This Job Is (Literally) Killing Me: A Moderated-Mediated Model Linking Work Characteristics to Mortality
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RESEARCH REPORT

This Job Is (Literally) Killing Me: A Moderated-Mediated Model Linking Work Characteristics to Mortality

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Research in applied psychology has found that job demands affect employee health outcomes. However, less is known about the mechanisms linking job demands to more distal health outcomes, such as death, and how other job characteristics (i.e., job control) and individual differences (i.e., cognitive ability) might buffer these relationships. Accordingly, we drew from theories from the work stress and medical literatures to argue that job control and cognitive ability moderate the positive relationship between job demands and the probability of mortality, via the mediating effects of poor physical (i.e., allostatic load) and mental health (i.e., depression) indicators. We tested our hypotheses using a 20-year time-lagged design in a sample of 3,148 individuals with mental health data (and a subsample of 754 with physical health data) from the Midlife in the United States Survey. We found that job control and cognitive ability buffered the positive relationship between job demands and poor mental health. Unexpectedly, we found that job control, but not cognitive ability, moderated the relationship between job demands and physical health, such that job demands were related to better physical health under conditions of high control, and unrelated to physical health under conditions of low control. In turn, physical and mental health mediated the moderated (by job control and cognitive ability) job demands-mortality relationship. Our findings suggest that job demands relate to death differentially via physical and mental health, and that these relationships are bounded in unique ways by job control and cognitive ability.

Keywords: stress, job characteristics, depression, allostatic load, mortality

Supplemental materials: http://dx.doi.org/10.1037/apl0000501.supp

Research in applied psychology and related fields has long recognized the inextricable link between individuals’ work context and health (Bliese, Edwards, & Sonmentag, 2017; Ganster & Rosen, 2013; Karasek, 1979). This research has largely focused on the effects that job characteristics have on proximal psychological (e.g., burnout; Crawford, LePine, & Rich, 2010) and physiological (e.g., blood pressure; Ilies, Dimotakis, & De Pater, 2010) health outcomes. This has led scholars to call for an expansion of this line of inquiry to include more distal health outcomes, such as serious psychological and physiological ailments and, ultimately, death (cf. Ganster & Rosen, 2013). For example, in their review of research on work stress published in the Journal of Applied Psychology, Bliese et al. (2017, p. 399) contended that “despite the expansion of strain-related variables over the last 100 years, few studies link work stressors to hard medical outcomes over time” and called for research linking stress processes to medically recognizable diseases (see also Demerouti, Bakker, Nachreiner, & Schaufeli, 2001; Heaphy & Dutton, 2008).

Recently, Gonzalez-Mulé and Cockburn (2017) addressed these calls by examining how job demands and control relate to mortality. They found that job demands were positively related to the likelihood of death in low control jobs, and negatively related to the likelihood of death in high control jobs. Despite the strengths of their study, two important unanswered questions remain regarding the job demands-mortality relationship. First, do job demands affect all individuals’ likelihood of death equally, or are some individuals at higher risk than others? In particular, cognitive ability, defined as the general ability to reason, solve problems, and learn (Gottfredson, 1997), is an individual difference construct central to applied psychology that, when high, could help employees meet the challenges of demanding jobs. Conversely, employees with low cognitive ability might be more susceptible to the deleterious health outcomes associated with job demands. Second, what are the mediating processes that explain why job demands influence death and, moreover, do job demands relate differen-
itially to these processes, depending on characteristics of the job or person? In other words, do job control and cognitive ability function similarly as buffers of the relationships between job demands, physical and mental health outcomes, and death? Investigating these questions will provide important guidance to practitioners regarding the potential benefits of job redesign interventions, changes to hiring and employee assignment practices, and employee well-being initiatives.

Thus, the purpose of our study is to examine when, for whom, and why job demands relate to mortality. Our study makes several contributions. First, we integrate the job demands-control (JDC; Karasek, 1979) and allostatic load (AL; McEwen & Stellar, 1993) models to suggest that the strength of the relationship between job demands and the likelihood of death depends on the levels of job control, and that these effects are mediated by more proximal physical and mental health outcomes. Relatively few studies have explicitly linked the work context to distal physiological outcomes such as death, with the few studies on this topic having been conducted in the epidemiology and medical literatures, albeit with some noteworthy methodological deficiencies (Ganster & Rosen, 2013; Gonzalez-Mulé & Cockburn, 2017). Second, we extend the JDC and AL models by suggesting that cognitive ability also buffers the effects of job demands on physical and mental health and, ultimately, death. There have been no studies (to our knowledge) investigating how cognitive ability buffers the effects of job demands on death, nor testing a theoretical model with intervening mechanisms linking work characteristics to death. We tested our theoretical propositions in a sample that is significantly younger than that in Gonzalez-Mulé and Cockburn’s (2017) study that, with a mean age of 44 years, more closely resembles the distribution of the age of employees in the United States. Further, we used a time lag of approximately 1 decade between measurement episodes. Thus, our study provides initial answers to the questions of when, for whom, and why job demands relate to death, while also providing important suggestions to managers hoping to improve their employees’ health. Given that the World Health Organization declared the relation between work-related stress and employee health to be one of the biggest challenges of the 21st century (Houtman, Jettinghoff, & Cedillo, 2007), exploring the relation similarly as buffers of the relationships between job demands, physical and psychological strain indicators (Demerouti et al., 2001) and subsequent anxiety and strain resolution. However, in cases of low control, individuals’ goal pursuit is stymied. This leads to an anxious state in which people do not have the necessary resources to respond to the demands of their job (Selye, 1978) and subsequent anxiety and strain resolution. Further, depression is a major mental illness which is often used in the medical literature as an indicator of overall mental health (Ganster and Rosen (2013).

Prevalence rates of depression are relatively high (between 12.8% and 16.6% across the life span; Blackmore et al., 2007) and depression contributes to a growing global increase of costly and damaging mental health disorders (World Health Organization, 2018).

### Linking Work Stress to Strain: The Job Demands-Control Model

According to the JDC model, strain results from the combination of the demands and control in one’s job. Job demands are defined as the psychological demands faced by employees in the form of concentration requirements, workload, and time pressure, while job control indicates the amount of discretion employees have to make decisions, schedule their work, and the like (Griffin & Clarke, 2011; Karasek, 1979). The JDC model suggests that job demands are “instigators of action” (Karasek, 1979, p. 287) that draw attention to the contrast between one’s current state and one’s desired end state. For example, receiving a challenging work assignment simultaneously causes individuals to be motivated toward goal attainment, while also causing anxiety about the work necessary to accomplish the assignment. Job control is a resource people can use to manage job demands, such that when control is high, demanding jobs allow individuals to learn and display competence because they have the necessary resources to accomplish their goals and meet their job’s requirements (Karasek, 1979; Ryan & Deci, 2000). This, in turn, leads to increased psychological fulfillment derived from the accomplishment of difficult work tasks (Selye, 1978) and subsequent anxiety and strain resolution. However, in cases of low control, individuals’ goal pursuit is stymied. This leads to an anxious state in which people do not have the necessary resources to respond to the demands of their job (Hobfoll, 1989), which can lead to the exacerbation of proximal physical and psychological strain indicators (Demerouti et al., 2001). If left unresolved, the JDC model suggests that these proximal strain indicators (e.g., anxiety; fatigue) will lead to the

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**Figure 1.** Theoretical model linking job demands to mortality. Control variables are not presented above for the purpose of clarity. Dashed lines designate the time point in which data was collected. Each MIDUS study was conducted 10 years after the previous study.
development of more distal strain outcomes, such as the deterioration of physical and mental health (Blackmore et al., 2007).

Past research has found support for the JDC model. Specifically, the extant evidence suggests that job demands are more strongly, positively related to strain when paired with low control, while high control attenuates the detrimental effects of job demands on strain (e.g., Fox, Dwyer, & Ganster, 1993; Ganster & Rosen, 2013; Ilies et al., 2010).

**Hypothesis 1:** Job control moderates the positive relationships between job demands and poor (a) physical and (b) mental health, such that the relationships will be stronger when job control is low than when it is high.

Although the JDC model is the dominant theoretical framework linking work stress to strain (Ganster & Rosen, 2013), it only focuses on job control as a moderator of job demands, and does not address the role of individual differences in the stressor-strain relationship. Past research has extended the JDC model by focusing on traits related to self-beliefs, such as self-efficacy (Jex & Bliese, 1999) and optimism (Xanthopoulou, Bakker, Demerouti, & Schaufeli, 2007). These studies suggest that when self-beliefs are high, the demands-strain relationship is buffered. This is because individuals with high self-beliefs perceive themselves as being capable of performing their jobs well and are thus less likely, compared to someone with low self-beliefs, to perceive demands as threatening to their demonstration of competence (Jex, Bliese, Buzzell, & Primeau, 2001).

Despite the important contribution of these studies, scholars (e.g., Xanthopoulou et al., 2007) have suggested that future research should also examine individual differences that reflect individuals’ ability to cope with demands, rather than simply their belief in their ability to do so. This distinction harkens to the idea of “will-do” versus “can-do” traits in the personnel selection literature (Borman & Motowidlo, 1993). “Will-do” traits reflect a motivational disposition to engage in job tasks, while “can-do” traits reflect the ability to accomplish job tasks (Gonzalez-Mulé, Mount, & Oh, 2014). In the context of job demands, self-beliefs are “will-do” traits that impact one’s appraisal of job demands as being threatening to one’s well-being and performance (Jex et al., 2001; Xanthopoulou et al, 2007), while cognitive ability is a “can-do” trait that impacts one’s ability to fulfill the requirements of a demanding job. Past research has shown the importance of cognitive ability for physical and mental health but has not considered its role as a moderator of the relationship between work stressors and health (cf. Calvin et al., 2011).

In line with this logic, we argue that cognitive ability moderates the relationship between job demands and health. Specifically, past research has shown that individuals who have the skills and abilities to meet the demands of their job experience less strain than those who do not (Edwards, 1996). Accordingly, demanding jobs require that employees effectively manage their time, organize priorities, and plan their workloads. Given that individuals with high cognitive ability acquire job knowledge at a faster rate than those with low cognitive ability (Schmidt & Hunter, 2004), they will be better able to learn ways to manage job demands and will perform better in demanding jobs than individuals low on cognitive ability. Because those with higher cognitive capacity perform well on difficult tasks, they are also more likely to appraise a demanding job as an opportunity to demonstrate competence. Just as Karasek (1979) suggested that learning occurs in high demands-high control jobs, cognitive ability provides the impetus for difficult work to be fulfilling rather than debilitating, resulting in strain resolution in more demanding jobs. On the other hand, individuals with lower cognitive ability do not have the cognitive capacity to meet the demands of a job with high workload, time pressure, and intense concentration requirements. These individuals are unlikely to perform well and will feel threatened by the same stymied goal pursuit that accompanies demanding jobs with low control; this, in turn, leads to deteriorations in physical and mental health.

**Hypothesis 2:** Cognitive ability moderates the positive relationships between job demands and poor (a) physical and (b) mental health, such that the relationships will be stronger when cognitive ability is low than when it is high.

As we noted previously, most studies investigating the job demands-health relationship have focused on short-term, proximal indicators of physical or mental health. We extend this research and discuss the more distal physiological and psychological processes and, ultimately, death that take place in response to work stressors.

**Job Demands and Distal Strain Outcomes**

The contention that stressors have long-term effects on distal strain outcomes is based on the AL model (McEwen & Stellar, 1993). According to the AL model, individuals’ bodily systems (e.g., metabolic, cardiovascular, and neuroendocrine) temporarily adjust their functioning in response to stressors by, for example, increasing blood pressure in response to a stressful situation (Sterling & Eyer, 1988). With repeated exposure to stress over an extended period of time, physiological and psychological bodily systems effectively adjust to treat these stressful states as the new baseline level of functioning (Selye, 1955). Juster, McEwen, and Lupien (2010, p. 3) referred to this as the “wear and tear” experienced by the body as a function of chronic stressors. Consequently, the AL model suggests that premature death is the ultimate result from long-term, unresolved stress, while more proximal intervening processes, like poor physical and mental health, are the mechanisms through which stress influences death.¹

In this study, we operationalize physical health using the AL index, defined by McEwen and Stellar (1993, p. 2093) as “the cost of chronic exposure to fluctuating or heightened neural or neuroendocrine response” that results from long-term stress exposure. The AL index captures a host of physiological responses to stress, through measures of cardiovascular, inflammatory, and metabolic functioning, which collectively provide a picture of how individuals’ bodily systems have adapted or deteriorated in response to chronic stress (Brooks et al., 2014). These measures are combined to provide an index of physical health that is congruent with the

¹ It is important to highlight that the allostatic load model suggests that both physiological and psychological systems will deteriorate over time in response to stress. Thus, indicators of physical and mental health are both germane to the model’s predictions. However, the allostatic load index only captures the physical aspect, and not the mental aspect, of an individual’s overall health.
setpoint-adjustment process outlined by the AL model, and provide a more valid and reliable measure of strain than any individual indicator taken in isolation (Robertson, Beveridge, & Bromley, 2017). Recent research has shown that poorer physical health, as measured by higher scores on the AL index, relates positively to mortality in varied age groups and geographic regions (Hwang et al., 2014; Seeman et al., 2010).

**Hypothesis 3:** Poorer physical health will be positively related to mortality.

We operationalize mental health using the incidence of depression, a major mental illness characterized by feelings of sadness and a loss of interest in activities once enjoyed (American Psychiatric Association, n.d.). Major depressive disorders are recognized by the World Health Organization as the leading cause of disability worldwide (World Health Organization, 2018). The medical literature has long recognized that depression has underlying physiological causes (e.g., Michelson et al., 1996). For example, the AL model suggests that chronic stress causes dysregulation of the hypothalamic system, which is associated with depression (PDQ Supportive & Palliative Care Editorial Board, 2017). Thus, in AL model parlance, depression is a “late stage” mediator that is proximal to disease endpoints, such as death (Juster et al., 2010). In line with this logic, several meta-analytic reviews have linked depression to an increased risk of mortality (e.g., Cuijpers & Smit, 2002; van Melle et al., 2018). The medical literature has long recognized that depression is a risk factor for mortality (e.g., Cuijpers & Smit, 2002; van Melle et al., 2018). For example, the AL model suggests that chronic stress causes dysregulation of the hypothalamic system, which is associated with depression (PDQ Supportive & Palliative Care Editorial Board, 2017). Thus, in AL model parlance, depression is a “late stage” mediator that is proximal to disease endpoints, such as death (Juster et al., 2010). In line with this logic, several meta-analytic reviews have linked depression to an increased risk of mortality (e.g., Cuijpers & Smit, 2002; van Melle et al., 2018).

**Hypothesis 4:** Poorer mental health will be positively related to mortality.

**A Process Model Linking Work Stress to Mortality**

Our hypotheses reflect a conditional indirect effects model where job control and cognitive ability moderate the effect of job demands on mortality through the mediating effects of physical and mental health. Hypotheses 1 and 2 are the “first stage” of our model, whereby job demands’ relationship with physical and mental health depend on job control and cognitive ability. Hypotheses 3 and 4 are the “second stage,” where poor physical and mental health have direct positive relationships with mortality.

**Hypothesis 5:** The positive indirect effect of job demands on mortality via poor (a) physical health and (b) mental health will be stronger when job control is low than when it is high.

**Hypothesis 6:** The positive indirect effect of job demands on mortality via poor (a) physical health and (b) mental health will be stronger when cognitive ability is low than when it is high.

**Method**

**Sample Description**

We used data from the Midlife Development in the United States (MIDUS) study to test our hypotheses (Brim, Ryff, & Kessler, 2004). The goal of the MIDUS is to better understand factors contributing to health and well-being in the United States. The MIDUS has been used for research in applied psychology, management, and related fields (e.g., Li, Zhang, Song, & Arvey, 2016; Marshall & Taniguchi, 2012; Patel, Wolfe, & Williams, 2019). To our knowledge, no studies have examined the focal relationships we examine in this study. The data we used were collected during three data collection waves (i.e., MIDUS 1, MIDUS 2, and MIDUS 3), spanning from 1995 through 2015. Participants were recruited via random-digit dialing from a nationally representative sample of adults. To be included in our study, individuals could not be retired in the MIDUS 1 and they had to have data on the control variables, job demands, job control, cognitive ability, measures of physical and mental health in the MIDUS 2, and mortality status in the MIDUS 3. Out of 7,108 participants, 3,148 met the inclusion criteria and were included in the mental health analyses. In the MIDUS 2, a subsample of participants (N = 1,054) were selected to provide biomarker data, and 754 of these participants met the inclusion criteria and were included in the physical health analyses. Individuals in our sample ranged in age from 20 to 74 (M = 43.79; SD = 10.48), and 50% were female.

**Measures**

**Job demands and job control.** We measured job demands using a five-item scale focusing on the workload, concentration demands, and time pressure in one’s work (α = .76). We measured job control using a six-item scale focusing on one’s ability to make decisions at work, decide what to work on, and decide how to complete one’s work (α = .86). The items for both measures were scored on a scale ranging from 1 (never) to 5 (all of the time).

**Cognitive ability.** We measured cognitive ability using the Brief Test of Adult Cognition, implemented via phone call in the MIDUS 2, which is made up of five subtests: word list recall, digits backward, category fluency, number series, and backward counting (Lachman & Tun, 2008; Tun & Lachman, 2006). Subtest scores were standardized and averaged (α = .71).

**Physical health.** We measured physical health using the AL index, which is based on risk scores computed from 24 biomarkers representing seven different bodily systems (Gruenewald et al., 2012; McEwen, 2000). Higher scores indicate poorer physical health. The average AL index score in our sample was 1.69 (SD = 1.02).

**Mental health.** We measured mental health using the depression scale from the World Mental Health Organization Composite Diagnostic Interview-Short Form (CIDI-SF; Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998). Participants meeting the criteria for clinical depression were assigned a score of “1,” or “0” otherwise. In our sample, 269 (8.5%) participants met the criteria for depression.

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2 It is important to note that the sample sizes differ for the first- and second-stage model equations because only a subsample of participants (N = 3,973 total; 2,612 of which met our inclusion criteria) were selected to complete the cognitive ability measure. Thus, we opted to also include individuals who did not complete the cognitive ability measure in the models with mortality as the dependent variable in order to maximize the data we used.

3 Please see Appendix B in the online supplemental material for more details regarding the items, origin, and scoring of the measures, and a confirmatory factor analysis of the job demands and job control scales.
Mortality. Mortality status was coded as 1 (deceased) or 0 (alive) as of the MIDUS 3. In our sample, 211 (6.7%) participants were deceased by 2015.4

Control variables. We controlled for several variables (all collected in the MIDUS 1) to rule out alternative explanations established in other studies for our findings, such as personality traits or socioeconomic status differences driving our results, and in the case of the physical and mental health analyses, to more strongly support the causal nature of the relationships in our model. Specifically, we controlled for gender (0 = male; 1 = female), age, race (0 = nonwhite; 1 = white), marital status (0 = not married; 1 = married), socioeconomic status (SES; α = .62), conscientiousness (α = .57), emotional stability (α = .74), and Time 1 measures of physical and mental health, in their respective models.

Results

Table 1 presents descriptive statistics and correlations for the control and study variables. Tables 2 and 3 present the results of the regression equations which we used to test our hypotheses.5 We used OLS regression for the AL index equations, logistic regression for the depression equations, and Cox proportional hazard regression for the mortality equations.6 We began our analyses by estimating an omnibus regression equations which we used to test our hypotheses.5 We used t tests of mediated-moderation. As shown in Model 3 of Tables 2 and 3, the interactions jointly improved model fit, followed by models testing each interaction separately to plot their shapes and, later, conduct tests of mediated-moderation. As shown in Model 3 of Tables 2 and 3, the interactions jointly improved the fit of the models predicting allostatic load (ΔR² = .01; p = .07) and depression (Δχ²(2) = 6.93; p = .03).

To test Hypothesis 1 and as shown in Model 1 of Tables 1 and 2, the interaction between job demands and control was significant in predicting allostatic load (B = −.11; β = −.05; p = .04) and depression (B = −.24; odds ratio (OR) = .78; p = .045). We plotted the relationships of job demands with the AL index and depression, at low (−1SD) and high (+1SD) levels of job control, and computed the simple slopes of job demands on the health outcomes at these levels of control (reported in the “First stage effects” portion of Table 4). As shown in Panel 1 of Figure 2, job demands were unrelated to allostatic load at low control (b = .00, 90% CI [−.11, .12]) and negatively related to allostatic load at high control (b = −.17; 90% CI [−.29, −.05]). As shown in Panel 1 of Figure 3, job demands were positively related to depression at low control (b = .43; 90% CI [.20, .67]) and unrelated to depression at high control (b = .06; 90% CI [−.22, .34]). These values correspond to odds ratios of 1 and 1.53, respectively, indicating that the likelihood of depression is the same across low and high job demands when job control is high, and increases by 70% when comparing low to high job demands when cognitive ability is low. Collectively, these results supported Hypothesis 2(b).7

As predicted by Hypotheses 3 and 4, and as shown in Model 4 in Tables 2 and 3, the AL index [B = .61; hazard ratio (HR) = 1.84; p = .00] and depression were both positively related to mortality (B = .83; HR = 2.30; p = .00). These results indicate that a one-unit increase in AL results in an 84% increased rate of death, and depression results in a 130% increased rate of death. Thus, Hypotheses 3 and 4 were supported.

To test Hypotheses 5 and 6, we computed 90% bias-corrected confidence intervals (CIs) around the conditional indirect effects using 1,000 bootstrapped samples (Edwards & Lambert, 2007). As shown in Table 4, the indirect effect of job demands on mortality via physical health was not significant at low job control (b = .00; 90% CI [−.07, .08]), but was negative at high control (b = −.10; 90% CI [−.23, −.02]). The difference between the indirect effects was significant (−.10; 90% CI [−.28, −.01]). The indirect effect of job demands on mortality via mental health was positive at low control (b = .36; 90% CI [.13, .67]) and not significant at high control (b = .05; 90% CI [−.19, .31]). The difference between the indirect effects was significant (−.31; 90% CI [−.71, −.02]). Thus, Hypotheses 5(a) was not supported, and Hypothesis 5(b) was supported. The indirect effect of job demands on mortality via physical health was not significant at low cognitive ability (b = −.00; 90% CI [−.09, .08]), but was negative at high cognitive ability (b = −.10; 90% CI [−.26, −.02]). The difference between the indirect effects was not significant (−.10; 90% CI [−.27, .00]). The indirect effect of job demands on mortality via mental health was positive at low cognitive ability (b = −.44; 90% CI [.17, .77]) and not significant at high cognitive ability (b = .00; 90% CI [−.24, .28]). The difference between the indirect effects was significant (−.43; 90% CI [−.88, −.09]). Thus, Hypothesis 6(a) was not supported, and 6(b) was supported.8

Discussion

Our study draws from the JDC and AL models (Karasek, 1979; McEwen & Stellar, 1993) to examine when, for whom, and why

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4 Please see Appendix B of the online supplemental material for an explanation and list of the causes of death in our sample.
5 We also estimated the equations without control variables, and our conclusions remain the same.
6 Please see Appendix C in the online supplemental material for additional details.
7 We also tested a three-way interaction between job demands, control, and cognitive ability predicting physical and mental health, but found that it was not statistically significant in either case.
8 Please see Appendix C of the online supplemental material for analyses conducted at the within-person level and additional robustness checks.
job demands relate to mortality. Taken together, our results suggest divergent pathways by which job demands influence death, as job demands are associated with an increase in the likelihood of death via poor mental health when job control or cognitive ability are low, and a decrease in the likelihood of death via better physical health when job control is high. Our study offers several theoretical and practical implications. In the case of mental health, we found support for our hypotheses derived from the JDC and AL models. Specifically, when job demands are greater than the control afforded by the job or the

Table 1

Descriptive Statistics and Variable Intercorrelations

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender</td>
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<td>.50</td>
</tr>
<tr>
<td>2. Age</td>
<td>43.79</td>
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<td>3. Race</td>
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<td>4. Marital status</td>
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<td>6. Conscientiousness</td>
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<tr>
<td>7. Emotional stability</td>
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</tr>
<tr>
<td>8. Physical health</td>
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<td>.89</td>
</tr>
<tr>
<td>9. Depression</td>
<td>.10</td>
<td>.30</td>
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<td>10. Job demands</td>
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<tr>
<td>11. Job control</td>
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<td>.76</td>
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<tr>
<td>12. Cognitive ability</td>
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<td>.96</td>
</tr>
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</tr>
<tr>
<td>14. Allostatic load</td>
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<td>.09</td>
</tr>
<tr>
<td>15. Mortality</td>
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<td>.25</td>
</tr>
</tbody>
</table>

Note. N = 754 for correlations with allostatic load; N = 704 for correlation between allostatic load and cognitive ability; N = 2,612 for other cognitive ability correlations. Alphas shown on the diagonal. Gender was coded 0 = male and 1 = female; race was coded 0 = nonwhite and 1 = white; marital status was coded 0 = not married and 1 = married; SES = socio-economic status; depression was coded 0 = not clinically depressed and 1 = clinically depressed; mortality was coded 0 = alive and 1 = deceased. 

Table 2

OLS and Cox Regression Results Predicting Allostatic Load and Mortality

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
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<td></td>
<td>B</td>
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<td>Control Variables</td>
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<td>.00</td>
<td>.00 (.07)</td>
<td>.00</td>
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<tr>
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<td>.03 (.00)</td>
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<td>.02 (.08)</td>
<td>.01</td>
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<td>-0.04</td>
<td>-0.06 (.06)</td>
<td>-0.04</td>
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<tr>
<td>Conscientiousness</td>
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<td>-0.03</td>
<td>-0.08 (.09)</td>
<td>-0.03</td>
</tr>
<tr>
<td>Emotional stability</td>
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<td>.02</td>
<td>.03 (.06)</td>
<td>.02</td>
</tr>
<tr>
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<td>-0.21 (.04)</td>
<td>-0.18</td>
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<td>Study Variables</td>
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<tr>
<td>Job demands (JD)</td>
<td>-0.08 (.06)</td>
<td>-0.08</td>
<td>-0.06 (.06)</td>
<td>-0.04</td>
</tr>
<tr>
<td>Job control (JC)</td>
<td>.03 (.05)</td>
<td>.05</td>
<td>.04 (.05)</td>
<td>.03</td>
</tr>
<tr>
<td>Cognitive ability (CA)</td>
<td>-0.06 (.05)</td>
<td>-0.06</td>
<td>-0.07 (.05)</td>
<td>-0.06</td>
</tr>
<tr>
<td>JD × JC</td>
<td>-0.11 (.06)</td>
<td>-0.05</td>
<td>.12 (.06)</td>
<td>.06</td>
</tr>
<tr>
<td>JD × CA</td>
<td>.11 (.06)</td>
<td>.05</td>
<td>.09 (.06)</td>
<td>.05</td>
</tr>
<tr>
<td>Allostatic load index</td>
<td>.16*</td>
<td>.15</td>
<td>.16*</td>
<td>.15</td>
</tr>
<tr>
<td>Model R² or χ²</td>
<td>.01*</td>
<td>.01*</td>
<td>.01*</td>
<td>.01*</td>
</tr>
<tr>
<td>ΔR² or Δχ²</td>
<td>.16*</td>
<td>.15</td>
<td>.16*</td>
<td>.15</td>
</tr>
</tbody>
</table>

Note. In Models 1–3, B = unstandardized regression coefficients and β = standardized regression coefficients. In Model 4, B = log hazard ratios, HR = hazard ratios. Standard errors are in parentheses. In Models 1–3, R² refers to the percent variance explained by the model and ΔR² refers to the incremental variance explained by the interaction terms. In Model 4, χ² refers to the chi-square statistic for the model and Δχ² refers to the incremental chi-square of the allostatic load index. Models 1–3 N = 704; Model 4 N = 754. Coefficient significance values are based on a one-tailed t-test (Models 1–3) or Z-test (Model 4); model significance results are based on an F- (Models 1–3) or χ²-test (Model 4). 

* p < .05.
individual’s ability to deal with those demands, there is a deterioration of the individual’s mental health and, accordingly, an increased likelihood of death. We found different, unexpected results in the case of physical health, such that job demands result in better health and a lower likelihood of death when paired with high control. It could be that high job control offers the opportunity to change task boundaries (i.e., job crafting) to benefit from high control. It could be that high job control offers the opportunity to change task boundaries (i.e., job crafting) to benefit from high control. It could be that high job control offers the opportunity to change task boundaries (i.e., job crafting) to benefit from high control.

This supports the notion that control in a stressful job results in experiences of mastery and competence, leading to improved physical health (e.g., Gonzalez-Mulé & Cockburn, 2017). Future research should investigate these possibilities.

We also unexpectedly found that job demands were unrelated to physical health when control was low, and that cognitive ability did not moderate the relationship between job demands and physical health. One explanation for the generally mixed findings in our study may be that our measure of job demands did not differentiate between challenge and hindrance demands. Specifically, challenge demands are work stressors (e.g., workload, responsibility, time

Table 4

<table>
<thead>
<tr>
<th>Path</th>
<th>-1 SD on the moderator</th>
<th>+1 SD on the moderator</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First stage effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Job Demands × Job Control → Allostatic load</td>
<td>.00 (.11, .12)</td>
<td>-.17* (-.29, -.05)</td>
<td>-.17* (-.33, -.003)</td>
</tr>
<tr>
<td>Job Demands × Job Control → Depression</td>
<td>.43* (.20, .67)</td>
<td>.06 (-.22, .34)</td>
<td>-.37* (-.76, -.02)</td>
</tr>
<tr>
<td>Job Demands × Cognitive Ability → Allostatic load</td>
<td>-.01 (-.12, .12)</td>
<td>-.17* (-.31, -.04)</td>
<td>-.16 (-.34, .03)</td>
</tr>
<tr>
<td>Job Demands × Cognitive Ability → Depression</td>
<td>.53* (.26, .77)</td>
<td>.00 (-.28, .30)</td>
<td>-.52* (-.91, -.11)</td>
</tr>
<tr>
<td><strong>Second stage effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allostatic load → Mortality</td>
<td>.61* (.24, 1.02)</td>
<td>.61* (.24, 1.02)</td>
<td>—</td>
</tr>
<tr>
<td>Depression → Mortality</td>
<td>.83* (.40, 1.16)</td>
<td>.83* (.40, 1.16)</td>
<td>—</td>
</tr>
<tr>
<td><strong>Conditional indirect effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Job Demands × Job Control → Allostatic load → Mortality</td>
<td>.00 (.07, .08)</td>
<td>-.10* (-.23, -.02)</td>
<td>-.10* (-.28, -.01)</td>
</tr>
<tr>
<td>Job Demands × Job Control → Depression → Mortality</td>
<td>.36* (.13, .67)</td>
<td>.05 (-.19, .31)</td>
<td>-.31* (-.71, -.02)</td>
</tr>
<tr>
<td>Job Demands × Cognitive Ability → Allostatic load → Mortality</td>
<td>-.00 (-.09, .08)</td>
<td>-.10* (-.26, -.02)</td>
<td>-.10* (-.27, .00)</td>
</tr>
<tr>
<td>Job Demands × Cognitive Ability → Depression → Mortality</td>
<td>.44* (.17, .77)</td>
<td>.00 (-.24, .28)</td>
<td>-.43* (-.88, -.09)</td>
</tr>
</tbody>
</table>

Note. Parameter estimates are unstandardized path estimates at low and high levels of the moderator. Values in parentheses are bias-corrected 90% confidence intervals from 1,000 bootstrapped samples.

* 90% confidence interval did not include zero.
pressure) that evoke feelings of fulfillment or achievement (Cavanaugh, Boswell, Roehling, & Boudreau, 2000), which is similar to Karasek’s (1979) original notion of job demands. On the other hand, hindrance demands are stressors (e.g., red tape, organizational politics) that involve excessive or undesirable constraints which interfere with individuals’ goal pursuit (Cavanaugh et al., 2000). Past studies have found that challenge demands are positively related to motivational constructs (e.g., engagement) and strain, but have a stronger relation with motivation than strain, while hindrance demands are related negatively to motivation, and are more strongly, positively related to strain than are challenge demands (Crawford et al., 2010; LePine, Podsakoff, & LePine, 2005). Thus, challenge demands may relate positively to health and negatively to the likelihood of death (or, at least, have weaker detrimental effects) when job control or cognitive ability is high, while hindrance demands are unlikely to ever be beneficial and may not be buffered even with high control or ability. These potentially differential relations of challenge and hindrance demands with health and death may have been obfuscated by the measure of overall job demands we used. Further, it is worth noting that the predictions offered by the JDC and AL models do not differentiate between challenge and hindrance demands, nor between mental and physical health. In fact, the JDC and subsequent theoretical models (e.g., job demands-resource model; Demerouti et al., 2001) were originally developed to explain mental strain. Thus, future research should investigate the ways challenge and hindrance demands are differentially bounded by work and individual characteristics, as well as how the predictions from the JDC and AL model apply to different types of health outcomes.

Our research offers several practical implications. First, managers should provide employees working in demanding jobs more control, and in jobs where it is unfeasible to do so, a commensurate reduction in demands. For example, allowing employees to set their own goals or decide how to do their work, or reducing employees’ work hours, could improve health. Further, organizations should select people high on cognitive ability for demanding jobs, as these people are less likely to suffer from depression and, ultimately, death. By doing this, organizations can benefit from the increased job performance associated with more intelligent employees, while also having a healthier workforce. Second, the divergent pathways we found could inform the use of targeted interventions. Specifically, in cases where the recommendations we outlined in the first point are untenable (e.g., increasing control for high demands jobs; reducing demands for low control jobs; hiring high cognitive ability employees for demanding jobs), managers should be aware of the risks and provide physical wellness programs (Conn, Hafdahl, Cooper, Brown, & Lusk, 2009) or mental health services (Charbonneau et al., 2005). For example, if an organization is unable to hire on cognitive ability for a demanding job, our study suggests that they should provide preventative...
mental health services or screening services for their employees and refer them to appropriate counseling (Harvey et al., 2011). Similarly, if an organization is unable to increase job control for demanding jobs, it should consider providing employees flexibility and incentives to attend to their physical health (Toker & Biron, 2012), such as weight control programs, healthy food options, or subsidizing gym memberships.

Our study suffers from several limitations. First, although the study takes place across two decades, there are only three measurement instances, which restricts our analysis to the work context provided by a single job reported during the first time point. This limitation is particularly salient when one considers that workers more commonly change careers and jobs compared to past generations (Bidwell, 2013; Hollister & Smith, 2014). Future research may consider the trajectory of changing job demands across a lifetime, as well as how this trajectory is related to health and mortality. Second, as mentioned earlier, our job demands measure combines elements of challenge and hindrance demands which we were unable to differentiate into separate scales. Third, a strength of our study was the objective measurement of distal indicators of physical health and mental health. However, the AL model suggests that other health outcomes also result from stress exposure, such as diabetes and bipolar disorder, which we were unable to examine (Ganster & Rosen, 2013). Similarly, other research suggests additional moderators of the relationship between job demands and mortality, such as social support (Theorell & Karasek, 1996). Given the nascent literature relating job demands to mortality, future research should seek to outline additional mechanisms and boundary conditions relating work to death.

References
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