The Role of Worry and Affectivity on Physiological Responses to an Acute Stressor

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The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

The authors have no relevant financial or non-financial interests to disclose.

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Abstract

The explanation for how acutely stressful experiences could result in proximal health outcomes has been lacking in occupational health research. Although scholars have argued that individual personality and affect could worsen health behaviors, we believe that these qualities also could intensify the experience of acute stressors, potentially explaining why acutely stress encounters result in poor health outcomes for some people, but not others. Our study examines three individual differences - worry, negative affect, and positive affect - that are relevant to differential stress anticipation, reactivity, and recovery. Study participants, who were full-time professional or managerial employees, attended a clinic where we gathered data on their trait worry, and state negative and positive affect. Then, they took part in an experimental exercise that should reflect stressful experiences at work (i.e., cognitive stressor with social pressure). The clinician collected measures of participant facial muscle tension, skin temperature, blood pressure, respiratory breathing, and heart rate, before, during, and after the stressful exercise. Results suggest that only positive affect magnified stress during the anticipation of the experiment, both worry and negative affect intensified the negative physiological effects of the stressor in two of the three experimental stages, and only negative affect delayed physiological recovery and relaxation. Our findings augment our understanding of how individual differences affect physiological responses to acute stress.

Keywords: Worry, Negative and Positive Affectivity, Acute Stress, Physiology

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Scholars have argued that physiology should be valued within our understanding of the work context (Heaphy & Dutton, 2008), particularly regarding occupational health and well-being (Ganster & Rosen, 2013). Many work roles involve extended work hours and high pressure (Lovelace et al., 2007), and the American Psychological Association argues that stress levels are presenting a mental health crisis (APA, 2020). Consequently, understanding the physiological ramifications of workplace stress is valuable to both research and practice.

Acute stressors have a specific onset and offset (e.g., minor workplace accidents), and research has demonstrated that physiological reactivity (e.g., elevated blood pressure) to these can lead to long-term health consequences, such as hypertension and poor health (Brown et al., 2017; Hamer & Steptoe, 2012). Moreover, the *magnitude* of acute stress response is related to future negative health and disease consequences (Turner et al., 2020). Although everyone experiences acute stressors occasionally, health consequences vary based on individual differences in physiological responses (Kiecolt-Glaser et al., 2020). Thus, when heightened physiological responses to acute stress occur, there could be long-term negative effects (e.g., poor health outcomes).

Further, personality affects individual responses to an acute stressor (Xin et al., 2017), and negative emotional experiences increase susceptibility to strain (Zellars et al., 2009) and illness (Friedman & Booth-Kewley, 1987). However, it is unclear which individual differences affect the response to (i.e., anticipation of, reactivity to, and recovery from) these acute work stressors such that they result in later health consequences. Research has posited that some individual differences could affect long-term stress responses (e.g., negative emotions; Brosschot & Thayer, 2003), but we believe these characteristics also could affect acute stress response, suggesting a link with later health outcomes.

Frequent and high physiological responding to acutely stressful experiences may lead to dysregulation and disease (Cacioppo et al., 1998; Gerin et al., 2000), and the results of a meta-analysis of acute laboratory stress support these conclusions (i.e., Marshland et al., 2017). Further, it has been argued that stress reactivity is due to one's biological sensitivity to context (Boyce & Ellis, 2005) and both positive and negative emotions have related to, for example, heart rate and blood pressure reactivity (Jacob et al., 1999). Thus, we believe that negative and positive affectivity and other related characteristics (e.g., worry) can represent one's biological sensitivity to acutely stressful situations. In other words, these same emotions and moods that have related to both prolonged activation (e.g., Brosschot & Thayer, 2003) and activation on typical workdays (e.g., Jacob et al., 1999) should also relate to one's response to acutely stressful encounters.

We address this gap by testing the physiological effects of heightened state negative and positive affectivity, as well as trait worry, on acute stressor anticipation, reactivity, and recovery in working professionals across a range of industries. In an experimental setting, after baseline and anticipation phases, we use a prototypical mental stressor test across three phases (i.e., out loud without pressure, in mind, and out loud with pressure), applying social pressure in the final phase to ensure our stressor's relevancy to typical work demands (Lovallo, 2015). Post-experiment, we examine both initial four-minute recovery and longer eight-minute (instructed) relaxation periods.

Our study examines multiple physiological outcomes, including muscle tension, blood pressure, respiratory rate, heart rate and skin temperature, as scholars recommend (e.g. Schwartz et al., 1996). The design overcomes weaknesses of prior occupational stress experimental research, such as examining only men (e.g., Wirtz et al., 2013), assessing acute stress retrospectively (Frazier & Parker, 2019), or conducting an experiment too briefly to find effects (e.g., Cendales-Ayala et al., 2017). Furthermore, framed by the appraisal model

(Lazarus & Folkman, 1984), we suggest that these individual differences that have been related to long-term health outcomes also influence acute stress responses.

Acute Stress and Health

Acute stress is typically considered to result from a discrete response to a stimulus. For a stimulus to be regarded as an acute stressor, it must be perceived as a threat (Lazarus & Folkman, 1984) resulting in what has been termed as a "fight-or-flight" physiological response (Canon, 1914), a shift toward sympathetic arousal (Selye, 1956 & 1978), and a deviation from homeostasis (McEwen, 2006). The acute stress response ends when the body returns to homeostasis.

Selye and McEwen similarly described the harm of continued stress response. According to Selye's General Adaptation Syndrome, continued acute stress response leads to the body's inability to resist the stressor, resulting in eventual exhaustion and disease (Selye, 1956 & 1978). Similarly, McEwen argued that repeated and frequent stress responses lead to an increased disease risk due to how the body adapts to this allostatic load (McEwen, 2006). As noted by other scholars (e.g., Smyth et al., 2013), both consequential stress theories posit that the risk of harm is due to an individual's cumulative stress response over time.

An important area of study is what causes heightened response to acute stress. Moreover, as implied in these models, the frequency of stress activation (e.g., repeated mental stressor representation), inability to adapt (e.g., continued threat appraisal), and delay in returning to a resting state (e.g., assessment of heightened general threat) result in a greater likelihood of acute stress persisting. Consequently, factors present in individuals' daily experiences (e.g., individual traits and affect) that could result in repeated and frequent intensified stress responses warrant investigation. In response to the call for investigations in stress response moderators (e.g., Smyth et al., 2013), we believe that individual differences

can augment acute stress response, such that, if frequently repeated, later health outcomes could be affected.

Individual Differences and Health

Research shows that individual differences are associated with acute stress reactivity (see Chida & Hamer, 2008, for a review). For example, in a recent study that compliments the current research, Schmid, Thomas, and Rentzsch (2024) investigated individual differences in physiological reactivity due to the acute stressor of time demands. Their research takes place in a real-life setting (rather than a laboratory), examining physiological and psychological outcomes several times each day. Specifically, it was demonstrated that reported increased time pressures were associated with lower heart rate variability, and that interindividual heart rate variability related to greater emotional exhaustion. These findings suggest that individual differences in physiological stress reactivity are not only associated with long-term but also with short-term negative effects (e.g., everyday emotional exhaustion).

These results are consistent with the transactional model of stress (Lazarus & Folkman, 1984), which suggests that stress reactivity is due to the individual appraisal process, which, in turn, is highly influenced by individual attributes. Characteristics (e.g., worry) affect the way individuals typically appraise their surroundings and cope with stressors (Semmer, 2003). Indeed, individual differences affect the interpretation of, response to, and recovery from a stressor, making them fundamental to understanding the stress process (Meurs & Perrewé, 2011).

Moreover, individual characteristics affect health. Depending upon individual perceptions and patterns of emotional responding (Kobasa, 1979), people experience certain physiological responses when confronted by environmental challenges. For instance, negative emotional experiences create a vulnerability to illness (Friedman & Booth-Kewley, 1987),

and emotional states associate with patterns of physiological functioning (Salovey et al., 2000). Further, dispositions have been linked to the development of physical diseases (Friedman & Booth-Kewley, 1987). Dispositions affect individual worldviews, leading to greater responses to stimuli and possible long-term detrimental outcomes.

However, reactivity cannot fully explain the individual stress response (see Schwartz et al., 2003; see Sonnentag & Fritz, 2015). Some individual differences can dramatically *prolong* physiological activation (e.g., Brosschot et al., 2006; Brosschot & Thayer, 2003), even outside of the stressor's presence, resulting in heightened anticipation of and decreased recovery from stressors (Brosschot et al., 2005). A more complete understanding of how individual differences impact the physiological response to acute stress would have valuable implications for research and practice. Theoretically, it is important to understand how dispositions affect stress experiences, and to what extent they affect the entirety of it (i.e., the anticipation of, reaction reactivity to, and recovery from such situations). Also, individual differences influence how employees experience workplace stress interventions (Ganster & Rosen, 2013), suggesting that this improved understanding could promote the adoption of more tailored work practices.

Although the majority of research on how individual differences affect health explores the long-term effects across many stressful encounters, we believe effects should also be found in acute stressor response. Recreating a past stressor or envisioning a future stressor activates physiological systems that could be observed in acutely stressful situations (Gerin et al., 2006). Thus, it is imperative to better understand how individual characteristics can affect acutely stressful situations, potentially lead to negative health outcomes.

In a conceptual model of PA and health, Pressman and Cohen (2005) proposed that state affect has main effects on health through pathways such as: positive health behaviors; protective or health-relevant physiological changes; and social, psychological/intellectual,

and physical resources. Although it might be surprising that experiencing affect "at the moment" could influence health decades later, short-term assessments can predict distal outcomes (Pressman, Jenkins, & Moskowitz, 2019). Thus, there is evidence that state NA and PA have extensive correlations with a plethora of health outcomes (Pressman et al., 2019).

In the current study, we examined acute stress responses, and we concur with other scholars that these proximal outcomes could influence latter daily (e.g., emotional exhaustion; Schmid et al., 2024) and longer-term health outcomes (Pressman, et al., 2019). Our research examines state negative affectivity and its cognitive correlate (Beck et al., 2001), trait worry, and we believe that both constructs are detrimental to anticipating, reacting to, and recovering from an acute stressor. Furthermore, we contend that, in response to an acutely stressful encounter, greater state positive affectivity will increase anticipatory and reactivity stress, but also improve physiological recovery. From a practical standpoint, in organizations, when those with high degrees of negative affectivity, positive affectivity, or worry experience heightened physiological stress activation, it not only affects the individual, but it also affects coworkers, because of the behaviors (e.g., poor decision-making or abusive behavior) that could result from this intensified physiological stress response.

Worry

Worry is the thoughts, emotions, and images of a negative nature that are not only repetitive, but uncontrollable (Brosschot et al., 2005). Worry can be centered on real and/or imaginary issues. The most worrisome individuals typically experience symptoms such as fatigue, difficulty concentrating, muscle tension, and sleep disturbance. Trait (chronic) worriers are more likely to lack confidence in themselves, perceive problems as threats (Llera & Newman, 2020), and become easily frustrated when dealing with a blocked goal (Hirsch & Mathews, 2012). Across three studies, Rosen and Hochwarter (2014) demonstrated more

negative psychological, attitudinal, and behavioral outcomes for individuals who were ruminators and worriers facing an occupational stressor.

Worry's effects in everyday life are associated with a plethora of negative health outcomes, such as coronary heart disease (Hamer et al., 2012), sleep problems (Takano et al., 2012), and overall mortality (Hamer, et al., 2012). Further, worry is the primary symptom of generalized anxiety disorder (Newman et al., 2013) and, in a meta-analysis, worry associated with higher blood pressure and heart rate (Ottaviani et al., 2014). Daily worrying leads to heightened cortisol in adolescents, suggesting an increased risk for negative health symptoms (Arbel et al., 2017). Rumination, conceptually similar to worry, increases physiological activation (Querstret & Cropley, 2012). Although Vahle-Hinz and colleagues (2014) found that work-related rumination positively related to nighttime heart rate variability during the weekend, the authors speculated that this finding could have been due to a stressful work week leading to an increased need to process (i.e., ruminate) and recover during the weekend. Worry and rumination also are associated with slower blood pressure recovery from experienced stress (Glynn et al., 2002; Gerin et al., 2006).

Felt stress mediates the relationship between the duration of ruminative and worried thinking and elevated diastolic and systolic blood pressure (Birk et al., 2019), and a literature review concluded that worry's link with coronary heart disease is partly due to its relationship with increased blood pressure (Tully et al., 2013). In an experimental study, a group that was instructed to worry for ten minutes maintained a heightened level of electromyographic (EMG; facial muscle tension) activity while worrying (Sykes, 2005). Moreover, although worry has been conceptualized as being future-oriented (Borkovec et al., 1998), research has shown that worrying can prolong physiological recovery from an experimental stressor (Capobianco et al., 2018).

Given that much of the experimental research induces worry and that some only induce worry for a few minutes (e.g., Davis et al., 2002; Fisher & Newman, 2013), we wanted to examine individual propensity to worry. Research has long demonstrated that worry can evolve (e.g., Borkovec et al., 1991) and can be viewed as a stable trait. Therefore, trait worry's effects on poor long-term health could be related to the detrimental impact of worrying on immediate physiological stress response. In sum, we argue that individuals with high levels of trait worry will be more likely to demonstrate intensified physiological anticipation of and reactivity to an acute stressor and will have a delay in their physiological recovery.

H1a: Individuals reporting higher levels of worry will show a greater rate of increase in physiological responses prior to the acute stressor

H1b: Individuals reporting higher levels of worry will show a greater rate of increase in physiological responses during the acute stressor

H1c: Individuals reporting higher levels of worry will show a less rapid reduction in symptoms during the recovery period

Negative Affectivity

We also examine individual state affect, as affect plays a primary role in human experiences (Gray & Watson, 2007), and describes individual feelings, moods, and emotions (Rosenberg, 1998). State negative affectivity (NA) is the assessment of a person's current experiences of nervousness and irritability (Watson et al., 1988). Their hyper-responsivity mechanism suggests that high NA individuals are more likely to have a heightened strain response to perceived stressors (Perrewé & Spector, 2002).

In contrast to worry, which operates within one's awareness, NA produces an emotional susceptibility to stress that does not require cognition, and, in an experience sampling study, momentary NA was positively related to blood pressure (Ilies et al., 2010).

The unconscious aspects of stress could be a considerable part of physiological stress responses (Brosschot et al., 2014), whether occurring before the anticipated stressor (Hall et al., 2001) or after an experienced stressor (Brosschot et al., 2007). For example, induced state NA in participants reduced blood pressure recovery after experienced stress (Radstaak et al., 2011).

Considerable evidence has linked NA to a heightened strain response to perceived stressors, such as those at work. Spector and Jex (1998) meta-analytically showed that NA related to physical health symptoms. NA also has related significantly to psychological strains of depression (Fortunato et al., 1999) and frustration at work (e.g., Fortunato et al., 1999; Fox & Spector, 1999), as well as with behavioral strains of abusive or aggressive behavior toward coworkers, sabotage, and stealing (Douglas & Martinko, 2001; Fox & Spector, 1999; Tepper et al., 2001). Given the negative health and behavioral problems associated with worrying and NA, a more thorough appreciation of how these (trait and state) characteristics affect physiological anticipation, reactivity, and recovery from acute stressors is warranted. Thus, we hypothesize that individuals with high levels state NA will be more likely to demonstrate intensified physiological anticipation of and reactivity to an acute stressor and will have a delay in their physiological recovery.

H2a: Individuals reporting higher levels of NA will show a greater rate of increase in physiological responses prior to the acute stressor

H2b: Individuals reporting higher levels of NA will show a greater rate of increase in physiological responses during the acute stressor

H2c: Individuals reporting higher levels of NA will show a less rapid reduction in symptoms during the recovery period

Positive Affectivity

Lastly, we investigate state positive affectivity (PA). PA is the tendency to experience positive emotions, such as enthusiasm, energy, and happiness. PA affects the way individuals perceive their jobs and their ability to function well in stressful situations. For instance, Fox and Spector (2000) found that high PA individuals had better interview performance. Further, Kraiger, Billings, and Isen (1989) experimentally manipulated positive affect and found that affective state had a positive influence on task perceptions and task satisfaction.

From a theoretical perspective, Fredrickson's broaden and build theory (2000) suggests that PA increases resilience and adaptation to stress. PA associates with physiological health (Fredrickson & Levenson, 1998) and psychological functioning (Nelson & Knight, 2010), and we argue that this is particularly helpful *after* the stressful encounter has concluded. In support, after a stressor was terminated, induced PA was beneficial to strain reduction (Fredrickson et al., 2000). Also, positive mood prior to an experiment related to increased recovery speed after a stressor ended (Tugade & Fredrickson, 2004). Thus, it seems that PA provides the ability to leverage positive emotions to experience better stress recovery (Cardon & Patel, 2015; Gallagher & Meurs, 2015).

Nevertheless, although PA has been used as an indicator of challenge appraisal (e.g., Ferguson et al., 1999), researchers have found that positive expectations (e.g., optimism or PA) manifested in approach behaviors can result in short-term psychological (Frank et al., 2022; Lee et al., 2022) and physiological costs (e.g., Nes et al., 2005; Segerstrom, 2005). Although PA could have protective functions after a stressor concludes, high PA individuals also have high activation and engagement when anticipating and experiencing stress, due to the tendency to experience strong (positive) emotions. In comparison to more mild feelings, the elation and happiness experienced by high PA individuals have related to blood pressure increases (Jacobs et al., 2007). Consequently, highly active and engaged responses to environmental challenges could be physiologically damaging. For example, both NA and PA

have *positively* related to individual activation (Brosschot, et al., 2005), such as elevated heart rate (e.g., Ilies et al., 2010).

These findings support a circumplex model of emotion, where contrary emotional states (i.e., NA and PA) have common biological pathways (Posner et al., 2005) and, thereby, could share similar physiological responses given similar activation. Therefore, high state PA could have short-term physiological costs, possibly due to the increased arousal of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis (Burgdorf & Panksepp, 2006; Lovallo, 2015). In support, higher levels of high arousal PA have associated with increased heart rate and systolic blood pressure (Armon et al., 2014).

In sum, we argue that individuals high on state PA will anticipate and react to stressors with exaggerated physiological responses. However, because PA increases resilience and adaptation to stressful situations (Fredrickson, 2004), we believe that high PA individuals will recover more quickly after the acute stressor than those low on PA.

H3a: Individuals reporting higher levels of PA will show a greater rate of increase in physiological responses prior to the acute stressor.

H3b: Individuals reporting higher levels of PA will show a greater rate of increase in physiological responses during the acute stressor.

H3c: Individuals reporting higher levels of PA will show a greater rate of decrease in symptoms during the recovery period.

Method

Sample

Data were collected over a two-year period from full-time employees from a wide range of organizations (i.e., manufacturing, finance, services, and health care) in the State of Rio Grande do Sul in Brazil. Participants were randomly selected from different occupational levels and various professional occupations. All companies emphasized to

employees that participation was voluntary, and there would be no penalty for refusing to participate. Participants were asked to sign a consent form explaining the purpose of the study, assuring confidentiality, and providing contact information for the Brazilian researcher. They also were offered two free stress management and biofeedback sessions. Ethics board approval was received for the study and individual informed consent was obtained. The project began with 191 participants, and complete data (i.e., both background survey data and experimental data, as described below) was collected from 189 participants, which formed our analysis sample. The sample had an average age of 40 years (SD = 8.31, range 24-61), and was 54% female.

Procedure

A clinical psychologist (i.e., one of the authors) collected data from each participant individually at a biofeedback clinic. During the first visit to the clinic, each participant completed a background survey containing demographic questions, as well as a measure of trait worry. The survey had been translated from English to Portuguese and back translated by two English teachers, fluent in both languages. The two translators worked independently. Only a few minor discrepancies in wording emerged, and the translators resolved these as they discussed the differences.

The participants returned to the clinic approximately one week later to take part in the study experiment. On arriving, participants took a seated, resting position and completed a short survey measuring state negative and positive affectivity. On completion of this survey, the clinician collected initial measurements of the participant's physiological condition, in the form of facial electromyography (EMG), blood pressure, respiratory breathing, skin temperature, and heart rate. Further measurements of each of these outcomes were taken synchronously at 120 second intervals throughout the laboratory session, which lasted 32

minutes, yielding 17 time points of data in total. Please see Figure 1 for illustration of our measurement time points.

<INSERT FIGURE 1 HERE>

The first 240 seconds of the laboratory session comprised the *baseline* period (i.e., time points 1-3), after which participants were given information about the forthcoming experiment. This marked the beginning of a second pre-experimental condition, the *anticipation* period (i.e., time points 3-5), which lasted a further 240 seconds. Participants then undertook *experiment condition 1* (i.e., time points 5-7), in which they had to perform a subtraction task out loud, counting backwards from 1,000 by 7's, for 240 seconds. The cognitive process of making mathematical calculations activates regions of the brain's cerebral cortex (Eger et al., 2003; Zarjam et al., 2013) and requires coordination between basic and complex cognitive processes, yielding a good example of high-level cognitive skill (Pesenti et al., 2001).

Although, on the surface, cognitive stressors, such as used in this study, might not have the appearance of real-life work, they include time pressure, demands on memory, analysis, and vigilance (Flynn & James, 2009), which are all core elements of professional occupations. Moreover, mental arithmetic tasks are thought to be a prototypical mental stressor due to not only their effectiveness as a stressor, but also minimal motor demands, resulting in cognitive demands that are analogous to the workplace (Lovallo, 2015).

Experiment condition 2 (i.e., time points 7-9), again lasting 240 seconds, involved the same subtraction task, but with participants required to attempt it only in their mind. *Experiment condition 3* (i.e., time points 9-11) again involved the same subtraction task for 240 seconds, but this time performed out loud. During this stage, the clinical psychologist spoke assertively and put individuals under pressure by repeatedly telling them to perform more quickly, suggesting to the person that he (she) was not doing well. By including social

pressure, we believe this phase adds an element (i.e., interpersonal demands) that makes the experiment even more reflective of workplace stressors. This concluded the active experimental periods of the study.

Two post-experiment conditions followed. First was *recovery* (i.e., time points 11-13), where participants were allowed to recover for 240 seconds. Last was *relaxation* (i.e., time points 13-17), where participations were told to attempt relax, and this period was a total of 480 seconds, to give participants ample time to relax. The individual was in a seated and relaxed position during all the conditions and measurements taken.

Individual Difference Measures

To measure trait worry, collected one week before the laboratory session, we used the Penn State Worry Questionnaire (Meyer et al., 1990). The 16-item Penn State Worry Questionnaire measures trait worry (e.g., "My worries overwhelm me") using a Likert rating from 1 (not at all typical of me) to 5 (very typical of me). The internal consistency reliability of this measure was high (Cronbach's alpha = 0.895, McDonald's omega = 0.931).

To measure state negative and positive affectivity, collected just before the laboratory session, we used Watson, Clark, and Tellegen's (1988) Positive and Negative Affect Schedule (PANAS) measure, one of the most widely used scales to measure affectivity. PANAS is comprised of 20 items, with 10 measuring negative affectivity (e.g., upset, afraid) and 10 measuring positive affectivity (e.g., inspired, excited). The internal consistency reliability of both positive and negative affect subscales was adequate (positive affect: alpha = 0.776, omega = 0.830; negative affect: alpha = 0.818, omega = 0.886).

Physiological Measures

Physiological acute stress reactivity involves a multisystem of responses by the autonomic nervous system, which regulates certain bodily processes without a person's conscious effort. Thus, our choice of study outcomes was designed to capture the autonomic

nervous system, which can be affected by individual differences, such as worry (e.g., Knepp et al., 2015).

Muscle tension was measured using facial EMG, involving a series of electrodes being applied to the skin to record the nerve's stimulation of muscle. The electrodes were centered on the forehead between the eyebrows and in the middle of each eyebrow (approximately 2.5 cm from the central electrode). EMG is a measure of muscular contraction/relaxation. It measures the electrical impulses that cause muscle fibers to contract, indicating muscle activity.

Blood pressure was measured using a sphygmomanometer placed on the upper right arm, which is a device with an inflatable cuff to collapse and then release the artery under the cuff in a controlled manner. Blood pressure is typically reported as two numbers; systolic is the pressure of the blood against the artery walls when the heart contracts, and diastolic is the pressure against the artery walls when resting.

Respiratory rate refers to an individual's respiratory pattern, specifically, the respiration rate in breaths per minute (inhalation-exhalation ratio). An elastic belt buckled horizontally around the upper chest was adjusted to fit snugly (not too tight nor too loose). The respiration sensor along the belt was positioned on the right side of the chest.

Heart rate, parameterized as the number of times the heart beats in the span of a minute (BPM), was measured using a stethoscope.

Skin temperature was recorded with a thermistor taped to the distal finger pad of the right index finger. Peripheral skin temperature is regulated by vasomotor control mechanisms. Emotional stress can cause sympathetic activation causing vasoconstriction in the hands and feet as the muscles contract around the blood vessels. As the blood flow is shifted to the tense muscles, the temperature gets colder, indicating physiological strain.

It is worth noting that, although cortisol is a widely used measure of experienced strain, it is inappropriate for our study. Cortisol levels do not rise until approximately 15 minutes after stress onset, and they do not decrease for several hours (Blackburn-Munro & Blackburn-Munro, 2003; Dedovic et al., 2009; Hanibal & Bishop, 2014). Given this pattern, cortisol measures would not allow us to test differences in anticipation, response, and recovery to acute stressors.

Control Variables

We controlled for the effects of age (years) and gender (1 = male vs 0 = female) in our models, since both have been shown to affect physiological measurements such as heart rate and muscle tension (Schwartz et al., 1980; Stoney et al., 1987).

Statistical Analysis

We modelled the change over time in each outcome using a piecewise (also known as discontinuous) multilevel growth model (Singer & Willett, 2003; Pinheiro & Bates, 2000; Grimm et al., 2016) where observations over time (the lower level, N1 = 3213) were considered as nested within subjects (the higher level, N2 = 189). As well as the separation of the variance into within and between subjects' components implicit in a multilevel growth model, such piecewise or discontinuous modelling allows the rate of within subjects change to vary between distinct periods of time (Singer & Willett, 2003). This makes the technique suitable for modelling repeatedly measured outcomes where the data collection points are divided by a series of interventions. In this study, our 17 study time points can be thought of as being divided into 7 segments or periods corresponding to the specific study conditions described above, as illustrated in Figure 1. The modeling of discontinuity in change across the course of a study is achieved by fitting separate indicator variables representing the distinct time periods (Singer & Willet, 2003).

For each of our six outcomes, our modelling strategy consisted of three distinct stages: first, modelling the pattern of change over time with random intercepts and piecewise fixed effects representing change in specific periods; second, allowing these period-specific slopes to vary by subject; and third, attempting to explain this slope variability by our hypothesized covariates of worry, negative affect, and positive affect.

In the first stage, we initially fitted an unconditional model (intercept only: model 1) to obtain a baseline for model fit and within- and between-subject variance estimates. To model the pattern of change over time, we began with a single coefficient estimating the linear effect of time across all time points (model 2a), and then sequentially separated change over time into distinct temporal pieces corresponding to the implementation of the experimental conditions within our study. Specifically, we modelled pre/during and post-experimental change (model 2b); then additionally separated the first 10 time points into separate pre- and during experiment periods (model 2c); then additionally segmented the experimental period according to the three experimental conditions (model 2d); then split the pre-experiment period into baseline and anticipation periods (model 2e); and finally, the post-experiment period into recovery and relaxation (model 2f). Where model fit was improved, the respective additional time variable was retained: this gave a model with up to 7 potential distinct periods of change, corresponding to the periods illustrated above in figure 1 (i.e. baseline: time points 1-3; anticipation periods: time points 3-5; experimental condition 1: time points 5-7; experimental condition 2: time points 7-9; experimental condition 3: time points 9-11; recovery: time points 11-13; relaxation: time points 13-17).

Having identified the best-fitting piecewise model (i.e., the “fixed effect” of change over time) for each outcome, the second stage of the modelling involved adding random effects (variance parameters). First, we fine-tuned the model by adding a within-participants autoregressive type-1 (AR1) covariance structure to the model (i.e., controlling for the lag

effect of the outcome at previous weeks, hence removing this "nuisance" variation; model 3). We then added between-subjects variance coefficients for the effect of each time-period variable to establish whether change within any period of the study varied between subjects (models 4a-4f). Where the addition of such variance parameters improved the fit of the model, they were retained and included together (model 4g) – otherwise the slope coefficient remained fixed. Subject-level covariances between intercept and slope were then fitted and retained if the model was improved (model 5).

Finally, where between-subjects variance in change over time had been identified in a specific period, we attempted to explain this variability from our hypothesized covariates – trait worry, negative affect, and positive affect. This was achieved by adding their main effects, as well as control variables of age and gender, followed by their interactions with the time variable representing the respective period (models 6 and 7 respectively). Testing the estimated coefficients for these interaction terms – and the between-subject variance in change over time within periods that they explain – provide a test and corresponding effect size for Hypotheses sets 1, 2 and 3.

The analysis sequence described above was performed separately for each outcome. We fitted our piecewise multilevel growth models using SPSS statistical software (version 26). The code used for fitting these models is available in a public research repository (https://researchbox.org/2152&PEER_REVIEW_passcode=TCNGHJ), along with the code used for producing the descriptive statistics and plots presented in our results section, and the study dataset itself. Maximum Likelihood Estimation was used for the fitting process. We compared competing models using chi-square difference tests between model deviances. When modelling heart rate, blood pressure, and respiratory rate, we divided the raw score by 10 to ease the fitting process (i.e., the convergence of each parameter to its final estimate under the iterative maximum likelihood fitting process). Our covariates, worry, negative and

positive affect were centered around their respective sample means to aid interpretation of coefficients in the final model. The $p < 0.05$ level of statistical significance was applied throughout when testing model improvement and model parameters, with exact p-values reported, along with confidence intervals. Given the use of multiple (6) related outcomes, p-values were Bonferroni-corrected (i.e., to obtain significance at the level, the value needed to be less than or equal to alpha/6).

Results

Table 1 gives mean scores, standard deviations, and bivariate correlations for the subject level variables within the study. Table 2 does likewise for our physiological outcomes, with the average and range of the bivariate correlations across the 17 time points reported given space restrictions (full correlation matrix for all six outcome measures across all 17 time points available on request). Figure 2 shows the sample mean scores at each time point for each outcome.

<TABLE 1 HERE>

<TABLE 2 HERE>

<FIGURE 2 HERE>

Rows 1 to 7 of Table 3 show the model comparisons as change across the study period was modelled by an increasing number of distinct segments (models 1 to 2f), with Appendix A showing the within and between subject variance component estimates.

The optimal model that emerged followed an identical pattern for each outcome. Separating change over time into two pieces (pre/during experiment and post experiment parts) improved model fit (model 2b vs model 2a). Fit was further enhanced by splitting the pre/during period by separating the growth terms for Pre and During (model 2c vs 2b); then by separating the ‘during’ experiment period into three segments, one for each experimental condition (model 2d vs 2c); and by separating the post-experiment period into recovery and

relaxation sections (model 2f vs 2d). However, separating the pre-experiment period into baseline and anticipation did not produce any improvement (model 2e vs 2d), hence the entire pre-experiment period (i.e., time points 1 to 5) was kept as a single period. For each outcome, adding an autoregressive type I within-subjects correlation structure to model the 'lag' effect also improved model fit (model 3 vs Model 2f).

When allowing the change over time in each period to vary between subjects, slightly different patterns of results were found across the outcomes (models 4a-4g: see table 3, rows 9 to 15). For all outcomes, allowing the rate of decrease in symptoms in the recovery and relaxation periods to vary between subjects improved the model fit. Additionally, for skin temperature and heart rate, there was evidence that the rate of increase in symptoms varied between subjects in the 'Out loud, under pressure' condition period (condition 3); and for diastolic blood pressure, the rate of increase in symptoms varied between subjects in the pre-experiment (i.e., baseline/anticipation) period.

For facial muscle tension, when periods were considered separately, model improvement suggested that the rate of increase in symptoms varied between subjects in each of the three experimental conditions. However, when these random (i.e., between-subjects variance) effects were combined within a single model, only that for experimental condition 1 significantly improved the model, hence this was retained alongside the random effects for recovery and relaxation periods.

Models 6 and 7 added covariates of worry, positive and negative affect, and then their interactions with specific time period indicator variables (e.g., BASE, ANTIC, EXP1, etc.) to explain this between-subjects' variation in change across specific periods. For each outcome, model 7 proved a superior fit to the data compared against preceding models. Model comparison tests are given in Table 3, rows 17 and 18; variance estimates in Appendix A.

The coefficients for the fixed effects in the final model for each outcome (model 7) are given in Table 4. In summary, each outcome exhibited a statistically significant increase in symptoms in all pre-experiment and experiment periods, and a statistically significant decrease in symptoms in the post-experiment (i.e., recovery and relaxation) periods.

Finally, for those periods and outcomes where we had found between subjects variation in change, we examined the effect of the three covariates (i.e. worry, negative affect, positive affect) in explaining this between-subject variation, by fitting the interaction terms between each of these variables and the time variable representing the respective period.

Worry intensified the increase in facial muscle tension during the first experimental condition. Negative affect magnified the decrease in skin temperature during the third experimental condition (out loud, under pressure). In the post-experimental segments, negative affect reduced the increase in skin temperature and decrease in blood pressure (both diastolic and systolic) during the recovery period and reduced the predicted decrease in diastolic blood pressure and respiratory rate during the relaxation phase. This pattern of higher levels of worry and negative affect associating with intensified increases in stress responses and delayed recovery from them supports Hypotheses 1b, 2b, and 2c.

The results for the impact of positive affect were less pronounced across multiple outcomes but still offered partial support for Hypotheses 3a and 3b. Positive affect magnified the increase in diastolic blood pressure that occurred in the pre-experimental period, the increase in facial muscle tension during the first experimental condition, and the increase in heart rate during the third experimental condition.

The hypothesized impact of positive affect on changes in stress responses in the recovery and relaxation segments (Hypothesis 3c) were not supported: the effects of positive affect on each outcome’s change over these periods were non-significant.

Figures 3, 4 and 5 illustrate the estimated model for change in each outcome as per model 7 at different levels of worry, NA, and PA respectively, with all other covariates held at their sample mean. Figure 3 plots the model at relatively low (-1 *SD*), medium (mean) and high (+1 *SD*) levels of worry; Figure 4 at low (-1 *SD*), medium (mean) and high (+1 *SD*) levels of negative affect; and Figure 5 shows the model plotted at low (-1 *SD*), medium (mean) and high (+1 *SD*) levels of positive affect.

<TABLE 3 HERE>

<TABLE 4 HERE>

<FIGURE 3, 4, 5 HERE>

Discussion

Findings

A systematic review of acute stress response measures revealed that acute stress has primarily been assessed retrospectively (Frazier & Parker, 2019). Our approach differed, and contributes to the literature by assessing physiological responses prior to, during, and immediately after the acute stressor. We examined characteristics, specifically heightened trait worry and state negative and positive affectivity, that make individuals more (or less) likely to show greater physiological responses to acute stress.

Regarding anticipation of stress, our results suggest that the increases in diastolic blood pressure were intensified by PA. Regarding reactivity to our experimental stressor, two of our three covariates (i.e., worry and PA) intensified the increase in facial muscle tension during the first experimental condition. In addition, during the third experimental phase, NA strengthened the decrease in skin temperature and PA likewise magnified heart rate increases. During recovery, NA slowed both the rebound in skin temperature and the decrease in systolic and diastolic blood pressures. Moreover, during our last phase (i.e., relaxation), NA diminished the decrease in respiratory rate and diastolic blood pressure.

The findings that PA increases reactivity and NA impairs recovery are in line with the Effort-Recovery Model (Meijman & Mulder, 1998). According to this model, stress reactivity alone is not necessarily harmful, but an enduring lack of recovery can be detrimental due to prolonged stress activation. Further, as noted by Geurts and Sonnentag (2006), this sustained activation can be the result of cognitive stress processing, which, in the case of our results, could be driven NA.

Some of our hypothesized relationships were not supported. We did not find that worry was related to a greater rate of increase in responses before our acute stressor. Since prior research often has induced participant worry (e.g., Sykes, 2005), perhaps our lack of instruction resulted in worry's inactivation before the experimental phases. We also did not find that heightened worry resulted in a less rapid reduction in symptoms during recovery and relaxation, which could be because worry is future-oriented (Borkovec et al., 1998). Moreover, NA did not relate to the rate of increase in responses prior to the acute stressor, and this could be due to NA needing a stressor to which it can be hyperresponsive (Perrewé & Spector, 2002). Lastly, PA did not relate to change in our outcomes after the acute stressor concluded, which could be due to the short time (i.e., 12 minutes) we were able to collect post-experimental data.

Theoretical and Practical Implications

Our results demonstrate the importance that scholars connect acutely stressful experiences with individual health. Study participants displayed elevated arousal on health indicators to the experimental stressor, which is a reminder that the repeated experience of acute stressors may yield long-term negative health outcomes (Smyth et al, 2013). Although stress researchers typically separate discussions of acute from chronic stress, it has been argued that both lie on the same continuum of physiological responding (Smyth et al, 2013), and we believe that stress research can be enhanced by incorporating this perspective into

stress models.

Our results from the "out loud, under pressure" experimental condition that the within-individual heart rate increase was intensified for those high on PA and that the skin temperature decrease was magnified for those high on NA are aligned with prior work (i.e., Ilies et al., 2010). Moreover, across the anticipation phase and the first and third phases of the experimental stressor, PA intensified some elements of the stress response. Although our results were not particularly strong, they partly affirm earlier findings (i.e., Armon et al, 2014), and provide modest support for the engagement model (Segerstrom, 2005). This stress perspective argues that high levels of approach behaviors (e.g., high PA) to stressors result in short-term physiological costs, which could be due to elevated arousal of the sympathetic nervous system and the HPA axis (Burgdorf & Panksepp, 2006). Since both state and trait affect are associated with mortality in healthy population studies (Chida & Steptoe, 2008), our results lend some support to the contention that PA can turn short-term negative effects into long-term adaptive effects. More work on PA could investigate this intriguing avenue of study.

Our results also show that elevated state NA delayed physiological recovery and relaxation following the stressor. This finding supports the suggestion by scholars that prolonged activation, rather than mere heightened anticipation and reactivity, could be the underlying mechanism explaining the role of negative emotions in somatic disease (e.g., Geurts & Sonnentag, 2006). An important part of this mechanism's effectiveness is likely due to the extended mental representation of the stressor following the stressor experience, as happens for those high on negative affectivity. Consequently, these results from our study not only reinforce the appraisal model's (Lazarus & Folkman, 1984) application to state affect, but also suggest a possibility that state affect, if repeated and frequent (McEwen, 2006), could be a factor in the prolonged activation leading to long-term health outcomes.

Negative affective experiences have been linked to long-term negative outcomes (Kubzansky & Kawachi, 2000), but PA relates to long-term positive results for individuals (e.g., Cardon & Patel, 2015). Given that PA is related to positive cognitive reappraisal (Fredrickson, 2004) but it did not affect the change in outcomes during the recovery or relaxation periods, it could be that the broadening-and-building of positive emotions that results in reduced strain occurs at a much later time than just a few minutes after the conclusion of a stressor. Studies could examine whether individuals need more time to cognitively reappraise the situation after a stressful encounter.

In practice, organizations can help employees with heightened negative affect or trait worry by examining contingency factors within the work environment. Both positive and negative affectivity influence job performance and worker well-being (Kaplan, Bradley, Luchman, & Haynes, 2009). Interventions aimed at reducing negative emotions (e.g., anxiety) and promoting positive emotions (e.g., excitement) are one way to help employees achieve success and better well-being. Although dispositional variables are reasonably stable, these characteristics are still somewhat malleable and evidence suggests that positive work events can affect long-term changes in employee well-being (Lucas, Clark, Georgellis, & Diener, 2004). For example, consistent and frequent positive feedback and helpful interactions with supervisors can facilitate a nourishing relationship with organizational leaders. Other mechanisms aimed at creating a positive work climate could include encouraging employees to take regular breaks, set realistic goals, and seek positive interactions with coworkers. Lastly, helping employees to understand the connections between self-care (e.g., healthy sleep and eating habits) and well-being also can help workers to cultivate positive health behaviors and outcomes.

Strengths, Limitations, & Directions for Future Research

A frequent concern among stress researchers is the confounding of stressors with

strains when both are subjectively-evaluated, and this has been a particular interest in regards to NA (Spector et al., 2000). But, due to our collection of experimentally induced stressors and objective physiological strain outcomes, one study strength is that our results are not tainted by this possibility. Furthermore, we collected data from working professionals, used a cognitive stressor, and applied social pressure, making our experiment a good analogue to workplace cognitive and social demands (Lovallo, 2015). Consequently, the results of our experiment should be applicable to many workplaces. Moreover, since prior stress experimental studies might have found nonsignificant results due to too short of a laboratory session (e.g., Cendales-Ayala et al., 2017), as recommended by scholars (Hauser et al., 2011), we extended our protocol to 32 minutes (i.e., 8-minute baseline and anticipation, 12-minute task, and 12-minute recovery and relaxation), to help ensure that physiological activation was detected and recovery was possible. Lastly, we used sophisticated statistical analyses that enabled us to model the effects of multiple interventions within the experiment by allowing discontinuity in change for individuals across time. Specifically, through our piecewise mixed linear modelling approach, we were able to examine change within each phase of the laboratory session, quantify between-subject variation in change within each phase, and control for the lag effect from the previous time point. These analyses provided a strong and precise hypothesis testing and should give scholars confidence in our results.

Regarding limitations, our experimental design did not allow for the collection of psychological stress along with physiological stress during the laboratory session. Robust future studies could collect both types of stress to better gauge their alignment in the stress process. Additionally, we were unable to collect our data using respondents exclusively from one organization/industry. Thus, although all participants were working professionals, we were unable to control for organizational and occupational effects on our outcome variables. Future research could test these relationships using only those employed in a single

occupation or organization. Also, although we assessed state NA prior to the collection of any physiological measurements, it is possible that knowledge that one would participate in an experiment could have distorted state NA. Lastly, as noted earlier, it is possible that our individual difference variables (e.g., PA) could demonstrate effects after the 12 minutes of post-experiment recovery and relaxation, but we were unable to collect such data. Future studies that examine a longer-term stress response of those high on PA could better test the broaden-and-build hypothesis (Fredrickson, 2004) to examine the effects of stress more closely on learning, as has been advocated by stress scholars (i.e., Meurs & Perrewé, 2011).

Conclusion

Although our hypotheses were only partially supported, each of the three individual differences that we tested yielded a unique pattern of significant results. As noted by other researchers (Smyth et al., 2013), differing individual stress response patterns could lead to varying degrees of experienced stress and subsequent disease risk. Therefore, future studies could examine when and how these response patterns contribute to poor health. Additionally, as has been noted by other organizational researchers (Heaphy & Dutton, 2008), future studies could make greater use of physiological measurements in the organizational sciences, and we hope our study contributes to this growing literature.

Conflict of Interest Statement

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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Figure 1

Study conditions and time points at which physiological data was collected

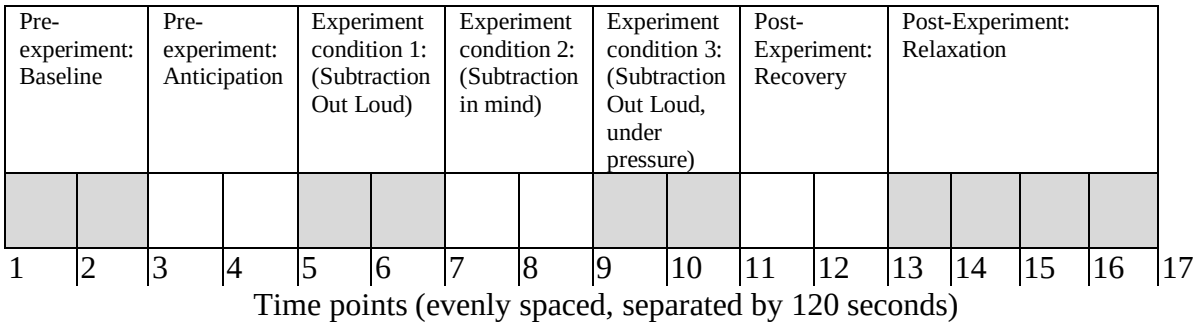


Table 1

Descriptive statistics and bivariate correlations for subject level study variables

Variable	Mean	Std Dev	1.	2.	3.	4.
1. Sex (1 = male, 0 = female)	0.460	0.500	---	---	---	---
2. Age (years)	39.529	8.306	0.100	---	---	---
3. Trait worry (grand mean centered)	0	0.790	-0.054	0.012	---	---
4. Negative affect (grand mean centered)	0	0.546	0.139	-0.059	0.074	---
5. Positive affect (grand mean centered)	0	0.423	-0.058	0.198	-0.124	0.071

N2 = 189 subjects

Table 2

Descriptive statistics and bivariate correlations for observation level study variables

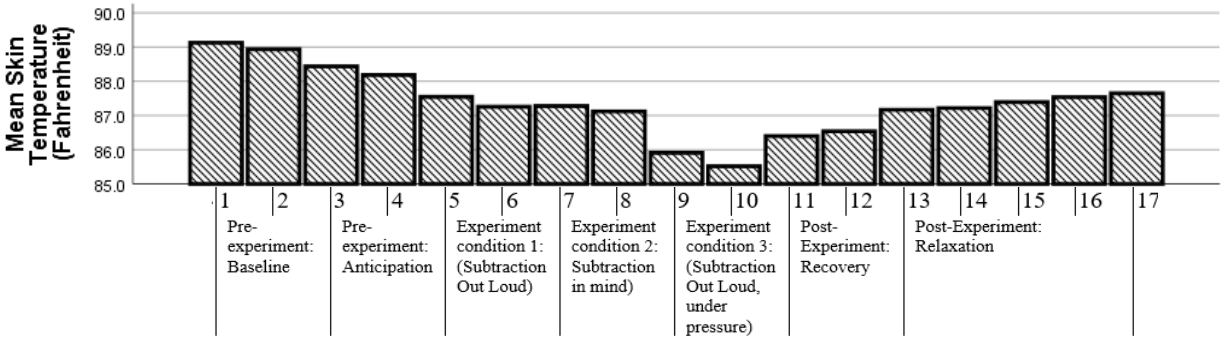
	Mean (average of time point-specific sample means)	Mean (Range of time point-specific sample means)	Std Dev (Range of time point-specific sample *SD*s)	Correlations (Range of time point-specific bivariate correlations)				
Variable		-	-	1.	2.	3.	4.	5.
1. Skin Temperature (Fahrenheit)	87.365	85.520 – 89.125	3.910 – 4.470	---	---	---	---	---
2. Face Muscle Tension (μV)	11.696	8.167 – 17.232	1.701 – 3.236	-0.253 – 0.107	---	---	---	---
3. Heart Rate (BPM)	86.020	76.862 – 100.947	8.764 – 10.999	-0.119 – 0.222	0.307 – 0.717	---	---	---
4. Blood Pressure Diastolic (mmHg)	89.340	80.085 – 102.037	7.670 – 11.801	-0.261 – 0.012	0.043 – 0.709	0.568 – 0.805	---	---
5. Blood Pressure Systolic (mmHg)	134.370	122.550 – 147.704	9.607 – 14.681	-0.275 -- -0.099	0.354 – 0.640	0.592 – 0.845	0.807 – 0.937	---
6. Respiratory rate (breaths per minute)	19.320	14.455 – 27.852	3.188 – 5.629	-0.344 – 0.095	0.644 – 0.787	0.247 – 0.717	0.127 – 0.733	0.374 – 0.695

N1 = 3213 observations from N2 = 189 subjects. Averages and ranges taken across the 17 study time points

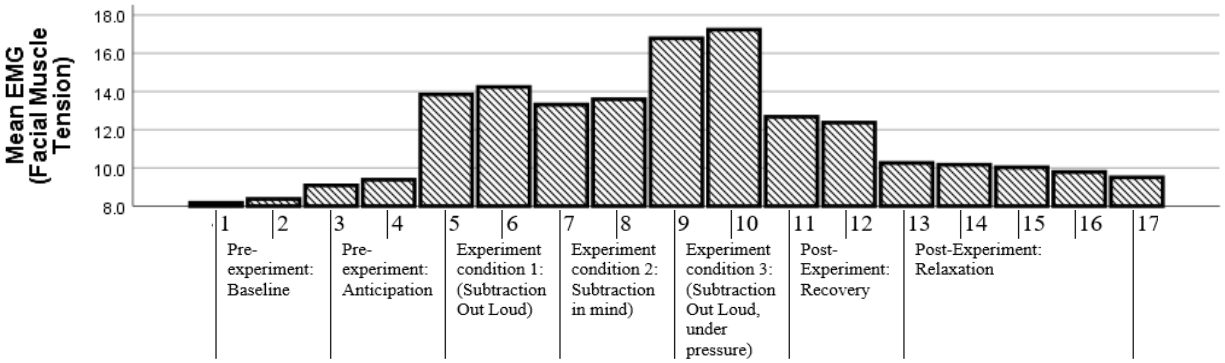
Figure 2

Sample mean scores for each outcome across the course of the study

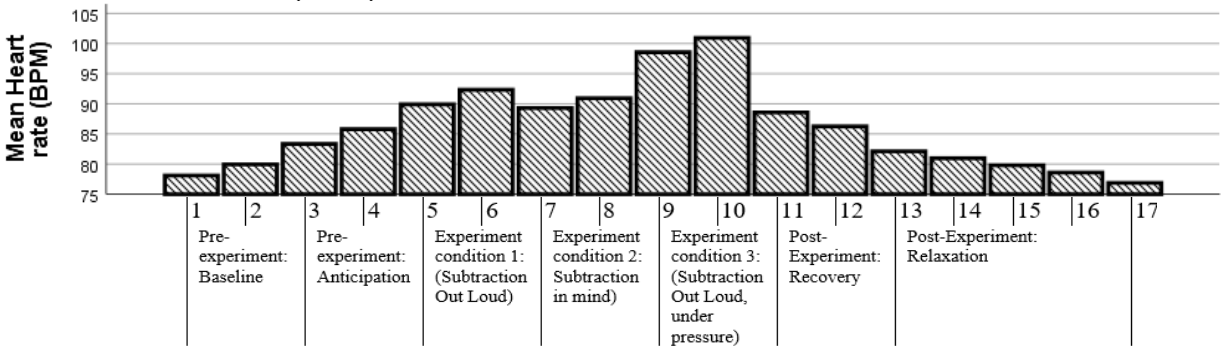
1. Outcome: Skin Temperature (Fahrenheit)



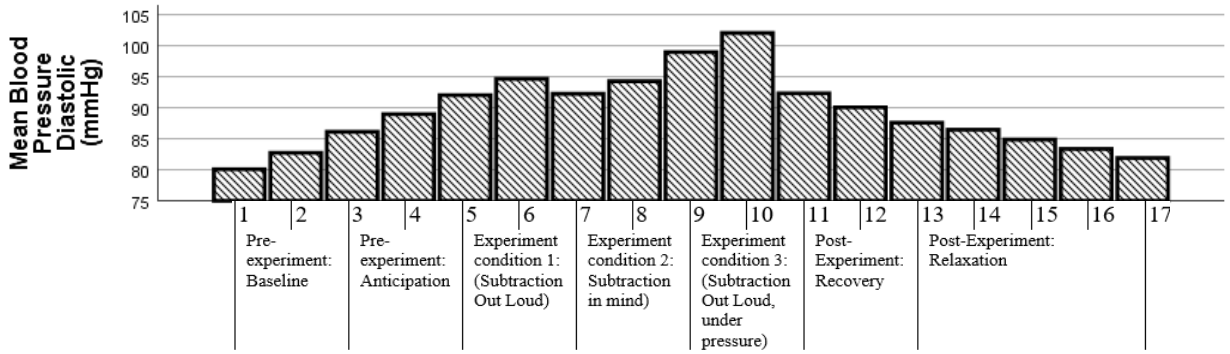
2. Outcome: Face Muscle Tension (µV)



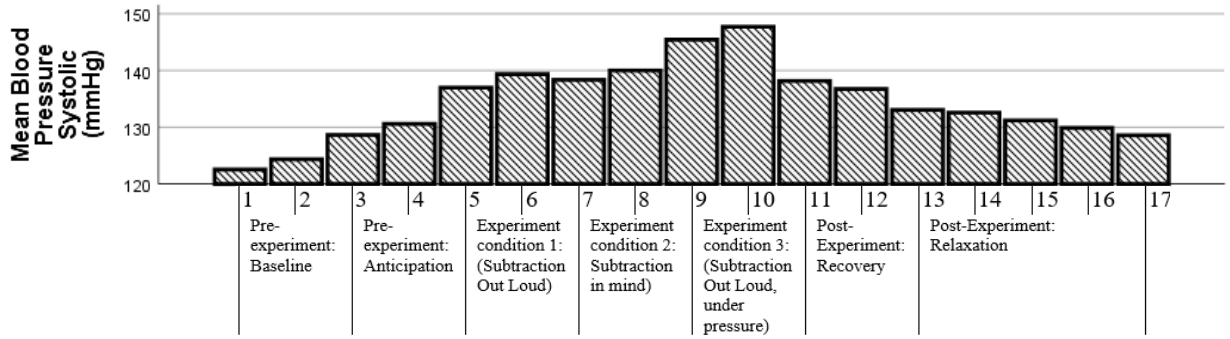
3. Outcome: Heart Rate (BPM)



4. Outcome: Blood Pressure Diastolic (mmHg)



5. Outcome: Blood Pressure Systolic (mmHg)



6. Outcome: Respiratory rate (breaths per minute)

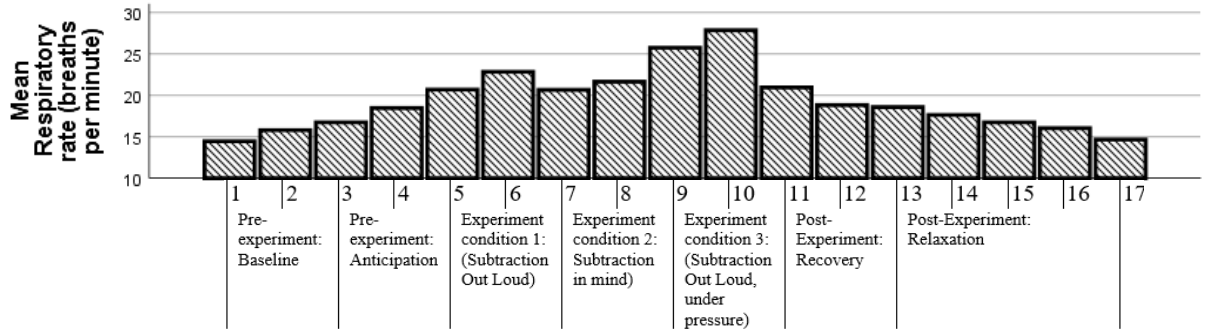


Table 3

Model comparisons of piecewise multilevel growth models for physiological outcomes

Outcome		Skin Temperature (Fahrenheit)			Face Muscle Tension (EMG) (µV)			Heart Rate (BPM/10)			Blood Pressure Diastolic (mmHG/10)			Blood Pressure Systolic (mmHG/10)			Respiratory rate (breaths per minute/10)		
Model	Test vs…	Dev.	Δ Dev, Δ df	*p*	Dev.	Δ Dev, Δ df	*p*	Dev.	Δ Dev, Δ df	*p*	Dev.	Δ Dev, Δ df	*p*	Dev.	Δ Dev, Δ df	*p*	Dev.	Δ Dev, Δ df	*p*
1. Unconditional model, random intercept	---	11974.437	---	---	16902.067	---		8358.458	---	---	7682.673	---	---	8559.889	---	---	4653.111	---	---
2a. Single period of linear change	1	11538.694	435.743, 1*	< 0.001	16893.841	8.226, 1*	0.004	8281.085	77.373, 1*	< 0.001	7677.817	4.856, 1	0.028	8496.07	63.819, 1*	< 0.001	4649.024	4.087, 1	0.043
2b. Pre/during and post experiment change periods	2a	9988.026	1550.668, 1*	< 0.001	14172.161	2721.68, 1*	< 0.001	5376.732	2904.353, 1*	< 0.001	4897.070	2780.747, 1*	< 0.001	5776.169	2719.901, 1*	< 0.001	2353.886	2295.138, 1*	< 0.001
2c. Pre, during, post periods	2b	9987.972	0.054, 1	0.816	14113.776	58.385, 1*	< 0.001	5255.95	120.782, 1*	< 0.001	4712.580	184.490, 1*	< 0.001	5644.394	131.775, 1*	< 0.001	2307.787	46.099, 1*	< 0.001
2d. Pre, condition-specific, post periods	2c	9959.249	28.723, 2*	< 0.001	13748.333	365.443, 2*	< 0.001	5213.125	42.825, 2*	< 0.001	4679.667	32.913, 2*	< 0.001	5538.536	105.858, 2*	< 0.001	2246.549	61.238, 2*	< 0.001
2e. Baseline, anticipation, condition-specific, post periods	2d	9956.554	2.695, 1	0.101	13739.676	5.657, 1	0.017	5209.428	3.697, 1	0.055	4678.801	0.866, 1	0.352	5533.987	4.549, 1	0.033	2246.378	0.171, 1	0.679
2f. Pre, exp'-condition-specific, recovery, relaxation periods	2d	9874.685	84.564, 1*	< 0.001	12945.024	803.309, 1*	< 0.001	4435.597	777.528, 1*	< 0.001	4251.558	428.109, 1*	< 0.001	5213.382	325.154, 1*	< 0.001	1713.609	532.94, 1*	< 0.001
3. Add within-subjects AR1 type autocorrelation	2f	7223.455	2651.23, 1*	< 0.001	12051.318	893.706, 1*	< 0.001	3263.653	1171.944, 1*	< 0.001	2663.379	1588.179, 1*	< 0.001	3203.647	2009.735, 1*	< 0.001	601.065	1112.544, 1*	< 0.001
4a. Allow Pre period change ONLY to vary by subject	3	7223.454	0.001, 1	0.975	12050.682	0.636, 1	0.425	3263.250	0.403, 1	0.526	2654.362	9.017, 1*	0.003	3202.519	1.128, 1	0.288	601.063	0.002, 1	0.964
4b. Allow exp' condition 1 change ONLY to vary by subject	3	7223.453	0.002, 1	0.964	12034.767	16.551, 1*	< 0.001	3261.076	2.577, 1	0.108	2663.379	<0.001, 1	0.999	3200.934	2.713, 1	0.100	600.316	0.749, 1	0.387
4c. Allow exp' condition 2 change ONLY to vary by subject	3	7223.455	<0.001, 1	0.999	12034.324	16.994, 1*	< 0.001	3262.941	0.712, 1	0.399	2657.226	6.153, 1	0.013	3198.687	4.96, 1	0.026	596.977	4.088, 1	0.043
4d. Allow exp' condition 3 change ONLY to vary by subject	3	7168.946	54.509, 1*	< 0.001	12025.726	25.592, 1*	< 0.001	3251.793	11.86, 1*	0.001	2659.083	4.296, 1	0.038	3203.647	<0.001, 1	0.999	600.962	0.103, 1	0.748
4e. Allow recovery change ONLY to vary by subject	3	7211.079	12.676, 1*	< 0.001	11903.604	147.714, 1*	< 0.001	3224.078	39.575, 1*	< 0.001	2656.168	7.211, 1*	0.007	3135.317	68.33, 1*	< 0.001	489.014	112.051, 1*	< 0.001
4f. Allow relaxation change ONLY to vary by subject	3	7202.134	21.321, 1*	< 0.001	12026.349	24.969, 1*	< 0.001	3253.881	9.772, 1*	0.002	2603.605	59.774, 1*	< 0.001	3196.676	6.971, 1*	0.008	554.276	46.789, 1*	< 0.001
4g. Significant variance terms from 4a-f all included (varies by outcome†)	3	7091.341	132.114, 3*	< 0.001	11810.043	241.275, 3*	< 0.001	3208.838	54.815, 3*	< 0.001	2561.364	102.015, 3*	< 0.001	3128.199	75.448, 2*	< 0.001	455.158	145.907, 2*	< 0.001

5. Add intercept-slope and slope-slope covariances (varies by outcome†)	4g	7050.623	40.718, 6*	< 0.001	11695.111	114.932, 6*	< 0.001	3171.978	36.86, 6*	< 0.001	2538.158	23.206, 6*	0.001	3128.199	<0.001, 3	0.998	411.199	43.959, 3*	< 0.001
6. Add main effects of controls, and of worry, pos and neg affect (each grand mean centered)	5	7036.221	14.402, 5	0.013	11677.149	17.962, 5*	0.003	3130.635	41.343, 5*	< 0.001	2524.049	14.109, 5	0.015	3084.132	44.067, 5*	< 0.001	377.201, 5	33.998, 5*	< 0.001
7. Add interactions of worry, pos and neg affect with those time period variables with random effects (varies by outcome†)	6	6974.952	61.269, 9*	< 0.001	11628.623	48.526, 9*	< 0.001	3102.353	28.282, 9*	0.001	2468.420	55.629, 9*	< 0.001	3055.306	28.826, 6*	< 0.001	359.966, 6	17.235, 6*	0.008

N1 = 3213 observations from N2 = 189 subjects., * $p < 0.05$, having Bonferroni-corrected for testing multiple (six) related outcomes

† For Skin temperature and heart rate (BPM), time period variables with random effects were: Experimental Condition 3, Recovery, Relaxation. For EMG, time period variables with random effects were: Experimental Condition 1, Recovery, Relaxation. For Diastolic Blood Pressure, time period variables with random effects were: Pre-experiment, Recovery, Relaxation. For Systolic Blood Pressure and Respiratory rate, time period variables with random effects were: Recovery and Relaxation.

Table 4

Unstandardized regression coefficients from final piecewise multilevel growth model for each physiological outcome (model 7)

Outcome	Skin Temperature (Fahrenheit)			Face Muscle Tension (EMG) (µV)			Heart Rate (BPM/10)			Blood Pressure Diastolic (mmHG/10)			Blood Pressure Systolic (mmHG/10)			Respiratory rate (breaths per minute/10)		
Predictor	B	95% CI	*p*	B	95% CI	*p*	B	95% CI	*p*	B	95% CI	*p*	B	95% CI	*p*	B	95% CI	*p*
(Intercept)	**89.286***	**86.630, 91.942**	**<0.001**	**7.557***	**6.477, 8.636**	**<0.001**	**7.456***	**6.911, 8.000**	**<0.001**	**7.536***	**7.038, 8.035**	**<0.001**	**11.346***	**10.674, 12.018**	**<0.001**	**1.193***	**0.958, 1.428**	**<0.001**
Time: Pre-experiment period	**-0.320***	**-0.364, -0.276**	**<0.001**	**0.618***	**0.536, 0.700**	**<0.001**	**0.262***	**0.237, 0.286**	**<0.001**	**0.295***	**0.268, 0.322**	**<0.001**	**0.275***	**0.250, 0.301**	**<0.001**	**0.131***	**0.115, 0.147**	**<0.001**
Time: Experiment condition 1 period	**-0.464***	**-0.522, -0.406**	**<0.001**	**2.357***	**2.217, 2.497**	**<0.001**	**0.315***	**0.281, 0.349**	**<0.001**	**0.272***	**0.241, 0.302**	**<0.001**	**0.432***	**0.398, 0.466**	**<0.001**	**0.209***	**0.187, 0.231**	**<0.001**
Time: Experiment condition 2 period	-0.066	-0.124, -0.008	0.025	**-0.398***	**-0.518, -0.278**	**<0.001**	**-0.067***	**-0.101, -0.033**	**<0.001**	-0.023	-0.054, 0.008	0.146	0.021	-0.013, 0.055	0.234	**-0.054***	**-0.076, -0.032**	**<0.001**
Time: Experiment condition 3 period	**-0.779***	**-0.867, -0.691**	**<0.001**	**1.633***	**1.513, 1.754**	**<0.001**	**0.474***	**0.433, 0.515**	**<0.001**	**0.377***	**0.346, 0.407**	**<0.001**	**0.366***	**0.332, 0.400**	**<0.001**	**0.306***	**0.283, 0.328**	**<0.001**
Time: Recovery period	**0.544***	**0.465, 0.623**	**<0.001**	**-2.915***	**-3.095, -2.734**	**<0.001**	**-0.774***	**-0.816, -0.732**	**<0.001**	**-0.605***	**-0.637, -0.573**	**<0.001**	**-0.565***	**-0.610, -0.520**	**<0.001**	**-0.442***	**-0.473, -0.412**	**<0.001**
Time: Relaxation period	**0.218***	**0.173, 0.264**	**<0.001**	**-0.383***	**-0.436, -0.330**	**<0.001**	**-0.164***	**-0.180, -0.148**	**<0.001**	**-0.150***	**-0.169, -0.130**	**<0.001**	**-0.153***	**-0.173, -0.133**	**<0.001**	**-0.074***	**-0.087, -0.061**	**<0.001**
Sex (1 = Male, 0 = Female)	-1.254	-2.344, -0.165	0.024	0.284	-0.156, 0.723	0.204	-0.183	-0.404, 0.039	0.105	-0.144	-0.348, 0.061	0.168	-0.048	-0.323, 0.227	0.732	-0.021	-0.116, 0.074	0.670
Age (years)	0.011	-0.055, 0.077	0.750	0.009	-0.018, 0.035	0.521	0.011	-0.003, 0.024	0.117	0.013	0.001, 0.026	0.034	**0.024***	**0.007, 0.040**	**0.006**	0.007	0.001, 0.012	0.027
Trait worry (grand mean centered)	0.028	-0.676, 0.733	0.937	0.368	0.071, 0.666	0.016	0.089	-0.071, 0.250	0.274	-0.073	-0.208, 0.062	0.286	0.000	-0.174, 0.173	0.997	0.055	-0.016, 0.127	0.127
Negative Affect (grand mean centered)	**-1.735***	**-2.761, -0.709**	**0.001**	**0.815***	**0.382, 1.248**	**<0.001**	0.237	0.003, 0.470	0.047	**0.368***	**0.172, 0.565**	**<0.001**	**0.455***	**0.202, 0.708**	**<0.001**	**0.174***	**0.071, 0.278**	**0.001**
Positive Affect (grand mean centered)	-1.099	-2.445, 0.247	0.109	**0.919***	**0.352, 1.487**	**0.002**	**0.482***	**0.176, 0.787**	**0.002**	-0.009	-0.266, 0.249	0.948	**0.685***	**0.353, 1.018**	**<0.001**	0.100	-0.036, 0.235	0.149
Trait worry* Time: Pre-exp' period										0.005	-0.026, 0.036	0.744						
Negative Affect* Time: Pre-exp' period										-0.015	-0.060, 0.029	0.498						
Positive Affect* Time: Pre-exp' period										**0.143***	**0.086, 0.201**	**<0.001**						
Trait worry*Time: Exp' cond' 1 period				**0.179***	**0.046, 0.313**	**0.008**												
Negative Affect*Time: Exp' cond' 1 period				0.240	0.047, 0.432	0.015												
Positive Affect*Time: Exp' cond' 1 period				**0.605***	**0.355, 0.855**	**<0.001**												
Trait worry*Time: Exp' cond' 3 period	-0.089	-0.199, 0.020	0.110				0.004	-0.043, 0.052	0.854									

Negative Affect*Time: Exp' cond' 3 period	**-0.312***	**-0.469, -0.154**	**<0.001**				0.035	-0.034, 0.104	0.316									
Positive Affect*Time: Exp' cond' 3 period	0.219	0.015, 0.424	0.036				**0.123***	**0.034, 0.213**	**0.007**									
Trait worry* Time: recovery period	-0.054	-0.154, 0.047	0.293	-0.018	-0.230, 0.194	0.866	-0.024	-0.078, 0.029	0.372	-0.021	-0.057, 0.015	0.257	-0.044	-0.098, 0.010	0.109	0.010	-0.026, 0.046	0.574
Negative Affect* Time: recovery period	**-0.202***	**-0.347, -0.057**	**0.007**	0.144	-0.162, 0.450	0.354	0.033	-0.044, 0.111	0.398	**0.095***	**0.043, 0.147**	**<0.001**	**0.196***	**0.118, 0.274**	**<0.001**	0.017	-0.035, 0.068	0.522
Positive Affect* Time: recovery period	-0.039	-0.226, 0.149	0.686	-0.354	-0.751, 0.043	0.080	-0.003	-0.104, 0.097	0.947	-0.034	-0.101, 0.034	0.326	-0.029	-0.130, 0.072	0.571	0.052	-0.015, 0.119	0.125
Trait worry* Time: relaxation period	0.075	0.017, 0.133	0.012	-0.040	-0.109, 0.028	0.245	-0.016	-0.036, 0.005	0.138	-0.011	-0.036, 0.013	0.360	0.001	-0.023, 0.026	0.934	-0.015	-0.031, 0.001	0.062
Negative Affect* Time: relaxation period	0.085	0.001, 0.169	0.047	0.090	-0.008, 0.189	0.071	0.030	<0.001, 0.060	0.048	**0.048***	**0.012, 0.084**	**0.008**	0.002	-0.034, 0.037	0.924	**0.033***	**0.010, 0.056**	**0.005**
Positive Affect* Time: relaxation period	-0.068	-0.177, 0.040	0.216	-0.103	-0.231, 0.024	0.112	-0.037	-0.076, 0.001	0.058	0.042	-0.004, 0.088	0.074	0.028	-0.018, 0.074	0.235	-0.018	-0.048, 0.012	0.231

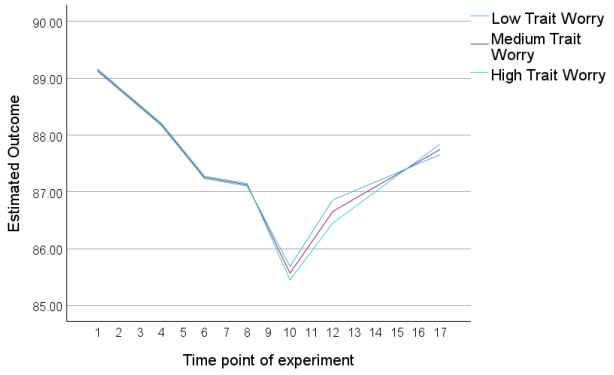
N1 = 3213 observations from N2 = 189 subjects* $p < 0.05$, having Bonferroni-corrected for testing multiple (six) related outcomes (hence p needs to be $< 0.05/6 = 0.0083$)

Variable 'Time: Pre-experiment period' coded 0, 1, 2, 3, 4 for time points 1-5 of the study respectively and 0 otherwise; 'Time: Experiment condition 1 period' coded 1, 2 for time points 6-7 of the study respectively and 0 otherwise; 'Time: Experiment condition 2 period' coded 1, 2 for time points 8-9 of the study respectively and 0 otherwise; 'Time: Experiment condition 3 period' coded 1, 2 for time points 10-11 of the study respectively and 0 otherwise; 'Time: Recovery period' coded 1, 2 for time points 12-13 of the study respectively and 0 otherwise; 'Time: Relaxation period' coded 1, 2, 3, 4 for time points 14-17 of the study respectively and 0 otherwise.

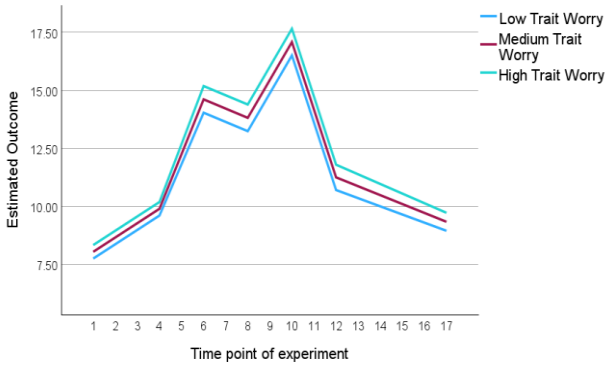
Figure 3

Estimated change over the study period in each outcome as per model 7, by worry, with all other covariates held at their sample mean.

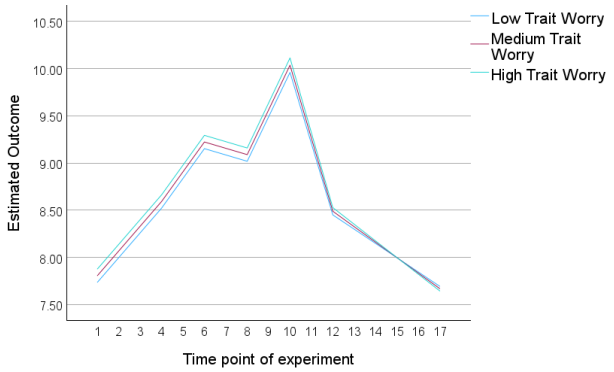
1. Outcome: Skin Temperature (Fahrenheit)



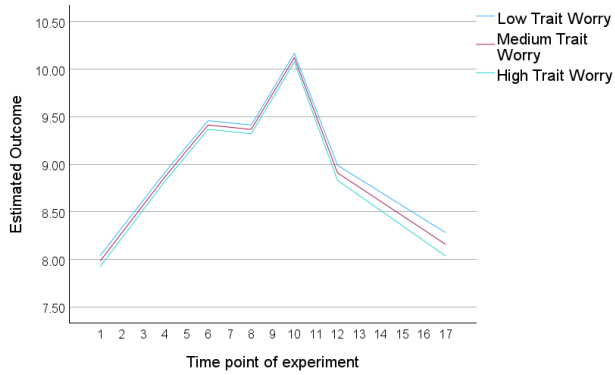
2. Outcome: Face Muscle Tension (EMG) (µV)



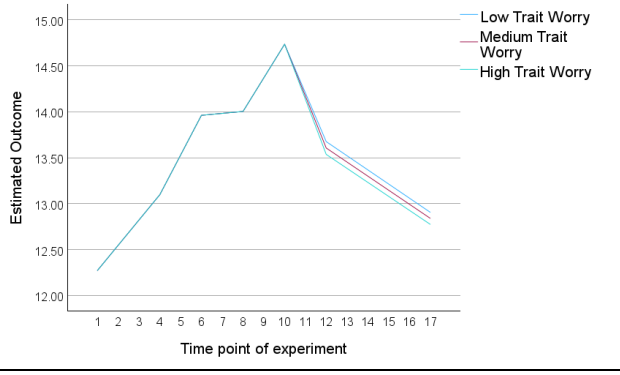
3. Outcome: Heart Rate (BPM/10)



4. Outcome: Blood Pressure Diastolic (mmHG/10)



5. Outcome: Blood Pressure Systolic(mmHG/10)



6. Outcome: Respiratory rate (breaths per min/10)

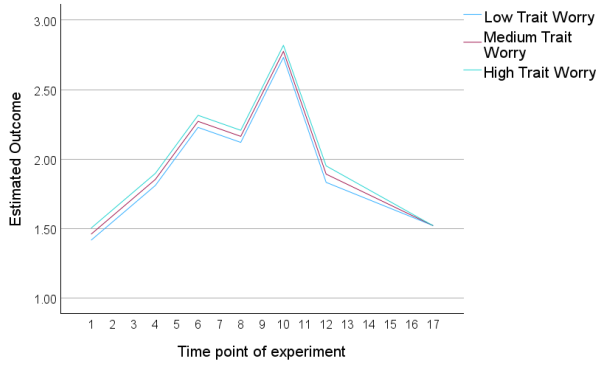


Figure 4

Estimated change over the study period in each outcome as per model 7, by negative affect, with all other covariates held at their sample mean.

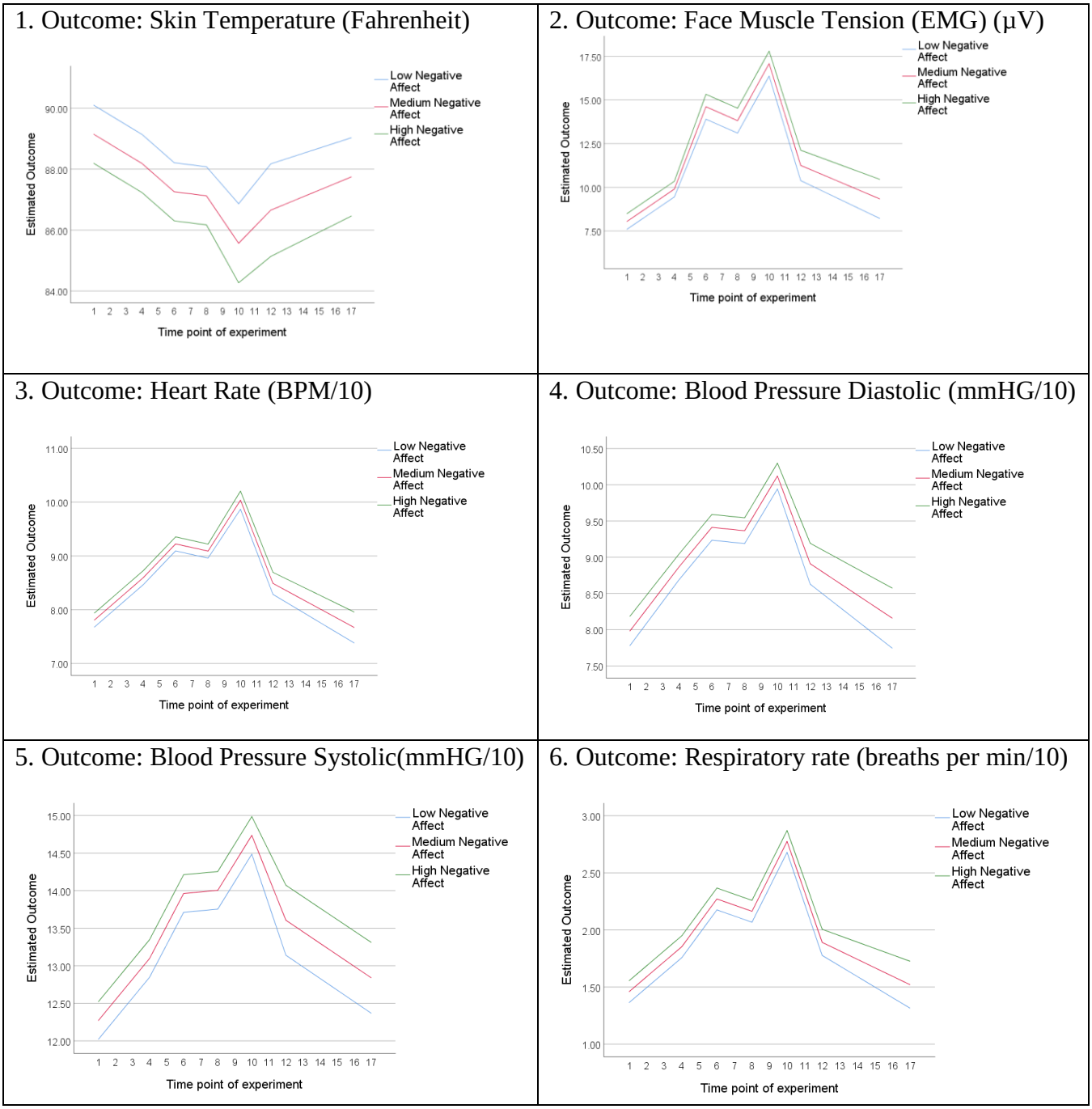
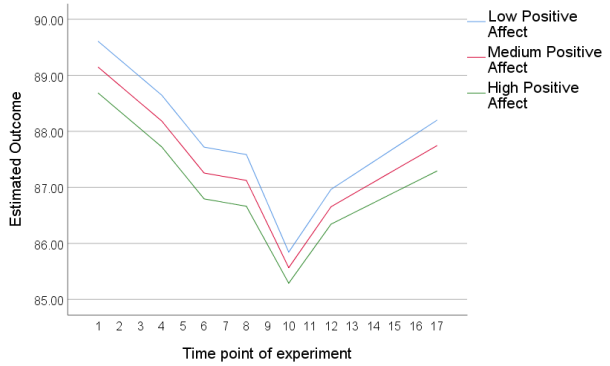


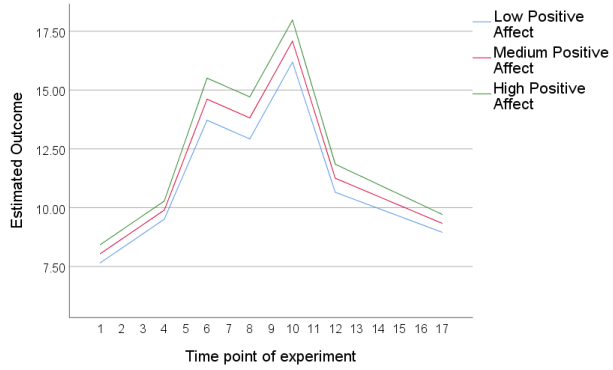
Figure 5

Estimated change over the study period in each outcome as per model 7, by positive affect, with all other covariates held at their sample mean.

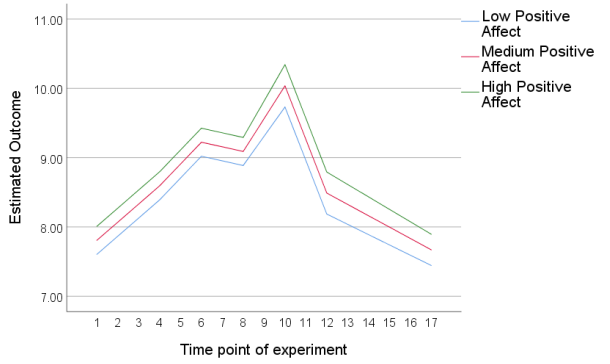
1. Outcome: Skin Temperature (Fahrenheit)



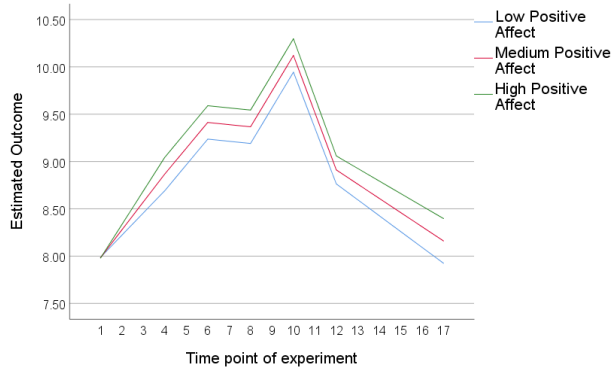
2. Outcome: Face Muscle Tension (EMG) (µV)



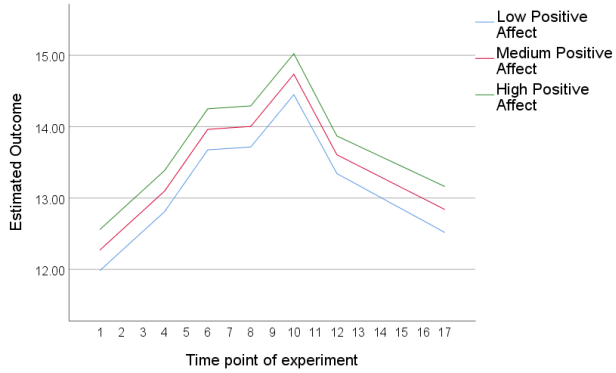
3. Outcome: Heart Rate (BPM/10)



4. Outcome: Blood Pressure Diastolic (mmHG/10)



5. Outcome: Blood Pressure Systolic(mmHG/10)



6. Outcome: Respiratory rate (breaths per min/10)