Physiological reactions to and recovery from acute stressors: the roles of chronic anxiety and stable resources

BACKGROUND
Research has shown that employees subjected to acute stressors at work can suffer devastating repercussions. However, little is known about how employees who are experiencing ongoing chronic anxiety or have stable resources respond to acute stressors, particularly regarding their physiological responses to these situations. This study examines the physiological effects of an acute stressor when workers are already under chronic anxiety (i.e., cognitive anxiety and somatic anxiety) or when they have a stable resource (i.e., job control).

PARTICIPANTS AND PROCEDURE
Data were collected from 230 full-time employees working at three major oil companies in Brazil. First, demographic, anxiety, and job control measures were collected via questionnaire. Later, muscle tension, skin temperature, and heart rate were collected during a simulated task to assess the physiological response to stress. Hypotheses were tested by repeated measures general linear modeling.

RESULTS
The findings indicated that when employees were exposed to an acute stressor, those with chronic cognitive and somatic anxiety exhibited more heightened physiological responses than those lower on chronic anxiety. Further, compared to those with low control, employees with stable, high control over their work experienced a lower physiological reaction to the acute stressor.

CONCLUSIONS
Chronic anxiety generates high levels of physiological arousal and hyper-responsiveness to acute environmental stressors. Also, employees possessing stable resources, such as job control, experience reduced physiological responsivity to an acute stressor.

KEY WORDS
anxiety; skin temperature; job control; acute stress; muscle tension
BACKGROUND

Psychological stress is associated with numerous health problems including headaches, weight control problems, sleeplessness, gastrointestinal problems, heart disease, compromised immune systems, difficulties with memory, and psychological disorders (Tetrick, 2002). It has been estimated that stress costs organizations billions of dollars in disability claims, absenteeism, and lost productivity, annually (e.g., Ryan & Watson, 2004; Xie & Schaubroeck, 2001). Moreover, the American Psychological Association concluded from the results of their 2020 survey that levels of stress are presenting a mental health crisis (APA, 2020).

Many employees experience stressors on an ongoing or near-constant basis (i.e., chronic), and various reviews of the extensive work stress literature have concluded that exposure to prolonged psychological stressors and job demands can have debilitating consequences for employees (Meurs & Perrewé, 2011; Tetrick, 2002). Moreover, physiological reactions to acute stress (i.e., intense, but short, stressor exposure) also can result in long-term health consequences (Hamer & Steptoe, 2012; O’Connor et al., 2021); but it seems that future negative health and disease are related to the magnitude of the person’s response to acute stress (Turner et al., 2020). Thus, although it appears that workers will continue to face increased levels of work pressure and corresponding risks of occupational illness (Landsbergis, 2003; Lovelace et al., 2007), there is significant variability among individuals in terms of the severity and length of the psychological stress they experience.

In addition, we posit that, much like those with chronic stressor exposure, some persons have stable, ongoing resources. Stable, work-related resources include aspects of the work context, such as perceived organizational support (Kurtessis et al., 2017) or supportive supervision (Hammer et al., 2009). In the present study, we argue that job control is a stable, work-related resource available for employees to better manage workplace stressors. Theoretically, conservation of resources theory provides the most detailed explanation of how resources can be used to manage stress (Hobfoll et al., 2018; Hobfoll & Shirom, 2000). In addition, another model of job stress has job control as one of its two focal constructs (i.e., the job demands-control model; Kain & Jex, 2010; Karasek, 1979). Therefore, we believe job control to be an important ongoing resource for employees in handling workplace stressors.

It has been argued that physiology should be appreciated in the context of employee health and well-being (Ganster & Rosen, 2013), including job resources and work stress (Kuykendall & Tay, 2015; Stueck et al., 2016). However, although prior studies have examined how a stressor or a resource affects employees’ stressful experiences, it is unclear how experiencing ongoing (i.e., chronic) stressors or resources influences the acute stress response process, particularly for workers. Consequently, our study makes four contributions. First, we measure employees’ physiological response to an acute stressor and test how chronic anxiety (i.e., cognitive and somatic anxiety) and a stable resource (i.e., job control) influence responses to acute stressors.

Moreover, researchers have called for more studies examining physiological recovery from stressors, since both physiological reactivity and recovery are essential components in the overall stress experience (Kühnel et al., 2020; Linden et al., 1997). Thus, our study makes a second contribution by examining our physiological outcomes before, during, and after our acutely stressful intervention, thereby investigating the experienced stress response through both physiological reactivity and recovery. A third contribution made by our study is that our experimental design utilizes a serial-7 cognitive stress test (Arena & Schwartz, 2003; Rosenthal et al., 1989) that is similar to workplace cognitive demands (Lovallo, 2015), and we increase the workplace relevance by placing social pressure on the participants to perform at their best. The fourth main contribution of our research is that we chose physiological measures (i.e., muscle tension, skin temperature, and heart rate) that have been shown to be important to the stress response.

THEORY AND HYPOTHESIS DEVELOPMENT

CHRONIC ANXIETY AND STABLE RESOURCES

Psychological stress can arise from a variety of sources. Some (e.g., Lazarus, 1999) have argued that experienced stress results from enduring threatening or harmful conditions, such as daily hassles. Others have suggested that characteristics of individuals could influence the stress response (e.g., Matthews et al., 2001). For instance, regarding cardiovascular illness, it has been argued (e.g., Yao et al., 2019), and results appear to suggest (e.g., Nabi et al., 2013), that some persons are over-reactive to their environment. In such instances, the interaction of the person with that situation produces the psychological stress response. In short, theoretical and empirical evidence from a variety of literature has suggested that experienced stress can result from both personal and environmental influences.

Based on the taxonomy of chronic stress offered by Baum et al. (1993), one group of researchers (i.e., Miller et al., 2007) defined chronic stress as an extended threat to the self that is posed by a stimulus, even if the stimulus is absent. Although chronic experienced stress could be measured via objective,
biological indicators, research findings suggest that its physiological effects also can be measured by subjective indicators, including among healthy workers (Boschi et al., 2017). Consequently, our study examines chronic experienced stress through the assessment of an individual’s ongoing anxiety, which has been linked to negative health outcomes, such that it could be an independent risk marker of cardiovascular disease (Karlsen et al., 2021) and heart failure (Siennicka et al., 2015). Other stress scholars also have characterized anxiety as a chronic stress producer (e.g., Chida & Hamer, 2008).

Cattell (1966) and Spielberger (e.g., 1966) were among the first to differentiate between state anxiety and trait anxiety. State anxiety is a transitory, unpleasant emotional state that varies in intensity across time, whereas trait anxiety, which is utilized in this study, describes a proneness to frequently experience state anxiety (Endler & Parker, 1990; Saviola et al., 2020; Spielberger, 1983). Anxiety has been argued to include such thoughts and feelings as fear, apprehension, respiratory distress, and dizziness (Kogan et al., 2016). Researchers have generally identified two components of anxiety: cognitive anxiety and somatic anxiety (Roberts et al., 2016).

As discussed by Viney and Caputi (2005), cognitive anxiety is caused by doubts about whether events and their implications can be understood. Individuals’ perceptions of events need to be reconciled with the cognitive systems and perceptions already in place. When these cannot be reconciled, people become cognitively anxious and worry. Thus, it could be argued that both the amount of perceptual material and its incongruity with expectations are at the root of cognitive anxiety (Lazarus, 1966; Ree et al., 2008). On the other hand, somatic anxiety concerns a person’s perception of physiological arousal. These perceptions are reflected in individual reports of experiences such as shortness of breath, clammy hands, or tense muscles. Therefore, anxiety is thought to be based on either worry (i.e., cognitive) or emotionality (i.e., somatic) (Ree et al., 2008).

Health researchers also have acknowledged that resources play an important role in an individual’s response to stressors. Within the health and wellbeing literature, the most elaborate discussion of resources has been provided by Hobfoll’s conservation of resources theory (see Hobfoll, 1989; Hobfoll & Shirom, 2000; Hobfoll et al., 2018). Being broadly defined, resources can be conditions, objects, personal characteristics, or energies. Hobfoll (1989) suggests that highly valued conditions (e.g., job control) provide individuals with a reservoir of resources, yielding greater potential resistance to stressors. On the other hand, some resources can be detrimental to individuals experiencing stress, depending on the context (Hobfoll et al., 2018). For instance, although in rare circumstances (e.g., immediate physical threat) anxiety can be helpful, ongoing heightened anxiety generally is harmful to individual stress management, and we concur with other scholars that it leads to chronic stress exposure for individuals (e.g., Chida & Hamer, 2008). Therefore, conservation of resources theory provides a theoretical framework for why our constructs of job control and anxiety influence the stress process.

In addition, other stress researchers have noted the importance of resources to stress management. For example, the widely cited and researched job demands-control model of stress suggests that job control is the specific resource of most usefulness to employee stress management when encountering workplace demands (Kain & Jex, 2010). Moreover, recent theoretical work argues that job conditions, such as job control, are resources that affect psychological well-being (Kuykendall & Tay, 2015). Therefore, we contend that the condition of job control and the personal characteristics of cognitive and somatic anxiety should influence the physiological response to acute stress.

**ACUTE STRESSORS**

In contrast to stressors that are stable and long-lasting (i.e., chronic stressors), acute stressors are those that have a specific onset and offset. Acute stressors could arise from a variety of experiences, such as the loss of a job, an automobile accident, or a mishap in the workplace, and research has linked acute stress reactivity to poorer physical health (Crosswell & Lockwood, 2020). A number of researchers have been interested in how individuals who experience chronic stress or stable resources respond when faced with an acute stressor, and, overall, these studies have yielded mixed results.

Some scholars have argued that chronic stress produces persistently high arousal levels and hyper-responsiveness to environmental demands, and several studies have supported these arguments (e.g., Fleming et al., 1987; McEwen, 2006; McEwen & Stellar, 1993). For instance, one study indicated that women with high levels of chronic stress, labeled background stress, exhibited greater responses in systolic blood pressure and heart rate ($p < .06$) than those with lower levels of chronic stress when experiencing an acute stressor (i.e., Lawler & Schmied, 1987). The results of another study indicated that those experiencing chronic stress had increased inflammatory responses to daily stressors (PACE et al., 2006). Also, Lepore et al. (1997) found that persons experiencing chronic stress had elevated systolic and diastolic blood pressure responses and delayed diastolic blood pressure recovery to acute, laboratory stressors. Finally, a group of researchers demonstrated that, under conditions of acute stress, marital sat-
isfaction for wives was lowest when chronic stress levels were high (Karney et al., 2005).

However, another group of researchers has argued what seemingly appears to be the contrary. Schaubroeck and Ganster (1993) found evidence supporting their hypotheses of reduced reponsivity and recovery to acute stressors for those with chronic exposure to stressful demands. They found that employees experiencing chronic occupational demands had lower cardiovascular and skin temperature responsiveness to a challenging situation. Another study found that individuals experiencing chronic stress had reduced physiological responses to stressful tasks, as measured via blood pressure and epinephrine levels (Matthews et al., 2001). Some results outside of the physiological stress literature could support these arguments. For instance, one interpretation of the findings of McGonagle and Kessler (1990) is that exposure to chronic stressors reduces the effects of acute stressors on depression, possibly by making the interpretation of the acute stressor more benign.

Therefore, some scholars (see Dienstbier, 1989; Schaubroeck & Ganster, 1993) have argued that chronic exposure to stressors leads to diminished physiological reactivity due to both reduced arousal and a weakened capacity for recovery. However, unlike prior work, the present study examines chronic anxiety — not situational stressors or demands to which individuals might adapt. We argue that persons experiencing high chronic anxiety will demonstrate more heightened physiological responses to acute stressors than those experiencing low chronic anxiety. Additionally, an individual’s stress response is not fully explained by reactivity to stressors, but also by delayed recovery, indicating the prolonging of the stressful experience (Brosschot et al., 2006). Therefore, we also will examine workers’ ability to recover (relax) after acute stressor exposure.

As noted earlier, it has been suggested that personality factors contribute to the chronic stress experience. Jackson et al. (2002) found that a pessimistic explanatory style moderated the relationship between perceived stress and physical illness. Moreover, persons with high negative affect were shown to experience increased muscle tension when experiencing stress (Zellars et al., 2009), and a meta-analysis found that adults with PTSD experienced greater physiological reactivity to a stressor than those without PTSD (Pole, 2007). Also, one study demonstrated that distress and trait anxiety were related to elevated cortisol levels (Van Eck et al., 1996), and another one that depressed women who were higher on trait verbal aggression had a greater response to a stressful task than those low on aggression (Betensky & Contrada, 2010). Finally, a meta-analysis revealed that anxiety, neuroticism, and negative affect reduced cardiovascular recovery to acute stress (Chida & Hamer, 2008).

We concur with other scholars who have argued that certain personality characteristics could make individuals more affected by the same level of stress as others without those characteristics (e.g., Gallagher et al., 2018). Further, we contend that the possession of some individual differences (i.e., chronic cognitive and somatic anxiety) exposes individuals to experienced stress on an ongoing basis and that they are unable to adapt, unlike how they could adapt to situational stress. Thus, these traits could expose individuals to chronic stress, and, in a typical acute stressor situation (e.g., workplace demands), they would detrimentally heighten the stress response due to their misalignment with the stressor context (Hobfoll et al., 2018). Consequently, we hypothesize that persons who report high chronic anxiety will demonstrate more heightened physiological responses (i.e., heart rate, muscle tension, and skin temperature) and a weakened ability to recover from an acute stressor than those reporting low levels of chronic anxiety.

Hypothesis 1(a): Individuals reporting high levels of cognitive and somatic anxiety will experience greater physiological arousal (i.e., increased heart rate and muscle tension, and decreased skin temperature) in the presence of an acute stressor than those reporting low levels of cognitive and somatic anxiety.

Hypothesis 1(b): Individuals reporting high levels of cognitive and somatic anxiety will recover more slowly from an acute stressor than those reporting low levels of cognitive and somatic anxiety.

As argued by Kuykendall and Tay (2015) in their framework for subjective well-being and physiological functioning in the work context, job conditions influence personal resources, which, in turn, influence physiological functioning. However, as opposed to chronic stress, less research has been directed toward the interaction of stable, ongoing resources with acute stressors, though these results seem more consistent. Regarding psychological outcomes, research by Ellering et al. (2005) demonstrated that job control strengthened the effects of coping on well-being in stressful situations, and the results of a study by Karney et al. (2005) suggested that couples with coping resources have elevated marital satisfaction in the presence of high acute stress. Also, greater positive affect was shown to prevent a reduction in job satisfaction, when in the presence of work overload (Gallagher & Meurs, 2015).

Concerning physiological responses, one group of researchers (i.e., Steptoe et al., 2003) found that men (but not women) with low job control showed larger increases in fibrinogen in response to an acute stressor than did those high on job control. In another study (Steptoe et al., 1993), men low on job control exhibited heightened blood pressure responsiveness to uncontrollable, acute stressor tasks. Lastly, bus drivers with greater job control were found to
have decreased physiological responses to job stress, compared to those with low job control (Cendales-Ayala et al., 2017). Thus, in line with the conservation of resources theory (Hobfoll, 1989; Hobfoll et al., 2018), we contend that having job control provides employees with a stable resource that helps them to better manage stressful experiences. Consequently, individuals reporting high levels of job control will experience less physiological arousal in response to and a greater ability to recover from acute stressors than persons reporting low levels of job control.

Hypothesis 2(a): Individuals reporting high levels of job control will experience less physiological arousal (i.e., decreased heart rate and muscle tension, and increased skin temperature) in the presence of an acute stressor than those reporting low levels of job control.

Hypothesis 2(b): Individuals reporting high levels of job control will recover more quickly from an acute stressor than those reporting low levels of job control.

PRESENT STUDY

The physiological stress literature has examined a number of different forms of physiological activity, such as muscular activity (e.g., EMG), sympathetic nervous systems (e.g., heart rate, skin temperature, and blood pressure) and hormonal responses (e.g., cortisol levels). A review (Somrentag & Fritz, 2006) concluded that exposure to acute stressors in both field and laboratory studies leads to an increase in cortisol levels. However, the effects of chronic stressors on cortisol are less clear, and we did not use a measure of hormonal response because these measures show a certain rhythm in the human body (Czeisler & Klerman, 1999). For instance, cortisol levels normally rise and peak during the early morning and drop throughout the day, with their lowest level in the evening. Thus, any activation (e.g., stressor intervention) is superimposed on the daily cortisol rhythmic cycle.

We tested the effects of experienced cognitive and somatic anxiety and job control on three physiological outcomes (heart rate, skin temperature, and electromyogram – EMG), before, during, and after a stressful intervention. We chose heart rate because prior research demonstrated that increased heart rate is associated with exposure to stressors (e.g., Lawler et al., 2003). We selected skin temperature because research has revealed that an individual’s stress propensity has been associated with skin temperature (Wofford, 2001). As blood pressure increases in stressful situations, blood volume decreases, and skin temperature is one way of measuring these blood volume reductions (e.g., Wofford, 2001). Further, Schaubroeck and Ganster (1993) in their examination of chronic exposure to stressors measured cardiovascular and skin temperature reactivity in response to acute stressors.

We also chose to measure muscle tension (assessed via EMG) because it is useful for gauging the valence and intensity of affective emotional reactions (Ensari et al., 2004). EMG is a measure of muscular contraction/relaxation and is frequently used to monitor stress levels in biofeedback programs (Pourmohammadi & Maleki, 2020). Electrical discharges of motor neurons produce contractions of muscle fibers, and the repeated discharge results in muscle contraction. The muscle relaxes as the discharge rate decreases. Both decreased skin temperature and increased EMG have been reported as indicators of increased experienced stress (Ganster et al., 2018; Zellars et al., 2009).

There is a reliable relationship between activity in specific facial muscles and the valence of affect (Barrett et al., 2019; Dufner et al., 2015). For example, some specific muscles are activated to frown, and others are activated to smile. Facial EMG measures non-voluntary muscular activity, thereby allowing for “assessment of the affective feelings that accompany such muscular activation” (Ensari et al., 2004, p. 59). For example, Kukde and Neufeld (1994) reported that medial frontalis (i.e., facial) EMG activity was associated with perceived threat.

PARTICIPANTS AND PROCEDURE

PARTICIPANTS

Participants in the study were fulltime employees working at three major oil companies in Brazil. The employees were randomly assigned to receive an invitation to participate. All companies made it clear that participation was voluntary and there was no penalty for not participating. The project began with 237 participants, primarily supervisors and managers, and complete data were collected from 230 participants. The sample was predominantly male (72.6%) and married (69.0%). The questionnaire was translated from English to Portuguese and back translated by two English teachers, fluent in both languages, who worked independently. Only a few minor discrepancies in wording emerged and were resolved by the translators as they talked through the differences.

PROCEDURE

Data were collected from each participant by a professionally educated clinical psychologist (i.e., one of the authors) at a professional biofeedback clinic. During the first visit to the clinic, everyone completed a questionnaire containing the demographic, anxiety, and job control measures. The participants returned to the clinic approximately one week later. During this visit, the individual entered a room where the
Clinician advised the participants that she would first collect the baseline measures of EMG, skin temperature, and heart rate, and then ask them to participate in a mental exercise while she continued to record the physiological measures. The individual was in a seated and relaxed position during the measurements.

Numerous studies have found mental stress to be associated with the stress response (e.g., Dupont et al., 2021; Ensari et al., 2004; Kukde & Neufeld, 1994), and previous studies have reported elevations in physiological measures when presented with a serial-7 stressor (e.g., Rosenthal et al., 1989; Stern et al., 2015). Being analogous to work demands, tasks involving mental arithmetic are considered a prototypical mental stressor (Lovalló, 2015). As noted by Ganster (2005), high levels of job demands result in physiological arousal that harms functioning on some tasks (Ganster, 2005, p. 493). Further, Schwebel and Suls (1999) reported significant correlations between cardiovascular measures and an arithmetic exercise with college students in a laboratory setting. Following a serial-7 approach, in our study, the clinical psychologist asked the individual to perform a [stress] task that required the subject to count backwards from 1,000 in multiples of 7 for 4 minutes.

To simulate arousal inducing work demands, and to enhance the stressful environment, the clinical psychologist spoke assertively and put the individual under pressure by repeatedly telling the individual to perform more quickly, thus suggesting to the person that he (she) was not doing well. Intervention physiological measures were collected during the stress task. The clinician then explained to the participant that the task exercise was complete, and they could relax. She then left the room for five minutes to allow the participant time to relax. Following the relaxation period, the clinician returned to the room and advised the participant that she would take the final readings to measure their relaxed state. Participants did not receive stress training or biofeedback management prior to any data collection. All measures were recorded by the same professional clinician using the same equipment.

**MEASURES**

**Heart rate.** Heart rate was measured as beats per minute using an automatic sphygmomanometer.

**Muscle tension.** Muscle tension was measured using electromyography (EMG). The facial muscles (i.e., frontalis EMG) were measured with 0.5-cm diameter silver-silver chloride electrodes placed above the eyebrow, over the pupil of each eye, with the ground centered. The skin was abraded to reduce resistance and cleaned with alcohol. EMG measures muscular contraction and relaxation via the electrical signals that cause muscle fiber to contract. Since muscle tissue does not normally produce electrical signals, if the muscles are completely at rest, there should not be any EMG activity. Also, EMG measures tension that is subtle and not under voluntary control (Ensari et al., 2004), and EMG is expected to increase in the presence of a stressor (Kukde & Neufeld, 1994).

**Skin temperature.** Muscle tension produces reflex vasoconstriction in the skin (hands and feet) to shift blood flow to the tense muscles such that colder temperatures indicate physiological arousal. Digital skin temperature was recorded with a thermistor taped to the distal finger pad of the right index finger.

**Cognitive anxiety.** Cognitive anxiety was measured via a 9-point Likert, 11-item measure developed by Lehrer and Woolfolk (1982) asking respondents how often they felt this way. It included items such as “I picture some future misfortune.” The Cronbach α reliability of the measure was .82.

**Somatic anxiety.** Somatic anxiety was measured via 16 items developed by Lehrer and Woolfolk (1982) asking respondents how often they felt this way. The 9-point Likert type scale had a Cronbach α reliability of .85. It included items such as “My throat gets dry.”

**Job control.** Job control was measured via a 9-item, 7-point Likert measure developed by Tetruck and LaRocco (1987). It included items such as “To what extent do you have influence over the things that affect you on the job?” The Cronbach α reliability of the measure was .87.

**CONTROL VARIABLES**

We controlled for the effects of age and gender in our analyses. Prior research has demonstrated that demographic variables can affect measurements of heart rate, muscle tension, and skin temperature (see Allen et al., 2014; Chatkoff et al., 2010; Jorgensen et al., 1996).

**RESULTS**

Means, standard deviations, and baseline period intercorrelations of the variables are presented in Table 1. As shown, cognitive and somatic anxiety were both positively correlated with baseline EMG, suggesting that, outside of an experimental context, individuals high in these anxieties experience greater muscle tension than those who are low in them. In addition, cognitive anxiety, somatic anxiety, and job control were all negatively associated with baseline skin temperature, but each was also unrelated to baseline heart rate.

The hypothesized effects of cognitive anxiety, somatic anxiety, and job control on the physiological measures were tested by repeated measures general linear modeling (GLM), using a procedure adopted...
Physiological reactions to and recovery from acute stressors from Lawler et al. (2003). The repeated measures GLM assesses within-subjects differences and between-subjects differences across three time periods (i.e., before, during, and after the intervention) for each physiological outcome. We extend this approach by controlling for the effects of age and gender, utilizing repeated measures GLM with covariates (c.f., Kozlowski & Bell, 2006).

As expected, because of our use of a stress test, sphericity was violated ($p > .01$) for each analysis. Therefore, degrees of freedom were adjusted using the Greenhouse-Geisser correction method (Howell, 2001), a conservative approach to sphericity violations. Regardless of the sphericity correction method employed, significance levels were relatively unchanged, and no significant results would have become non-significant. Contrast analyses for our focal constructs compared the physiological response (i.e., EMG, skin temperature, and heart rate) between the baseline resting state and the intervention (i.e., level 1 vs. level 2), and between the intervention and the subsequent post-experiment resting state (i.e., level 2 vs. level 3). Control variables were entered prior to the focal variable in each analysis.

As shown in Table 2, the rate of decrease in skin temperature from the baseline reading to that during the stressful intervention shows no significant interaction with cognitive anxiety. However, the rate of increase in temperature from the stressful intervention to relaxation has a significant interaction with cognitive anxiety ($p < .05$), in partial support of hypothesis 1(b). In addition, from both the baseline to the stressful intervention and from the stressful intervention to relaxation, cognitive anxiety demonstrated a significant interaction with EMG ($p < .001$), explaining 15.0% additional variance in the rate of increase and over 10.0% in the rate of decrease, respectively. The findings regarding EMG lend support for both hypotheses 1(a) and 1(b). However, our analysis did not find a significant interaction between heart rate and cognitive anxiety. Therefore, we found partial support for both hypotheses 1(a) and 1(b) regarding cognitive anxiety.

Table 3 provides details on the interactions of somatic anxiety with skin temperature, muscle tension (i.e., EMG), and heart rate. These results are similar to those for cognitive anxiety. Specifically, somatic anxiety did not significantly interact with the acute stressor intervention in the effect on heart rate, but it did demonstrate a significant interaction effect on the relaxation period of skin temperature ($p < .05$), partially supporting hypothesis 1(b). In addition, somatic anxiety exhibited significant interactions with EMG from both the baseline to the intervention ($p < .001$) and from the intervention to the relaxation ($p < .001$) periods, explaining additional variance of over 14.0% and 9.0%, respectively, providing support for both hypotheses 1(a) and 1(b). Similar to the results concerning cognitive anxiety, our results for somatic anxiety were partially supportive of hypotheses 1(a) and 1(b).

Table 4 provides the interactions with job control. Similar to the other focal constructs, job control did not have a significant interaction with heart rate. However, unlike the others, it also did not have a significant interaction with skin temperature. Job control did exhibit a significant interaction with EMG,
explaining an additional variance of nearly 8.0% from the baseline to the intervention and nearly 6.0% from the intervention to the relaxation period. Thus, our EMG results provide support for both hypotheses 2(a) and 2(b).

Figures 1 to 5 illustrate the differences for skin temperature and EMG. In the repeated measures GLM analyses, cognitive anxiety, somatic anxiety, and job control were continuous variables. However, for illustration purposes, these figures display

Table 2

Repeated measures GLM results for effects of cognitive anxiety on skin temperature, muscle tension, and heart rate

| Factor                | Between subjects effects | Within subjects effects | 1,2 |
|-----------------------|--------------------------|------------------------|------|
|                       | df | Mean square | F | p  | df | Mean square | F | p | partial η² |
| Temperature (Temp)    | 1.00 | 55186.18 | 2398.64 | < .001 | 1.57 | 16.00 | 11.78 | < .001 |
| Age                   | 1.00 | 16.31 | 0.71 | .401 | 1.57 | 1.44 | 1.06 | .333 |
| Gender                | 1.00 | 380.32 | 16.53 | < .001 | 1.57 | 8.16 | 6.01 | .006 |
| Cognitive anxiety (CA)| 1.00 | 86.30 | 3.75 | .054 | 1.57 | 9.81 | 7.23 | .002 |
| Error                 | 226 | 23.01 | 45.02 | .019 |
| CA × Temp: 1 vs. 2    | 1.00 | 1.83 | 1.72 | .191 |
| CA × Temp: 2 vs. 3    | 1.00 | 15.84 | 5.34 | .022 |
| Muscle tension (EMG)  | 1.00 | 357.36 | 55.92 | < .001 | 1.16 | 45.02 | 5.18 | .019 |
| Age                   | 1.00 | 2.60 | 0.41 | .524 | 1.16 | 3.91 | 0.45 | .532 |
| Gender                | 1.00 | 4.53 | 0.71 | .401 | 1.16 | 0.17 | 0.02 | .916 |
| Cognitive anxiety (CA)| 1.00 | 344.88 | 53.97 | < .001 | 1.16 | 281.48 | 32.36 | < .001 |
| Error                 | 226 | 6.39 | 8.70 |
| CA × EMG: 1 vs. 2     | 1.00 | 585.84 | 39.95 | < .001 | 0.15 |
| CA × EMG: 2 vs. 3     | 1.00 | 369.44 | 26.19 | < .001 | 0.10 |
| Heart rate (HR)       | 1.00 | 36551.57 | 527.87 | < .001 | 1.57 | 2218.32 | 84.55 | < .001 |
| Age                   | 1.00 | 168.21 | 2.43 | .120 | 1.57 | 119.08 | 3.55 | .041 |
| Gender                | 1.00 | 503.10 | 7.27 | .008 | 1.57 | 34.29 | 1.02 | .345 |
| Cognitive anxiety (CA)| 1.00 | 151.86 | 2.19 | .140 | 1.57 | 40.52 | 1.21 | .290 |
| Error                 | 226 | 69.23 | .019 |
| CA × HR: 1 vs. 2      | 1.00 | 0.326 | 0.01 | .943 |
| CA × HR: 2 vs. 3      | 1.00 | 89.39 | 1.30 | .255 |

Note. 1Within subjects effects utilized Greenhouse-Geisser sphericity correction; 2contrast levels are: 1 – baseline, 2 – intervention, 3 – relaxation.
the differences in the outcomes for those with low, mean, and high cognitive anxiety, somatic anxiety, and job control, using a standard deviation split. As shown in Figure 1, the skin temperature of those high in cognitive anxiety increased significantly less than those who were low in cognitive anxiety following the stressful intervention. Similarly, as illustrated in Figure 2, those high in cognitive anxiety experienced greater muscle tension (EMG) than those low in cognitive anxiety during the stressful

Table 3

Repeated measures GLM results for effects of somatic anxiety on skin temperature, muscle tension, and heart rate

| Factor                        | Between subjects effects | Within subjects effects | Level contrasts |
|-------------------------------|--------------------------|-------------------------|----------------|
|                               | df  | Mean square | F   | p   | df  | Mean square | F   | p   | partial η² |
| Temperature (Temp)            | 1.00 | 53.696.41  | 2339.83 | < .001 | 1.57 | 16.74 | 12.36 | < .001 |
| Age                           | 1.00 | 16.14     | 0.70 | .403 | 1.57 | 1.42 | 1.05 | .337 |
| Gender                        | 1.00 | 349.97    | 15.25 | < .001 | 1.57 | 7.10 | 5.24 | .010 |
| Somatic anxiety (SA)          | 1.00 | 99.52     | 4.34 | .038 | 1.57 | 9.67 | 7.14 | .002 |
| Error                         | 226  | 22.95     |      |     | 354.94 | 1.35 |
|                               |     |           |      |     |           | SA × Temp: | 1 vs. 2 |
|                               |     |           |      |     |           | 1.00 | 0.69 | 0.65 | .421 |
|                               |     |           |      |     |           | SA × Temp: | 2 vs. 3 |
|                               |     |           |      |     |           | 1.00 | 18.51 | 6.27 | .013 | 0.03 |
| Muscle tension (EMG)          | 1.00 | 308.37    | 49.18 | < .001 | 1.16 | 37.79 | 4.31 | .033 |
| Age                           | 1.00 | 2.82     | 0.45 | .503 | 1.16 | 4.20 | 0.48 | .517 |
| Gender                        | 1.00 | 10.19    | 1.63 | .204 | 1.16 | 0.18 | 0.02 | .916 |
| Somatic anxiety (SA)          | 1.00 | 371.55   | 59.24 | < .001 | 1.16 | 271.26 | 30.96 | < .001 |
| Error                         | 226  | 6.27     |      |     | 261.83 | 8.76 |
|                               |     |           |      |     |           | SA × EMG: | 1 vs. 2 |
|                               |     |           |      |     |           | 1.00 | 56.74 | 38.59 | < .001 | 0.15 |
|                               |     |           |      |     |           | SA × EMG: | 2 vs. 3 |
|                               |     |           |      |     |           | 1.00 | 34.75 | 24.41 | < .001 | 0.10 |
| Heart rate (HR)               | 1.00 | 35155.07  | 507.93 | < .001 | 1.57 | 2749.51 | 81.80 | < .001 |
| Age                           | 1.00 | 166.86   | 2.41 | .122 | 1.57 | 120.78 | 3.60 | .040 |
| Gender                        | 1.00 | 465.02   | 6.72 | .010 | 1.57 | 37.24 | 1.11 | .319 |
| Somatic anxiety (SA)          | 1.00 | 155.95   | 2.25 | .135 | 1.57 | 83.87 | 2.50 | .097 |
| Error                         | 226  | 69.21    |      |     | 350.81 | 33.62 |
|                               |     |           |      |     |           | SA × HR: | 1 vs. 2 |
|                               |     |           |      |     |           | 1.00 | 21.61 | 0.34 | .561 |
|                               |     |           |      |     |           | SA × HR: | 2 vs. 3 |
|                               |     |           |      |     |           | 1.00 | 12.27 | 1.79 | .183 |

Note: 'Within subjects effects utilized Greenhouse-Geisser sphericity correction; 'contrast levels are: 1 – baseline, 2 – intervention, 3 – relaxation.'
intervention, as well as a weaker ability to recover from the intervention.

Figure 3 illustrates that those high on somatic anxiety had a decreased ability to recover skin temperature from the intervention when compared to those low on somatic anxiety. As indicated in Figure 4, those high on somatic anxiety showed a heightened EMG response to the stressful intervention and a decreased ability to recover. Finally, Figure 5 illustrates that those high on job control exhibited a decreased

| Table 4                                                                 |
|------------------------------------------------------------------------|
| **Repeated measures GLM results for effects of job control on skin temperature, muscle tension, and heart rate**              |
| **Factor** | **Between subjects effects** | **Within subjects effects** |
|            | df          | Mean square | F    | p    | Level contrasts | df  | Mean square | F    | p    | partial η² |
| Temperature (Temp)          | 1.00        | 37870.03    | 1649.87 | < .001 |   | 1.57 | 8.59 | 6.17 | .005 |
| Age                     | 1.00        | 6.12        | 0.27   | .606  |   | 1.57 | 0.89 | 0.64 | .492 |
| Gender                  | 1.00        | 351.24      | 15.30  | < .001 |   | 1.57 | 8.67 | 6.23 | .005 |
| Job control (JC)          | 1.00        | 98.50       | 4.29   | .039  |   | 1.57 | 2.61 | 1.87 | .164 |
|                          |             |             |        |       | JC × Temp: 1 vs. 2 | 1.00 | 0.00 | 0.00 | .951 |
|                          |             |             |        |       | JC × Temp: 2 vs. 3 | 1.00 | 5.95 | 1.98 | .161 |
| Muscle tension (EMG)      | 1.00        | 892.60      | 121.53 | < .001 |   | 1.16 | 365.28 | 39.19 | < .001 |
| Age                     | 1.00        | 13.53       | 1.84   | .176  |   | 1.16 | 14.03 | 1.51 | .224 |
| Gender                  | 1.00        | 2.32        | 0.32   | .575  |   | 1.16 | 0.27 | 0.03 | .900 |
| Job control (JC)          | 1.00        | 129.28      | 17.60  | < .001 |   | 1.16 | 151.64 | 16.27 | < .001 |
|                          |             |             |        |       | JC × EMG: 1 vs. 2 | 1.00 | 309.73 | 19.50 | < .001 | 0.08 |
|                          |             |             |        |       | JC × EMG: 2 vs. 3 | 1.00 | 204.97 | 13.82 | < .001 | 0.06 |
| Heart rate (HR)          | 1.00        | 30016.75    | 430.05 | < .001 |   | 1.57 | 1927.77 | 57.44 | < .001 |
| Age                     | 1.00        | 139.95      | 2.01   | .158  |   | 1.57 | 129.79 | 3.87 | .031 |
| Gender                  | 1.00        | 537.13      | 7.70   | .006  |   | 1.57 | 34.27 | 1.02 | .346 |
| Job control (JC)          | 1.00        | 23.54       | 0.34   | .562  |   | 1.57 | 41.55 | 1.24 | .285 |
|                          |             |             |        |       | JC × HR: 1 vs. 2 | 1.00 | 2.88 | 0.05 | .832 |
|                          |             |             |        |       | JC × HR: 2 vs. 3 | 1.00 | 79.42 | 1.16 | .283 |

Note. ‘Within subjects effects utilized Greenhouse-Geisser sphericity correction; ‘contrast levels are: 1 – baseline, 2 – intervention, 3 – relaxation.’
response to the intervention and an increased ability to recover from it.

POST-HOC ANALYSIS

The control variables of age and gender explained a sizeable amount of variance in the analyses for the heart rate and skin temperature outcomes. It seemed possible that a significant amount of this variance could be explained by our focal constructs once we removed our control variables of age and gender. Therefore, we conducted post-hoc analyses of the relationships that cognitive anxiety, somatic anxiety, and job control have with skin temperature and heart rate, without controlling for age and gender. However, removing the controls of age and gender did not make the non-significant relationships significant.
DISCUSSION

This study demonstrates a heightened physiological response to an acute stressor for those experiencing cognitive and somatic anxiety. The findings support the research that has found chronic stress to produce consistently high levels of physiological arousal and hyper-responsiveness to environmental demands and acute stressors. Further, our research shows that employees who reported having stable resources, namely, job control, experience reduced physiological responsivity to an acute stressor. This provides additional evidence that stable resources can serve as buffers from acute stressors.

CONTRIBUTIONS OF THE STUDY

The present research contributes to the study of job stress by examining the roles of both job control and chronic anxiety in physiological reactivity. Regarding our theoretical implications, conservation of resources theory argues that resources are helpful to individuals managing the stress process. Our study supports this contention by showing that job control assisted employees to better manage a stressful experience. Moreover, we extend research understanding by showing that job control was beneficial to individuals when experiencing a stressor outside of the workplace (i.e., in a laboratory setting). Hobfoll and colleagues (2018) argued that more work should be done on the characterization of resources, and our results suggest that resource application can extend beyond its domain of origin, possibly due to the stable, ongoing nature of job control. We believe this finding contributes to our theoretical understanding of the value of resources.

Although little research has investigated the potential for resources that are detrimental to stress management (e.g., Russell et al., 2017), our findings regarding the exacerbation of the stress response and the decreased ability to recover following stress for those high on anxiety could lend support to the conservation of resources theory contention that resource value is context dependent. Resources that are relevant and helpful in one domain could be salient but harmful in another (Hobfoll et al., 2018), and our results contribute to this emerging literature on the context-dependence of resources.

In addition, the job demands-control model (Kain & Jex, 2010; Karasek, 1979) posits that job control plays a pivotal role in the experience of stress at work, and our results support this model, particularly since our acute stressor produces cognitive demands similar to those in the workplace (Lovallo, 2015). It has also been suggested that stress that is controllable may activate the hypothalamic-pituitary-adrenal (HPA) axis because it allows for active coping efforts (see Miller et al., 2007), and others have emphasized that job control allows for more successful coping strategies (i.e., Elfering et al., 2005). Thus, in line with Kuykendall and Tay (2015), one explanation for our results is that job control is a resource available to individuals confronting acute stressors, both physiologically because of the activation of the HPA axis and behaviorally because of the more effective coping efforts it allows individuals to utilize.

On the other hand, as we have argued, somatic and cognitive anxiety may be detrimental to stress management because it reinforces poor coping mechanisms. Worry, rumination, and other related phenomena, such as cognitive anxiety, have been found to be critical factors in health and well-being (Brosschot et al., 2006). Specifically, worrying and cognitive anxiety have been linked to physiological activation that can lead to long-term health consequences, such as cardiovascular disease (Brosschot et al., 2006). Further, these researchers argued that chronic worrying or repetitive negative thoughts are responsible for the effects on health and may act as a stressor itself, as well as mediating the effect of psychosocial stressors. This study provides evidence that chronic anxiety can exacerbate physiological reactivity when an acute stressor is induced. This is important because if physiological activation is prolonged, there is evidence to suggest that there is a direct link with cardiovascular, immune, endocrine, and neurovisceral systems (Brosschot et al., 2006; Thomsen et al., 2004). Overall, our study demonstrates the important role of perceived job control and chronic anxiety in physiological reactivity due to acute stressors.

STRENGTHS AND LIMITATIONS

Notable strengths of this study include the fact that our study is one of the few that have examined physiological reactions of employees. In addition, we studied the physiological stress response to acute stressors under varying conditions, namely, chronic stressors and stable resources. Moreover, following the guidance of prior scholarship (e.g., Crosswell & Lockwood, 2020), we chose measures (e.g., heart rate) that, although imperfect, are helpful for use in stress and health research. Also, as recommended by Kuykendall and Tay (2015), our research design includes both individual (i.e., chronic and somatic anxiety) and organizational (e.g., job control) factors, recognizing that both domains play an important role in worker experienced stress.

This study is not without limitations. First, the sample was predominately male and all were employees from Brazil. Clearly, additional research is needed to study different populations. For instance, since our sample was primarily composed of managers and supervisors, it is possible that their work stressors and
responses could be different from other work populations. Another limitation is that chronic cognitive and somatic anxiety could be impacted by contextual elements outside of the workplace (e.g., home stressors), and, in the present study, we were unable to control for such effects. Further, we know very little about potential cultural effects that may play a role in experienced physiological arousal. The acute stressor that was manipulated was rather short in duration, so it is difficult to determine whether acute stressors that last longer may have differential effects on physiological reactivity. In addition, it is not entirely clear why heart rate was not differentially affected by the acute stressor for those reporting chronic anxiety or stable resources. Perhaps it is not heart rate per se, but the variability in heart rate that should be examined. Heart rate variability (HRV) has attracted considerable attention in psychology and medical sciences and has become an important dependent measure referring to the beat-to-beat alterations in heart rate (Pham et al., 2021). HRV seems to be a marker of mental load (e.g., solving complex tasks) and HRV appears to be sensitive and responsive to acute stressors (Kim et al., 2018).

**DIRECTIONS FOR FUTURE RESEARCH**

Although our acute stressor intervention seems to be analogous to work demands (Lovallo, 2015), because it was conducted in a clinic, we cannot be certain that it is generalizable to context-specific acute stressors. Since it is possible that different sources of stress could elicit differential effects on the stress process, future research might examine acute stressors that occur directly in the workplace (e.g., sudden deadlines, mergers, or other organizational changes), or those that occur in the home domain (e.g., marital conflict, financial hardship, death of a loved one). Also, since our study found the resource of job control to have effects outside of the workplace, it suggests that scholars should investigate other possible resources (i.e., conditions, objects, personal characteristics, or energies) that are powerful enough to assist stress management in different life domains. Such research would expand our theoretical understanding of the value of resources to individuals’ experience of stress (Hobfoll et al., 2018).

Further, we examined only Brazilian employees in our sample. Future research might examine the extent to which cultural issues play a role in the chronic anxiety–acute stressor relationship. Although the preponderance of studies of work stress have been conducted with American workers, the psychological and psychological effects of work stress have also been reported in the United Kingdom and Latin America (e.g., Bianchi, 2004; Moulton, 2003; Ryan & Watson, 2004; Stacciarini & Troccoli, 2004). In 1999, the United Kingdom issued the *Management of Health and Safety at Work Regulations*, requiring all organizations with five or more employees to conduct regular risk assessments of workplace hazards, including stress, and comply with rigorous guidelines, or face fines and possible prosecution (Moulton, 2003; Spiers, 2004).

Spector and colleagues (2004) presented evidence that work-family pressures were universally reported and were related to well-being, especially mental well-being, among workers in Asian, Latin American, and Anglo countries. Health problems have been associated with long working hours in many countries, including Brazil, the United Kingdom, Japan, and the United States (Landsbergis et al., 2001; Portela et al., 2004; Sekine et al., 2006). Thus, although stress appears to be a universal concern, the role of culture may affect individual responsivity to acute stressors. Clearly, much additional research is needed before definitive and universal statements about chronic and acute stressors can be developed.

**PRACTICAL IMPLICATIONS**

The management of acute stress is a challenge for many employees. We examined the job control of workers and found that this job condition is a personal resource for employees, supporting prior theoretical frameworks (e.g., Kuykendall & Tay, 2015). Similarly, one study demonstrated that organizational support helped improve the well-being of ambulance personnel (i.e., Soh et al., 2016), workers who likely regularly experience acute stressors. Therefore, it suggests that organizations may be able to promote resistance to acute stressors by providing employees with favorable work conditions (e.g., organizational support and control over their work), and interventions that address the job-related causes of employee stress are recognized as essential elements for improving employee health and well-being (National Institute of Occupational Safety and Health, 2015).

**CONCLUSIONS**

Our study provided support for enhanced physiological responsivity to and reduced recovery from an acute stressor for individuals suffering from chronic anxiety. Further, our results supported the neutralizing effects of perceived job control on physiological reactivity and recovery when individuals are exposed to an acute stressor. Individuals who have more resources appear to be more resilient to acute stressors than individuals without these resources. Research examining different types of resources is still needed, and organizations that provide resources for employees (e.g., job control) may benefit from having more resilient employees.
Disclosure

The authors declare no conflict of interest.

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