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Challenge and Hindrance Stressors and Metabolic Risk Factors

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The current study investigates differential relationships between challenge and hindrance stressors and metabolic risk factors using data from the National Survey of Midlife Development in the United States (MIDUS II). Guided by the challenge–hindrance stressor model and the allostatic load model, we test two theoretically driven paths: a direct physiological path and an indirect path via health behaviors (i.e., high-risk eating, cigarette smoking, and alcohol consumption). Challenge stressors versus hindrance stressors were hypothesized to differentially predict health behaviors and metabolic risk factors. Results favor the health behavior-mediated pathway in comparison with the direct physiological pathway. High-risk food consumption served as a link between hindrance stressors and metabolic risk factors. Some evidence supported smoking as a link between hindrance stressors and metabolic risk factors, and alcohol consumption as a link between challenge stressors and metabolic risk factors. The pattern of findings supported the challenge–hindrance distinction, particularly in relation to health behaviors. By combining the challenge–hindrance and allostatic load literatures, our study theoretically and empirically extends knowledge of how work stressors relate to physiological outcomes. Moreover, we also extend the nomological network of challenge and hindrance stressors to behavioral and physiological outcomes.

Keywords: challenge, hindrance, work demands, allostatic load, health behavior

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Work stressors are damaging to worker health and well-being. This relationship is supported by decades of research (Ganster & Rosen, 2013; Häusser, Mojzisch, & Schulz-Hardt, 2011) and founded upon several theoretical perspectives (Hobfoll, 1989; Karasek, 1979). Indeed, national initiatives and organizational policies and practices have been developed that target reduction of work stressors and/or their associated strains (e.g., Total Worker Health; Anger et al., 2015).

Although concerns with regard to the negative impact of work stressors are warranted, recent findings suggest not all work stressors are equally deleterious. Meta-analytic and primary study investigations support a distinction between challenge and hindrance workplace stressors (Cavanaugh, Boswell, Roehling, & Boudreau, 2000; Rodell & Judge, 2009). Challenge stressors are defined as stressors associated with gains, such as learning and goal achievement, and hindrance stressors are defined as stressors that interfere with or prevent growth and accomplishment (Cavanaugh et al., 2000). Consequently, challenge stressors are associated with beneficial outcomes, such as higher job satisfaction and job performance, relative to hindrance stressors (Lepine, Podsakoff, & Lepine, 2005; Podsakoff, Lepine, & Lepine, 2007). The challenge–hindrance pat-

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tern suggests challenge stressors have salutary effects on worker well-being, whereas hindrance stressors have relatively more negative well-being effects. However, much of the challenge–hindrance distinction has been established using psychological constructs. It is therefore unclear whether these two stressors also predict divergent behavioral or physiological outcomes. In addition, much of the literature is based on cross-sectional self-report designs, which is subject to same-source bias. As a result, our knowledge with regard to the pathways linking challenge and hindrance stressors to strains is narrow in scope.

Based on a time-lagged, multisource research design, the purpose of the current study is to expand challenge and hindrance stressor theory and research. Specifically, we investigate the differential relationships that challenge and hindrance stressors have with a key physiological strain, metabolic risk factors. Using the allostatic load model (McEwen, 1998; McEwen & Seeman, 1999) as a guiding theoretical framework, we test and compare two pathways between stressors and metabolic risk factors. First, we examine a path in which challenge and hindrance stressors directly relate to metabolic risk factors. Second, we examine an indirect path, in which challenge and hindrance stressors are related to metabolic risk factors through health behaviors. A visual representation of the study hypotheses is presented in Figure 1.

The current study contributes to the literature in several ways. First, we combine the challenge–hindrance and allostatic load literatures to yield new theoretical and empirical insights into both. We extend the challenge–hindrance literature by testing novel behavioral and physiological outcomes. We also extend the allostatic load and occupational health literature by bringing attention to the importance of stressor type. Literature on work stressors and behavioral and physiological health is almost exclusively based on the job demands–control model or the effort reward imbalance model (Juster, McEwen, & Lupien, 2010; Kivimäki, Singh-Manoux, Nyberg, Jokela, & Virtanen, 2015; Siegrist & Rödel, 2006). These theories group job stressors together, overlooking the importance of stressor type (Dawson, O'Brien, & Beehr, 2016). This lack of differentiation could contribute to why research on behavioral and physiological outcomes associated with work stressors has produced weak and inconsistent findings (Bergmann, Gyntelberg, & Faber, 2014; Ganster & Rosen, 2013; Siegrist & Rödel, 2006). For example, Bergmann and colleagues' (2014) review showed inconsistent relationships between work demands and a variety of metabolic risk factors. They concluded certain work stressors (e.g., job insecurity, low social capital, and injustice) were most strongly associated with physiological outcomes such as elevated blood pressure. These stressors are all indicative of hindrances. Overall, these findings suggest distinguishing challenge and hindrance stressors is important for understanding the relationship between workplace stress and health.

Second, we compare two distinct, theoretically driven pathways between work stressors and physiological health: the direct physiological path (McEwen & Seeman, 1999) and the health behavior-mediated path (Taylor, Repetti, & Seeman, 1997). The latter has rarely received attention, as studies primarily assess bivariate relationships between job stressors and either health behavior or physiological health (Bergmann et al., 2014; Ganster & Rosen, 2013; Juster et al., 2010; Siegrist & Rödel, 2006). Both pathways are untested within the challenge–hindrance literature. In testing the direct physiological and indirect health behavior pathways, we are able to identify the efficacy of each in isolation and in relation to one another. In addition, we test multiple health behaviors simultaneously, allowing us to examine the relative efficacy of multiple plausible paths. Thus, our study advances theoretical understanding of how challenge and hindrance stressors are linked to physiological health.

Third, our study methodologically advances both the challenge–hindrance and allostatic load literatures. Challenge–hindrance studies are primarily cross-sectional and use self-reported stressors and self-reported psychological outcomes. This is a notable limitation in that same-source bias is known to amplify stressor–strain relationships (Burke, Brief, & George, 1993; Ganster & Rosen,

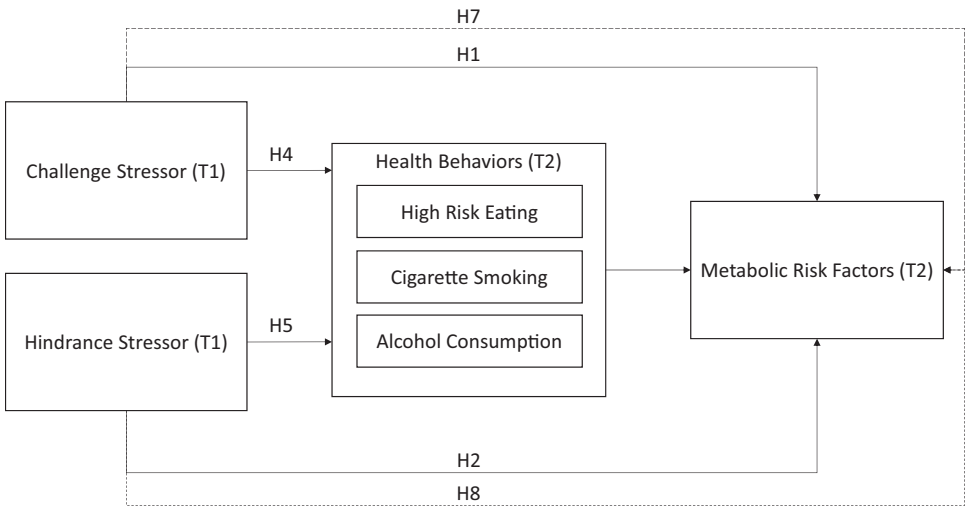

Figure 1. Visual representation of the study hypotheses. T1 = variables measured at Time 1. T2 = variables measured at Time 2. Dashed lines represent hypothesized indirect effects. Comparison hypotheses (H3, H6) not shown for parsimony.

2013). Our study uses a lagged multisource design to connect challenge and hindrance stressors with objectively measured physiological outcomes. In doing so, our study builds upon previous work by mitigating same-source bias.

Fourth, our research contributes to the nascent literature that connects work stressors to physiological health implications, and more specifically to metabolic risk factors (Chandola, Brunner, & Marmot, 2006; Ganster & Rosen, 2013; Vrijkotte, Van Doornen, & De Geus, 1999). Metabolic risk factors are well-established predictors of cardiovascular and metabolic diseases and conditions, such as diabetes, obesity, and stroke (Ervin, 2009; Mozaffarian et al., 2015). Such conditions are among the leading health problems in the United States and around the world, especially among the aging population (Aguilar, Bhuket, Torres, Liu, & Wong, 2015). For example, it is estimated that in the United States, about 11% of the population deals with diabetes (Guariguata et al., 2014), more than 795,000 people experience a stroke every year (Mozaffarian et al., 2015), and about 35% of men, 40% of women, and 35% of adults above the age of 65 are obese (Centers for Disease Control & Prevention, 2016; Fakhouri, Ogden, Carroll, Kit, & Flegal, 2012). Given national concern for worker health and the aging workforce, our study has implications for understanding how work stressors contribute to critical worker health issues within contemporary U.S. society.

Hypothesis Development

Challenge and Hindrance Stressors

Cavanaugh and colleagues (2000) first developed the challenge versus hindrance stressor distinction in an effort to clarify the relationships between work stressors and work-related outcomes such as job satisfaction and job search. They found different types of work stressors could be sorted reliably into two categories: challenge stressors or hindrance stressors. Challenge stressors are associated with gains, such as learning and goal achievement. Challenge stressors are theoretically grounded in stress appraisal research, which suggests some stressors may be viewed as motivating (i.e., eustress; Selye, 1955) and may result in positive emotions or attitudes, such as satisfaction, happiness, or a sense of achievement (Lazarus & Folkman, 1984). Challenge stressors are considered stressful, but are also likely to stimulate motivation and positive emotion, as they provide potential opportunities for growth and demonstration of competence (Crawford, Lepine, & Rich, 2010). Examples of challenge stressors include time pressure and high levels of job responsibility. Hindrance stressors interfere with or prevent growth and accomplishment. Hindrance stressors are theoretically akin to distress (Selye, 1955) or hassles (Lazarus & Folkman, 1984), which are negatively evaluated stressors that demotivate and evoke negative emotions such as frustrations or anger. Hindrance stressors are also considered stressful, but thwart learning or achievement opportunities and are perceived as frustrating nuisances (Crawford et al., 2010). Examples of hindrance stressors include organizational politics, hassles, job insecurity, and poor supervision (Cavanaugh et al., 2000; Lepine et al., 2005; Zhang, Lepine, Buckman, & Wei, 2014).

Challenge and hindrance stressors consistently demonstrate a distinct pattern of relationships with work outcomes. Challenge stressors are associated with desirable outcomes such as increased job satisfaction (Cavanaugh et al., 2000), motivation, performance (Lepine et al., 2005), organizational citizenship behaviors (Rodell & Judge, 2009), engagement (Crawford et al., 2010), resilience (Crane & Searle, 2016), and decreased withdrawal and intention to quit (Boswell, Olson-Buchanan, & Lepine, 2004). In contrast, hindrance stressors typically relate to undesirable outcomes, such as lower performance, organizational commitment, and job satisfaction (Lepine et al., 2005; Podsakoff et al., 2007) and higher burnout (Crawford et al., 2010) relative to challenges.

The challenge–hindrance pattern is similar for psychological strain-related outcomes. By definition, both challenge and hindrance stressors may elicit psychological strain (Boswell et al., 2004). However, challenge–strain relationships tend to be weaker than hindrance–strain relationships. This pattern has been replicated across a variety of psychological strain outcomes, including burnout (Crawford et al., 2010), psychological strain (Lepine et al., 2005; Podsakoff et al., 2007), perceived stress (Edwards, Franco-Watkins, Cullen, Howell, & Acuff, 2014), anxiety (Tuckey, Searle, Boyd, Winefield, & Winefield, 2015; Wood & Michaelides, 2016), anger (Rodell & Judge, 2009; Tuckey et al., 2015), emotional exhaustion, and physical symptoms (Webster, Beehr, & Love, 2011).

The differential relationships between challenge stressors and strain and hindrance stressors and strain is based on research investigating psychological strain. However, psychological strain is empirically and conceptually distinct from physiological strain (Ganster & Rosen, 2013), so it remains unclear whether the pattern of differential relationships extends to physiological outcomes. Further, theoretical perspectives on the challenge–hindrance distinction use psychological constructs such as energy, affective, or cognitive resources (Lazarus & Folkman, 1984; Lepine et al., 2005; Podsakoff et al., 2007; Selye, 1955), which do not readily explain to relationships with physiological strain. We introduce the allostatic load model to the challenge–hindrance literature as our guiding theoretical framework for examining relationships between challenge and hindrance stressors and physiological strain outcomes.

The Allostatic Load Model

The allostatic load model is a contemporary stress model that explicates how psychosocial stressors get "under the skin" and ultimately develop into diseases and disorders (Juster et al., 2010). The model suggests physiological changes are adaptive, and physiological systems are dynamic. Although efficient physiological change (i.e., allostasis) is considered adaptive and healthy, repeated activation of physiological systems can be physiologically taxing, resulting in suboptimal physiological functioning that is termed allostatic load (McEwen, 1998). Allostatic load develops into diseases and disorders through a three-phase temporal process. First, stressors trigger immediate physiological changes in the neurological and endocrine systems, resulting in elevated hormones (epinephrine, norepinephrine, and cortisol). The hormones produced immediately during acute stressor exposure are known as *primary mediators* (Juster et al., 2010; McEwen & Seeman, 1999). Primary mediators prepare the body to cope with the stressor by regulating cardiovascular, immune, and metabolic systems (e.g., increasing heart rate, releasing glucose into the blood stream). Chronic exposure to stressors can overactivate primary mediator systems, resulting in dysfunctional patterns, such as

failure to return to prestressor levels (McEwen & Seeman, 1999). Over time, dysregulation manifests in *secondary outcomes*, which are subclinical levels of metabolic, immune, and cardiovascular functioning such as elevated cholesterol, C-reactive protein levels, or elevated resting blood pressure (Juster et al., 2010). Secondary outcomes are considered risk factors that may evolve over time into *tertiary outcomes*, or major diseases and disorders such as cardiovascular disease, depression, or morbidity (Juster et al., 2010).

A small body of research has examined work stressors and allostatic load. This research shows work stressors such as long work hours and workload are associated with increases in the cortisol awakening response (Chida & Steptoe, 2009), blood pressure (Landsbergis, Dobson, Koutsouras, & Schnall, 2013), metabolic risk factors (Li et al., 2007), and allostatic load (Juster et al., 2010). However, effect sizes are often small and several studies fail to find significant relationships between work stressors and allostatic load composites or individual physiological strain indicators (Ganster & Rosen, 2013). For example, a meta-analysis of laboratory studies found a meta-analytic correlation of −.23 between work-related stressors and acute sympathetic nervous system reactivity (Chida & Hamer, 2008). Cross-sectional and lagged studies find effect sizes similar in magnitude when using job stress to predict allostatic load and indicators such as cholesterol and development of Type 2 diabetes (Chandola et al., 2006; Heraclides, Chandola, Witte, & Brunner, 2009; Li et al., 2007). There are two possible reasons for small and nonsignificant findings that we address in the current research.

First, failure to consider the challenge–hindrance stressor distinction may contribute to the weak, inconsistent relationships between work stressors and physiological strain found in previous research. Challenge and hindrance stressors distinguish stressors that are nonthreatening and beneficial (i.e., challenges) from stressors that are threatening and harmful (i.e., hindrances; Cavanaugh et al., 2000). Threatening stressors yield larger primary physiological stress responses than less threatening stressors, resulting in more extreme fluctuations across physiological systems (Dickerson & Kemeny, 2004; McEwen, 1998; McEwen & Seeman, 1999). Repeated exposure to extreme fluctuation amplifies wear and tear on primary and secondary outcome systems relative to mild fluctuations associated with nonthreatening stressors.

Second, failure to theoretically isolate the most appropriate physiological systems influenced by work stressors may contribute to the observed weak relationship between work stressors and physiological strain. Because allostatic load involves a complex interplay of multiple stress-related symptoms, it is typically operationalized as a composite variable consisting of indicators from both primary and secondary systems (Juster et al., 2010; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). However, recent studies show stressor effects may vary by system, and therefore deeper insights might be gained by focusing on specific systems rather than on a total allostatic load index (Juster et al., 2010). To address the need to better isolate specific physiological systems, we focus on metabolic risk factors for several reasons. First, secondary systems (such as the metabolic system) are best aligned with our interest in chronic challenge and hindrance stressor exposure, compared with more acutely reactive primary system indicators. Second, metabolic risk factors are a key, consistent linking mechanism between work stressors and tertiary outcomes (i.e., major diseases and disorders) when compared with other systems (Ganster & Rosen, 2013; Juster et al., 2010). Moreover, metabolic risk factors are empirically distinct from and explain variance above and beyond other neuroendocrine, immune, and cardiovascular indicators in outcomes such as cardiovascular disease and performance on complex cognitive tasks (McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012; Seeman, McEwen, Rowe, & Singer, 2001).

On the basis of theoretical reasoning and empirical research on challenge and hindrance stressors and the allostatic load model, we propose challenge and hindrance stressors are directly related to metabolic risk factors. Because both challenge and hindrance stressors are expected to elicit strain, we expect both types of stressors are positively associated with metabolic risk factors. Because hindrance stressors are harmful and threatening and challenge stressors are beneficial and nonthreatening, we hypothesize this positive relationship is stronger for hindrance stressors compared with challenge stressors.

Hypothesis 1: Challenge stressors positively relate to metabolic risk factors.

Hypothesis 2: Hindrance stressors positively relate to metabolic risk factors.

Hypothesis 3: Hindrance stressors have a stronger positive relationship with metabolic risk factors compared with challenge stressors.

Health Behaviors as Mediators

The allostatic load model proposes physiological dysregulation can develop not only due to physiological stress responses, but also as an indirect outcome of behavioral stress responses (McEwen, 1998; McEwen & Seeman, 1999). Stressor-induced health behavior changes are considered a form of allostasis in which an individual attempts to adapt his or her behavior to cope with a stressor. Despite short-term coping benefits, repeated engagement in such behaviors may result in dysregulation of secondary outcome systems and ultimately disease (McEwen, 1998). We focus on high-risk eating, cigarette smoking, and alcohol consumption.

Research investigating health behaviors as mediating variables between stressors and physiological strain is sparse. Within the allostatic load literature, health behaviors are most typically used as control variables (see Juster et al., 2010, for a review). The extant research examining health behaviors and either work stressors and/or physiological strain predominantly originates in epidemiology and focuses on main effects with little theoretical underpinning. This body of research yields tentative evidence for direct relationships. For example, some research shows work stressors are related to health behaviors such as cigarette smoking and alcohol consumption (Frone, 1999; Siegrist & Rödel, 2006) and food consumption (O'Connor, Jones, Conner, McMillan, & Ferguson, 2008). However, results across studies are mixed, indicating moderators are likely present (Frone, 1999; Siegrist & Rödel, 2006). We propose the challenge–hindrance distinction as a moderator that may help explain relationships between workplace stressors and health behaviors.

Ample research suggests high-risk food consumption and cigarette smoking are associated with increased metabolic risk, although the strength of associations increase with dosage (Grundy et al., 2005; Oh et al., 2005; Riccardi, Giacco, & Rivellese, 2004; Zhu, St-Onge, Heshka, & Heymsfield, 2004). Findings suggest

alcohol consumption is consistently associated with metabolic risk but that the association is curvilinear, such that moderate use is beneficial for metabolic health, whereas heavy use is detrimental (Alkerwi et al., 2009; Baik & Shin, 2008; Freiberg, Cabral, Heeren, Vasan, & Curtis Ellison, 2004). Thus, we investigate a direct association between alcohol and metabolic risk, but we do not propose a specific directionality.

We directly test this mediated pathway based on propositions of the allostatic load model, which frames health behaviors as a form of allostasis and source of allostatic load (McEwen, 1998). Like psychological or physiological strain, health behaviors are considered a stress response in the allostatic load framework (McEwen & Seeman, 1999). Therefore, we expect health behaviors will follow the challenge–hindrance pattern. Because challenge and hindrance stressors both elicit strain responses, we hypothesize both challenge and hindrance stressors positively relate to engaging in health behaviors used to cope with stressors (high-risk eating, smoking, and alcohol consumption). Because hindrance stressors are threatening and challenge stressors are nonthreatening and beneficial, we hypothesize that this relationship is stronger for hindrance stressors compared with challenge stressors. Further, chronic engagement in these health behaviors is a form of allostatic load. Consequently, we hypothesize that engagement in such behaviors will be associated with physiological dysregulation in the form of increased metabolic risk factors. Thus, the relationship between challenge and hindrance stressors and metabolic risk factors will also be mediated by health behaviors.

Hypothesis 4: Challenge stressors positively relate to (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption.

Hypothesis 5: Hindrance stressors positively relate to (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption.

Hypothesis 6: Hindrance stressors have a stronger positive relationship with (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption compared with challenge stressors.

Hypothesis 7: There is an indirect positive relationship between challenge stressors and metabolic risk factors through increased (a) high-risk eating and (b) cigarette smoking, and an indirect negative relationship between challenge stressors and metabolic risk factors through increased (c) alcohol consumption.

Hypothesis 8: There is an indirect positive relationship between hindrance stressors and metabolic risk factors through increased (a) high-risk eating and (b) cigarette smoking, and an indirect negative relationship between hindrance stressors and metabolic risk factors through increased (c) alcohol consumption.

Method

Participants

We tested the hypotheses using data from the second wave of the National Survey of Mid-Life Development in the United States (MIDUS; Ryff et al., 2012). Funded by the MacArthur Midlife Research Network, MIDUS included a random sample of midlife adults between the ages of 25 and 74 across the United States, contacted via random digit dialing. All MIDUS II participants participated in a general survey phase, consisting of a phone survey and a self-administered mail-in survey (N = 4,963). Following the general survey phase, participants were offered participation in a variety of subsequent phases (e.g., cognitive testing, daily diary, biomarkers). For the current study, we use participants who participated in Phase 4, the biomarker study (Ryff, Seeman, & Weinstein, 2013). This phase involved a two day, one night visit to one of the three participating medical clinic sites to assess physical and biological indicators of health (e.g., health behaviors, immunological health, cardiovascular health, stress response). On Day 1, staff members recorded participant medical history, conducted a bone densitometry scan, and conducted a physical exam. Fasting blood samples were collected from each participant before breakfast on the second day of the hospital stay. All samples were collected and processed using a standardized protocol and stored in a −60°C to −80°C freezer until shipped on dry ice to be analyzed in the MIDUS lab. Samples were kept at −65°C until assayed. Participants from the general survey phase were compensated up to $60, and participants who completed the biomarker phase were compensated an additional $200.

Of the 1,255 participants who participated in the general survey and biomarker phase, we excluded those who did not have data on the variables of interest (526 participants removed), were not working (216 additional participants removed), worked 20 hr per week or less (52 additional participants removed), reported unemployment within the past year (133 additional participants removed), and reported no personal wages in the past calendar year (10 additional participants removed). These exclusion criteria ensured all participants in the sample had sufficient long-term exposure to challenge and hindrance stressors at work.

Study analyses were based on a final sample of 318 participants. Approximately half of the participants were male (51.90%) with an average age of 49.32 years (SD = 8.61, minimum = 34, maximum = 81). Participants primarily identified themselves as White (92.50%), followed by Black and/or African American (2.80%), and other (0.90%). Most participants were well-educated (modal education level was a four-year college degree), and employed in a professional specialty (31.70%), executive, administrative, and managerial (21.60%), or administrative support (14.90%) occupations. Other occupations included operator, laborer, or military (6.60%), precision production, crafts, and repair (7.6.60%), service (6.00%), or sales occupations (6.00%). Participants worked 43.06 hr per week on average (SD = 8.57). Mean household income was $95,747/year (median = $80,250/year, SD = $61,069/year, minimum = $9,000/year, maximum = $300,000/yr).

Timing of Measures

Variables from the current study were taken from two studies within MIDUS II: the general survey phase and the biomarker phase. Challenge and hindrance stressors were measured during the MIDUS II general survey phase, which was collected between 2004 and 2006. Demographic factors (e.g., gender, ethnicity, work status) were also assessed in the general survey phase. The remaining measures were assessed during the biomarker phase, which took place between 4 and 62 months after completing the general survey phase (mean time lag = 27.55 months, SD = 15.17). High-risk eating, cigarette smoking, and alcohol consumption were measured during the medical history exam on the first

day of the biomarker phase. Metabolic risk factors were assessed during the physical exam on the first day and using fasting blood draw assays on the morning of the second day.

Measures

Challenge stressors. Challenge stressors were measured with three items ($\alpha = .63$). These items were adapted from Karasek and colleagues' (1998) Job Content Questionnaire. The items were consistent with the conceptual definition of challenge stressors and tapped into job responsibility and time pressure (Cavanaugh et al., 2000; Lepine et al., 2005; Rodell & Judge, 2009; Zhang et al., 2014). Items were rated on a 5-point frequency scale that ranged from 1 (*never*) to 5 (*all of the time*). Items were identified and validated using subject matter expert ratings and a quantitative Mturk validation study (see online supplemental materials for details and a full list of items).

Hindrance stressors. Hindrance stressors were measured using eight items ($\alpha = .72$). Two items were from the Job Content Questionnaire (Karasek et al., 1998) and the remaining six were developed for the MIDUS II study. Items reflect work overload, interruptions, unfair treatment, harassment, and poor supervision. These items are consistent with the conceptual and operational definitions of hindrance stressors (Cavanaugh et al., 2000; Lepine et al., 2005; Rodell & Judge, 2009; Zhang et al., 2014). Two items were rated on a 5-point frequency scale that ranged from 1 (*never*) to 5 (*all of the time*), and the remaining six items were rated on a 5-point frequency scale that ranged from 1 (*never*) to 5 (*once a week or more*). Items were identified and validated using subject matter expert ratings and a quantitative Mturk validation study (see online supplemental materials for details and a full list of items).

High-risk eating. High-risk eating was measured with a three-item formative scale. Items were chosen based on empirical research on metabolic risk factors (Yoo et al., 2004) and national recommendations from the U.S. Department of Health and Human Services and U.S. Department of Agriculture (2015). High-risk foods include average number of daily sugared beverages, average number of times fast food was consumed per week, and average number of times beef or high-fat meat was consumed per week. All items were scored on 5-point frequency scales, with greater values indicating higher consumption frequency. The daily scale ranged from 1 (*none*) to 5 (*5 or more servings per day*), and weekly scales ranged from 1 (*none*) to 5 (*5 or more times per week*).

Cigarette smoking. Cigarette smoking was measured with one item, "do you currently smoke cigarettes regularly?" Participants responded either no (coded as 0) or yes (coded as 1).

Alcohol consumption. Alcohol consumption was based on one item. Participants indicated on a frequency scale how often they drank alcoholic beverages during the past month on average. The scale ranged from 1 (*never drink*) to 6 (*everyday*).

Metabolic risk factors. Metabolic risk factors included a combination of eight general, lipid, and glucose indicators of metabolic allostatic load: body mass index, waist-hip ratio, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein (HDL) cholesterol, fasting glucose levels, homeostasis model of insulin resistance, and glycosylated hemoglobin levels (HbA1c). These indicators have been used in published studies in which the MIDUS II data set was used to examine allostatic load; the indicators in the current study were chosen a priori to represent the metabolic system (Gruenewald et al., 2012). Factor analysis studies confirm such indicators are empirically distinct from alternative system (cardiovascular, immune) indicators (McCaffery et al., 2012), and our data show small-to-moderate correlations among the eight indicators (average $r = .20$, $\alpha = .66$). Further, these indicators include known risk factors that make up metabolic syndrome (National Heart, Lung, and Blood Institute, 2016). Consistent with operationalization of allostatic load in the extant literature (Juster et al., 2010), participant scores for each indicator were dichotomized based on whether or not they fell into the high-risk quartile of the sample. Quartiles were computed separately for men and women to account for sex differences in metabolic risk factors (see Table 1), consistent with previous allostatic load research (Seeman et al., 2004). There are documented gender differences in metabolic risk factors, such that risk factors are more prevalent for women than for men (Beigh & Jain, 2012; Regitz-Zagrosek, Lehmkuhl, & Weickert, 2006), although gender differences vary by the type of metabolic risk factor (Regitz-Zagrosek et al., 2006). Within our data set, independent t tests showed waist-hip ratio, $t(316) = 16.49$, $p < .01$, triglycerides, $t(316) = 6.05$, $p < .01$, HDL cholesterol, $t(316) = -8.34$, $p < .01$, fasting glucose, $t(316) = 3.87$, $p < .01$, and homeostasis model of insulin resistance, $t(316) = 2.26$, $p = .02$, significantly differed by sex. For all biomarker components except HDL cholesterol, scores that fell within the upper quartile were coded as 1, indicating the participant had high risk levels, and scores that did not fall within the upper quartile were coded as 0, indicating no presence of risk. For HDL cholesterol, scores that fell within the lower quartile were coded as 1, indicating high risk, and scores that did not fall within the lower quartile were coded as 0, indicating no presence of risk. Dichotomized scores were then averaged to create a score for metabolic risk factors, ranging from 0 to 1, with higher values indicating more risk.

Demographic variables. Demographic variables such as sex, age, gender, work hours, race, education, income, occupation, and industry were assessed with single-item measures in the general phase phone and self-administered questionnaire surveys. Participants reported whether or not they were taking four classes of medication (blood pressure, cholesterol, corticosteroids, and/or depression). For parsimony, the correlation matrix includes an index for the number of participants who were taking at least one of these mediations (1 = yes, 0 = no). Each type of medication was entered separately as control variables in the supplemental analyses to avoid confounding the unique effects of each medication (1 = yes, 0 = no). We also created an index of hereditary risk for metabolic-related diseases and disorders. Participants who had a blood relative diagnosed with heart disease, hypertension, high cholesterol, circulation problems, diabetes, or stroke were coded as 1 for hereditary risk, and participants who did not have a blood relative with these diagnoses were coded as 0 for hereditary risk.

Results

Hypothesis Testing

All descriptive statistics, correlations, and path analyses for hypothesis testing were conducted using the lavaan package in R (Rosseel, 2012; Version 3.2.3, R Core Team, 2015). Descriptive statistics and correlations among study variables were computed to

Table 1
High-Risk Cutoff Values for Metabolic Risk Indicators

Indicator	Clinical cutoff	Gruenewald et al., 2012 (N = 1,000–1,008)	Current study	
			Men (N = 165)	Women (N = 153)
Body mass index[a]	≥25.00	≥32.31	≥32.53	≥32.78
Waist-hip ratio[b]	≥.90 (M), ≥.85 (W)	≥.97	≥1.00	≥.87[c]
Triglycerides (mg/dL)[d]	≥200.00	≥160.00	≥190.50	≥130.00[c]
Low-density lipoprotein cholesterol (mg/dL)[e]	≥160.00	≥128.00	≥132.00	≥125.50
High-density lipoprotein cholesterol (mg/dL)[e]	<40.00	≤41.37	≤35.00	≤48.00[c]
Fasting glucose (mg/dL)[f]	≥125.00	≥105.00	≥106.00	≥98.00[c]
Insulin resistance[g]	≥2.60	≥4.05	≥4.27	≥3.68[c]
Glycosylated hemoglobin[h]	≥6.50	≥6.10	≥6.04	≥6.02

Note. (M) = cutoff for men, (W) = cutoff for women.
[a] Centers for Disease Control. (2015). *About adult BMI*. Retrieved from https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/. [b] World Health Organization. (2011). *Waist circumference and waist-hip ratio*. Retrieved from http://apps.who.int/iris/bitstream/10665/44583/1/9789241501491_eng.pdf. [c] Independent samples t test showed significant differences between men and women, $p < .05$. [d] Berglund, L., Brunzell, J. D., Goldberg, A. C., Goldberg, I. J., Sacks, F., Murad, M. H., & Stalenhoef, A. F. H. (2012). Evaluation and treatment of hypertriglyceridemia: An Endocrine Society clinical practice guideline. *Journal of Clinical Endocrinology and Metabolism, 97,* 2969–2989. [e] National Heart, Lung, and Blood Institute. (2001). *ATP III at a glance: Quick desk reference*. Retrieved from https://www.nhlbi.nih.gov/health-pro/guidelines/current/cholesterol-guidelines/quick-desk-reference-html. [f] Mayfield, J. (1998). Diagnosis and classification of diabetes mellitus: New criteria. *American Family Physician, 58,* 1355–1362. [g] Ascaso, J. F., Pardo, S., Real, J. T., Lorente, R. I., Priego, A., & Carmena, R. (2003). Diagnosing insulin resistance by simple quantitative methods in subjects with normal glucose metabolism. *Diabetes Care, 26,* 3320–3325. [h] World Health Organization. (2011). *Glycated haemoglobin (HbA1c) for the diagnosis of diabetes*. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK304271/#__NBK304271_dtls__

examine variable relationship patterns (see Table 2). Correlations show some initial support for the challenge–hindrance pattern, such that hindrances are associated with greater metabolic risk (continuous operationalization r = .14, clinical cutoff operationalization r = .11), unhealthy eating (r = .19), and smoking (r = .12), whereas challenges are not (correlations with these variables range from −.02 to .02). To test the study hypotheses, we conducted path analysis using WLSMV estimation (diagonal least squares weighted), which accounts for the ordinal nature of smoking and alcohol consumption behavior. Our approach allowed us to examine the theoretically driven multivariate relationships between all variables simultaneously. The model included all direct and indirect hypothesized paths (Hypotheses 1, 2, 4, 5, 7, and 8). In addition, path analysis allowed us to parsimoniously compare the strength of model paths (Hypotheses 3 and 6).

Significance for direct and indirect effects (Hypotheses 1, 2, 4, 5, 7, and 8) was interpreted using the bias-corrected percentile (BCa) bootstrapped 95% confidence intervals (CIs; 5,000 samples; Preacher, Rucker, & Hayes, 2007). To directly compare the strength of challenge and hindrance paths (Hypotheses 3 and 6), we tested a series of models in which the paths from challenge stressors were constrained to be equivalent to paths from hindrance stressors for each of the behavioral mediators and the metabolic risk factors. We tested constraints separately to ensure significant constraints were not masked by nonsignificant constraints. A significant decrease in χ^2 fit from the hypothesized model to the constrained model indicated the constrained parameters statistically differed from one another.

Standardized parameter estimates are displayed in Figure 2. Fit statistics for the hypothesized path model indicated sufficient fit, $\chi^2(3) = 4.02$, $p = .26$, confirmatory fit index = .98, root mean square error of approximation = .03, standardized root mean square residual = .02. Hypothesis 1, which stated challenge stressors positively relate to metabolic risk factors, was not supported by the direct effects in the model ($\beta = -.01$, $p = .87$). Hypothesis 2 stated hindrance stressors positively relate to metabolic risk factors. This hypothesis was also not supported by the direct effects in the model ($\beta = .00$, $p = .97$). Hypothesis 3 stated hindrance stressors have a stronger positive relationship with metabolic risk factors compared with challenge stressors. There was no significant decrease in model fit after constraining the path from hindrance stressors to metabolic risk factors to be equal to the path from challenge stressors to metabolic risk factors, $\Delta\chi^2(1) = 0.01$, $p = .94$. Hypothesis 3 was therefore not supported.

Hypotheses 4a–4c proposed direct positive relationships between the challenge stressors and the health behaviors (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption. Challenge stressors were not associated with high-risk eating ($\beta = -.01$, $p = .85$) or cigarette smoking ($\beta = .01$, $p = .93$). Challenge stressors were associated with alcohol consumption in the expected direction ($\beta = .13$, $p = .03$). Thus, Hypotheses 4a and 4b were not supported, and Hypothesis 4c was supported. Hypotheses 5a–5c proposed direct positive relationships between hindrance stressors and the health behavior mediators of (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption. Hindrance stressors were significantly related to high-risk eating ($\beta = .20$, $p < .01$) and smoking ($\beta = .13$, $p = .03$). Hindrance stressors were not significantly related to alcohol consumption ($\beta = -.03$, $p = .60$). Thus, Hypotheses 5a and 5b were supported, and Hypothesis 5c was not supported.

Hypotheses 6a–6c proposed the relationships between hindrance stressors and (a) high-risk eating, (b) cigarette smoking, and (c) alcohol consumption would be stronger than the parallel relationships between challenge stressors and health behaviors. Results indicated hindrance stressors had a stronger positive relationship with high-risk eating behavior, $\Delta\chi^2(1) = 5.47$, $p = .02$, compared with challenge stressors. Thus, results supported Hypothesis 6a. Hindrances and challenges were not differentially related to cigarette smoking,

Table 2
Means, Standard Deviations, and Correlations for the Primary Analyses

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Age	49.32	8.61	—															
2. Work hours	43.06	8.57	−.11	—														
3. Gender[a]	0.48	0.50	−.01	−.21*	—													
4. Race[b]	0.05	0.21	−.05	−.05	.07	—												
5. Education[c]	8.31	2.42	−.06	.06	.04	−.02	—											
6. Income	95,747	61,070	−.00	.27*	−.09	.00	.32*	—										
7. Lag Phase 1 to Phase 4[d]	27.55	15.17	−.11	.01	.07	−.07	.18*	.05	—									
8. Hereditary risk	0.96	0.20	−.03	.12*	.04	−.03	.05	−.05	.08	—								
9. Medication use	0.45	0.50	.27*	.05	.02	.02	.01	.08	.01	.12*	—							
10. Challenge stressor	3.77	0.62	−.14*	.25*	.03	−.10	.26*	.17*	−.01	.01	−.01	—						
11. Hindrance stressor	2.19	0.60	−.18*	.11	−.07	.07	−.13*	−.15*	−.04	.05	−.01	.11	—					
12. Metabolic risk factors[e]	0.26	0.24	.06	.01	−.00	.04	−.25*	−.04	−.13*	.04	.12*	−.03	.07	—				
13. Metabolic risk factors z[e]	0.00	0.60	.05	.12*	−.40*	.00	−.17*	−.03	−.16*	.08	.14*	−.02	.14*	.77*	—			
14. Metabolic risk factors C[e]	0.29	0.21	.04	.10	−.37*	−.03	−.18*	.03	−.09	.09	.13*	−.00	.11*	.73*	.85*	—		
15. High−risk eating	2.37	0.70	−.20*	.13*	−.26*	.10	−.22*	−.03	−.10	−.03	−.03	.02	.19*	.28*	.39*	.33*	—	
16. Smoking[f]	0.10	0.30	−.01	.04	−.15*	−.02	−.08	−.09	−.06	−.04	−.04	.01	.12*	.12*	.19*	.13*	.08	—
17. Alcohol consumption	2.60	1.51	.13*	.14*	−.20*	−.06	.09	.14*	.08	.01	.10	.13*	−.01	−.16*	−.10	−.08	−.08	.07

Note. $N = 318$ for all variables except income and race ($N = 308$).
[a] 0 = Male, 1 = Female. [b] 0 = White, 1 = Non-White. [c] 8 = 2-year college degree, 9 = 4-year college degree. [d] Lag time in months. [e] Metabolic risk factors computed as the average of the dichotomized high-risk empirical quartiles computed separately for males and females for each indicator. Metabolic risk factors z is computed as the average of the standardized values (*z* scores) for each indicator. Metabolic risk factors C (clinical) is computed as the average of the dichotomized clinically defined high-risk groups. [f] 0 = Not a current smoker, 1 = Current regular smoker.
* $p < .05$.

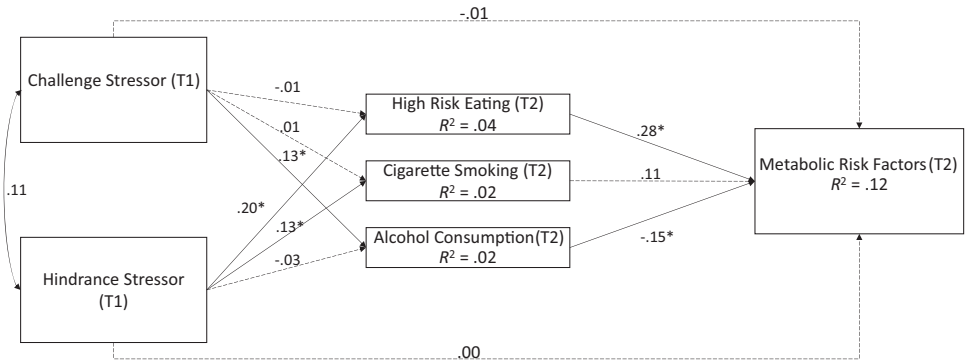

Figure 2. Hypothesized model parameter results. Standardized parameter estimates are presented. Indirect paths are not displayed to ease interpretation. * $p < .05$. Dashed lines represent nonsignificant parameters. T1 = variables measured at Time 1. T2 = variables measured at Time 2.

$\Delta\chi^2(1) = 1.99$, $p = .16$, or to alcohol consumption, $\Delta\chi^2(1) = 3.20$, $p = .07$. Hypotheses 6b and 6c were not supported.[1]

Hypothesis 7 proposed significant indirect effects from challenge stressors to metabolic risk factors through the health behavior mediators. The indirect effect from challenge stressors to metabolic risk factors via alcohol consumption was significant (standardized effect = −.020, unstandardized indirect effect = −.008, 95% CI [−.021, −.001]). The indirect effects from challenge stressors to metabolic risk factors through high-risk eating (standardized effect = −.003, unstandardized effect = −.001, 95% CI [−.016, .012]) and smoking (standardized effect = .001, unstandardized indirect effect = .000, 95% CI [−.004, .008]) were nonsignificant. Hypothesis 7 was partially supported. Hypothesis 8 proposed significant indirect effects from hindrance stressors to metabolic risk factors through health behavior mediators. High-risk eating mediated the negative relationship between hindrance stressors and metabolic risk factors (standardized effect = .056, unstandardized effect = .022, 95% CI [.009, .041]). The indirect effects for hindrance stressors through smoking (standardized effect = .014, unstandardized effect = .006, 95% CI [.000, .018]) and alcohol consumption (standardized effect = .005, unstandardized effect = .002, 95% CI [−.004, .011]) included zero and were therefore not significant. Hypothesis 8 received partial support.

Supplemental Analyses

Alternative metabolic risk factor operationalizations. We recognize our dichotomous measure of metabolic risk may truncate meaningful variability in participant scores on the metabolic risk factor components. To addresses this issue, different operationalizations are often tested in the allostatic literature to check the robustness of findings and ensure results are not dependent on one particular operationalization (Juster et al., 2010). We ran our hypothesized model using *continuous* metabolic risk factor scores, as well as metabolic risk factors computed using *clinical cutoff* scores. We computed continuous scores by creating *z* scores for each indicator and averaging the standardized indicators. Results from this analysis did not differ from our original results with two exceptions. First, the direct relationship from alcohol consumption to metabolic risk became nonsignificant (β = −.09, $p = .08$), and consequently the indirect effect from challenge stressors to metabolic risk via alcohol consumption became nonsignificant (standardized effect = −.012, unstandardized effect = −.012, 95% CI [−.038, .000]). The direct association from challenge stressors to alcohol consumption remained significant (β = .13, $p = .03$). Second, the direct relationship from smoking to metabolic risk (β = .13, $p = .03$), and the indirect relationship from hindrance stressors to metabolic risk via smoking became significant (standardized effect = .022, unstandardized effect = .022, 95% CI [.003, .062]). Clinical cutoff scores used for the MIDUS data in previous studies are displayed in Table 1. We created dichotomous high/low risk categories for each metabolic risk component based on whether the participant met or exceeded the clinical cutoff for each component; dichotomous indicators were averaged. When using the clinical cutoff scores, two changes occurred. The direct relationship between alcohol consumption and metabolic became nonsignificant (β = −.08, $p = .16$), although the relationship between challenges and alcohol consumption remained significant (β = .13, $p = .03$). Consequently the indirect effect from challenge stressors to metabolic risk through alcohol consumption also became nonsignificant (standardized effect = −.010, unstandardized effect = −.004, 95% CI [−.013, .001]). Additionally, the direct relationship between smoking and metabolic risk became significant (β = .12, $p = .048$).

Time lag variation. There was wide variation in the amount of time between participant reports of challenge and hindrance stressors and assessment of the behavioral mediators and metabolic risk factors (between 4 and 62 months). Given this time lag variability, we might expect a simplex pattern such that participants with shorter time lags have stronger relationships between

[1] We selected our measure of alcohol consumption to reflect habitual alcohol consumption that we might expect with chronic stressor exposure, in line with our theoretical rationale. However, other measures of alcohol consumption include the average number of drinks on days when individuals drink, binge drinking (>5 drinks on one occasion), or an average of weekly frequency and average number of drinks on days when drinking (Frone, 1999, 2015). We tested each of these three measures of alcohol consumption (measured at Time 2) in the hypothesized model. None of the associations between alcohol consumption measures and challenges, hindrances, or metabolic risk were significant. Further, contrasts comparing hindrance and challenge paths to alcohol consumption were not significantly different. Full results are available from the first author upon request.

the Time 1 challenge and hindrance stressors and the Time 2 behavioral mediators and metabolic risk factors than participants with longer time lags. Alternatively, we might predict that time increases the potential for stressor accumulation, such that participants with longer time lags have stronger relationships between Time 1 challenge and hindrance stressors and Time 2 behavioral mediators and metabolic risk factors compared with participants with shorter time lags. To test whether time lag influenced results, we first entered time lag as a control predictor for each behavioral mediator and metabolic risk factor in the hypothesized model. Two parameters changed statistical significance. Specifically, the relationship between alcohol consumption and metabolic risk factors became nonsignificant ($\beta = -.03$, $p = .61$), and consequently the indirect effect from challenge stressors to metabolic risk factors through alcohol consumption also became nonsignificant. Additionally, the direct effect from smoking to metabolic risk ($\beta = .14$, $p = .02$), as well as the indirect effect from hindrance stressors to metabolic risk through smoking became statistically significant (standardized effect = .018, unstandardized effect = .007, 95% CI [.001, .022]). All other results were consistent with the hypothesized model results. We also tested time lag as a moderator for the relationships between challenge and hindrance stressors and each behavioral mediator and metabolic risk factor. To achieve model convergence, all variables were estimated as continuous and maximum likelihood estimation was used with bias-corrected bootstrapped CIs (5,000 iterations). None of the interaction parameters were significant ($p > .05$). Overall, we conclude time lag variation had minimal systematic influence on our results.

Demographic covariates. Typically allostatic load research controls for a number of variables that are known to predict physiological health and tertiary disease endpoints, such as age, race, socioeconomic status, medication use, and/or hereditary risk (Ganster & Rosen, 2013; Juster et al., 2010). We ran the hypothesized model, entering age, race (White vs. Non-White), income, education, use of blood pressure, cholesterol, depression, or corticosteroid medication, and hereditary risk covariates as predictors of metabolic risk factors. To achieve model convergence, all variables were estimated as continuous and maximum likelihood estimation was used with bias-corrected bootstrapped CIs (5,000 samples). This model allowed us to assess whether hypothesized relationships held after controlling for demographic variation in the outcomes. Only education ($\beta = -.21$, $p < .01$) and blood pressure medication ($\beta = .14$, $p = .02$) explained significant variability in metabolic risk factors. After entering demographic covariates, the relationship between hindrance stressors and smoking became nonsignificant ($\beta = .10$, $p = .09$), and the relationship between smoking and metabolic risk became significant ($\beta = .14$, $p < .01$). All other results remained the same.

Controlling for Time 1 smoking and alcohol consumption. We tested for the possibility that challenges and hindrances predict change in health behaviors. Identical smoking and alcohol consumption measures were available in the MIDUS Phase 1 survey (smoking Time 1–Time 2 $r = .83$, alcohol consumption Time 1–Time 2 $r = .81$). We added smoking at Time 1 as a predictor of smoking at Time 2, and alcohol consumption at Time 1 as a predictor of alcohol consumption at Time 2 to our hypothesized model. All challenge and hindrance associations with smoking and alcohol consumption were nonsignificant ($p > .05$). We therefore did not find support for the association between challenges and hindrances and change in smoking or alcohol consumption.

Discussion

The current study investigated differential relationships between challenge and hindrance stressors and metabolic risk factors with time-lagged data from MIDUS II. Our results show challenge and hindrance stressors are differentially related to eating, smoking, and alcohol consumption, but not directly to metabolic risk factors. Specifically, hindrances were associated with high-risk eating and smoking but not alcohol consumption, whereas challenges were associated with alcohol consumption, but not high-risk eating or smoking. In addition, the results support an indirect path between hindrance work stressors and metabolic risk factors as mediated by high-risk eating. Some results support tenuous pathways between hindrance stressors and metabolic risk via cigarette smoking. Similarly, some results support alcohol consumption as a linking mechanism for the relationship between challenge stressors and metabolic risk. With the exception of the indirect effects via cigarette smoking and alcohol consumption, our results were consistent across different operationalizations of metabolic risk factors and after modeling variations in time lag.

Our study shows support for the indirect pathway linking workplace stressors and metabolic risk, as opposed to a direct pathway. There was a significant bivariate relationship between hindrance stressors and metabolic risk factors in the expected direction when using a continuous and clinical cutoff measure of metabolic risk factors; no significant bivariate association was found for challenge stressors. However, this relationship was not significant in the path analyses after controlling for health behaviors. In combination, these results indicate work stressors are indirect, distal predictors of metabolic risk via mediators such as health behaviors. This finding helps to clarify how work stressors are related to physiological outcomes. Additionally, these results suggest previous bivariate studies may have found weak associations due to inclusion of challenge stressors and/or failure to consider health behavior mediators. Consistent with the allostatic load model (McEwen, 1998; Taylor et al., 1997), our findings suggest health behaviors, particularly high-risk eating habits, may be forms of behavioral strain that occur in response to stressors, which in turn contribute to physiological dysregulation.

The pattern of relationships observed between challenge and hindrance stressors and health behaviors demonstrated additional support for proposed challenge–hindrance distinction. Results suggest individuals who are exposed to chronic hindrance stressors are more likely to eat foods high in fat and/or sugar, whereas exposure to challenge stressors had no such relationship with eating behavior. Planned contrasts show hindrance stressors are more strongly associated with high-risk food consumption relative to challenge stressors. This pattern falls in line with the allostatic load model and the challenge–hindrance framework (Cavanaugh et al., 2000; McEwen, 1998). It may be that individuals feel a stronger need to behaviorally cope with chronic hindrance stressors compared with challenge stressors, due to their frustrating quality (McEwen, 1998). As an alternative explanation, hindrance stressors may be more likely to drain cognitive and emotional energy compared with challenge stressors (Crawford et al., 2010; Rodell & Judge, 2009). Such energy is necessary for self-control in

making healthy food choices (Baumeister, Vohs, & Tice, 2007). Although a similar pattern was found for smoking cigarettes, the relationship was small in magnitude and not significant across all robustness checks. Nonetheless, results indicate smoking may be a pathway by which work stressors are associated with metabolic risk.

A different pattern was found for alcohol consumption. Challenge stressors were associated with increased alcohol consumption, whereas hindrance stressors were unrelated. We also found evidence that challenge stressors were negatively associated with metabolic risk factors through increased alcohol consumption. However, the direct and indirect relationships associated with alcohol and metabolic risk were again small in magnitude, and not significant across all robustness checks. The possible indirect negative effect is attributable to the negative association between alcohol consumption and metabolic risk factors found in our data. Research indicates alcohol consumption has a curvilinear relationship with metabolic risk, such that light drinking is associated with decreased metabolic risk factors, and heavy drinking is associated with increased metabolic risk factors (Alkerwi et al., 2009; Baik & Shin, 2008; Freiberg et al., 2004). Given that the alcohol consumption frequency in our sample was low and positively skewed, our results are consistent with these findings. Consistent with the allostatic load model, alcohol use is typically framed as a coping response to work stressors (Frone, 1999; Wang, Liu, Zhan, & Shi, 2010). However, empirical support for this notion is surprisingly mixed (Frone, 1999), and more recent studies show results may depend on nuanced environmental and individual factors such as the time of day (Frone, 2008), environmental norms and support (Wang et al., 2010), individual reactions and expectations (Frone, 2015; Frone, 2016), or alcohol consumption operationalization (Frone, 2015). Our study reveals that one potential reason for mixed previous findings could be failure to consider the type of work stressor. Motivational theories of alcohol consumption suggest both positive and negative states predict alcohol use (Cooper, Frone, Russell, & Mudar, 1995; Frone, 2015; Kuntsche, Knibbe, Gmel, & Engels, 2005). Given that challenge stressors are associated with motivation and positive attitudes, such as job satisfaction (Cavanaugh et al., 2000; Lepine et al., 2005), our findings suggest challenge stressors may trigger positive motivational pathways that facilitate frequent alcohol consumption.

Theoretical Implications

Our study has several theoretical implications. First, we combine two currently independent literatures (i.e., challenge–hindrance stressors and allostatic load) to yield new insights in the broader work stress literature. By combining these theories, we extend the nomological network of challenge and hindrance stressors to behavioral and physiological strain outcomes. Thus, our findings demonstrate further validity for challenge and hindrance stressor constructs. Combining these literatures also is also a start toward clarification of the relationships between work stressors and additional physiological strain indicators, such as cortisol, blood pressure, or immune markers (Ganster & Rosen, 2013). Our findings demonstrate that the type of work stressor matters and should be considered in future studies. Second, we delineate a theoretical pathway connecting challenge and hindrance stressors via health behaviors, particularly high-risk food consumption. In testing this mediated pathway, we add credence to propositions of the allostatic load model (McEwen, 1998; Taylor et al., 1997) and extend theoretical knowledge of how work stressors are differentially related to physiological strain. Further, we demonstrate that mediating mechanisms are necessary to connect workplace stressors with distal physiological outcomes.

Practical Implications

Our study suggests challenge and hindrance stressors have implications for employee health behaviors and metabolic health. Further, hindrance stressors appear to have relatively more negative consequences compared with challenge stressors, and hindrance stressors primarily damage metabolic health via high risk eating. Eating behavior is largely under the control of individuals. Workers could support one another in making healthy choices and try to encourage more restorative behaviors as a stress response, such as exercise or mindfulness-based practices (Good et al., 2015). Organizations could support these efforts by providing access to healthy foods (e.g., onsite vending machines can be stocked with healthier alternatives to candy bars and sugary drinks such as nutritional bars and nut mixes). Our findings are also important for organizations, given the steadily aging workforce (Aguilar et al., 2015) and substantial worker costs associated with metabolic risk factors such as obesity and stroke (Steinberg, Scott, Honcz, Spettell, & Pradhan, 2015). Organizations can make efforts to reduce hindrance stressors such as unfair treatment and interruptions. For example, leaders can create and promote policies and practices that encourage fair treatment and reduce the risk of mistreatment (Tepper, 2007). Alternatively, research shows training and visual cues can help to reduce interruptions (Relihan, O'Brien, O'Hara, & Silke, 2010). Our results suggest such interventions will not only improve performance and employee psychological health, but also health behaviors and ultimately metabolic health.

Limitations and Future Directions

Study limitations reveal promising avenues for future research. First, as is often the case when using archival data from a large-scale probability study, not all measures are ideal. For example, cigarette smoking was measured on a dichotomous scale, rather than a frequency scale, which may truncate variance and the potential to find associations among study variables. Stronger relationships between challenge and hindrance stressors and health behaviors may emerge with frequency scales. Similarly, our measure of chronic alcohol consumption differs from other types of alcohol consumption, such as binge drinking or problem drinking. Another consideration is generalizability. Our sample may be biased, as healthy individuals or those with the time and means to participate may be more likely to continue to opt into the MIDUS II and biomarker substudy. Given this study is the first to link challenge–hindrance stressors with health behaviors and metabolic risk factors, we recommend replication with other assessments of challenge and hindrance stressors to determine generalizability. This is important in that a variety of demands have been used to represent challenge and hindrance stressors, yet our study only taps into a subset (e.g., time pressure for challenges, and interpersonal interactions for hindrances).

Much of the founding work in this area is based on meta-analytic research, which combines different job demands into a single effect size (Crawford et al., 2010; Lepine et al., 2005). Thus, the specific demands researchers use to assess challenge and/or hindrance stressors varies from study to study. For example, studies have focused exclusively on time pressure (Pearsall, Ellis, & Stein, 2009; Tuckey et al., 2015; Widmer, Semmer, Kälin, Jacobshagen, & Meier, 2012), task complexity (Tuckey et al., 2015), workload (Richardson, Yang, Vandenberg, DeJoy, & Wilson, 2008), and/or responsibility (Webster et al., 2011) to assess challenge stressors. Researchers have taken a similar approach to operationalizing hindrance stressors (Pearsall et al., 2009; Tuckey et al., 2015; Webster et al., 2011). Our results indicate that time pressure and high job responsibility combined with hindrances that primarily reflect interpersonal interactions produce the expected challenge–hindrance differences in relation to behavioral health and metabolic risk. This specificity is a strength in that our results are conceptually precise, yielding specific theoretical and practical conclusions for two stressors that are well-known and prevalent in today's workplace. However, replication across a wider variety of challenges and hindrances is needed.

Many of the effect sizes found in the present study were small in magnitude. In particular, the indirect effects on metabolic risk were weak, and for alcohol consumption and smoking behavior these effects were not robust to minor changes in our analytic procedure. This is not surprising given that health behaviors and metabolic risk are determined by numerous external and internal factors. Although the challenge and hindrance distinction is important to consider, additional moderators (e.g., expectations around coping with health behaviors; Frone, 2015) and mediators (e.g., fatigue) in the work and nonwork interface could also be considered. Overall, a more thorough understanding of both the direct associations in each stage of our mediated model could be expanded with more elaborate models.

Although our workplace stressors are assessed several months before behavioral and physiological strains, our design lacks longitudinal assessment. Our supplemental analysis did not find support for the idea that challenges and hindrances are associated with change in smoking or alcohol consumption, likely due to the consistency in health behaviors over time. Therefore, we cannot conclude a change in workplace stressors produces a change in strains over time, but rather that workplace stressors are associated with behavioral and physiological strain. Lack of support for longitudinal effects is likely due to the relatively stable and in part genetically determined nature of health behaviors and physiological functioning in adulthood (Chassin, Presson, Pitts, & Sherman, 2000; Frone, 2013). In chorus with recently published reviews (Ganster & Rosen, 2013; Ilies, Aw, & Lim, 2016), we suggest future research build upon these findings by examining the accumulation of workplace stressors and related strains over time using true longitudinal designs. Research that focuses on times of career and developmental transitions, such as early career/teenage years, or midlife adults transitioning into different career stages may be particularly likely to see change in both work stressors and health behaviors.

The challenge–hindrance framework is also limited in that it does not consider individual perceptions of or reactions to stressors (Webster et al., 2011). Individual reactions to hindrance or challenge stressors may differ as a function of individual differences, or within person over time as individuals learn from and adapt to stressors in their environment (Meurs & Perrewé, 2011). Our theoretical rationale implies health behaviors are a coping response used in reaction to chronic stressor exposure. However, we did not directly assess cognitive processes underlying this response, such as if individuals expect health behaviors to alleviate work-related stressors. Future research might use diary methodology to better capture whether there are differential cognitive and learning processes associated with challenges and hindrances over time.

Finally, our study does not assess primary mediator systems (i.e., sympathetic, parasympathetic, and immune system stress responses). Dysregulation of primary mediator systems theoretically mediates the relationship between stressors and secondary outcomes such as metabolic risk factors (McEwen, 1998; McEwen & Seeman, 1999). However, little evidence for this process exists in the literature (Ganster & Rosen, 2013; Juster et al., 2010). Primary mediator systems are those involved in acute stress response (Juster et al., 2010). Because we focus on chronic, rather than acute, stressors and assess outcomes months after stressors are evaluated, we do not expect to see meaningful variation in primary mediator systems in relation to challenge and hindrance stressors. Examining differential acute primary mediator responses in relation to challenge and hindrance stressors would be a meaningful next step. For example, an episodic approach could be taken in which individuals record blood pressure, heart rate, and cortisol following a work stressor episode. The episode could be both subjectively appraised and objectively categorized as a challenge or as a hindrance, and differential relationship patterns assessed. Recent work shows support for associations between hindrance stressors and physiological outcomes using this type of design. For example, Yang, Bauer, Johnson, Groer, and Salomon (2014) reported incidents of injustice increased salivary cortisol. Similarly, Shockley and Allen (2013) found work–family conflict episodes related to elevated heart rate.

Conclusion

Our study investigates the differential predictive relationships between challenge and hindrance stressors and metabolic risk factors. Overall, we find challenge and hindrance stressors differentially relate to engagement in health behaviors, particularly eating high-risk food, smoking, and alcohol consumption. Hindrance stressors are typically associated with deleterious health behaviors, whereas challenge stressors are unrelated to health behaviors with the exception of alcohol consumption. Further, hindrance stressors are positively associated with metabolic risk factors via consumption of high-sugar, high-fat foods. Our findings pave the way for additional research that investigates the link between work stressors and physiological health based on challenge and hindrance stressor theory.

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