MALLEABILITY BELIEFS OF ANXIETY: IMPACT ON TREATMENT PREFERENCES AND EMOTION REGUALTION

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ABSTRACT

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Beliefs about how much people can change their attributes influence cognitive, affective, and motivational responses to challenging situations. Most research on this topic has focused on academic contexts, but newer work suggests these types of beliefs may relate to clinical phenomena as well. Specifically, recent studies show that the belief that anxiety is changeable (the growth mindset of anxiety) relates to a preference for individual therapy versus medication and greater engagement in psychosocial treatments for anxiety disorders. A working model proposes that the growth mindset of anxiety promotes a motivation to engage in effortful strategies to experience and learn from uncomfortable emotions. However, all research to date on this construct has been correlational in nature. Therefore, the present investigation sought to examine the impact of a brief experimental manipulation promoting the growth mindset of anxiety. Study 1 was an online study and examined the causal impact of this belief on treatment preferences (therapy vs. medication), willingness to initiate in future treatment, and anticipated efficacy of such treatments. The intervention successfully increased growth mindset of anxiety endorsement, increased participants' willingness to initiate future treatment, and increased expected efficacy at a trend level. It did not have an effect on treatment preference. Study 2 examined the impact of the mindset manipulation on electrophysiological correlates of emotion regulation. Baseline differences (before the intervention) between groups made comparisons difficult, but there was some evidence that those in the mindset condition were less reactive to

negative stimuli overall after the intervention. Overall, the two studies suggest that the anxiety mindset can be induced and may have implications for treatment motivation and emotion regulation processes that deserve further study.

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KEY TO ABBREVIATIONS

EEG Electroencephalogram

ERP Event-Related Potential

SPN Stimulus-Preceding Negativity

LPP Late Positive Potential

INTRODUCTION

Anxiety and its disorders constitute the most common mental health problems worldwide (Baxter, Scott, Vos, & Whiteford, 2013) and contribute to significant individual and societal costs. Although treatments for anxiety disorders are often touted as some of the best of all psychosocial interventions (Barlow, 2002), two key problems prevent most individuals getting the help they need. First, nearly 90% of individuals with anxiety disorders do not seek help for their anxiety, and among those who do, there is over a 15-year delay between age of onset and initial treatment seeking behavior (Johnson & Coles, 2013 Wang et al., 2007). Second, for those who do seek treatment, nearly one in five terminates treatment prematurely and one in three treatment completers are considered "non-responders" in that they do not fall below clinical thresholds by the end of treatment (Taylor, Abramowitz, & McKay, 2012).

Certainly these are multifaceted problems involving structural and political factors, but, at the individual level, patient *motivation* is considered a key predictor for both treatment seeking and achieving successful treatment outcomes. Broadly defined, motivation is a "process whereby goal-directed activity is instigated and sustained", and most motivational constructs map onto one of two questions: "Can I do this?" and "Why do I want to do this?" (Schunk, Meece, & Pintrich, 2014, p. 5). Previous attempts to increase patients' motivation to change, such as motivational interviewing, have produced mixed findings (see Romano & Peters, 2015 for a meta-analysis). I contend that a possible reason for these mixed and modest results is that previous studies have overlooked a crucial precursor to motivation for change: the belief that change is possible in the first place.

Thus, the current investigation focuses on a set of beliefs – called mindsets – about the changeability of self-attributes, that can influence subsequent motivations, cognitions, emotions,

and behaviors across a variety of contexts, but have been under-examined in clinical psychological research (Burnette, O'Boyle, VanEpps, Pollack, & Finkel, 2013; Dweck, 1999). Two studies evaluated the impact of a novel manipulation that induces the belief that anxiety is changeable – i.e., the growth mindset about anxiety. In keeping with modern definitions of motivation (Schunk et al., 2014), the studies examined distinct correlates of motivation to engage in effortful behaviors aimed at improving anxiety, including self-reported willingness to initiate treatment and expected efficacy of treatment (i.e., therapy; Study 1) as well as greater success in instigating and sustaining effortful emotion regulation strategies, assessed with electroencephalogram (Study 2). Before detailing the studies further, a brief review of the work on mindsets and motivation is provided.

Lay Theories and Mindsets

Years of research in social and personality psychology have uncovered so-called "lay theories", which refer to how individuals create meaning from their experiences (Haslam, 2017; Kelly, 1955; Plaks, Levy, & Dweck, 2009). These theories, which are often implicit or unconscious, are thought to "operate in the background" and serve to facilitate the understanding of complex information (Cimpian & Salomon, 2014; Plaks et al., 2009). These and other meaning-making structures can be considered to be distal from many other psychological phenomena. That is, there are often many processes thought to moderate and mediate relations between lay theories and more observable behaviors (Burnette et al., 2013).

There are important cognitive, developmental, cultural, and social-interactive processes that contribute to the adoption of lay theories (Haslam, 2017). Research suggests that young children may adopt one set of lay theories – essentialist theories – in which they attribute observable behaviors and appearances of an animal to an underlying "essence" (Cimpian &

Salomon, 2014). For example, "a cat's underlying essence causes it to have whiskers, soft fur, sharp claws, and the tendency to purr when satisfied. Essence constrains visible characteristics but is not defined by them. There may be changes in the observable characteristics of members of a category (e.g., hairless cats) but these do not necessarily imply changes in the essence of these members" (Dar-Nimrod & Heine, 2011, p. 801). Metaphysically speaking, few categories actually have one underlying essence that defines them (Haslam, 2017); nonetheless, many people hold these implicit lay theories assuming the existence of such underlying essences.

Essentialist thinking can relate to assumptions of *immutability* of traits. For instance, when individuals apply essentialist thinking to groups of people, the perceived homogeneity and immutability of the members of the group is increased (Dar-Nimrod & Heine, 2011). That is, if members of a group are thought to share an underlying essence, they are assumed to have similar traits that cannot change because of this underlying essence. Essentialist thinking can be reinforced by certain communications between parents and their children. Parents who use more generic statements such as "girls are kind" with their children may reinforce ideas of categorical representations that have inherent properties (Haslam, 2017). The adoption of essentialist lay theories leads individuals to attribute behaviors to underlying and immutable traits, rather than historical, contextual, and situational forces at play. This tendency to infer dispositional characteristics based upon observable behaviors is similar to other social psychological constructs. For instance, in his description of the fundamental attribution error, Ross (1977) shows how individuals will attribute subjects' behavior in the Milgram experiments to personal dispositions rather than the "potency of situational pressures and constraints acting upon all subjects" (p. 184; see also Bierbrauer, 1973). Indeed, the lay theories described next were created, in part, out of attribution theory.

One set of lay theories related to essentialist beliefs is *mindsets.* Mindsets refer to beliefs about the malleability of self-attributes, such as intelligence, personality, or anxiety. These beliefs lie along a continuum of malleability. On one end, the fixed mindset holds that these attributes are unchangeable and are largely determined by innate factors; on the other, the growth mindset holds that attributes can be changed with learning and effort (Dweck, 1999). Mindsets can be about any attribute – athletic ability, personality, music ability, and so on – and people can hold different mindsets simultaneously. For instance, a person can believe that intelligence is quite malleable but that personality is unchangeable (Dweck, Chiu, & Hong, 1995). Like most motivational constructs, mindsets are domain-specific, meaning that a mindset about math ability is more related to math grades than to social studies grades, for example (Dweck et al., 1995; Romero, Master, Paunesku, Dweck, & Gross, 2014; Schroder, Dawood, Yalch, Donnellan, & Moser, 2016).

In general, mindsets have been related to multiple aspects of motivation, including goal adoption, attributions, and effort expenditure (for reviews, see Burnette et al., 2013; Dweck, 1986, 1996, 1999). Whereas individuals with a growth mindset[1] tend to attribute performance to unstable and controllable factors like effort, strategy, and mood, those with a fixed mindset tend to attribute performance to stable and internal attributes like intelligence. Because of these attributional styles, mindsets are also thought to promote distinct motivations for approaching, engaging, and reacting to various performance situations (Dweck & Leggett, 1988). Those with more of a growth mindset tend to prefer challenging tasks in order to learn, develop mastery, and strengthen ability (i.e., they adopt learning goals). Growth-minded individuals tend to also

[1] I refer to "fixed-minded" and "growth-minded" people here for simplicity; in reality, mindsets lie along a continuum. It may be more accurate to say "those with more or less of a growth mindset".

believe that effortful behaviors will lead to success (i.e., have higher self-efficacy beliefs; Diseth, Meland,& Breidablik, 2014) . When mistakes occur, growth-minded individuals tend to redouble their efforts, consider alternative strategies, and ultimately strive to improve their learning. In contrast, those with a fixed mindset often prefer easier tasks in order to demonstrate their presumably innate skills to others (i.e., they adopt performance goals). Challenging tasks quickly elicit anticipatory anxiety for those with a fixed mindset, because mistakes are threatening to their self-esteem. When mistakes occur, fixed-minded individuals experience a sense of helplessness, disengage from the task, and ironically commit more mistakes (e.g., Moser, Schroder, Heeter, Moran, & Lee, 2011; Mueller & Dweck, 1998).

Mindsets and Mental Health

The vast majority of mindset research comes from social, personality, and educational psychology, which shows that mindsets have impacts on a range of academic behaviors, resilience, and adjustment (Blackwell, Trzesniewski, & Dweck, 2007). However, accumulating research indicates that mindsets are also applicable to mental health. For instance, just as the growth mindset of intelligence promotes the engagement of effortful strategies during a challenging academic task (Hong, Chiu, Dweck, Lin, & Wan, 1999), growth mindsets of emotion relate to adaptive and effortful emotion regulation strategies such as cognitive reappraisal (De Castella et al., 2013; Kneeland, Dovidio, Joormann, & Clark, 2016; Tamir et al., 2007; Schroder, Dawood, Yalch, Donnellan, & Moser, 2015).

The most recent work suggests that mindsets of *anxiety*, in particular, are most relevant to mental health-related motivation. Like the emotion mindset, the anxiety mindset also predicts the use of cognitive reappraisal (Schroder et al., 2015). Two additional lines of evidence indicate the anxiety mindset is particularly important for mental health-related motivation. First, the growth

mindset of anxiety corresponds to hypothetical preferences for psychological treatment (Schroder et al., 2015). Individuals were asked if they themselves suffer from mental health problems or to imagine that they suffer from a mental health problem and to choose between individual therapy (presumably the more effortful option) and medication (the less effortful option). Those who chose the therapy option were significantly more growth-minded than those who opted for medication. Mindsets of intelligence, personality, and emotion were not as reliably related to the hypothetical treatment choice (Schroder et al., 2015). Second, the growth mindset of anxiety –and a related construct called "anxiety change expectancy" - is related to adaptive engagement in psychological treatments, including increased homework compliance and enhanced treatment outcomes (De Castella et al., 2015; Westra, Dozois, & Marcus, 2007; Valentiner et al., 2013). Together, these findings indicate that growth mindsets of anxiety may promote motivation to engage in effortful behaviors to gain a sense of control and mastery over one's anxiety, much like mindsets in academic domains. That is, this mindset may help promote the very motivation needed for individuals to seek out and stay with mental health treatments.

Figure 1 depicts a working model of how growth and fixed mindsets of anxiety lead to mastery or avoidance strategies, and finally psychological health and distress, respectively. Those with more of a growth mindset of anxiety are hypothesized to use more adaptive emotion-regulation strategies in the face of anxiety, and over time have better psychological health. This, in turn, reinforces the idea that anxiety is changeable. In contrast, those with a fixed mindset of anxiety use less helpful or maladaptive ways of managing anxiety, which leads to greater psychological distress. This distress, in turn, reinforces the belief that anxiety is fixed in nature.

Despite this promising accumulation of evidence for the advantages of the growth mindset of anxiety, all of the evidence to date has been correlational, so conclusions regarding

directional causality are inherently limited. One logical next step in this line of research would be to experimentally induce the growth mindset of anxiety and examine its impacts on mindset endorsement and motivation. This was the primary aim of the current study.

Mindset Interventions

Given the potential consequences of individual differences in mindsets, with growth mindsets generally relating to more favorable outcomes in some domains, a plethora of social psychological studies have examined how this belief can be induced. Almost all of these studies have examined mindset interventions in the intelligence or personality domain, and most have been conducted in academic settings. This research has exploded in recent years. Here, a brief review of these intervention studies is provided in order to contextualize this research area and set the stage for the present mindset intervention in the anxiety domain.

First, it is important to emphasize that mindset interventions "are not magic" – they "are not inputs that go into a black box and automatically yield positive results" (Yeager & Walton, 2011, p. 293). Instead, they are entirely dependent upon the context in which they are implemented. For instance, academic mindset interventions are dependent upon the quality of the teaching, the academic content, and the skills offered to students. Without these basic structures in place, academic mindset interventions will likely have very little effect. These interventions are not meant to replace conventional teaching strategies; they are intended to complement them.

The earliest mindset induction research relied on small laboratory studies in which participants read brief "scientific articles" describing intelligence as primarily genetically influenced or primarily due to a challenging environment (Bergen, 1991; Hong et al., 1999). Theorizing suggests that inducing genetic beliefs about intelligence would lead to a fixed mindset, whereas promoting the notion that environmental influences shape intelligence would

lead to a growth mindset. One notable aspect of this research is that not all mindset inductions actually measure post-intervention mindset beliefs (i.e., the endorsement of a fixed or growth mindset). Instead, the outcome variables are behaviors or attitudes that would be consistent with a fixed or a growth mindset. For instance, Hong et al (1999) had students read a scientific article and then perform a challenging ability task. Participants were then either given satisfactory feedback indicating they had scored in the 66th percentile or unsatisfactory feedback indicating they had scored in the 20th percentile. Hong et al (1999) found that students who received the growth mindset article were more likely to indicate an interest in taking a remedial tutorial designed to improve skill in the task, even if they had been given the unsatisfactory feedback. These results are consistent with theory that suggests the growth mindset promotes effortful behaviors in the face of setbacks (Dweck, 1999). Experimental laboratory studies that do measure mindsets have found that reading the articles does, in fact, change mindset endorsement (Schroder, Moran, Donnellan, & Moser, 2014).

Another way to induce fixed or growth mindsets is by praise of performance. In a highly cited study, Mueller & Dweck (1998) provided children who were performing an increasingly difficult task with *person* or *process* praise. Person praise (e.g., "You got that right, you must be smart") is thought to promote a fixed mindset because it praises an inner ability. Process praise (e.g., "You got that right, you must have tried hard") is thought to promote a growth mindset because the emphasis is on transient effort. This was another study that did not measure post-intervention mindsets. Instead, the outcome variable was how many problems the children solved correctly after encountering failures as the task became more difficult. Children who received process praise did better after failure than children who received person praise. Similar findings have been provided from other groups (e.g., Brummelman et al., 2014). However, one recent set

of studies failed to replicate the original Mueller and Dweck findings (Li & Bates, 2017). In a sample of 624 10-12 year olds, Li and Bates (2017) were unable to find significant effects of the growth mindset manipulation of effort praise. In fact, in one of their studies, growth mindset endorsement was negatively correlated with adaptive response to challenge. Li and Bates also did not measure mindset endorsement after the intervention.

Another class of mindset interventions uses in-person workshops to promote the growth mindset (Blackwell, Trzesniewski, & Dweck, 2007; Donohoe, Topping, & Hanna, 2012; Orosz et al., 2017). In one of the most highly cited mindset intervention studies (Blackwell et al., 2007), 91 middle school students transitioning to seventh grade with declining math grades either attended a mindset workshop or a control study skills workshop. The workshops were eight weekly 25-minute sessions. The two conditions were identical except for three sessions. The mindset workshops consisted of information about brain plasticity and activities and reading assignments inducing the growth mindset of intelligence. All participants' grades were declining prior to the intervention, and the grades of the students in the mindset condition – but not in the control condition - stopped declining after the intervention. Growth mindset endorsement and positive attributions about effort were also higher for the mindset condition three weeks after the intervention.

In an attempt to replicate and extend the findings of Blackwell et al (2007), Orosz et al. (2017) had 55 10th grade students with high grade point averages engage in a mindset intervention consisting of five weekly workshops. Teachers who were trained on the material carried out the workshops. Although there were small increases in growth mindset endorsement immediately after the intervention, these changes did not last at a later assessment at the end of

the semester. Moreover, there were no differences in grade point averages at the end of the semester between conditions.

A few key differences between the Blackwell and Orosz studies should be noted. First, whereas the Blackwell et al study had mindset experts deliver the content, the Orosz study had teachers carry out the interventions (i.e., a "train the trainer" study). Certainly this was meant to increase generalizability, but the effects could have been attenuated. Second, the samples were entirely different between studies. In the Blackwell study, the sample was composed of low-achieving inner-city middle school students struggling with math; the Orosz sample consisted of high-achieving high school students. Mindset theory is predicated on the idea that individuals with some disadvantage (e.g., historically underrepresented minority background, lower socioeconomic status, academically struggling) benefit more from mindset interventions (Broda et al., 2018; Claro et al., 2016; Dweck, 1999; Sisk, Burgoyne, Sun, Butler, & Macnamara, 2018; however, see Li & Bates, 2017). When viewed from this perspective, the data from the Orosz study demonstrate the boundary conditions of mindset interventions as predicted by theory.

One small study (N = 33 students ages 13-14) from Scotland used four 40-minute workshop sessions along with activities students completed at home to induce the growth mindset (Donohoe et al., 2012). Results showed initial gains in mindset from the intervention but these gains were not sustained three months later. Grade point averages were not assessed in this study and little information was provided on the demographic characteristics or achievement standing of the sample. One difference between this and the Blackwell study was the number of sessions – Blackwell et al used eight weekly sessions whereas this study used four. Although the total number of minutes of intervention time was similar (160 minutes in Donohue vs. 200 minutes in Blackwell), it may be the case that mindset workshops are most effective when they

are delivered across more weeks. Overall, the methods and results from workshop studies are mixed and require further study to understand long-term effects.

A final class of mindset interventions uses self-administered tutorials (either online or in-person) in one or two sessions to induce the growth mindset (e.g., Broda et al., 2018; Paunesku et al., 2015; Yeager et al., 2014; Yaeger, Walton, et al., 2016). Generally speaking, these interventions condense the materials from previous workshop studies (e.g., Blackwell et al., 2007) to an online platform with 45-60 minute sessions. These studies have examined a wide variety of outcomes – including grade point average, self-reported well being, depressive symptoms, and response to a social challenge (i.e., Cyberball). Online studies are particularly appealing because of their ease of dissemination (e.g., Paunesku et al., 2015; Yeager, Walton, et al., 2016).

Yeager et al (2014, Study 2) had ninth-graders who were struggling academically complete a one-time 25-minute self-administered growth mindset of personality intervention during the first few weeks of school. Students in the intervention condition were handed envelopes that contained neuroscience information about the malleability of the brain which can change behaviors, read quotes from past ninth graders about their experiences encountering peer conflicts, and then wrote about their own experiences. One to two days after the intervention, participants in the intervention condition reacted less negatively to a Cyberball exclusion paradigm compared to the control condition. Moreover, students in the mindset condition reported lower global stress eight months post-intervention. Finally, students in the mindset condition reported higher grades at the end of the academic year than students in the control condition.

In another study, high school seniors at a charter school were administered an online growth mindset of intelligence tutorial before going to college (Yeager, Walton, et al., 2016). The primary outcome assessed was full-time enrollment during the first semester of college, and secondary outcomes were utilization of academic support services, extracurricular group participation, and living on campus status. Although a social-belongingness intervention improved these outcomes, the mindset condition had no effects on any of these; in fact, students assigned to the growth mindset intervention had significantly lower outcomes compared to those assigned to the social belongingness condition. The authors suggested that the mindset intervention may have been redundant with mindset messages students already received at their high school, or that the growth mindset message was "represented as a private belief, not a reflection of their college's values" (Yeager, Walton, et al., 2016, p. E3343). Study 2 in the same paper compared mindset, social belongingness and control conditions in a much larger sample of undergraduate freshmen just before beginning college. In this intervention, the growth mindset was characterized as the ethos of the university. The authors combined growth mindset and social belongingness interventions in the presentation of results. This time, lay theory interventions lead to improved outcomes compared to controls, especially for those from a disadvantaged background. Grades were not assessed in either study.

A recent meta-analysis examined effects of trait mindsets and mindset interventions on academic outcomes (Sisk et al., in press). This study was very well powered to assess historic effects because it examined data from over 365,000 participants for trait mindsets and data from over 57,000 participants for intervention effects. Notably, this study only examined relations between mindsets and the more distal outcome of academic achievement - i.e., exam grades, course grades, average grades, standardized tests and laboratory measures of achievement. The

overall meta-analytic effect between mindset and academic achievement was significant yet very small (meta-analytic r = .10). This was nearly identical to that of an earlier meta-analysis by Burnette et al (2013, meta-analytic r = .095) that examined many more achievement domains and outcomes. Sisk et al (in press) suggested researchers and policy-makers caution against implementing mindset interventions in academic settings given these small effect sizes. However, consistent with previous data and theory, Sisk et al (in press) also found suggestive evidence that mindset interventions may be more effective for at-risk populations.

In sum, a variety of mindset interventions have been conducted over the years consisting of brief scientific articles, variations of praise, weekly workshops, and self-administered tutorials. These studies have assessed a number of outcomes and not all studies examine post-intervention mindset endorsement. As can be seen above, the results of these studies have produced mixed results. The most comprehensive meta-analyses on mindsets and mindset interventions to date (Burnette et al., 2013; Sisk et al., in press) indicate that they are more related to proximal outcomes (e.g., goal adoption, learning strategy, effortful behaviors) than distal outcomes (grade point average). Moreover, mindset interventions tend to produce small effect sizes. Yet, small effects do not indicate that interventions are not helpful for the population at large (see Meyer et al., 2001). Critical to the present study, the most recent studies (e.g., Paunesku et al., 2015; Yeager, Walton, et al., 2016) are suggestive that online tutorials teaching the growth mindset may help improve motivational tendencies to persist in school (i.e., measured with full-time college enrollment). This is important because of the current study's focus on motivational tendencies related to anxiety.

Also critical to the present study is the ease of dissemination that online tutorials have (Paunesku et al., 2015), which is much needed for individuals struggling with anxiety and related

disorders. If a scalable, theory-driven intervention could be delivered, it may help promote motivation and willingness to seek and engage in effective therapies. Despite the work demonstrating mindsets can be induced in other domains, no studies have induced a growth mindset of anxiety. Establishing causality for mindsets of anxiety would be important for a few reasons. It would inform theories of the nature of the relations between beliefs and distress, emotion regulation and motivation, inform extant treatment approaches, and inspire novel treatment avenues. For these reasons, the current studies deliberately recruited participants with clinically high levels of anxiety.

The Current Studies

The current investigation sought to advance the understanding of the growth mindset of anxiety by evaluating the impact of a novel anxiety mindset induction on distinct correlates of motivation. The intervention, described in detail below, is entirely self-administered and can be completed in less than 30 minutes. Both studies assessed the effect of the intervention relative to a carefully constructed control condition that also focused on anxiety, but that excludes the crucial content that anxiety is malleable. In order to evaluate the intervention's impact among those who may need it the most, both studies recruited individuals who have clinically-elevated anxiety symptoms who have not previously sought treatment. Because some previous research indicates that mindset interventions are more effective for individuals with some noticeable disadvantage (e.g., lower socioeconomic status, impoverished background, or academic struggles; Claro et al., 2016), highly anxious participants were recruited for the current studies. Anxious individuals were also targeted because the long-term focus of this work is to develop interventions that are easily disseminated to individuals who may benefit the most (i.e., individuals struggling with anxiety). Study 1 investigated the mindset induction's impact on

hypothetical treatment choice (individual therapy vs. medication), anticipated efficacy of their chosen treatment, and their willingness to initiate treatment. Study 2 investigated how the procedure influences the implementation and continued engagement of emotion regulation using event-related brain potentials (ERPs) derived from the human electroencephalogram (EEG).

STUDY 1

In other domains, growth mindsets lead participants to engage in more effortful behavior in order to develop mastery, even if participants had experienced failure or setbacks (Dweck & Leggett, 1988; Hong et al., 1999; Mueller & Dweck, 1998; Schumann, Zaki, & Dweck, 2014). Study 1 (an online study) sought to examine if this finding extends to the anxiety mindset domain in clinical decision-making scenarios. Specifically, Study 1 provided an experimental test of our previous correlational findings that individuals with a growth mindset of anxiety prefer more effortful treatments (individual therapy) to alleviate anxiety, relative to those with more of a fixed mindset of anxiety (Schroder et al., 2015). After the intervention (or control condition), participants indicated whether they would rather choose individual therapy or medication if they were to seek treatment in the future. They were also asked to rate how willing they were to initiate their treatment of choice and how effective they believed that treatment would be in reducing their symptoms.

The growth mindset in other domains is correlated with higher efficacy beliefs and more positive attributions about effort (e.g., Diseth et a., 2014; Dweck, 1999; Hong et al., 1999). Based on previous research, it was hypothesized that 1) individuals in the growth mindset condition would endorse higher growth-mindset beliefs after the intervention than the control condition participants; 2) a higher proportion of individuals in the growth mindset condition would prefer therapy versus medication; 3) those in the growth condition would endorse greater willingness to initiate the treatment; and 4) those in the growth group would indicate a greater expected efficacy of their treatment choice.

Study 1 Method

Participants

A sample of 1,624 adults recruited from Amazon's Mechanical Turk (MTurk; Shaprio, Chandler, & Mueller, 2013) completed a consent form and the screening survey, which consisted of one item from the Penn State Worry Questionnaire (Meyer et al., 1990; Schroder, Clark, & Moser, in press) and a treatment history question. To be eligible for the main part of the study, participants needed to endorse a 4 or 5 on the PSWQ item, indicative of clinically problematic worry (Schroder et al., in press), and no history of treatment of any kind. Participants also completed the Generalized Anxiety Disorder -7 (GAD-7; Spitzer et al, 2006) as an ancillary measure of anxiety symptom severity. Eligible participants were then directed to a second consent form and completed the second part of the survey – which consisted of the anxiety mindset intervention or the psychoeducation control condition. A total of 286 participants (*M* age = 33.49, *SD* = 11.48, range 11-79; 74% female) completed this second part of the survey and constituted the analysis sample. Table 1 presents demographic variables for Study 1. A power analysis (with power = 0.80) considering the effect size (Cohen's *d* = 0.46) from previously published and unpublished data on hypothetical treatment choice indicated that a sample size of at least 152 (76 per group) was necessary. Thus, the sample of 286 participants exceeded the necessary sample size. Participants were compensated $0.10 for the screening survey and $3.00 for completing the tutorial. It should be noted that participants were not randomly assigned to the conditions described below; rather, they either completed the anxiety mindset intervention or the control condition, as these conditions were posted online at separate times. Titles and advertisements of each of the conditions were identical, so there were no concerns of a self-selection bias. No participants completed both conditions.

Procedure

Anxiety Mindset Intervention. The anxiety mindset intervention was modeled closely after personality and intelligence mindset interventions that have been implemented in previous studies (e.g., Miu & Yeager, 2015). In fact, each of the following procedures has been systematically tested in past experimental work in other mindset domains and is considered important to promoting the growth mindset message (Blackwell et al., 2007; Chiu, Hong, & Dweck, 1997; Paunesku et al., 2015; Yeager et al., 2011, 2013, 2014, 2016). The key intervention message is that anxiety is changeable and that anxious people are not necessarily stuck being anxious. Participants are taught that people can strengthen the brain's "stress coping muscle", which "lives" in the prefrontal cortex, by confronting the situations that make them anxious and by learning that anxiety decreases over time with practice. Participants are provided with information about the prefrontal cortex and amygdala, and how anxiety worsens when the prefrontal cortex is unable to override reactivity from the amygdala. Examples of how the brain's response to anxiety changes with practice and effort were given. Specific techniques to reduce anxiety were described. The concept of the growth mindset was then described along with examples of putting this mindset to use. Quotes from celebrities who have recovered from anxiety disorders and from past individuals who had taken the growth mindset tutorial (these quotes were generated by the experimenter) were included to reinforce the message that anxiety is changeable. Participants were asked to write a paragraph explaining the growth mindset of anxiety for high school students struggling with anxiety at the end of the intervention. They were also asked to write one or two steps they could take in order to "get on the growth mindset path". Such writing prompts are thought to invoke a "saying is believing" effect (people believe what

they say to others), and are a staple of mindset interventions (Aronson, Fried, & Good, 2002; Miu & Yeager, 2015; Yeager et al., 2014).

Control Condition. Although some studies have compared growth-mindset with fixed-mindset inductions (e.g., Chiu et al., 1997; Hong et al., 1999; Schumann et al., 2014), inducing the fixed mindset of anxiety may be considered unethical. For this reason, the control condition was carefully crafted to be as similar to the intervention condition as possible (i.e., about anxiety), without the key message that anxiety is changeable. Similarities between control and intervention conditions include a focus on anxiety, neuroscience findings relevant to anxiety, writing prompts, celebrity quotes, and quotes from other individuals with anxiety problems. The primary message in the control condition is that anxiety is *common*. Descriptive information about anxiety disorders (symptoms, prevalence rates, gender ratios, etc.) from the NIMH Anxiety Disorders webpages was provided. This information is accessible to the public and commonly provided in initial therapy sessions of manualized treatment protocols (e.g., Foa, Hembree, & Rothbaum, 2007). Simply knowing that anxiety is a common condition and normalizing the experience is often thought to be helpful. Neuroscience findings are also presented, highlighting the fact that anxiety disorders can impact the brain. Quotes from celebrities and other individuals with anxiety reinforce the message that anxiety is common. As in the intervention condition, participants are prompted to write how anxiety has impacted their life and to write to a high school student struggling with anxiety about what they learned from the tutorial. After completing the tutorial, participants completed the following questionnaires.

Self-Report Measures

Implicit Theories of Anxiety (TOA [anxiety mindset scale]; Schroder et al., 2015). The anxiety mindset scale is a four-item measure of the anxiety mindset. Items (e.g., "You have a certain amount of anxiety and there is really not much you can do to change it.") are rated on a scale of 1 (Strongly Disagree) to 6 (Strongly Agree). Items are reverse-coded such that higher scores indicate more endorsement of the growth mindset.

Generalized Anxiety Disorder – 7 (GAD-7; Spitzer et al, 2006). The GAD-7 is a seven-item scale of GAD symptom severity (e.g., "feeling nervous, anxious, or on edge") in which items are scored on a 0 "not at all" to 3 "nearly every day" scale regarding the past two weeks.

Hypothetical Treatment Choice (Cochran, Pruitt, Fukuda, Zoellner, & Feeny, 2008). Hypothetical treatment choice was measured with one item: "If you struggle or if you were to struggle with mental health problems (e.g., anxiety, depression) and had a choice between individual therapy or medication to help you with your mental health problems, which would you choose?"

Treatment Expectations and Willingness to Engage. Two items modified from the Credibility and Expectancy Questionnaire (Devilly & Borkovec, 2000) were used: "On a scale from 0% to 100%, how much improvement in your symptoms do you think will occur if you were to partake in the treatment you chose?". On this scale, a response of 1 corresponds to 0% and a response of 11 corresponds to 100%, so the possible range was from 1-11. Willingness to engage in treatment was measured with the item, "On a scale from 0 (Not at all likely) to 5 (Extremely likely), how likely are you to begin this treatment option in the next year?".

At the end of the tutorials, to check for similar levels of interest and learning potential between conditions, two questions were given at the end of the surveys: "For you personally,

how interesting was the activity you completed today?" and "How much do you feel that you learned from the activity you completed today?" The items were rated on 1 ("Not interesting at all" and "Nothing at all", respectively) to 5 ("Extremely interesting" and "An extreme amount", respectively) response scale.

Analyses and Anticipated Results

Analyses consisted of one-way univariate ANOVAs, t-tests, and chi-square tests. The four primary hypotheses were that individuals in the growth mindset condition would 1) endorse more of a growth mindset of anxiety; 2) be more likely to prefer individual therapy vs. medication; 3) rate their treatment of choice as more likely to improve symptoms; and 4) indicate higher likelihood of beginning their treatment of choice in the next year.

Study 1 Results

Manipulation Checks

Duration of Intervention. On average, participants spent 25.68 minutes (*SD* = 15.16; range 3.15 to 123.08) on the control tutorial and spent 27.21 minutes (*SD* = 13.58; range 7.98 to 95.60) on the anxiety mindset tutorial. There was no significant difference between conditions in duration of intervention $t(284) = 0.89$, $p = .37$). Extreme values noted in the range were likely caused by an error in recording timing. Results were unchanged excluding extreme values and when only participants with duration values of 15 minutes or more were included in analyses. Analyses were conducted with and without duration of the intervention as a covariate presented below.

Interest and Learning Ratings. The groups did not differ significantly in terms of interest (Mindset $M = 3.93$, $SD = 1.02$; Control $M = 4.03$, $SD = 0.94$, $t(283) = 0.86$, $p = .39$) or the amount they felt they learned (Mindset $M = 3.77$, $SD = 0.94$, Control $M = 3.66$, $SD = 0.99$,

$t(284) = 0.96$, $p = .34$); participants indicated that the information from both conditions was interesting and novel.

Self-Report Results

Table 2 presents descriptive statistics, internal reliabilities, and bivariate correlations for all Study 1 variables across the entire sample. Table 3 presents these variables separated by experimental condition.

Mindsets and Anxiety. Univariate ANOVAs with Condition (Mindset vs. Control) as the fixed factor were used to evaluate baseline differences. There were no significant differences in baseline self-report measures (see Table 3 for group means and standard deviations), including prescreen trait worry (PSWQ item 15, $F(1, 284) = 0.50$, $p = .48$, $\eta^2_p = .002$) or prescreen growth mindset endorsement (TOA; $F(1, 284) = 1.87$, $p = .17$, $\eta^2_p = .007$). However, after the intervention, the Mindset group endorsed significantly higher TOA scores than the Control group, corresponding to higher endorsement of the growth mindset ($F(1, 284) = 37.40$, $p < .001$, $\eta^2_p = .12$, $d = 0.72$).

To evaluate change in GAD-7 symptoms before vs. after the intervention, a 2 (Time: Pre vs Post Intervention) x 2 Condition (Mindset vs. Control) repeated-measures ANOVA was carried out. The main effect of Time was not significant ($F(1, 284) = 0.04$, $p = .84$, $\eta^2_p = .0001$). However, the interaction between Time and Condition was significant ($F(1, 284) = 6.78$, $p = .010$, $\eta^2_p = .023$). Follow-up t-tests indicated that the Mindset condition had a larger decrease in GAD-7 symptoms from pre to post (*M change* = -0.27, *SD* = 1.78, $t(148) = -1.93$, $p = .06$) compared to the Control condition (*M* change = +0.32, *SD* = 2.00, $t(136) = +1.77$, $p = .08$; difference in difference between conditions: $t(284) = 2.60$, $p = .01$, $d = 0.16$). See Table 5 for descriptive statistics.

Hypothetical Treatment Choice, Expectations, and Willingness to Engage. For the hypothetical treatment choice item, 82% (111 of 136) of participants in the Mindset group chose therapy (vs. 18% who chose medication), compared to 77% (114 of 149) of participants in the Control condition. Although the percentage was higher in the Mindset group, the chi-square test was not statistically significant, $X^2 = 1.12$, $df = 1$, $p = .18$.

Univariate ANOVAs with Condition (Mindset vs. Control) as the fixed factor were used to evaluate post-intervention differences. As predicted, those in the Mindset group endorsed greater expected improvement with their chosen treatment compared to those in the control condition (see Table 3), and this difference approached statistical significance ($F(1, 284) = 3.70$, $p = .055$, $\eta^2_p = .013$, $d = 0.23$). Individuals in the Mindset condition also indicated they would be more likely to begin treatment in the next year compared to those in the Control condition, and this difference reached statistical significance ($F(1, 284) = 3.94$, $p = .048$, $\eta^2_p = .014$, $d = 0.23$).

Univariate ANOVAs with Condition as a fixed factor and key outcome variables as dependent variables were conducted with Duration of Intervention included as a covariate. For post-intervention TOA scores, the main effect of Condition was still significant ($F(1, 285) = 36.45$, $p < .001$, $\eta^2_p = .114$). For expected improvement, the main effect of Condition approached statistical significance ($F(1, 285) = 3.60$, $p = .059$, $\eta^2_p = .013$), as it had without the covariate ($p = .055$). For treatment likelihood, which had reached statistical significance without the covariate ($p = .048$), the main effect of Condition also approached statistical significance ($F(1, 285) = 3.86$, $p = .051$, $\eta^2_p = .013$). Thus, including duration of intervention as a covariate did not change main study results in terms of effect size, although the treatment likelihood test did not reach statistical significance after controlling for duration.

Exploratory Moderation Analyses

An exploratory moderation analysis was conducted to assess whether the intervention had a differential impact for higher vs. lower anxious participants. The sample was selected for high anxiety so the range was somewhat restricted. Hayes' (2013) PROCESS macro was used in these analyses in which Condition (Mindset vs. Control) was used as the independent variable, and pretreatment GAD-7 score was used as the moderator variable. GAD-7 scores were used because of the higher variability compared to the PSWQ prescreening item (which only had responses of 4 or 5, by design). Moderation would be suggested with a significant Condition x GAD-7 interaction. Four dependent variables from the post-intervention assessment were examined: anxiety mindset (TOA), hypothetical treatment choice (0 = medication, 1 = therapy), treatment expectancies, and treatment likelihood.

For TOA scores, the main effect of Condition was still significant ($b = 0.94$, $p < .001$) and the interaction between Condition and GAD-7 scores was not ($b = .03$, $p = .31$). For hypothetical treatment choice, the main effect of Condition was not significant ($b = .32$, $p = .28$) and neither was the interaction term ($b = -.02$, $p = .75$). In terms of expected improvement, the main effect of Condition was marginally significant, as above ($b = .47$, $p = .06$), and the interaction term was not significant ($b = -.06$, $p = .18$). Finally, for treatment likelihood, the main effect of Condition was significant ($b = .41$, $p = .04$) and the interaction term was not significant ($b = -.01$, $p = .89$). In sum, this exploratory analysis revealed that primary outcomes related to the intervention were not moderated by anxiety severity.

Study 1 Discussion

Study 1 found, for the first time, that an intervention designed to induce the growth mindset of anxiety lead to greater endorsement of this belief compared to a Control condition.

The intervention, which on average took less than 30 minutes to complete, also lead to greater reported likelihood of beginning treatment in the next year, and higher expectancies for successful treatment at a trend level (p = .055). The intervention did not relate to preference for therapy vs. medication, as hypothesized. Together, findings of this study suggest that the anxiety mindset intervention may improve motivation for high-anxious individuals who have never had treatment.

STUDY 2

Whereas Study 1 examined the impact of the anxiety mindset manipulation on hypothetical (i.e., imagined) treatment choices, willingness, and expectancy, Study 2 was designed to assess the intervention's impact on the effectiveness of instigating and sustaining an effortful emotion regulation strategy. Difficulties regulating emotions are evident among those with anxiety disorders (Mennin, Heimberg, Turk, & Fresco, 2002), and are targeted in many contemporary therapies (Clark & Beck, 2010). Whether or not someone engages in a particular emotion regulation strategy depends on a number of motivational factors, including perceived efficacy to use that strategy (Bigman, Mauss, Gross, & Tamir, 2016) and emotion-regulation goals (Tamir, 2016). As reviewed above, mindsets also influence the tendency to use different emotion regulation strategies (e.g., Kneeland, Nolen-Hoeksema, Dovidio, & Gruber, 2016; Tamir et al., 2007). All research to date linking mindsets with emotion regulation has examined self-report measures of emotion regulation. However, emotion regulation is a dynamic process that unfolds over time (Gross, 1998) and so understanding *when* mindsets impact emotion regulation may provide rich insights into the motivational mechanisms involved. For example, does a growth mindset protect against unnecessary anticipatory anxiety prior to initiating an emotion regulation strategy? Or, does a growth belief facilitate adaptive emotion regulation after encountering an emotional stimulus?

Event-related brain potentials (ERPs) – which have high temporal resolution (Luck, 2014) - may help shed light on emotion regulation dynamics across time. ERPs are electric brain signals measured with electroencephalogram (EEG) and are elicited by internal or external events in a given task context. There are two well-studied ERPs that index separable processes of emotion regulation: the stimulus-preceding negativity (SPN) and the late positive potential

(LPP). Described in more detail below, many emotion regulation tasks first present participants with a *cue* instructing participants what to do (e.g., "Look" vs. "Reappraise"), followed by an emotional *stimulus* to which participants carry out the instructions. The SPN is time-locked to the cue and immediately precedes the impending emotional image. The SPN is a frontally maximal ERP and is related to anticipatory and preparatory processes (e.g., Brunia, van Boxtel, & Böcker, 2012). Increases in SPN are thought to reflect increased anticipation and preparation to act on the upcoming emotional image (e.g., Moser, Krompinger, Dietz, & Simons, 2009; Shafir, Schwartz, Blechert, & Sheppes, 2015). It is important to note that not all types of emotion regulation elicit increases in SPN because not all emotion regulation strategies require anticipatory action. When participants are instructed to *distract* or *distance* themselves, the SPN is increased (Moser et al., 2009; Thiruchselvam, Blechert, Sheppes, Rydstrom, & Gross, 2011). This is because there is some preparation for the upcoming emotional image that can be done: participants can begin to "mentally check out" before the image is even displayed. In contrast, there is no SPN increase when participants are instructed to reappraise the upcoming image in a positive way (positive reappraisal; Moser, Hartwig, Moran, Jendrusina, & Kross, 2014) or to increase negative emotions by imagining themselves or a loved-one in the presented negative scene (Moser et al., 2009). In both of these latter cases (positive reappraisal and personalization), "emotional content is necessary to generate alternative representations of the pictured scene for the purposes of modulating emotional reactions" (Moser et al., 2014, p. 98). That is, participants must first view the emotional image prior to engaging in these strategies to alter meaning-making processes; anticipatory activity prior to image presentation is thus unnecessary and potentially unhelpful.

The LPP, on the other hand, is time-locked to the emotional stimulus itself. It reaches maximal amplitude between 300-700ms at centroparietal electrode sites and lasts up to several seconds, depending on the stimulus duration (Cacioppo, Crites, Bernston, & Coles, 1993; Cuthbert et al., 2000). Generally, early LPP windows reflect attention allocation and later ERP windows index meaning-making stages. LPP amplitudes in both windows are larger in the context of emotionally arousing images (either unpleasant or pleasant in valence), compared to emotionally neutral stimuli (e.g., Lang & Bradley, 2010; Schupp et al., 2000). Importantly, the LPP is modulated by top-down cognitive control. Specifically, the LPP is decreased when participants are instructed to direct attention to non-affective aspects of the stimulus (Dunning & Hajcak, 2009; Hajcak, Dunning, & Foti, 2009) or are instructed to reappraise the emotional response (Hajcak & Nieuwenhuis, 2006; Moser, Hajcak, Bukay, & Simons, 2006; Moser et al., 2014). Thus, the LPP may serve as a gauge for how successfully a particular emotion regulation strategy is implemented, with more success resulting in a smaller LPP. In sum, two ERPs have been studied in the context of emotion-regulation processes: whereas the SPN reflects anticipatory activity prior to image onset, the LPP reflects longer-lasting attention allocation and meaning-making processes that unfold over time after image onset. A smaller SPN prior to reappraise trials would indicate less anticipatory activity and smaller LPP on reappraisal trials would indicate more successful emotion regulation.

Critical to the present study is a recent finding that chronic worry interferes with positive reappraisal and is associated with both increased SPN and increased LPP (Moser et al., 2014). Individuals with low levels of worry showed no such increase of SPN prior to reappraisal and successfully reduced LPP amplitudes during reappraisal (versus passive viewing). Increased SPN prior to reappraisal among worriers may indicate unnecessary effortful processing and

anticipatory anxiety prior to engaging in positive reappraisal, which they may have perceived as very difficult. Moreover, increased LPP amplitude during reappraisal may reflect increased emotional arousal — i.e., unsuccessful emotion regulation. These findings – increased anticipation and unsuccessful implementation of positive reappraisal among individuals with chronic worry - are likely the result of years of unsuccessful emotion regulation attempts.

This conceptualization helps bridge emotion-regulation impairments associated with worry and mindset theory. Just as those with more of a fixed mindset of intelligence attribute school failures to internal and unchangeable characteristics like innate ability, individuals with chronic worry may attribute their failures in positive reappraisal to a stable internal deficit in emotion regulatory abilities – they may also come to accept that their anxiety cannot change. Just as those with more of a fixed mindset learn to avoid if possible or endure with great anxiety challenging academic tasks, worriers may come to expect that engaging in positive reappraisal is likely to be a highly aversive and ineffective endeavor. Indeed, increased SPN just before reappraisal trials is suggestive of increased anticipatory preparation and enlarged LPP on these trials is indicative of higher affective arousal experienced.

Thus, Study 2 tested the hypothesis that a growth belief of anxiety may buffer the impact that worry has on the neural correlates of positive reappraisal. The belief that control over anxiety can be cultivated may encourage individuals with worry to employ a more adaptive approach to regulating emotions during the task – one characterized by little anticipatory anxiety or preparation before reappraising and successful implementation of positive reappraisal. Specifically, worriers exposed to the growth mindset of anxiety should demonstrate no anticipatory increase in SPN and reduced LPP on positive reappraisal trials whereas those in the control condition should show anticipatory increase in SPN and either no change in LPP or a

paradoxical increase in LPP on reappraisal trials. Results from Study 2 will supplement those of Study 1 by providing insight into how the anxiety mindset impacts the actual ability of worriers to initiate and sustain a difficult emotion-regulation strategy. In this way, Study 2 will shed new light on how mindsets impact the moment-to-moment mechanisms involved in effortful emotion regulation. This tendency to engage in more effortful emotion regulation strategies may, in turn, relate to preferences for therapy and an expectation for more success in treatment. That is, the growth mindset induction should promote a willingness to engage in effortful strategies to overcome anxiety.

Study 2 Method

Participants

Seventy-five female participants (see Table 1 for demographics) were recruited from the Michigan State University Human Participation in Research subject pool to participate in a "Brain Activity and Emotion Regulation Study" between September 2016 and May 2017. Participants were recruited using similar screening criteria used in Study 1, except that in Study 2, given no time restriction for prescreening, the full 16-item PSWQ was used instead of the single item[2]. Note that the full 16-item PSWQ and item 15 have virtually identical screening properties (Schroder et al., in press). Notably, in Study 2, the prescreening instrument was given at the beginning of the semester and participants completed the study throughout the semester. As a result, the delay between pre-intervention measurement occasion (anxiety symptoms and mindset endorsement) and post-intervention measurements was much longer than in Study 1.

[2] Due to a computer error in prescreening, N=8 participants were recruited who did not meet the clinical cutoff for college students on the PSWQ (a score above 61, Behar et al., 2003). The minimum PSWQ score was 50, which was still above the cutoff for the general population but not for students (Behar et al., 2003) . Analyses were conducted with and without these participants and none of the results changed. Therefore, these participants were included in the final analyses to increase statistical power.

Participants received partial course credit for their participation. Data from 19 participants were excluded prior to EEG analysis because of an excessive number of artifacts in the EEG data in either of the tasks (pre- or post-intervention). The final sample for EEG analysis consisted of 56 participants (N=28 in each group). Despite the data loss, the final EEG sample size was still larger than the needed sample size of 42 determined by a power analysis (power = 0.80) using the effect size of Cohen's d = 0.90 from the Moser et al (2014) study.

Procedure

Upon arrival to the laboratory, participants were introduced to research assistants and provided a description of the procedures. After completing a written consent form, they were fitted with an electrode cap and EEG sensors, as described below. Participants were randomly assigned to condition in Study 2. They completed the emotion-regulation task, the anxiety mindset intervention (or control condition), and then the emotion-regulation task again. There was one additional item in the Control condition for the EEG study following the description of the anxiety disorders: "Write a few sentences about your anxiety. In which situations are you most anxious? Is your experience similar to some of the descriptions you have just read?" The experimenters were blind to the condition (Mindset or Control) to which participants were assigned.

Emotion Regulation Task

The emotion-regulation task was identical to the one used by Moser et al (2014). The stimulus set consisted of 60 color International Affective Picture System images (IAPS; Lang, Bradley, & Cuthbert, 2008): 30 neutral, low-arousal images and 30 negative, high-arousal images. On each trial, participants first viewed an instruction phrase ("REAPPRAISE NEGATIVE", "LOOK NEGATIVE", or "LOOK NEUTRAL") for 2 seconds that directs them

how to respond to the following picture. The "REAPPRAISE NEGATIVE" phrase indicated that participants should imagine that the pictured scene improves and to think of the image in a more positive light so as to decrease the intensity of their negative emotions. The instruction phrase "LOOK NEGATIVE" and "LOOK NEUTRAL" indicated that participants should respond naturally to the presented negative and neutral images, respectively. After the instruction phrase, a blank screen for 500ms and then a black screen with white fixation cross for 500ms was presented. Following the fixation cross, the IAPS image was displayed for 6 seconds. An intertrial interval of 2.5 seconds allowed for participants to relax and clear their minds of the previous image. Participants first completed two practice blocks of the reappraisal task to familiarize themselves with the timing of events and instructions. The entire task consisted of 90 trials (30 LOOK NETURAL, 30 LOOK NEGATIVE, and 30 REAPPRAISE NEGATIVE), presented in random order. LOOK NEUTRAL trials were included as fillers to buffer against possible habituation to the negative images and were not included in analyses.

Task Rating Questionnaire. As a manipulation check, after the task, participants rated their experiences of the positive-reappraisal task for each of the trial types (Look-Neutral, Look-Negative, Reappraise-Negative) along the following dimensions: engagement, effort, attentiveness, understanding, difficulty, and emotional reactivity. They used a 1 (Not at all) to 7 (Very) Likert-type scale.

Psychophysiological recording and data reduction

Psychophysiological recording, analyses, and reporting were carried out in accordance with recommended guidelines (Keil et al., 2014). For EEG set-up, participants were fitted with a stretch-lycra cap. The cap had 66 electrode ports to which electrodes were connected. Continuous electroencephalogram (EEG) activity was recorded using the ActiveTwo BioSesmi

system (BioSemi, Amsterdam, The Netherlands). Recordings were taken from 64 Ag-AgCl electrodes placed in accordance with the 10-20 system. The "10-20 system" refers to the standardized method of applying the location of each of the scalp electrodes – each electrode is spaced apart from adjacent electrodes at a distance of either 10% or 20% of the total front-back or right-left distance of the skull. Figure 2 provides a schematic of the BioSemi system and electrode placement. To fit the cap on participants such that the positioning was standardized and centered on the participants' scalp, measurements were first taken from the nasion (the distinctly depressed area between the eyes) and the inion (the lowest point of the skull from the back of the head and indicated by a prominent bump). Once the cap was applied, an additional measurement from the tip of the left ear to the tip of the right ear was used to center the cap. The chinstrap was then tightened to a snug but comfortable fit. Sixty-six labeled ports in the cap indicate where each of the scalp electrodes is to be plugged in. The labels of the scalp electrodes roughly correspond to areas of the cerebral cortex. The letters F, T, C, P, and O stand for frontal, temporal, central, parietal, and occipital lobes, respectively. Odd numbers correspond to left hemisphere sites and even numbers correspond to right hemisphere sites. Z sites correspond to central sites along the midline of the scalp. For example, the site "Pz" is placed in the center of the cap over parietal cortex, "P1" is placed above the parietal cortex, just left of center, "P3" is placed just left of P1, and so on.

Two electrodes were placed on the left and right mastoids – the mastoid is the bony part just behind the ears. The mastoid electrodes were used as a common reference for all other electrode sites during offline data collection. Electrooculogram (EOG) activity generated by eye movements and blinks was recorded at Fp1 (the site directly above the left eye, see Figure 2) and at three additional electrodes placed inferior to the left pupil and on the left and right outer canthi

(the outer or inner corner of the eye, where the upper and lower lids meet), all approximately 1 cm from the pupil). During data acquisition, the common-mode sense (CMS) active electrode and the driven right-leg (DRL) passive electrode formed the ground, as per BioSemi's design specifications. The function of the CMS-DRL loop, in addition to forming a reference, is to constrain the common mode voltage (that is, the average voltage of the participant), which limits the amount of current that can possibly return to the participant. All signals were digitized at 1024 Hz – 1,024 samples of data taken per second for millisecond precision – using ActiView software (BioSemi).

Offline analyses were performed using Brain Vision Analyzer 2 (BrainProducts, Gilching, Germany). Scalp electrode recordings were re-referenced to the numeric mean of the mastoids. That is, the average activity recorded at the mastoids was subtracted from activity at all other scalp electrodes. Data were then bandpass filtered with cutoffs of 0.1 and 30 Hz (12 dB/oct rolloff). Only data in this frequency band (between 0.1 and 30 Hz) were analyzed because this is where the relevant signals exist (Hajcak et al., 2012; Luck, 2014).

Ocular artifacts were corrected using the method developed by Gratton, Coles, & Donchin (1983). Ocular correction refers to the procedure in which eye movement (e.g., eye blinks or eye movement irrelevant to the experiment) is accounted for in the EEG data before analysis. Eye movement can modify scalp recorded electrical activity and this procedure is aimed at correcting this modification. Importantly, most eye movement interference occurs at the front of the scalp (i.e., near the eyes). The Gratton et al (1983) method allows for the inclusion of all ERP trials as it computes separate estimates of eye blink and eye movement correction during the experimental session and estimates a propagation factor that accounts for the differential

impact of eye movement across the scalp. To further minimize blink interference, subjects were instructed to blink as little as possible while the images were displayed.

Stimulus-locked data were segmented into individual epochs beginning 200ms before stimulus onset and continuing for 6,000ms following the stimulus. To detect physiologic artifacts, a computer-based algorithm scanned each 6,200ms segment and looked for three criteria and if any of these were met, the trial was rejected. First, trials were rejected if they contained a voltage step exceeding 50 μV between consecutive sampling points – such a steep change is unlikely to reflect naturally occurring brain activity. Second, trials were rejected if they contained a difference of more than 200 μV within a trial – again, it is unlikely that such an excessive change would occur naturally. Finally, trials were rejected if the maximum voltage difference was less than 0.5 μV within a trial; this could reflect a problem with the scalp electrode recording properly.

To calculate the SPN, segments were averaged across trials at six electrode sites from the frontal and central part of the scalp (F1, Fz, F2, FC1, FCz, and FC2) in two time windows and then averaged together across electrode sites – this procedure is known as pooling (i.e., averaging across electrode sites; Luck, 2014). Pooling is helpful for improving the precision of ERP measurement by capitalizing on the high inter-channel correlations of adjacent electrodes. It also helps increase statistical power. See Figure 2 for a depiction of the electrode pools used in the ERP analyses. As per convention, a baseline of the average activity in the -200 to 0ms pre-cue window was subtracted from each data point subsequent to stimulus onset. Baseline corrections are used to minimize offsets and drifts that occur just before stimulus onset. As in previous studies (Moser et al., 2014), the early SPN was quantified as the average activity in the 300-2000ms post-cue ERP window and the late SPN as the average amplitude in the 2300-

3000ms post-cue window. The early window captures orienting and processing of the cue and the later window most likely reflects anticipation of the impending stimulus (e.g., Moser et al., 2009; van Boxtel & Bocker, 2004).

The LPP was quantified using two electrode pools: a frontal electrode pool (F1, Fz, F2, FC1, FCz, FC2) and a parietal electrode pool (CPz, P1, Pz, P2, POz). Both frontal and parietal LPPs were considered in order to observe any differences across the scalp; although most studies of the LPP focus on parietal electrode sites (Foti & Hajcak, 2008), effects of interventions (e.g., exposure therapy) have been observed for the frontal LPP (e.g., Leutgeb et al., 2009, 2012). Whereas the parietal LPP may reflect attention allocation to emotional stimuli, some researchers suggest the frontal LPP may reflect participants' "genuine efforts to decrease emotional expression" (Murata, Moser, & Kitayama, 2013). As efforts to decrease emotional responding was a key component of the current study of positive reappraisal, the frontal LPP was also considered. Both frontal and parietal LPPs were defined as the average amplitude in successive ERP windows after picture onset (400-700ms, 700-1000ms, 1-2s, 2-3s, 3-4s, 4-5s, and 5-6s) per convention (Cuthbert et al., 2000; Moser et al., 2014). The average amplitude in the -200 to 0ms pre-picture onset was used as the baseline for the LPP. See ERP figures below for a depiction of the different windows used in analyses.

Analyses and Anticipated Results

Repeated-measures ANOVAs were used to evaluate changes from pre- to post-intervention on measures of anxiety symptoms and mindset endorsement. These were 2 Time (Pre vs. Post) x 2 Condition (Mindset vs. Control) ANOVAs. It should be noted that given the structure of the online recruitment procedure in Study 2, that pre-intervention measures were collected days to weeks before the post-intervention measures were. The SPN analysis then

consisted of a 2 Trial Type (Look-Negative vs. Reappraise Negative) x 2 ERP Window (300-2000ms vs. 2300-3000ms) x 2 Time (Pre vs. Post-Intervention) x 2 Condition (Mindset vs. Control) ANOVAs. The early LPP analysis consisted of a 2 Time (Pre vs. Post-Intervention) x 2 ERP Window (400-700ms, 700-1000ms) x 2 Trial Type (Look-Negative vs. Reappraise Negative) x 2 Condition (Mindset vs. Control) ANOVAs. The late LPP analysis consisted of a 2 Trial Type (Look-Negative vs. Reappraise Negative) x 5 ERP Window (1-2s, 2-3s, 3-4s, 4-5s, 5-6s) x 2 Time (Pre vs. Post-Intervention) x 2 Condition (Mindset vs. Control) ANOVA.

A significant Time x Trial Type x Condition interaction was expected to emerge in both SPN and LPP analyses. The predicted interaction would show increased SPN on Reappraisal trials compared to Look-Negative trials in both groups at Time 1, but a decreased SPN on Reappraisal trials at Time 2 would emerge in the Mindset condition such that the difference between Reappraise and Look-Negative trials would only remain in the Control condition. Similarly, for the LPP analysis, the predicted interaction would show increased LPP on Reappraisal trials compared to Look-Negative trials for both groups at Time 1, but a decrease in LPP after the Mindset condition would show a smaller LPP on Reappraise trials compared to Look-Negative trials in this group only at Time 2. Reduced SPN prior to reappraisal trials would suggest reduced anticipatory effort, whereas reduced LPP would reflect more successful positive reappraisal. Some of the degrees of freedom vary slightly across statistical tests due to missing questionnaire data. Prior to analysis, data were screened to ensure that general linear model assumptions (normality, linearity, homogeneity of variance) were satisfied. None of the study variables violated these assumptions.

Study 2 Results

Manipulation Check

Duration of Intervention. Data from five participants were excluded from the calculation of the duration of intervention due to experimenter error (N = 69). The average length of completing the control tutorial was 16.59 minutes (*SD* = 7.20; range 8.55 to 36.42) and for the anxiety mindset condition the length was *M* = 16.91 minutes (*SD* = 7.74; range 7.75 to 41.02; these times were not significantly different between conditions ($t(67) = 0.18$, $p = .86$). As in Study 1, covariate analyses are presented below controlling for duration of intervention.

Self-Report Results

For self-report measures, the full sample size of available data (N=74; anxiety mindset *n* = 39, control *n* = 34) was used (one participant's Condition assignment was not identifiable). Degrees of freedom vary slightly due to missing questionnaire data. Descriptive statistics and bivariate correlations of all Study 2 self-report variables across the entire sample are presented in Table 4, and descriptive statistics for variables for both conditions separately are presented in Table 5.

Mindsets and Anxiety. First, 2 Time x 2 Condition repeated-measures ANOVAs were conducted to assess changes from pre- to post-intervention effects between conditions for self-reported anxiety and mindset measures. Recall that the delay in pre- to post-measurement occasions was larger than that in Study 1 in that pre-intervention assessments were collected online earlier in the semester, days to weeks before post-intervention measurement collection. For PSWQ scores, neither the main effect of Time ($F(1, 71) = 0.66$, $p = .42$, $\eta^2_p = .01$) nor the interaction between Time and Condition ($F(1, 71) = 2.08$, $p = .15$, $\eta^2_p = .03$) was significant. For GAD-7 scores, the main effect of Time was significant ($F(1, 71) = 9.66$, $p = .003$, $\eta^2_p = .12$)

which indicated that all participants endorsed higher GAD-7 scores after the intervention compared to the online prescreening assessment. The interaction between Time and Condition was not significant (F (1, 71) = 0.30, p = .59, η^2_p = .004). For TOA (anxiety mindset) scores, the main effect of Time was significant (F(1, 70) = 10.90, p = .002, η^2_p = .14) –participants across both conditions endorsed higher growth mindset after the intervention compared to before. The main effect of Condition was also significant (F(1, 70) = 9.64, p = .003, η^2_p = .12) indicating that individuals in the Mindset condition had higher endorsement of mindset scores compared to the Control condition at both pre- and post-intervention(see Table 5). However, the interaction between Time and Condition was not significant (F(1, 70) = 1.42, p = .24, η^2_p = .02).

Hypothetical Treatment Choice, Expectations, and Willingness to Engage. For the hypothetical treatment choice item, 79% of participants in the Mindset condition chose therapy, compared to 71% of participants in the Control condition. The chi-square test was not statistically significant, X^2 = 0.65, df = 1, p = .30.

Scores on the treatment expectancy and treatment likelihood items were not significantly different between groups (see Table 5). Univariate ANOVAs revealed no significant main effect of Condition for treatment expectancy (F(1, 72) = 1.72, p = .19, η^2_p = .02, d = 0.31), nor a significant main effect of Condition for treatment likelihood (F(1, 72) = 0.33, p = .56, η^2_p = .01, d = 0.13).

As in Study 1, duration of intervention was controlled for with covariate analyses. Adding duration as a covariate did not impact the results of the self-reported outcomes. The Time x Condition interaction was still not significant for TOA scores (F(1, 69) = 1.60, p = .21, η^2_p = .023). The main effect of Condition was not statistically significant for expected

improvement ($F(1, 68) = 2.23$, $p = .14$, $\eta^2_p = .033$), nor for likelihood to begin treatment ($F(1, 68) = 0.48$, $p = .49$, $\eta^2_p = .007$).

Event-Related Potentials (ERPs)

As described above in the Participants section, data from 56 participants were available for ERP analysis.

Stimulus-Preceding Negativity (SPN). Descriptive statistics for SPN amplitudes are presented in Table 6. All ERP figures display grand-averaged waveforms. Grand averages are computed by taking the average of all participants' averaged waveforms (averaged across several trials) for each condition separately. Grand-averaged waveforms for the cue-locked ERPs are presented in Figure 3, where Time 0 represents the onset of the cue (i.e., the moment when the phrase "Look-Negative" or "Reappraise-Negative" was on the screen). Results from the ANOVA are presented in Table 7. For regulation-related effects on the SPN, there was only one significant main effect of ERP Window, in which the SPN became smaller from the early to later ERP window (see Table 7). No further interactions or main effects of interest were found for regulation trials. Thus, for the SPN, the predicted Time x Trial Type x Condition interaction did not emerge.

Late Positive Potential (LPP). Descriptive statistics for frontal and parietal LPP amplitudes are presented in Tables 8 and 9, respectively. Results from the frontal and parietal LPP ANOVAs are presented in Tables 10 and 11, respectively. Grand-averaged waveforms for picture-locked ERPs (i.e., the ERPs elicited after the emotional image) are presented in Figures 4 and 5. In these figures, Time 0 represents the onset of the picture stimulus. For brevity, only the effects involving Condition are discussed in detail here, although all effects from the ANOVAs

are listed in the aforementioned Tables. Note that no interactions involving Condition were found in the 400-700ms or 700-1000ms windows.

The predicted Time x Trial Type x Condition interaction did not emerge (see Tables 10 and 11). Again, this interaction effect would have shown that the reappraisal trials elicited a smaller LPP than the look-negative trials, and that this difference would have been evident at Time 2 for the Mindset condition only. Thus, the main prediction of the study was not supported. Second, there were baseline differences observed in the LPP (i.e., differences before the intervention occurred), rendering between-group differences after the intervention difficult to interpret. Therefore, interactions involving Condition, discussed below, were followed-up by examining effects for each condition separately.

A significant Time x Condition interaction emerged at the parietal LPP pool in the 1-6s ERP window $F(1, 54) = 5.37$, $p = .024$, $\eta^2_p = .091$; see Table 11). Follow-up paired t-tests were conducted to further probe this interaction. The LPP was averaged across windows and trial types and then the Time 1 vs. Time 2 LPPs were compared for each condition separately. For the Control condition, the LPP was not different from Time 1 to Time 2 ($t(27) = 0.21$, $p = .84$). However, in the Mindset condition, an unexpected decrease in the LPP from Time 1 to Time 2 was found ($t(27) = 3.09$, $p = .005$). That is, across both trial types (look and reappraise) and ERP windows, the overall LPP was decreased from Time 1 to Time 2 in the Mindset condition only.

There was also a Trial Type x Condition interaction in the 1-6s time range at both frontal ($F(1, 54) = 7.91$, $p = .006$, $\eta^2_p = .13$) and parietal ($F(1, 54) = 11.01$, $p = .002$, $\eta^2_p = .17$) electrode pools. To further probe this interaction, Look-Negative vs. Reappraise-Negative trials (averaged across Time and the 1-6s ERP windows) were compared in paired t-tests for each Condition separately. In the Control condition, the LPP on Reappraise-Negative trials was significantly

smaller than on Look-Negative trials at both frontal sites ($t(27) = 2.55$, $p = .017$) and parietal sites ($t(27) = 3.98$, $p < .001$; see Figures 4 and 5). This indicates the Control participants were able to decrease the LPP on Reappraise-Negative trials at both pre and post time points. This is also apparent in the headmaps displayed in Figure 6; cooler colors indicate smaller LPP for Reappraise versus Look-Negative trials. In contrast, there was no such difference in the Mindset condition at frontal ($t(27) = 1.23$, $p = .23$) or parietal sites ($t(27) = -.14$, $p = .89$).

Finally, in the parietal 1-6s analysis, there was a marginal four-way interaction between Time, ERP Window, Trial Type, and Condition ($F(4, 216) = 3.02$, $p = .052$, $\eta^2_p = .053$). To further understand this interaction, the Time x ERP Window x Trial Type ANOVA was conducted in each Condition separately. The three-way interaction emerged for the Control condition ($F(4, 108) = 4.58$, $p = .002$, , $\eta^2_p = .15$) but not for the Mindset condition ($F(4, 108) = 0.78$, $p = .54$, , $\eta^2_p = .028$). The two-way ERP Window x Trial Type ANOVA was then conducted for Time 1 and Time 2 separately in the Control condition. This analysis revealed no ERP Window x Trial Type interaction at Time 1 ($F(4, 108) = 0.98$, $p = .42$, $\eta^2_p = .035$), but a significant interaction at Time 2 ($F(4, 108) = 9.68$, $p < .001$, $\eta^2_p = .26$). To further decompose this interaction, paired-samples t-tests were conducted comparing Reappraise and Look-Negative trials for each of the ERP windows (1-2s, 2-3s, 3-4s, 4-5s, 5-6s) at Time 2. This analysis indicated the reappraisal effect, in which the LPP was smaller on the Reappraise trials, was non-significant in the 1-2s window ($t(27) = 1.23$, $p = .23$) but was significant in subsequent windows and the effect grew steadily larger across time, peaking in the 4-5s window (2-3s: $t(27) = 2.58$, $p = .016$; 3-4s: $t(27) = 4.41$, $p < .001$; 4-5s: $t(27) = 4.71$, $p < .001$; 5-6s: $t(27) = 4.31$, $p < .001$). This is evident in Figure 6, in which the Control condition headmaps at Time 2 grow steadily cooler

across ERP windows, indicating the regulation effect gets larger across windows at the post-test assessment.

To summarize the LPP findings, the predicted Time x Trial Type x Condition interaction was not observed. This effect would have shown that the reappraisal condition elicited a smaller LPP than the look-negative condition, and that this difference would have been evident at Time 2 for the Mindset condition only. Baseline differences in LPP made comparisons between groups difficult, however. Participants in the Control condition were able to reappraise their emotions (as indicated by reduced LPP on reappraise trials) at both time points. This was unexpected, as the Control group looked like the low-worry group in the Moser et al. (2014) study by showing the reappraisal effect. Participants in the Mindset condition did not show this reappraisal effect at either time point. However, the Mindset condition showed an overall decrease in LPP amplitude (on both Look-Negative and Reappraise trials) from pre-to-post intervention. To further understand the ERP results, a series of ancillary analyses were conducted, which are presented next.

Ancillary Analyses

To help contextualize the findings in Study 2, two ancillary analyses were carried out. First, task-rating data from the emotion regulation task were examined. The purpose of this analysis was to provide a validity check on the emotion regulation task by considering participants' subjective, self-reported experiences about the task. These data were collected at the end of the emotion regulation task and asked participants to rate on a scale of 1-7 how well they understood the task, how difficult they found the task to be, how much effort and attention they put into the task, and their emotional reactions to the task. Each of these questions was asked for each trial type (Look-Neutral, Look-Negative, and Reappraise-Negative). The task rating data

are presented in Table 12. As expected, participants rated Reappraise-Negative trials as more difficult, more effortful, and more attention demanding than Look-Negative trials.

However, an unexpected finding was that participants reported more emotional reactivity for Reappraise-Negative trials compared to Look-Negative trials. A 2 Time x 2 Trial Type x 2 Condition ANOVA on emotion reaction ratings revealed a main effect of Trial Type ($F(1, 54) = 22.09$, $p < .001$, $\eta^2_p = .29$), indicating that reappraise negative trials were more emotionally reactive than look-negative trials. A Trial Type x Time interaction indicated that the effect was stronger at Time 2 ($t(55) = 5.44$, $p < .001$) than at Time 1 ($t(55) = 2.08$, $p = .043$). Few participants rated Reappraise trials as less emotionally reactive than Look-Negative trials (N = 8 at T1 and N = 6 at T2). These findings are in direct contrast to previous research using the identical task (Moser et al., 2014), where participants reported less emotional reactivity on reappraisal trials. A significant main effect of Time ($F(1, 54) = 13.92$, $p < .001$, $\eta^2_p = .21$) indicated that emotional reaction ratings were lower at Time 2 than at Time 1. The lack of effects involving Condition indicates that ratings became lower at Time 2 for both the Control and Mindset groups[3].

The second ancillary analysis considered electromyogram (EMG), which measures the electrical activity from the corrugator muscle of the face (see Cacioppo, Petty, Losch, & Kim, 1986). EMG data allow for another indicator of task validity by examining whether the Reappraisal trials were less physiologically arousing than the Look-Negative trials. Corrugator EMG is a reliable indicator of online physiological response to emotional valence, such that a

[3] Only two small but significant differences emerged at Time 1 between the two experimental conditions – participants in the Mindset condition endorsed greater understanding for Look-Negative and Reappraise-Negative trials compared to the Control group (see Table 12). None of the ERP analyses reported above changed when taking these into consideration (with understanding ratings covaried out in the ANOVAs).

larger EMG response has been found for negative compared to neutral images (Cacioppo et al., 1986). EMG is also sensitive to emotion regulation such that it is reduced for regulation trials (Bernat et al., 2011). EMG data were segmented into successive 500ms bins beginning from time 0 (stimulus onset) and extending to 6000ms. A 2 Time x 12 EMG Window x 2 Trial Type (Look Negative vs. Reappraise Negative) x 2 Condition ANOVA was conducted[4]. A significant Trial Type x EMG Window interaction ($F(11, 594) = 8.76$, $p < .001$, $\eta^2_p = .14$) indicated that the Reappraise-Negative trials had less EMG reactivity than Look-Negative trials and that this difference became larger across time. This interaction is depicted in Figure 7. A significant main effect of Time ($F(1, 54) = 33.26$, $p < .001$, $\eta^2_p = .38$) indicated that overall EMG reactivity was smaller at Time 2 than at Time 1. The only effect involving Condition was a marginal main effect ($F(1, 54) = 3.32$, $p = .074$, $\eta^2_p = .058$) which revealed that, overall, those in the Mindset condition showed marginally more EMG reactivity across both trial types and across both time points.

Study 2 Discussion

Study 2 did not find the self-reported increase in growth mindset endorsement following the anxiety mindset intervention (i.e., the interaction between Time and Condition was not significant). Moreover, Study 2 did not replicate the increased effectiveness and likelihood ratings in the Mindset condition that was found in Study 1.

With regards to the ERPs, between-group differences at baseline made interpretations difficult when comparing Control and Mindset conditions. Importantly, the predicted interaction effects were not observed. Examining each condition separately is necessary to understand the effects. The Control group unexpectedly showed the reappraisal effect on the LPP – it was

[4] Results were unchanged when only EMG windows between 1000-6000ms were analyzed.

reduced on reappraise trials at both time points. The Mindset condition showed no such reappraisal effect at either time point. Again, it was hypothesized that the Mindset condition would show the reappraisal effect at Time 2. However, those in the Mindset condition showed a general decrease in overall LPP (on both trial types) from Time 1 to Time 2. This may indicate a less intense emotional reaction to the negative stimuli (regardless of trial type) after the intervention.

The self-report task rating data unexpectedly showed that most participants found Reappraise-Negative trials to be more emotionally reactive than Look-Negative trials. This contrasts with previous findings (Gross, 1998; Moser et al., 2014; Ray, McRae, Ochsner, & Gross, 2010), which indicated that participants found Reappraise trials to be less emotionally reactive. This is also in contrast to the EMG data, which suggested that participants had less physiological reactivity to Reappraise trials. Finally, from Time 1 to Time 2, across both trial types, participants showed both a decrease in EMG and a decrease in self-reported emotional reactivity.

At first blush, the results of Study 2 are somewhat difficult to piece together. Regardless of experimental condition, reappraisal trials elicited *lower* physiological reactivity indexed by EMG, yet these trials elicited *higher* self-reported emotional reactivity. Participants in both conditions also reported higher GAD-7 symptoms after the tutorials; however, there was a longer amount of time in between GAD-7 assessments (e.g., in some cases, weeks) so it is difficult to attribute this increase to either tutorial rather than the passage of time. This discrepancy between levels of analysis may be specific to the sample of individuals with chronic worry. Perhaps on the autonomic nervous system level, participants were experiencing some down-regulated emotional experience but this was not translated consciously and the ultimate evaluation of

reappraisal trials was more subjective emotional reactivity. This is noteworthy considering some theories of generalized anxiety disorder hypothesize that worries dampen autonomic reactivity (Borkovec, Lyonfields, Wiser, & Diehl, 1993). It is also possible that the time interval between the viewing of the pictures (i.e., the "online" EMG data during the task) and the "offline" self-reported emotional experience after the task was sufficiently long for individuals to "forget" that they felt down-regulated during reappraisal trials. Future studies with simultaneous EMG data collection and self-reported emotional experience built into the task (e.g., trial-to-trial assessment, as opposed to assessment after the task) may help elucidate these differences. Understanding the time course of this discrepancy between physiological and self-reported levels of analysis is an interesting area for future research.

The two conditions differed on LPP amplitudes at baseline, and these differences need to be considered for any between-groups comparisons. Within-groups comparisons (across time) are likely to be more helpful in understanding the data presented here. There was a significant decrease in overall LPP amplitude in the Mindset group from Time 1 to Time 2; this decrease was absent in the Control condition. Together with the self-report and EMG data, this suggests that participants in the Mindset condition decreased emotional reactivity in all three levels of analysis across time – subjective experience (self-report), autonomic reactivity (EMG), and attention allocation to negatively arousing emotional information (LPP). All of these changes occurred across all trials, rather than a function of emotion regulation trial type. In this way, then, participants in the Mindset group had more synchrony across systems after the intervention. Those in the Control condition had no overall change to the LPP despite an overall decreased subjective emotional experience and decreased autonomic reactivity (EMG) (again, not as a function of trial type).

It is not obvious what the decrease in LPP after the Mindset condition may reflect. It is unlikely to be simply fatigue, as there was no decrease in the Control condition. Considering the literature on the LPP, the decrease may indicate that participants in the Mindset condition allocated less attention to the most arousing aspects of the negative emotional images (e.g., Dunning & Hajcak, 2009) after the intervention. If this were the case, it would indicate that the growth mindset of anxiety message prompts (either consciously or unconsciously) individuals to allocate fewer resources to negative stimuli. This may help explain why individuals with growth mindsets seem to fare better in the face of negatively valenced situations such as difficult academic transitions and interpersonal distress (Yeager et al., 2013, 2014). However, this suggestion is speculative at this stage, particularly because this was not a hypothesized effect.

GENERAL DISCUSSION

Despite decades of research demonstrating that the growth mindset cultivates intrinsic motivation and a willingness to engage in effortful behaviors in many domains, the application of this knowledge to clinical psychology is limited. The two current experiments were designed to advance understanding about the links between the anxiety mindset and motivations for engaging in adaptive strategies that promote mental health. The online Study 1 evaluated the effects of a novel anxiety mindset intervention on treatment preferences and motivations to engage in treatment. The in-lab Study 2 examined this intervention's impact on these motivations as well as the neural correlates of positive reappraisal, an adaptive emotion regulation strategy.

Study 1

Study 1's findings indicated that the mindset intervention lead to a greater endorsement of the growth mindset of anxiety compared to the control condition. This is the first study to show this, as all previous research on this construct was cross-sectional (Schroder et al., 2015, 2016, 2017; Yalch, Schroder, Dawood, & Donnellan, in press). The evidence is accumulating that the anxiety mindset may function similarly to other mindsets that have been studied for decades such as the intelligence and personality mindsets, but is more closely linked with mental health outcomes. Specifically, the anxiety mindset was first demonstrated to be a reliable measure (Schroder et al., 2015) and psychometrically distinct from other mindsets of intelligence, personality, and emotion (Schroder et al., 2015, 2016). The anxiety mindset has also been shown to be predictive of mental health-related outcomes, (Schroder et al., 2015, 2016, 2017; Yalch et al., 2017) and most recently, shown to be predictive of future distress even when controlling for baseline levels of anxiety and depression (Schroder, Callahan, Gornick, & Moser, *manuscript in*

revision). The current study provided the logical next step in this line of work and demonstrated that this belief can be induced.

Study 1 also found that individuals with anxiety exposed to the growth mindset intervention endorsed increased willingness to engage in treatment (although this was at a trend level once duration of intervention was controlled for) and rated the hypothetical treatment as more effective than those who received the control tutorial (although this was also at a trend level). Although the effect sizes were small, these findings are suggestive that inducing a growth anxiety mindset leads to perceptions that may be helpful for treatment.

Neither Study 1 nor Study 2 found that the anxiety mindset induction lead to a preference for therapy over medication. This hypothesis was based on the assumption that therapy would be perceived as more effortful than medication, and previous mindset interventions found that individuals exposed to the growth mindset were more likely to engage or prefer effortful strategies (Blackwell et al., 2007; Hong et al., 1999). Moreover, previous correlational work found the anxiety mindset was related to a preference for therapy vs. medication (Schroder et al., 2015). The null finding of treatment preference may be due to a number of reasons. First, it is possible that the intervention was simply not potent enough to sway perceptions. Indeed, some previous mindset intervention studies have failed to find an impact on effort and motivation (e.g., Hunt, 2017; Li & Hunt, 2017; Orosz et al., 2017; Sisk et al., in press). Second, consistent with a meta-analysis of treatment preferences (McHugh et al., 2013), over three quarters of participants across both studies chose the therapy option; perhaps there was not enough variance to detect effects of the intervention on this variable. Examining the intervention's impact on treatment preference in a larger sample with greater variability in treatment preference may help address this potential ceiling effect. Finally, the control condition was an anxiety education condition and

did not induce a fixed mindset of anxiety – this may have attenuated differences between intervention and control conditions. Future studies inducing a fixed mindset of anxiety may find that individuals are more likely to choose medication.

Study 2

Study 2's findings were less straightforward. The growth mindset was not induced successfully in Study 2 and the increased self-reported motivation indices after the mindset intervention were not replicated. This may have been due to a number of reasons as well. The sample size was much smaller than in Study 1. The sample was also qualitatively different from that of Study 1 in that it was composed of younger anxious female undergraduates vs. a community sample of anxious male and female adults in Study 1. It is possible the intervention's effects on motivation to engage in treatment are more potent for older individuals who have had more time with untreated anxious distress. Moreover, there was an additional task (the emotion-regulation task) in between the intervention and the assessment of motivational indices; it is possible that the additional time that the task took interfered with the intervention's effects on self-reported motivation.

None of the predicted ERP results was found. There were no differences in the SPN between trial types, between time points, or between conditions. This is in contrast to previous studies using the identical task (Moser et al., 2014). The SPN reflects anticipatory activity prior to the trial (Brunia et al., 2012); the null findings here may suggest the mindset intervention does not impact anticipatory effort prior to reappraisal. The LPP was difficult to interpret due to baseline differences between conditions prior to the intervention being administered. At baseline, the Control condition showed a reduced LPP for Reappraise trials, even before the intervention. A smaller LPP on Reappraise trials suggests more successful emotion regulation, if the LPP is

seen as an index of attention allocation to emotional information (Cuthbert et al., 2000; Moser et al., 2014). This was unexpected as all participants were anxious. In contrast, those in the Mindset condition looked more similar to the high-worry group in the Moser study in that the two trial types were not as well differentiated.

Moreover, the self-report task ratings showed that participants found the reappraisal trials more emotionally reactive than the Look-Negative trials. This is in contrast to previous studies that used an identical task (Moser et al., 2014). This is particularly interesting given that the corrugator EMG data revealed that on the autonomic nervous system level, participants were experiencing a decreased physiologic response on reappraise trials. There appears to be disconnects between autonomic and central nervous physiology and self-report data that future studies will need to tease apart.

Despite these peculiarities, the most striking difference from pre- to post- intervention was the overall decrease in the late parietal LPP in the Mindset condition. This was observed across Look-Negative and Reappraise-Negative trials. The lack of differentiation by trial type (i.e., no significant effects involving Condition, Time, and Trial Type) indicates this was a broad and non-specific decrease in LPP amplitude to negative stimuli. It is difficult to speculate as to what this decrease reflects, but a few interpretations may be offered. Considering the literature on the LPP, the decrease may indicate that participants in the Mindset condition allocated less attention to the most arousing aspects of the negative emotional images (e.g., Dunning & Hajcak, 2009) after the intervention. Whether this was a conscious effort to decrease overall reactivity is not clear; if so, this may indicate participants in the Mindset condition were more motivated to down-regulate their emotions during the task regardless of trial type. It may also mean that participants in the Mindset condition were less engaged with the task after the intervention.

However, the Control condition did not show an overall decrease in LPP amplitude; the decrease was specific to the Mindset condition and so it is unlikely that fatigue or task engagement would explain the decrease. Finally, there were was an overall decrease across levels of analysis in the Mindset condition, as self-reported emotional reaction, EMG, and ERP amplitudes were all decreased at Time 2 compared to Time 1, regardless of trial type. This coherence was not found in the Control condition – although self-report and EMG reactivity was decreased at Time 2, the LPP was unchanged at Time 2.

Limitations and Future Directions

There were a number of limitations to the current studies that future work will need to address. First, outcomes were collected only immediately after the intervention and thus the duration of the effects will need to be assessed at longer intervals in future studies. It will be important to understand how long the increase in growth mindset of anxiety is maintained, as well as the increase in motivation for treatment. Second, Study 1 did not utilize true random assignment; rather, conditions were posted online at separate times. However, the description of the study was identical across conditions, eliminating any potential self-selection bias. Moreover, no participants completed both conditions. Larger-scale studies utilizing true random assignment will be necessary to see if effects hold. Third, baseline differences in the ERP study made it difficult to compare between conditions. This may be an issue of inadequate power to wash out chance noise generated from random assignment. A number of subjects were lost due to excessive EEG artifacts and had this not happened it is possible these baseline differences would have not been present.

Despite these limitations, the two studies presented here provide the first evidence that the growth mindset belief can be induced with a brief intervention discussing the fact that

anxiety can change. There was also evidence that motivations to engage in treatment and perceptions that treatment would be helpful were also increased after this novel intervention (only in Study 1). Some evidence was presented to suggest that the mindset intervention also decreased attention allocation to negative aspects of the emotional stimuli, although this suggestion is speculative given the above-mentioned limitations in baseline differences.

The current studies help point to future directions investigating the causal effects of the anxiety mindset. As this was the first study to design and implement an anxiety mindset intervention, future studies will need to fine-tune the intervention's message to assess whether more or different information may make the message more potent. It will also be important to distill the most important points from the intervention to create an even more concise intervention. Future studies could also assess whether effects would be stronger if the control condition had induced the fixed mindset of anxiety. Because the anxiety mindset is also correlated with a number of other psychological symptoms (e.g. depression, perfectionism, interpersonal problems; Schroder et al., 2015), understanding the intervention's effects on other distressed populations is another next step.

These are important future directions because an increasingly recognized asset of mindset interventions is their scalability (Paunesku et al., 2015) – they are easily disseminated interventions that have the potential to provide measurable, albeit small (Burnette et al., 2013; Sisk et al., in press) benefits to many individuals at little to no cost. This is especially relevant given the high prevalence of anxiety disorders and the fact that most of these individuals do not seek treatment at all. Given the somewhat mixed results of previous mindset intervention studies (see review in Introduction), it will be important to continue to collect data and assess different outcomes from this and related interventions. In this first anxiety mindset intervention study, the

focus was on increasing motivation to engage in effortful behaviors related to overcoming anxiety, measured with self-report items and ERPs indexing engagement in effortful emotion regulation strategies. Future studies will need to assess how this intervention actually translates to motivated behavior (i.e., following up with therapy appointments, engagement in treatment, symptom reduction across time). Understanding the key messages that are most impactful could be beneficial in terms of future efforts to scale these types of interventions (Kenthirarajah and Walton, 2015). This is particularly important given recent data indicating mindset interventions may not directly relate to distal outcomes as strongly as previously thought (Sisk et al., in press).

In terms of examining the effects of the intervention on the neural correlates of emotion regulation, a larger sample size may be necessary to wash out baseline differences that were present here. It will also be useful to examine other emotion regulation strategies other than positive reappraisal, such as emotional suppression and distraction, which have slightly different effects on the LPP.

In conclusion, a brief self-administered intervention designed to induce the growth mindset of anxiety was developed and tested in two samples of individuals with high anxiety. Results from the online Study 1 were suggestive that the intervention lead to increased growth mindset endorsement, greater willingness to engage in treatment and increased expectations for treatment (at a trend level). The much smaller Study 2 did not replicate these self-reported findings and did not find hypothesized effects on event-related brain potentials. However, Study 2 provided suggestive evidence that the intervention decreased attention allocation to negative stimuli overall (regardless of emotion regulation instruction). Despite these mixed findings, this research helps lay the groundwork for more studies to come examining whether the intervention developed here leads to meaningful changes in motivation, treatment preferences, and

engagement in emotion regulation processes The ultimate goal in this line of work is to further understand how beliefs about how much self-attributes can change impacts mental health and to leverage this knowledge to help individuals who may need it the most.

APPENDICES

APPENDIX A

TABLES

Table 1. Sample demographics of Study 1 and Study 2

	Study 1		Study 2	
Variable	*M*	*SD*	*M*	*SD*
Age	33.49	11.48	18.97	1.15
% Female	74.1	--	100	--
Native Language	*N*	*%*	*N*	*%*
English	277	96.85	66	89.2
Other	9	3.15	7	9.5
Ethnicity	*N*	*%*	*N*	*%*
White	217	75.87	49	66.22
African American	24	8.39	7	9.46
Hispanic or Latino/a	10	3.50	5	6.76
Asian	23	8.04	4	5.41
American Indian or Alaska Native	3	1.05	0	0
Other	9	3.15	9	12.16

Table 2. Means, standard deviations, and bivariate correlations among variables across the entire sample in Study 1

	M	*SD*	*Range*	1.	2.	3.	4.	5.	6.	7.	8.
1. Prescreen PSWQ Item 15	4.41	0.49	4.00-5.00	--							
2. Prescreen TOA item	2.89	1.50	1.00-6.00	-.21**	--						
3. Prescreen GAD-7	13.09	5.21	0.00-21.00	.48**	-.25**	(.89)					
4. Hypothetical Treatment Choice	0.79	0.41	0.00-1.00	.01	-.003	-.11	--				
5. Expected Improvement	6.94	2.11	1.00-11.00	-.04	.15**	-.10	.09	--			
6. Likelihood to Begin Treatment	2.36	1.72	0.00-5.00	.05	-.06	.17**	-.11	.24**	--		
7. Post TOA	3.94	1.39	1.00-6.00	-.12	.57**	-.23**	.17**	.21**	.02	(.94)	
8. Post GAD-7	13.13	5.26	0.00-21.00	.48**	-.25**	.93**	-.11	-.11	.18**	-.23**	(.90)

Note. $N = 286$. PSWQ (Penn State Worry Questionnaire) Item 15: "I worry all the time"; TOA: theories of anxiety (higher scores indicate greater growth mindset endorsement); TOA Item: "You have a certain amount of anxiety and you really cannot do much to change it" (reverse scored such that higher scores indicate greater growth mindset endorsement); GAD: Generalized Anxiety Disorder; Hypothetical Treatment Choice scored Medication = 0, Therapy = 1. Cronbach's alpha values for measures with more than 1 item are listed on the diagonal in parentheses. *p<.05, **p<.01

Table 3. Means and standard deviations for the two conditions in Study 1

		Control N =149			Anxiety Mindset N=137	
Variable	*Range*	*M*	*SD*	*Range*	*M*	*SD*
Prescreen PSWQ Item 15	4-5	4.39	0.49	4-5	4.43	.50
Prescreen TOA item	1-6	2.77	1.46	1-6	3.01	1.55
Prescreen GAD-7	0-21	12.86	5.26	0-21	13.34	5.17
Hypothetical Treatment Choice	0-1	0.77	0.43	0-1	0.82	0.39
Expected Improvement	1-11	6.71	2.16	2-11	7.19	2.03
Likelihood to Begin Treatment	0-5	2.17	1.71	0-5	2.60	1.71
Post TOA	1-6	3.50	1.33	1-6	4.44	1.27
Post GAD-7	1-21	13.18	5.28	0-21	13.07	5.25

Note. PSWQ (Penn State Worry Questionnaire) Item 15: "I worry all the time"; TOA: theories of anxiety (higher scores indicate greater growth mindset endorsement); TOA Item: "You have a certain amount of anxiety and you really cannot do much to change it" (reverse scored such that higher scores indicate greater growth mindset endorsement); GAD: Generalized Anxiety Disorder; Hypothetical Treatment Choice scored Medication = 0, Therapy = 1.

Table 4. Means, standard deviations, and bivariate correlations among variables across the entire sample in Study 2

	M	*SD*	*Range*	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Prescreen PSWQ	66.28	5.99	50.00-80.00	(.75)								
2. Prescreen TOA	3.85	1.16	1.00-6.00	-.28*	(.91)							
3. Prescreen GAD-7	8.43	4.11	0.00-19.00	.34**	-.25*	(.81)						
4. Hypothetical Treatment Choice	0.76	0.43	0.00-1.00	-.02	.36**	-.09	--					
5. Expected Improvement	7.28	1.81	3.00-11.00	.03	.26*	-.08	.12	--				
6. Likelihood to Begin Treatment	1.63	1.50	0.00-5.00	.08	.13	.06	.03	.28*	--			
7. Post PSWQ	65.49	7.82	34.00-80.00	.20	-.08	.23*	.10	-.15	-.06	(.84)		
8. Post TOA	3.92	1.30	1.00-6.00	.06	.36**	-.17	.18	.17	.04	-.10	(.94)	
9. Post GAD-7	10.11	4.19	3.00-18.00	.25*	-.09	.48**	.08	-.12	-.06	.42**	-.18	(.80)

*Note. N*s = 73-75. PSWQ: Penn State Worry Questionnaire; TOA: theories of anxiety (higher scores indicate greater growth mindset endorsement); GAD: Generalized Anxiety Disorder; Hypothetical Treatment Choice scored Medication = 0, Therapy = 1. Cronbach's alpha values for measures with more than 1 item are listed on the diagonal in parentheses. *p<.05, **p<.01

Table 5. Means (With Standard Deviations) for Self-Report Measures for the two conditions in Study 2

	Control N=35						Anxiety Mindset N=39					
	Prescreen			Post-Intervention			Prescreen			Post-Intervention		
Variable	*Range*	*M*	*SD*	*Range*	*M*	*SD*	*Range*	*M*	*SD*	*Range*	*M*	*SD*
TOA	1-6	3.04	1.22	1-6	3.54	1.39	1-5	3.24	1.11	1.50-6	4.28	1.13
PSWQ	50-80	65.83	6.27	53-60	66.49	6.97	51-79	66.66	5.84	34-78	64.44	8.51
GAD-7	0-16	8.57	3.99	3-18	10.37	4.54	1-19	8.49	4.27	3-18	9.79	3.92
% Choosing Therapy	--	--	--	0-100%	71%	--	--	--	--	0-100%	79%	--
Expected Improvement from Treatment	--	--	--	4-11	7.60	1.50	--	--	--	3-11	7.05	2.03
Likelihood to Begin Treatment	--	--	--	0-5	1.74	1.50	--	--	--	0-5	1.54	1.54

Note: TOA: Implicit theories of anxiety (higher scores indicate higher growth-mindset endorsement). PSWQ: Penn State Worry Questionnaire; GAD-7: Generalized Anxiety Disorder-7 questionnaire. Measures not collected at the prescreen assessment are indicated with "--".

Table 6. Means (With Standard Deviations) for Stimulus-Preceding Negativity (SPN) (μV)

	Control								Anxiety Mindset							
	Time 1				Time 2				Time 1				Time 2			
	Look		Reappraise		Look		Reappraise		Look		Reappraise		Look		Reappraise	
ERP Window	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
200-2300ms	1.67	3.22	0.88	3.68	0.27	2.70	0.87	2.88	0.56	3.89	0.06	3.41	0.86	2.79	-0.90	3.66
2300-3000ms	3.02	3.92	2.85	5.63	0.88	4.27	1.74	5.14	0.38	5.00	0.28	3.70	1.93	3.18	-0.44	5.39

Note: Values are in microvolts (μV) and represent the pooled average of six scalp electrodes: F1, Fz, F2, FC1, FCz, and FC2 time-locked to the cue. ms: millisecond.

Table 7. ANOVA results for the regulation effects on the SPN

Effect	*F*	df	*p*	η^2_p
300-2000ms				
Trial Type	1.52	1, 54	.222	.027
Time	2.58	1, 54	.114	.046
Condition	2.05	1, 54	.158	.037
Trial Type x Time	0.01	1, 54	.938	<.001
Trial Type x Condition	0.19	1, 54	.662	.004
Time x Condition	1.84	1, 54	.180	.033
Trial Type x Time x Condition	2.66	1, 54	.109	.047
2300-3000ms				
Trial Type	1.17	1, 54	.284	.021
Time	0.95	1, 54	.334	.017
Condition	3.89	1, 54	.054	.067
Trial Type x Time	0.28	1, 54	.599	.005
Trial Type x Condition	3.34	1, 54	.073	.058
Time x Condition	2.98	1, 54	.090	.052
Trial Type x Time x Condition	1.93	1, 54	.170	.035

Note. Bolded values indicate statistically significant effects at $p < .05$

Table 8. Means (With Standard Deviations) for the Frontal Late Positive Potential (LPP) (μV)

	Control								Anxiety Mindset							
	Time 1				Time 2				Time 1				Time 2			
	Look		Reappraise		Look		Reappraise		Look		Reappraise		Look		Reappraise	
ERP Window	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
400-700ms	-4.77	7.85	-4.39	7.28	-5.72	5.86	-5.50	6.83	-3.61	4.99	-3.60	4.47	-7.36	5.64	-5.65	5.65
700-1000ms	0.67	7.24	1.51	6.74	0.17	4.89	0.47	6.29	1.43	4.53	1.62	4.27	-1.43	4.41	-0.30	4.63
1000-2000ms	2.56	6.16	3.44	3.95	0.81	3.23	2.13	4.98	2.44	5.09	3.44	3.95	-0.79	3.71	1.27	4.72
2000-3000ms	2.31	5.68	0.82	5.77	1.26	3.90	0.71	4.67	3.09	4.63	3.41	3.95	-0.12	3.80	1.54	5.98
3000-4000ms	2.02	5.22	-0.68	6.96	1.50	3.63	-0.98	4.19	3.04	4.60	2.50	4.11	-1.18	5.08	0.80	5.52
4000-5000ms	1.42	5.77	-0.83	7.03	1.43	5.02	-1.84	4.28	2.52	5.05	1.70	4.23	-1.01	5.25	0.63	5.06
5000-6000ms	0.85	5.20	-1.08	7.77	1.55	5.53	-1.57	5.93	2.94	5.91	1.05	4.22	-0.25	4.57	-0.30	5.27

Note. Values are in microvolts (μV) and represent the pooled average of six scalp electrodes – F1, Fz, F2, FC1, FCz, and FC2 time-locked to the stimulus. ms: millisecond.

Table 9. Means (With Standard Deviations) for the Parietal Late Positive Potential (LPP) (μV)

	Control								Anxiety Mindset							
	Time 1				Time 2				Time 1				Time 2			
	Look		Reappraise		Look		Reappraise		Look		Reappraise		Look		Reappraise	
ERP Window	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
400-700ms	4.29	5.75	4.44	6.29	4.03	4.35	3.81	5.28	5.84	5.38	6.10	5.16	3.87	5.89	5.23	5.25
700-1000ms	3.01	5.44	2.86	6.02	3.51	3.88	2.21	5.09	4.39	4.84	3.99	4.86	2.87	4.90	3.32	4.66
1000-2000ms	1.63	4.96	-0.55	5.12	1.61	3.51	0.56	4.70	2.02	4.91	2.30	4.65	0.07	4.42	0.80	4.80
2000-3000ms	0.79	5.33	-2.11	5.45	0.92	4.45	-1.61	4.63	2.03	5.46	1.88	4.06	-0.23	4.17	-0.13	5.49
3000-4000ms	0.79	5.37	-2.20	6.29	1.23	4.85	-2.60	3.71	2.37	4.92	1.86	4.30	-1.12	5.19	-1.42	4.66
4000-5000ms	1.07	5.23	-1.52	6.16	1.98	5.97	-2.87	4.00	2.58	5.20	2.21	4.15	-0.18	5.22	0.66	4.83
5000-6000ms	0.72	4.95	-0.95	7.05	2.20	5.64	-2.17	5.03	3.16	5.70	2.65	3.61	0.59	4.95	0.04	4.98

Note: Values are in microvolts (μV) and represent the pooled average of five scalp electrodes: CPz, P1, Pz, P2, and POz time-locked to the stimulus. ms: millisecond.

Table 10. ANOVA results for regulation effects on the frontal LPP

Effect	*F*	df	*p*	η^2_p
400-1000ms				
Time	**10.866**	**1, 54**	**.002**	**.168**
Time x Condition	2.634	1, 54	.110	.047
ERP Window	**230.905**	**1, 54**	**<.001**	**.810**
ERP Window x Condition	0.316	1, 54	.576	.006
Trial Type	3.802	1, 54	.056	.066
Trial Type x Condition	0.285	1, 54	.595	.005
Time x ERP Window	2.763	1, 54	.102	.049
Time x ERP Window x Condition	0.295	1, 54	.589	.005
Time x Trial Type	0.558	1, 54	.458	.010
Time x Trial Type x Condition	1.626	1, 54	.208	.029
ERP Window x Trial Type	0.019	1, 54	.892	<.001
ERP Window x Trial Type x Condition	0.749	1, 54	.391	.014
Time x ERP Window x Trial Type	1.651	1, 54	.204	.030
Time x ERP Window x Trial Type x Condition	0.172	1, 54	.680	.003
Condition	0.016	1, 54	.900	<.001
1-6s				
Time	**7.183**	**1, 54**	**.010**	**.117**
Time x Condition	3.745	1, 54	.058	.065
ERP Window	**7.542**	**4, 216**	**.001**	**.123**
ERP Window x Condition	1.155	4, 216	.316	.021
Trial Type	2.205	1, 54	.143	.039
Trial Type x Condition	**7.912**	**1, 54**	**.007**	**.128**
Time x ERP Window	0.615	4, 216	.548	.011
Time x ERP Window x Condition	0.239	4, 216	.796	.004
Time x Trial Type	1.760	1,54	.090	.032
Time x Trial Type x Condition	1.080	1,54	.303	.020
ERP Window x Trial Type	**11.654**	**4, 216**	**<.001**	**.178**
ERP Window x Trial Type x Condition	2.312	4, 216	.094	.041
Time x ERP Window x Trial Type	0.787	4, 216	.469	.014
Time x ERP Window x Trial Type x Condition	2.795	4, 216	.059	.049
Condition	0.457	1,54	.502	.008

Note. Bolded values indicate statistically significant effects at $p < .05$

Table 11. ANOVA results for regulation effects on the parietal LPP

Effect	*F*	df	*p*	η^2_p
400-1000ms				
Time	2.02	1, 54	.161	.036
Time x Condition	0.874	1, 54	.354	.016
ERP Window	**27.733**	**1, 54**	**<.001**	**.339**
ERP Window x Condition	0.460	1, 54	.501	.008
Trial Type	0.003	1, 54	.954	<.001
Trial Type x Condition	1.593	1, 54	.212	.029
Time x ERP Window	1.739	1, 54	.193	.031
Time x ERP Window x Condition	0.007	1, 54	.935	<.001
Time x Trial Type	0.030	1, 54	.863	.001
Time x Trial Type x Condition	1.964	1, 54	.167	.035
ERP Window x Trial Type	**9.893**	**1, 54**	**.003**	**.155**
ERP Window x Trial Type x Condition	0.047	1, 54	.830	.001
Time x ERP Window x Trial Type	1.759	1, 54	.190	.032
Time x ERP Window x Trial Type x Condition	0.464	1, 54	.499	.009
Condition	0.638	1, 54	.428	.012
1-6s				
Time	**4.077**	**1, 54**	**.048**	**.070**
Time x Condition	**5.374**	**1, 54**	**.024**	**.091**
ERP Window	**4.556**	**4, 216**	**.015**	**.078**
ERP Window x Condition	1.484	4, 216	.232	.027
Trial Type	**9.938**	**1, 54**	**.003**	**.155**
Trial Type x Condition	**11.008**	**1, 54**	**.002**	**.169**
Time x ERP Window	1.170	4, 216	.317	.021
Time x ERP Window x Condition	0.372	4, 216	.708	.007
Time x Trial Type	0.022	1, 54	.883	<.001
Time x Trial Type x Condition	1.153	1, 54	.288	.021
ERP Window x Trial Type	**3.682**	**4, 216**	**.018**	**.064**
ERP Window x Trial Type x Condition	2.185	4, 216	.102	.039
Time x ERP Window x Trial Type	2.645	4, 216	.075	.047
Time x ERP Window x Trial Type x Condition	3.024	4, 216	.052	.053
Condition	1.954	1, 54	.168	.035

Note. Bolded values indicate statistically significant effects at $p < .05$

Table 12. Emotion regulation task ratings

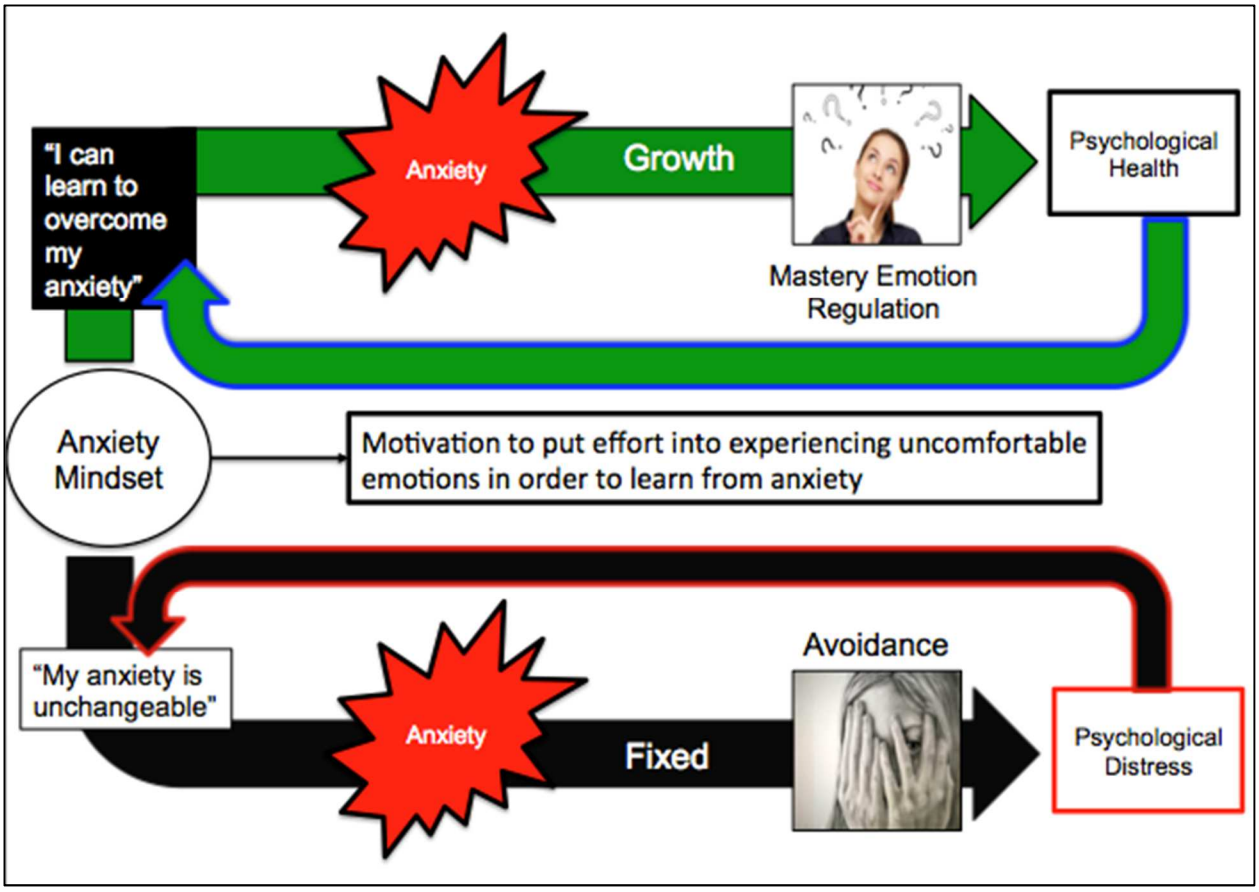
	Control		Anxiety Mindset		Analysis	
Time 1	*M*	*SD*	*M*	*SD*	*t*	*p*
Understand						
Look- Neutral	6.21	0.96	6.54	1.17	1.13	.265
Look- Negative	**6.39**	**0.79**	**6.79**	**0.50**	**2.23**	**.030**
Reappraise-Negative	**6.29**	**0.90**	**6.71**	**0.60**	**2.10**	**.040**
Difficulty						
Look- Neutral	2.36	1.47	2.50	1.82	0.32	.748
Look- Negative	2.50	1.17	2.32	1.47	0.50	.617
Reappraise-Negative	4.25	1.18	4.00	1.76	0.62	.535
Effort						
Look- Neutral	2.29	1.49	2.29	1.61	0.00	1.00
Look- Negative	2.57	1.53	1.93	0.94	1.90	.063
Reappraise-Negative	4.39	1.55	4.39	1.63	0.00	1.00
Attention						
Look- Neutral	4.50	1.71	4.50	1.53	0.00	1.00
Look- Negative	5.75	1.24	5.61	1.23	0.43	.666
Reappraise-Negative	6.39	0.96	6.39	0.83	0.00	1.00
Emotional Reaction						
Look- Neutral	2.21	1.52	2.36	1.34	0.37	.711
Look- Negative	5.21	1.17	5.04	1.35	0.53	.598
Reappraise-Negative	5.43	1.23	5.43	1.10	0.00	1.00
Time 2						
Understand						
Look- Neutral	6.54	0.74	6.79	0.63	1.36	.181
Look- Negative	6.64	0.62	6.82	0.48	1.21	.233
Reappraise-Negative	6.64	0.62	6.86	0.36	1.58	.119
Difficulty						
Look- Neutral	2.89	2.10	2.61	1.73	0.56	.580
Look- Negative	3.00	1.96	2.54	1.53	0.99	.328
Reappraise-Negative	4.57	1.60	3.89	1.64	1.57	.123
Effort						
Look- Neutral	2.82	1.79	2.25	1.60	1.26	.213
Look- Negative	2.82	1.72	2.04	1.17	2.00	.051
Reappraise-Negative	4.43	1.93	4.04	1.82	0.78	.436
Attention						
Look- Neutral	3.71	2.07	4.18	1.95	0.87	.391
Look- Negative	5.18	1.39	5.04	1.62	0.35	.725
Reappraise-Negative	5.96	1.11	5.86	1.18	0.35	.727
Emotional Reaction						
Look- Neutral	2.00	1.52	2.14	1.35	0.37	.711
Look- Negative	4.18	1.44	4.43	1.57	0.62	.538
Reappraise-Negative	4.89	1.20	5.21	1.17	1.02	.313

Note. Bolded rows indicate statistically significant effects at $p < .05$

APPENDIX B

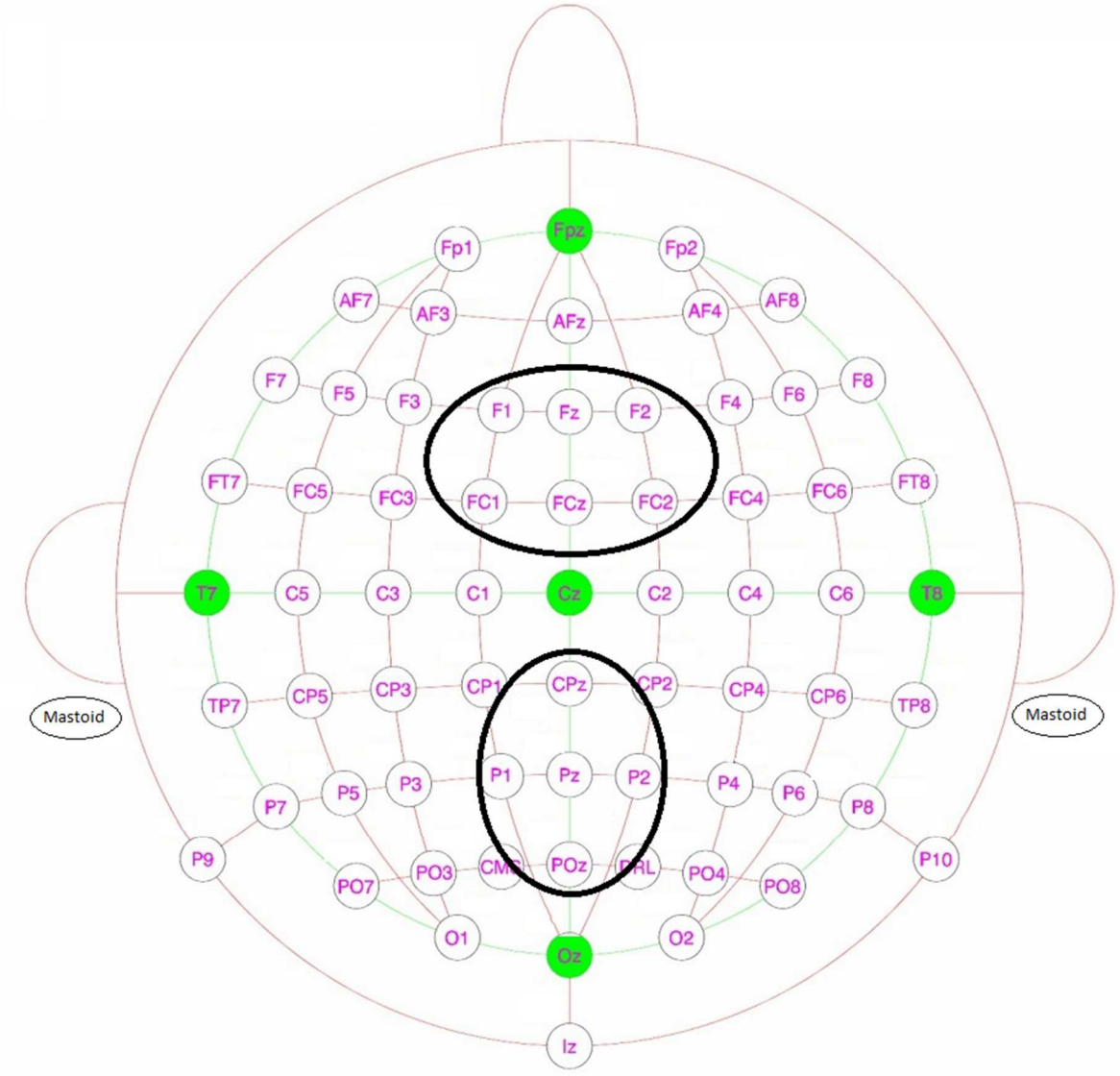
FIGURES

Figure 1. Simple conceptual model linking the anxiety mindset to emotion regulation, motivation, and psychological outcomes



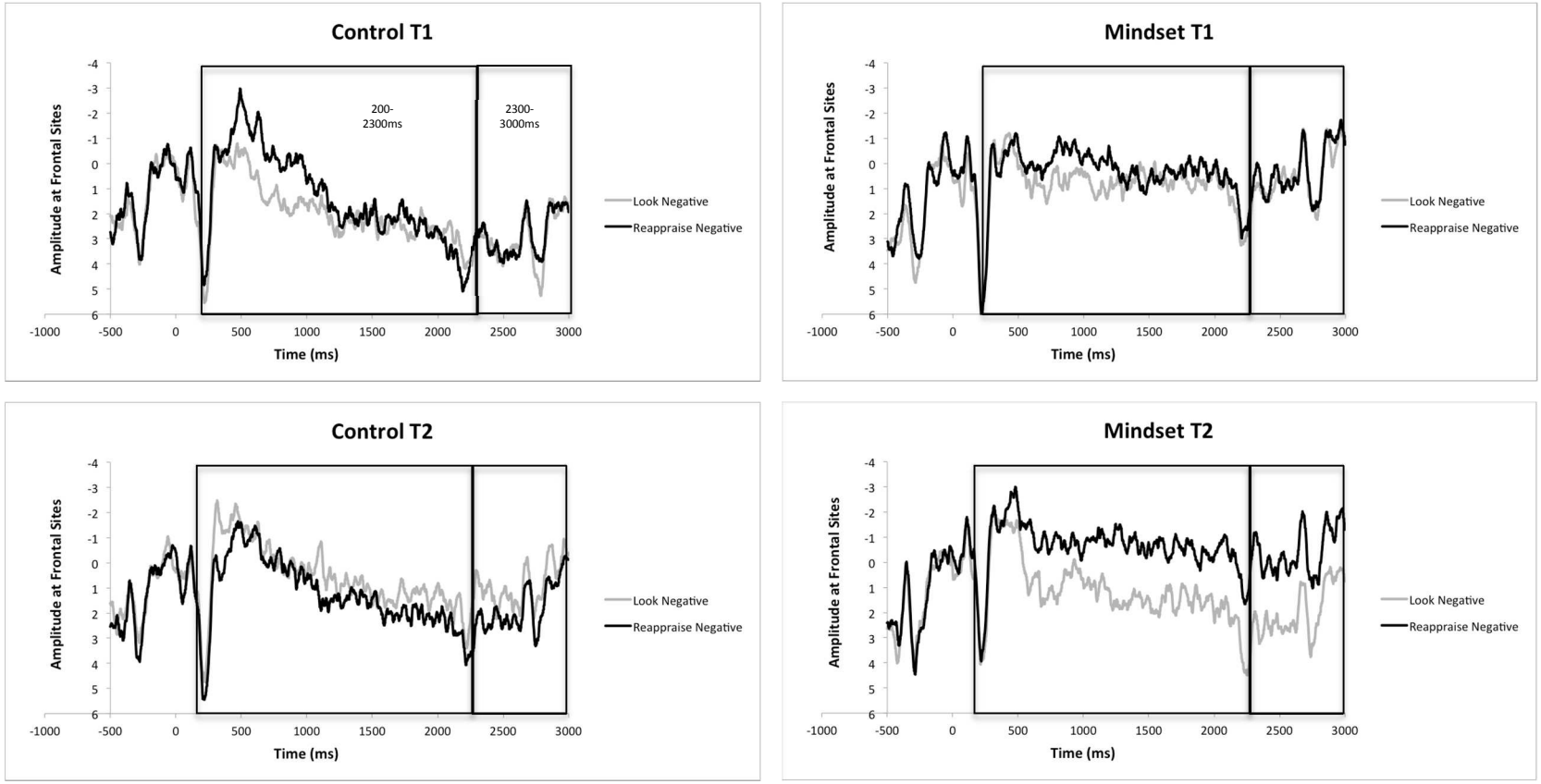
Note. The primary concept is that growth anxiety mindset endorsement promotes a motivation to put effort into experiencing uncomfortable emotions in order to learn from anxiety. This will, on average, lead to greater use of emotion regulation strategies that are adaptive and that promote emotion experience (e.g., therapy), rather than avoidance. Backwards arrows indicate that health or distress reinforces growth or fixed beliefs. Also note that growth and fixed arrows represent averages of individuals endorsing more of a growth or more of a fixed mindset (that is, people are not "fixed minded" or "growth minded", but vary on the degree to which they endorse a growth mindset).

Figure 2. EEG setup and electrode pool depictions



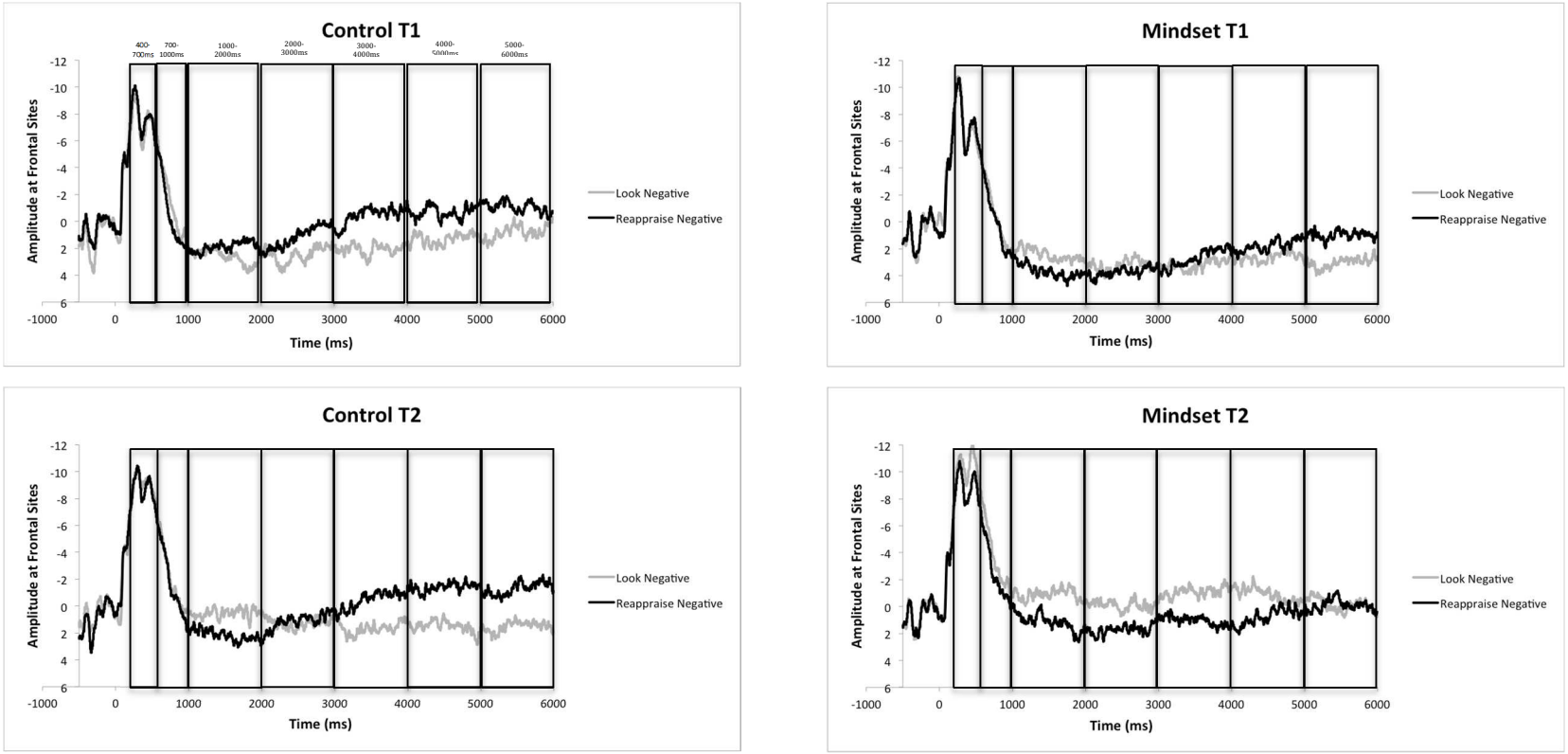
Note. Schematic of BioSemi EEG cap, electrode placement, and electrode pools used in statistical analyses. Nose is plotted up. Additional electrodes were placed behind each ear on the mastoid process. The top bolded circle indicates the electrodes used in the SPN and frontal LPP analyses (F1, Fz, F2, FC1, FCz, and FC2). The bottom bolded circle indicates the electrodes used in the parietal LPP analyses (CPz, P1, Pz, P2, and POz). Note also the CMS and DRL electrodes, which formed the ground during data collection. Modified from https://www.biosemi.com/headcap.htm

Figure 3. SPN waveforms



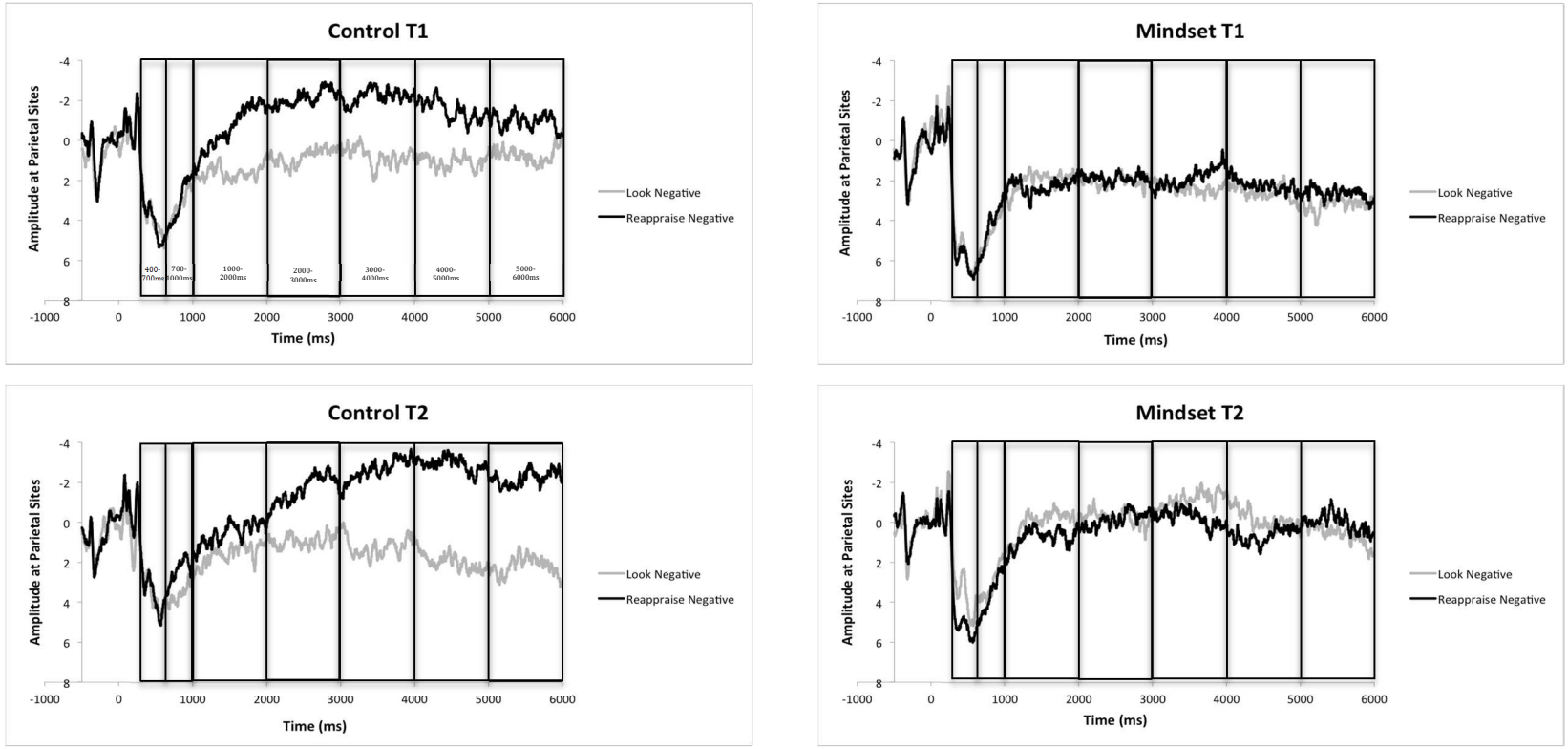
*Note***.** Cue-locked grand average waveforms depicting the SPN. Grand average waveforms are computed by averaging each participant's average waveform, and then averaging the averages. Here, Time 0 represents the onset of the cue ("Look-Negative" or "Reappraise-Negative"). Note that negative amplitude values are plotted up on the y-axis, as is custom in ERP research. Boxes illustrate ERP windows used in the analyses: 200-2300ms and 2300-3000ms windows were used in the analyses of the SPN.

Figure 4. Frontal LPP waveforms



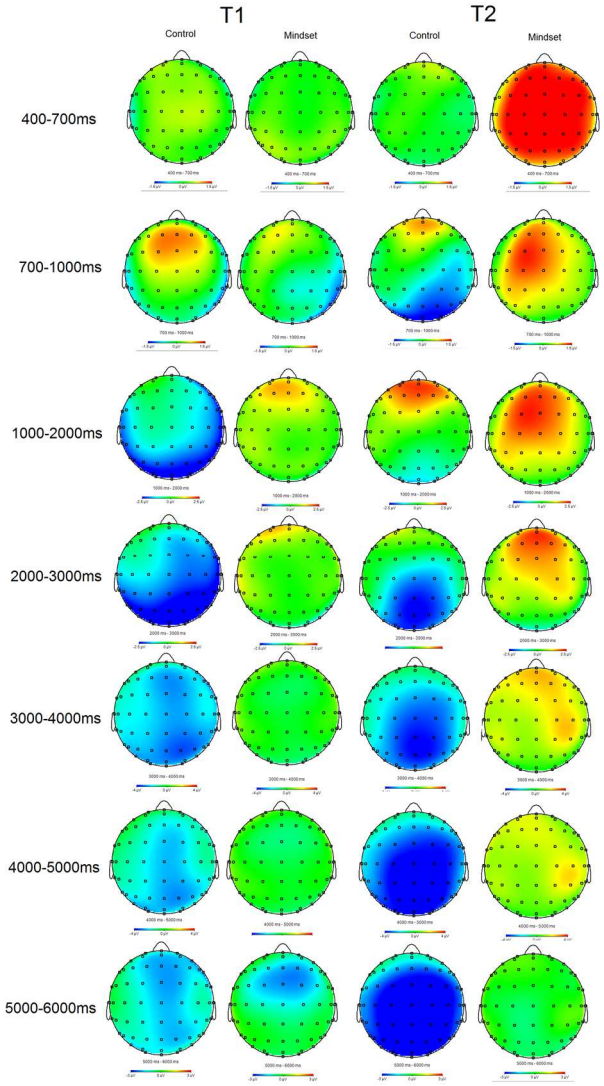
Note. Stimulus-locked grand average waveforms depicting the frontal LPP. Grand average waveforms are computed by averaging each participant's average waveform, and then averaging the averages. Here, Time 0 represents the onset of the stimulus. Note that negative amplitude values are plotted up on the y-axis, as is custom in ERP research. Boxes illustrate ERP windows used in the analyses: 400-700ms, 700-1000ms, 1000-2000ms, 2000-3000ms, 3000-4000ms, 4000-5000ms, and 5000-6000ms.

Figure 5. Parietal LPP waveforms



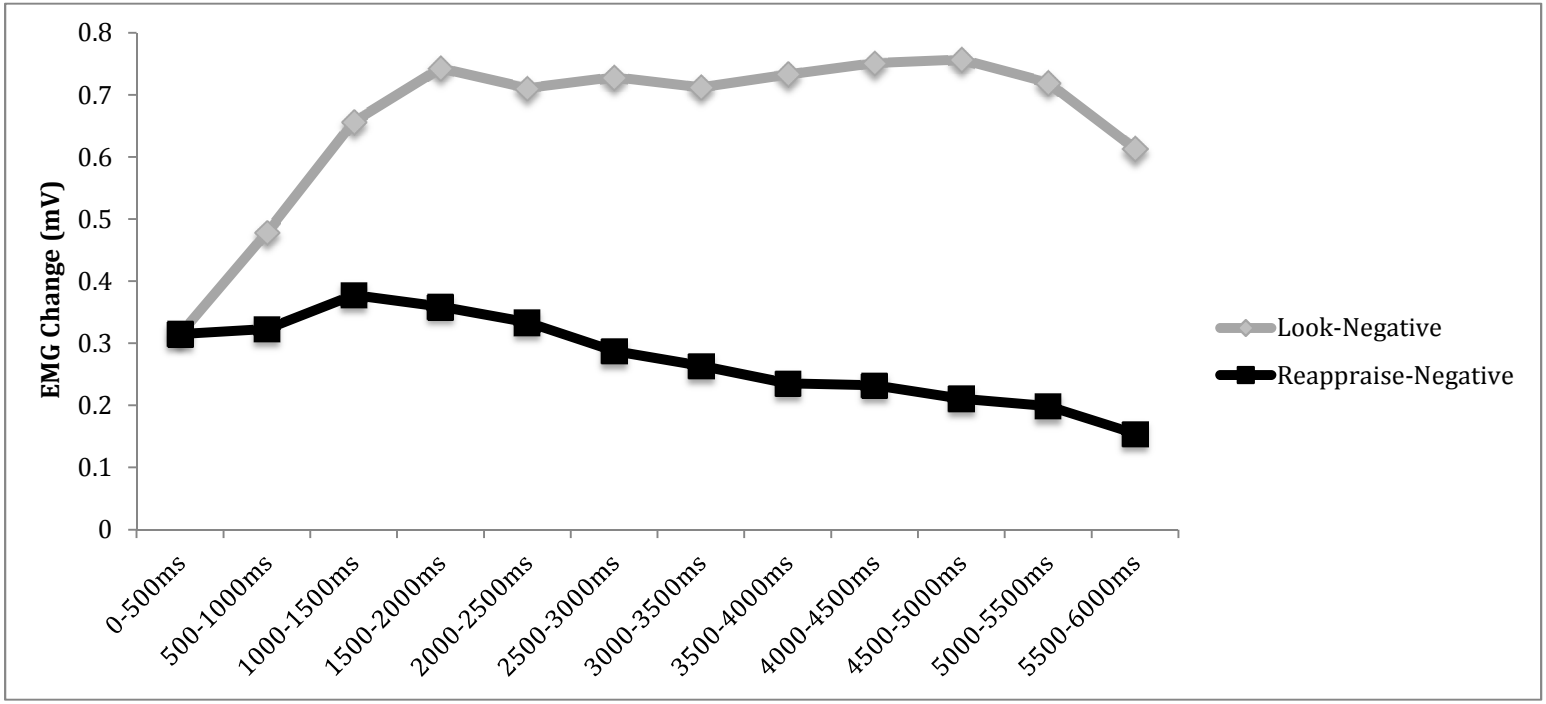
Note. Stimulus-locked grand average waveforms depicting the parietal LPP. Grand average waveforms are computed by averaging each participant's average waveform, and then averaging the averages. Here, Time 0 represents the onset of the stimulus. Note that negative amplitude values are plotted up on the y-axis, as is custom in ERP research. Boxes illustrate ERP windows used in the analyses: 400-700ms, 700-1000ms, 1000-2000ms, 2000-3000ms, 3000-4000ms, 4000-5000ms, and 5000-6000ms.

Figure 6. Scalp maps depicting topography of Reappraise minus Look-Negative stimuli



Note. Scalp maps depicting the difference wave (Reappraise minus Look-Negative). Warmer colors indicate more positivity for reappraise versus look trials; cooler colors indicate more positivity for look trials.

Figure 7. EMG data for Look-Negative and Reappraise-Negative trials averaged across Time 1 and Time 2



*Note***.** Stimulus-locked EMG data depicting averaged EMG change (mV) across all participants in both conditions across the time windows. Time 0 here represents stimulus onset.

REFERENCES

REFERENCES

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