commonly observed in multisession skill acquisition studies (Newell, Mayer-Kress, & Lui, 2001; Rickard, 2007).

Sliwinski et al. (2010) provided a real life example of the type of learning (retest) effects that are analogous to those one should observe in a measurement burst study. An older adult who takes up cross-country skiing for the first time might display considerable and rapid improvement in her skiing performance during her first season as indexed by the time taken to ski around the lodge. Then spring arrives, the snow melts and 8 months pass before the next snowfall allows her to resume skiing. On her first day out in her second season, she is a bit 'rusty' (out of practice) and a bit slower than she was at the end of the previous season. But after a few skiing sessions, she quickly recovers the skill that was 'lost' during the warmer months and then continues to improve with additional practice. After a prolonged temporal disruption in practice, performance becomes a function not only of the total amount of practice, but of how much practice has recently occurred. The double exponential (described more fully in the Analytic Approach to Modeling Retest Effects session) captures the temporal dynamics of clustered bouts of intensive practice (within-bursts) separated by lengthy temporal gaps (between-bursts).

In addition to performance gains attributable to cumulative and recent practice, aging related effects may exert themselves during the interval that separates measurement bursts. To follow the above example, although the older skier is becoming more skilled every season due to her ongoing practice, her potential best speed might be decreasing across seasons because she is aging. To bring this example back to the present study, overt performance on a speeded cognitive tasks could improve across sessions within bursts, due to the benefit of practice, but that estimates of individuals' latent potential (i.e., the asymptote) could reveal slowing across bursts, due to aging or other long-term processes (such as the cumulative effects of stress). We applied the double-exponential learning model to obtain estimates of long-term (across burst) declines in asymptotic performance across bursts that are distinguished from short-term (within burst) retest related gains. Therefore, tests of our primary hypotheses involve evaluating whether GPS predicts changes in asymptotic cognitive performance that are not confounded by retest effects.

Method

Participants

One hundred sixteen older adults from senior residence centers in the Syracuse, NY metropolitan area were recruited for participation in a longitudinal study of health and cognition through advertisements in local newspapers and flyers posted in their residence centers. The average age at baseline was 80.38 years ($SD = 6.40$, range 67–96) and 72% of the sample was female. Participants had on average 14.90 years of education ($SD = 2.40$) and 97% were white, 2% were African American, and 1% Asian American.

Materials

Global perceived stress—We measured global perceived stress using Cohen and colleagues' Perceived Stress Scale (PSS; Cohen et al., 1983). The PSS is a 14-item measure assessing an individual's subjective appraisal of how stressful, overwhelming, and uncontrollable his or her life has been over the past month. Responses to questions such as "In the past month, how often have you felt nervous or 'stressed'?" and "In the past month, how often have you felt difficulties were piling up so high that you could not overcome them?" were made on a 5-point scale (1 = *never* to 5 = *very often*). Positively worded questions were reverse coded, and a total score was obtained by summing the values of all the items, with higher scores reflecting greater levels of perceived stress. Cronbach's alpha for the PSS in this sample was .81.

Depressive symptoms—We used the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977) to assess participants' depressive symptoms. The CES-D is an established measure consisting of 20 items assessing negative mood and depression. Example items include the following: "I feel depressed," and "I am happy". Responses were provided on a 4-point scale (0 = *not at all* to 3 = *very much*) with positively worded items being reverse coded. A total score was calculated by summing the responses on the 20 items, with higher scores indicating greater depressive symptoms. Cronbach's alpha for the CES-D in the current sample was .86.

Health related functional limitations—Participants' physical symptoms reports were assessed daily using a brief version of the Larsen & Kasimatis (1991) physical symptom checklist. This checklist assessed five constellations of symptoms: aches/pain (headaches, backaches, joint pain, and muscle soreness), gastrointestinal symptoms (poor appetite, nausea/upset stomach, constipation/diarrhea), symptoms associated with cardiovascular functioning (chest pain, dizziness, heart pounding), upper respiratory symptoms (cold/flu symptoms, allergy/hay fever symptoms) and a category for "other" physical symptoms or discomforts. Four follow-up questions asked participants whether their physical symptoms limited 1) the amount of time they spent on work or other activities, 2) the extent to which they accomplished less than what they would like, 3) whether they felt limited in the activities they did, and 4) whether they experienced difficulty performing their work or other activities. Participants responded on a three-point scale: "not at all", "slightly", or "very much". Responses were summed across the four items to produce a daily functional limitations score that ranged between 0–12 with higher scores indicating greater limitations.

Somatic health—To measure reported somatic health, participants completed the 12-item Health Survey-Short Form (SF-12; Ware, Kosinski & Keller, 1996). The SF-12 assesses the state of individuals' health and limitations they have experienced for a broad range physical and social activities in the past 4 weeks. For the present analysis, we used the physical health component score, which ranges in scores from 0–100 scale, with higher scores indicating greater physical health and fewer health-related limitations.

Number Match—Two variations of Salthouse's (1996) number match–processing speed task were used as indices of processing speed efficiency. In the easy variation, participants had to make a decision as to whether two 3-digit strings were composed of the same numbers or not. Responses were indicated by pressing the “/” key if the digit strings were the same and the “z” key if the digit strings were different. In the difficult variation, participants had to decide whether two 5-digit strings were composed of the same numbers. Responses were indicated in the same manner as stated earlier, and trials were separated by a 500-ms intertrial interval. Thirty-two trials were completed for both the three- and five-item versions of the task. Response times (RTs) for each variation were calculated by averaging across trials on which a correct response was made; RTs served as the dependent variable.

N-Back—A 1-back and a 2-back version of the n-back task (Awh et al., 1996; Smith & Jonides, 1997) were used as markers of working memory performance. Individuals decided whether the currently presented stimulus was the same or different than a stimulus presented one screen back (1-back) or two screens back (2-back). Stimuli (the digits 1 through 9) were presented one at a time in random order in the center of a computer screen. The stimulus digits appeared in white on a black screen. Individuals were instructed to press one of two keys as accurately and quickly as possible indicating whether the current stimuli was the same (“/” key) or different (“z” key) than the stimulus observed one or two screens back. Stimuli appeared immediately upon a participant's response; no interstimulus interval (ISI) was included. Half of the trials required a response of same, half required a response of different. Three blocks of 20 items were presented for each version of the n-back for a total of 60 trials for each task. The dependent variables were the RTs for each version of this n-back tasks that were calculated by averaging across trials on which a correct response was made.

Keep-Track—A serial counting task (Keep-Track 1) was used to as an index of simple processing speed efficiency. Participants were presented with one of two geometric shapes (a circle and a diamond) on a computer screen, one at a time, in random order. The task was to count only one of the two objects (the circle) while ignoring the other (the diamond). After a shape was displayed, participants were instructed to press the space bar as quickly as possible after they had counted the shape. A new stimulus would appear immediately after the space bar had been pressed (i.e., no ISI). At the end of each trial participants reported the number of targets they counted. Five trials with between 8 and 14 items per trial were administered (for a total of 60 RTs). The average time to count an object served as the dependent measure, and only RTs from trials on which counting was accurate were included.

The hard version of this task consisted of a variation of Garavan's (1998) serial-attention task (Keep-Track 2) which was used to assess the accuracy in which individuals could keep separate running counts of two distinct items simultaneously. Participants were presented with one of two geometric shapes (i.e., a rectangle or a triangle) presented in the center of a computer screen, and the task was to press the space bar each time they counted the displayed object. The following item appeared immediately after the space bar was pressed (i.e., no ISI). Count totals for each shape were reported after each trial ranging between 8 and 16 items in length. Accuracy rates were pooled across trials and were arcsine transformed to normalize the distribution.

Procedure

Participants were given a brief introduction to the study, and the experimenter obtained informed consent as approved by the Syracuse University Institutional Review Board. Participants were told that they were participating in a longitudinal study examining health and cognition in adulthood. Participants were scheduled to visit the research site six times within a 10-day period during which they completed the cognitive tests that were administered by a trained research assistant. These bursts of six daily cognitive assessments were repeated every 6 months for a two-year period, yielding up to five bursts and 30 daily cognitive assessments. Participants completed a GPS and depressive symptoms measure at the start of every burst and they completed a physical symptoms checklist daily within each burst prior to their cognitive assessments. The retention rates were as follows: 78% ($n = 90$) completed all five bursts, 87% ($n = 101$) completed at least four bursts, 88% ($n = 102$) completed three or more bursts and 93% ($n = 108$) completed at least one follow-up burst. Of the 26 participants who missed at least one burst the reason for missing them varied, eight died prior to their next burst, eight dropped out because of serious illness, six canceled appointments because of illness, two canceled appointments because of scheduling conflict, and two moved out of state.

Analytic Approach for Modeling Retest Effects

The following double-negative exponential model, described in detail by Sliwinski, Hoffman, and Hofer (2010), was fit to each cognitive task assessed during the measurement bursts:

$$RT_{ti} = a_i + \Delta a_i (burst_{kj}) + g_i \exp[-r_i (occasion_{ti})] + (Burst_{kj} > 1) + g_i^* \exp[-r_i^* (occasion_{tki})] + e_{ti}$$

According to this model, response time (RT) for a given individual (i), on a given assessment (t) is a function of four parameters, 1) the person's initial or baseline asymptotic response time (a_i), 2) the amount by which their asymptote has changed, $\Delta a_i (burst_{kj})$, and two negative exponential learning functions described next. 3) Learning across all sessions and bursts is accounted for by the term $g_i \exp[-r_i (occasion_{ti})]$ where g_i reflects the difference between initial RT and asymptote (i.e., the total gain in performance due to practice), and a second term, 4) $(Burst_{kj} > 1) \times g_i^* \exp[-r_i^* (occasion_{tki})]$ that captures "warm-up" related improvements in each burst subsequent to the first (i.e., $Burst_{kj} > 1$). That is, some of the practice related performance gains are lost between burst and some re-

learning or “warm-up” occurs at the beginning of follow-up bursts. There were no practice trials for any of the assessments, including the first assessment, therefore warm up effects were not present for the first burst. The r parameter denotes the rate of practice related improvement across all sessions and bursts, and r^* signifies the rate of “warm-up” related gains at follow-up bursts. PROC NLMIXED (SAS Institute, 2008) was used to fit this double-exponential nonlinear model and we outputted random asymptote (a_i) and delta asymptote (Δa_i) parameters for each individual and for each of the six cognitive tasks (see Supplemental Table 1 for a sample generic SAS code). The baseline asymptote (a_i) and change in asymptote (Δa_i) parameters served as the primary outcome variables in the analyses described in the results section.

Results

Baseline Asymptote and Change in Asymptote

Figure 1 illustrates the sample means for each task on each session across all five measurement bursts (i.e., a total of 30 session means for each task). The fitted curves in each panel were obtained by plotting the average of the predicted RTs across all individuals for each session and burst. Visual inspection of the average RTs across all tasks show practice effects (faster RTs) across sessions within burst, especially for the baseline burst, as well as some loss of practice gains (slower RTs) during the initial sessions of follow-up bursts. The fitted curves capture both practice related gains as well as the “warm-up” effects at follow-up bursts. In addition to the practice and warm-up effects, parameter estimates indicated that estimates of asymptotic RT increased significantly across bursts for most of the tasks. Table 1 shows the average baseline asymptotes, change in asymptotes, and standard errors for each cognitive task obtained by fitting the double-exponential model to the measurement burst data. All fixed and random effect parameter estimates from the double-exponential model are provided in a supplementary table (Supplemental Table 2). Estimates on Table 1 show that asymptotic response time (RT) for the easy version of the speed task (Number Match 3) was 1500 milliseconds at the baseline burst, and that asymptotic RT for this task significantly ($p < .05$) increased (i.e., became slower) by an average of 53 milliseconds per burst. Asymptotic RT for the difficult version of the speed task (Number Match 5) was about 3221 milliseconds at baseline and significantly ($p < .05$) increased by an average of 60 milliseconds per burst. RTs for the working memory tasks exhibited a similar pattern of results, with significant ($p < .05$) slowing of asymptotic RT by an average of 9 milliseconds and 29 milliseconds for the N-Back 1 and N-Back 2, respectively. For the Keep-Track task, only easy version (Keep-Track 1) showed significant asymptotic slowing (7 milliseconds per burst). Although the point estimate of asymptotic change for the difficult version of the attention tasks (Keep-Track 2) was positive (13 milliseconds per burst), this effect did not attain statistical significance ($p = .08$).

To facilitate data reduction described below, person-specific random effects for baseline asymptote (a_i) and change in asymptotic RT (Δa_i) were outputted for each of the tasks. These estimates were then standardized to T-scores with a mean of 50 and standard deviation of 10; our analyses henceforth will use these standardized asymptote (a_i) and delta asymptote (Δa_i) parameters as the cognitive indicators.

Descriptive Statistics and Correlations

Descriptive statistics for GPS and age are presented on Table 2. The average depressive score at baseline was 10.41 ($SD = 7.69$). We averaged participants' total daily functional limitation scores across the six days within each burst to obtain burst-level scores of functional limitation; participants reported an average functional limitation score of 5.46 ($SD = 1.67$) during the baseline burst assessment. Average somatic health at baseline was 44.92 ($SD = 9.77$); a supplementary table presents the baseline means and standard deviations of each cognitive task (Supplemental Table 3). Table 3 presents the correlations between each cognitive estimate (i.e., asymptote, delta asymptote), GPS, baseline age, depressive symptoms, health limitations, somatic health, sex, and education. Age was significantly correlated with greater baseline response time and increases in response time for both processing speed tasks (number match tasks) and the easy version of the working memory task (1-back; r s range from 0.21 to .33, $p < .05$). Age was significantly correlated with increases in response time for the easy version of the attention task (Keep-Track 1; $r = .22$, $p < .05$). Age was also positively, but not significantly, correlated with baseline response times for both Keep-Track attention tasks (r s = .18 and .19, $p < .10$). GPS at each burst was not significantly associated with age at baseline but was positively associated with baseline depressive symptoms (r s range from .41 to .50, $p < .05$) as well as with functional limitations (r s range from .22 to .36, $p < .05$) and somatic health (r s range from $-.23$ to $-.35$, $p < .05$). GPS was unrelated to years of education and gender, but sex was associated with cognitive function. Males exhibited faster baseline RTs in the N-Back 1 ($r = -.22$, $p < .05$) and Keep-Track 1 tasks ($r = -.27$, $p < .05$) and exhibited slower cognitive decline in the difficult variation of the processing speed task (Number Match 5; $r = -.24$, $p < .05$). GPS at each burst was also more consistently associated with change in asymptote parameters compared to the baseline asymptote parameters.

Data Reduction

To reduce the number of parameters produced from the double-exponential learning models, we first averaged the standardized parameter estimates of baseline asymptote and asymptotic change across the easy and difficult versions of each of the tasks (i.e., number match, n-back, and keep track) producing three asymptote and three change in asymptote parameters. The correlations among the pooled estimates presented on Table 4 show significant intercorrelations among the baseline asymptote scores (r s range from 0.32 to .49, $p < .05$) and asymptotic change scores (r s range from 0.25 to .37, $p < .05$). We verified the pattern on intercorrelations by conducting an exploratory factor analysis, which suggested a two factor structure with baseline asymptote scores loading on one factor and change in asymptote scores loading on the second factor. We fit a confirmatory factor analysis to formally assess the fit of this two factor structure presented on Figure 2. The first latent factor was comprised by the three baseline asymptote scores and the second factor (labeled as "change in asymptote" in the figure) was comprised by the asymptotic change scores. The two factors were positively correlated with each other ($r = .32$, $p < .05$) indicating that individuals with slower asymptotic RTs at baseline also experienced more rapid increases in their asymptotic RTs across bursts. These two factors (i.e., baseline asymptote and change in asymptote) were used as our outcomes in the remainder of the analyses.

GPS and Asymptotic Performance

Level and change in GPS—To test our predictions regarding whether both level and change in GPS predicted cognitive change, we first fit a linear growth curve centering data at the midpoint (i.e., burst 3) to model average level (intercept) and change (slope) in GPS. The intraclass correlation (ICC) of GPS was .68, indicating that GPS scores were relatively stable across bursts. The mean and variance component for the GPS intercept were significant (Intercept = 18.13, Variance = 30.90, $p < .05$). The fixed GPS slope was positive and significant (Slope = 0.12, $p < .05$) showing modest but reliable increases in average GPS across the two-year follow-up period. However, the variance component of the GPS slope was not significant (Slope Variance = 0.06, $p = .30$), indicating that there were no significant individual differences in rates of GPS change. The lack of significance in the slope variance does not necessarily imply that everyone changed at equal rates, but may indicate a lack of power to detect significant individual differences in rates of GPS change¹. In addition to a linear slope, we also explored the possibility for a quadratic slope in GPS that was not statistically significant.

Perceived stress and asymptotic performance—Figure 3 provides a schematic of the growth model used to examine whether GPS level predicted baseline asymptote and asymptotic change. We also modeled the association between GPS slope and asymptotic change and accounted for the possible association between lower baseline asymptote to changes in GPS. Residual bootstrapping on 5000 bootstrap samples was used to produce bias-corrected standard errors for tests of statistical significance. An initial model indicated that GPS slope was not predicted by baseline asymptotic performance and that the GPS slope did not predict asymptotic change; this was not surprising given that there was no evidence of reliable individual differences in rates of GPS change. Therefore, subsequent models did not include the GPS slope as an outcome or predictor. Table 5 (Model 1) shows results from a model that included average GPS level (i.e., GPS intercept) as a predictor of baseline asymptotic performance and rate of change in asymptotic performance. GPS level was not significantly related to asymptotic RT at baseline ($B = 0.03$ (0.12), *ns*), but was positively related to change in asymptotic performance ($B = 0.31$ (0.15), $p < .05$). These results indicate that those individuals with higher overall stress exhibited more rapid slowing across bursts².

Next, we examined whether the effect of stress level on cognitive slowing remained after adjusting for age, education, sex as well as for baseline levels of depressive symptoms, functional health limitations, and somatic health. We first examined the univariate associations between each of the covariates of interest and baseline asymptotic performance and change in asymptote. Older age was associated with slower baseline performance ($B = 0.27$ (0.12), $p < .05$) and with greater decline in performance ($B = 0.29$ (0.11), $p < .05$). Males had faster baseline RTs ($B = -3.49$ (1.62), $p < .05$) and greater functional limitations were

¹A likelihood ratio test showed that model fit was not statistically different ($p = .23$) between a full model and a model constraining the slope variance to zero.

²It is worth mentioning that the analyses presented on table 5 were also conducted while centering the GPS observations at baseline and that the pattern of results did not change—GPS intercept at baseline, and not slope, predicted more rapid cognitive slowing indicating a true prospective association between greater GPS baseline level and cognitive change overtime.

associated with slower RTs at baseline ($B = 0.71$ (0.41), $p = .08$), though this association was not statistically significant. Poorer somatic health was significantly associated with faster cognitive slowing across bursts ($B = -0.150$ (0.07), $p < .05$). Education and depressive symptoms were unrelated to baseline asymptote and change in asymptote. We decided to retain these variables as covariates due to their association with GPS. Table 5 (Model 2) shows parameter estimates after accounting for demographic variables and baseline depression and health. Older age was significantly associated with more rapid cognitive decline ($B = 0.21$ (0.10), $p < .05$) and positively (but not significantly) associated with slower asymptotic performance at baseline ($B = 0.22$ (0.13), $p < .09$). Men exhibited significantly faster asymptotic RTs ($B = -3.21$ (1.49), $p < .05$), but there were no gender differences in rates of cognitive change. Years of education, depressive symptoms and the health variables were not significantly related either to baseline or rate of change in cognition. GPS remained a significant predictor of increases in the asymptotic slope ($B = 0.27$ (0.12), $p < .05$) across bursts³. We also examined interactions among demographic and mental health variables. These supplementary analyses showed that GPS effects on asymptotic change did not varied across age ($B = .01$ (.03), $p = .97$), gender ($B = .43$ (.36), $p = .97$), years of education ($B = -.03$ (.05), $p = .59$), and depressive symptoms ($B = .01$ (.02), $p = .77$).

Finally, due to the robust conceptual and statistical association among depressive symptom, health, and GPS, we conducted a set of exploratory analyses to determine whether changes in these measures were associated with cognitive change. Neither changes in depressive nor physical symptoms were significantly related to cognitive change.

Discussion

In this study we examined the prospective association between level and change in GPS and cognitive slowing among 116 adults aged 67 years and older, who participated in a two-year measurement-burst study. In support of our first hypothesis, we found that higher average levels of GPS across the study predicted cognitive slowing in a latent factor of attention, working memory, and speed of processing performance and that this effect remained after accounting for age, education, sex, depressive symptoms, and physical health. Contrary to our second hypothesis, however, changes in GPS did not predict changes in cognitive performance.

Global Perceived Stress and Cognitive Change

Despite the relatively small sample size ($n = 116$) and short follow-up period (2 years), the present study found evidence of significant cognitive slowing on most of the cognitive tasks, and that the rate of decline was positively related to both chronological age and GPS. One reason that we were able to detect significant cognitive slowing was that our sample was relatively old (mean age of 80). Another reason is that our use of an intensive measurement burst design afforded increased precision, resulting from the large number of measurements (up to 30) on each individual, as well as applying a model that allowed distinguishing retest

³We examined whether there was differential prediction of GPS intercept on asymptotic slope for the multiple cognitive tasks by fitting a series of models which tested unique paths from stress to each of the cognitive tasks. None of these unique paths were significant, indicating that none of the tasks were differentially sensitive to GPS over and above the shared variance among them.

performance gains from age-related cognitive decline. Specifically, by using a double-exponential learning model which separately modeled practice related gains within bursts and aging related slowing across bursts, we demonstrated significant declines in asymptotic speeded performance on most of the cognitive tasks. Although they present logistical challenges, intensive measurement designs offer potential for addressing some of the important challenges that face longitudinal cognitive aging research, such as accounting for retest effects and sensitive detection of cognitive decline (Sliwinski, 2008; 2011).

The present study is among a few to examine prospective associations between GPS and cognitive performance. Our results are in line with two previous studies that demonstrated a longitudinal association between greater baseline GPS and cognitive decline. Rönnlund and colleagues (2013) examined a decline in self-reported cognition across 15 years and Aggarwal and colleagues (2014) examined declines in objective cognition across a 6 year period. Specifically, Aggarwal and colleagues showed that baseline GPS predicted decline in accuracy performance across various cognitive tasks. Our results extend this previous work in several ways. First, we demonstrated that perceived stress also relates to less efficient (slower) cognitive performance as evidenced by increased reaction time on the attention demanding tasks used in this study. Second, we separately modeled retest effects which allows us to rule out the possibility that stress relates to prospective cognitive decline simply by reducing the ability of individuals to benefit from practice on repeatedly administered cognitive tests. That is, our results indicate that stress predicts cognitive decline independent of retest effects. And third, we examined whether not only level but change in stress related to cognitive decline.

Global Perceived Stress Level versus Change

We found that average GPS level predicted cognitive decline, whereas increases in GPS did not. This raises the question: why does (average) level but not change in GPS predict cognitive decline? One possible answer is that biannual changes in psychological stress might reflect relatively transient fluctuations that do not exert a substantial cumulative effect on cognitive function. That is, increases or decreases in levels of stress are less important than whether the absolute level of psychological stress is high or low. In contrast, higher levels of GPS may reflect relatively enduring chronic sources of stress that may accumulate overtime and impair cognitive function (e.g., Korten, Penninx, Pot, Deeg & Comjies, 2014). The relatively high ICC for GPS (.68) implies stable individual differences in stress levels across the study period and is consistent with this interpretation.

Because the measure of perceived stress used in this study asked participants to report on how they perceived their life over “the past month” it is worth considering why individual perceptions of stress during the last 30 days is both stable over time and predicts future cognitive changes. We consider three possible explanations for this question. One possibility is that greater GPS levels reflect enduring influences of a person’s environment. For instance, living in a disadvantaged neighborhood characterized by low socioeconomic status or high levels of violence may promote concerns or worries about an individual’s safety that, if chronic, can contribute to cognitive decline over time (Baum, Garofalo, & Yali, 1999). A second possibility that is specifically related to older adulthood is the experience of

bereavement, that entails having to adapt to the loss of a spouse, or caring for a spouse who is chronically ill can increase feelings of stress within individuals that can be enduring and promote cognitive decline (Bonanno & Kaltman, 1999; Pinquart & Sörensen, 2003).

Lastly, higher levels of GPS reports may also reflect individual differences in coping mechanisms that may exacerbate stress responses (e.g., Scott, Sliwinski, & Blanchard-Fields, 2013) and accumulate over time. For example, daily diary studies show that individuals who report greater GPS exhibit greater negative emotional reactivity to daily hassles (van Eck et al., 1998; Sliwinski et al., 2009), which in turn predict long-term negative mental and physical health outcomes (Charles et al., 2013; Piazza et al., 2012), and worse cognitive function (Stawski, Mogle, & Sliwinski, 2013). A measure of personality trait neuroticism was not available and thus not accounted for in this study. Trait neuroticism, operationalized as the tendency to experience emotional distress in previous studies, has indeed been shown to predict cognitive decline over time (Wilson et al., 2005). Aggarwal and colleagues (2014) did account for trait neuroticism and found consistent results with greater GPS level predicting cognitive decline across a six year period. Although emotional distress is a construct that is tightly linked with GPS and should be accounted for, we did account for depressive symptoms which capture the tendency to experience negative mood. We show that individual differences in GPS remained a significant predictor of cognitive decline after adjusting for physical and depressive symptoms. Therefore, we can rule out that the association between GPS and cognitive decline was a by-product of depression or health related functional limitations.

Limitations and Future Directions

Some limitations to this study are worth noting. First, one reason for the lack of association between rates of change in GPS and cognitive decline could include our relatively small sample size and short follow up duration, especially if the longitudinal associations between GPS and cognitive decline are relatively small. However, this lack of association between GPS increases and cognitive slowing argues against the possibility that decreases in cognitive abilities may lead to increases in feelings of stress and vice versa. Second, our study included only six assessments of cognition within each burst which may have reduced the precision of estimate asymptotic performance. Third, our failure to detect differential sensitivity of stress effects across different cognitive domains could be due to the considerable amount of shared variance among our tasks given the speeded nature of these tasks. Thus, we cannot conclude that there is equivalence across perceptual speed and working memory, for example. Future studies considering both accuracy and response time across various tasks will be informative in determining differential sensitivity of stress on cognitive domains. Lastly, we did not examine local effects of GPS within each burst. A previous study examining effects within and across bursts found that participants experienced more negative affect during burst in which they reported GPS scores that were greater than their average (Sliwinski et al., 2009). Although such analyses were beyond the scope of our study, future studies should examine whether similar within person effects of GPS on cognition exist in this and other measurement-burst studies.

Other future directions include an analysis of the time course through which GPS may change and exert negative effects on cognitive function. It is possible that GPS changes at a slower pace compared to asymptotic performance. Studies that incorporate multiple time-scales (e.g., across decades, years, months, weeks, days), may thus be necessary to facilitate understanding of the time course through which changes in stress may impair cognitive function. Our sample of participants was relatively homogeneous consisting of well-educated, white female participants residing in senior residence centers, which does not allow for a generalization of results to more racially and socioeconomically diverse individuals. In supplementary analyses we found non-significant moderation of the link between GPS level and cognitive slowing by gender and years of education which was not surprising given the sample's homogeneity. Future studies that incorporate more socioeconomically diverse participants should examine the role of social and demographic moderators of the stress-cognition link.

Identification of modifiable indirect pathways through which perceived stress relates to cognitive function represents an additional future direction. For example, stress-related cognitive interference has been linked with poorer cognitive performance among older adults (Stawski, Sliwinski, & Smyth, 2006). Experiencing intrusive thoughts related to a recent negative event, may momentarily impair cognitive performance by occupying attentional resources (e.g., Sliwinski, Smyth, Hofer, & Stawski, 2006). Over time, a tendency to experience these form of thoughts (i.e., stress-related intrusive and repetitive thoughts), may contribute to chronic stress by prolonging the stress response and result in impaired cognitive function (Brosschot, Gerin, & Thayer, 2006). Individual differences in self-regulatory processes, such as levels of perceived control and mastery may also comprise an important indirect pathway. Greater GPS levels may reduce feelings of control and mastery that may promote cognitive decline possibly due to reduced engagement in health-promoting behaviors such as poor sleep and decreased participation in physical activities (Ballis, Segall, Mahon, Chipperfield, & Dunn, 2001; Lachman & Weaver, 1998).

In spite of its limitations, the present study is the first to demonstrate a prospective association between perceived stress and cognitive slowing. These results were held after accounting for demographic, mental, and physical health variables. The unique association between perceived stress and cognitive slowing emphasizes the utility of measuring perceptions of stress to complete assessments of mere exposure to negative events.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Average and predicted response times from dual-exponential learning model. Average and predicted RTs are presented across sessions separated by bursts. RTs are presented in seconds. The y axes for each plot were modified to optimize representation of data.

Figure 2.

Two-factor latent variable model of cognition ($\chi^2(8) = 7.30, p = .50, CFI = 1.00, RMSEA = 0.00$). Standardized parameters are presented and are all statistically significant ($p < .05$).

Figure 3.

Representation of growth model used to examine the prediction of perceived stress intercept and slope on baseline asymptote and change in asymptote factors. GPS=Global Perceived Stress (denoted by the burst number). Speed = processing speed, WM = working memory, Atten. = attention.

Table 1

Estimated asymptotes and change in asymptotes across bursts

	<u>Baseline Asymptote</u>	<u>Change in Asymptote</u>
	Estimate (SE)	Estimate (SE)
NM3	1500.42 (48.21)*	53.59 (7.44)*
NM5	3220.55 (96.90)*	60.29 (13.22)*
NB1	832.18 (29.71)*	9.06 (4.20)*
NB2	1289.86 (66.14)*	28.69 (10.49)*
KT1	581.61 (20.57)*	6.89 (3.11)*
KT2	984.55 (37.25)*	12.91 (7.34) [†]

Note. Estimates in milliseconds. WM = Working Memory; Atten. = Attention; NM3 = Number Match 3; NM5 = Number Match 5; NB1= N-Back 1; NB2 = N-Back 2; KT1 = Keep-Track; KT2 = Keep-Track 2.

*
p<.05.

[†]
p<.10

Table 2

Descriptive statistics for observed scores at each burst

	Burst 1	Burst 2	Burst 3	Burst 4	Burst 5
GPS (<i>n</i>)	116	105	104	97	92
<i>M</i>	17.26	18.44	18.77	18.41	18.15
<i>SD</i>	6.68	7.29	7.37	7.12	7.38
Age (<i>n</i>)	116	106	105	97	92
<i>M</i>	80.33	80.66	81.19	81.57	82.08
<i>SD</i>	6.43	6.22	6.20	6.16	6.16

Note. GPS = Global Perceived Stress

Table 3

Pearson correlation coefficients

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. GPS1	-												
2. GPS2	.66*	-											
3. GPS3	.58*	.60*	-										
4. GPS4	.70*	.67*	.66*	-									
5. GPS5	.66*	.59*	.66*	.73*	-								
6. NM3 (Asymp)	.05	.04	.06	.04	.07	-							
7. NM5 (Asymp)	.05	.01	-.03	-.05	-.02	.90*	-						
8. NB1 (Asymp)	.03	.05	.03	.04	.07	.57*	.49*	-					
9. NB2 (Asymp)	-.01	-.03	-.03	-.08	-.04	.28*	.24*	.55*	-				
10. KT1 (Asymp)	-.04	.03	-.01	-.01	.08	.41*	.38*	.62*	.34*	-			
11. KT2 (Asymp)	.01	.05	.11	-.02	.07	.52*	.50*	.62*	.34*	.81*	-		
12. NM3 (Delta)	.13	.27	.04	.19†	.09	0.10	.17†	.07	-.11	.12	.08	-	
13. NM5 (Delta)	.13	.34*	.19†	.26*	.23*	.03	.06	.10	-.06	.10	.08	.76*	-
14. NB1 (Delta)	.15	.25*	.10	.15	.21*	.12	.17†	.27*	.05	.35*	.19*	.36*	.29*
15. NB2 (Delta)	.01	.18†	-.05	.08	-.03	-.10	-.010	.10	.04	.11	.01	.27*	.31*
16. KT1 (Delta)	.20*	.21*	.21*	.20*	.18†	.09	.12	.16†	-.06	.14	.14	.25*	.23*
17. KT2 (Delta)	.08	.14	.09	.06	.10	.17†	.23*	.32*	.04	.33*	.23*	.25*	.18
18. Age (Baseline)	.13	.02	.08	.13	.14	.26*	.33*	.21*	.08	.18†	.19†	.29	.21
19. Education (Baseline)	.00	-.04	-.01	.01	-.03	-.06	.06	-.03	.08	.09	.05	-.17†	-.16
20. Sex (0 = female, 1 = male)	.04	-.15	-.11	-.04	-.10	-.07	-.11	-.22*	-.15	-.27*	-.17†	-.17†	-.24*
21. Dep Symp (Baseline)	.50*	.43*	.46*	.41*	.49*	-.09	-.08	-.10	-.01	-.18†	-.08	.07	.13
22. Funct Lim (Baseline)	.32*	.36*	.22*	.29*	.29*	.12	.14	.14	.12	.07	.16	.05	.08
23. Som Hlth (Baseline)	-.24*	-.27*	-.35*	-.23*	-.20†	-.06	-.05	-.14	.00	-.16	-.18	-.10	-.21*

Note. GPS = Global Perceived Stress; Asymp = Asymptote; NM=Number Match; NB=N-Back; KT=Keep Track; Dep Symp=Depressive Symptoms; Funct Lim = Functional Limitations; Som Hlth = Somatic Health.

.01>p<.10
+
>p<.50
*
d

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Table 4

Pearson correlations among pooled estimates

	1	2	3	4	5	6
1. Number Match (Asymptote)	-					
2. N-Back (Asymptote)	.32*	-				
3. Keep Track (Asymptote)	.47*	.49*	-			
4. Number Match (Delta)	.10	-.06	.10	-		
5. N-Back (Delta)	.00	.04	.10	.37*	-	
6. Keep Track (Delta)	.21*	.07	.20*	.26*	.25*	-

Note. Speed = Processing Speed; WM = Working Memory.

* p<.05.

Table 5

Model estimates of GPS predicting baseline asymptote and change in asymptote

	Model 1		Model 2	
	<u>Baseline Asymptote</u>	<u>Change in Asymptote</u>	<u>Baseline Asymptote</u>	<u>Change in Asymptote</u>
	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)
GPS Intercept	0.03 (0.12)	0.31 (0.15)*	−0.06 (0.14)	0.27 (0.12)*
Age			0.22 (0.13) [†]	0.21 (0.10)*
Sex			−3.21 (1.49)*	−1.34 (1.31)
Education Years			0.27 (0.23)	−0.04 (0.24)
Depressive Symptoms (Baseline)			−0.05 (0.11)	−0.03 (0.65)
Functional Limitations (Baseline)			0.73 (0.52)	−0.13 (0.65)
Somatic Health (Baseline)			−0.02 (0.08)	−0.06 (0.07)

Note. GPS=Global perceived Stress. Sex, 0 = female, 1 = male. Residual bootstrap sample size = 5,000.

*
 $p < .05$.

[†]
 $p < .10$.